Preoperative Assessment of the Acute Critically Ill Trauma Patient in the Emergency Department

4

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Key Points

- In the acutely ill trauma patient, a rational, sequential, and rapid approach is mandatory: time is critical but rationality should always be used.
- To secure the airway is a "class one priority": a delay in securing the airway may lead to rapidly progressing hypoxemia and risk of life-threatening complications.
- A comprehensive approach to manage hemodynamic instability is warranted, including management of hemorrhagic hypovolemic shock, hemodilution, hypothermia, coagulopathy, electrolyte abnormalities, and acid-base derangements and alternative etiologies (if present) of shock after trauma.
- The role of ultrasound (US) in the acute care of trauma is rapidly evolving and gaining a pivotal role: it should be implemented and expanded according to the most recent guidelines.
- Acute coagulopathy of trauma (ACT) is receiving an ever-increasing attention due to the relevant impact researches on the outcome: viscoelastic tests (TEG/ROTEM) seem to have a relevant impact on the management of the bleeding trauma patients. Antifibrinolytics are becoming a mainstay in the trauma management.

4.1 Introduction

Trauma, defined as a "serious bodily injury or shock caused by an external source," is nowadays a leading cause of morbidity and mortality worldwide, and it is predicted to become the third largest contributor to the global burden of disease by 2020. The initial evaluation of an acute critically ill trauma patient is a challenging task, particularly in the bleeding, unstable patient. Time is of utmost importance (Grade 1A in the last release of the European guideline on management of major bleeding and coagulopathy following trauma) [1]: in some cases, seconds make the difference between life and death [2, 3]. A great number of traumatized patients require emergent resuscitation, surgical management for temporary stabilization (or definitive treatment) of injuries, and perioperative critical care management [1, 2]. Accordingly, the time available for preassessment varies, and a multidisciplinary approach is warranted to care for individuals who have suffered a severe traumatic injury: anesthesiologists, as perioperative care physicians, should have, when present, a leading role in managing trauma patients, enabling early airway management, appropriate and timely resuscitation, basic (or advanced) neurologic evaluation, hypothermia prevention, analgesia and sedation as needed, and transfer of the patient to the operating room without delay [2, 3].

The main goals of preoperative assessment in the trauma setting are:

1. To evaluate the patient's conditions
2. To determine if lab or instrumental tests are needed (testing should be performed based on clinical suspicion and limited to those able to change the management)
3. To estimate the risks
4. To consider any intervention able to reduce or impact on the perioperative surgical and medical risks

Depending on the urgency, the interventions are categorized since 2004 in lifesaving, urgent, expedited, and elective

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[4]. Among interventions able to reduce injuries from the environmental exposure, hypothermia (defined as a core temperature below 35 °C, mild hypothermia being classified as a core temperature between 32 and 35 °C) should be addressed and treated immediately or, if possible, prevented. As a matter of fact, hypothermia may heavily impact on both coagulopathy and the development of multiple organ dysfunction [3]. Whenever possible, a medications list, particularly of drugs able to impact on hemostasis (antiplatelet, anticoagulant drugs and particularly the new direct anticoagulants) and the cardiovascular system (antihypertensives, particularly ACE inhibitors and beta-blockers), should be obtained.

4.2 Pulmonary Assessment

In case of emergency, surgical or interventional procedures, postoperative pulmonary complications, (reported in up to 6% of the cases after major abdominal surgery), along with cardiac, thromboembolic, and infectious complications, are among the major postoperative medical sequelae: they contribute significantly to overall perioperative morbidity and mortality in the acute trauma setting [4, 5] and are almost always associated with longer hospital stay, higher treatment costs, and worst outcome [6].

4.2.1 Airway Assessment

Following the ABC algorithm, to secure the airway is the priority. This is especially true in acute critically ill patients who are at risk for respiratory distress or aspiration. In specific trauma patients (e.g., patient with head and midface trauma or neck injuries), a fast and accurate evaluation of the airway might be lifesaving. Every patient should be assessed for mask ventilation, tracheal intubation, and / or surgical airway access. As most of these patients are at increased risk for aspiration, the supraglottic airway devices play a minor role in the airway management. Since the LEMON score has been included in the 8th edition of the *Advanced Trauma Life Support* (ATLS) Manual in 2009, it should be incorporated as a standard in the assessment of the airway of every trauma patient (or at least an effort has to be done) [3, 7] (Table 4.1).

Commonly used criteria for endotracheal intubation (ETI) include:

1. Inability to maintain airway or oxygenation
2. Respiratory distress (also without hypoxia or hypoventilation)
3. Moderate cognitive impairment

Table 4.1 LEMON score

L	Look externally (Facial trauma, large incisors, beard or mustache, large tongue)
E	Evaluate the 3-3-2 rule – Incisor distance: 3 FB – Hyoid-mental distance: 3 FB – Thyroid-to-mouth distance: 2 FB
M	Mallampati score ≥ 3
O	Obstruction Presence of any condition like epiglottitis, peritonsillar abscess, trauma
N	Neck mobility (limited neck mobility)

4. Glasgow Coma Scale < 9
5. Inability to protect the airway against aspiration
6. Unstable midface trauma and/or airway injuries

Furthermore, early ETI may be indicated in cervical spine injury with any evidence of respiratory failure. Complete cervical spinal cord injury or incomplete injury above C6 will lead, due to a decreased innervation of the respiratory muscles (especially intercostal muscles), to breathing difficulties. Instead, lesions above C3 determine complete and immediate respiratory arrest. Eligible patients undergo an immediate rapid sequence intubation and/or surgical airway access, depending on the underlying trauma [8]. Any form of cervical spine pathology should be excluded if to secure the airway is not time-critical and an emergent priority: even if rare, intubation-related cervical spine injuries have been described [3, 9, 10]. If cervical spine injuries cannot be excluded, manual in-line stabilization (MILS) is the gold standard, as direct laryngoscopy might become more difficult if not impossible. Videolaryngoscopes are well-recognized alternatives, often offering, compared to conventional direct laryngoscopy, a better view. However, a possible longer intubation time has to be considered [8]. Other devices for a backup plan might include flexible fiberscope or optical stylets such as Bonfils or Bullard: all are mentioned in international guidelines [8, 11, 12]. However, all those alternatives imply an experienced user and a laryngeal view not compromised by blood or other secretions [3]. More important, indirect optical devices may be useless in ongoing bleeding, as to be expected, for example, in midface or neck injuries. According to ASA (American Society of Anesthesiologists) recommendations [2, 12], in case of problematic intubations, alternatives include (but are not limited to) combinations of solutions or techniques: among them, laryngeal mask airway as an intubation conduit (with or without flexible scope guidance), flexible scope intubation, intubating stylet or tube changer, and light wand. In patients with maxillofacial, laryngeal or tracheal trauma, oral or nasal blind intubation is discouraged, and surgical airway facilities should be immediately available [3]. For the most updated algorithms, see those endorsed by ASA [2, 8, 12].

