Jan KOLLER

BURNS

TEXTBOOK FOR STUDENTS OF GENERAL MEDICINE AND DENTISTRY

COMENIUS UNIVERSITY BRATISLAVA
BURNS
Textbook for students of general medicine and dentistry

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1 INTRODUCTION

Human skin seems to be simply external coverage of the human body. This statement is not completely true, as human skin, in addition to its protective function from the external environment, does have a range of other functions such as mechanical barrier, external immune barrier, participation in thermoregulation, excretion of sweat and sebum, sensory functions, and identification functions for the human being. Skin is the largest organ of the human body, as its average surface in adult persons is about 2 m² and its total mass is about 5 kg. An extensive burn injury affecting more than 20% of the body surface area, can lead to temporary, or permanent dysfunction or failure of the skin organ which could become a life threatening situation.

Every physician shall be capable to provide first aid and primary emergency care of the burn victims. Furthermore, every physician should know the basic issues of burns pathophysiology, burn patient assessment, diagnostics, classification of burns, principles of emergency treatment, primary burn wound care and indications for transfer of more severe cases to higher level hospital facilities and burn centres as well as indications and principles for outpatient treatment of minor burns.

Main goals of this textbook are to provide basic knowledge for understanding „the burn problem“ from pathophysiological points of view and to serve as a guide for diagnostics and therapeutic modalities, as well as treatment guidelines for care of the burn victims for students of medical faculties.
2 THE BURN PROBLEM

2.1 BURN DEFINITION

There is no unique and widely accepted definition of the burn injury. One of the most commonly used ones is as follows:

“Burn injury is the local response of a tissue, with or without systemic response, to an energy transfer from a physical (mechanical, thermal, electrical, radiation) or chemical source.”

This definition includes not only all the potential sources of the burn injuries, but also the extent of the body damage which does influence by major part the severity of the injury. As it will be explained later, it is a big difference between a minor burn causing just local damages at the injured body parts, and an extensive burn which can cause, in addition to damaging large amounts of skin and subcutaneous tissues, also dysfunctions of several organs and systems of the human body.

2.2 BURN AETIOLOGY

The majority of thermal injuries are caused by excessive heat such as hot water and steam (scalds), flame, explosion, contact of body parts with excessively hot surfaces (burns), but also by exposure to freezing temperatures (frostbites). Less often sources include exposure to electrical current, UV or ionizing irradiation, or corrosive chemical substances (corrosions), see Tab. 1.

Tab. 1 Burns aetiology

<table>
<thead>
<tr>
<th>PHYSICAL FACTORS</th>
<th>Burns (combustiones)</th>
<th>Cold injuries (congelationes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thermal energy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Electrical current</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(direct/alternating current)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Radiation</td>
<td>Ultraviolet (UV)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Infrared</td>
<td></td>
</tr>
<tr>
<td></td>
<td>X-ray</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ionizing</td>
<td></td>
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</table>

<table>
<thead>
<tr>
<th>CHEMICAL FACTORS</th>
<th></th>
</tr>
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<tbody>
<tr>
<td>Acids</td>
<td>(corrosions – corrosiones)</td>
</tr>
<tr>
<td>Alkalis</td>
<td></td>
</tr>
<tr>
<td>Other corrosive chemical substances</td>
<td></td>
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</table>
Burns are quite common injuries. According to statistics, there is expected that roughly 1% of the human population will each year suffer some kind of burn injury. Fortunately, one half of these injuries are so minor, that the victims do not visit a physician and they care about these injuries at home. This means that roughly $\frac{1}{2}$% of each country population will be treated yearly medically. The majority of these cases will be treated as outpatients, only 4% of them will be treated in local hospitals and 2% (the most severe cases) in specialized burns centres.

In our region (Slovakia) the majority of burn injuries occur at homes. The most dangerous place at home is the kitchen. Work – related injuries represent around 10% of all the burns. Pertaining the sources, the majority of burns are caused by hot liquids and contact with hot sources (minor injuries). Burns caused by flame are more severe, and represent around 30% of the hospitalized patients. All the other sources represent together less than 10% of all burns.

### 2.3 Pathophysiology

#### 2.3.1 Local Changes (Damages)

They occur at sites where the burn source does reach the body surface. They can affect mostly skin, but in some cases also underlying deeper tissues and/or structures (Tab. 3, p. 26). The surface area of damaged skin represents the extent of burns (Body Surface Area Burned, BSAB). In smaller burns the extent can be measured in centimetres (such as 5 x10 cm) or square centimetres (such as 155 cm$^2$). If the burn affects multiple body areas, the extent is calculated as percentage of body surface area of skin affected, i.e. total body surface area burned (TBSAB). In cases, where the TBSAB exceeds 15% of total body surface area (TBSA) in adults, or 10% TBSA in children, extensive local damage triggers a systemic body reaction which can lead to development of shock (burn shock) in emergency period and will continue in form of burn disease, or syndrome later.

There are 3 mechanisms by which thermal energy can cause tissue damage:

- Initial direct damage of cells and tissues by thermal energy,
- Cells and tissue damage caused by action of inflammatory mediators triggered by the initial thermal insult,
- Ischemic tissue damage caused by disturbances of local blood circulation initiated by the burn shock.
Initial direct damage of cells and tissues by thermal energy

Normal temperature of the human body assuring optimal function of cells, tissue and organs is around 37 °C. Increase of tissue temperature **above 45 °C** can cause **temporary disturbances** of cells and tissues functions. If the **temperature increase does not exceed 55 °C** this **damage** can be **reversible**. Increase of tissue temperature **above 55 °C** causes proteins denaturation and **irreversible damages** to cells and tissues (cell death, tissue necrosis). The extent of damages to cells and tissues depends on the amount of thermal energy which is delivered from the energy source to tissues and cells of the body. The **tissue damage is proportional directly to temperature of the heat source and duration (time) of energy transfer** to tissues. For example, during scalding by 80 °C hot water of body areas not covered by clothing, the heat source (hot water) does not accumulate at the body surface, but flows down and allows rapid cooling of the affected tissues. This way the exposure of body parts to heat source is quite short and the deeper layers of tissues are not damaged. The resulting burns are usually superficial. On opposite, by scalding of clothed body parts, or falls into hot water of the same temperature, due to longer exposure to heat caused by additional exposure time necessary for clothing removal, or escape from the hot tub, usually the burned areas become deeper (Tab. 3, p. 26).

Cells and tissue damage caused by action of inflammatory mediators triggered by the initial thermal insult

There are two types of this damage:

**Early damage** (first to third post burn day)

Nowadays there is quite clear, that relatively significant part of damage to tissues, which have not been damaged by the initial thermal insult, can be contributed to toxic inflammatory mediators activated by damages to tissues caused by burning. Even it is well known, that the process of healing is triggered by inflammation, excessive production of inflammation mediators, such as free radicals, or proteinases, can lead to damages of endothelial cells and skin cells not affected initially by the burning insult. It was demonstrated, that the inflammatory response initiated by burn can be responsible not only for generalized increase of capillary permeability, but for early damages of healthy tissues as well. In cases, when the inflammatory process will become excessive, and will eventually get out of control, it can cause also conversion of initially superficial burns to deep ones. The knowledge of these mechanisms can draw attention of physicians to better care of both the patients and their wounds in order to eliminate the risk of excessive inflammatory reaction post injury. This
would include early analgosedation of the patients, early administration of fluid substitution therapy and elimination of painful stimuli by early coverage of open wounds.

Delayed damage (later than 3 days post burn)

It follows the initial thermal damage to cells and tissues in a couple of days and is initiated by continuing systemic inflammatory response in the body accelerated locally by necrotic cells and tissues, increasing bacterial colonization of the burn wound, mechanical forces and/or application of more aggressive topical antibacterial agents (mostly disinfectants). Neutrophil leukocytes which are always present in wound exudate as first line cellular immunity cells can during their breakdown release great range of various proteolytic enzymes and oxygen free radicals which could cause damage even to healthy cells and tissues. If the inflammation continues locally, concentration of metalloproteinase increases gradually in the wound area. Their presence and increasing activities was demonstrated both in superficial partial thickness burns and in deeper dermal layers, which can result in deactivation of growth factors produced by undamaged cells. This can lead to deceleration of the healing process and increased synthesis of collagen which will result in increased scar formation. This sequence of events can be influenced by part by early removal of necrotic tissues, improved drainage of exsudates from the wound, burn wound infection control and, particularly, by temporary wound coverage and/or permanent wound closure.

