Contemporary Intensive Care Treatment for Patients with Severe Multiple Trauma

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7.1 Introduction

The primary reason for mortality in patients younger than 40 years is still severe injuries induced by multiple trauma. Improvement of the rescue system with shorter intervals for rescue and transport and early respiratory and circulatory support significantly reduced early death resulting from brain damage, hypoxia, and the typical reasons of death such as exsanguinating hemorrhage in combination with acidosis, hypothermia, and coagulopathy, referred to as the "lethal triad." Consequently, complex and sequential multiple organ dysfunction (MOD) and multiple organ failure (MOF) have advanced to being the predominant reasons for increased mortality. Interestingly, the triggers for subsequent MOD/MOF are the same as those factors that accounted for the early casualties.

On the basis of the realization that multiple trauma is not characterized by the sum of the individual injuries, but predominantly reflects the consequences of severe systemic changes that expand the severity of the initial injuries, the preclinical management and emergency room management (e.g., advanced trauma life support), including surgical treatment concepts (damage control surgery, bail-out surgery), were developed (Fig. 7.1). The improvements substantially influence the subsequent intensive care of patients suffering from multiple trauma.

Apart from the basic treatment concepts of intensive care medicine (e.g., volume management, lung-protective respiratory support, nutrition, and anti-infectious therapy), treating physicians must be familiar with the trauma-induced cascades and trauma-associated MOD/MOF (e.g., coagulopathy, metabolism, thermoregulation). Insufficient cognition will promote mortality and morbidity caused by the alterations inherent to MOD/MOF.

7.2 Pathophysiology of Trauma

Severe trauma is characterized by a systemic reaction characterized by immunologic, neuroendocrine, microcirculatory, and coagulatory alterations. The functionally interwoven cascades are activated sequentially and in parallel. The typical findings are:

- Acute phase reaction with the goal of activating the immune system, initiating a host defense and promoting reparative processes
- Hyperinflammation (i.e., systemic inflammatory response syndrome [SIRS]) and increased endothelial permeability
- Hypoinflammation progressing to immunoparalysis (i.e., compensatory anti-inflammatory response [CARS]) subsequent to the initial SIRS
- Recruitment of leukocytes
- Activation of the plasmatic coagulation cascades
- Neuroendocrine response and metabolic alterations

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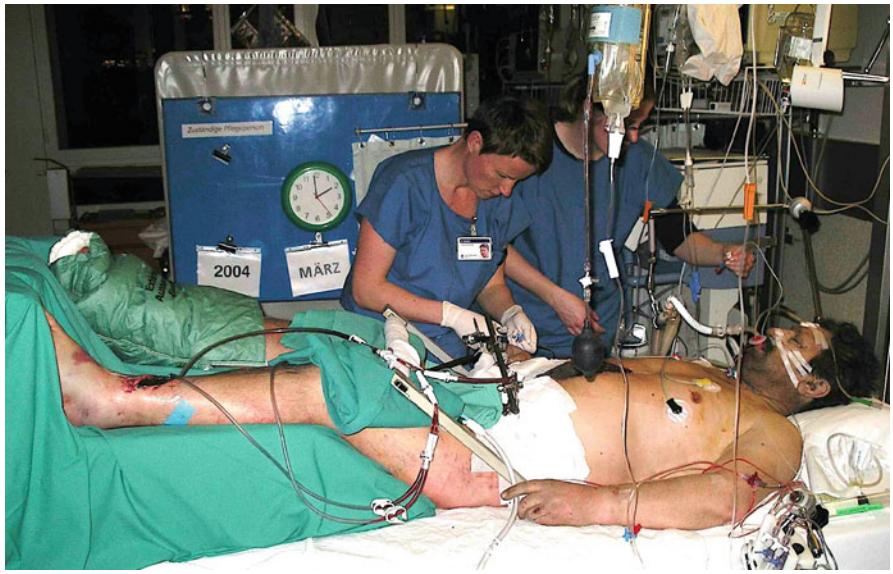

Fig. 7.1 Polytrauma patient after damage control surgery: Pelvic clamp, external fixator, open abdomen with Vacu-seal

Triggering, as well as modulation, of these trauma-related reactions results from disturbed microcirculation and alterations induced by ischemia/reperfusion.

To date, these trauma-induced cascades are best explained by a so-called two-hit model. While the first hit is induced by the initial trauma with soft-tissue damage, organ injury, and fractures, the second hit is triggered by the subsequent SIRS [1]. The second hit is caused by secondary insults resulting from endogenous or exogenous causes. Endogenous reasons are hypoxia, repetitive cardiovascular instability/hypovolemia, metabolic acidosis, ischemia/reperfusion, tissue necrosis, and infections associated with an antigenic load that activates the immune response (Fig. 7.2). Common exogenous reasons are extensive surgical interventions with additional tissue damage, extensive blood loss, disturbed coagulation, hypothermia, acidosis, mass transfusions, inadequate or delayed surgery, and inadequate or delayed intensive care treatment. Insufficient timely surgery and intensive care are reflected by the term "neglected trauma".

These diverse alterations amplify inflammatory, neuroendocrine, and metabolic reactions [2].

The trauma and subsequent "antigenic load" induce local and systemic liberation of primary pro-inflammatory mediators followed by subsequent or parallel release of anti-inflammatory mediators.

The resulting extensive SIRS that can induce multiple organ dysfunction and progress to MOF substantially contributes to an increased morbidity and mortality. In this context, levels of cytokines as well as duration of elevated cytokine concentrations correlate with the severity of injury and are associated with an increased susceptibility to subsequent infections and mortality [3, 4]. In addition, the subsequent impaired cellular immunocompetence is clearly associated with an increased risk of developing sepsis that in turn is associated with an aggravated mortality following trauma [5, 6]. This explains why additional insults known to amplify destructive cascades must be avoided. Furthermore, perfect timing of potentially damaging interventions is indispensable.

