Evaluation and Critical Care Management of the Burn Patient

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Franz W. Baruffaldi Preis and Antonella M. Citterio

Abbreviations

ABA	American Burn Association
BLS	Basic Life Support
ECM	extracellular matrix
EGF	Epidermal growth factor
IGF	insulin-growth factor
IL	Interleukin insulin-growth factor
MOFs	Multi-Organ Failure Syndrome
PDGF	Platelet-derived growth factor
TBSA	total body surface area
TGFβ	β Transforming growth factor
TNF	tumour necrosis factor
VEGF	vascular endothelial growth factor
W	Weight
WHO	World Health Organization

Key Points

- Burn injuries are acute trauma that can affect people in different situations and ages.
- Burn injuries are classified according to the burning agent, the depth and the extent of lesions. All these factors are connected with the severity of the trauma.
- Lound and Browder chart is used to calculate the size of burn wounds and is utilized for children.
- The burn shock is a distributive and hypovolemic shock mediated by cytokines, chemokine, platelet, and inflammatory factors. There are two phases after trauma: the ebb (hypovolemic and hypometabolic) and the flow (hypermetabolic) phase.
- Fluid resuscitation follows the Parkland formula with crystalloids (Ringer Lactate). The use of colloids (albumin or fresh frozen plasma) is indicated at least 8 h after trauma.
- Nutrition must be started early to prevent catabolic loss and to prevent infection.
- Occlusion, humid, and heat preserving medications are indicated.
- Early excision and wound closure is the standard of care. Enzymatic debridement can be used for fast and safe debridement.

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5.1 Introduction

Burn trauma, the acute and rehabilitation phase, affects the patient on a physical and psychological level. This is directly related to the resultant scarring which modifies the appearance. The psychological factors may influence the patient's possibility of resuming normal social and working life. According to the WHO, before the COVID 19 pandemic, 11 million burn traumas had occurred worldwide and of these, 180,000 deaths. In Italy, it is estimated that around 100,000 cases suffer burned injuries each year, with approximately 10,000 requiring hospitalisation, and a total of 500 deaths a year. In 2020, the goal was to reduce the number of deaths by 50%, although current data show that the target is yet to be reached. Burn agents, in agreement with the ABA report, most frequent causes are fire, 40% of cases; followed by scalds, 30% of cases; and followed by contact, chemical and electric. Seventy percent of all accidents happen in domestic areas, and the paediatric population is the most vulnerable, including the elderly and persons with chronic disease. Burn trauma is no longer considered an unpredictable wound by the WHO but rather the result of factors that lead to the burn trauma itself. For this purpose, the Haddon Matrix for burn prevention, like other similar applications in public health, is used. The Haddon Matrix is a tool to assist in the development of ideas for preventing many types of injuries and used

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Table 5.1 Matrix protocol

Contributing factors	Before injury	During injury	Post injury
Host and human factors			
Object or substance			
Physical environment			
Socio-cultural environment			

by governments for burn prevention. The Matrix Protocol has four columns and three rows. In the columns, contributing factors are described, in the rows, the time period is indicated (Table 5.1).

5.2 Burn Category

The burn trauma classification takes into account the burning agent, where there is transferred energy that causes immediate, due to fire, or delayed, scarring, and tissue necrosis. Burn trauma is generally associated with heat generated by liquids, solids, fire or, in other cases, friction, cold, radiation, electricity, or chemical agents.

Thermal burns are injuries caused by excessive heat, typically from contact with hot surfaces, liquids, steam, or flames. A minimum of 44 °C of direct heat is required to cause a burn injury. The duration of the contact, the degree of the temperature, and the body area involved, determine the degree of burn.

Electrical burn injury depends on the voltage, notably the injury is not limited to a superficial level but penetrates beyond the fascia. The systemic consequences may include heart arrhythmia, myoglobinuria, acute kidney injury, muscle, and bone necrosis, nerve damage.

Chemical burns due to acid or alkali salts. The burn is due to the corrosive effects of these substances. Chemicals induce harm due to pH fluctuation, which is gradual, and long-lasting until the substance is inactivated (Fig. 5.1).

Recognising the agent is of paramount importance as gold standard treatment is differentiated accordingly. For example, early surgical treatment is required with flame burn, however, not in frostbite burn. In addition, the depth and extent of the burn determine the severity of the clinical outcome and the necessity for surgical treatment. Burn depth classification is in four degrees (Table 5.2).