Ischemic damage of tissues caused by disturbances of blood supply at the burn site

Compromise of local blood flow at the burn site is caused usually by unstable circulation in the shock period with subsequent redistribution of blood flow in the body with preference to most important vital organs such as brain and myocardium and reduction in other organs and systems (see also Tab. 2, p. 13) Second most important factor compromising local circulation is excessive formation of interstitial tissue oedema in the burned area and its vicinity. Formation of microtrombi in capillaries in the zone of stasis can cause conversion of initially more superficial burns to deep ones. Preventive measures include early initiation of substitution therapy of the burn shock and efforts to avoid administration of excessive amounts of intravenous fluids which would cause fluid overload and increased oedema not only around burned tissues, but also in non-burned body regions. Particularly in abdominal cavity area the fluid overload can cause abdominal compartment syndrome, which could become a life threatening situation if it is not recognized and treated early.
2.3.2 **Burn Pathology**

Noxious insults from the burn sources cause damage or even destruction of cells and tissues in different layers of skin and/or deeper tissue layers. Depending on the intensity of the burn sources the damages caused to cells and tissues can be reversible, or irreversible with formation of cells and tissue necrosis. The severity of subsequent pathophysiological events depends on the severity of cells and tissue damages at the site of injury. The symptoms of local damages can vary from mild and/or moderate changes such as painful redness (erythema) which does not disappear with time, accompanied by swelling (oedema), through epidermal necrosis and formation of blisters, to most severe changes as necrosis of deeper dermal layers, or necrosis spreading through full skin thickness, or into deeper layers of subcutaneous structures.

**Burn depth**

Severity of local tissue changes serves as a base for burn depth classification (Tab. 3, p. 26; Fig. 1, p. 10). There are 3 (in fact 4) grades of burn depth classification, according to involvement of particular layers of skin and subcutaneous structures:

- 1\(^{\text{st}}\) degree (grade) burns include necrosis of epidermis only (Fig. 1a),
- 2\(^{\text{nd}}\) degree burns include necrosis of epidermis and superficial (2\(^{\text{nd}}\) a), or also deeper parts of the dermis (2\(^{\text{nd}}\) b; Fig. 1a, 1b, 1c),
- 3\(^{\text{rd}}\) degree burns include full thickness necrosis of skin and eventually also progression of necrotic changes to deeper laying tissues and structures (Fig. 1c, 1d, 1e).

The **burn wound is three dimensional.** According to Jackson 3 zones of the burn wound can be recognized (Fig. 2, p. 11):

- central zone of tissue necrosis,
- surrounding zone of capillary stasis,
- peripheral zone of hyperaemia.

The central necrotic zone is formed by irreversibly damaged tissues. Tissues in the capillary stasis zone can undergo necrotic changes, if the capillary stasis turns into ischemia (due to compromise of the capillary blood flow). On the other side, if the capillary circulation in the stasis zone would recover, the tissue damage could be reversible. The peripheral zone of hyperaemia is caused by local inflammatory reaction of surrounding tissues triggered by mediators released from blood cells and cells from the central zones of necrotic tissues and capillary stasis.
Fig. 1 Examples of burns according to their depth

- **I\(^{st}\) degree**: red, no blisters
- **II\(^{nd}\) a. degree**:
  - thin epidermal blisters
  - positive capillary refill
- **II\(^{nd}\) b. degree**:
  - dry dermis and no epidermis
  - no capillary refill
- **II\(^{nd}\) b. and III\(^{rd}\) degree mixed**:
  - partial thickness skin loss (whitish and reddish areas)
  - full thickness loss (dark areas)
- **III\(^{rd}\) degree**: hand & wrist
- **III\(^{rd}\) degree**: thighs
The burn depth is decisive for assessment of burn wounds healing options as well. Both at initial burn evaluation, and from healing time point of view, there are 2 burn types:

- **superficial burns** include 1\(^{st}\) and 2\(^{nd}\) a. degree. The healing time of those burns is usually less than 14 days and they usually heal without any scars,

- **deep burns** include 2\(^{nd}\) b. degree (deep partial thickness) and 3\(^{rd}\) degree (full thickness). 2\(^{nd}\) b. grade burns will heal usually within more than 2 weeks and up to 8 weeks, or more time frame. 3\(^{rd}\) degree burns, if their extent is small, can heal within several weeks, or even months with appropriate treatment methods. In more extensive 3\(^{rd}\) grade burns usually surgical intervention including necrotic tissues excision followed by skin grafting is necessary for successful wound healing. Deep burns usually leave permanent sequelae – scars.

### 2.3.3 Extent of Burns

For **burn severity assessment**, in addition to burn depth, the **extent of burns** is of utmost importance. The TBSAB in extensive burns is calculated according to **rule of nines** (according to Wallace-Pulaski, for adult patients only), by **burn charts** (e.g. Lund-Browder charts for both adults and children, (Fig. 4 and 5), or by **rule of the patient's hand** (1\%, Fig. 3).
Fig. 3 Rapid assessment of the extent of burns

a) Rule of the palm   b) Rule of 9 (Wallace-Pulaski)

2.4 SYSTEMIC CHANGES

Extensive tissue damage along with massive painful stimuli trigger pathophysiological mechanisms of systemic response to severe burn injury. Neurohumoral pathways are triggered first in a similar way as in other kinds of extensive injuries. In addition to other kinds of injuries, toxic products originating in devitalized tissues get into the blood stream, and are distributed to all body areas. They can by major part contribute to stimulation of target cells in different tissues, organs and systems, which results in excretion of a range of mediators, cytokines and growth factors which trigger successive systemic reactions resulting in changes in the target organs and systems. Most significant consequences of these changes are leading to generalized increase of capillary permeability causing massive losses of fluids and electrolytes from systemic circulation into interstitial space and resulting in significant decrease of the circulatory volume accompanied by initiation of systemic inflammatory response syndrome (SIRS). In addition to rapid and massive transfer of body fluids between particular body compartments extensive release of a great variety of inflammatory and other mediators will cause significant disturbances of most of the organs and systems. One of the
mostly affected systems is the cardiovascular system what results in rapid development of burn shock.

**Tab. 2 Circulatory volume redistribution during burn shock**

<table>
<thead>
<tr>
<th>VOLUME DISTRIBUTION</th>
<th>physiologic</th>
<th>redistribution during shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO</td>
<td>5.4 l/min. (100%)</td>
<td>CO = 2.7 l/min (50%)</td>
</tr>
<tr>
<td>ORGAN</td>
<td></td>
<td></td>
</tr>
<tr>
<td>brain</td>
<td>1.3 l</td>
<td>1.3 l (100%)</td>
</tr>
<tr>
<td>kidneys</td>
<td>1.5 l</td>
<td>150 ml (10%)</td>
</tr>
<tr>
<td>liver + GIT</td>
<td>1.5 l</td>
<td>590 ml (40%)</td>
</tr>
<tr>
<td>muscles</td>
<td>600 ml</td>
<td>300 ml (50%)</td>
</tr>
<tr>
<td>skin</td>
<td>200 ml</td>
<td>100 ml (50%)</td>
</tr>
<tr>
<td>heart</td>
<td>60 ml</td>
<td>60 ml (100%)</td>
</tr>
<tr>
<td>others</td>
<td>240 ml</td>
<td>200 ml (83%)</td>
</tr>
</tbody>
</table>

Consequences of this complex and rapid sequence of events are as follows:

- Rapid decrease of intravascular volume,
- Decrease of heart minute volume,
- Generalized increase of peripheral vascular resistance triggered for compensation of intravascular volume decrease,
- Increase of pulmonary vascular resistance,
- Redistribution of blood flow between organs and systems (Tab. 2, p. 13),
- Ischemic damage to selected organs and systems.

Without appropriate, rapid and aggressive fluid replacement therapy the changes mentioned above changes can gradually lead to development of multiple organ and systems dysfunction (MOSD) which, if not treated early an properly can progress into multiple organs and systems failure (MOSF) - a life threatening situation with very high mortality rate.

Even by rapid and correct fluid replacement therapy a variety of problems and complications can occur. Massive fluid load (particularly by substitution of crystalloids) can increase generalized oedema formation. The oedema itself is not a severe complication, but it can cause disturbances of tissue capillary blood flow, decrease of oxygen diffusion rate to tissues and cells thus causing progressive tissue and cell hypoxia which can result in temporary or permanent damage resulting in tissue and cell death. Hypoxia along with disturbances of microcirculation can predispose to development of wounded area infection.
Clinically, these events will result in decrease of circulatory volume together with development of severe shock (burn shock) with all its deleterious consequences.