4.3 Assessment of Breathing

4.3.1 Physical Examination

The primary tool for diagnosis and differential diagnosis is still the physical examination. Auscultation may display normal bronchial bilateral breath sounds, crackles, pleural rubs, wheezing, or silent chest. Respiratory depth and rate have to be determined, chest wall asymmetry and paradoxical chest wall motion are to be evaluated, and the patient has to be examined for penetrating wounds, seat belt, or steering wheel marks. Chest wall tenderness or other signs indicating rib fractures have to be excluded; subcutaneous emphysema or tracheal deviation has to be ruled out. Hyperresonance or dullness to chest percussion may indicate tension pneumothorax or hemothorax. Studies give evidence that experienced users in lung ultrasound (LUS) will be able to rule out in a fast, safe, and radiation-free way life-threatening conditions like hemothorax and tension pneumothorax, large pleural effusions, or misplaced tubes [13–15]. Furthermore, lung ultrasound in some situations (e.g., identification of pneumothorax after blunt trauma, misplacement of endotracheal tubes or pleural effusion) might be even superior to conventional imaging techniques [13–16].

4.3.2 Imaging: From Chest X-Ray to Chest Ultrasonography and CT Scan

One of the first routine examinations in trauma patients is still a standard chest radiograph [3]. In the further clinical course, or according to the clinical scenario, plain radiographs may be foregone in favor of immediate chest CT scanning [15]. Although both methods are still important and necessary, chest ultrasonography (US) plays more and more a role in trauma patients, especially in time-critical situations. Among major advantages of US are lack of radiations and immediate availability. Studies give evidence that the quality of radiography and ultrasonography, when US is used by experienced and/or specifically trained examiners, is similar [13–16].

One of the risks in trauma patients is a post-intubation tension pneumothorax, to be immediately ruled out by all means for the dramatic and life-threatening hemodynamic and respiratory effects. The fastest and therefore safest method in the operating theater will be the ultrasonography. For pneumothorax, the sensitivity is 91% and the specificity 98%. In an expert review done by Wilkerson et Stone and quoted by Blanco et al. [14] and Volpicelli et al. [16], the AAs concluded that bedside thoracic US is an even more sensitive screening test than supine AP chest radiography for the detection of pneumothorax in adult patients with blunt chest trauma. Since 2012, the lung US for pneumothorax has been incorporated in the international evidence-based recommendations for point-of-care lung ultrasound [16]. With a *level A* evidence, the signs of pneumothorax include the presence of lung point(s), the absence of lung sliding, the absence of B-lines, and the absence of lung pulse. In the OR, another application for ultrasonography is the confirmation of a correct endotracheal intubation. Particularly in trauma patients, when intubation under direct vision could be difficult, and the help of capnography is lacking, US seems to be a useful tool to confirm correct intubation, as proved in a very recent experience [17].

4.3.3 Gas Exchange

Patients with aspiration, hemo- or pneumothorax, and lung contusion are at extreme risk of developing rapidly progressing hypoxemia and/or acute lung injury [18, 19]. Additional risks may arise from massive transfusion (by transfusion of packed red blood cells, fresh frozen plasma, and/or platelets), volume overload, and fluid shift. These conditions may result in ventilation/perfusion mismatch, increased intrapulmonary shunt, increased extravascular lung water, segmental lung damage, and/or loss of compliance, further aggravating pulmonary gas exchange [18–20]. Protective ventilation is warranted in this scenario.

4.3.3.1 Monitoring Gas Exchange and Acid-Base Equilibrium (ABE)

(a) **Arterial Oxygen Saturation (SaO_2)**—Continuous oxygen saturation monitoring should be standard in all trauma patients. In case of mechanical ventilation, $SaO_2 > 92$ is the target [8].
(b) **End-tidal Carbon dioxide ($EtCO_2$)**—$EtCO_2$ monitoring should be used in all intubated patients. In non-intubated patients the micro-stream, nasal capnographic monitoring as a semi-quantitative measurement should be considered both during spontaneous and, when indicated, during noninvasive ventilation (NIV).
(c) **Blood Gas Analysis (BGA)**—As in the acute critically ill patients, arterial line placement should be standard. An experienced provider, guided by US in problematic or difficult cases, should insert the arterial line within few minutes (often less than one). Intermittent blood gas analysis immediately reveals changes in oxygenation and acid-base balance. Base excess (BE) and lactate blood level provide a way to assess the severity of shock. Blood gas analysis and acid-base equilibrium allow within seconds decisions concerning ventilation, replacement of volume, administration of electrolytes, and/or packed red blood cells (PRBCs). Metabolic acidosis is not infrequent in the

hyperacute setting: adequate fluid resuscitation and, when needed, pressor or inotropes (and not bicarbonate!) are the mainstays to restore blood pressure and to maintain tissue perfusion [8, 15].

4.3.4 Lung Protective Ventilation

Patients intubated in the prehospital triage have to be assessed for adequate ventilation. Most of the trauma patients, particularly those with accompanying blunt chest trauma due to fall from height or motor vehicle accident, suffer from single or multiple, uni- or bilateral rib fractures, flail chest, hemothorax or tension pneumothorax, as well as lung contusion: they require immediate intubation and lung protective ventilation, to be continued both in the OR and in the ICU [8, 21]. Ventilation parameters have to be assessed and, if necessary, appropriately adjusted. Typically, the ventilator settings would require tidal volume of 6–8 mL/kg (predicted body weight) and a respiratory rate (RR) of 12–16 breaths/min using a volume- or pressure-limited ventilation. RR at 8–10 breaths/min, with adequate expiratory time to reduce air trapping (i.e., inspiratory-to-expiratory [I:E] ratio of 1:3), might also be considered, allowing a permissive hypercapnia ($PaCO_2$ 40–45 mmHg) if not otherwise contraindicated (e.g., evidence of brain injury or metabolic acidosis) [8]. Mean airway pressure should be kept as low as possible and ideally below 25–30 mmHg. An individualized "open-lung PEEP" (OL-PEEP, the level resulting in maximal dynamic compliance during a decremental PEEP trial) should be applied [21, 22]. According to some Authorities [8], in the specific setting of trauma, no PEEP has to be used until having achieved a hemodynamic stability after adequate resuscitation. After stabilization, PEEP could be increased to 5–10 cm H_2O, main being the balance between minimization of lung injury and hemodynamic optimization. FiO_2 should be titrated aiming at SaO2 > 92% [8, 21, 22].