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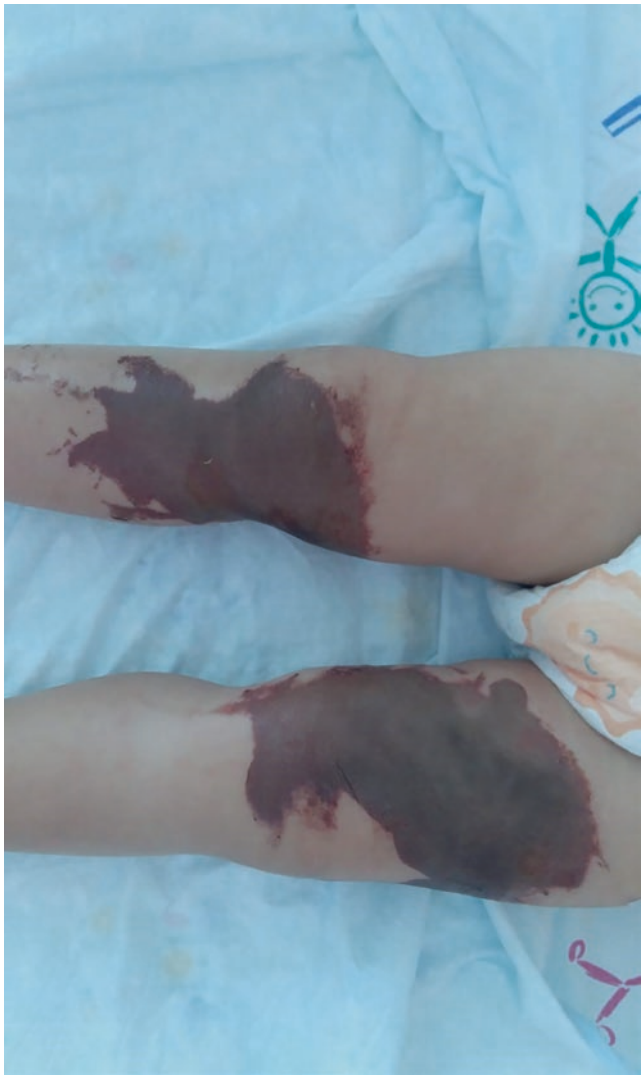

Fig. 5.1 Chemical burn

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Table 5.2 Burn depth classification according to estimation

First degree	Second degree	Third degree	
Superficial thickness	Partial or Intermediate thickness	Full thickness	Fourth degree
Affecting only part of the epidermis	Involvement of epidermis and superficial part of the dermis	Full thickness	Muscles and/or bone involvement
• no blisters	• blisters	• dry	• dry
• no scars	**If superficial**	• contractures	• ontractures
• pain	• moist	• compartment syndrome	• compartment syndrome
	• hyperaemic	• scarring	• scarring
	If deeper		
	• drier		
	• with a reticular pattern		
	• pain		
No surgery required	Increasing involvement correlates to augmented risk for surgery, scarring and infection	Skin graft required	Risk of losing involved areas
		High risk of infection	
		Increased mortality	

5.3 Burn Extention Measurement

The burn wound sides are calculated using the Rule of Nine (Table 5.3). More accurate estimation can be made using the Lound and Browder chart, which can also be used for children, and is more accurate (Fig. 5.2).

Table 5.3 Rule of Nine or Wallace

9%	Head and neck
9%	Upper limb
18%	Lower limb
18%	Trunk (thorax and abdomen)
18%	Trunk (back and buttocks)
1%	Genitals

Age in Years	0	1	5	10	15	Adult
A-½ of head	9½	8½	6½	5½	4½	3½
B-½ of one thigh	2¾	3¼	4	4¼	4½	4¾
C-½ of one leg	2½	2½	2¾	3	3¼	3½

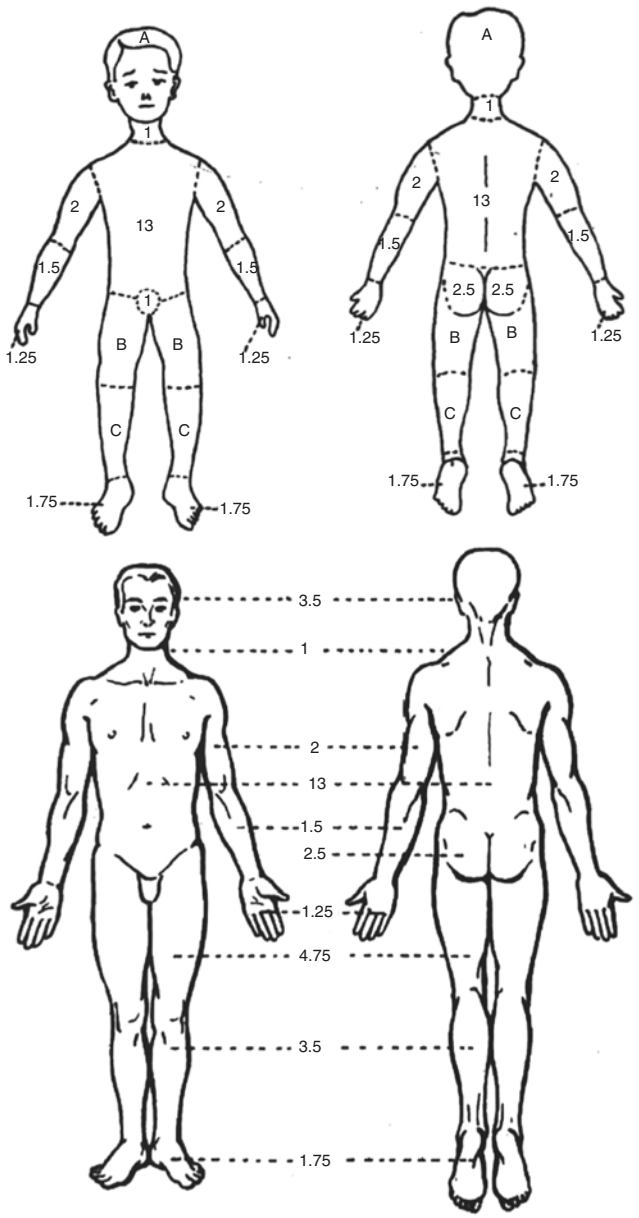

Fig. 5.2 Lound and Browder chart in use in Niguarda hospital

5.4 Pathophysiology of Wound Healing [2–5]

There are three different zones in the burned injury, the Coagulation Zone, where there is necrosis; the Ischaemic Zone, where perfusion is decreased, and the Hyperaemic Zone, where there is vasodilatation and inflammation in the outermost area (Figs. 5.3 and 5.4).

