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REVIEW ARTICLE

# Surgical intensive care unit—the trauma surgery perspective

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**Abstract**
*Purpose* This review addresses and summarizes the key issues and unique specific intensive care treatment of adult patients from the trauma surgery perspective.
*Materials and methods* The cornerstones of successful surgical intensive care management are fluid resuscitation, transfusion protocol and extracorporeal organ replacement therapies. The injury-type specific complications and unique pathophysiologic regulatory mechanisms of the traumatized patients influencing the critical care treatment are discussed.
*Conclusions* Furthermore, the fundamental knowledge of the injury severity, understanding of the trauma mechanism, surgical treatment strategies and specific techniques of surgical intensive care are pointed out as essentials for a successful intensive care therapy.

**Keywords** Surgery · Intensive care unit · Trauma · Injury · Critical care · Polytrauma

## Introduction

In the industrialized world, trauma is the leading cause of death and disability in patients less than 45 years of age [1, 2]. The temporal profile of trauma-related mortality clearly displays clearly two peaks with an early and late mortality. Clinical problems in the early phase after trauma are predominantly due to exsanguination, refractory hemorrhagic shock, respiratory failure and traumatic brain injury (TBI). In the later phase, inflammatory-related diseases, such as systemic inflammatory response syndrome (SIRS) and sepsis, leading to capillary endothelial leakage, are typically observed. Inflammation and shock-induced microvascular perfusion failure frequently results in organ- [3] or multiple (remote) organ failure (MOF), which can be viewed as the triggering pathways for late trauma mortality. Therefore, restitution of normal physiology, i.e. stabilization of the circulatory macrohemodynamic situation, recovery of capillary microcirculatory perfusion with maintenance of tissue oxygenation and most importantly, restoration of respiratory/pulmonary function are the main goals of intensive care treatment during the early stage after trauma [1].

The intensive care of trauma patients has unique and specific aspects with staged treatment phases that markedly differ from those in non-trauma critical care patients, underscoring that a trauma-specific intensive care unit (ICU) is associated with improved clinical outcome [4, 5]. The specific features that have led to substantial improvement in outcome and standard of intensive trauma care in the last three decades include [6]:

- Increased quality in prehospital trauma care
- Permanent integration and availability of surgical therapy, resources and expertise in an interdisciplinary approach with establishment of specific trauma ICUs
- Enhanced understanding of the posttraumatic pathophysiology with implementation of organ replacement therapies
- Consideration of time course in posttraumatic systemic inflammation with introduction of the "damage control" principle concerning timing and performance of surgical interventions

Thus, the ICU has proven to be the optimal place for resuscitation and restoration of physiology after initial life-

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saving surgery and damage control procedures following trauma [7]. However, adequate, effective and outcome-orientated intensive care medicine is very expensive, often conflicting with requirements of cost-effective economic management concepts. This problem becomes even more obvious and relevant due to the current trend of an increasing demand in critical care capacity and ICU personnel staff in the future [8]. The most common diseases requiring intensive care treatment after trauma are acute lung injury and severe sepsis [8]. In this review, we evaluate the available literature on critical care medicine in adult trauma patients using online PubMed-listed research.

The intensive care treatment of trauma patients is divided into two major phases:

*Resuscitation phase (<24 h after trauma)* Effective blood volume is restored and tissue perfusion is optimized. Life- or limb-threatening injuries should be excluded and sufficiently treated in this phase. During the first 24 h, life-saving surgeries such as thoracotomy, laparotomy or craniotomy are performed in 6% of all trauma patients treated in the ICU. The intensive care treatment should focus on rewarming, correction of lactate acidosis and coagulopathy [7].

*Restoration of normal physiology (<48 h after trauma)* Normal body temperature and coagulation is established with concomitant treatment of intracranial hypertension, temporary respiratory failure, stabilization of hemodynamic and prevention of infection and MOF.

*Organ dysfunction and replacement phase* After manifestation of impending organ failure or during organ failure, due to infectious complications, SIRS, sepsis and MOF, this phase is the domain of organ replacement therapy.

*Recovery phase* Normalization of organ function or persisting organ failure (i.e. kidney) with weaning, anabolic metabolism, spontaneous negative fluid balances are the principal objectives.

*Regeneration phase* The aim is to regain complete organ function, muscle strength and mobility. These phases address the major problems and therapeutic goals. However, they merge dynamically to each other, have variable durations and are partially overlapping.

## Classification of injury severity

Several different classification systems (injury severity score (ISS), Hannover polytrauma score, organ injury score, revised trauma score, Glasgow coma score (GCS)) already exist in order to estimate the injury severity and outcome (trauma injury severity score, revised injury severity classification) of polytraumatized patients. These scores should reflect and quantify the injury severity, assist in decision making and prognostic anticipation of the clinical course. Additional valuable critical care scoring systems (simplified acute physiology score (SAPS II), sequential organ failure assessment score, acute physiology and chronic health evaluation) are further focussing on the physiology of the diseases and risk of developing organ failure.

The survey of all injuries, the consideration of the physiologic status, the pre-existing diseases and the completed scoring rates are necessary to capture the total injury severity and perform a priority-adapted and goal-directed intensive care treatment.

## Strategies in trauma care

For the treatment of polytraumatized patients, there are basically two different therapeutic principles available: The damage control and the early total orthopaedic care concept. The damage control principle is designated for extremely injured patients nearly exhausting their normal physiology [7]. It consists of three different phases of treatment: the initial life-saving surgery (control of haemorrhage and contamination), restoration of normal physiology on ICU and subsequent reoperation with definitive repair of all injuries [7]. Patients who are assigned to the damage control approach suffer from [9–12]:

- High-energy trauma
- Multiple torso injuries
- Hemodynamic instability
- Coagulopathy
- Major vascular injuries
- Severe metabolic acidosis (pH<7.1)
- Hypothermia (<35°C)
- Severe brain and/or lung injury
- ISS >40
- Lactic acidosis >5 mmol/L
- Base deficit <−8 mmol/L (>55 years) and <−18 mmol/L (<55 years)
- Resuscitation and operative time >90 min
- Need of massive transfusion (>10 units RBC)

On the contrary, the early total orthopaedic care concept aims for early definitive orthopaedic surgery and stabilization of long-bone fracture even in the primary phase of polytrauma treatment. Early total care is indicated in patients who have sustained multiple long-bone fractures and moderate thorax/abdomen injuries and whose overall hemodynamic and general status allows for immediate

definitive treatment and can cope with additional surgical trauma, i.e. surgical approaches, prolonged surgeries, etc. Furthermore, time-saving simultaneous operations are performed. Therefore, better functional outcome and decreased length of hospitalization in these patients is expected.

## Timing of operations and decision making

The primary decision making for early total care or damage control is done usually in the resuscitation room before admitting the patient to the operation room or ICU. Therefore, the surgical critical care specialist has to consider the above-mentioned points, know about the chosen strategy and continually ask the following question: is the bleeding and the contamination sufficiently stopped [13]?

Pursuing that approach also implies that continuous/serial re-evaluation of the patient's clinical course is mandatory and crucial to the final treatment success. Emergency reoperations should be performed in patients with ongoing bleeding, evolving infection, deteriorating neurological status and/or development of compartment syndrome [14]. Indications for emergency operations include the following [7]:

- Necessity of blood substitution with more than 2 units of red blood cells (RBC)/h for 3 h
- Blood loss via chest drain after initial evacuation exceeding more than 1,000–1,500 ml/h
- Renal or respiratory failure due to abdominal compartment syndrome
- And worsening mental or neurological (pupil) status.

An important aspect of the ICU management is the integration and timing of secondary operations/programmed revisions in the general treatment concept of the trauma patient [2]. If possible, planned/scheduled definitive surgical treatment should only be done in fully re-warmed, hemodynamically stable and physiologically recaptured patient [7]. Indicators are recovered organ function (creatinine, bilirubine), oxygenation (Horovitz index), lactate clearance, declining inflammation parameters and spontaneous negative fluid balance (flow phase).