4.3.4.1 ECMO: The Case for Very Selected Indications

Evaluation of patients who are "critical" concerning the pulmonary gas exchange should be immediately performed, and their referral to extracorporeal membrane oxygenation (ECMO) team might be considered [23, 24]. As a matter of fact, its use in ARF after trauma is controversial and seldom reported [24]: the decision to activate the ECMO team differs from center to center. Nevertheless, accepted criteria refer commonly to the Murray score (see Table 4.2) [18]. Usually patients with potentially reversible causes and a Murray score ≥ 3.5 might benefit from ECMO and should be discussed with the ECMO team [23, 24]. According to the most recent experience, mortality was higher in patients with high injury severity score and lower arterial pH on arrival. Ability to tolerate systemic anticoagulation was associated with improved survival [24].

Table 4.2 Murray score

Point(s)	0	1	2	3	4
PaO2/FiO2 FiO2 100% For 20 min	≥300	225–299	175–224	100–174	≤100
PEEP	≤5	6–8	9–11	12–14	≥15
CRX, number of quadrants infiltrate	0	1	2	3	4
Compliance (cm H_2O)	80	60–79	40–59	20–39	≤19

4.3.5 Previous History

Underlying injuries, especially neck or midface injuries, may comprise the airway and/or breathing capacity of a trauma patient. Besides that, additional preexisting patient-related factors can aggravate this condition. Therefore, if possible, the past and present history of the patient should be taken, to be aware of other comorbidities able to complicate perioperative ventilation and/or oxygenation further. Age >50 years [3, 4], chronic obstructive pulmonary disease [25], congestive heart failure, poor general health status (American Society of Anesthesiologists [ASA] class >2), obstructive sleep apnea, smoking [26], pulmonary hypertension [27–29], low oxygen saturation, and serum albumin < 3.5 g/dL are patient-related risk factors able to heavily impact on postoperative pulmonary complications [5]. As in every kind of risk assessment in surgery, the patient's comorbidities and the type surgery are the two main drivers able to qualify the patient at "high risk." Emergency surgery increases the risk for pulmonary complications above average [5, 30]. The incidence of complications is inversely related to the distance of the surgical incision from the diaphragm [5]. Thus, the complication rate is significantly higher for thoracic and upper abdominal surgery than for lower abdominal and all other procedures [31].

Therefore close communication between anesthesia, surgery, radiology, and operating room staff should be continuous, concise, clear, and guaranteed: a dedicated communication training is suggested by some authors [3, 8].

4.4 Circulation (Cardiac) Assessment

Among the causes of shock (classically defined as the acute imbalance between oxygen supply and demand but practically defined in the trauma setting as an acute, critical hypotension) in the critically ill acute trauma patient, hemorrhage

is reported as the most common [1]. Classified in four classes, hemorrhage becomes critical when blood losses involve ≥30% of the blood volume (class III), with critical fall of systolic blood pressure [15]. Chest, abdomen, retroperitoneum, and thighs are reported to be the most frequent sites of (massive) hemorrhage [3]. In case of urgent surgical intervention, anesthesia and the surgical procedure can influence cardiac performance in several ways. Decreased cardiac preload due to blood loss, fluid shifts, and compressed venous blood flow and afterload changes due to anesthetic agents, bowel manipulation, pulmonary emboli, as well as stress on cardiac compensation caused, for example, by stress, pain, tachycardia, and/or anemia can adversely impact on intraoperative hemodynamics. Other potential causes of hemodynamic impairment to be considered (and possibly ruled out) are cardiac tamponade, tension pneumothorax, hemothorax, spinal cord injury, and fat or air emboli [15]. Initially, detailed patient history (including drugs taken by the patient), skin temperature, and capillary refill (press centrally for 5 s and release: normal color should return within 2 s) and, if possible, auscultation and electrocardiogram (ECG) should be the first steps in evaluating a trauma patient [8, 15].

4.4.1 Auscultation

Besides all types of heart murmurs, which might be present in any patients, special attention has to be paid to patients with blunt chest trauma. Patients with suspected myocardial contusion might display, besides usually very unspecific signs, *pulsus paradoxus*, muffled heart sounds, and pericardial friction rub. This is the setting where US, the real new millennium stethoscope, could make the difference.

4.4.2 EKG

When possible, a 12-lead EKG should be recorded in every trauma patients: on the one hand to exclude acute conditions (pulmonary embolism, myocardial infarction, cardiac contusion, electrolyte imbalances), on the other hand as a baseline for future comparison. In suspected cardiac contusion, EKG is a class I recommended test in the initial treatment of a patient with significant chest trauma. One might get a hint if the probability of a traumatic contusion is high [31–34]: as a matter of fact, 40–83% of the patients will have an abnormal EKG. There is no correlation between the severity of contusion and any kind of arrhythmia. Most likely contusion of the left ventricle will present as an abnormality of ST-T and pathological Q waves. Also common is a right bundle branch block, whereas AV-Block I°, left bundle branch block, hemiblocks, and AV-Block III° are less common.

4.4.3 Biomarkers

In general serum lactate, arterial blood gas analysis, and renal and liver function tests are included in an emergency routine [8]. Measuring biomarkers (troponin I and troponin T, N terminal pro BNP) initially and after a 4–6-h interval, particularly in patients with blunt chest trauma, belongs to the tests with negative predictive value of class II and should be assessed [35–38].

4.4.4 Echocardiography

If ECG and the results of biomarkers are not within normal limits, further investigations (mainly echocardiography) are recommended as a class II recommendation and should be considered [39]. In *time-critical cases,* the bedside cardiac ultrasound is a useful tool in the emergency environment [14]. The subxiphoid four-chamber view is an excellent cross-longitudinal section of the heart. The size of the right ventricle can easily be compared to the left ventricle size. Contractility of each chamber, mitral valves, and the descending thoracic aorta in cross section next to the left atrium can be evaluated. Simple question concerning the overall activity of the heart as well as a possible pericardial effusion/tamponade can be ruled out or confirmed quickly and precisely. Adequate image quality on transthoracic echocardiography (TTE) may be technically challenging in the acute trauma patients. Consequently, TTE might be required in some of the more acutely ill patients and have relevant information also of the aortic pathology (i.e., dissection). In *less time-critical cases,* and if and when possible, transesophageal echocardiography (TEE) could find its strength in the emergency department for the evaluation of hemodynamically unstable patients, providing selective functional and anatomic information to differentiate the underlying disturbance, being perhaps superior to other invasive monitors. As a semi-invasive method to assess the left ventricular (LV) volumes and ejection fraction, TEE provides fast information about heart pump function, acute myocardial infarction (MI) or pulmonary embolism (PE), heart and systemic volume status (hypovolemia), obstructive flow patterns (dynamic LV flow obstruction, cardiac tamponade), and unexplained hypoxemia. Because the management of MI, PE, hypovolemia, or obstruction is different, the early placement of TEE can significantly assist in forming and optimizing the treatment plan. TEE has already been well established for patient monitoring during cardiac surgery. In 2010 the American Society of Anesthesiologists updated their practice guidelines for perioperative TEE use [40]. Recommendations for TEE use for non-cardiac surgery were established. The American Society of Anesthesiologists (ASA) practice guidelines recommend the use of TEE when the nature of surgery or the patient's

