Multiple Trauma Part II: Triage and initial resuscitation in trauma

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2026 Update 2026

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Intended Learning Outcomes

Multiple Trauma Part II: Triage and initial resuscitation in trauma

1. Describe the principles of triaging trauma patients
2. Summarize the key steps in assessing trauma patients
3. Describe the principles of resuscitation in trauma patients
4. Describe principles of haemodynamic support in major trauma patients
5. Identify the key management principles of caring for patients in traumatic cardiac arrest
6. Describe indications and practices of massive transfusion in major trauma patients
7. Describe the concepts of coagulation management in major trauma patients

eModule Information

Relevant Competencies from CoBaTrICE

Multiple Trauma Part II: Triage and initial resuscitation in trauma

- **1.1** Adopts a structured and timely approach to the recognition, assessment and stabilisation of the acutely ill patient with disordered physiology.
- **1.5** Assesses and provides initial management of the trauma patient
- **2.1** Integrates clinical findings with laboratory investigations to form a differential diagnosis
- **3.1** Manages the care of the critically ill patient with specific acute medical conditions
- **3.4** Recognises and manages the patient with, or at risk of, acute kidney injury
- **6.1** Manages the pre- and post-operative care of the high-risk surgical patient
- **6.5** Manages the pre- and post-operative care of the trauma patient under supervision

1. Trauma triage

Trauma triage systems are structured protocols used by emergency medical services (EMS), emergency departments, and trauma centers to quickly assess the severity of injuries and ensure that patients receive care at the appropriate level facility. These systems are critical in optimizing outcomes, minimizing delays, and conserving resources by directing patients to the most suitable trauma care setting based on injury severity.

1. 1. Purpose and Importance

The overarching goal of trauma triage is to **"get the right patient to the right place at the right time."** Triage systems are designed to distinguish between patients with minor injuries who can be safely treated in non-specialized hospitals and those with major trauma who require the expertise and resources of a designated trauma center. Inaccurate triage – either undertriage (missing a severe injury) or overtriage (overestimating severity) – can have significant consequences. Undertriage may result in preventable morbidity or mortality, while overtriage can overburden trauma centers and waste valuable resources.

1. 2. Prehospital and Hospital Triage

Triage occurs at two main stages:

1. **Prehospital triage** is guided by national or regional criteria. ATLS 11 refers to the 2021 **National Guideline for the Field Triage of Injured Patients**, which classifies high-risk ("RED") and moderate-risk ("YELLOW") criteria (Figure 1). These criteria incorporate:
    - Physiologic status (e.g., abnormal RR, SpO_2 <90%, inability to follow commands)
    - Key injury patterns (e.g., penetrating injuries to head/neck/torso, pelvic fracture, chest wall instability)
    - Mechanism of injury (e.g., high-risk motor vehicle impact, pedestrian struck, falls >10 ft)
    - EMS judgment and special considerations (e.g., anticoagulant use, pregnancy, older adults, suspected abuse)
2. **Hospital triage** occurs upon ED arrival and reassesses the need for trauma team activation, definitive care, or transfer to a higher-level trauma center.

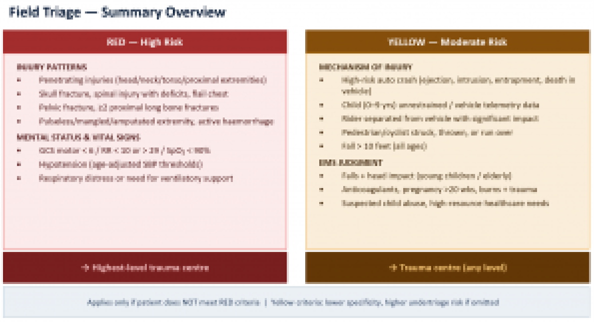

Figure 1: Field Triage - Summary Overview.

1. 3. Triage Tools and Scores

Several **trauma scoring systems** assist with triage:

- **Revised Trauma Score (RTS)**: Uses GCS, systolic BP, and respiratory rate.
- **TRISS (Trauma and Injury Severity Score)**: Combines RTS, Injury Severity Score (ISS), and age to predict survival.
- **START** (Simple Triage and Rapid Treatment): Used in mass casualty incidents (MCIs).

The TRENAU ABC Triage System

A notable example of a regional triage tool is the **TRENAU ABC system** used in the Rhône-Alpes region in France and adopted in several European trauma networks. It provides a **graded, dynamic classification** of trauma patients and guides care priorities and transport decisions:

- **A – Absolute Criteria:** Major trauma with life-threatening injuries (e.g., GCS ≤8, systolic BP <90 mmHg, penetrating trauma to head/torso). Immediate activation and transport to a level I trauma center.
- **B – Borderline Criteria:** Serious injuries without immediate threat to life but requiring advanced care (e.g., pelvic fracture, severe limb injuries, multiple rib fractures). Transport to a trauma center, depending on local logistics and proximity.
- **C – Conditional Criteria:** Moderate injury patterns that may evolve or are high-risk due to comorbidities or age (e.g., elderly with head trauma). Requires observation or reassessment; transfer if condition worsens.

The TRENAU system supports **graduated decision-making**, especially in regions with varying access to trauma resources. It aligns triage with **clinical presentation, risk stratification, and local capabilities**, emphasizing **dynamic reassessment**, which is essential in the early phase of trauma care.

Recent prospective data from Italy show that the TRENAU triage system substantially reduces overtriage compared with the ACS-COT scheme (23% vs 59%) while maintaining an acceptable undertriage rate of 3.9%. TRENAU performs particularly well in mature, inclusive trauma systems where graded trauma centers (levels I–III) allow safe redistribution of patients. Although its sensitivity is lower than ACS-COT, most undertriaged patients would still be directed to an intermediate-level trauma center, preserving safety. These findings support the use of TRENAU as a more resource-efficient triage tool within European trauma networks.

1. 4. Challenges and Future Directions

Despite standardized tools, triage remains complex. Provider variability, atypical presentations (e.g., in elderly), and environmental factors can affect decision-making. Emerging solutions include m**achine learning algorithms, wearable monitoring**, and **mobile triage apps** to support real-time, data-informed triage decisions.
The integration of **region-specific tools like TRENAU ABC** with national guidelines helps tailor triage to local trauma system design, balancing precision, safety, and efficiency.

Triage in older adults presents additional challenges that merit explicit consideration. Geriatric trauma patients are frequently under-triaged, as subtle physiological changes and low-energy mechanisms may mask severe injury. Older adults may not exhibit typical compensatory responses to shock, and an apparently normal systolic blood pressure may represent hypotension. Frailty is present in at least one-third of older adults and significantly worsens outcomes, requiring closer monitoring and a lower threshold for trauma center referral. Because inadequate triage contributes to higher mortality in this population, modern triage systems increasingly incorporate age-specific modifiers and frailty assessments.

In Text References

(Gianola et al. 2021; American College of 2025)

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2. Management principles

2. 1. Anticipation and preparation

Like the prehospital phase, in-hospital trauma care is under intense **time pressure. Delays**, particularly in managing **haemorrhage** and **neurological injury**, can be fatal. This is why trauma management – especially in trauma centres – requires **multidisciplinary and multiprofessional coordination**. Few conditions mobilize so many healthcare professionals so rapidly and unpredictably.

Thus, care should be delivered in a dedicated location, by a trained and cohesive team.

Management ideally begins **before the patient's arrival**. A structured pre-arrival team activation, with early role assignment and rapid preparation of equipment and resources, improves coordination and reduces delays in the resuscitation phase.

Regardless of transport method, it’s crucial that the receiving team receives **minimum essential information** (age, Glasgow Coma Scale, mechanism – penetrating or not – presence of shock, blood pressure, heart rate) to **prepare in advance**.