From a clinical point of view, the first 24 h are characterized by the *haemostasis phase*, which includes vasoconstriction, platelet activation, and release of growth factors by different cells, keratinocytes, and fibroblasts, which provide fibrin clot deposition as a provisional matrix, platelet activation and aggregation and release of PDGF, EGF, and TGFβ.

After 24 h, macrophages and neutrophils are recruited to the wound site inducing vasodilatation. The *inflammatory phase* [4] initiates with the release of cytokines, chemokines, including IL–1, IL-8, and TNF, growth factors, importantly, IGF, and VEGF, removing debris from the wound. P*roliferation* is the next phase in which granulation, angiogenesis, epithelialization occurs, and the conversion from fibroblasts to myofibroblasts is involved in the deposition of extracellular matrix. This final stage is the *remodelling phase,* where granulation tissue matures and the ECM is remodelled.

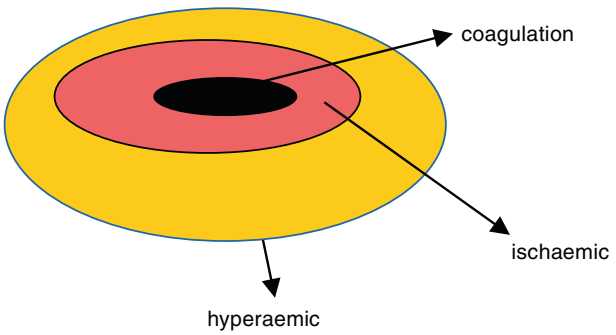

Fig. 5.3 Wound healing

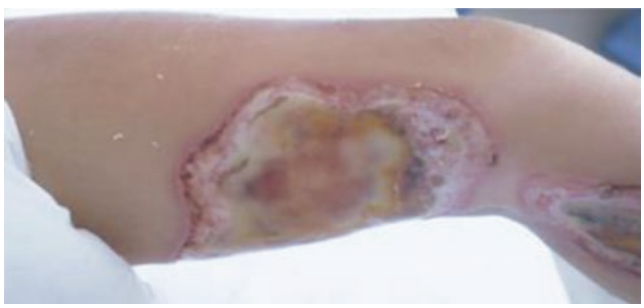

Fig. 5.4 Picture with coagulation, ischaemic, and hyperaemic area

5.5 Shock: Ebb and Flow Phase

Cytokines and inflammatory mediators have an effect both locally and systemically that may result in burn shock. This is defined as a lack of oxygen delivery to the tissue as required metabolically. Oedema of the tissue occurs due to fluid shift from the circulating plasma into the interstitial space, as proteins and plasma are sequestered to burned and non-burned tissue, most common when the burn injury TBSA is greater than 25%. Inflammatory mediators and stress hormones increase microvascular permeability, alter membrane permeability impact renal function, reduce cardiac contractility, and cause vasoconstriction. In a 3-h period following a 30% or greater TBSA burn injury, almost 50% of total plasma fluid may be lost from the vascular system. The resulting hypovolaemia causes a decrease in circulating blood volume, a decrease in venous return, arterial hypotension, hyperthermia, tachycardia, hyperventilation, and decreased urine output. Oedema in the burned and a non-burned wound increases with resuscitation. This distributive shock causes alterations in the perfusion of organs and tissue thus increasing the risk of infection as cellular and immunologic response is suppressed.

Disseminated intravascular coagulation can occur. This results in the deposition of fibrin clotting at the injury site and possibly resulting in thrombosis or a haemorrhage. Following this *ebb phase*, within 96 h, a hypermetabolic state is typically observed and can persist for up to 36 month post-trauma. Stress hormones increase blood pressure, insulin resistance, energy expenditure, organ function catabolism, protein loss, and muscle wasting.

5.6 Sepsis

Severe burn injuries result in immunosuppression with the damaged tissue being fertile ground for the growth of pathogens. Microorganisms can rapidly colonise the burn injury, as there is no physical barrier protecting the body. The avascular necrotic tissue inhibits the healing process that involves fibroblast; growth factor-induced endothelial cell proliferation by creating a breeding ground for bacterial colonization. This creates a biofilm that protects bacteria from the host immune system as well as antimicrobial agents or antibiotics. The American Burn Association Consensus Conference agreed on the definition for Sepsis and Infection in Burn Injury [6, 7], where Sepsis is a change in the burned patient that triggers the risk for infection. This is a presumptive diagnosis as antibiotics are usually administered and the cause of infection must be investigated. Despite the necessity to adhere to the discovery of the infection, the definition of sepsis is also age-dependent, where adjustments are necessary for children.