known or suspected cardiovascular pathology might result in severe hemodynamic, pulmonary, or neurologic compromise. TEE should be used when unexplained life-threatening hemodynamic instability persists despite corrective therapy if equipment and expertise are available. According to the ASA, patients with potential benefit of TEE monitoring might include those with known or suspected cardiovascular compromise, patients with unexplained persistent hypotension, or unexplained persistent hypoxemia, as well as patients with major thoracic or abdominal trauma. However, depending on comorbidities, clinical condition and surgical invasiveness, intraoperative TEE can help to optimize perioperative care, assessing LV function and volume load, recognizing early regional wall motion abnormalities, and guiding fluid, vasopressor or inotropic therapy [41].

4.4.5 Evaluation of Volume Status and the Impact of Renal Function

Assessment of the volume status is of the utmost importance and one of the most challenging tasks in the acute critically ill trauma patient. Fluid resuscitation with respect to optimal volume and type of fluids is under constant debate [15]. Strategies which include delayed fluid resuscitation and/or controlled hypotension are relevant in some trauma scenarios but, according to very recent reviews, are to be taken and applied wisely (cum grano salis!) [42]. On the contrary, volume overload might have a devastating impact not only on kidney function but also on the overall mortality. It is known that acute kidney injury (AKI) develops in 55–66% of critically ill patients with a mortality rate ranging 15–80%, depending on the severity of renal dysfunction [43, 44]. Recent reports highlight that fluid overload is an independent risk factor for AKI in the critically ill patients. It is biologically plausible that volume overloading leads to organ congestions thus resulting in decreased renal blood flow [45–50]. Furthermore, it has been shown that fluid overload is related to an increase in overall mortality with critical illness. If both factors are combined, fluid overload in an already established AKI has been correlated with lower renal functional recovery and lower survival [51, 52]. Patients with chronic kidney disease (CKD) are at increased risk of perioperative morbidity and mortality. Preexisting chronic kidney diseases can be ruled out reviewing serum creatinine, glomerular filtration rate, and blood urea nitrogen (BUN). Acute trauma and urgent/emergent trauma surgery increase the risk of acute kidney injury (AKI). In trauma patients, an indwelling urinary catheter is the rule, allowing, on the one hand, an immediate detection of urinary tract injuries and, on the other hand, continuous measurement of urine output (UO). In a large ICU population, intensive UO monitoring was associated with improved detection of AKI and reduced 30-day mortality in patients developing AKI; less fluid overload was also reported for all monitored patients [53].

Among the methods used to assess volume status, measurement of central venous pressure (CVP)**,** equivalent to right atrial pressure (RAP) when the vena cava is continuous with the right atrium, is still considered a standard of monitoring [54, 55]. This is confirmed by studies, which give evidence that large part of the anesthesiologists uses the CVP to assess volume status and to guide volume therapy and fluid management decisions [54, 55]. This in spite of some relevant studies clearly shows limitations of this use of CVP in this setting [56]. Other concerns expressed by experts are the risk of complications (among them, infection, catheter-induced thrombosis, and arrhythmias). Furthermore, occasionally, in time-critical situations or in very peculiar conditions when central venous catheterization is not possible, the use of noninvasive or dynamic techniques to assess the volume status is crucial to rule out (or in..!) hypovolemia [57]. As a matter of fact, preload static parameters such as CVP or wedge pressure may not predict volume status and/or fluid responsiveness. RAP is related to venous return, which in turn depends on venous capacitance gradient. Cyclic changes of ventricular filling induced by increased intrathoracic pressure variations and heart-lung interaction during mechanical ventilation lead to delta-up and delta-down variations of stroke volume and inferior vena cava (IVC) size. Fluid responder is usually defined the patient whose stroke volume increases by 10–15% following a 3 mL/kg (usually 250 mL) crystalloid or colloid fluid bolus. Dynamic parameters (systolic pressure variation, SPV; stroke volume variation, SVV; pulse pressure variation, PPV), instead of the time-honored "static" parameters (CVP is an example), are in the last few years on the rise both in anesthesia and ICU settings [58]. An alternative and quick method to assess the volume status is the sonographic assessment of the venous collapsibility index (VCI) using respiratory variation of IVC (inferior vena cava) diameter. VCI measures the fractional change in major venous diameters through the respiratory cycle rather than relying on a single measurement of venous diameter. Results of a recent review article confirm that sonographic measurement of IVC diameter and collapsibility is under certain circumstances a valid method of estimating CVP and RAP (right atrial pressure) [59]. Technically, the measurement of the IVC diameter can be performed in the subxiphoid, lateral, and even supra-iliac position. However, ICV diameter can be affected by high intra-abdominal pressure and is therefore limited in patients with blunt abdominal trauma or patients scheduled for emergency re-laparotomy. In case of problematic conditions, superior vena cava (SVC) collapsibility index might be an alternative: SVC needs of course of a TTE monitoring, neither always possible in the trauma setting not everywhere available. Vignon et al. in a very recent experience tried to identify responders to volume expansion using 10 mL/kg of colloid solution among septic shock patients under mechanical

ventilation. Using for SVC a threshold of 36%, collapsibility had sensitivity of 90% and specificity of 100% in detecting an 11% rise in cardiac index after volume expansion [60].

4.5 Neurologic Assessment

Once addressed and, if possible, solved the problems associated with the safety of the airway, breathing, and circulation, a focused neurologic examination is needed. Neurologic assessment should be performed according to the Glasgow Coma Scale (Table 4.3), able also to grade the severity of neurological impairment. Also to be addressed any lateralizing signs and the level of sensation if a spinal cord injury is present (Table 4.4).