In unscheduled or self-presenting cases, the team may be **caught off guard** and must **adapt quickly**.

This preparation phase typically involves a brief team huddle that allows:

- Team awareness: All personnel (core team and partners like surgeons, radiologists, anaesthetists) are informed
- Task assignment: Roles and responsibilities are clarified
- Scenario planning: Based on initial data, potential deterioration is anticipated
- Resource mobilization: Equipment for airway management, vascular access, blood products, operating theater/interventional radiology availability
    - For blood product readiness, prior coordination with the French Blood Establishment (EFS) and protocols is needed
- Psychological readiness: Briefing improves team resilience in unpredictable situations

For example:

- The TRENAU network developed an A/B/C triage scale to stratify patients during emergency call regulation.
- The RED FLAG score helps predict patients likely to require massive transfusion due to acute hemorrhage .

Cognitive aids, protocols, and checklists incorporating this graded response are essential to standardize team preparation and resource mobilization. These protocols foster collective adoption and generate shared mental models and communication frameworks, which are critical for team cohesion and performance.

2. 2. Reception phase

2. 2. 1. Handover and patient transfer

The handover by the **prehospital team** is a critical moment — not only for the **clinical information transfer** but also because it marks the **transition of responsibility** between the prehospital and hospital teams. This interface is a **vulnerable moment** for the patient, particularly if they are clinically unstable. It is essential for **both teams to pause** and allow time for the handover, ensuring **all team members hear the same information**.

The handover must be concise and structured. The (AT)MIST(ER) format is recommended: ATLS describes the MIST mnemonic (Mechanism, Injuries, Signs, Treatment), while NICE major trauma guidelines endorse the extended ATMISTER structure (Age, Time, Mechanism, Injuries, Signs, Treatment, Estimated arrival time, Requirements). Regardless of the variant used, the aim is a brief, standardized, and audible transfer of critical information.

The **transfer of the patient** onto the trauma bay stretcher and connection to monitoring devices (e.g., cardiac monitor) can be carried out in several ways, depending on **prehospital immobilization** and the **layout of the trauma bay**. The key is that the **receiving team uses the same standardized method** each time to maintain **workflow consistency**.

2. 2. 2. Team Organization and Communication

Team structure and communication in the trauma bay are critical to effective trauma care. While the full scope of human and organizational factors is beyond this document, Figure 2 summarizes key principles.
Essential steps include:

- **Identifying the trauma team leader** and team members
- **Assigning roles clearly**
- **Limiting personnel** to essential staff
- Using **closed-loop communication**
- **Avoiding task interruptions**
- **Employing cognitive aids** when needed
- Performing **regular situation updates (time-outs)** — brief (≤2 min), repeatable if needed

The **trauma leader** should ideally remain **physically withdrawn** from procedures to maintain **a broad situational overview**, thereby avoiding **task fixation (tunnel vision)**.

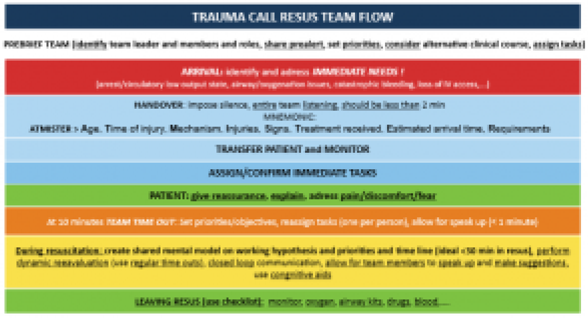

Figure 2: Key principles

2. 2. 3. Immediate Priorities (xABCDE)

Handover and initial clinical assessment should identify **life-threatening conditions** needing **immediate intervention** (e.g. tension pneumothorax, uncontrolled external haemorrhage, respiratory failure).
The **modified ATLS algorithm** (https://www.facs.org/quality-programs/trauma/education/advanced-trauma-life-support/atls-11/) guides this phase:

- **X** (control of external exsanguinating hemorrhage): Identify and control visible, life-threatening bleeding using **rapid haemostatic measures** such as direct pressure, wound packing with haemostatic dressings, limb tourniquets, or junctional haemorrhage control devices.
- **A (Airway)**: Check for obstruction (blood, vomit, debris), verify endotracheal tube or supraglottic airway, and use **end-tidal CO_2 monitoring**.
- **B (Breathing)**: Assess ventilation effectiveness, chest deformities, subcutaneous emphysema, thoracic asymmetry.
- **C (Circulation)**: Evaluate haemodynamic — radial pulse, heart rate, systolic pressure, capillary refill, extremity warmth/colour, peripheral pulses.
- **D (Disability)**: Assess neurological status via **Glasgow Coma Scale (GCS)** and pupil exam (anisocoria, mydriasis).
- **E (Extended)**: Examine abdomen, pelvis, extremities, or any **other trauma signs** detectable on clinical exam.

> **Note**
>
> In traumatic peri-arrest or arrest states, recent evidence supports prioritising circulation-first (C-A-B) when hypovolaemia or obstructive causes are likely, as airway manoeuvres may worsen hypotension and delay haemorrhage control.

Point-of-care ultrasound (POCUS) may support the primary survey by rapidly identifying pericardial tamponade, loss of cardiac motion, pneumothorax, or intra-abdominal free fluid. According to the ERC 2025 traumatic arrest guideline, ultrasound may be used if it does not interrupt life-saving procedures such as bilateral thoracostomy or haemorrhage control. POCUS should therefore never delay decompression or thoracotomy when obstructive shock is suspected.

This **primary survey is not a full clinical exam** and should take **only a few minutes**. Its **sole objective** is to determine if **immediate interventions** are required. The evaluation may be performed by the trauma leader or delegated. Ideally, it leads to a **brief situation update** and an **action plan for the next 10 minutes**.

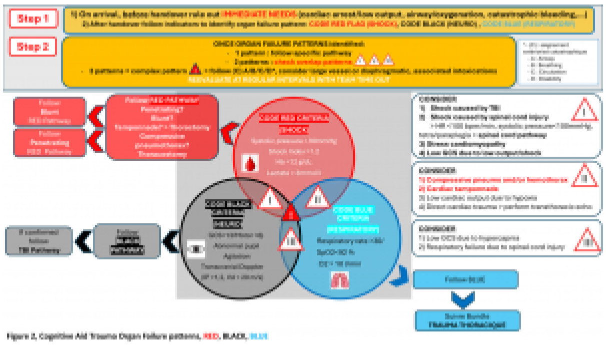

Figure 3: Provides an organ failure prioritization framework.

2. 2. 4. Clinical Examination

A **systematic clinical exam** must be performed (including the **posterior side** of the body, especially in penetrating trauma).
This **brief but comprehensive exam** complements the primary survey by identifying **additional injuries**, especially **peripheral or extremity injuries**. The goal is to identify clinical signs of **lesions needing urgent — if not immediate — intervention**.
This exam **should not exceed three minutes** and **must not delay other care**. It is particularly important in **penetrating trauma**, where identifying **all entry points** is critical. Entry wounds in the abdomen may also indicate **thoracic injuries**, and vice versa.
Because projectiles and blades can follow unexpected paths, a **CT scan is often helpful** in surgical planning, **if the patient is stable**.