## Trauma re-checks

Undetected injuries such as ongoing or occult haemorrhage, compartment syndrome and rhabdomyolysis should be considered in patients with failed resuscitation and unsuccessful lactate clearance. With an incidence of 2–9% overlooked injuries are common, especially in patients with polytrauma (high-energy/high-velocity accident victims), in initial unconsciousness, substance abuse and/or hemodynamic instability. Recognition and delayed diagnosis of these injuries can be made at any stage of the trauma management [2, 15–17]. Therefore, repeated physical examinations and radiographic imaging/CT-and/or MRI-scans diagnosis during ICU stay are necessary for complete diagnosis and to prevent adverse and poor outcome. The tetanus immune status has to be checked and, if necessary, an immunization must be administered.

## Hemodynamic and fluid resuscitation

One of the most common tasks in trauma patients is to achieve hemodynamic stabilization. The classic parameters for monitoring of macrohemodynamic circulatory function such as ECG, blood pressure, oxygen saturation, as well as urine output, have been shown to be of limited reliability and validity in predicting nutritive perfusion and tissue oxygenation at the microcirculatory level. Furthermore, these parameters have failed to predict mortality and did not turn out to be valid indicators for life-saving interventions before cardiovascular collapse in trauma patients [18]. Notably, blood pressure poorly reflects microcirculatory perfusion and central venous pressure does not reliably predict fluid responsiveness. More reliable methods to determine the fluid necessity and responsiveness is the base deficit, lactate clearance and PICCO catheter system, measuring the pulse pressure variation, stroke volume variation, systolic pressure variation and the global end-diastolic volume [19]. Trauma patients with short episodes of massive hypotension are at increased risk to either die during ICU stay or need a markedly prolonged recovery time [20]. Therefore, the patients at risk for hypotension and secondary shock have to be identified and grouped according to accepted criteria such as: borderline, BP 80–100 mmHg, blood units received 2–8, lactate <2,5 mmol/L; normal, BP >100 mmHg, blood units received <2, lactate and base deficit normal[21]. Furthermore, hypovolemic shock can be classified according to ATLS manual. At-risk patients should have several large lumen intravenous lines, a central line to control fluid resuscitation and central venous pressure as well as an arterial line for continuous measurement of the blood pressure [7]. The fluid resuscitation management is guided by the urinary output as a parameter of end-organ perfusion, restoration of vital signs, cardiac output measuring, mixed venous oxygenation saturation ($SvO_2$) and lactate clearance [7]. Upon ICU arrival, the typical tests are performed: blood gas analysis, complete blood counts, coagulation profile or thrombelastography and blood chemistry [22]. Lactate clearance and arterial base deficit have been shown to be superior indicators of sufficient tissue perfusion when compared to blood pressure and urine output [23]. Trauma patients with

a normalized lactate 24 h after trauma have been identified to reach a 100% survival rate [7, 22]. Therefore, the lactate clearance is widely accepted as a prognostic marker of successful resuscitation and indicator for decreased mortality in hemorrhagic shock [7, 22]. Lactate should be measured every 4 h until two consecutive measurements are less or equal to 2 mmol/L [13]. Moreover, the arterial base deficit should be normalized or kept over −2 mmol/L [10]. In trauma victims, whose base deficit was not cleared within 48 h after trauma, only 13% survived [24]. It is generally accepted that the traumatized patients need approximately >600 ml/min/$m^2$ oxygen delivery index ($DO_2I$) and >150 ml/min/$m^2$ oxygen consumption index ($VO_2I$) within 12 h after admission to ICU. These parameters correspond to 68–72% $SvO_2$ [7]. Crystalloid or colloid intravenous fluids can be used for the initial fluid resuscitation [7]. However, there is an ongoing controversy about the type of fluid resuscitation which should be used [1, 19]. No clear advantage for hypertonic fluid resuscitation compared to isotonic crystalloid in trauma patients was found in a recent Cochrane review [19]. In hypertonic fluid resuscitation, less amount of fluid is necessary, and therefore, the probability of developing edema is reduced [19]. Further beneficial effects on immune modulation and reducing intracranial pressure are described [19]. Also, colloids are not superior to crystalloids in treating hypovolemia in the trauma patient and showed no survival benefit [19]. However, extensive fluid resuscitations can lead to dilution of clotting factors, acute respiratory distress syndrome, abdominal compartment syndrome and brain edema [3, 19]. In addition, large amounts of crystalloid contribute to the development of abdominal compartment syndrome [25]. Aggressive fluid administration restores normal blood pressure and maintains tissue oxygenation despite increasing the hydrostatic pressure at the wound site with eventually increased bleeding, clot dislocation and dilution of clotting factors with coagulopathy [26]. The permissive hypotension concept respects these potential problems and was proven to have a better outcome especially in patients with penetrating trauma. As a temporary concept, blood loss can be reduced until definitive bleeding control is achieved. Permissive hypotension is not suitable in TBI because of adequate perfusion pressure maintenance [27]. Inotropic and vasoactive agents in trauma should be avoided in the early resuscitation phase because of a two-fold higher mortality rate that seems to result from aggravation of the microcirculatory breakdown due to arterial vasoconstriction with lowered tissue oxygen delivery [19]. The primary successful endpoint in resuscitation is a stoppage in the source of bleeding with sufficiently resituated macrohemodynamic situation/-hourly urinary output and restored tissue oxygenation measured by recompensate base deficit and lactate clearance.

## Coagulopathy

Uncontrolled haemorrhage and exsanguination are the leading cause of early death in trauma patients [28]. The accompanying coagulopathy develops with an incidence of 10–34% and is a multifactorial condition initiated by excessive bleeding, consumption of clotting factors and platelets, dilution, fibrinolysis, hypothermia, acidosis and transfusion-associated citrate accumulation [1, 3, 29]. This leads to the dreaded manifestation of the so-called lethal triad consisting of hypothermia, coagulopathy and metabolic acidosis, which is associated with up to four-fold elevated mortality [9, 30]. The new understanding of the traumatic coagulopathy as a dynamic and reversible disease, taking course in different stages (hypercoagulopathy, coagulopathy, hyperfibrinolysis) has tremendously influenced the management of traumatic haemorrhage [31]. Risk factors for traumatic coagulopathy are an ISS >25, pH<7.1, systolic BP<70 mmHg and temperature <34°C [32]. Beside clinical judgment of ongoing bleeding and coagulopathy, laboratory tests such as platelet count, platelet function analysis, activated partial thromboplastin time (PTT), prothrombin time, international normalized ratio (INR) and thrombelastography are recommended in order to comprehensively assess impairment of coagulatory and thrombocyte function [33]. Formation of a stable and sufficient blood clot requires an internal milieu (normal body temperature, hemoglobin 7–9 g/dL, hematocrit >21–24%, platelets >50–100.000/nL, fibrinogen >1.5–2 g/L, PTT <1.5-fold normal ranges, INR <1.5, normal ionised calcium) as precondition for sufficient hemostasis. Patients with platelets less than 150.000 nL have significantly higher risk of mortality after severe trauma [34]. Posttraumatic hypothermia is also associated with an increased mortality rate, cardiac dysrhythmias, reduced cardiac output, reduced oxygen disposal (left shift of the hemoglobin saturation curve) and impaired blood clotting cascade [9, 35]. Therefore, early rewarming and maintenance of the normal body temperatures is essential, thereby attenuating coagulopathy and improving outcome. This might be conflicting in situation with concomitant severe TBI or after cardiac arrest. With that in mind, all transfusions, as well as humidified breathing air, should be warmed to 37°C. Furthermore, the patient should be kept warm by means of special rewarming inflatable air mats to minimize thermal loss [6]. To prevent traumatic coagulopathy, immediate bleeding control must be achieved. In the meantime, a systolic blood pressure 80–100 mmHg is recommended in the absence of TBI or spinal cord injury [36]. Early applications of thawed fresh-frozen plasma (FFP; 10–15 ml/kg) and fibrinogen concentrate or cryoprecipitate (50 mg/kgBW) are useful and valuable concepts in severely bleeding trauma patients [36, 37]. The RBC/FFP ratio represents a controversial

topic. Recent studies recommend RBC/FFP ratio <1:1 [33]. Further small studies revealed the important role of platelets in the initial resuscitation of severe bleeding trauma patients and showed a better outcome for patients with a liberal transfusion protocol (RBC/FFP/platelets 1:1:1) [33]. In patients with substantial hyperfibrinolysis detected by thrombelastography tranexsamic acid (bolus 10–15 mg/kg, followed by 1–5 mg/kg/h) or ε-aminocapronic acid (bolus 100–150 mg/kg, followed by 15 mg/kg/h) should be administered [36]. Vitamin K-dependent oral anticoagulation in polytraumatized patients is best treated with prothrombin complex concentrate. In patients with acetylsalicylic acid intake over a longer period, desmopressin application can be useful to reverse the anticoagulatory effects. The substitution of antithrombin III along with FFP in trauma patients is not recommended [32, 36].