The trigger includes at least three of the following [8, 9]:

- Temperature > 39° or < 36, 5 °C.
- Progressive tachycardia >110 bpm; in children>2SD above age-specific.
- Progressive tachypnea >25 bpm non ventilated or > 12 L/min ventilated; in children>2SD above age-specific.
- Thrombocytopenia will not apply until 72 h from initial resuscitation; <100,000/mcl; in children>2SD above age-specific.
- Hyperglycaemia in the absence of pre-existing diabetes mellitus, plasma glucose >200 mg/dL; Insulin resistance (requirements >25% over 24 h).
- Inability to continue enteral feeding 24 h, residual >2× feeding rate or abdominal distension or diarrhoea >2500 mL/d for adults, residual 150 mL/h or diarrhoea >400 mL in children.
- In addition, infection is demonstrated by at least one of the following: positive culture for infection, pathological tissue source identified clinical response to antimicrobial agents.

Clinical state must be evaluated, minor vital signs, IV access, check serum lactate, and blood cultures before administering antibiotics. The initial resuscitation provides haemodynamic support which includes fluid resuscitation with crystalloid fluid, vasopressors if hypotension present to maintain MAP>65 mmHg. Then, start empiric therapy with broad-spectrum antibiotics and continue reassessment for fluids. Taking precautions to prevent and treat sepsis and MOFs include optimize resuscitation and haemodynamic status, prevent organ hypoperfusion, prevent intestinal barrier deterioration with enteral nutrition, performing escharectomy to take away all necrotic tissue.

5.7 Inhalation Injury

Inhalation *is suspected* when a burn injury occurs in a closed space. Signs include hoarseness, carbonaceous sputum, cyanide; *upper airway injury* due to oedema subsequent to heat; *lower respiratory system* due to chemical wheeze and dyspnoea, being indicators for inhalation injury. It is possible to identify three different situations: *systemic toxicity* due to inhalation of gas produced by combustion, such as carbon monoxide or hydrogen or micro-particle inhalation. The treatment is oxygen, in a semi-upright position. Endotracheal intubation or tracheostomy is sometimes suggested if airway patency is compromised, as oedema could have been in

progress for many hours. Chest radiograph does not exclude the diagnosis of inhalation. Prophylactic antibiotics and corticosteroids are not indicated for the treatment of smoke inhalation injury.

5.8 First-Line Treatment

Observation and removal of hazards and risks for operators are mandatory before treating the patient in pre-hospital. Triage depends on the resources and the team must follow the BLS rules for patient monitoring and treatment. If water is readily available, it should be poured directly into the burn area. Ice packs should never be used. During the transfer to the hospital the patient, after the burn injury site has been irrigated with water, should be lain on dry sheets to avoid further temperature loss. It is difficult in the trauma scenario or in the ambulance to calculate the extension of the burned area. The ABA with other International Societies for burn care [8, 9] have developed a formula for the infusion that patients need during transport to the burn centre (Table 5.4). Humidified oxygen should be given to all patients.

Table 5.4 Crystalloid resuscitation during transport using *Lactated Ringer Solution*

<5 years	125 mL LR/h
6–13 years	250 mL LR/h
≥14 years	500 mL LR/h

5.9 On Admittance

5.9.1 Primary Survey

In the emergency room evaluation starts with the primary survey: **A**irway management, **B**reathing and ventilation, **C**irculation and cardiac status, **D**isability, **E**xposure as in the general evaluation of trauma.

5.9.2 Secondary Survey

First, stabilise the patient for trauma and then for burns is mandatory. A specialist usually performs the evaluation of burn depth and size, based on TBSA. If the circulation of the extremities is compromised by circumferential or near circumferential eschar, the underlying tissues could be Ischaemic and needed escharotomy, decompresses cuts interesting only the superficial tissue over the fascia. Pulse oximetry, measuring oxyhaemoglobin saturation, may be helpful to perform escharotomy (Table 5.5). Escharotomy is also indicated for trunk or neck if ventilation or breathing is compromised. Fasciotomy is rarely performed as a first-line procedure in burn injury.

Adult patients with burns greater than 20%TBSA and paediatric patients having greater than 10% should be resuscitated with fluids (Table 5.6).

Table 5.5 Limits for escharotomy

O_2 saturation (%)	Compartmental pressure (mmHg)	Escharotomy
>95	<25	NO
90–95	24–40	Further investigation
<90	>40	YES

Table 5.6 Monitoring

TBSA > 20% or unstable patient	TBSA < 20%
Check for inhalation injury:	Check for inhalation injury:
Intubation, bronchoscopy, carboxyemoglobin evaluation	Intubation, bronchoscopy, carboxyemoglobin evaluation
Central venous catheter	Two accesses lines
Arterial line for continuing blood pressure monitoring	BP hourly
Invasive monitoring for unstable patient via arterial lines, SvO_2, PiCCO, …	
Enteral feeding	
Urinary catheter	Urinary catheter
Core temperature	Core temperature
Chest X-ray	Chest X-ray
Blood draw: Electrolytes, BUN, creatinine, blood counts, platelets, coagulation, lactates, albumin or total serum proteins	Blood draw: Electrolytes, BUN, creatinine, blood counts, platelets, coagulation, lactates, albumin or total serum proteins
Check other injuries	Check other injuries
Clean, check the need for escharotomy or fasciotomy	Clean, check the need for escharotomy or fasciotomy
Recalculate TBSA and fluid requirements	Recalculate TBSA and fluid requirement