2. 2. 5. Diagnostic Workup in the Trauma Bay

Available tools include:

- **Chest X-ray**
- **Pelvic X-ray**
- **Extended Focused Assessment with Sonography for Trauma (eFAST)**

2. 2. 5. 1. Chest and Pelvic Radiographs

Until the 2010s, chest and pelvic X-rays were **standard for all severe trauma patients**.
Chest X-ray can identify **pneumothorax, haemothorax,** pulmonary contusions, **aortic**

injury signs (e.g. widened mediastinum), or **diaphragmatic rupture**.
Sensitivity/specificity for pneumothorax detection: **47% / 100%** (95% CI: 31–63%, and 97–100%).
A 2001 study of 400 cases found **19% of chest X-rays led to urgent decisions**, all **clinically appropriate**.
Pelvic X-ray detects **major fractures** that may cause haemorrhage. A 2002 reference cohort showed **68% sensitivity / 98% specificity**, with a **30–40% false-negative rate**.
A 2001 study found pelvic X-ray led to **urgent decisions in 2%** of cases, all appropriate.
However, only **CT scan** reliably detects **complex fractures** and **active bleeding** via **contrast extravasation**.
Due to **limited accuracy** and the rise of **FAST ultrasound**, many centres have **abandoned routine trauma radiographs**, though some still use them in **Grade A (most critical) patients**, in **uncertain FAST results**, or **suspected diaphragmatic rupture**.
When performed, X-rays should be done **immediately after patient arrival** to **avoid delays or team interference** and to **inform early clinical decisions**.

2. 2. 5. 2. eFAST (Extended Focused Assessment with Sonography for Trauma)

> **Note**
>
> **eFAST** is now the **standard ultrasound exam** in trauma to identify **lesions requiring immediate intervention** or to guide patient orientation (e.g. radiology vs OR).
> FAST is not meant to provide detailed injury descriptions. It takes <3 minutes and can occur alongside the clinical exam.

Historically, **classic FAST** assessed the **abdominal cavity and pericardium**.
eFAST expands to include **pleura, lungs**, and optionally **transcranial Doppler (TCD)**.
eFAST detects:

- **Abdominal fluid** as a reliable indicator of **intra-abdominal bleeding**
  - 2015 Cochrane review (n = 2,755): sensitivity **90%**, specificity **98%**
  - In shock: sensitivity drops to **62%**, specificity **83%**
- **Pubic diastasis**, suggestive of **pelvic fracture**
- **Thoracic abnormalities** (pneumothorax, haemothorax, contusions)
  - Pneumothorax detection: sensitivity **91%**, specificity **99%**

2. 2. 5. 3. Transcranial doppler

Transcranial Doppler may be added for patients with **moderate to severe traumatic brain injury**. It provides rapid insights into **cerebral perfusion**, potential **brain death (backflow pattern)**, or **need for perfusion pressure adjustment**.
TCD indicators of low cerebral blood flow:

- **Pulsatility Index > 1.2**
- **Diastolic flow velocity < 20 cm/s**

2. 2. 6. Prioritization

All information gathered from clinical tools (physical exam, X-rays, eFAST ultrasound) contributes to forming a **diagnostic and decision matrix** for the trauma leader and team. This matrix helps prioritize which injuries to treat and determines the patient's destination. There are **three major priorities: respiratory, circulatory, and neurological failure**.

- **Respiratory threats,** particularly hypoxemia, must be addressed immediately through oxygenation, airway control, and, if necessary, pleural decompression.
- **Circulatory failure** is most often due to haemorrhage. Initial assessment aims to **identify haemorrhagic sources** (haemostatic targets) for **targeted therapy** to control bleeding. Haemorrhage is not the only cause of circulatory instability but is usually the most treatable.
- **Neurological threats**, such as cerebral herniation or spinal cord compression, are equally critical but, in terms of timing, come **after haemorrhage control**.

The **ultimate goal** of prioritization is to decide whether to send the patient to a **whole-body CT scan** for full injury assessment or directly to the **operating room (OR)** or **interventional radiology (IR)**.
This decision depends on:

- **Hemodynamic stability** (dynamic, influenced by resuscitation response).
- **Initial findings**, including the nature of the haemorrhagic target.
- **Trauma bay layout**, including proximity to scanner, OR, or IR suite.
- **Team experience**, including surgical and radiological support availability.

Although general rules are difficult to set, a **whole-body CT scan is preferred** to map injuries, including in penetrating trauma (e.g., ballistic), especially as part of **integrative damage control**. If the CT is close to the trauma bay, it's often faster and more informative.
If the patient is **very unstable**, direct OR transfer is needed, especially for **penetrating trauma**.
The **trauma bay stay** (including initial assessment and treatment of threats) should ideally **not exceed 30 minutes**.

2. 3. Interventions in the Resuscitation Room

It is not possible to offer universal recommendations regarding vascular access or other devices for equipping a severely injured trauma patient in the resuscitation room. At a minimum, two well-secured large-bore peripheral intravenous (IV) lines are required. A central venous catheter can be very useful, especially for administering catecholamines, but is not essential. Norepinephrine administration via peripheral IV access is considered safe up to 2 mg/h. Note that central lines are not ideal for rapid

volume resuscitation due to the Hagen-Poiseuille law; a large peripheral IV line is more effective.
If a central venous line is chosen, the femoral site is usually preferred, ideally combined with an arterial catheter. Even in cases of pelvic trauma, the vascular structures at this site are usually intact. Other options include the jugular or subclavian sites. In all cases, ultrasound-guided placement is strongly recommended to identify anatomical variations and secure faster, safer access.

The placement of an arterial catheter enables much closer and more reliable blood pressure monitoring, especially in low-output states. However, the benefits of central venous access and invasive blood pressure monitoring must be weighed against the delay they often introduce—typically 10 to 15 minutes. Vascular access placement must not delay or hinder other critical interventions, such as immediate surgery for haemorrhage control or CT imaging in suspected brain herniation. In the case of technical difficulties, attempts should be stopped after 10–15 minutes.
Intraosseous access, preferably at the humeral site (or tibial as second-line), is a reliable and easily performed alternative. The humeral site provides better flow and faster drug action.
A fluid warmer and infuser is also a critical piece of equipment for managing severe trauma. This device allows rapid infusion of large fluid volumes, including blood products. It should be prepared systematically before the arrival of a suspected haemorrhagic shock patient.
The placement of urinary or nasogastric tubes is not mandatory but can be considered after lesion assessment. Visible urethral bleeding should contraindicate urinary catheterization and prompt urology consultation. In such cases, a suprapubic catheter may be preferred. An orogastric tube is preferred over a nasogastric one in patients with suspected basal skull fractures.

2. 4. Damage Control Concept

The concept comes from naval warfare (e.g., the Falklands War) and refers to a ship's ability to survive damage and continue fighting. Analogously, **damage control in trauma** means **prioritizing survival over definitive repair**.

Patients with **limited physiological reserve** (due to age, trauma severity) may **not tolerate prolonged surgery.** Damage control surgery (DCS) uses **short, targeted procedures** to stop bleeding and contamination (e.g., liver packing, splenectomy, retroperitoneal packing).

Damage control resuscitation (DCR) complements this, minimizing iatrogenic harm from volume overload and focusing on **early coagulopathy treatment**.

The approach applies to a **subset of patients with rapid circulatory and metabolic failure** (e.g., hemorrhagic shock, coagulopathy, acidosis, hypothermia). It should be initiated by a **multidisciplinary decision** with clear objectives.

For more detail see section 7 in this module: Damage Control surgery

3. Patterns of organ failures: Circulatory, Respiratory, Neurological

3. 1. Circulatory failure and haemorrhagic shock

Causes of circulatory failure
Most commonly due to active haemorrhage, but also vasoplegia (e.g., spinal shock) or obstructive causes (e.g., tamponade, tension pneumothorax). Rarely cardiogenic (e.g., cardiac contusion). Rule out haemorrhage first as it is time-sensitive and treatable.

Definition of haemorrhagic shock
A type of hypovolemic shock due to massive blood loss, defined clinically by sustained systolic BP 30% drop from baseline, with signs of hypoperfusion (e.g., mottling, oliguria).