Apart from the more obvious findings that can be measured and observed at the bedside, several additional factors have been identified. In this context, genetic predisposition and gender dependency influence morbidity and mortality:

- Men show an increased morbidity and mortality compared with women [7]
- Men show a significantly increased incidence of bacterial infections [8]
- Women develop a sepsis significantly less often and thus have a better prognosis [9]
- Male gender is a risk factor for developing pneumonia and septic complications following trauma [10]
- Polymorphism of the interferon-γ-receptor-1-Gens is closely correlated with posttraumatic infections [11]

Aimed at reducing the "antigenic load" and thereby decreasing release of trigger factors, novel surgical strategic concepts during primary surgical care were developed and implemented in clinical routine. In this context, "damage control" has advanced to being an integral component in contemporary surgery [12]. A central element is to postpone the definitive surgical

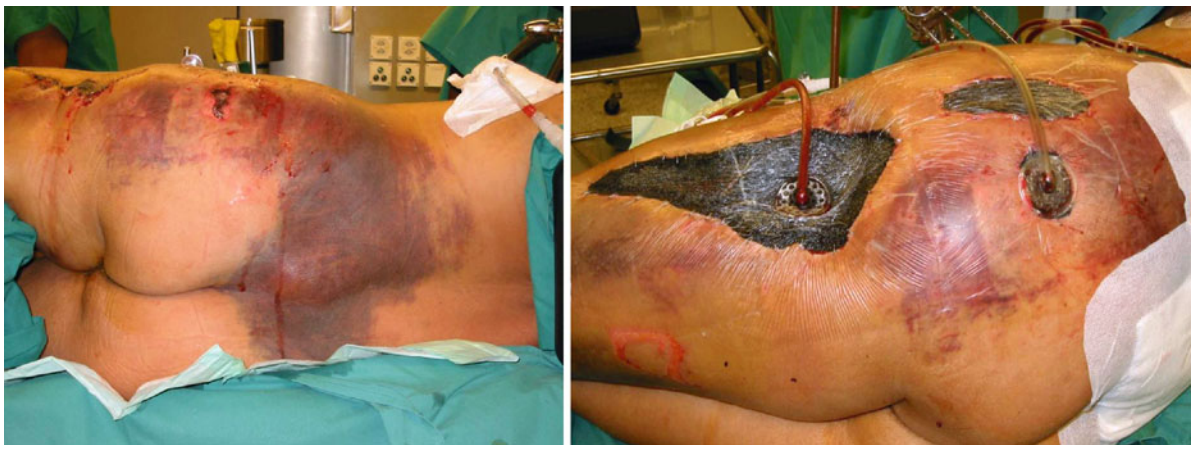

Fig. 7.2 Morel-Lavallee lesion of the pelvis (subcutaneous degloving) after débridement and vacuum sealing

care of the severely injured patient and to initially perform less invasive primary surgical stabilization procedures (i.e., external fixation, tamponade for hemostasis). Definitive surgical care should be performed after the patient has been stabilized in the ICU. The different surgical steps are strongly influenced by the individual reactions with their individual temporal development (SIRS, CARS, infections, sepsis, and hemodynamic and cardiopulmonary instability) [13]. Consequently, intensive care is crucial in stabilizing and improving the condition of the patient, thereby preparing the patient for subsequent surgical interventions.

7.3 Intensive Care

In close cooperation with trauma surgeons, intensive care medicine entails the following duties and responsibilities:

- Maintenance and restoration of vital and organ functions, including homeostasis concomitantly avoiding overcorrection
- Optimization of overall condition for subsequent surgical treatment
- Defining optimal time point for subsequent surgical treatment in cooperation with the trauma surgeons
- Stabilization, prevention, and early diagnosis and treatment of general as well as trauma surgery-related complications

Successful treatment of severely injured patients in the ICU is comprised of different parts that can only be performed using an interdisciplinary approach. The following points are of particular importance:

- *Intensive nursing care/surveillance*: Support of, and where necessary, taking over of activities of daily life (e.g., food intake, personal hygiene, movement, bedding) and medical attention and actions (e.g., analgesia, administration of fluids and drugs, complete control of vital functions and organ systems based on clinical surveillance, registration of monitored vital signs, handling of devices, etc.). Standardized physiotherapy aimed at improving and reinstituting breathing, mobilization, and movement.
- *Intensive care therapy*: Consists of supporting the endogenous compensation mechanisms and reparative processes by optimizing substrate delivery (e.g., oxygenation, perfusion, nutrition), temporary artificial support of organ functions in the event of reversible organ failure, and prevention of secondary damage. The overall purpose is to create a condition allowing subsequent healing and recovery.

In this context it is of utmost importance to practise a holistic approach (i.e., to understand the patient) in its complete complexity and to guarantee an adequate and timely flow of information between the different disciplines involved. For the trauma patient, the interdisciplinary approach is indispensable to identify problems quickly, to react adequately, and to develop a strategy based on individual development and regression.

Many new procedures are considered impossible without contemporary intensive care medicine. A good example of this is the non-surgical management of patients with injuries to the liver, spleen, and kidney and is considered the best approach in hemodynamically stable patients. As recently reported, this non-surgical management of patients who are not actively bleeding is successful in >90 % of patents with isolated trauma and

94 % of multiply injured patients. This is only possible in the event of minimal volume administration, absence of brain injury, and additional abdominal trauma, as well as additional injuries requiring surgery and age below 55 years [14]. This, however, requires a continuous, competent, dedicated, and immediate readiness in the ICU.

7.4 Special Aspects in Trauma Intensive Care

Patients with traumatic brain injury (TBI), severe trauma, and multiple injuries are usually incapable of providing detailed information regarding the circumstances of the accident and their own personal medical history. Thus, additional injuries as well as concomitant pre-existing diseases and regular medication will remain unknown during the early posttraumatic phase and can be overlooked if information is not gathered from relatives and the treating general practitioner.

7.4.1 Hemorrhage

During the early posttraumatic phase, longstanding volume deficit and the hypovolemic-hemorrhagic shock are the most deleterious alterations that determine subsequent development and incidence of potentially devastating consequences. The most prominent pathophysiologic consequence is microcirculatory impairment, resulting from hypovolemia and subsequent sympathic-adrenergic precapillary vasoconstriction. These changes are accompanied by nitric oxide-induced vasodilation that in turn causes shunts and impairs nutritive capillary perfusion, explaining heterogenous capillary perfusion. As a consequence, impaired organ perfusion with evolving tissue acidosis and lactate production with sustained increase in endothelial permeability will aggravate the underlying condition as a result of progressive edema formation. In case of persisting ischemia, degradation of energetically rich phosphates in conjunction with free oxygen radical induced mitochondrial damage will result in irreversible structural and functional cell injury. The degree of the damage strongly depends on the extent and duration of the underlying hypovolemia. Reperfusion injury resulting from restored perfusion is feared for its generation of highly toxic free oxygen radicals. These, in turn, are known to damage cell membranes by peroxidation of cell membrane lipids, accounting for resulting vasoplegia and swelling.