5.10 Resuscitation from Burn Shock

Fluids resuscitation aims to support the patient in the first 24–48 h after the trauma [2, 3]. The first gold standard is the replacement of the fluid sequestered in the third space due to burn injury. The massive fluid shift occurs even if the total body water remains unchanged, but intracellular and interstitial volume of fluid increases at the expense of plasma and blood volume [10, 11]. Notably, the oedema process is amplified by fluid resuscitation. The initial resuscitation is carried out with crystalloids due to the proteins leakage from capillary after thermal injury. Lactated Ringer solution is the most popular resuscitation fluid utilized with a concentration of 130 mEq/L in sodium. The used formula for crystalloid resuscitation is the Parkland formula and the modified Brooke one (see Table 5.7). The aim of the Parkland formula is the replacement of fluid loss with crystalloids, but plasma volume is not maintained without proteins and massive oedema results. Protein replacement is necessary and there are different formulas: (1) proteins should not be infused in the first 24 h as they can produce massive water accumulation; (2) albumin should be added to salt water from the beginning to reduce water infusion; and (3) hypoproteinemia may increase tissue oedema, therefore, and proteins should be infused after 12 h from the trauma (Fig. 5.5).

Parkland formula does not accurately predict the fluid requirements in large burned patients that frequently exceed

Table 5.7 Fluid resuscitation

Formula	First 8 h	Second 16 h	Electrolyte	Colloids
Parkland 2–4 mL/%TBSA/kg	50% of total mL	50% of total mL	Ringer lactate	20% plasma volume circulation. During the second 24 h
Modified Brooke 2 mL/%TBSA/kg			Ringer lactate	0.3–0.5 cc/kg/%TBSA of albumin 5%. During the second 24 h
Demling				0.5–1 cc/kg/%TBSA of fresh frozen plasma during the first 24 h beginning after 8–10 h after burns

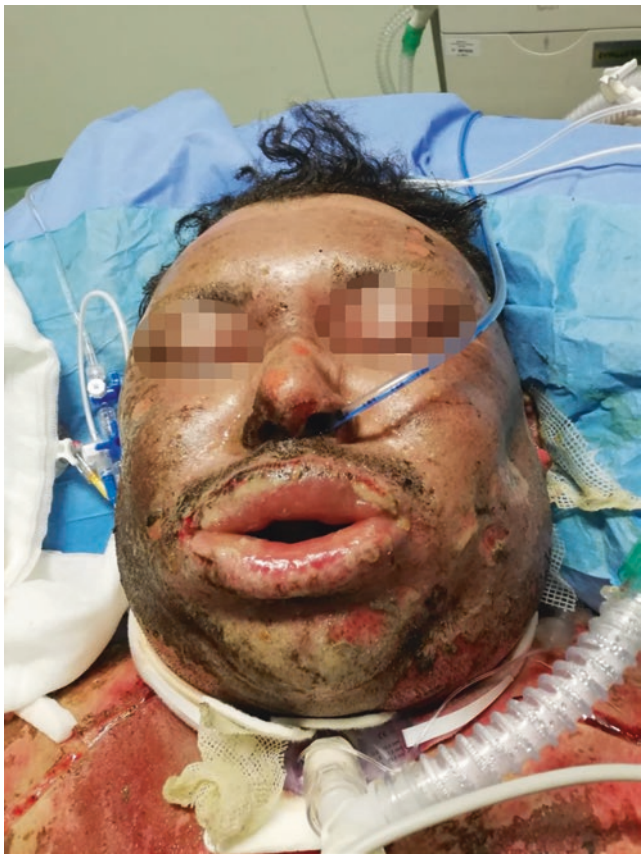

Fig. 5.5 Burned patient 24 h after trauma

the fluid volume predicted so, in 2000, Pruitt wrote about the "Fluid Creep" [11] to describe the increasing resuscitation volumes and urged the clinicians to "push the pendulum back". Over-resuscitation can produce abdominal and extremity compartment syndrome, pulmonary and cerebral oedema, acute pulmonary distress syndrome, and MOFs. Blumetti (2008) published a retrospective study that suggests the problem could be on parameters used to guide the resuscitation rather than calculated formula volumes. Resuscitation may require the use of colloids if the plasma volume is reduced and in patients with more than 40% TBSA burned. To avoid over and under resuscitation is important to define the minimum and maximum volume of crystalloid that can be infused and then after every 4 h check the point. Invasive haemodynamic monitoring is requested.

5.10.1 Standard Monitoring Criteria

- **Fluid balance**
- ***Vital signs***: **invasive BP, HR < 140/min, invasive monitoring if required**, **SpO_2, Internal temperature, urinary output**
  (30–50 mL/h or 0.3–0.5 mL/kg/h adult Patients)
- ***Laboratory***: Electrolytes, Ht, enzymes, Serum lactate, pH

5.11 Nutrition [5, 8, 9, 12]

In burn injury, there is an increase in the catabolic hormones epinephrine, cortisol, and glucagon in contrast with the insulin effects. Therefore, blood sugar increases, protein synthesis, and lipogenesis are inhibited. To prevent protein wasting, the diet must be rich in protein, about 2 g/kg/day in adults and 3 g/kg/day for paediatric patients. Among the amino acids with an important role in energy, delivery there is Glutamine. Notably, Glutamine is an important fuel for muscle, liver, wounds, and lymphocytes thus extra support of 0.2 g/kg/day could be important for large burns. The caloric needs in nutritional support are shown in Table 5.8. It is preferable to use enteral nutrition over parenteral as this is associated with a lower infectious risk. Furthermore, enteral feeding has a more effective role in gastrointestinal and mucosal barrier protection.