Therapeutic objective: rapid haemorrhage control
Haemorrhagic shock evolves in two phases:

1. Sympathetic phase: compensatory tachycardia and vasoconstriction.
2. Parasympathetic phase: sudden hypotension and paradoxical bradycardia, triggered by intense hypovolemia (via vagal reflex).

External bleeding must be controlled early (e.g., scalp clips, pelvic binder, limb tourniquets). The patient should be transferred promptly to a trauma center.

eFAST ultrasound is crucial for early hemorrhage detection (sensitivity: 62–89%, specificity: ~95–98%). If no source is found, CT scan allows lesion mapping and prioritization. Whole-body CT is associated with reduced mortality.

Surgical and interventional techniques (e.g., embolization) should be selected via multidisciplinary discussion. In some cases, REBOA (aortic balloon) may temporize bleeding while definitive treatment is prepared.

Blood Pressure Targets
In traumatic hemorrhagic shock, aim to maintain perfusion without worsening bleeding (i.e., permissive hypotension). Target:

- SBP 80–90 mmHg, or MAP 60–65 mmHg.
- In TBI patients, aim for SBP >110 mmHg.

For detailed hemodynamic management see Module on Haemostatic resuscitation

3. 2. Respiratory Failure and Airway Management

Respiratory failure is uncommon but accounts for about 10% of preventable trauma deaths, often due to hypoxemia. Tachypnea (>30 breaths/min) is a warning sign and may also indicate early circulatory insufficiency.

Causes of respiratory failure include:

- **Neurologic:** Airway obstruction, aspiration, impact apnea.
- **Mechanical:** Flail chest, multiple rib fractures, pneumothorax, hemothorax, hemopneumothorax, diaphragmatic rupture/herniation.
- **Parenchymal:** Pulmonary contusion.

Obstructive causes are typically managed with airway clearance, aggressive oxygenation, and securing the airway. This latter step—induction and orotracheal intubation—is high risk in severe trauma. Every intubation should be considered difficult due to positioning, cervical spine precautions, bleeding, vomiting, etc. Preoxygenation is essential but less effective in this context, increasing the risk of hypoxemia. Therefore, thorough preparation and anticipation are crucial (see spinal trauma section). The MACOCHA score helps assess intubation difficulty. Video laryngoscopy is a preferred technique for this high-risk population.
For induction agents, drugs with minimal hemodynamic impact like ketamine or etomidate are preferred. Recent meta-analyses suggest a trend against etomidate; a detailed discussion is beyond this scope. Crucially, in trauma patients with shock, anesthetic doses should be reduced to blunt sympathetic inhibition.

Among mechanical causes, tension pneumothorax requires **immediate decompression** via thoracostomy at the 4th intercostal space, mid-clavicular line. Needle decompression is not recommended, as it's ineffective in ~50% of cases. Thoracostomy should be performed in any patient with circulatory or respiratory compromise, especially under positive pressure ventilation. In some cases, it must be done even without confirmed pneumothorax on imaging.
Chest tube placement is not always required afterward but omitting it risks pneumothorax recurrence. If no tube is placed, stoma patency must be monitored.
Chest tube insertion can also aid diagnosis by quantifying thoracic bleeding. An initial output >1000–1500 mL or ongoing bleeding >150–200 mL/hour should prompt

consideration of damage-control thoracotomy
In the early phase, intubation and mechanical ventilation usually stabilize mechanical issues such as flail chest or multiple rib fractures.

3. 3. Neurological Injury and Traumatic Brain Injury

A 2016 French epidemiological study found a 6% overall hospital mortality among 144,000 trauma patients. This increased to 12% when traumatic brain injury (TBI) was present, affecting around 40% of cases.
The first step in TBI management is severity assessment, based primarily on the Glasgow Coma Scale (GCS: eye, verbal, and motor responses), combined with pupillary reactivity. This informs early management strategies such as osmotherapy. This assessment must be repeated frequently due to the evolving nature of brain injuries.
The second step is to optimize cerebral hemodynamic to reduce the risk of hypoperfusion. Transcranial Doppler (TCD) can detect cerebral hypoperfusion via the pulsatility index (PI); a PI > 1.2–1.4 suggests altered perfusion. Assessing middle cerebral artery velocities and PI via TCD helps guide blood pressure targets!
A combination of clinical and paraclinical findings (pupil exam, TCD, pupillometer) may warrant osmotherapy (e.g., mannitol, hypertonic saline) as a temporary measure to control intracranial hypertension while awaiting surgery (hematoma evacuation, decompressive craniectomy). Routine prophylactic osmotherapy is not currently recommended!
Control of cerebral homeostasis includes preventing and correcting systemic secondary insults to the brain (ACSOS). Hypotension and hypoxemia are independently associated with increased mortality and poor neurologic outcomes—even from the first episode. Therefore, early airway control with endotracheal intubation should be considered in severe TBI to prevent aspiration, address hypoxemia, and maintain normocapnia. CO_2 levels strongly affect cerebral arteriolar reactivity; hypocapnia causes vasoconstriction and ischemia, while hypercapnia causes vasodilation and increased cerebral blood volume. Hence, continuous end-tidal CO_2 monitoring is recommended from intubation onward, with a target $EtCO_2$ of 35–40 mmHg, avoiding fluctuations that could impair cerebral perfusion
All these steps must occur simultaneously to minimize delays in care and enable early brain imaging for lesion characterization and potential surgical intervention.
In cases of TBI associated with skull base or cervical spine fractures, complex facial fractures (Le Fort II/III), soft tissue neck injuries, or when initial imaging does not explain the neurologic picture, further imaging should assess the supra-aortic trunks for vascular injury
Primary brain injuries (from the trauma itself) and secondary injuries (e.g., ischemia or oedema) combine to create additional intracranial volume within the fixed cranial vault, leading to intracranial hypertension (ICH). ICH reduces cerebral perfusion, worsening

ischemia and causing neuronal death. Therefore, intracranial pressure (ICP) monitoring is crucial to assess TBI severity, diagnose ICH, and monitor cerebral perfusion pressure (CPP = MAP - ICP) when combined with invasive blood pressure monitoring.
ICP monitoring is widely indicated, as elevated ICP is hard to predict clinically. It is achieved by inserting an intracranial pressure sensor. Current guidelines recommend maintaining ICP < 20 mmHg and CPP between 60–70 mmHg. When ICH is detected, it constitutes a medical emergency requiring a tiered response involving medical measures (deep sedation, ACSOS control) and surgical procedures (hematoma evacuation, craniectomy, ventricular drainage). These interventions fall within the expertise of specialized centers and are covered in expert recommendations.

3. 4. Special populations

3. 4. 1. Pregnant patients

Trauma is the leading non-obstetrical cause of maternal death during pregnancy. Across all severities, trauma affects 6–8% of pregnant patients, with a high risk of underestimating the severity, both maternal and fetal. Morbidity and mortality vary depending on gestational age and injury mechanism. Falls are more common due to weight gain, balance changes, and ligament laxity.
Overall, the management does not differ significantly, but when pregnancy is known, careful evaluation and monitoring of obstetric and fetal complications are essential. Any delay in maternal care negatively impacts the fetus.
Main obstetric complications include:

- Miscarriage
- Premature rupture of membranes
- Uterine rupture
- Placental abruption
- Intrauterine fetal death

A reassuring maternal clinical state does not guarantee fetal integrity.

Management must be multidisciplinary, coordinated, and rapid, posing several

Challenges

- Balancing maternal and fetal risks
- Rare and stressful situation involving teams unaccustomed to severe trauma (obstetricians, emergency teams), or working together; this should be anticipated before patient arrival with clearly defined roles. Ideally, a protocol should guide the management of these patients.