The criteria defining diagnosis of hemorrhagic shock have been refined over the years. The early definition of hemorrhagic shock consisting of a lost blood volume of 1–2 l [15] was substituted by the systolic blood pressure <90 mmHg [16] and the vital parameters were used to estimate prognosis [17]. Based on the observation that the cardiovascular system is able to maintain an adequate systolic pressure by a compensatory increase in heart rate despite a progressive volume loss, the so-called shock index was introduced [18]. The shock index consists of an easy-to-calculate formula dividing systolic blood pressure by heart rate. A shock index <1 is highly suggestive of hemodynamic instability resulting from hypovolemia. In addition to the absolute values, duration of shock is also of crucial importance. In this context, a threshold of 70 min appears to be clinically relevant [19, 20]. Additional predictive factors are heart and breathing frequency upon admission to the hospital [21–23]. Especially in younger patients with well preserved compensation mechanisms macrocirculation (blood pressure, heart frequency, filling pressures) may appear "normal" although microcirculation still is impaired (i.e. serum lactate concentrations 12 hours after admission). This condition is referred to as "occult" hypoperfusion and is accompanied by an increase in infectious and other complications [24–25].

Diuresis can also be used as a parameter to estimate volume depletion, provided urine production and urine release are not hampered by pre-existing renal disease and injuries to the urinary tract system.

The overall accepted goal is to swiftly restore sufficient circulation, maintain hemodynamic stability by improving organ perfusion and microcirculation using volume administration via large bore catheters, and by reducing further temperature loss. The primary goal is quantitative restoration guided by hemodynamic parameters, restoration of peripheral perfusion, restitution of sufficient diuresis (0.5–1 ml/kg body weight, >2 ml/kg body weight in case of rhabdomyolysis, that is, creatin kinase (CK) >5,000 IU/ml), and reduction or normalization of arterial lactate, pH, and base excess values. In this context, lactate >2.5 mmol/l and negative base excess that has a higher negativity than – 8 mmol/l represent important threshold values of acidosis (pH<7.2), and negative base excess values were shown to significantly predict outcome in traumatized patients [26].

Because low pH values did not unanimously correlate with the outcome, pH alone should not be used as a basis to limit therapeutic interventions. Predictive sensitivity is increased by the presence of other factors such as blood loss, hypothermia, increased lactate levels with negative base excess, and coagulopathy.

Perhaps more important than the absolute values is how long it takes to normalize lactacidosis and negative base excess during adequate treatment consisting of volume management, hemodynamic support, and rewarming. Persisting negative base excess and lactacidosis exceeding 24 h is clearly associated with significantly increased morbidity and mortality [24, 27]. Lactate-guided volume management was associated with significant reduction in mortality despite absence of improved signs of vasopressor-driven hemodynamic stabilization determined by measurements using the pulmonary artery catheter [24, 28].

7.4.2 Hypovolemia and Management of Hypovolemic/Hemorrhagic Shock

Qualitative volume replacement consists of substituting oxygen carriers (hemoglobin), factors of hemostasis (plasmatic coagulation factors, platelets), and correcting existing intravascular volume depletion. While a hemoglobin target of 9–10 g/dl had been targeted for many years, a lower hemoglobin count of approximately 7 g/dl was shown to improve outcome [29]. In this context, bedside point of care analysis of hemoglobin as well as glucose and lactate have significantly influenced morbidity and mortality and have reduced the number of resources consumed [30].

7.4.3 Disturbed Coagulation

Loss of coagulation factors and platelets as a result of uncontrolled hemorrhage, as well as dilution of coagulation factors and platelets resulting from excessive fluid replacement and reduced ionized calcium concentrations, hypothermia, and acidosis, in conjunction with the type and extent of injury (e.g., brain) all contribute to disturbed hemostatic mechanisms [31]. Contrary to the diffuse intravascular coagulopathy which includes thrombus formation we are confronted with traumatic intravascular coagulopathy (TIC) in which coagulation is hampered. In this context, the clinical picture of coagulopathy is not always reflected by laboratory values in a timely fashion. It is critical to base the subsequent administration of various coagulation factors on clinical judgment that, in turn, is strongly influenced by individual experience. Obvious bleeding requires immediate correction during the process of obtaining laboratory values that can take up to 60 min before results of plasmatic coagulation can be integrated into the fine-tuning of correcting TIC. Bedside analysis using thrombelastography may aid in faster and differentiated decision making. It is important to keep in mind that other elements apart from the concentration of coagulation factors and platelets are responsible for hemostatic failure [32]. An important devastating factor is underlying hypothermia that will disturb the entire coagulation cascade [33]. Inhibition of enzymatic reactions is reflected by prolonged prothrombin- and thromboplastin time during hypothermia even when the measured coagulation factors are normal. Another important technical detail is that functional coagulation tests are performed at 37 °C and not corrected for the actual temperature of the injured patients. This, in turn, will underestimate the extent of disturbed coagulation [34]. In addition, platelet function is impaired by hypothermia via reversible, temperature-dependent disturbance of thromboxane B2 production that will prolong the hemorrhage time [35]. Additional changes of the enzyme kinetics will delay initiation and propagation of platelet aggregation despite adequate platelet substitution [36]. This, in turn, explains the often seen poor correlation between platelet count and progressive bleeding in patients receiving massive transfusions and can be seen as an indication for platelet transfusion despite normal platelet count [37].