2.4.1 **BURN SHOCK**

Burn shock is caused by **massive continuous leak of fluids from circulation into interstitial space due to rapid pathological increase of capillary permeability. The extent of fluid losses** from circulation to third space in the body **depends proportionally on the burn extent.** In very extensive burns (exceeding 50% TBSA) the circulatory volume can decrease by 50% within 30 minutes post burn without any clinical symptoms of fluid loss from the body.

Due to massive stress reaction resulting in increased production of suprarenal medullar hormones and redistribution of blood flow for body organs and systems (see Tab. 2, p. 13), the clinical symptoms of massive hypovolaemia can be masked initially (for example, instead of initial hypotension, normotension, or even mild hypertension can be present).

For these reasons the physicians shall be very cautious, as the clinical signs of the burn shock can be hidden initially and different from those of other kinds of post traumatic shock states.

Generalized increase of capillary permeability post burn usually persists from 24 to 72 hours post injury and so far there are no means (drugs etc.) which could influence this fact. The massive losses of fluids from the circulation can be compensated by massive intravenous fluid replacement (substitution) therapy only during the entire period of continuing capillary leak. Approaches to substitution therapy will be described later (see p. 29).

2.4.2 **BURN DISEASE (BURN SYNDROME)**

Clinical syndromology of systemic responses to massive burn injury is called burn disease (burn syndrome). There are 3 periods of the disease:

1. **Emergency (shock) period**

The shock period starts at the moment of burn injury in extensive burns. The main symptoms in this period include symptoms of shock and of dysfunctions of different organs/systems of the body. The duration of the period is identical with duration of the burn shock. It varies usually between 24 and 72 hours post burn.

Restoration of pathological capillary permeability and stabilization of organ/systems functions will mean the end of this period and start of the second – acute period.
II. Acute period (see also p. 55)

The acute period starts as soon as the shock symptoms resolve and the patient’s general condition and circulation are stabilized. Its duration depends on the time necessary for closing the majority of patient’s open burn wounds. Although the period is called acute, its duration can exceed four to six weeks, which are characteristic for other kinds of acute diseases.

The acute period is characterized by continuing presentation of signs, symptoms and consequences of extensive open burn wound and by permanent threat of systemic complications originating in the large wound surface area.

In this period the majority of surgical procedures such as necrotic tissue excisions and wound coverage/grafting procedures are provided. In most of the cases intensive or resuscitative care of the patient is performed along with regular meticulous intensive wound care procedures (dressing changes, conservative wound debridement, application of topical medications. During the entire acute period it is of utmost importance to provide all the available measures for prevention of infection and cross-contamination of the burn wounds. Intensive care shall provide particularly monitoring along with maintenance of all organs and systems functions, particularly maintenance of adequate cardiovascular, respiratory, renal, gastrointestinal, metabolic functions and maintenance of adequate tissue oxygenation and homeostasis.

Patients with extensive burns are in severe catabolic state and they require maintenance of their nutrition and energy balance as well preferably by natural ways (i.e. oral or enteral nutrition). Intensive nursing care shall provide proper patient positioning, prevention of pressure sores, splinting of extremities, respiratory therapy and physical therapy. Rehabilitation should be initiated as soon as possible. The duration of this period can be usually in a range from several weeks to several months. The acute period will finish as soon as the patient is stabilized and does not require further organs and/or systems support (such as artificial ventilation, circulatory support by vasopressors, permanent monitoring of vital signs and functions, continuous intravenous fluids administration etc.). The burn wound shall be healed, or covered by permanent coverage (autologous skin) to such an extent, that the residual area of the open wound would be less than 10% to 15% of the body surface area. The end of the acute period will be the start of the last – rehabilitation and restitution period.

III. Restitution and rehabilitation period (see p. 89)

Following closure of the major part of patient’s wound, it is necessary to continue by active rehabilitation procedures, realimentation, and by care of both the healed wounds (scars) and still open wounds. The patient is taught to re-gain his lost skills and prepared for
dismission from the hospital care and for return back home to his/her family and all other activities. In this period it is very important to provide psychological support to the patient and his family as well. In cooperation with his/her family the patient´s return to work and his previous social activities should be also prepared. The care of his/her residual wounds and scars will be provided both at home and at outpatient departments (ambulatory care).
3 FIRST AID AND EMERGENCY TREATMENT AT THE ACCIDENT SITE

3.1 FIRST AID

Appropriate first aid is one of the most important issues immediately after the accident occurred. Main goals of the first aid include stopping the burning process, to cool immediately the burned body areas to evacuate the victims from the accident scene to a safer place, to watch the vital signs, to cover the open wounds, and to call for emergency services.

Non-medical first aid

If the victim is alone and did not lose consciousness, he/she needs to take immediate actions mentioned above. Otherwise, other persons present at the accident scene do usually participate at provision of the first aid in the same order of actions, and they call at the same time for emergency medical services. In case, where vital functions are endangered, appropriate resuscitative measures should be taken by the persons present until the ambulance will arrive.

Medical first aid

It depends on the emergency services organization, if the ambulances are equipped by personnel including paramedics only, or a medical doctor as well. Main goals of the medical first aid provision include initial assessment of the injury severity, assessment and maintenance of the vital functions (A-Airway, B-Breathing, C-Circulation), pain management, initiation of intravenous fluid replacement therapy, provisional wound coverage by sterile blankets, prevention of heat loss, and rapid transport to the nearest hospital capable to provide appropriate primary patient management. In cases, when the burn occurred in enclosed space, or suspicion of inhalation injury is present, orotracheal intubation with subsequent artificial ventilation is necessary (if medical doctor is member of the rescue team).

Rationale of immediate cooling of burns

Excessive thermal energy from the heat source is accumulating in human tissues directly depending on the source temperature and time of exposure. When the temperature in human tissues exceeds 45 °C (critical temperature), wide range of disturbances and damages to tissue
components occur. The tissue damage could be reversible, if the temperature does not exceed the temperature of protein molecules denaturation, which is from 55 to 57 °C. Temperatures exceeding this range cause cell death leading to complete tissue death (necrosis).

Thermal energy accumulated by tissues causes that temperatures exceeding the critical ones persist in tissues for longer time period thus increasing the extent and depth of tissue damage. This sequence of deleterious events can be reverted by immediate rapid cooling of the burned areas which can remove the excessive thermal energy accumulated in tissues and speed up the decrease of tissue temperature below the critical level. This is the mechanism by which immediate rapid cooling of the burned areas can substantially decrease the extent and depth of tissue damage. The best method of cooling is the use of ambient temperature drinking water (usually from the tap). Drinking water is clean enough for preventing secondary contamination of the burned areas. If the burned body parts are covered by dressing, then cooling shall be done first and only after cooling shall the dressing be removed. Moreover, water is an excellent accumulator of thermal energy and is capable to cool rapidly the overheated tissues. Cooling can also alleviate pain from the burned body areas.

In small burns cooling can be provided longer time to improve the comfort of the patients. One should be cautious with the use of cooling in extensive burns, as prolonged cooling can cause decrease in core body temperature, which shall be avoided. Anyway, even in extensive burns rapid cooling is effective, but should be stopped as soon as the temperature of burned body parts returns to normal range. Ice should be not use for cooling of burns, as extended cooling by ice can cause frostbites. There exist also commercially available systems for cooling of the burned body parts such as Water Jel®, or Burn Wrap® etc.

3.2 BASIC PRINCIPLES OF FIRST AID PROVISION IN BURNS

First aid principles in burn injuries are by part similar to the ones in other kinds of injuries. However, circumstances accompanying them have several differences such as:

- **Evacuation of the burn victim** from dangerous environment should be done first. One needs to keep in mind the security of the rescue personnel (rescuers) as well, in order not to put their lives in danger.

- **Extinguishing of burning clothing, cooling of the body parts burned**
  In case the clothing of the victim is burning, and there are no fire fighting or provisional devices (blankets) available at the scene, the rescuers should aim that the
victim would fall down to the ground and roll over several times to extinguish the flames. It should be kept in mind, that the victim must not run, as this would accelerate the burning process. Very useful is to use water for both extinguishing the flames and for rapid cooling of the burned body areas.

- Escape from the burning spaces with simultaneous protection of the airways
  The second major threat for the fire victims during escape from burning spaces is inhalation of toxic gases and smoke particles which can result in inhalation injury with subsequent respiratory dysfunction/failure and, eventually, in intoxication by toxic gases and substances produced by burning of different materials as well. Persons during escape should move as close to the ground as possible, as the less toxic and coldest air is nearest to the ground. Most of the toxic gases, such as carbon monoxide or hydrogen cyanide, are lighter than air and therefore concentrate in the upper enclosed space areas. If possible, all the synthetic material clothes should be removed and the persons should protect themselves by wrapping in moist cotton or wool blankets. The respiratory areas shall be protected by moist towels, although they can protect just from inhalation of smoke particles, they are not able to protect against inhalation of toxic gases.