Many physiological and anatomical changes occur during pregnancy, some of which vary with gestational age. These changes can delay hypovolemia diagnosis and reduce physiological compensation. Laboratory values are altered, complicating interpretation. Airway management requires special care due to increased risks of desaturation and difficult intubation. Additionally, from 20 weeks onward, supine positioning can compress the inferior vena cava, necessitating a left lateral tilt to improve venous return.
In addition to the standard eFAST, a rapid fetal assessment can be performed by the obstetric team upon admission.
A full-body CT scan can be performed using an adapted protocol with no significant increase in fetal risk, after informing the radiology team. In some cases of maternal and fetal stability and in experienced centres, a targeted scan based on clinical findings may be appropriate.

3. 4. 2. Elderly Patients

Representing up to 20% of the global population, patients over 65 admitted for major trauma require special consideration. In 2015, the American Association for the Surgery of Trauma (AAST) identified geriatric trauma as a growing issue linked to high hospital admission and readmission rates. These patients are frequently under-triaged, despite better outcomes when admitted to trauma centers.
Important considerations in elderly trauma patients include:

- Baseline functional status, physiological age, or frailty scores, which reflect physiological reserve and adaptive capacity
- Pre-existing conditions and medications, which may influence diagnosis and management

At the same injury severity, elderly patients have significantly higher mortality, especially when moderate-to-severe traumatic brain injury (TBI) is involved.
The spine, particularly the upper cervical vertebrae (C1–C2), is vulnerable due to degeneration or arthritis. Early evaluation and immobilization are important. However, cervical collars can cause discomfort, skin breakdown, or delirium. When spinal injury is suspected, risks and benefits of immobilization, surgical intervention, and early mobilization with bracing must be considered.
Each intervention's benefits and risks must be assessed early in a multidisciplinary context including geriatrics and rehabilitation. While early invasive treatments may offer short-term benefits, they can also lead to medium-term complications (e.g., immobility). Even with stable fractures, mortality increases with age, transfusion needs, cognitive impairment, or mechanical ventilation. Attention to infection, delirium, and electrolyte imbalance is critical.
Some authors advocate for aggressive early management using dedicated teams for trauma patients over 70, which may reduce hospital stay and mortality. This strategy may help mitigate age-related excess mortality.
However, such patients are often less likely to be admitted to trauma centres. Reasons may include unconscious age bias, low-energy injury mechanisms, altered

physiological responses, or lack of specific triage scores.

During early care, particularly in intensive care, decisions around treatment intensity and duration often arise. Scores like the Geriatric Trauma Outcome Score can support such decisions, but morbidity and mortality remain hard to predict. Therefore, a holistic management approach, accounting for the patient's previous condition, recovery potential, support system, and including specialized teams (geriatrics, rehab, social workers), is strongly recommended.

A reasonable strategy involves:

- Aggressive triage and management guided by physiological frailty scores
- Early correction of coagulopathy
- A reassessment at 72 hours to evaluate treatment responsiveness, supported by decision tools

In Chapter References

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4. Haemodynamic management

4. 1. Cardiac arrest management

Historically, traumatic cardiac arrest (TCA) had extremely poor survival, but recent data show survival rates of **7–17%**, particularly when reversible causes are rapidly treated. The ERC 2025 guidelines emphasise that **TCA is potentially reversible** and must be approached differently from medical cardiac arrest.

Chest compressions must not delay life-saving interventions. It is essential to identify and treat reversible causes of cardiac arrest:

- Hypoxaemia
- Tension pneumothorax
- Massive haemothorax
- External compressible haemorrhage (tourniquets, wound packing, haemostatic dressings)
- Non-compressible haemorrhage (pelvic binder, splinting, rapid blood-product resuscitation)
- Cardiac tamponade

In TCA or peri-arrest states, corrective actions for reversible causes should occur simultaneously whenever personnel allow, as hypoxia, hypovolaemia, tension pneumothorax, and tamponade frequently coexist and worsen each other's physiology.

> ★ **Important**
>
> Bilateral finger thoracostomy should be performed as a priority to ensure chest decompression in case of traumatic cardiac arrest.

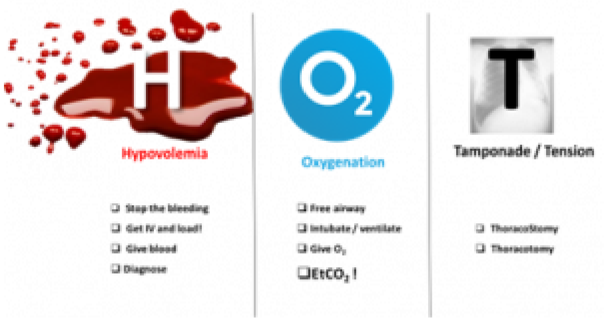

Figure 4: Reversable causes of traumatic cardiac arrest.

4. 1. 1. Intravenous cannulation and monitoring

- Establishment of intravenous access is a key intervention.
- Short but large-bore intravenous cannulas promote rapid fluid infusion. In patients with severe trauma, early insertion of at least two such cannulas may be lifesaving since venous filling can dramatically decrease as haemorrhage progresses.
- In hospital, central venous and arterial cannulation can be performed in certain circumstances. They allow continuous and reliable monitoring of blood pressure, safe drug and vasopressor administration, easy blood sampling and large bore venous access for high volume resuscitation. The lines for the arterial and central catheter should be prepared in a sterile manner by a trained trauma nurse before the arrival of the patient. In blunt trauma, the femoral site is a good option for arterial and venous access, since large vessel injuries are uncommon and pelvic trauma only exceptionally impairs femoral cannulation.
- Early starting with blood products transfusion in case of apparent haemorrhage.
- When peripheral access is difficult or impossible due to severe hypovolaemia, early escalation to central venous access can be considered. Evidence from pre-hospital trauma cohorts shows that up to 75% of patients in exsanguinating shock have no peripheral IV access on arrival, and large-bore central catheters allow substantially higher flow rates than intraosseous devices. Successful placement is associated with greater delivery of blood products and higher rates of ROSC and survival to the emergency department, with an acceptable complication rate (~4%). Landmark-guided subclavian access is often preferred because the vessel remains patent even in low-flow states.

4. 1. 2. Emergency haemorrhage control

Controlling haemorrhage is crucial to treat the underlying cause of hypovolaemic shock. In external haemorrhage, tourniquets, external compression, stapling, stitching and splinting are used to achieve this goal. Recent experience from military medicine suggests that haemostatic compression dressings can enhance local clot formation and increase the chances of haemorrhage control by external compression.

If external compression cannot control haemorrhage from extremities, a tourniquet may be used. Caution should be given when a tourniquet is applied due to ischaemia of distal tissues and neuropathic injury, but it remains a life-saving intervention in case of uncontrollable bleeding.

In patients with shock, a pelvic binder should be applied even in the absence of clinical signs of pelvic fracture. Pelvic binder minimises motion at the fracture sites and reduces venous haemorrhage.