The following additional and preexisting coagulation disorders can aggravate the acute coagulation disorder:

Release of tissue factors because of severe TBI, pharmacologic anticoagulation, functional disturbance of platelet functions due to pharmacological and endogenous (hepatic and renal insufficiency) influences, hemophilia and deficit in von Willebrand factor. Moreover, consumption of factors due to massive transfusions (MT) and underlying hemorrhage resulting in low 2,3-DPG concentrations, low activity of factors IV (ionized calcium), V, VIII, and XIII, low fibrinogen levels, dilution thrombocytopenia, functional platelet disturbance, hypothermia, and acidosis have to be taken into consideration. Massive transfusion is thought to increase citrate concentration which chelates calcium that in turn will impair the coagula-

tion cascade and is also believed to increase protein C that can inhibit plasminogen activator inhibitor, thereby resulting in sustained activation of plasminogen; which can generate hyperfibrinolysis, and in turn, promote the development of microvascular hemorrhages at the mucosa, injuries, puncture sites, and sutures. Microvascular hemorrhages generally result from dilution coagulopathy and an increased consumption of hemostatic factors that can lead to diffuse bleeding that cannot be managed surgically. Consequently, the diagnosis of such a hemostatic disturbance must be made early to allow timely correction and prevention of aggravated complications. The diagnosis of such a hemostatic disturbance strongly depends on the vigilance and experience of the treating physicians in all involved disciplines.

Contrary to dilution coagulopathy, the combination of consumption and dilution coagulopathy will result in more severe disturbances seen in laboratory parameters reflected by a larger decrease in platelets, international normalized ratio, and fibrogen, as well as a pronounced prolongation of the activated prothrombine time (aPTT). Therefore, in patients requiring massive transfusion use of fresh frozen plasma (FFP) in order to provide a balanced set of activating and inhibitory coagulation factors (including Factor V) still may be indicated particularly because blood donors carrying a higher risk for induction of transfusion-related lung injury have been identified and excluded from FFP donation. This procedure is supported by a meta-analysis published in 2010 reporting that plasma infusion at high plasma: RBC ratios in patients undergoing MT was associated with a significant reduction in the risk of death [odds ratio (OR), 0.38; 95 % confidence interval (CI), 0.24–0.60] and multiorgan failure (OR, 0.40; 95 % CI, 0.26–0.60) [38]

Correction of disturbed coagulation occurs by supplementing the different components. To date there has been considerable controversy regarding correction strategies of impaired coagulation in trauma patients. Whereas in slight or moderate bleeding, targeted supplementation of coagulation factors monitored by means of thrombelastography or preferably thrombelastomety (i.e. ROTEM) as point of care testing (POCT) might be feasible, in severe and life-threatening bleeding this approach might fail because of the time lag between sample drawing and availability of the results. Therefore, as mentioned above use of fresh frozen plasma (FFP) in severe bleeding still is an important option in order to achieve hemostasis. However, certain factors, frequently fibrinogen and sometimes factor XIII must be additionally supplemented because provision by FFP alone might be insufficient. Factor XIII is a protein responsible for stabilizing the formation of a blood clot. In the absence of Factor XIII, a clot will still develop but it will remain unstable. If Factor XIII is deficient, the tenuously formed clot will eventually break down and cause recurrent bleeds. Suspicion in Factor XIII deficiency should be raised if in a patient with clinical relevant bleeding (i.e. diffuse micro vascular bleeding without clearly identifiable bleeding source if FXIII-activity is below 60 %). In such circumstances administration of FXIII in a dose of 30 IE/kg BW may be indicated. Furthermore, in trauma patients a positive influence on the development of the systemic inflammatory response syndrome (SIRS) could be demonstrated [39, 40, 41].

7.4.4 Hypercoagulability

Apart from possibilities of uncontrolled bleeding, trauma patients are exposed to a considerably elevated risk of thromboembolic complications in part because of an imbalance of pro- and anticoagulating factors and an increase in pro-coagulant factors (i.e., fibrinogen) resulting from a post-traumatic acute phase reaction.

Venous thromboembolic complications lead to a significant increase in morbidity and mortality and present in two forms:

- Deep vein thrombosis
- Lung embolism

A prophylaxis in trauma patients is important because clinical investigations for establishing a diagnosis are not sensitive enough. In a meta-analysis of 73 studies, however, it was found that none of the prophylaxis used was superior to another, even compared with no prophylaxis. Moreover, spinal injuries, spinal cord injuries, and age were identified as major risk factors for thromboembolic complications [42]. A prophylactic placement of caval filters may reduce the incidence of lung embolism [43, 44]. Our own experience demonstrates that cava filters can temporarily be inserted and removed in a high percentage of patients, and that they provide reliable protection against clinically relevant lung embolism [45–47].

7.4.5 Quantitative Volume Substitution

The choice of the type as well as of the amount of volume substitutes is still a matter of controversy. Young, healthy, trauma patients generally tolerate large amounts of intravenous fluids. In elderly patients, on the other hand, fluid and sodium overload may induce congestive heart failure, impaired blood–gas exchange, and hypoxia leading to the classic day 3 myocardial infarction and increased mortality, particularly if they are already suffering from a preexisting heart condition or renal insufficiency [48, 49].

It must be stressed that the classic 0.9 % sodium chloride solution is somehow toxic because it leads to hyperchloremic and dilution acidosis that exacerbates the existing acidosis in patients [48, 50, 51]. If the situation goes unrecognized, the attempt to correct the acidosis with additional fluid replacement will lead to a volume overload, with all its consequences [44, 52]. Additional unwanted effects of pure crystalloid fluid replacement are the decrease of cardiac output [53] and stroke volume by as much as 20 % even if adequate end diastolic pressure is achieved [44]. Furthermore, it has been shown that after pure crystalloid infusion – as opposed to higher concentration hydroxy ethyl starch – higher concentrations of proinflammatory cytokines, a depression of peritoneal macrophage function, and a higher expression of adhesion molecules can be found [54].

Patient's microcirculation is very vulnerable to become compromised during the acute phase. Once a SIRS is induced, fluid overload and edema potentiate capillary leak leading to a local loss of control of inflammatory mediators and an increase of edema [55]. Furthermore, volume substitution with crystalloids alone leads to a reduced colloidosmotic pressure by up to 50 % with a consecutive fourfold increase of pulmonary transendothelial flux. Corrective administration of colloid fluids will consecutively reduce inflammation and edema [56–58].

In summary, volume expansion will improve cardiac output to a certain point via the Starling mechanism. Beyond that point it will, however, worsen cardiac function and promote edema formation, particularly if based solely on crystalloids [59]. Therefore, monitoring of stroke volume and cardiac output before and after fluid challenge is a more reliable alternative than assessing heart frequency and blood pressure alone [60–62].