Table 5.8 Caloric formulas [8, 9]

Adults formulas	Kcal/day
Kcal/kg	35 Kcal/kg
Curreri	Age 16–59: 25(W) + 40(%TBSA)
	Age > 60: 20(W) + 65 (%TBSA)

In general, in order to avoid infusion of a high volume of nutritional feeding through a nose-gastric tube and meliorate residual feeding rate, high protein nutritional mixtures are indicated.

5.12 Wound Covering [4, 5, 8–10, 12]

Loss of epidermal barrier permits the evaporation of fluids, with microorganisms having unimpeded access to microcirculation due to systemic infection. Deep tissues become dry with secondary cell death and progression of wound depth. In the first-period post-burn, wounds are generally sterile or at the stage of superficial bacterial colonization, in the fourth–fifth day, bacterial involvement is evident predisposed by the avascular nature of the burn. Occlusive, humid, and heat preserving dressings are indicated. The partial-thickness burn has moderate-to-high exudate from wounds; thus, it is appropriate to use an absorbable dressing.

Cleansing with gentle washing with saline water is the most important component of burn wound cleansing in order to remove the non-adherent necrotic material. Subsequent cleansing of injures area are carried out by means of antiseptic. Chlorhexidine Gluconate in soapy solution at 0.5–1.0% is the most used product due to its broad spectrum of action against low toxicity and interaction with other antiseptics. Then, the coverage of the wound is performed with gauze impregnated with Vaseline. The dressing should be maintained approximately 48 h to prevent contamination, dryness, evaporation and negative mechanical effects. This conventional dressing, with non-adherent gauze or gelling contact mesh, has the advantage of being easily changed, according to the degree of soaking. Adsorbent dressing as gels and alginates are able to prevent the accumulation of fluids, which favour the process of bacterial proliferation.

Antimicrobial dressing should facilitate reepithelisation by providing a moist, clean environment. Silver-based dressing is a mainstream of treatment as the silver ions cutoff DNA replication and the electron transport chain. Nanocrystalline dressing improves anti-microbial activity. Despite these products being more costly, they are able to remain on the wound for several days, unlike the conventional dressing (Figs. 5.6 and 5.7).

Whereas, blister management requires the vesicles to be snipped open, its content evacuated, leaving the walls as a biological dressing [8].

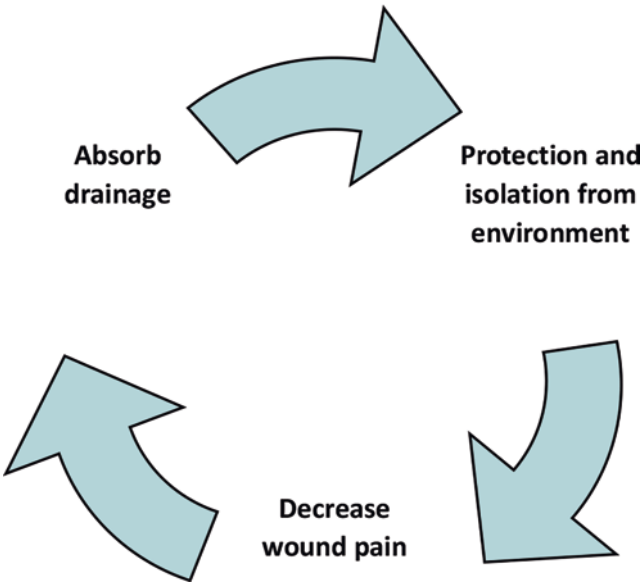

Fig. 5.6 Dressing purpose

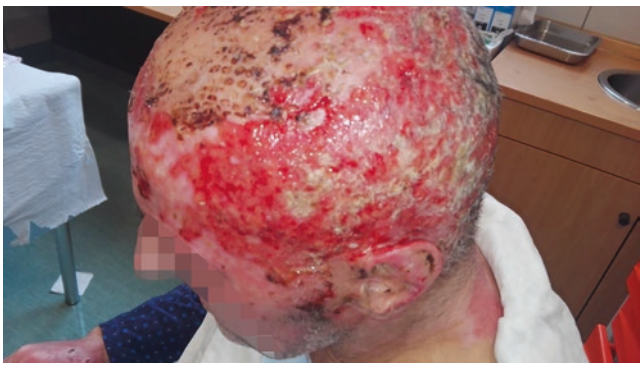

Fig. 5.7 Ghost graft and local infection with essudation

5.13 Surgical Management

In general, surgical technique for burn excision is defined by the timing: early in the first few days after burn to day 10; delayed after 10 days to 3 weeks after injury. Early excision [4, 5, 9, 12] and grafting reduces hospital stay, improves long-term outcome, reduces infectious complication and improves overall survival. The most important determinant of hypertrophic scar is delayed wound healing. In patients with insufficient autograph, skin substitutes provides temporary coverage. Tangential eschar excision is the standard technique, while fascia excision is indicated for deep burn or electrical injuries. Eschar excision is accompanied by significant bleeding which requires appropriate treatment. Once necrotic eschar has been removed, it is necessary to cover the painful area with dermal epidermis grafts. The first choice is represented by auto grafts, dermal epidermis grafts taken from non-injured areas, which can be placed on the cleansed base of the burn.