4. 1. 3. Principles of haemorrhagic shock resuscitation

In case of haemorrhagic shock, it is paramount to maintain adequate tissue perfusion. Fluid replacement constitutes an important element of haemorrhagic shock resuscitation. Excessive fluid administration, however, may result in haemodilution and cause relative anaemia. However, the organism disposes of more physiologic mechanisms to compensate for anaemia and haemodilution than hypovolaemia-

induced tissue hypoperfusion. Therefore, appropriate fluid resuscitation remains a crucial element. There is, however, a fine balance between appropriate and excessive fluid resuscitation. Excessive fluid loading can raise the blood pressure to levels that may disrupt clots and enhance bleeding, contribute to coagulopathy, and precipitate organ damage such as pulmonary or kidney injury. There is no ideal resuscitation fluid; colloids should probably be avoided altogether, particularly in the most severely injured patients with high risk of organ failure. Normal saline is under scrutiny, because its saline and chloride load may lead to hyperchloremic acidosis and kidney injury. Balanced crystalloids are a better option, but not in patients with severe TBI if they are hypotonic (like Ringer Lactate).
For all of these reasons and especially in uncontrolled haemorrhage or until haemorrhage is controlled, a fluid restrictive approach should be applied, and fluid be administered to target a systolic pressure between 80 and 90 mmHg. This approach is known as permissive hypotension or damage control resuscitation. A prolonged permissive hypotension beyond 90 minutes may increase the risk of organ damage.
Early administration of a complete array of blood products is preferable to fluid resuscitation. In the event of concomitant TBI, targeting a higher systolic pressure between 100 and 120 mmHg might be beneficial, but this has to be individualised, balancing benefits and risks. Vasopressor-inotropes such a Noradrenaline can be used to achieve this objective. Noradrenaline is also the agent of choice to maintain perfusion pressure in severe or prolonged haemorrhage induced hypotension.

> **Note**
>
> Research is required to elucidate the place of fluids, the optimal kind of fluids and the place of noradrenaline in the resuscitation of haemorrhagic shock and which strategy to apply in which clinical scenario.

> **Note**
>
> Current recommended practice is restrictive fluid management, avoiding excessive amounts of crystalloids. In case of apparent haemorrhage starting with blood products is paramount and continue as long as indicated.

In case of severe shock, uncontrolled haemorrhage and/or severe anaemia, transfusion of O negative or O positive packed red blood cells can be necessary. No absolute haemoglobin value can be stated as a definite indication for uncrosses matched transfusion. The decision is based on clinical judgment integrating the dynamic character of severe haemorrhage.

4. 1. 4. REBOA

Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA) is an endovascular technique intended to provide temporary haemorrhage control in patients with severe non-compressible torso bleeding. A balloon catheter is inserted via the femoral artery and advanced into the aorta, where inflation occludes distal flow and transiently augments central perfusion. Its purpose is purely bridging, buying time for definitive surgical or endovascular haemorrhage control.

However, its role has been significantly de-emphasised in current international guidelines. The 2025 European Resuscitation Council (ERC) Guidelines explicitly state that REBOA is de-emphasised and is no longer included in the traumatic cardiac arrest management algorithm. Moreover, due to signals of potential harm (including increased mortality in the UK-REBOA randomized trial) the ERC does not recommend the use of REBOA outside clinical trials.

Accordingly, REBOA should be considered only in highly selected patients and only within systems that have established protocols, expert operators, and immediate access to operative or interventional radiology resources. Definitive haemorrhage control must follow without delay, and occlusion time should be minimised due to the risk of distal ischaemia and reperfusion injury.

Ongoing research continues to refine the indications, contraindications, and operational safety of this intervention.

4. 1. 5. Relief of obstructive shock

In trauma, obstructive shock results from tension pneumothorax, cardiac tamponade and, in rare cases, by tension pneumomediastinum. Tension pneumothorax results from a one-way valve effect in which air is forced into the pleural cavity during inspiration but is unable to escape during expiration. The accumulation of intrathoracic air increases the intrathoracic pressure to a point that intrathoracic organs and vessels are compressed, and consequently venous return and cardiac output are impaired.
The first sign of obstructive shock is hypoxia. Other clinical features include arterial hypotension, tachycardia, respiratory distress, tachypnoea, ipsilateral absence of breath sounds, tracheal deviation from mediastinal distortion to the opposite side and jugular venous distension.

> ★ **Important**
>
> Tension pneumothorax is a clinical diagnosis and should be decompressed immediately (without waiting for X-ray confirmation) by performing a finger thoracostomy. Finger thoracostomy should be performed at the intercostal space 4-5 (above the inferior rib) on the anterior axillary line. Finger thoracostomies may require re-fingering if the compression reoccurs. Needle decompression can be attempted as a bridging measure but may not prove efficient and should not delay finger thoracostomy. If needle decompression is performed, it should be done at the intercostal space 4-5 (above the inferior rib) on the anterior axillary line where chest wall thickness is the thinnest and where the decompression failure rate is the lowest (13%). In in-hospital setting, preparations should then be made to insert a chest drain as soon as possible.

In the pre-hospital setting, the diagnosis of cardiac tamponade in blunt trauma is extremely difficult to make. In the ED, clinical signs may be strongly suggestive, and the definitive diagnosis can be immediately made by ultrasonography (see clinical imaging chapter). Ideally an extended FAST should be performed rapidly to confirm the suspicion without delaying the intervention to treat tamponade. Although often suggested, needle pericardiotomy is impractical in trauma patients and rarely drains blood clots from the pericardium. Emergency clamshell thoracotomy may be indicated in select patients with penetrating chest trauma and witnessed cardiac arrest in order to treat cardiac tamponade or stop central haemorrhage by aortic cross-clamping and perform open cardiac massage.

5. Damage control strategy

Haemorrhagic shock represents the second major cause of early death among the injured regardless of the mechanism of injuries. Haemorrhagic shock combined with central nervous system (CNS) injuries causes high rates of prehospital mortality. Most trauma deaths occur in the first few hours following injury, often before the injured patient reaches the hospital. Haemorrhage contributes to death during the prehospital period in 33-56% of cases and is the most common cause of death upon arrival of the Emergency Medical Service (EMS). Exsanguination accounts for the largest proportion of mortality within the first hour of trauma centre arrival. Thereafter, CNS injury replaces haemorrhage as the leading cause of trauma mortality. Very few haemorrhagic deaths occur after the first day. The presence of early haemorrhagic shock in the pre-hospital setting or ED is associated with high rates of multiple organ failure (24%) and sepsis (39%). Critically injured trauma patients are treated in three, often overlapping phases:

- the resuscitation phase, with initial haemorrhage control and lifesaving stabilization
- the interventional phase, when definitive control of bleeding is obtained
- the critical care phase, when support and restoration of normal physiology are accomplished

Various recommendations for the control of acute traumatic haemorrhage have been published in recent years. The European consensus guidelines include operative interventions for acute bleeding control: stabilization/closure of the pelvic girdle (1B); intra-abdominal surgical haemostasis with abdominal packing and vascular embolization (1B/C); damage control measures (early repositioning and fracture stabilization of long tubular bones and control of internal organ injury/haemorrhage) (1C); local haemostasis combined with other surgical procedures or packing in the case of venous or moderate arterial haemorrhages with parenchymal injury (1B). Death from massive traumatic haemorrhage may occur within minutes, particularly if a major artery is involved. The primary goal of haemorrhagic shock resuscitation is to arrest haemorrhage and re-establish effective circulating blood volume and tissue perfusion. The ability to arrest the haemorrhage will in part depend on its location (e.g., extremity versus intracavitary). A source inaccessible to immediate, direct haemostasis control can exacerbate the state of shock and lead to death very quickly. The secondary goals of resuscitation are to avoid rebleeding and to counter any physiological derangements (e.g., coagulopathy, metabolic acidosis, hypothermia) induced by the traumatic injury, state of shock, or resuscitative intervention. The combination of coagulopathy, metabolic acidosis, and hypothermia has been termed the “lethal triad of death” and is associated with a high mortality rate.