- Assessment of vital signs of the victim
  Following flames extinction and evacuation from the accident scene the next step is evaluation of the victim’s vital signs – consciousness, breathing, cardiac function. In case the victim is not unconscious, it is important to cool the burned body surface areas in order to remove the accumulated heat energy from the affected tissues and to cover thereafter the burned areas by provisional cover (clean blanket) or some kind of prefabricated dressing. In case the victim is unconscious and without spontaneous breathing, or cardiac function, immediate start of adequate resuscitation procedures is of utmost importance. At the same time emergency service shall be notified describing the location of the accident, number of victims and circumstances of the accident.

- Electrical burns
  In electrical injuries it is of utmost importance to take care about switching off the current, otherwise the rescuers are endangered and shall not touch the victim unless this is done.

- Chemical burns – corrosions
  In chemical burns the most important issue is splashing, rinsing or showering the affected areas by large amounts of water in order to reduce the concentration of the
chemicals at the affected sites. Using of neutralizing substances (such as acids, if the caustic agent is alcalic and vice versa) is not recommended, as the chemical reaction during neutralizing is exothermic generating various amounts of added heat. If the caustic chemical was concentrated, then rinsing with water should be prolonged (20 minutes and more).

- **Wound coverage and prevention of hypothermia**

Affected open wound areas should be protected from the outer environment (risk of contamination – infection) by provisional coverage or prefabricated dressings from first aid kits. There should be taken preventive measures for rapid heat loss of the victims by use of protective shields or warming blankets or eternal heat sources.

- **Preliminary rapid assessment of burn extent and depth at the accident scene**

Rapid burn extent evaluation is provided using the rule of palm (opened patient’s hand) which represents 1% of the body surface (Fig. 3a, p. 12), or by the rule of 9 (Fig. 3b, p. 12) according to Wallace and Pulaski. Rapid burn depth assessment is done according to Tab. 3, p. 26.

### 3.3 INITIAL BURN SEVERITY ASSESSMENT AT THE ACCIDENT SITE

At the accident scene a very rapid preliminary burn severity assessment should be provided by emergency physician, as according to this assessment, different transportation and therapeutic modalities should be thereafter decided upon. The victims should be categorized preliminarily into two groups as follows:

**I. Minor burns**

**Extent:**
- 15% of the Total Body Surface area (TBSA) and less in adults,
- 7-10% TBSA and less in children, according to their age.

**Depth:**
- superficial burns (degree I\textsuperscript{st} and II\textsuperscript{nd} a., i.e. epidermal and superficial dermal burns),
- deep burns (degree II\textsuperscript{nd} b. and III\textsuperscript{rd}) 2% TBSA and less in adults and 1% TBSA and less in children.

**No other severity factors** (see also Tab. 4, p. 27) present:
- critical age groups,
- co-morbidities,
- burns involving functional areas (face, hands, feet, genitalia),
- concommitant injuries.
II. Extensive burns

Extent: - more than 15% TBSA in adults,
- more than 7-10% TBSA in children.

Depth: - deep burns (degree II\textsuperscript{nd} and III\textsuperscript{rd} i.e. deep dermal and full thickness skin loss) exceeding 2% TBSA in adults and 1% of TBSA in children.

Additional severity factors present (Tab. 4, p. 27):
- critical age groups,
- co-morbidities,
- burns involving functional areas (face, hands, feet, genitalia),
- concomitant injuries,
- electrical injuries, severe corrosions.

3.4 EMERGENCY CARE AND PROCEDURES

Initial diagnosis and decision taking provided by emergency physician at the accident site

The most important issue in diagnosis and decision taking is to decide, if the burn falls into a category of „minor burns“ or „extensive burns“ (Tab. 4, p. 27).

I. Minor burns

Priorities include pain management and emergency wound care at the accident site followed by transportation to specialized physicians (surgeons, plastic surgeons, traumatologists) who will provide primary wound care and subsequent outpatient treatment procedures, mainly dressing changes.

II. Extensive burns

In all extensive burns priorities include vital signs evaluation and, if indicated, provision of resuscitation procedures. As all the extensive burn victims are threatened by rapid development of burn shock, preventive measures include the following:

- insertion of secure large bore i.v. cannula,
- initial intravenous administration of pain medications and all other medications,
- in case of signs of inhalation injury endotracheal intubation and artificial ventilation should be provided,
- initiation of intravenous fluid replacement therapy by Ringer lactate solution,
- wound coverage by sterile dressing,
- prevention of massive heat loss of the patient,
- depending on the burn severity primary transportation of the patient to the nearest local hospital, or, following agreement, transportation of the victim to the nearest burn centre by road or air ambulance,
- continuation of fluid replacement therapy during transportation is mandatory.

3.5 SUMMARY OF FIRST AID EMERGENCY MANAGEMENT AT THE ACCIDENT SITE AND PRIMARY TRANSPORTATION

The impact of burn injury to a patient can range either from just a minor injury to one or several parts of his/her body (i.e. “small burn”) which will require outpatient care only and will heal within several days or up to 2 weeks. Or, there can be a major injury (i.e. “extensive burn”) with extensive damage to larger body surface areas accompanied by systemic changes, necessity of hospital treatment, threat of complications, permanent sequellae, or even death, with a major impact not only on the patient itself but on his family as well.

Main tasks of physicians who will treat such patients shall include:

- correct patient assessment, diagnosis and emergency care of the patient, primary care of the burn wound,
- provision of therapeutic interventions such as life-saving procedures, pain management, fluid replacement therapy, initiation and continuation of intensive care, emergency wound care,
- decision of subsequent treatment measures (like admission to local hospital or transfer to burn centre) in cases of major burns,
- provision of wound care and patient instructions of subsequent outpatient care in small burns.

In order of physician’s ability to provide correct assessments, diagnostics and decisions, it is necessary that he/she will be familiar with the principles of pathophysiology, diagnostic and therapeutic modalities of the burn injuries.
4 PRIMARY CARE AT HEALTH CARE ESTABLISHMENTS

It is usually provided by hospital emergency departments. In hospitals without emergency departments it should be provided by surgical and/or traumatology departments with close cooperation of anaesthesia and intensive care departments. Emergency care of extensively burned victims should be provided in clean, adequately heated environment and by aseptic approach, preferably at operation theatres. During all the diagnostic and therapeutic procedures all the efforts should aim to minimize pain (analgesedation, general anaesthesia) and secondary contamination of extensive burn wounds by aseptic techniques. At the same time procedures for advanced burn life support should be provided as well. The burn care team is composed of surgeons, anaesthesia and intensive care physicians, scrub nurses and auxiliary personnel. Consultants from other specialties (paediatricians, ophthalmologists, ENT physicians, neurologists) can participate, where appropriate.

Basic principles of primary burn care are as follows
(Ranking of particular steps is according to importance of the procedures):

1. Evaluation, support and maintenance of basic vital functions,
2. Detailed physical examination of the patient,
3. Evaluation of burn severity (see also p. 27, Tab. 4; steps 4-12 in extensive burns only),
4. Insertion of large bore intravenous line,
5. Pain management,
6. Fluid replacement therapy,
7. Ventilation and oxygenation of the patient,
8. Thermal comfort of the patient,
9. Urine output measurement,
10. Blood, urine, bacteriology, and other samples for laboratory investigations,
11. Primary surgical management of extensive burn wounds,
12. Prevention of gastric/duodenal stress ulcers,
13. Tetanus prophylaxis,
14. Other necessary procedures in extensive burns,
15. Diagnosis,
16. Decision about subsequent care of the patient,
17. Transportation of burn victims.
4.1 Evaluation, Support and Maintenance of Basic Vital Functions

Evaluation is provided by same methods as in other kinds of severe injuries (Airway, Breathing, Circulation – ABC). In case of any compromise of basic vital functions resuscitation procedures shall be initiated (or continued) until the patient will become stabilized.

For evaluation and scoring of consciousness, the most frequently used system is the Glasgow Coma Scale (GCS). Continuous monitoring of basic vital signs and functions shall be provided starting from patient admission and through all diagnostic and therapeutic procedures. In patients with respiratory system dysfunction/failure ventilatory support is mandatory.

Basic monitoring of vital functions includes (see also Tab. 6, p. 36):

- heart rate (ECG),
- pulse oximetry (pulse rate and wave, capillary blood O₂ saturation,
- respiratory rate,
- temperature,
- blood pressure – non-invasive.