7.4.5.1 Fluid Replacement and the Bowel System

In recent years, several studies have shown that an increased administration of sodium and water may have numerous detrimental effects on the gastrointestinal system: The edema in the splanchnic area leads to an increase in intraabdominal pressure that in turn can lead to a decrease of tissue oxygenation. Apart from intestinal permeability dysfunction, which is suspected to account for increased bacterial translocation, a protracted dysmotility of the bowel can be observed, causing intolerance to enteral nutrition. The resulting gastrointestinal dysfunction increases the risk of ventilation-associated pneumonia and therefore increases morbidity and mortality, as well as length of stay in the ICU and time to discharge [49, 59]. It is well recognized that the excessive use of crystalloids constitutes a risk factor for the development of abdominal compartment syndrome (ACS) in trauma patients. It has been shown that supranormal volume replacement, as it is often applied, will lead to an increase of the amount of crystalloid fluids infused, an increased incidence of ACS, and an elevated rate of MOF with a consecutively higher mortality rate [63–66]. Liberal infusion of crystalloid fluids in young trauma patients may lead to the secondary development of ACS even in the absence of abdominal injuries [67]. In most studies, the mortality rate resulting from ACS-induced lung failure or MOF is more than 50 % despite aggressive surgical abdominal decompression [63–66]. Administration of more than 3 l of crystalloid fluids prior to transfer of the patient from the Emergency Department to the ICU is highly predictive for the development of primary or secondary ACS.

Given these pathophysiological effects of crystalloid fluid infusion, alternative volume replacement strategies are warranted using colloid fluids or hypertonic saline, and considering early administration of vasopressors in order to restore vascular tone [63–66, 68].

7.4.6 Hypothermia

There is an increased risk for hypothermia leading to elevated mortality during volume substitution particularly during massive transfusions, commonly necessary in multiple trauma patients. It has been shown that prolonged hypothermia during volume therapy has deleterious effects on cardiovascular parameters and

hepatocellular function, and additionally leads to an upregulation of cytokines. Conversely, heating to 37 °C during volume restitution improves cardiovascular and hepatocellular function and reduces cytokine concentrations [69]. Furthermore, rewarming increased hepatic blood [70]. Heating of the patient to normothermia, therefore, must accompany volume substitution following traumatic hemorrhage.

Thermal homeostasis depends on the balance of factors leading to heat loss (conduction, convection, evaporation, and radiation) and the capacity of the body to produce heat. It is important to note that heat loss and the drop in body temperature begin immediately following trauma and are further propagated by hypoperfusion, shock, prolonged exposure, immobility, and old age. If preventive measures are not implemented, cooling continues in the emergency department and the ICU, especially if the injured patient is exposed in a temperature gradient of more than 15 °C and if he or she is not immediately covered by warm blankets. A drop of core body temperature to 35 °C is regarded as being clinically significant, and a drop to below 34 °C indicates that early intraabdominal packing may be warranted [20]. More than 20 % of trauma patients and 50 % of injured patients undergoing laparotomy are hypothermic upon exit from the operating room [71]. These patients require significantly more volume substitutes and transfusions, and their need for catecholamine is increased, which leads to a higher incidence of organ dysfunction, a longer stay (LOS) in the ICU, as well as a higher mortality [72]. However, hypothermia may possibly be a surrogate marker for the severity of the injury and the consecutive shock, making it difficult to account for the true effect of hypothermia alone in the clinical setting [73].

Rewarming of the patient in the ICU can be achieved through warming of ventilation gas, warmed intravenous fluids, heating blankets, or by invasive heating devices (i.e., Cool Guard®).

7.4.7 Acidosis

Acidosis resulting from hypovolemia can contribute to coagulopathy, which in turn may promote hemorrhage and hypovolemia. Correction of coagulopathy therefore requires not only hemostasis but also restoration of tissue perfusion and oxygenation through adequate volume substitution, transfusion, and pharmacological circulatory support. Apart from clinical signs (e.g., peripheral perfusion and adequate diuresis) the endpoints of restoration of sufficient tissue perfusion are normalization of serum lactate levels, base deficit, and central venous O_2 saturation. However, they remain controversial [74–76].

7.4.8 Prophylaxis and Therapy of Organ Damage

7.4.8.1 Circulation

Volume substitution for stabilization of the circulatory system is not an issue only at the start of intensive therapy. Apart from the primary hypovolemia resulting from hemorrhage, secondary trauma reactions can cause protracted hypovolemia and fluid distribution disturbances. They include vasodilation and a capillary leakage because of posttraumatic inflammation caused by inflammatory mediators (SIRS). The use of vasopressors is often unavoidable because of SIRS-associated vasodilation.

Despite adequate volume substitution, intractable shock, particularly in the context of thoracic trauma, may indicate cardiac injury (valvular injuries, cardiac tamponade, or coronary dissection) or acute cardiac decompensation resulting from concomitant heart disease (coronary heart disease, cardiomyopathy). Transesophageal echocardiography has proved to be a useful bedside examination in such situations.

7.4.8.2 Respiration

Respiratory failure primarily resulting from pulmonary insufficiency or from extrapulmonary causes is commonly encountered in the trauma patient. If the cause is of extrapulmonary origin (abdominal hypertension, left heart failure, etc.), these problems should be addressed and non-invasive ventilatory support strategies should be employed. Respiratory failure resulting from a pulmonary disturbance (lung contusion, aspiration), endotracheal intubation, and ventilation using positive end expiratory pressure often cannot be avoided. In general, however, modern ventilation strategies aim at keeping ventilator support as short and least invasive as possible. Therefore, controlled ventilation of the trauma patient is mandatory only if an acute TBI is present or in cases of severe hypovolemic shock. In the latter case, maintaining spontaneous ventilation will require a blood flow of 20–30 % of cardiac output. Therefore, relieving the respiratory muscles by controlled ventilation is essential in this phase. In all other cases of respiratory insufficiency, the goal currently is to preserve at least parts of

spontaneous breathing activity or the use of non-invasive support [77, 78].