Further assessment should include pupillary size and responsiveness. In less time-critical cases, assessment of gross motor and sensory function in all four limbs should be carried out. If a spinal cord injury is present, a full neurological assessment is vital at the earliest opportunity—an example is the check for priapism, loss of anal sphincter tone, and the bulbocavernosus reflex.

For the experienced physician, most of the cranial nerves can be easily assessed as reported below (Table 4.5).

In conscious trauma patients, motor function can also be easily assessed using the "American Spinal Injury Association (ASIA) Motor Key" (Tables 4.6 and 4.7).

Table 4.3 Glasgow Coma Scale (GCS)

Behavior	Response	Score
Eye opening response	Spontaneously To speech To pain No response	4 3 2 1
Best verbal response	Oriented to time, place, and person Confused Inappropriate words Incomprehensible sounds No response	5 4 3 2 1
Best motor response	Obeys commands Moves to localized pain Flexion withdrawal from pain Abnormal flection (decorticate) Abnormal extension (decerebrate) No response	6 5 4 3 2 1
Total score	Best response Comatose patient Totally unresponsive	15 8 or less 3

Table 4.4 Level of consciousness according to GCS

GCS 13–15, mild	Normal to lethargic, mildly disoriented
GCS 9–13, moderate	Lethargic to obtunded, follows commands with arousal, confused
CGS 3–8, severe	(Comatose, no eye opening or verbalization, does not follow commands with arousal)

Table 4.5 Cranial nerve assessment

Cranial nerves (CN)	
CN I	Olfactory Nerve
	Non-noxious stimulus (e.g., coffee) testing each nostril separately
CN II	Optic nerve (visual fields, pupillary reaction to light)
CN III, IV, VI	Oculomotor, trochlear, abducens
CN V	Trigeminal nerve (motor and sensory divisions to be tested)
CN VII	Facial nerve (facial expression and taste sensation)
	Muscles for facial expression (smile and wrinkle the brow)
CN VIII	Vestibular-auditory nerve (snapping the fingers together to compare the two sides)
CN IX, X	Glossopharyngeal and vagus nerves IX, mainly sensory; X, mainly motor
Gag reflex:	Test the posterior third of the tongue and oropharynx
Hoarseness:	CN X or lesion nucleus ambiguous
Unilateral lesion CN X:	Uvula deviation
Bilateral lesions CN X:	Palate elevates poorly when saying "Ah" but rapidly when gag reflex elicited
CN XI	Spinal accessory (ipsilateral SCM and trapezius muscle)
	(weakness of turning the head to the opposite side)
CN XII	Hypoglossal nerve
	Tongue protrudes toward the side of lesion

Table 4.6 Function and muscles involved in movements

Root	Function	Muscle
C5	Elbow flexors	Biceps
C6	Wrist extensors	Extensor carpi radialis longus and brevis
C7	Elbow extensors	Triceps
C8	Finger flexors	Flexor digitorum profundus
T1	Finger abductors	Abductor digiti minimi
L2	Hip flexors	Iliopsoas
L3	Knee extensors	Quadriceps
L4	Ankle dorsiflexors	Tibialis anterior

Table 4.7 Muscle grading

Grade	Strength
0	Total paralysis
1	Palpable or visible contraction
2	Active movement, full range of motion, gravity eliminated
3	Active movement, full range of motion, against gravity
4	Active movement, full range of motion, against gravity, and provides some resistance
5	Active movement, full range of motion, against gravity, and provides normal resistance
NT	Not tested

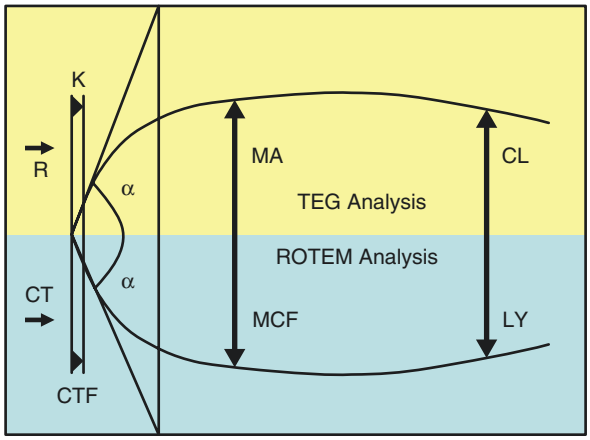
Patients with neurotrauma are at extreme risk. Hypotension (one single systolic blood pressure measurement <90 mmHg) doubles mortality. Hypoxia, defined as apnea or cyanosis, or PaO_2 < 60 mmHg on the arterial blood gas analysis, is also known to increase mortality. Combination of both usually triples mortality in patients with neurotrauma. Patients have to be assessed for neurogenic shock and basal skull fracture. Neurogenic shock refers to a hemodynamic condition characterized by hypotension, bradycardia, and hypothermia: usually occurs in spinal cord injuries above T1. Below the level of injury, loss of vasoconstrictive effect might be lost. Its incidence increases in injuries above T6. As parasympathetic effects are relatively unopposed, this condition usually results in bradycardia, lower systemic vascular resistance, and venous pooling. Basal skull fractures are usually heralded by some characteristic signs and symptoms. Among them (the list is not complete) are Raccoon's eyes, Battle's sign, cerebrospinal fluid rhinorrhea (patients usually describe salty or metallic taste)/otorrhea, hemotympanum, or laceration of external auditory canal.

4.5.1 Coagulation and the Trauma-Associated Coagulopathy

Close to one third of the severely injured trauma patients develop an "evident" coagulopathy with relevant hemorrhage [61–63]. Hypothermia, acidosis, and hemodilution ("the lethal triad") are among the physiologic derangements often present in the acute phase of trauma. Bleeding after major trauma has been associated with hemostatic alterations associated with the "lethal triad": (1) acidosis (due to inadequate tissue perfusion and able to impact in part on coagulation process), (2) hypothermia (due to conduction/convection mainly associated with physical exposure and cold fluids), and (3) hemodilution (multiple hemostatic defects due to large volumes of unbalanced fluids and/or unbalanced administration of blood components). Disseminated intravascular coagulation (DIC) occurs early after the injury and is mainly associated with consumption coagulopathy (increased consumption vs. inadequate repletion) together with diffuse microvascular thrombosis [61]. However, together with DIC, there is an early "biological" response to trauma and shock, the acute traumatic coagulopathy (ATC), not associated with the "triad", and possibly overlapping to DIC. As a matter of fact, trauma-induced coagulopathy (TIC) is defined as the acute traumatic coagulopathy (ATC, a sort of "innate" component) together with "coagulopathies" which further develop, frequently iatrogenic and related to unbalanced fluid resuscitation, suboptimal temperature control, or delayed hemorrhage control [63].