Extended monitoring in more severe cases includes additionally (Tab. 7):

- blood pressure – invasive by arterial line,
- central venous pressure – invasive by central venous line,
- additional cardiovascular functions: - pulmonary artery wedge pressure,
- hemodynamic calculations,
- respiration (in artificially ventilated patients): - tidal volume (TV),
- minute volume (MV),
- peak inspiratory pressure,
- inspiratory O₂ fraction (FIO₂).

All the parameters obtained by continuous monitoring shall be recorded in regular intervals in written form in patient record files. The interval is usually one hour. Critical changes in the parameters shall be recorded immediately after they occur.
4.2 DETAILED PHYSICAL EXAMINATION OF THE PATIENT

Physical examination of the patient at admission is mandatory. In areas of the body under examination, all the clothing shall be removed during examination and all the non-physiologic findings shall be recorded. This applies for all the natural orifices of the body such as are the oral cavity, nasal cavity, external ears, eyes, perigenital and perianal areas as well.

For recording of burned skin areas specially designed burn charts are used. The most commonly used burn charts are those suggested by Lund and Browder separately for adults and for children. These charts contain also tables for BSAB calculations (Fig. 4 and 5).

Rapid check of all the organs/systems shall be provided as well.

4.3 EVALUATION OF BURN SEVERITY

For evaluation of burn severity, see also Tab. 5.

There are two basic criteria and 5 other criteria for burn severity evaluation.

The basic criteria are:

- burn extent,
- burn depth.

Burn extent

It is calculated as percentage of the body surface area burned (see also p. 15).

Burn depth

It is diagnosed according to the Table 3 (See also p. 8). For evaluation, diagnostic and prognostic issues there are two basic burn categories:

- **Superficial burns** include degree I\(^{st}\) and II\(^{nd}\) a. (superficial partial thickness skin loss) which are capable of spontaneous healing without any scars, if managed properly.

- **Deep burns** include II\(^{nd}\) b. (deep partial thickness loss) where healing is prolonged and sometimes conversion to full thickness loss can occur. In III\(^{rd}\) degree burns (full thickness skin loss and deeper) healing can occur in small burns only by epithelialisation from the wound edges. In more extensive burns surgical procedures including necrotic tissue removal and skin grafting for coverage/closure of these wounds are necessary.
The other criteria include:

- Patient’s age,
- Localization of burns,
- Burns aetiology,
- Concomitant diseases,
- Concomitant injuries.

### Tab. 3 Symptomatology of local changes in skin burns

<table>
<thead>
<tr>
<th>Degree</th>
<th>Damage</th>
<th>Appearance</th>
<th>Clinical signs</th>
<th>Common cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>I (Fig. 1a)</td>
<td>Superficial and deeper parts of epidermis</td>
<td>Fresh burn: redness, swelling, no blisters Later: colour change of epidermis, desquamation</td>
<td>Burning pain of several hours duration, relief by cooling</td>
<td>Sunburn, Explosion, scalding by lower temperature hot water</td>
</tr>
<tr>
<td>IIa (Fig. 1b)</td>
<td>Complete loss of epidermis down to basement membrane</td>
<td>Epidermal blisters, following their removal pink and wet wound bed, positive blanching phenomenon</td>
<td>Sharp pain of several hours duration</td>
<td>Contact burns, scalding by boiling water of uncovered body areas, electrical arc (flash)</td>
</tr>
<tr>
<td>IIb (Fig. 1c)</td>
<td>Complete loss of epidermis, necrosis of deeper dermal layers with preservation of some dermal elements containing epidermal structures (skin adnexa)</td>
<td>Fresh burn: pale appearance, less blisters, purple or whitish wound bed, blanching phenomenon negative, Later: less discharge, dryer wound bed</td>
<td>Less intensive pain, less sensitive wound bed, sharp pain by pin prick recognized as pain</td>
<td>Explosions without secondary clothing ignition, falls into hot liquids, scalding by hot oil or fat</td>
</tr>
<tr>
<td>III (Fig. 1d)</td>
<td>Complete loss of both epidermis and dermis, necrotic changes can extend to subcutaneous tissues and even to deeper structures</td>
<td>In fresh scalds pearly white, in burns brownish, or dark to black, firm, thrombotic blood vessels underneath the skin can be visible</td>
<td>Painless, pinprick feels like touching</td>
<td>Flame, clothing ignition, high voltage electrical current, concentrated chemicals (acids, alkali etc.)</td>
</tr>
</tbody>
</table>

1Blanching phenomenon: by pressing the wound bed with sterile spatula, or other similar object for 10-15 seconds and quick relieve of pressure, blanching is observed initially, followed by reappearance of the original pink colour of the wound bed. Blanching is present only if the dermal capillary network is not occluded. In deep dermal burns the capillary network is occluded by thrombosis and blanching cannot be evoked.

**Patients’ age**

The age of the patients is very important in the process of severity evaluation. Burns in children aged 2 years and less are more severe than in the older ones, as functional reserves in organs and systems are less developed than in the older age groups. Patients aged 60 years
and more represent a significant mortality risk increase due to various complications. Their organs/systems functional reserves are decreased as well which increases the severity of their burn injuries.

Tab. 4 Burn severity evaluation and classification of burn injuries

<table>
<thead>
<tr>
<th>Severity Factors</th>
<th>Burn classification</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Small/minor</td>
</tr>
<tr>
<td>Extent Adults</td>
<td>&lt; 15%</td>
</tr>
<tr>
<td>Extent Children</td>
<td>&lt; 10%</td>
</tr>
<tr>
<td>Depth Adults</td>
<td>Deep &lt; 5%</td>
</tr>
<tr>
<td>Depth Children</td>
<td>Deep &lt; 1%</td>
</tr>
<tr>
<td>Age</td>
<td>&gt;2y. &lt;60 y.</td>
</tr>
<tr>
<td>Localisation</td>
<td>NO: face, hands, feet, genitalia</td>
</tr>
<tr>
<td>Aetiology</td>
<td>NO: electric Chemical</td>
</tr>
<tr>
<td>Concomitant diseases</td>
<td>No</td>
</tr>
<tr>
<td>Concomitant injuries</td>
<td>No</td>
</tr>
<tr>
<td>TREATMENT &amp; subsequent care</td>
<td>Outpatient</td>
</tr>
</tbody>
</table>

Burns localization

Burns affecting functionally and/or aesthetically important areas increase the severity of the injuries as well. These areas include face, hands, feet and genitalia. In more severe face burns the subsequent oedema (swelling) can extend to respiratory areas with major threat of their obstruction. Oedema of the eyelids can prevent them to open which can cause temporary blindness. Burns of the hands and feet are painful and need to be dressed, which can prevent the victims to care of themselves. Burns in genital and anal areas are threatened by immediate or later contamination by residential gram negative bacteria and are threatened by development of wound infection. All the above mentioned factors thus increase the burn severity.

Co-morbidities

Several severe chronic diseases such as diabetes, atherosclerosis, heart diseases, malignancies, etc. can increases the severity of the burn injuries. This can be applied as well to long-term intake of several medications (such as hormonal therapy, immunosuppressive therapy etc.).
Concomitant injuries

Burn injury can be associated to other injuries such as mechanical trauma (fractures, large open wounds, craniocerebral injuries, thoracic injuries, abdominal injuries etc.). By flame burns and explosions in enclosed spaces there should be always a suspicion of simultaneous inhalation injury.

Cause of burn

Several aetiological factors such as electric current or concentrated chemical caustic agents will increase the burn severity as well.

4.4 **INSERTION OF LARGE BORE INTRAVENOUS LINE**

If it was not done already at the accident site, or during transportation, large bore venous cannula should be inserted preferably by unburned skin area. The cannula will serve as a direct vascular access for intravenous administration of various medicaments along with large amounts of intravenous fluids (substitution therapy) in the shock period. It should be the preference of the intensive care specialists, which access would be selected. In cases where problems with peripheral venous access will occur, central venous cannula should be inserted (such as v. subclavia, or internal jugular vein). As soon as the intravenous line will become functional, initial doses of analgesic medications should be administered, and followed by initiation of massive fluid replacement therapy by balanced crystalloid solutions (such as Ringer lactate, or Hartmann’s solution. **Administration of hypotonic solutions** (like 5% or 10% dextrose) is contraindicated.