## Inflammation, SIRS, sepsis

SIRS, sepsis and infection are common disorders typically developing in critical care trauma patients. From several studies, it is now becoming increasingly clear that a posttraumatic dysbalance of pro- (IL-1, IL-6, IL-8, TNF-α) and anti-inflammatory cytokines (IL-4, IL-10, IL-13, HLA-DR) is triggering the onset of these complications. Predominating levels of pro-inflammatory cytokines lead to SIRS, organ- and MOF, whereas prevailing anti-inflammatory parameters predispose to infection and sepsis. Common infectious complications are nosocomial or ventilator-associated pneumonia, lung abscess or empyem, urinary tract infections, surgical infections, cholecystitis, sinusitis, otitis media and catheter-associated infections. Consequently, early empirical antibiotic intervention is expected to result in significant benefits in patients with sepsis or septic shock [38, 39]. Conversely, immunosuppression using low-dose hydrocortisone and fludrocortisone treatment in septic shock patients with adrenal insufficiency improved survival [40]. The different critical care organisations developed a precise guideline, summarizing the diagnosis and treatment of sepsis, the surviving sepsis campaign, which is also valid in trauma patients [41].

## Organ/multiple organ failure and extracorporeal organ support/replacement therapy

The lungs, liver and kidneys are the primary dysfunctional organs following severe shock or trauma. Hemorrhagic shock is the inciting factor for early MOF (<72 h after trauma). Infection, SIRS and sepsis are the principal reasons for the late multiple organ dysfunction syndromes (MODS), which develops in 21.1–36.6% of all polytraumatized patients. The blood transfusion requirement is another independent predictor for MOF, especially when the administered blood is older than 2 weeks [42, 43].

### Ventilation, acute lung injury and adult respiratory distress syndrome (ARDS)

The traumatized and ventilated patient has an increased susceptibility and risk to develop ventilator-associated pneumonia, ALI and ARDS, which in turn further aggravates tissue hypoxemia [7]. The hypoxemia is caused by impaired oxygenation with accumulation of protein-rich edema fluid in the distal air space of the lung due to alveolar endothelial disintegration with increased microvascular permeability and macromolecular endothelial leakage [44]. In addition, evolving pulmonary edema is also perpetuated due to excessive fluid resuscitation, transfusion-related acute lung injury and hypercapnia. The goal of ventilator strategy is to maintain oxygenation and ventilation as well as prevention of secondary volu- or baro-trauma. The lung protective ventilation has reduced the ALI mortality from 40% to 20% [45]. Therefore, the patient should be ventilated with an initial tidal volume of 6–8 ml/kg, maintained peak inspiratory pressure less than 40 mbar and the fraction of inspired oxygen ($FiO_2$) should be adapted to achieve an oxygenation saturation >93% [46]. This often requires additional positive endexspiratory pressure (PEEP), and eventually, the adaptation of the inspiration to exspiration time (I/E). In patients with ALI and ARDS, the I/E ratio is reduced in favour of a better oxygenation, however, with simultaneously developing hypercapnia. Therefore, permissive hypercapnia with pH >7.2 is tolerated in trauma patients with severe lung injury. However, this concept is not applicable in patients with TBI and elevated intracranial pressure because of cerebral vasodilatation and therefore, aggravation of the cerebral hypertension.

Other therapeutic tools to enhance pulmonary function and to reduce alveolar fluid accumulation include the introduction of rotokinetic beds or conventional bedding devices. They improve the ventilation/perfusion mismatch by opening contused lung areas and atelectasis. The initial CT scan can assist the choice of bedding therapy especially in patients with a.p. gradients (contusion, no contusion). The risk and frequency of nosocomial pneumonia is reduced by using a semi-recumbent body position, especially in patients with enteral nutrition [47]. One innovative concept to normalize the ventilation/perfusion mismatch is the application of inhaled nitric oxide [18]. Several experimental studies have shown that acute posttraumatic lung injury and endothelial damage is associated with depressed activity of inducible nitric oxide synthetase that inevitably leads to increased leukocyte endothelial cell

interaction (adherence), capillary narrowing and impaired overall microvascular blood flow. Endpoints of successful ventilator support are good oxygenation, a conscious patient, uncompromised airways, sufficient respiratory parameters (tidal volume, vital capacity and inspiration force), effective couch and adverse-effects reflex, finally allowing uneventful extubation of the patient. However, extubation failure has been shown to be associated with increased morbidity and mortality [48]. Corticosteroid significantly reducing laryngeal edema is one approach for decreasing the reintubation rate (≤50%). Before extubation, an upper airway obstruction should be excluded by using the cuff-leak test [49].

In case of long-term ventilation, severe TBI and repeated extubation failure, a percutaneous dilatational tracheostomy should be considered to decrease anatomical dead space and allow improved secretion and mucus mobilization. Despite the fact that tracheostomy is beneficial compared to long-term translaryngeal intubation (less nosocomial pneumonia, shorter mechanical ventilation, less dead space, bronchial toilet, reduced work of breathing and ICU stay), the optimal time to perform tracheostomy remains controversial, although there is a trend towards early application [50]. The percutaneous dilatational tracheostomy is an easy and safe procedure that can be performed in the ICU. Open tracheostomy should also be considered in specific patients with anatomic conditions (extreme cervical spine kyphosis, i.e. Bechterew patients, massive struma, previously performed cervical surgeries) and ARDS. To prevent wound infections, a necessary tracheostomy should be performed after surgical stabilization of cervical spine injuries. This means also that in patients with cervical spine injuries that may require tracheostomy due to simultaneous chest/lung trauma, an early anterior spinal stabilization should be performed in order to reach consolidated surgical wound conditions for the subsequent tracheostomy.

In case of mechanical ventilation failure, the extracorporeal lung support is the only available option to secure oxygenation and decarboxylation [41]. Several studies have shown a survival advantage for trauma patients treated with extracorporeal membrane oxygenation (ECMO) and pumpless extracorporeal lung assist [41]. Extracorporeal gas exchange by a capillary membrane oxygenator for oxygenation and decarboxylation, such as ECMO, ECLA and pumpless interventional lung assist (iLA) can help in such conditions to reduce the inspiratory plateau pressure, $PaCO_2$, improve the oxygenation and survival of the patients (Fig. 1) [48].