As a matter of fact, according to the most recent theories [61–63], the so-called ATC is evident prior to and independent from the "lethal triad," worsens with hypotension and head injury, and differs from the hemostatic alterations observed in other potentially problematic settings (e.g., liver surgery, cardiac surgery, or obstetrics). When compared with trauma patients without ATC, individuals with ATC show to three- to fourfold greater mortality, particularly in the first 24 h following the trauma [61–63]. ATC should be defined an early and endogenous hemostatic alteration triggered by endothelial hypoperfusion associated with massive tissue injury and characterized by hypocoagulation, hyperfibrinolysis, early and enhanced fibrinogen depletion, and platelet dysfunction [62–65]. Interestingly enough, minor prolongations of clotting times and preserved thrombin generation are associated with reduced clot generation and clot strength, as if an endogenous process could be responsible for the hemostatic derangements. The exact mechanism of ATC is still a matter of debate [61–63, 66, 67]. It is postulated a "switch" of thrombin from the "usual" procoagulant action toward an anticoagulant effect: the switch is sustained by an increase in soluble thrombomodulin levels secondary to endothelial hypoperfusion and glycocalyx disruption [62, 63, 66]. Thrombomodulin-bound thrombin gives rise to an "excess" activation of protein C (activated protein C, aPC), able to cleave FV and FVIII [62, 63]. Fibrinolytic state is enhanced by (1) increase in tissue plasminogen activator (tPA)-mediated conversion of plasminogen to plasmin, (2) decreased plasminogen activator inhibitor-1 (PAI-1), and (3) decreased thrombin-activatable fibrinolysis inhibitor (TAFI). The hyperfibrinolytic state observed in trauma patients with ATC is associated with increased D-dimer level and reduced level of native PC [61].The PC depletion could suggest a potential mechanism to explain, after the early hypocoagulable phase lasting up to 24 h, the second, hypercoagulable phase observed up to 48–72 h. Hypofibrinogenemia and enhanced fibrinolysis are associated with elevated D-dimer and reduced fibrinogen, usually documented with conventional lab tests (D-dimer, Clauss fibrinogen). As abovementioned, increasing severity of injuries correlates with elevated markers of endothelial cell and glycocalyx damage, protein C activation, clotting factor consumption, and fibrinolysis. In spite of that, as reported by Johansson et al., [67] INR and aPTT values (everywhere available and then always to be considered in first-line diagnostics) were within the normal range. Interestingly enough, when prolonged (in 10–30% of the traumatized patients), aPTT was more specific than PT-INR to define the severity of coagulopathy [61, 67].

Nowadays, the viscoelastic approach (more dynamic than conventional lab tests) is increasingly used, due to the relevant advances in thromboelastography/thromboelastometry made possible by the newest release of both TEG (Haemonetics, USA) and ROTEM (TEM International, Germany). Thromboelastography is an "holistic approach," able to provide simultaneously on a small sample of whole or citrated fresh blood and in a short timespan (15 min), information usually obtained from multiple time-consuming tests (PT, PTT, thrombin time, fibrinogen level, and platelet count) regarding clot initiation/formation, clot strength, and (hyper) fibrinolysis [1, 68, 69] (Fig. 4.1) (table of reference values for TEG and ROTEM).



Reference values for TEG					
	r- time reaction time (min)	*K* time (min)	*α*-angle	MA maximum amplitude (mm)	LY lysis (%)
Whole blood	4–8 min	1–4 min	47–74°	55–73 mm	
Citrated blood+ kaolin	3–8 min	1–3 min	55–78°	51–69 mm	
r-TEG	0–1 min	1–2 min	66–82°	54–72	0.0–7.5
Citrated blood+ kaolin and heparinase	Comparison with kaolin test: a shortening in CT in kaolin and heparinase indicates the presence of heparin or heparin-like substances				
ROTEM	EXTEM (tissue factor)		Extrinsic activator (tissue factor); not affected by aprotinin, sensitive to heparin; fast assessment of clot formation		
	INTEM (contact activator)		Intrinsic activator: sensitive to heparin: assessment of clot formation and fibrin polymerisation		
	HEPTEM (contact activator + heparinase)		Neutralises heparin effects; specific detection of heparin		
	FIBTEM (tissue factor+ platelet antagonist cytochalasin D)		Pharmacologic inactivation of platelet cytoskeleton by cytochalasin D; FIBTEM represents the strength of the clot under platelet inhibition		
	APTEM (tissue factor + aprotinin)		Inhibition of premature lysis by the addition of aprotinin: fast detection of fibrinolysis when compared at the same time to EXTEM		
	ECATEM (ecarin)		Used in the management of direct thrombin inhibitors		

Reference ranges for ROTEM					
	CT Clotting time (s)	CFT Clot formation time (s)	A 10 Amplitude 10 min after CT (mm)	MCF Maximum clot firmness (mm)	ML Maximum lysis (% of MCF)
INTEM	100–240 s	30–110 s	44–66	50–72 mm	<15 %
EXTEM	38–79 s	34–159 s	43–65	50–72 mm	<15 %
FIBTEM			8–24	9–25 mm	
APTEM	Comparison with EXTEM: a stable MCF confirms hyperfibrinolysis				
HEPTEM	Comparison with INTEM: a shortening in HEPTEM CT indicates the presence of heparin or heparin-like substances				

Fig. 4.1 Nomenclature, reference values, and "signatures" of TEG and ROTEM [69]

In trauma patients, early traces show a reduction in clot amplitude (reduced MA) and reduced alfa angle. Low fibrinogen (fibrinogen monitoring is available on both TEG and ROTEM) in turn can also influence the platelet function thus contributing to the reduced MA (Fig. 4.2).

Thromboelastography/elastometry is the only way to clearly demonstrate hyperfibrinolysis (both primary or secondary) (see Figs. 4.3 and 4.4) for TEG and ROTEM hyperfibrinolysis traces.

Also possible is the demonstration of a "laboratory detectable autoheparinization" (heparin-like effect) due to liberation of endothelial components (glycosaminoglycans): this condition could be documented in vitro and reversed with heparinase. The clinical meaning and the correction of heparin effect in specific surgical or medical settings is under discussion (Fig. 4.5).