4.5 **PAIN MANAGEMENT**

Analgesia and sedation of the patients by intravenous administration is integral part of shock treatment in order to decrease the severe stress reaction and to sedate the intubated patients undergoing artificial ventilation. Effective initial pain management helps to decrease the extent of the burn shock as well.
4.6 **FLUID REPLACEMENT THERAPY**

4.6.1 **SUBSTITUTION THERAPY OF BURN SHOCK**

Only a few decades ago, when the pathophysiological mechanisms of the burn shock have not been understood, the mortality of burn injuries exceeding 30% TBSAB was extremely high (50% and more) and the majority of them was dying due to hypovolemic shock and its consequences. Even at the present time, the main causes of death in 50% of the extensively burned patients who die within 10 days post injury will occur due to complications resulting from inadequate initial fluid replacement, or errors which occur in relation to substitution therapy. So far there does not exist a worldwide consensus about provision and methods of fluid replacement therapy during burn shock (see Tab. 5).

As it was mentioned already (pp. 11-12), the burn shock starts to develop in a very short time frame (within several minutes) following the accident. Massive leak of fluids from intravascular space to surrounding interstitial tissues is enabled by generalized pathological increase of capillary permeability in areas surrounding the burn wounds in less extensive injuries, and in the entire human body in extensive burns. The fluid loss is invisible, as the majority of fluids escape from the circulation into the interstitial space and not outside of the body. So far there are no medicines, which could prevent or stop this massive capillary leak. The duration of the generalized capillary permeability is from 24 to 72 hours, and then it starts to decrease gradually to normal values. Therefore the only option how to replace the massive losses of intravascular volume is massive continuous fluid replacement by intravenous administration. Massive fluid replacement shall continue until the capillary permeability will return to normal values. Maintenance of adequate blood flow (and, of course, simultaneous good oxygen and nutrients supply) to organs and systems compromised by blood flow redistribution (see Tab. 5) will be the only possibility for prevention of early organ and system failures which increase tremendously mortality of such patients.

4.6.2 **MAIN GOALS OF SUBSTITUTION THERAPY**

- Maintenance of adequate circulatory volume by rapid replacement of the initial loss of fluids and/or prevention of further fluid losses from circulation into interstitial space.
- Effective replacement of electrolytes, water, and albumin losses,
- Maintenance of homeostasis (fluids and electrolytes balance),
- Prevention or mineralisation of organs dysfunction/failure,
- Aim at maintaining adequate circulation, tissues oxygenation and organs function with the minimal amount of fluids administered,
- Aim at prevention of fluid overload, which can cause other severe or even life threatening complications (such as cerebral oedema, pulmonary oedema, abdominal compartment syndrome).

4.6.3 SPECIFIC GOALS OF FLUID REPLACEMENT THERAPY

- To maintain normal, or near-normal hydration of the human body,
- Normalisation of acid/base balance,
- Maintenance of adequate functions of all organs (kidneys, heart, lungs, liver, brain, etc.),
- Restoration and maintenance of adequate tissues perfusion in order to assure good oxygen supply to both reversibly damaged and undamaged tissues. This can enhance the natural healing processes and prevent deepening of the burn wounds.

There are several calculations (formulae) (Tab. 6) for calculation of quantity of fluids for substitution therapy. They fall into two groups:

- For crystalloid solutions: isotonic and hypertonic,
- For colloid solutions: proteins (plasma, albumin), polysaccharides starches.

4.6.4 FORMULAE FOR CRYSTALLOIDS

At the present time the use of crystalloid solutions is preferred compared to colloid solutions for initial resuscitation (first 24 hours). The most popular formula used worldwide is the Parkland formula developed by prof. Baxter at Parkland Memorial Hospital in Dallas, USA. The solution used is Ringer lactate (RL) in English speaking countries and Hartmann solution (HS) which is popular in German speaking countries. Their composition is almost identical.

**PARKLAND FORMULA FOR FIRST 24 HOURS**

\[ 4 \text{ ml RL} \times \text{kg patient’s body mass (BM)} \times \% \text{TBSAB} \]

Parkland formula is calculated to achieve an hourly urine output of 1 ml/kg body mass.

As the fluid leak is most extensive in the first few hours post burn, it is recommended to administer half of the calculated fluid amount during the first 6 hours post burn and the rest during following 16 hours. One of the advantages of crystalloid fluid resuscitation is its availability and low cost. Disadvantages include more pronounced hypoproteinaemia in very
extensive burns and more extensive interstitial tissue oedema formation. For imagination of how large fluid amounts need to be given in the first 24 hours below is a sample calculation.

**Patient:** male, body mass 100 kg, TBSAB 50%

**Calculation:** \( 4 \times 100 \text{ (kg)} \times 50 \text{ (\%)} = 20000, \text{00 ml} \)

As the above calculation have shown, the patient will need to receive 10 000 ml of RL (10 litres) within the first 8 hours and additional 10 000 ml of RL (10 litres) during the following 16 hours.

### 4.6.5 FORMULAE USED FOR COLLOIDS (PROTEINS – PLASMA, ALBUMIN)

Plasmatic proteins are important for generation of oncotic pressure necessary for compensation of capillary hydrostatic pressure. Those in favour of colloid resuscitation recommend including colloids into resuscitation formulae. The first quite widely used formula was suggested by Evans and calculated 1 ml colloids for 1 kg body mass and 1% of TBSAB together with 1 ml crystalloids for kg and % TBSAB. This way the total amount of calculated fluids was one half of that of fluids calculated by Parkland formula.

Even if it seems that lower fluid load would improve the success of initial fluid replacement therapy of the burn shock, randomized controlled studies confirmed lower mortality with the use of crystalloid fluid resuscitation. The recommendations from these studies can be summarized as follows:

- Proteins should not be used during the first 24 hours, as in this period of excessive capillary leak, they are not more effective in maintaining circulatory volume compared to crystalloids. On opposite, they can increase extravascular lung water accumulation and they can hamper early mobilisation of oedema fluid accumulated in tissues.
- Some of the proteins, particularly albumin, can be used mixed together with crystalloids early following injury.
- Protein administration in selected cases can be started already from 8 to 12 hours post injury.
- Proteins, particularly fresh frozen plasma can be administered earlier in case of planned early surgical intervention.
Tab. 5 Survey of most frequently utilized formulae for substitution therapy

<table>
<thead>
<tr>
<th>Formula</th>
<th>First 24 hours</th>
<th>24-48 hours</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Colloids (ml)</td>
<td>Crystalloids (ml)</td>
<td>Colloids (ml)</td>
</tr>
<tr>
<td>Parkland</td>
<td>0</td>
<td>4 x kg x % TBSAB</td>
<td>0.3-0.5 x kg x % TBSAB</td>
</tr>
<tr>
<td>Brooke update</td>
<td>0</td>
<td>3 x kg x % TBSAB</td>
<td>0.3-0.5 x kg x % TBSAB</td>
</tr>
<tr>
<td>Evans</td>
<td>1 x kg x % TBSAB</td>
<td>1 x kg x % TBSAB</td>
<td>0.5 x kg x % TBSAB</td>
</tr>
<tr>
<td>Brooke</td>
<td>0.5 x kg x % TBSAB</td>
<td>1.5 x kg x % TBSAB</td>
<td>0.25 x kg x % TBSAB</td>
</tr>
<tr>
<td>Slater</td>
<td>75 x kg FFP (fresh frozen plasma)</td>
<td>2 000</td>
<td>?</td>
</tr>
<tr>
<td>Galveston</td>
<td>5000 ml x m² TBSAB + 2000 ml x m² BSA (12.5g albumin / 1 l RL)</td>
<td>3750 ml x m² TBSAB + 1500 ml x m² BSA (12.5g albumin / 1 l RL)</td>
<td>Children only!</td>
</tr>
<tr>
<td>Monafo</td>
<td>0</td>
<td>Volume keeping HD around 30 ml</td>
<td>As Parkland</td>
</tr>
<tr>
<td>Warden</td>
<td>0</td>
<td>Volume keeping HD around 30 - 50 ml</td>
<td>As Parkland</td>
</tr>
<tr>
<td>Demling</td>
<td>1.-8. hrs: 2ml x kg x hr Dextran 9.-24. hrs: 0.5 ml x kg x % FFP</td>
<td>RL keeping HD around 30 ml</td>
<td>?</td>
</tr>
</tbody>
</table>

RL = Ringer lactate  BSA (Body Surface Area)
FR = 0.9% saline  BSAB (BSA Burned)
FFP = fresh frozen plasma  G 5% = 5% dextrose solution
HD = diuresis per hour

4.6.6 COLLOID RESUSCITATION USING PLASMA EXPANDERS

A couple of decades ago the only polysaccharides for intravenous administration were dextranomers – Dextrane and Rheodextrane, which have been used as plasma substitutes. Their major disadvantage was patients’ sensitisation which could result in allergic reactions. Recently developed colloid solutions for therapeutic use are modified starch products. In the last few years hydroxyethylstarch was used as plasma expander. Although this product proved to be effective in maintaining circulatory volume, recent studies confirmed increased mortality with its use in critically ill patients.