These devices can help to bridge patient until their pulmonary function or cerebral edema has improved or in rare cases until a lung transplant is available. Known side effects of these devices are mechanical hemolysis, transfusion requirement and right heart failure. Additionally, the

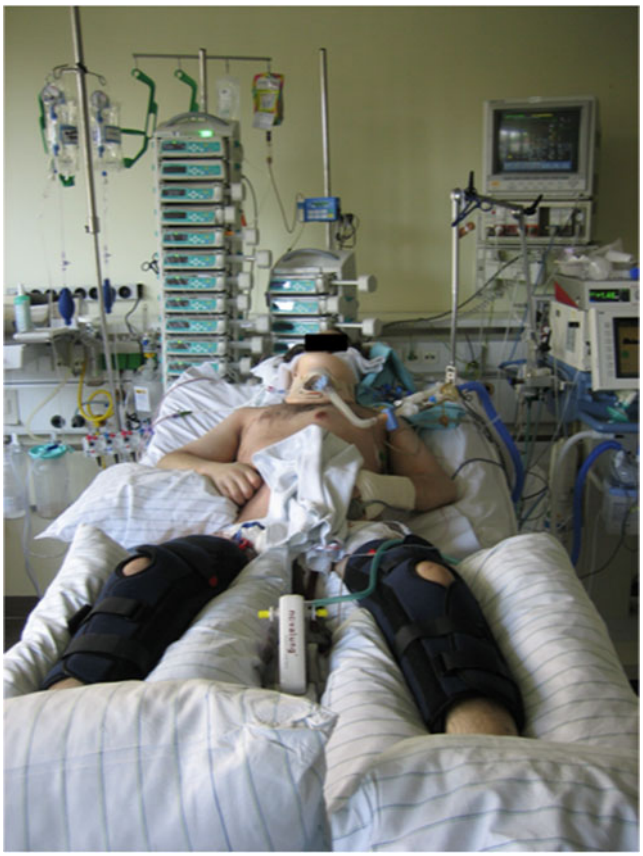

**Fig. 1** Successful control of $PaCO_2$ by iLA in a polytraumatized patient with combined severe traumatic brain injury, ARDS and renal failure

required heparinization of trauma patients is a challenging topic. Due to the development of new oxygenation technologies with heparin bonding capillaries, a low-dose heparinization (150–220/IU/kg bodyweight/day) is sufficient, once more decreasing the risk of bleeding [51]. Nevertheless, contraindications for ECMO include severe head injury, terminal chronic or malignant disease, severe haemorrhage, disseminated intravascular coagulation. In heart failure, septic shock and severe peripheral arterial occlusive disease the pumpless iLA is contraindicated [52]. In simultaneous renal and liver failure, continuous hemofiltration and extracorporeal liver support device can be connected to ECMO [53]. In patients with additional heparin-induced thrombocytopenia, an anticoagulation using hirudin, argatoban and prostacyclin are useful pharmacotherapeutic alternatives [48]. Notably, the heparin-coated capillaries of ilA and ECMO in HIT are contraindicated.

## Acute renal failure and rhabdomyolysis

The risk of acute renal failure in trauma patients is low. However, direct tissue damage by traumatic laceration, renal vessel injury with subsequent necrosis, rhabdomyol-

ysis resulting from compartment syndrome and crush injury, obstructive uropathy, hypotension and the use of iodinated contrast mediums during radiographic imaging (CT scan) have the potential to induce renal failure. Acute renal failure in trauma patients has a mortality up to 60% and combined with lung failure up to 80% [54]. The acute renal failure and its severity classified by risk, injury, failure, loss, end-stage criteria is directly associated with higher mortality rates of critical care trauma patients [55]. Incidence of renal failure can be decreased by aggressive i.v. fluid resuscitation [55]. Continuous veno-venous haemodialysis seems to be superior to haemodialysis because no systemic anticoagulation is needed and consecutive hypotension is less likely [56]. Undoubtedly, renal replacement therapy has improved the survival of trauma patients with acute renal failure, but the timing of its initiation is a controversial debate. Biochemical (urea, creatinine) and clinical parameters (fluid volume status, onset of oliguria) are described as decisive factors determining the start of haemodialysis. Therefore, uncontrolled electrolyte abnormalities, metabolic acidosis, fluid overload and uremic encephalopathy are indications for renal replacement therapy. In trauma patients, early renal replacement therapy has attracted major interest, as small studies of combat victims have proven there is an advantage for early or even prophylactic haemodialysis [41]. New filters with large pore diameters (high cut-off membranes) and antibiotic-coated membranes (polymyxin B) are suitable in the prevention of septicaemia and treatment of rhabdomyolysis. The aetiology of rhabdomyolysis in trauma patients is most frequently caused by trauma-induced ischemia, infection or drug-induced, toxic cell death. Common complications are electrolyte imbalances (calcium, potassium), hypotension, coagulopathy, compartment syndrome, tubular obstruction from myoglobin and tubular damage and renal vasoconstriction. Normally, CK, myoglobin, white blood count (WBC), aspartate aminitransferase, alanine aminitransferase (ALT), lactate dehydrogenase are elevated and myoglobinuria exists [48, 57]. The most important treatment in rhabdomyolysis is the effective treatment of its cause in terms of emergency fasciotomies, early revascularization and preservation of renal function. Consequently, aggressive fluid application, mannitol, loop diuretics and bicarbonate for urine alkalization (pH>6.5) are recommended [57].

### Acute liver failure

Acute liver failure in response to severe trauma is rare, but associated with a mortality rate of 15–50% [58]. Known factors that induce posttraumatic acute liver failure are shock, direct liver trauma (contusion), partial hepatectomy, abdominal compartment syndrome, hepatic ischemia resulting from hepatic artery or portal vein damage or parenchymal compression by inadequate packing, endotoxemia and hepatotoxic drugs [59]. In addition to established laboratory parameters, not reliably reflecting liver function (INR, PTT, fibrinogen, ammonia, bilirubine, AST, ALT, lactate, blood glucose), the functional liver capacity can be detected by LiMAx and LiMON test [60, 61]. A serum bilirubine level exceeding >300 μmol/L has been demonstrated to predict death with a specificity and sensitivity of 87.8% and 90.3%, respectively [41]. Attendant complications in liver failure are hypoglycaemia, renal failure, hepatic encephalopathy and coagulopathy. To clear albumin-bound toxins from the systemic circulation, haemodialysis is ineffective and, therefore, not suitable for that purpose. Nowadays, different systems are available (single pass albumin dialysis, molecular adsorbents recirculation system, Prometheus), which can help to bridge the time gap of trauma patients until liver regeneration starts or transplantation is feasible [53]. Although these liver replacement therapies are considered to systemically clear ammonia, bile acids, bilirubine, copper, iron and phenols from the blood, the evidence for their effectiveness in trauma patients is low [41]. Often, the only causal and lasting treatment option that leads to survival in trauma patients with liver failure is transplantation.

## Metabolism

There are different reasons for acid–base deficits in trauma patients. Besides the traditional classification of respiratory versus metabolic acidosis or alkalosis, the surgical critical care specialist must also consider the possibility of an iatrogenically (hyperchloremic) induced acidosis, that is due to pre-existing diseases (chronic renal failure) or acute disorders like hemorrhagic shock and sepsis. The most common acid–base abnormality in trauma patients is the lactic acidosis following hypovolemia. The trauma-induced acidosis is normally corrected without further interventions, if the patient is sufficiently rewarmed and resuscitated. Rarely, the acidosis must be corrected (pH<7.2) using bicarbonate or TRIS-buffer (respiratory failure, no carbon dioxide increase). The hypermetabolic response to trauma with hyperglycaemia is well known, indicative of endocrine dysfunction and associated with increased morbidity and mortality [62]. Among other factors, the trauma-induced activation of the sympathoadrenal system, increased levels of catecholamines and glucocorticoids, glucagon, increasing insulin resistance, while decreased insulin production contribute to the posttraumatic hyperglycaemia [63]. Adverse effects of hyperglycaemia are abnormal immune function, growing infection rates and poor outcome. Interestingly, in a study by Mohr et al., the blood glucose variability turned out to be an independent factor of patient

outcome in male [62]. In this context, additional studies have shown beneficial effects of a strict blood glucose (<150 mg/dl) control protocol [64]. Likewise, the intensive insulin therapy maintaining the blood glucose level at 110 mg/dl was found to markedly reduce morbidity and mortality in critically ill surgical ICU patients [65]. However, reservations towards an intensive insulin therapy should be expressed in patients with TBI because hypo-/hyperglycaemia can cause secondary brain injury. In conclusion, moderate blood glucose target levels between 120 and 150 mg/dl are recommended [64, 66]. In poorly controlled diabetic patients, the target range should be increased to 100–200 mg/dl [66].