Enzymatic debridement [8] of burn wounds is a non-surgical conservative debridement based on application of bromeline. It is a safe and selective debridement, which helps to avoid the surgical complications related to the first surgical debridement. Consequently, the skin treated with bromeline can be grafted or treated with other medications or undergo a new escharectomy Fig. 5.8.

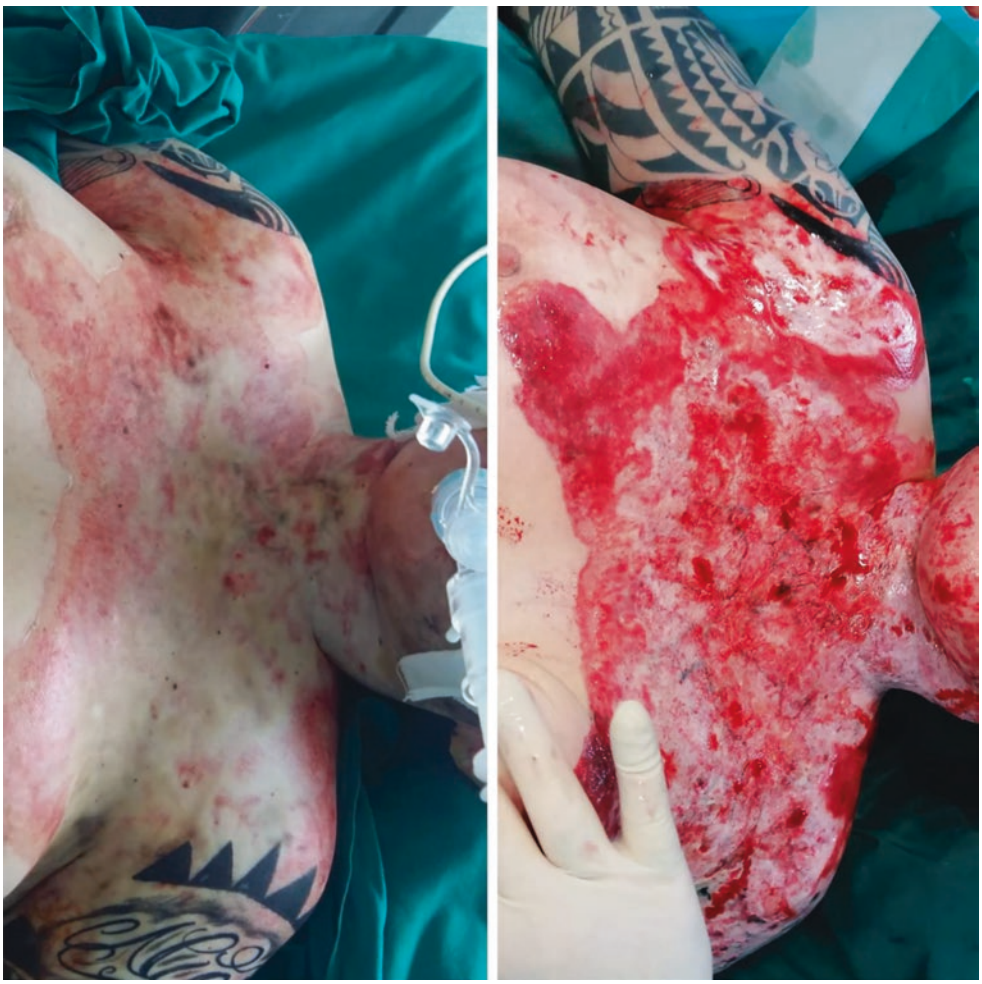

Fig. 5.8 Pre- and post-escharectomy with bromeline

5.14 Criteria for Transfer a Patient to a Burn Centre [8, 9]

- Patients requiring burn shock resuscitation
- Burns that involve head, hands, genitalia or major joints
- Deep partial thickness burns and full thickness burns in any age group
- Circumferential burns irrespective of age group
- Burns of any size with concomitant trauma or diseases, which might complicate treatment, prolong recovery, or affect mortality
- Burns with suspected in inhalation injury
- Any type of burn when treatment options are not clear
- Significant electrical burns
- Significant chemical burns

5.15 Clinical Scenario

5.15.1 Put the Actions in Order

Emergency call for an explosion in a habitation. The house owner, on entering his home, was thrown to the ground from an explosion. He got up and ran to salvage what he could from the fire. The firefighters arrived on the scene, they entered the house to find **a man unconscious lying on the ground**:

(A) The patient was undressed, washed with warm liquids and placed on dry sterile sheets.