Fluid replacement therapy in paediatric burns

In comparison with substitution therapy of adult burns, fluid replacement management of paediatric burns is more difficult, as the physiological reserves of paediatric population are
much more limited than in normal adult population. There are evidences that with the same TBSAB involvement children require higher amounts of fluids to be replaced than adults. In children aged less than 3 years fluid substitution therapy is indicated in burns involving 5% of TBSAB already, whereas in adults oral fluid substitution is satisfactory up to TBSAB of 15%. Parkland or Brooke formulas can be used in children as well, but better alternatives are special paediatric formulae such as Galveston or Cincinnati formulae where body mass (in kgs) is replaced by body surface area in m². Example of the Galveston formula is below.

**Galveston formula:**

\[ RL \times 5000 \text{ ml} \times \text{TBSAB (in } m^2) + 2000 \text{ ml} \times m^2 \times \text{BSA (in } m^2) \]

\[(12,5 \text{ g albumin is added to } 1000 \text{ ml of RL solution)}

\begin{tabular}{ll}
RL=Ringer-lactate; BSA= Body Surface Area in m² & \\
\end{tabular}

**4.6.7 FLUID REPLACEMENT THERAPY IN INHALATION INJURY**

Inhalation injury increases the fluid losses in burn patients requiring higher volumes of substitution therapy for burn shock resuscitation. This increase, depending on the severity of the inhalation injury, can vary between 20 and 50% of the calculated volumes during the first 24 hours, as compared to patients without inhalation injury. As inhalation injury can predispose the patients to development of more rapid and extensive pulmonary oedema, the volume of fluids administered should be kept at the minimal level which can assure stability of circulation and basic organ functions.

**4.6.8 RECOMMENDED PRACTICES AND TACTICS OF BURN SHOCK SUBSTITUTION THERAPY**

**FIRST 24 HOURS**

**Adult patients**

In our burn centre we start fluid substitution according to calculations by Parkland formula. Instead of RL solution we use Hartmann solution, which composition is almost identical to RL. The clinical response of patients to fluid administration is evaluated at least each hour (parameters measured and evaluated see Tab. 6, p. 36) in order to enable making corrections of the rate of fluid administration.

**Paediatric patients**

In children aged 3 years and older Parkland formula can be used for calculations, but we prefer to use Galveston formula in ages up to 10 years. Galveston formula is recommended in
all paediatric patients younger than 3 years. It is very important to keep in mind, that excessive fluid administration in children younger than 3 years is much more dangerous than insufficient one. The reason is that renal functions of this age group are not developed fully, and their kidneys are not capable to excrete rapidly large amounts of fluids, which then accumulate in tissues and contribute to excessive oedema formation. Extremely dangerous for children is oedema formation in brain, lungs, and abdominal cavity, which can lead to irreversible ischemic damages. Administration of hypotonic solutions (such as 5% dextrose, or ½ saline solutions) is strictly contraindicated as they enhance oedema formation in tissues and organs.

**FROM 24 TO 48 HOURS POST BURN**

The replacement therapy fluids demand is lower and is roughly 50% of that in the first 24 hours.

By Parkland formula calculations crystalloids are no more used for substitution in this period. Instead, colloids administration (fresh frozen plasma or 5% albumin solution) is started in amounts of 0.5 ml x kg x % TBSAB supplemented by 10% dextrose solution. The amount of dextrose solution should be corresponding to the amount of urine excreted during the previous 24 hours.

In children where the Galveston formula was used for the first 24 hours, the calculation for the second 24 hours is as follows: 3 750 ml x m² TBSAB + 1 500 ml x m² BSA (fluid loss according to burn surface) + (normal fluid loss).

**Tactics of substitution therapy**

All the calculations (formulae) for fluid replacement therapy serve only as good guides for initiation of fluids substitution. Each patient is a unique individual, whose response to therapy can be quite different from other patients. Therefore, following fluid replacement initiation, the subsequent fluid replacement management shall be strictly tailored to the particular patient and guided by clinical response of the patient to therapy administered (see also Tab. 7, p. 37). In patients of same age, same TBSAB, these differences can vary on average by 50% or even more (see below in Clinical response part). There are some situations which can influence the patient’s demand to fluid replacement volume. In superficial burns of same TBSAB the fluid demand can be lower, in very deep burns (such as if the entire TBSAB is IIIrd degree) the fluid demand is usually higher. Fluid demand is usually increased also in patients with inhalation injuries and high tension electrical burns.
4.6.9  **Clinical Response and Patients Follow-Up**

It is of utmost importance to provide regular evaluations of clinical response of each patient to therapy administered and make appropriate corrections. The patients require continuous monitoring of basic vital signs and functions and regular monitoring of laboratory and other parameters. According to the results obtained, the subsequent fluids, electrolytes and other medications are corrected and adjusted (see Tab. 6, p. 36).

Major stress reaction post burn initiates compensatory mechanisms against massive circulatory volume losses, which can mask clinical symptoms of severe shock state initially, particularly hypotension. The clinicians need to keep this in mind by diagnosing if the patient is in shock, or not (see also Tab. 7, p. 37).

**Blood pressure and pulse rate values of burned patient** do not need to signal differences from normal ranges initially due to severe stress reaction accompanied by massive release of suprarenal medullar hormones into blood circulation initially. Normal blood pressure can be measured initially thus altering one of the most important sign of hypovolaemia. On the other side, if rapid drop of blood pressure is detected, it signals severe hypovolaemia due to failure of effective compensatory mechanisms and requires a rapid therapeutic intervention - increase of the speed of fluids administration.

**Central venous pressure** (CVP) is usually very low, or even negative in the first 24 hours of substitution therapy due to decreased cardiac preload. Any attempts to “hunt” the CVP by increasing fluids intake shall not be provided, as this can lead to over resuscitation with serious consequences, which have been mentioned already (see also pp. 29-31). **There shall be never tried to normalize low CVP during the first 24 hours post burn!** Normal or elevated CVP in the first 24 hours is usually a signal of cardiac dysfunction or failure, or a signal of excessive fluid replacement therapy. If this occurs, invasive and more detailed cardiovascular function parameters need to be monitored by inserting of Swan-Gang catheter into pulmonary artery and by measuring pulmonary artery wedge pressure and cardiac output. Increasing the invasivity of monitoring methods can lead to increase of their complications as well.
### Tab. 6 Selected parameters for burn shock monitoring

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Monitoring method</th>
<th>Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consciousness</td>
<td>Clinical</td>
<td>Each ½ hour</td>
</tr>
<tr>
<td>Pulse</td>
<td>Monitoring</td>
<td>Continuous</td>
</tr>
<tr>
<td>Oxygenation</td>
<td>Pulse oximetry, Acid – base balance, PaO₂</td>
<td>Continuous, At least à 12 hrs.</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Manual or automatic measurements, Monitoring by arterial line</td>
<td>½ hr, Continuous</td>
</tr>
<tr>
<td>Ventilation</td>
<td>Monitoring</td>
<td>Continuous</td>
</tr>
<tr>
<td>Central Venous Pressure (CVP)</td>
<td>Manual measurement, Monitoring by central venous line</td>
<td>½ hr, Continuous</td>
</tr>
<tr>
<td>Urine output</td>
<td>Collection by urinary catheter to bag, Specific gravity measurement, Biochemical investigation of urine</td>
<td>1 hr, 1 hr, daily</td>
</tr>
<tr>
<td>Peristaltic</td>
<td>Clinically / monitoring, clinically auscultation</td>
<td>½ hr, Continuous</td>
</tr>
<tr>
<td>Acid base balance</td>
<td>Capillary blood sample, Or: arterial &amp; venous blood sample</td>
<td>At least 2x/day, Or more frequently</td>
</tr>
<tr>
<td>Serum electrolytes</td>
<td>Venous blood sample</td>
<td>At least 2x/day, More frequently, if necessary</td>
</tr>
<tr>
<td>Glycaemia</td>
<td>Venous blood or capillary blood sample</td>
<td>Usually 1x / day, By diabetes more frequently</td>
</tr>
<tr>
<td>Blood count</td>
<td>Venous blood sample</td>
<td>1x / day, + following each operation</td>
</tr>
<tr>
<td>Biochemical screening¹)</td>
<td>Venous blood sample</td>
<td>Usually daily</td>
</tr>
<tr>
<td>Inflammation Markers ²)</td>
<td>Venous blood sample</td>
<td>Usually daily</td>
</tr>
<tr>
<td>Sepsis Markers ³)</td>
<td>Venous blood sample</td>
<td>Usually daily</td>
</tr>
<tr>
<td>CK (creatins-kinase)</td>
<td>In high-tension electrical burns, Venous blood sample</td>
<td>Usually twice daily</td>
</tr>
<tr>
<td>COHb (carboxyhaemoglobin)</td>
<td>In case of intoxication suspicion, (burns in enclosed spaces), Venous blood sample</td>
<td>Hourly until normalisation of COHb level in blood</td>
</tr>
</tbody>
</table>