Obesity (body mass index >30 kg/m$^2$) occurs in approximately 5% of blunt trauma patients and is associated with prolonged ICU stays and ventilation time [67]. Moreover, obesity is an independent predictor of poor outcome in critical care patients. These patients suffer more frequent complications like MOF, ARDS, myocardial infarction and renal failure. In obese patients, the blood volume, cardiac output and total respiratory resistance are increased, while the chest wall compliance is decreased. The tidal volume should be calculated on the ideal body weight rather than total body weight and a prophylactic PEEP (5–10 cm$H_2O$) should be administered to improve oxygenation. To improve the weaning process, a reverse Trendelenburg positioning is useful. Obesity is a further independent risk factor for deep vein thrombosis, which lends support to the concept of more generous administration of low-molecular-weight heparin (LMWH) therapy. Caution must be taken in application of lipophilic drugs (propofol, benzodiazepines, fentanyl, neuromuscular blockers), because their distribution volume is unpredictable and accumulation of the drug and rebound effects can occur [67].

## Nutrition

Trauma, sepsis and burns cause a clinically relevant increase in the resting energy expenditure, between 40% and 80%. In contrast, clinical deterioration due to shock, severe sepsis and septic shock can lead to a drop in the resting energy expenditure, to a level of only 20% above a healthy individual. Critical care patients in the acute stage should also be supplied equally to the total energy expenditure [68]. In the acute phase, 20–25 kcal/kg bodyweight (BW)/day should be administered, whereas during recovery phase, the values should be increased up to 25–30 kcal/kgBW/day [69].The early enteral nutrition preserves the intestinal function and limits bacterial translocation and infectious complications [69]. An enteral nutrition should start within the first 24 h using a standard high-protein formula in hemodynamically stable patients with functioning gastrointestinal tract, who are however, not expected to be able to take a full oral diet within 3 days [69]. For this, jejunal or gastric feeding tubes can be used with equal efficiency. Parenteral nutrition is a reserve tool and can be administered to those patients not reaching their target nutrition level on only an enteral diet [69]. After trauma and in mild sepsis, an immune-modulating diet enriched with arginine, nucleotides and ω-3 fatty acids is superior to normal enteral nutrition [69]. Immune-modulating diets can improve immune function and host defence mechanisms, modulate inflammation and reduce the risk of infection, wound complications and length of hospital stay.

Antioxidant therapy and free radical scavenging used in the enteral diet was proven to reduce MOF, ventilation days and survival after trauma [70]. Patients with ARDS should receive ω-3 fatty acids and antioxidants-enriched enteral nutrition [69]. An immune-modulating diet with fish oil improves the outcome in critically ill patients with SIRS, sepsis and ARDS [69]. Obese patients may benefit from a hypocaloric, high-protein enteral nutrition [71]. Notably, continuous renal replacement therapy leads to an excessive loss of proteins and amino acids. Therefore, in those patients, higher caloric and protein delivery (25–35 kcal/kgBW/day) is required [72]. Among other trace elements, the micronutrient selenium was identified to exert antioxidantional support (200–1,000 μg/day) and beneficial effects in trauma, sepsis and burn patients [58].

## Deep venous thrombosis and pulmonary embolus

Critically ill and, particularly, trauma patients are at high risk for deep venous thrombosis (DVT) and consecutive pulmonary embolism. Additional risk factors are mechanical ventilation, sedation, paralysis, central venous catheterization, spinal cord injury, pelvic/acetabular fracture, combined long-bone fracture and TBI [73]. Thrombosis prophylaxis should be started as early as possible based on the risk of bleeding and accompanying coagulopathy (hemorrhagic TBI, epidural haematoma, diffuse tissue bleeding). Unfractionated heparin (UFH) and LMWH are both feasible methods for sufficient thromboprophylaxis. UFH can be neutralized by protamine and therefore, has a practical advantage in patients at risk to develop bleeding. In patients with a high risk of bleeding, mechanical thromboprophylaxis with compression stockings, foot pumps and intermittent pneumatic compression is recommended. A Doppler ultrasound or duplex should be performed on patients with clinical symptoms of DVT or based on screening protocols (<48 h after admission and every 5–7 days on ICU). Furthermore, a temporary inferior

vena caval filter placement should be considered for the highest-risk patients with contraindications for full anti-coagulation and severe lung-injured patients with right heart failure (ARDS), dying from a small pulmonary embolism [74].

## Thrombocytopenia

Thrombocytopenia is one of the most common laboratory abnormalities in critical care patients and is another predictor for increased mortality and morbidity. Potential reasons for thrombocytopenia in trauma patients include bleeding, sepsis, disseminated intravascular coagulation (DIC) and heparin-induced thrombocytopenia (HIT) [75]. Patients after orthopaedic surgery and treated with unfractionated heparin are at higher risk to develop HIT compared to patients treated with LMWH. The clinical diagnosis of HIT (drop of platelets >50%, new thrombosis, pulmonary embolism) in trauma patient is challenging because of the trauma-related initial thrombocytopenia and compensatory thrombocytaemia, in which a secondary drop of the platelet count can be masked [76]. Therefore, in patients with the suspicion of evolving HIT additional serologic tests (antibody test) should be performed to allow early diagnosis, prevent thrombembolic complications and change the anticoagulative protocol to hirudin, argatoban or prostacyclin [75].

## Specific injury-related intensive care

### Traumatic brain injury (TBI)

Concomitant severe head trauma/traumatic brain injury is observed in about 60% of all polytraumatized patients. The severity of traumatic brain injury (TBI) is classified by the GCS and was shown to have a negative impact on the mortality rate [77]. The TBI is divided into primary (contusion, hematoma, diffuse axonal injury) and secondary brain damage due to a variety of post-primary processes, e.g. ischemia, inflammation, edema formation, perifocal apoptotic cell death in surrounding areas (penumbra). Especially polytraumatized patients are in danger of developing secondary brain damage due to hemorrhagic shock and hypoxemia after severe thoracic trauma [78]. Hypotension (systolic BP<90 mmHg) occurs in 35% of patients with TBI. Combined with hypoxemia ($PaO_2$< 60 mmHg) hypotension is an independent predictor of poor outcome and disability [79]. Therefore, aggressive fluid resuscitation and providing adequate blood pressure and oxygen delivery is absolutely essential in such patients. Norepinephrine as vasopressor should be used in case of hypotension with insufficient cerebral perfusion pressure (CPP), despite aggressive fluid resuscitation. Measuring intracranial pressure (ICP) and CPP is recommended for patient >40 years of age, and with abnormal CT scan, motor posturing and systolic BP<90 mmHg [80]. A cerebral blood flow threshold to diagnose cerebral ischemia is not generally recommended [80]. Further jugular venous oximetry and microdialysis, monitoring the cerebral and brain tissue oxygenation can help to optimize the brain oxygen delivery [80]. Clinical neurophysiological test (electroencephalogram (EEG), evoked potentials, electromyography) are useful to diagnose epilepsy, brain death, neurologic disorders and estimate the prognosis of anoxic ischemic encephalopathy after TBI [81]. The ICP is to be kept below 20 mmHg, while the CPP should be maintained above 70 mmHg. Apart from decompressive craniectomy, potential treatment options to reduce ICP are deep sedation, drainage of cerebrospinal fluid, administration of mannitol and hypertonic saline, moderate hyperventilation ($PaCO_2$ 30–35 mmHg), protective hypothermia and EEG guided burst suppression sedation (barbiturates) [82]. Additional beneficial effects of 7-day posttraumatic seizure prophylaxis using phenytoin were shown by Temkin et al. [83]. Possibly, promising interventions to improve neurological outcome in the future are early induction of prophylactic hypothermia and exogenous erythropoietin therapy [84]. Finally, one should be aware of accompanying clinically relevant hypothalamic–pituitary dysfunctions (adrenal deficiency, diabetes insipidus) after TBI, which in turn require secondary corrective interventions [85].