Fluid resuscitation has continuously evolved over the past few decades. Traditionally, large volumes of non-blood-containing fluids (e.g., crystalloids and synthetic colloids), have been used to restore lost circulating volume; however, over the past few decades, this large-volume approach has shown to be deleterious. At least for human trauma casualty care, Damage Control Resuscitation (DCR) has been promoted as a more optimal resuscitative approach for the most severely injured patients suffering from uncontrolled haemorrhagic. DCR is a systematic approach that starts in the emergency room and continues throughout the operating room and the Intensive Care Unit (ICU). It is designed, along with damage control surgery, to promptly and aggressively reverse the lethal triad. This approach is based on the use of "haemostatic resuscitation" and "permissive hypotension". Haemostatic resuscitation is characterised by using combined blood product components with very limited crystalloid infusion to avoid iatrogenic haemodilution and the worsening of metabolic acidosis and hypothermia while countering any coagulopathy. Permissive hypotension is the strategic decision to delay the initiation of fluid resuscitation and provide limited resuscitation with fluids/blood products to bleeding trauma patients by targeting a lower-than-normal blood pressure to allow adequate perfusion without disrupting thrombus formation. Then, during the past decade, the practice of massive transfusion (MT) has expanded appreciably from a simple definition to a more comprehensive management strategy for haemorrhagic shock referred to as DCR. In patients requiring MT, it is critical to maintain adequate blood flow and arterial pressure to maintain tissue oxygenation to vital organs. In DCR, volume resuscitation is performed with RBC, FFP and platelets in a 1:1:1 fashion restricting crystalloid administration just to keep lines open. Colloids should be avoided. DCR is carried out most effectively if directed by a specific protocol or algorithm that delineates authority and clinical criteria for activation, outlines the responsibilities of various members of the resuscitation team, lists how and at what time points the patient is assessed, directs the timing and coordination of certain interventions, suggests resuscitation endpoints to monitor, and specifies under what circumstances a DCR is deactivated. The efficiency of a protocol-directed process decreases the time to transfusion, which might be a significant factor in reducing organ failure, other post-injury complications, and mortality. Protocols direct transfusion of blood components either in fixed ratios or as a goal-directed strategy. Early recognition and prompt treatment results in improved outcomes in exsanguinating patients. Upon arrival in the ED, it might be unclear which patient will need massive transfusion (MT). Models have been developed using both clinical and laboratory parameters to predict who would need MT. Several definitions of MT exist based on the volume of transfused blood products per time unit. The three most common definitions of MT in adult patients are:

- transfusion of ≥10 Units of Packed Red Blood Cells (PRBCs), which approximates the Total Blood Volume (TBV) of an average adult patient, within 24 h
- transfusion of at least 4 Units of PRBCs in 1 h with anticipation of continued need for blood product support
- replacement of 50% of the TBV by blood products within 3 h.

Identification of patients in need of MT are critical tasks for future research since appropriate MT guidelines seems to improve survival. Prediction tools for MT in adult trauma patients have been developed for both military and civilian trauma patients with predominantly penetrating or blunt injuries respectively. The predictive ability ranges from 80% to 90%. These prediction equations typically include blood pressure, heart rate, base deficit, INR, and haemoglobin values, and FAST examination. The proposed advantage of using these models is that they increase the ability to determine which patients will require a DCR upon admission. Several Scoring systems have been developed for adult and paediatric trauma populations to predict early which patients are more likely to require MT, defined as the transfusion of 10 units or more of pRBC within 24 hours of admission. Examples of such models are:

- ***Trauma Associated Severe Haemorrhage (TASH) Score***, which incorporates several physiological and injury-related variables (e.g., haemoglobin, base excess, systolic blood pressure, heart rate, gender, injury pattern). The aim of this score is to assess the persistence of bleeding by using the probability of massive transfusion as a surrogate index.
- ***Vandromme Score*** links some clinical measurements with the need for MT: blood lactate (BL) ≥ 5 mmol/l, heart rate >105 bpm, INR >1.5, haemoglobin ≤11 g/dl, and systolic blood pressure < 110 mmHg. (Alternative models are PWH (Prince of Wales Hospital) score, ABC (Assessment of Blood Consumption) score, Schreiber score and Larsen score)
- ***Red Flag***, a score predicting severe haemorrhage from the prehospital setting has recently been proposed. It gathers five criteria: unstable pelvic fracture, shock index (heart rate/ systolic arterial pressure) ≥1, on-scene point-of-care haemoglobin ≤13g/dl, mean arterial pressure <70 mmHg and orotracheal intubation. In practice, the presence of any combination of at least two criteria during the pre-hospital care phase activates the Red Flag and provides a powerful signal to initiate an adequate intra-hospital standardized haemorrhage control response (massive transfusion protocol and/or immediate haemostatic procedures).

6. Permissive hypotension

Traditionally, resuscitative strategies for countering haemorrhagic shock have focused on replacing extracellular fluid deficits with rapid infusion of large volume of crystalloids to restore normal haemodynamic parameters. Over the last few decades, increasing evidence has shown that large-volume crystalloid resuscitation is not innocuous. Large-volume resuscitation may actually exacerbate the haemorrhagic shock, induce an inflammatory response, and cause immune dysregulation and coagulopathy. Evidence from experimental animal models has shown that resuscitation with large volumes of crystalloids with the aim of achieving normotension in acute uncontrolled haemorrhage may worsen haemorrhage, exacerbate blood loss, and increase mortality when compared to deliberate hypotensive fluid resuscitative approaches.

Haemodilution via large-volume fluid resuscitation significantly worsens the severity and duration of trauma-induced coagulopathy and contributes to developing the classical “lethal triad”.

Current recommendations and guidelines are trending toward reducing prehospital or presurgical fluid volume resuscitation, an approach referred to as deliberate hypotensive resuscitation or permissive hypotension. The premise is to achieve and maintain adequate vital organ perfusion and tissue oxygenation without further exacerbation of blood loss by allowing thrombus formation. The blood flow should allow sufficient oxygen delivery to tissues. This is verified by monitoring serum lactate levels and central venous oxygen saturation.

Several experimental studies have shown that maintaining a systolic blood pressure of approximately 90 mm Hg and a mean arterial pressure (MAP) of around 60 mmHg until definitive surgical haemostasis has been achieved, results in increased oxygen delivery, decreased blood loss, and reduced mortality. However, strong evidence from randomised trials is lacking.

The current European guidelines on management of major bleeding and coagulopathy following trauma without TBI recommend in the initial phase a target systolic blood pressure of 80–90 mmHg until major bleeding is stopped. The pressure target must be achieved with small amounts of fluids. Haemorrhage-induced hypotension can progress into irreversible haemorrhagic shock resulting in end-organ damage and mortality if untreated for several hours. Therefore, the time between injury and bleeding control should be minimized. Contraindications to hypotensive resuscitative approach include TBI and spinal cord injuries. In these patients, maintaining adequate cerebral perfusion pressure (CPP) is vital to avoid further harm and for the recovery of the neuronal tissues. Hypotension or hypoxia in patients with TBI contributes to increased mortality rates. CPP depends on MAP and intracranial pressure (CPP = MAP – Intracranial pressure). Too low CPP in the case of severe TBI will worsen the secondary brain injury. The current European guidelines on the management of major bleeding and coagulopathy in trauma recommend that a MAP should be maintained at ≥ 80 mmHg in patients with severe TBI (GCS ≤8). In addition, the concept of permissive hypotension should be carefully considered in elderly patients and patients with chronic arterial hypertension. In presence of life-threatening hypotension, the administration of vasopressors is recommended in addition to fluids to maintain target arterial pressure. Damage control resuscitation and permissive hypotension are strategies awaiting definitive treatment, which should be the aim of the total treatment team.

7. Haemostatic resuscitation

Over the last 10 years, the management of major haemorrhage in trauma patients has changed significantly. Traumatic coagulopathy has been demonstrated in patients who received little or no intravenous fluid therapy, negating the long-held belief that iatrogenic haemodilution is the main causative factor of traumatic coagulopathy. The term “trauma-induced coagulopathy” (TIC) has been launched, divided in two chronological phases:

The first is “the acute traumatic coagulopathy (ATC”, caused by immediate activation of multiple haemostatic pathways, (including fibrinolysis), in association with tissue injury.