According to what was previously discussed, increased fibrinolytic activity and rapid fibrinogen depletion character-

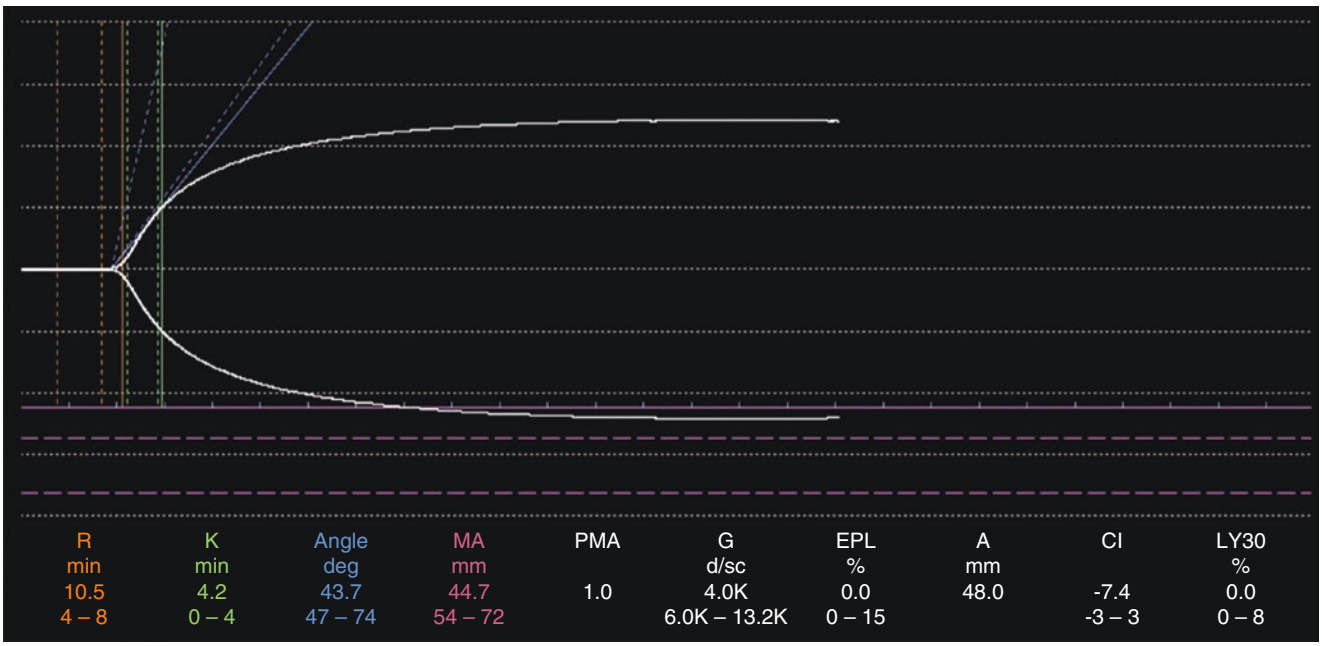


Fig. 4.2 TEG in a trauma patient (upon ED arrival)