## Thoracic/cardiovascular injury

In severe thoracic trauma, differential diagnosis to hypotension and tachyarrhythmia such as blunt cardiac injury (incidence of 8–76%) must be excluded [86]. For more detailed diagnosis of blunt heart contusion electrocardiography, analysis of enzymes indicative of cardiac muscle injury analysis, as well as echocardiography, are available [86]. In case of an abnormal ECG (ST changes, arrhythmia, ischemia, heart block, unexplained ST), the patient must be continuously ECG-monitored for 24–48 h. In hemodynamic unstable patient, an echocardiography (transthoracic, tranesophageal) should be performed. Neither creatinine phosphokinase isoenzymes [87] nor troponin-T levels or temporal profiles are useful in predicting blunt cardiac injury-related complications [86]. However, Troponin-I (cut off >1.05 μg/L) is a valuable tool to distinguish between patients at risk of developing arrhythmia or ventricular dysfunction after trauma. Moreover, troponin-I should be measured at admission and 6 h after trauma [88]. Characteristic indications for echocardiography in trauma patients are severe thoracic

trauma, patients with pre-existing valvular or myocardial disease, suspected cardiac or aortic injury (in particular, dissecting aneurysms), suspicious widening of the mediastinal silhouette on the chest radiograph as well as critically ill, hemodynamically unstable patients [89]. Because of the limitations of the TTE (subcutaneous emphysema, positive pressure ventilation, surgical incisions and drains, artefacts by ribs) the tranesophageal echocardiography (TEE) is more suitable in trauma patients. Indications for TEE are hemodynamically unstable patients (evaluation of right/left heart pump function/ejection rate), major trauma with the inability to position the patient for transthoracic echocardiography and suspected aortic dissection [89]. In addition to structural lesions, the volume status in response to fluid resuscitation and cordial function can be investigated. In hemodynamically unstable patients, a cardiac shock can be reliably ruled out (sensitivity 100%, specificity 95%) [90]. In acute decompensation scenarios the echocardiography offers a suitable bedside alternative especially when a transport to the CT scan is not feasible. The TEE has a 97% sensitivity and specificity to detect traumatic aortic injuries [91]. In some studies, TEE was superior in detecting small aortic lesions (intima flap) not requiring surgical repair compared to aortography and CT scan [91]. Therefore, a negative echocardiography and hemodynamic stability rule out an aortic injury.

In flail chest patients due to segmental rib fractures, a non-invasive ventilator support is useful and can help to avoid invasive ventilation and atelectasis. The basis for this procedure is sufficient pain control, mobilization and intensive physical therapy. Fractures of the one to two ribs/upper thoracic aperture are often indirect signs for possible concomitant aortic or tracheobronchial injuries. Notably, both injuries have been typically missed and overlooked injuries in the past, but are nowadays more often identified as polytrauma CT scans have become more routine. Especially the tracheobronchial injury can be camouflaged by sputum and becomes symptomatic/obvious in the later phase of intensive care treatment. Clinical signs are thoracic soft-tissue emphysema, pneumothorax on the left side, bloody sputum, high fistula volume over the chest drain and air leakage alarm on the ventilator. Using a tracheos-/bronchoscopy, a tracheobronchial injury can be ruled out. Another potential cause of the abovementioned clinical symptoms is the oesophageal injury, which can be diagnosed by dynamic radiographic biplanar views with contrast medium administered via a nasogastric tube to detect contrast extravasations. If not already assessed at the CT scans, a concomitant diaphragmatic rupture with gastroduodenal hernia would then also be seen. Attention should be paid on fractures of the 9–12 ribs, which are at risk to cause either liver or spleen injuries. Independent of the size, a pericardial effusion in trauma and especially in penetrating trauma is a pathologic feature, the source of which must be ascertained. Traumatic hematothorax, mostly accompanying the rib fractures, can easily be treated if necessary by a chest tube (5th intercostal space, middle axillar line). Initial blood loss of more than >400 ml/h and 1,000–1,500 ml within every following hour via chest drainage are generally accepted indications for thoracotomy, surgical revision or rarely embolization. When in doubt of active bleeding, blood gas analysis of the drain fluid might help to differentiate between active bleeding (Hb approximately >5 mg/dl) and pleural effusion.

## Abdominal injury

Abdominal trauma with either injury to parenchymal organs, intraperitoneal bleeding or perforation/penetration of bowel, stomach, etc. with free intraabdominal air is one of the most frequent reasons for life-saving emergency laparotomy. The predominant injury locations are the spleen, liver, bowel and stomach, vascular injuries, diaphragm and kidney. In the last decades, the non-operative management, especially of spleen and liver injuries, was proved to be a successful concept in properly selected patients [92]. These patients have to be carefully monitored in the ICU for at least 48 h (AAST grade I–II) and 5–7 days (AAST grade III–V) with repeated ultrasound controls and laboratory testing. Severe liver injuries (AAST grade III–V) are predisposed to develop SIRS, tachycardia, ileus due to large peritoneal hematoma and biloma. The signs of failure of conservative management are:

- Increasing abdominal pain, tenderness, peritonitis, ileus
- Ongoing bleeding (decreased hematocrite, tachycardia, hypotension) with blood transfusion requirement (>2–4 units RBC) within the first 48 h and expansion of the liver or spleen pathology
- Sepsis due to infected hematoma or biloma

The diagnosis of posttraumatic biloma usually is delayed because the sterile bile induces only a modest peritonitis. In patients suspected with jaundice, acitis, colic and inanition ultrasound, CT, MRI or retrograde cholangiography should be performed to exclude a biliary duct injury, hemobilia, biloma or abscess. Successful treatment options are percutaneous drain, embolization, biliary catheterization and laparotomy. Increased levels of bilirubine and lipase in the drain fluid after splenectomy indicate pancreatic injury and should carefully be monitored and analysed. The traumatic or iatrogenic pancreatic injury is a common attended injury in those patients. After splenectomy, an immunization for haemophilus influenza B, meningococci and pneumococci to prevent overwhelming post-splenectomy infection is required. The vaccination should be performed approximately

2 weeks after splenectomy and before hospital discharge [93]. A reactive thrombocytosis with platelets >1,000,000/nL is at high risk of thrombembolic complications. Therefore, a prophylactic application of acetylsalicylic acid (100 mg/day) is recommended.

Traumatic colon injuries (≤33%) have the highest rate of septic complications [94]. Independent risk factors for abdominal complications are severe faecal contamination, ≥4 units of blood transfusions within the first 24 h and single-agent antibiotic prophylaxis. Thus, traumatic colon injuries should be treated by combinational antibiotic prophylaxis. After surgical treatment, the colon leaks and anastomosis insufficiencies remain the major problem. Depending on the type of repair, a leakage rate of 2.2–3.2% in primary anastomosed colon, 5.5–9% in resection and primary anastomosis, 4% in ileocolostomies and 8.9–13% in colocolostomies occur [94]. Risk factors for anastomotic leakage are ≥6 units of blood transfusion and hypotension in the emergency room and an abdominal trauma index score ≥25. Interestingly, the method of anastomosis (hand-sewn, stabled) did not influence the incidence of abdominal complications. The non-destructive colon injury should be repaired by debridement and primary anastomosis. In destructive colon injury, segmental resection and primary anastomosis should be performed [94].