Restriction of controlled ventilation lies in the fact that it causes a myriad of undesired effects and carries risks (circulatory depression, ventilator-associated pneumonia, alveolar volutrauma and barotraumas, etc.) that can lead to increased morbidity and mortality, as well as a prolonged LOS in the ICU and hospital. Recent studies have shown that assisted spontaneous breathing (ASB), as opposed to controlled mechanical ventilation, increases gas exchange, systemic blood flow, and tissue oxygenation. Computed tomography studies have revealed that the improved gas exchange is a result of a re-distribution of ventilation and end expiratory gas distribution in dependent lung areas. ASB, therefore, avoids the unwanted cyclic end expiratory alveolar collapse in dependent zones [79]. Another advantage lies in the lower requirement for sedation, which again may lead to a shorter LOS in the ICU [78]. Early ASB calls for intensive support such as breathing and physical therapy, posturing, and early mobilization. Therefore, surgical treatment must aim for early fracture stabilization, even if only temporary (e.g., by external fixation).

In the event of controlled mechanical ventilation, lung injury as a result of the ventilation, must be avoided. Central elements comprise limitation of tidal volume to 6 ml/kg body weight, depending on the severity of lung injury, in order to avoid volutrauma, as well as limiting peak inspiratory pressure to below 30 mbar to avoid barotrauma (so-called "Lung Protective Ventilatory Strategy") [80]. Switching to assisted spontaneous breathing modes as early as possible should remain a high priority in these cases.

Techniques such as pumpless extracorporeal lung assist up to extracorporeal mebrane oxygenation (ECMO) (Fig. 7.3) may be needed in order to allow for sufficient gas exchange obeying lung protective ventilatory strategies.

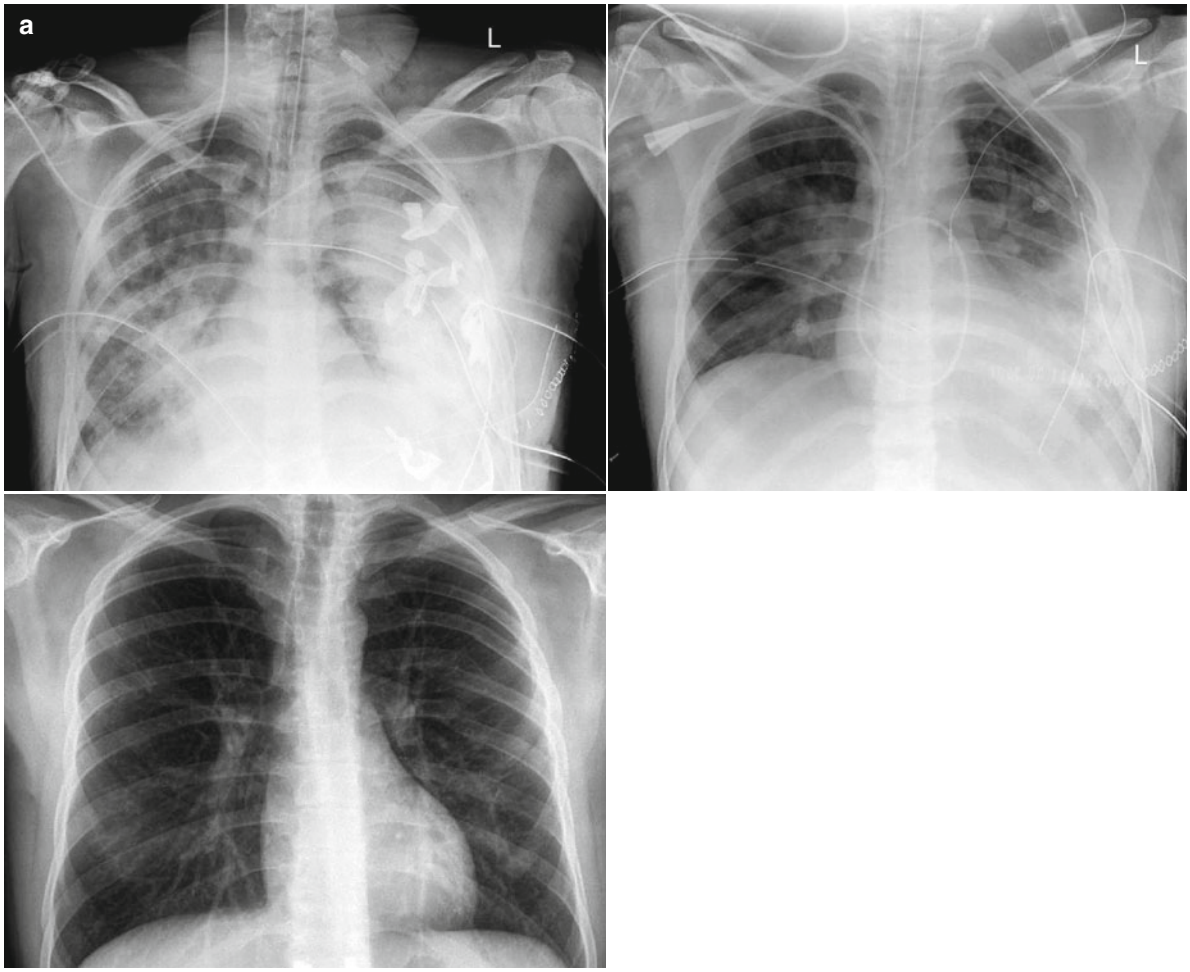

Fig. 7.3 (**a**) Severe chest trauma: Sequence of chest radiographs. (**b**) Patient with PECLA (Pumpless ExtraCorporal Lung Assist)