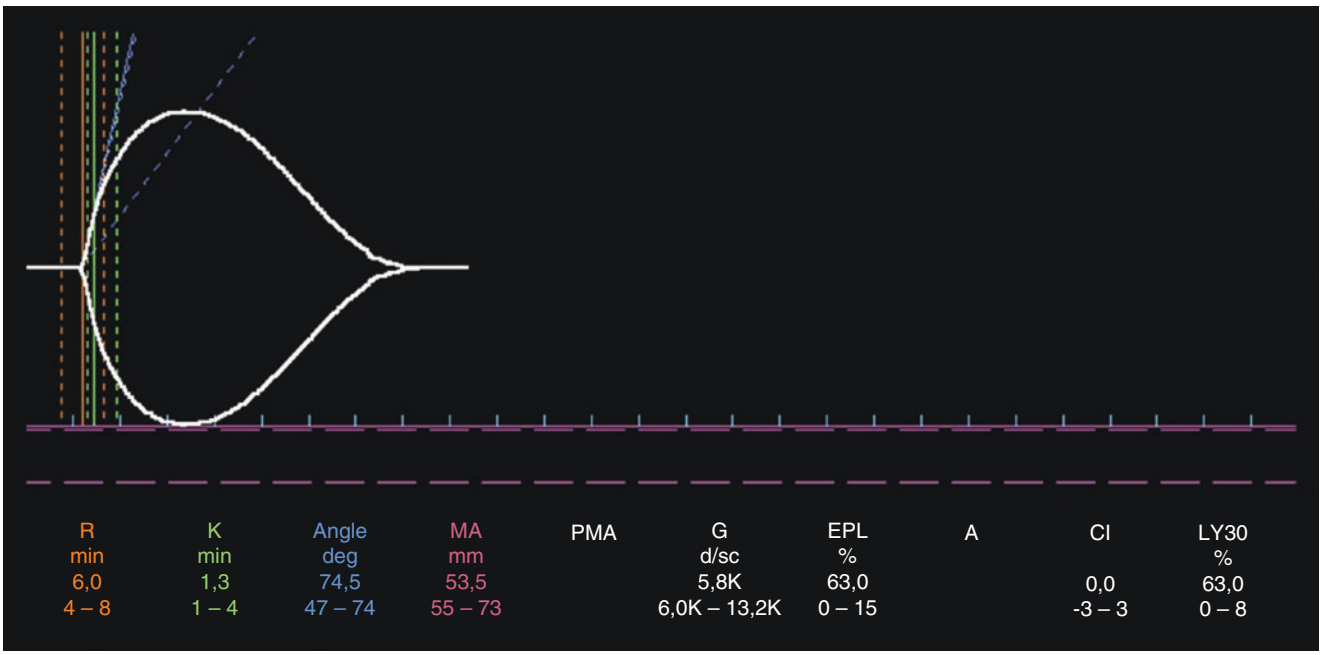


Fig. 4.3 Hyperfibrinolysis on TEG

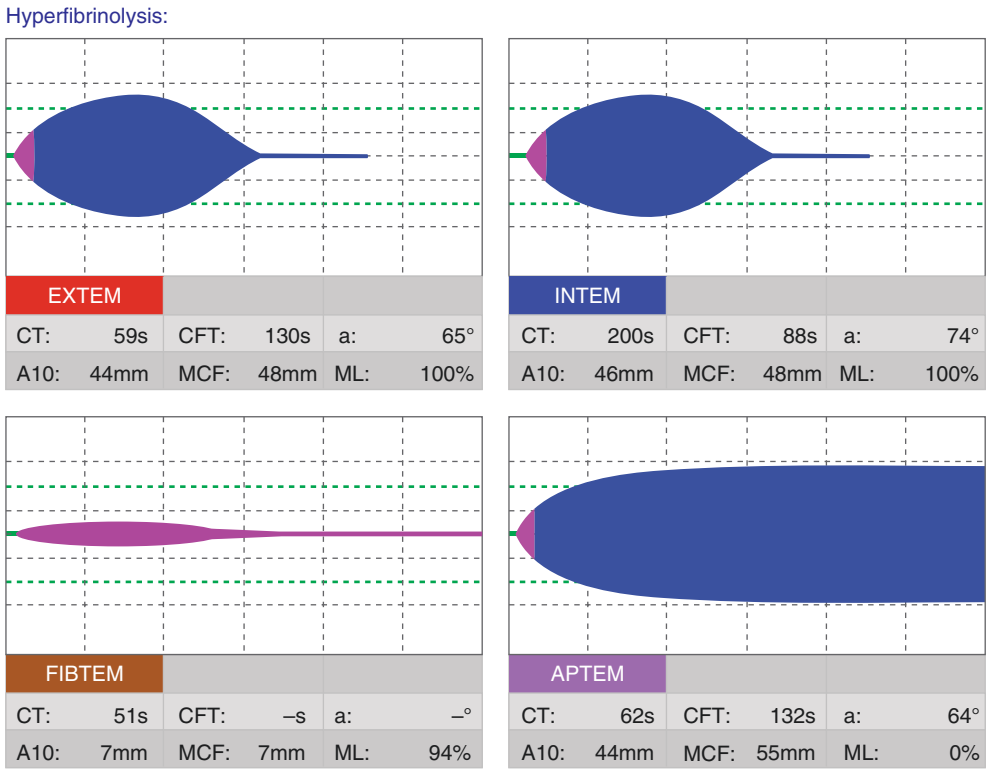


Fig. 4.4 Hyperfibrinolysis on ROTEM

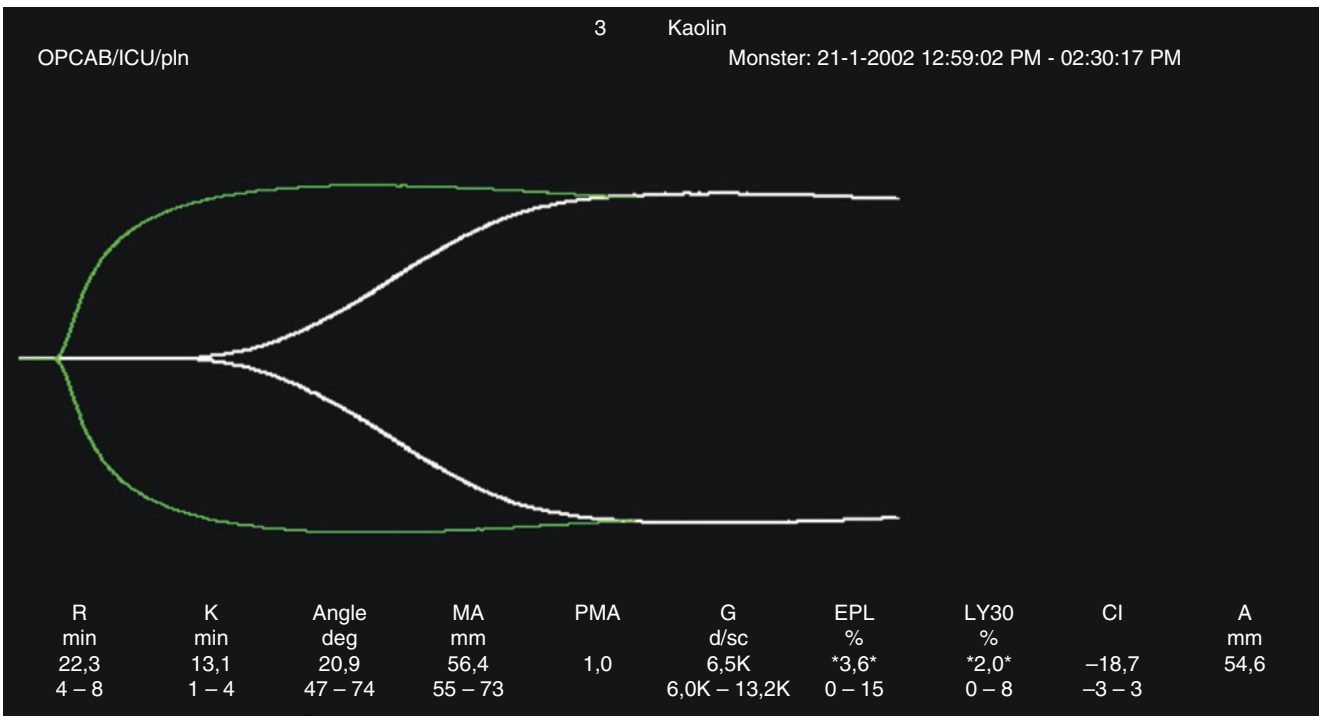


Fig. 4.5 Heparin-like effect (white line) and correction with heparinase (green line)

ize the ATC: as very recently demonstrated by Davenport et al. [62, 63], PC pathway should provide a "mechanistic link between the two main theories of early trauma coagulopathy", (1) DIC with fibrinolytic phenotype and (2) primary aPC-driven fibrinolysis (and possible fibrinogenolysis) [62, 63]. As stated by Davenport et al. [62], a unifying hypothesis defined by aPC generation and widespread fibrinolytic activation provides support to the improved outcomes

in trauma patients (both in cohort studies and in randomized controlled trial) when the early empiric administration of an antifibrinolytic drugs was used [69]. Since long time antifibrinolytic drugs were available to minimize blood loss in various surgical setting (orthopedic, cardiac, liver transplant surgeries, obstetrics). Since the publication of the CRASH-2 trial (the first randomized, placebo-controlled, multicenter trial of tranexamic acid in trauma patients) [68, 70], it is standard to infuse 1 g of tranexamic acid (TXA) over a 10-min period followed by an infusion of another gram of TXA over an 8-h period. This has been incorporated as recommendation number 25 (Grade 1A recommendation) in "The European Guideline on the management of major bleeding and coagulopathy following trauma: fourth edition" [1]. This, as well as the infusion of fibrinogen, is standard of care in most of the centers dealing with trauma patients. However, once the main source of bleeding is controlled by damage control surgery (DCS) and additional factors like hypothermia and acidosis (together with hypotension, the already mentioned components of the "lethal triad") are excluded or corrected, TEG or ROTEM are excellent tools to guide further administration of blood products. It turned out to be of extraordinary value in patients with massive crush injury (e.g., caused by rollover), where one or more extremities are without blood supply for several hours. Once the supplying vessels are reanastomosed, the reperfusion may severely compromise the coagulation within minutes, leading to significantly prolonged R-times (reperfusion syndrome or ischemia-reperfusion injury).

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