The abdominal compartment syndrome (ACS)-induced MOF has a mortality rate of 83% [95]. ACS can be understood as a mechanical cause of MOF related to an increased intraabdominal pressure [96]. Clinical problems are the impaired ventilation (restrictive disorder, atelectasis, ventilation/perfusion mismatch), tissue, gut and organ perfusion, declined cardiac and decreased urinary output [96]. The secondary ACS caused by massive transfusion and endothelial leakage with visceral and retroperitoneal edema occurs without an abdominal injury [97]. The intraabdominal pressure >20 mmHg measured at endexspiration by a bladder catheter in supine position (zero reference point midaxillary line at iliac crest, 25 ml instillation volume) is defined as ACS [71]. Normal IAP is 5–7 mmHg. Intraabdominal hypertension starts at IAP≥12 mmHg and is associated with the beginning of organ failure [72]. In respect to the mean arterial pressure, the abdominal perfusion pressure is calculated and levels >60 mmHg are required for sufficient organ perfusion [96]. The ACS is associated with severe abdominal and pelvic trauma but also after damage control procedures with abdominal or retroperitoneal packing to control haemorrhage [95]. The standard treatment of ACS parallels the treatment of extremity and thoracic compartment syndrome performing a decompressive laparotomy with temporary abdominal wall closure [13]. However, in patients with abdominal or retroperitoneal packing due to haemorrhage and accompanied coagulopathy re-bleeding after decompression of the abdominal wall/retroperitoneal space may occur. In these situations, coagulation should be restored prior to performance of decompressive laparotomy. Depending on the patient's condition, the abdomen must be opened in the ICU or in the operating room and remain open. Visceral coverage can be achieved by several techniques such as vacuum-assisted closure, zippers, blankets and sandwich techniques. The fluid resuscitation might be underestimated in those patients due to leakage of several liter of fluid a day [7].

Trauma patients in the ICU are at risk to develop stress ulceration with upper gastrointestinal bleeding. Vulnerable patients have a previous history of ulceration, renal failure, sepsis, MODS, mechanical ventilation, coagulopathy and burns [98]. Prophylaxis should be done by administration of cytoprotective agents (sucralfate), H2- receptor blockers (ranitidine) or proton-pump inhibitors and early intragastric enteric nutrition [99].

## Spine injury

Spinal injury is common in polytrauma patients. Patients with hypotension, critical injury, ankylosing spondylitis, closed head injuries and low GCS are at higher risk for delayed diagnosis or as being an overlooked injury [100]. Undetected spinal injuries are 4.5 times more common in the cervical spine compared to the thoracolumbar spine and are associated with progressive neurologic deficit [100]. Ischemia is an important factor to develop or aggravate neurological deficits. Direct spinal vascular injury and spinal cord trauma with vasoplegia can lead to hypotension. Hypotension can reduce the spinal cord blood flow and perfusion aggravating neurological dysfunction (secondary spinal cord injuries) [101]. Therefore, aggressive fluid management with high–normal MAP (85–90 mmHg) for 7 days is safe, improves spinal cord perfusion and may enhance neurological outcome [101]. In persisting hypotension, despite aggressive fluid resuscitation, norepinephrine is the vasopressor of choice to elevate blood pressure [102]. Due to overwhelming fluid resuscitation in vasoplegia, serious complications, such as pulmonary edema and hyponatremia, can occur [103]. Additionally, due to vasoplegia with low blood pressure, spinal cord-injured patients can be misinterpreted as ongoing bleeders and mistakenly receive vicarious amounts of fluid [103]. On the other hand, a patient that has significant bleeding might be underestimated due to a lack of pain and a misinterpretation of the low blood pressure. Patients with spinal cord injuries are in danger of acquiring an autonomic dysfunction syndrome [103]. In the acute phase, effects on the cardiovascular system, respiratory system, temperature regulation, paralytic ileus and bladder dysfunction are seen.

Bradycardia often occurs within the first 2–3 weeks after spinal cord injury [103]. Controlling body temperature is disturbed by the inability of the patient to sweat and shiver below the spinal cord lesion level. Due to lack of sensation, spinal cord-injured patient are at risk of acquiring pressure sores. Urinary output can be influenced by an inappropriate anti-diuretic hormone secretion [103]. Therefore, low urinary output in the first days and polyuria (5–6 L/day) after 6–7 days can be normal and should not be misunderstood as an inability to concentrate urine and therefore, treated with vasopressin. In such cases, severe hyponatremia and renal failure can result.

Urinary tract infections are very common in spinal cord-injured patients due to incomplete bladder emptying. In 1940, 40% of the spinal cord-injured patients died following renal dysfunction. Thanks to bladder emptying protocols and antibiotic therapy, the mortality has been reduced to 3–5% [103]. Intermittent catheterization is the gold standard to avoid urinary tract infections. Further suprapubic cystostomy is the second method of choice [104]. Up to 77% of patients with indwelling catheters acquire a urinary tract infection within 7 days after injury. Mostly, Gram-negative infections of more than one bacterium are diagnosed.

Patients with cervical spine fracture should be intubated either by in-line head traction, cervical collar or fiberoptic assistance [105]. Cervical spine fractures and discoligamentous injuries can often be treated successfully by initial collar immobilization and anterior surgery (discectomy and anterior spondylodesis/plating). Fractures with uni-/bilateral facet luxation and incomplete paraplegia are emergency cases requiring surgical reduction, spinal cord decompression/laminectomy and stabilization, sometimes in a sequential anterior–posterior approach [106]. Management of thoracolumbar spine strictly depends on the general status of the patient, the degree of neurological deficits and biomechanical instability. It has been shown that patients with highly unstable burst/luxation (AO-type C-fractures) substantially benefit from early stabilization, mostly from posterior approach, in order to prevent additional neurological damage, reduce spinal instability-associated intractable pain and allow better nursing with prone/supine positioning of the patient. Using the "spinal damage control" approach, the patient is expected to have a decreased duration of required ventilation, less secondary neurological deficits, less complications, hospital costs, mortality and better prerequisites for subsequent rehabilitation [106, 107]. Thus, decisions should be made on an individual case-by-case basis knowing that extensive surgical intervention may harm the traumatized patient [108]. Postoperatively, cerebrospinal fluid leaks can be diagnosed by investigation of the drain fluids for beta-2 transferrin and beta-trace protein content compared to the blood levels. Neuroprotective pharmacotherapy is still under investigation, whereas methylprednisolone is the only effective agent tested in a multicenter clinical trial (NASCIS scheme II) [109]. NASCIS is widely considered to be the gold standard in spinal cord-injured patients. Recent studies criticize the small effects on clinical outcome and adverse side effects such as a two-fold increase in the risk of pneumonia [109, 110]. To summarize, NASIC should not be a standard treatment and indications should be made on an individual basis [110].

Therefore, we only use NASCIS in patients with complete paraplegia. Patients with spinal cord contusion and neurological deficit receive a dexamethasone 40 mg bolus and 8 mg application three times a day for 3 days. In our experience, there is not an increased incidence of complications such as pneumonia.

Cervical spine injuries are especially at risk of developing respiratory failure. One reason is the 40% loss of vital capacity due to intercostal muscle paralysis. Furthermore, the ability to sympathetically dilatate the bronchi are not possible, and therefore, inhalation with bronchial dilatators is needed. These autonomic nervous imbalances cause excessive mucus production, secret stagnation and hypersensibilty of the trachea. Therefore, cardiac arrest by vagal stimulation during tracheal suctioning is reported [103]. A respiratory protocol can reduce the ventilation time in spinal cord-injured patient. Patients with cervical spine fractures above C5 usually need mechanical ventilator support. Because most often the cervical spine is anteriorly stabilized, tracheostomy, if possible, should be performed after complete wound healing, to prevent wound infection and postoperative complications [111]. Paralytic ileus ceases within the first 1–2 weeks after trauma with risk of vomiting, aspiration and meteoristic abdomen. Therefore, protocols for bowl emptying are advised, and it is recommended that full enteral nutrition should be initiated as early as the first bowel movement [103].

## Musculoskeletal injuries and pelvic trauma

Pelvic injuries are often accompanied by severe bleeding (85% venous) and intraabdominal injuries resulting in a high mortality rate. Apart from life-saving surgery with mechanical stabilization of the pelvis (external fixator, C-clamp, belts, wraps), ligation of vascular bleeding and/or pelvic packing, the angiographic embolization, is a widely used method to stop the bleeding. The generally accepted algorithm for hemodynamically unstable pelvic injuries includes the emergency reduction initially by a pelvic binder, followed by external fixation and or pelvic clamp. According to our experience, the indication for retroperitoneal packing and/or subsequently followed angiographic embolization is given by

persisting macrohemodynamic instability, despite adequate reduction and stabilization of the pelvic ring. Because of the chimney effect, the retroperitoneal bleeding is not self-tamponading. An expanding retroperitoneal haematoma causes ACS, renal failure, coagulopathy and restrictive respiratory failure. Sometimes, confusion about the predominant bleeding source can occur, due to rupture of the peritoneum and secondary intraperitoneal haematoma. In extreme circumstances, an aortic balloon occlusion can be administered to bridge time until surgical or control of interventional bleeding is achieved [112].