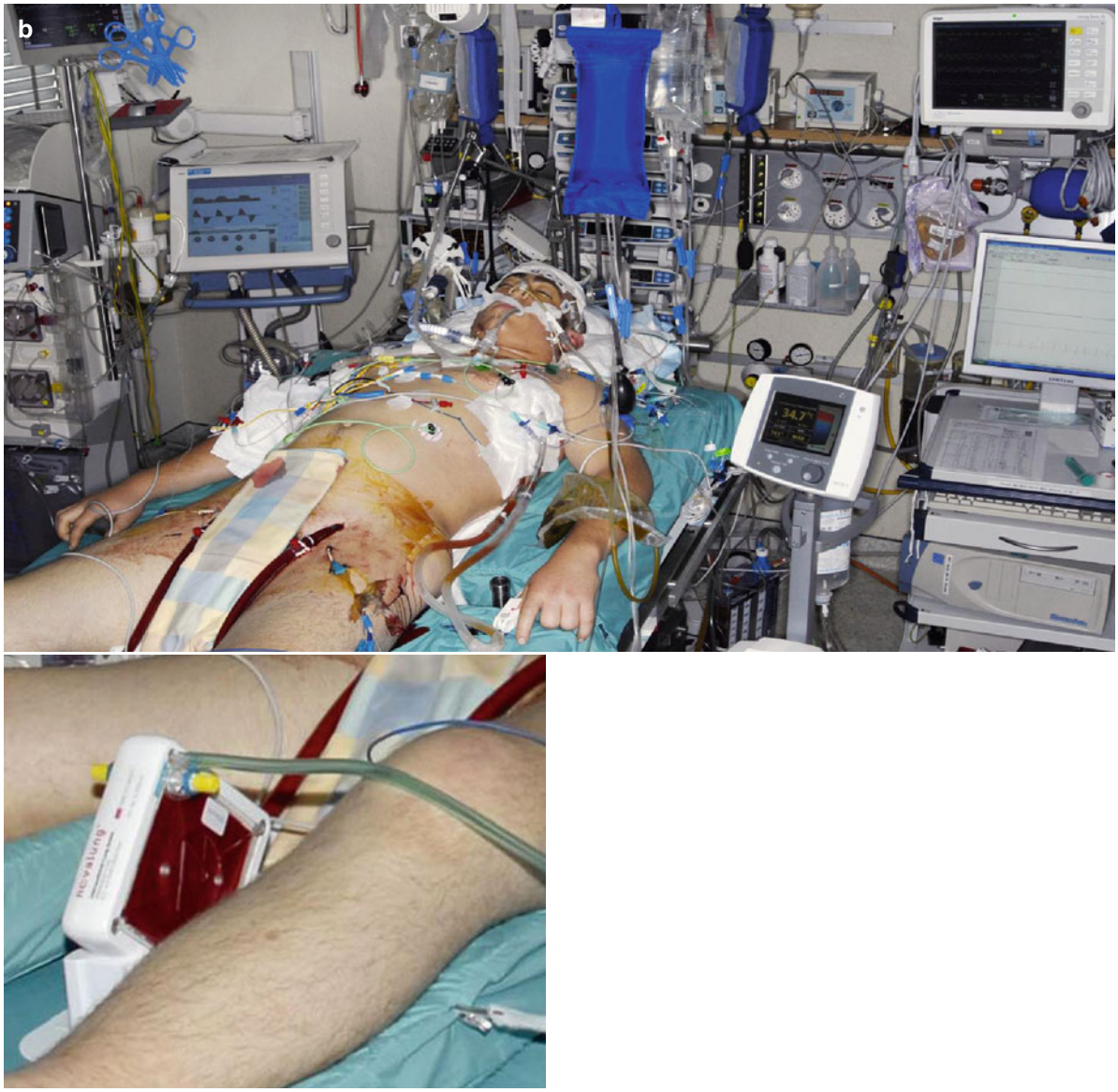

Fig. 7.3 (continued)

7.4.8.3 Bowel System

The bowel system acts a classic shock organ and is rather inaccessible to clinical diagnostic tests and leads to compromising events that are not being noted and treated in a timely manner. The occurrence of stress ulcers is often the result of organ dysfunction because of a relative or absolute hypoperfusion of the splanchnic area. It can therefore not be prevented by a pharmaceutical ulcer prophylaxis.

Complications such as SIRS, severe infections, or sepsis are characterized by a splanchnic hyperperfusion and increased oxygen transport caused by a stress hormone and cytokine-induced hypermetabolism, as well as an increased hepatic gluconeogenesis. Various cytokines, such as Interleukin-6, induce an acute phase reaction with synthesis of acute phase proteins by the liver. This hypermetabolism in turn can lead to a mismatch between actual oxygen consumption and availability.

The gut mucosa, lined with enterocytes, probably plays an important role in the pathogenesis of MOF, and is a controversial topic [81]. In order to limit damage to the mucous membrane, restoration of splanchnic