The second is the “resuscitation-related coagulopathy” or “dilutional coagulopathy” caused by the dilution of haemostatic factors and by the development of hypothermia.

The recognition of TIC is important, it is a prognostic factor associated with poor clinical outcome. Clinical management has changed, the focus in DCR is both to re-establish oxygen-carrying capacity and the ability to coagulate. Haemostatic resuscitation should be initiated as soon as possible.

7. 1. Acute traumatic coagulopathy

Acute traumatic coagulopathy is a distinct, endogenous coagulopathy that can develop within 30 minutes after trauma. It develops before resuscitative efforts are initiated; regardless of loss, consumption, or dilution of haemostatic factors or platelets; it develops prior to any influence from physiological factors such as metabolic acidosis and hypothermia. ATC manifests as systemic hypocoagulation and hyperfibrinolysis. Hypotheses of its origin are: 1) a disseminated intravascular coagulation (DIC) with a fibrinolytic phenotype, 2) an enhanced thrombomodulin-thrombin protein C pathway, and 3) a marked sympathoadrenal response leading to catecholamine induced endothelial and glycocalyx damage. Studies have demonstrated that ATC occur when significant hypoperfusion (base deficit > 6 mmol) is combined with severe injury (Injury severity score > 15). As shock severity increases, the PT and INR rise, and coagulation factor levels fall. Fibrinogen < 1,5 g\L in 41 % of the patients who were hypotensive on admission. It has been reported that up to 25-34% of trauma patients suffering major injuries and significant haemorrhage are hypocoagulant upon presentation in emergency room.

7. 2. Standard coagulation tests

The most commonly used tests for defining TIC include a 50% prolongation of aPTT or PT, an INR ≥1.5, PT ratio >1.2, or fibrinogen <1,5 g/L. PT-based tests (PT, INR), aPTT and Clauss fibrinogen are useful in guiding transfusion and predicting mortality. Originally, these tests were designed to evaluate clotting factor deficiencies rather than acquired multiple factor-based coagulopathies, and so they are not bleeding predictors in these cases. Moreover, they do not assess the contribution of platelets to haemostasis, the role of fibrinolysis, thrombin generation, or the interactions between coagulation enzymes and cellular phospholipid surfaces. Furthermore, these are not point-of-care assays and turnaround times often negate the value of the results. Therefore, plasma-based coagulation assays are rarely helpful in the immediate management of ATC, and do not have an important role in monitoring ongoing bleeding or as a guide to the appropriate use of blood products. Increasingly, TEG thromboelastography (Hemonetics Corporation, Braintree, MA, USA) and ROTEM thromboelastometry (TEM International GmbH, Munich, Germany) are being used to guide trauma resuscitation, although we are still lacking evident data from RCTs or other clinical trials. Whole-blood viscoelastic tests can more sensitively and reliably assess the complex nature of TIC. These laboratory methods describe initiation, formation and stability of the blood clot and can give more comprehensive insight into the pathologies of the coagulation system during acute haemorrhage. Advocates for viscoelastic monitoring suggest that the capacity to distinguish specific haemostatic abnormalities provides a means of individualizing coagulation and transfusion management. In addition, thrombelastometry/-graphy are point-of-care assays with the ability to identify systemic hyperfibrinolysis. Limitations are the inability to integrate temperature effects (measurements are performed at 37 °C) or assess local coagulation pathologies (e.g., local hyperfibrinolysis). Platelet dysfunction and the effects of various anticoagulant drugs (e.g., vitamin K-antagonists) are also difficult to evaluate. These point-of-care devices are still being studied to properly target their role in the resuscitation process.

7. 3. Therapy

In all trauma patients with, or considered to be at high risk of, significant haemorrhage (systolic arterial blood pressure <90 mmHg, or heart rate >110 bpm, or both) coagulation support should be initiated immediately. As soon as coagulation monitoring is available, it must be used to guide the therapy. Early therapeutic interventions reduce the need for transfusion of RBC, fresh frozen plasma and platelets, reduce the incidence of post-traumatic multi-organ failure, shorten the length of hospital stay, and may improve survival.

7. 3. 1. Initial haemostatic resuscitation

We define “initial resuscitation” as the period between arrival in the emergency department and availability of results from coagulation monitoring. Tranexamic acid should be administered, first as a bolus dose of 1 g within 3 hours of injury. A second dose of 1 g (infusion) is administered over the next 8 hours. The Clinical Randomisation of Antifibrinolytic therapy in Significant Haemorrhage (CRASH-2) trial assessed the effects of early administration of TXA on survival, vascular occlusive events and the receipt of blood product transfusion in trauma patients. All causes of mortality were significantly reduced with TXA by 1.5 %, and the risk of death due to bleeding was significantly reduced by 0.8 % There was a reduction in bleeding deaths by one-third, mainly through preventing exsanguination within the first 24 h. The early treatment (≤1 h injury) significantly reduced the risk of death due to bleeding by 2.5 %. Plasma (FFP or pathogen-inactivated plasma) in a plasma–RBC ratio of at least 1:2 (as needed). As an alternative, fibrinogen concentrate or cryoprecipitate 2 gr and RBC (according to Hb level) can be used. Levels of fibrinogen lower than 1.5 g/l are detected in more than 70% of patients with an admission Hb lower than 100 g/l and in more than 60% of those with a BE lower than -6. Fibrinogen was low (< 1,5 g\L) in 41% of patients who were hypotensive on admission. Hypotension, increasing shock severity and a high degree of critical injury (ISS ≥25), were all associated with a reduction in fibrinogen levels. Fibrinogen depletion is associated with poor outcome. Fibrinogen is by far the coagulation protein with the highest plasma concentration. The sources of fibrinogen are fresh frozen plasma and fibrinogen concentrate. A major disadvantage of fresh frozen plasma is the time delay related to thawing and administration. Administration of plasma to bleeding patients help stabilise fibrinogen levels, but cannot contribute to a significant increase in fibrinogen levels unless high volumes are infused (dilutional effect). One litre of plasma contains on average 4 g of fibrinogen. For initial coagulation support, 2 to 4 g of fibrinogen is recommended to correct hypofibrinogenemia already present on arrival in the exsanguinating patient. The 2016 European Guidelines introduced a new restrictive approach providing fibrinogen concentrate and RBCs according to haemoglobin concentration, with or without viscoelastic tests’ guidance.

7. 3. 2. Further haemostatic resuscitation

As soon as results from coagulation monitoring (Standard coagulation tests and\or viscoelastic tests) are available, they should be used to guide further haemostatic therapy. In the bleeding patient, we recommend maintaining Hb levels between 8 and 10g/dL. If a plasma-based coagulation resuscitation strategy is used, plasma (FFP or pathogen-inactivated plasma) must be administered to maintain PT and APTT <1.5 times the normal control. Plasma transfusion must be avoided in patients without significant bleeding. If a concentrate-based strategy is used, fibrinogen concentrate, or cryoprecipitate must be used if significant bleeding is accompanied by viscoelastic signs of a functional fibrinogen deficit or a plasma fibrinogen level of less than 1.5–2.0 g/L. We suggest an initial fibrinogen supplementation of 2–4 g. Repeat doses must be guided by viscoelastic monitoring and laboratory assessment of fibrinogen levels.

Platelets must be administered to maintain a platelet count above 100 × 10^9/L. Platelet count must be kept above 100 × 10^9/L in patients with ongoing bleeding and/or TBI. If administered, an initial dose of four to eight single platelet units or one aphaeresis pack is suggested. Ionised calcium levels must be monitored and maintained within the normal range during massive transfusion.

In Chapter References

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