Early fixation of long-bone fractures in polytraumatized patients lead to a reduction of fat embolism and prevalence of ARDS and MOF compared to patients with prolonged tractions [113]. But there is also evidence that early definitive fixation of long-bone fractures in patients with TBI and severe thoracic trauma can cause a secondary brain damage or ARDS [114]. Dependent on the injury severity and physiologic status of the patient, an individual decision making (damage control vs. early total care) is effective. Primary goals are stabilization of fractures and instabilities achieved by external fixation, plating or nailing and treatment of contamination with radical debridement and definitive or temporary wound closure (Fig. 2).

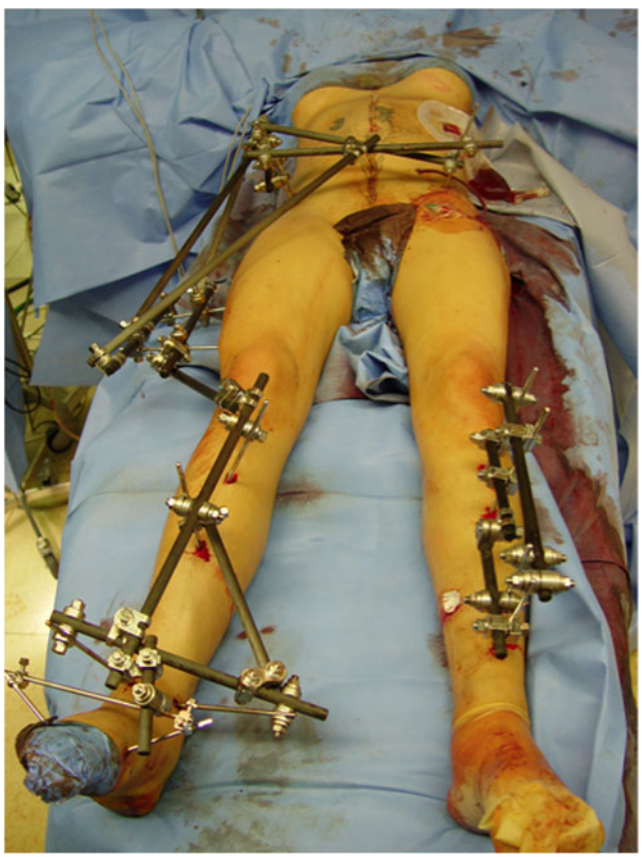

**Fig. 2** Damage control of multiple fractures (pelvis and both legs) in polytraumatized patient with external fixator

Compartment syndrome must be recognized and treated urgently with emergency decompressive fasciotomy. In polytraumatized or unconscious patients, clinical examination and compartment monitoring should be performed. Perfusion pressure levels <30 mmHg calculated by the diastolic blood pressure minus the compartment pressure, indicate compartment syndrome and require emergency fasciotomy. Patients with crush injury and >4-h warm ischemia time are at high risk of developing a compartment syndrome.

Soft tissue injury recovery and wound healing is further affected by coagulopathy, inflammation, hypermetabolism, nutrition and drug therapy. These mechanisms are impaired due to sepsis, tissue hypoxia, edema, excessive and prolonged local pressure in critical care patients [85].

## Burn injury

Burn injuries with the unique pathophysiology are best cared for in burn centres. Early operative therapy, early enteral nutrition and intensive blood-glucose control have lead to better survival of burn patients. Known complications of burn patients are hypothermia, infections, hypermetabolism and compartment syndrome. The cornerstone of effective intensive care treatment in burns is sufficient and balanced fluid and electrolyte resuscitation. The amount of crystalloid fluid is individual for each patient but can be estimated by the known Parkland formula [115]. Colloids and hypertonic saline have adverse effects on the burned patient, due to disturbances in the endothelial barrier function and are not recommended initially [116]. Risk factors for non-response to fluid management are inhalation injury, electrical burns, delayed resuscitation, alcohol or drug abuse [117]. The fluid resuscitation is guided as usual by the hourly urinary output (<0.5 ml/kg/h), lactate clearance and vital signs [116]. Interestingly, vitamin C substitution within 24 h after burns reduce the fluid requirement (40%), tissue water content (50%) and demand of ventilator support [118]. Inhalation trauma is the major comorbidity in burn patients, significantly increasing the mortality rate [116]. Patients at risk present clinical symptoms such as stridor, facial burns, history of unconsciousness, carbonaceous sputum and voice changes. The upper airway is endangered, and in most cases, intubation has to be performed. Inhalation injury and mechanical ventilation are associated with a 70% incidence of pneumonia and cannot be influence by prophylactic antibiotics [116]. Preventable secondary complications are hypothermia, compartment syndrome, ACS, stress ulcers, adrenal insufficiency, neutropenia and DVT. The compartment syndrome is released by immediate escharotomy and fasciotomy. Due to hypermetabolism, better wound healing

and prevention of mucosal atrophy with bacterial infections early enteral nutrition via a transpyloric feeding tube should be administered [116].

## Trauma in pregnancy

In 4.6–8.3% of all pregnant women, trauma occur thus indicating that this is a rare condition [119]. For detection of an unknown pregnancy or pregnancy in the first trimester, ultrasound and a pregnancy test should be performed in child-bearing aged women (16–50 years) during the resuscitation phase [120]. Predictors for fetal loss following trauma are a high ISS, elevated base deficit, high abdominal or thoracic-abbreviated injury score and abnormal uterine activity [120]. After estimation of the gestational age, in pregnancies with ≥24 weeks of gestation, an immediate uterine (activity) and fetal monitoring (heart rate) should be performed under the direction of the obstetrics service [120]. For known pregnancy, typical alterations of laboratory values (WBC↑, pH↑, bicarbonate↓, $PaCO_2$↓, fibrinogen↑) and typical pregnancy diseases (preeclampsia, gestational diabetes) have to be considered. Especially in Rh-negative women, the possibility of a feto-maternal transfusion syndrome must be ruled out and if necessary treated with Rh-immune globulin application. After 22–24 week of gestation, premature labor occurs in 25% of the traumatized women. These women are at high risk of placental abruption. Furthermore, pregnant women have an increased risk to develop deep vein thrombosis and pulmonary embolism aggravated by the trauma-related modulations of the coagulation system. Therefore, thrombosis prophylaxis is highly recommended. In case of cardiopulmonary arrest, emergency caesarean should be performed within 4 min to result in a viable normal infant.

## Psychiatric

Due to improved trauma management, more people survive and psychiatric disorders, such as acute stress disorder, posttraumatic stress disorder and depression are more prevalent. [121]. Therefore, 10–45% of traumatized patients suffer from acute or posttraumatic stress disorder and 8–35% suffer from depression. Potential risk factors are the ICU admission, female gender, burns, personality characteristics and coping styles, simultaneous intoxication prior to trauma, subjective feeling of life-threatening and invasive critical care procedures [121]. Early attention to prevent or treat posttraumatic psychiatric disorders is essential in order to achieve a better long-term outcome.

## Conclusion

The intensive care treatment of trauma patients has unique features and pathophysiologic regulatory mechanisms. Fundamental knowledge of the injury severity, understanding of the trauma mechanism and specific techniques of surgical intensive care are essentials for a successful critical care. Cornerstones of intensive care management are effective fluid resuscitation, transfusion protocols, extracorporeal organ replacement therapies, early and adequate performance of indicated surgical procedures and the injury-specific complications.

**Conflicts of interest** None.

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