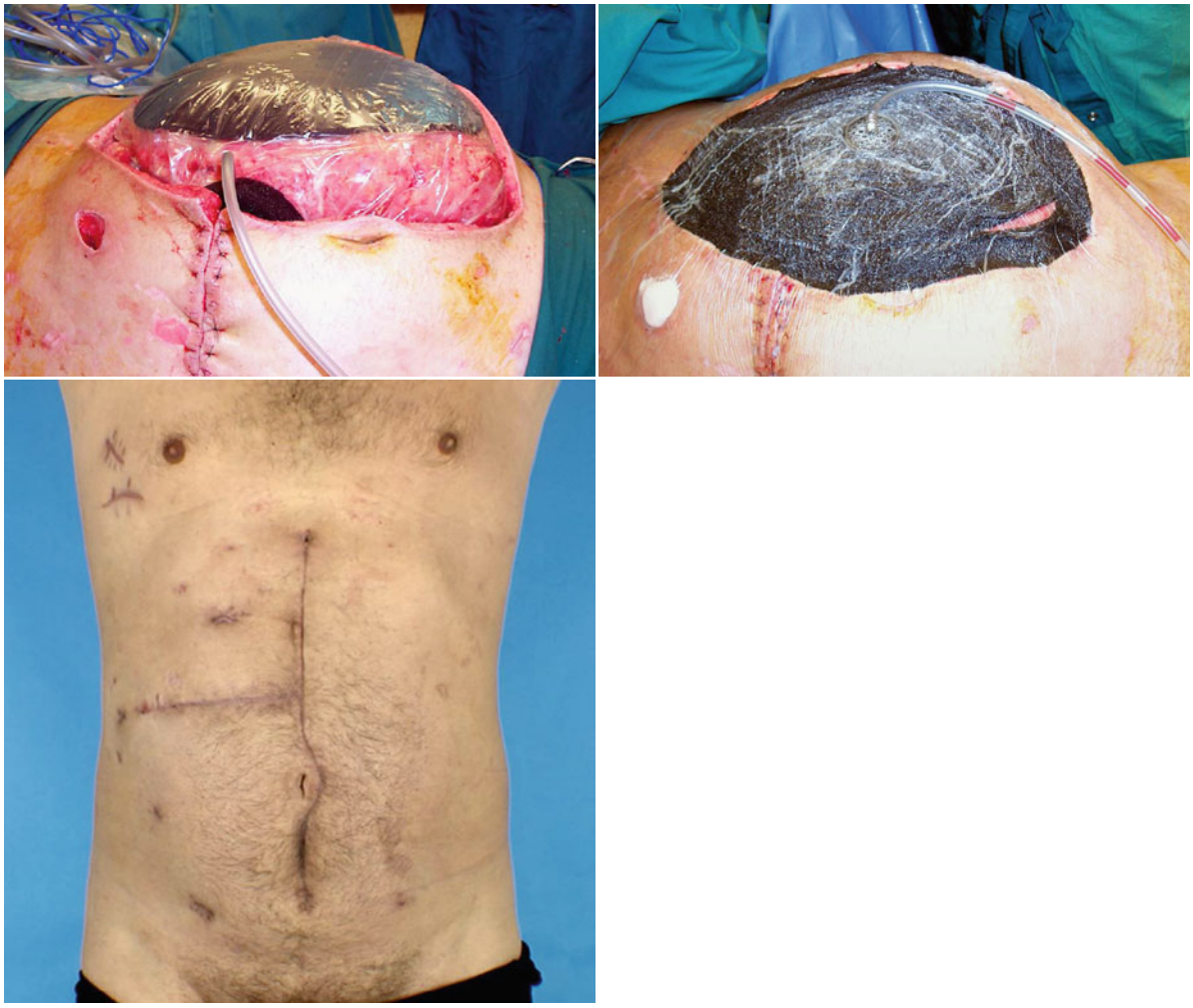

Fig. 7.4 Abdominal gunshot wound, perihepatic packing, open abdomen with Vacu-seal. Healed abdominal wall

perfusion through volume therapy and shock treatment, as well as endoluminal administration of substrates by early enteral nutrition, is essential.

Abdominal Compartment Syndrome

An elevated intraabdominal pressure, termed ACS, may heavily impair systemic circulation and perfusion of abdominal organs [82].

ACS can be induced by abdominal trauma with consecutive gut edema, intra- and retroperitoneal hematomas, intra-abdominal packing (in damage control surgery) (Fig. 7.4), and by excessive infusion of crystalloid fluids (see above). Venous backflow decreases because of direct compression of the inferior vena cava and venous pooling of blood in the pelvic area and the lower extremities. Additionally, elevated intra-abdominal pressure leads to diaphragmatic elevation and a relative increase of cardiac afterload, causing a decrease of cardiac output [83].

Visceral blood flow to the liver, the gut, and the kidneys is impaired. Renal function is particularly at risk because elevated intra-abdominal pressure also impairs renal run-off and also because the kidneys are particularly vulnerable to direct organ compression, leading to an elevated vascular resistance, and therefore, further impairment of kidney perfusion [84]. So renal dysfunction up to anuria is a common complication of ACS.

Elevated intra-abdominal pressure causes a decrease of thoracic volume and pulmonary compliance. Ventilation-perfusion mismatch and impaired blood oxygenation follow.

Perihepatic packing as a damage control procedure may be a reason for a significant ACS. Compression of

the suprarenal vena cava impairs kidney function, in which case a second look at abdominal decompression through hematoma evacuation and/or (partial) removal of the packing may be necessary [83, 85]. This will also improve visceral perfusion, cardiac function, and respiratory mechanics [86]. In the absence of ACS, packing should be left in place until the patient is hemodynamically stable, acidosis has been corrected, the patient is normothermic, and sufficient coagulation has been restored. Premature removal of abdominal packing may otherwise only be warranted in order to decrease the risk of abscess formation if the abdominal cavity has been substantially contaminated [87]. Moreover, ACS still can occur even if an open abdomen strategy has been applied. This is referred to as "tertiary ACS" and also may require premature return to the operating theatre for decompression (i.e. exchange of soaked packing tissue).

Kidneys

The pathophysiology of acute renal insufficiency in the polytrauma patient is driven by shock resulting from hypovolemia. Ischemia, focal hypoxia, and dysfunction of the coagulation system lead to functional and structural damage presenting itself clinically as oligo/anuria. Therefore, a central element of preventing acute renal failure is urgent as also decisive correction of hypovolemia and avoiding hypotensive states. There are no other possibilities of protecting the kidney; neither application of dopamine nor administration of diuretic drugs has any scientific basis. On the contrary, it is most likely that such interventions will lead to a deterioration of renal function (e.g., because of worsening of renal energy homeostasis).

A trauma-specific form of renal failure known as crush syndrome is the result of the destruction of large amounts of muscle mass where myoglobin reaches the intravascular space. Myoglobin is toxic for the kidneys because it is filtrated and may then mechanically clog the tubular system because of precipitation. Additionally, small amounts of myoglobin can be absorbed through endocytosis into the tubular cells which will lead to the formation of highly toxic hydroxyl radicals through the release of porphyrin complexes of iron [88]. The standard therapy for crush syndrome entails the liberal administration of large quantities of isotonic crystalloid fluids (correction of hypovolemia, increase of diuresis), bicarbonate (increases solubility of myoglobin in the urine through alkalinization), and mannitol (increases tubular urinary flow). As long as systemic arterial blood pressure can be maintained, substances (i.e. calcium antagonists) may be used to inhibit the myoglobin-induced vasoconstriction (i.e., calcium antagonists). Any type of vasoconstriction during hypovolemic shock is detrimental and worsens the prognosis of acute renal insufficiency.

In the event of acute oligo/anuric renal failure, continuous renal replacement therapy should be employed. It has been shown that for crush syndrome, continuous hemofiltration is superior to dialysis.

7.5 Summary

As the trauma surgeon is responsible for all surgical aspects of trauma and decides whether, and at what stage of treatment, support from other specialties is needed, the trauma ICU specialist aims for restitution of vital and physiological functions taking into account the specific host response by the patient. Major goals in the initial phase include rewarming, correction of coagulation, acidosis, hypovolemia, monitoring/avoidance of compartment syndromes in order to optimize oxygenation, and tissue perfusion. Therapy is later directed toward reestablishing physiological functions, including early enteral nutrition, infection control, and avoiding secondary injuries and complications.

Consultation between the trauma surgeon, ICU staff, and other medical specialties guarantees optimal assessment, diagnostics, and treatment tailored to the specific needs of the individual patient. The major goal is maintenance and/or restoration of all functions that are needed to assure a good long-term quality of life.

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