(B) They intubated the patient on the spot and two venous infusion routes was inserted, and an infusion of Lactate Ringer started at 500 mL/h

(C) He was transported out of the flames and taken to a safe area
(D) Breathing was superficial, about 4 min
(E) The medical crew approached the patient. He was unresponsive
(F) Airway was obstructed by soot

5.15.2 Choose the Most Correct Answer

The Operations Centre was notified and the patient was referred to a Hospital with a Burns Centre. He reached the shock room in the ER Trauma Unit, where a heated station had been set up. The patient was evaluated by
(A) Anaesthesiologist, Plastic surgeon, Nurse
(B) Trauma Surgeon, Radiologist, Plastic Surgeon
(C) Plastic surgeon, Nurse
(D) Anaesthesiologist, Trauma Surgeon, Radiologist, Plastic Surgeon, Nurse

ACTIONS in the Emergency Room **ABC of trauma, laryngoscopy**, insertion of a **central venous catheter** and **arterial catheter**, **nasogastric tube, urinary catheter**, performed **biochemical samples** for major trauma, **chest, spine**, **pelvis X-ray, abdominal ultrasound** and **brain CT scan.**

5.15.2.1 Choose the Correct Answer

The evaluation by the plastic surgeon, diagnosed deep second-degree burn and third-degree burn with **45% TBSA**. There was no vaccination card, and an anti-tetanus immunoglobulin jab was given.

The Plastic Surgeon decided to apply the **Parkland formula**:

$$4\,\text{mL} \times 85\,\text{kg} \times 45\%\text{TBSA} = \textit{15,300 mL to be infused over 24 h}.$$

(A) The infusion started at 637 mL/h
(B) The infusion started at 956 mL/h
(C) The infusion started at 1021 mL/h

7650 mL for the first 8 h

The patient had already infused 500 mL in the pre-hospital phase, so there are 7650 mL for the first 8 h; an hour had passed, so the real volume was 7150 mL in the first 7 h. Lactate Ringer was set to the speed of 1021 mL/h.

The patient was gently washed with warm saline and medicated with fatty gauze and covering gauze. Hourly diuresis is monitored (range of 30/50 mL/h), electrolyte and blood lactated checks are scheduled every hour, enteral nutrition, and analgesic and sedative therapy start. Blood pressure, HR, cardiac monitoring hourly.

If diuresis was low than 30 mL hourly a fluid bolus is performed (250/500 mL), but in the second 16 h, a new check for fluid input must be done. The patient after 500 mL of bolus administered 3× consecutively, had a urine output of 50 mL hourly, blood lactated and red cells decreased.

Enteral nutrition was started at low speed, sedation and painkillers began.

5.15.3 Choose the Correct Solution

After 8, the patient had an infusion of 9150 mL, 1500 mL more than planned (Table 5.9).

In the second 16 h, 7650 mL was planned
(A) The infusion with ringer lactated continued at 478 mL/h
(B) The infusion was planned at 7650–1500 mL = 6150 mL: 16 = 384 mL/h
(C) Fresh Frozen Plasma about of 850 mL was planned with a reduction of the infusion controlled by urine output, blood lactate, red cell count

Table 5.9 Results over 24-h monitoring with fluid resuscitation in a burned patient being treated

Value	2 h post-burn	8 h post-burn	24 h post-burn
HR/min	125	115	110
TC °C	34.5	35.1	35.9
MAP mmHg	85	75	70
CI L/min/m^2	1.62	2.1	3.22
ITBVI mL/m^2	455	468	744
ELWI mL/kg	6.0	6.9	8.0
Blood lactate mmol/L	4.8	3.96	2.45
HCT%	58%	59%	49%
Urine output	80 mL	240 mL total	960 mL total
Warm fluid infused	1521 mL crystalloids	9150 mL crystalloids	16,000 mL crystalloids + 850 mL FFP

PiCCO pulse index contour cardiac output, *HR* heart rate, *MAP* medium arterial pressure, *CI* cardiac index, L/min/m^2, *ITBVI* intrathoracic blood volume index, mL/min, *ELWI* extravascular lung water volume index, mL/kg, *TC* internal body temperature measured with PiCCO

References

1. Yin S. Chemical and common burns in children. Clin Pediatr (Phila). 2017;56(5 Suppl):8s–12s.
2. Carlotto R, Greenhalgh D. Colloids in acute burn resuscitation. Crit Care Clin. 2016;32:507–23.
3. Demling RH. The burn edema process, current concepts. J Burn Care Reabil. 2005;26(3):207–27.
4. Jeschke MG, van Baar ME, et al. Burn injury. Nat Rev Dis Primers. 2020;6:11.
5. Keck M, Herndon DN. Pathophysiology of burns. Wien Med Wochenschr. 2009;159(13–14):327–36.
6. Mayo Clinic. Sepsis. Scottsdale, AZ: Mayo Foundation for Medical Education and Research; 2021.
7. Singer M, et al. The third international consensus definition for sepsis and septic shock (sepsis3). JAMA. 2016;315:801–10.
8. Herndon DN. Total burn care. 4th ed. Edinburgh: Elsevier Saunders; 2012.
9. ISBI Practice Guidelines Committee. ISBI practice guidelines for burn care. Burns. 2016;42:953–1021.
10. Rae L, Fidler P. The physiologic basis of burn shock and the need for aggressive fluid resuscitation. Crit Care Clin. 2016;32:491–505.
11. Saffle JI. The phenomenon of "fluid creep" in acute burn resuscitation. J Burn Care Res. 2007;28(3):382–95.
12. Lee KC, et al. History of burns: the past, present and the future. Burns Trauma. 2014;2(4):169–80.
13. Lachiewicz AM, Hauck CG, et al. Bacterial infections after burn injuries: impact of multi drug resistance. Clin Infect Dis. 2017;65:2130–6.