¹) Biochemical screening: electrolyte, urea, kreatinin, glucose, total proteins, albumin, AST, ALT - serum
²) Inflammation Markers: CRP
³) Sepsis Markers: blood count (neutrophils, thrombocytes), PCT (procalcitonin), presepsin

CVP = central venous pressure

**Urine output** measuring each hour is one of the best and simple monitoring methods of fluid replacement therapy adequacy. It is relevant if there were no dysfunctions and/or chronic renal diseases in the past. Optimal values of hourly urine output should be in adults in a range of 0.3 – 0.5 ml x kg (body mass), in children it should be in a range of 0.5 – 1 ml x kg. In addition to the quantity of urine obtained per hour measurements of urine specific gravity shall be done as well. During the shock period by normal kidney functions the specific gravity of the urine should be above 1020. Specific gravity of urine below 1020 can indicate either
excessive fluid intake (or administration of diuretic medications, which is not recommended until normal circulatory volume is restored), or it can be a signal of renal dysfunction (both chronic and acute). Low urine specific gravity is usually one of the signs of kidneys damage (loss of urine concentrating capacity). In high-tension electrical injuries the damage of tissues can extend into the muscles causing disruption of myocytes and massive release of myoglobin into blood circulation (myoglobinaemia). Resulting myoglobinuria itself can cause severe kidney damage by obstructing the renal tubular system. Therefore in cases of massive myoglobinuria it is recommended to protect the kidney functions by increase of substitution therapy fluids administration and by attempting to alkalize urine by addition of sodium bicarbonate (NaHCO$_3$) to fluids administered. Osmotic stimulation of diuresis by Manitol infusions can be helpful as well.

Tab. 7 Interpretation of clinical parameters during burn shock monitoring

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Optimal values</th>
<th>Pathological values</th>
<th>Interpretation of differences</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensorium</td>
<td>Clear</td>
<td>Impaired</td>
<td>Even in very extensive burns the patient is usually conscious. Different steps of unconsciousness can be observed in concomitant head injury or intoxication</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>Within normal limits</td>
<td>Hypotension</td>
<td>Hypotension can be found in cases of extensive or inadequately replaced fluid losses, vasomotor dysfunction and/or heart failure</td>
</tr>
<tr>
<td>Peripheral blood flow</td>
<td>Good (acral parts warm, pink, rapid capillary refill)</td>
<td>Compromised (acral parts cold, cyanotic, slow capillary refill)</td>
<td>Compromised blood flow at the periphery is present in deep shock state, or severe compensatory vasoconstriction</td>
</tr>
<tr>
<td>CVP</td>
<td>Normal values or decreased</td>
<td>Increased values</td>
<td>Increased CVP is a sign of excessive replacement therapy (over resuscitation) or dysfunction of the myocardium</td>
</tr>
<tr>
<td>Pulse</td>
<td>Regular, strong, &lt; 100/min.</td>
<td>Weak, irregular, &gt; 100/min.</td>
<td>Tachycardia accompanied by weak pulse together with drop of blood pressure and diuresis are usually signs of inadequate fluid replacement. Bradycardia and/or arrhythmia are usually signs of myocardial dysfunction</td>
</tr>
<tr>
<td>Hourly urine output</td>
<td>0.5-1ml x kg</td>
<td>&lt; 0.5ml x kg</td>
<td>These values are relevant only if there are no signs of kidneys dysfunction (urine specific gravity should be above 1020)</td>
</tr>
<tr>
<td>Urine specific gravity (SG)</td>
<td>&gt; 1020</td>
<td>&lt; 1020 during first 24 hours</td>
<td>Low urine SG indicates renal damage initially, later in polyuric phase it is normal</td>
</tr>
</tbody>
</table>

*CVP = central venous pressure*
4.6.10 **WHEN TO FINISH FLUID REPLACEMENT THERAPY OF SHOCK**

Fluid replacement therapy of shock should be finished as soon as the pathologically increased generalized capillary permeability will return to normal. This will stop the uncontrolled fluid leak from circulation into interstitial space and excessive accumulated fluids from generalized oedemas will start to be mobilized back into the blood flow. They are then going to be excreted by kidneys out of the body. Clinical evidences of these events show return of circulatory stability and maintenance of normal values of urine output by fluid intake corresponding to fluid losses by burned areas, urine and respiration. Administration of colloids should continue according to clinical and laboratory findings after the cessation of shock resuscitation. The same applies for continuing monitoring and maintenance of fluid and electrolytes balance throughout the acute period of burn disease.

4.6.11 **FAILURE OF FLUID SUBSTITUTION THERAPY**

There are situations where the fluid replacement therapy of burn shock is not successful and fails. The most common causes of treatment failure can include:

- delayed start of fluid substitution treatment,
- extensive and excessive tissue damage not compatible with survival of the victim,
- severe inhalation injury,
- acute failure of suprarenal glands,
- extreme age groups (less than 2 years and more than 60 years) by very extensive burns,
- co-morbidities which affect several organ systems such as cardiovascular system, severe metabolic diseases, severe immunological disturbances etc.

4.6.12 **SUMMARY OF SUBSTITUTION THERAPY MANAGEMENT**

Well managed substitution therapy is one of the key factors for successful outcome of extensive burns. As there are options for selection of different formulae, the best practice should be to use the formula which the physician has enough experience already with. As it was mentioned already, the formulae should be used for initial fluid replacement calculations only. The subsequent guidance of fluids substitution therapy should be individually adjusted according to patient’s response and needs. It is advisable, particularly in children and older individuals to keep the patient on the “dry side” that means the use of minimal amounts of fluids for maintenance of basic vital functions during the first 48 hours post burn. Excessive
administration of fluids is much more dangerous to the patient as its complications include excessive fluid leak into interstitial space and their accumulation in organs and tissues resulting in very serious complications such as cardiac failure (in older individuals), pulmonary oedema, brain oedema (particularly in children), intermediate burns deepening, abdominal compartment syndrome and a variety of other complications as well.

4.7 Ventilation and Oxygenation of the Patient

In patients without inhalation injury that is able breathing spontaneously respiratory parameters should be monitored. It is important to aim at keeping arterial (capillary) blood oxygen saturation ($\text{SaO}_2$) above 95%. In cases where there is suspicion of inhalation injury, early intubation followed by artificial ventilation should be provided. If the intubation is not done in time, before oedema formation, the airways can be obstructed by rapid swelling making intubation difficult or impossible. If this occurs, fiberoptic intubation or urgent tracheotomy shall be performed to assure free airways. In intubated and heavily sedated patients artificial ventilation shall be instituted. Unconscious patients and patients where there is suspicion of carbon monoxide intoxication shall be intubated and ventilated as well. Inspired oxygen fraction ($\text{FiO}_2$) shall be adjusted to a level assuring $\text{SaO}_2$ above 95% in order to achieve good oxygen supply of organs and tissues.

4.8 Thermal Comfort of the Patient

By all the emergency procedures provided at hospital admission of severely burned patients good thermal comfort of the patients is mandatory in order to prevent hypothermia, which can worsen the extent of the burn shock. Optimal room temperatures should be kept between 30 °C and 33 °C. It is necessary to avoid large body areas left exposed for a longer time. All the body areas where procedures are not provided should be covered by sterile blankets. If these measures are not sufficient to maintain body core temperature in normal range, heating blankets combined by infrared heaters should be used.

4.9 Urine Output Measurements

In all extensive burns which require fluid replacement therapy hourly urine output monitoring is mandatory, therefore a urinary catheter should be inserted. The catheter shall be connected
to a closed collecting system to a calibrated container. The volume and the specific gravity of the excreted urine shall be measured hourly and recorded in patient file.

4.10 **BLOOD, URINE, BACTERIOLOGY, AND OTHER SAMPLES FOR LABORATORY INVESTIGATIONS**

Blood samples for routine basic laboratory investigations should be obtained at admission of the patient to hospital (see also Tab. 7). In addition to basic checks of blood count and biochemistry investigations (such as blood glucose; electrolytes – Na⁺, K⁺, Ca²⁺, Mg²⁺; kreatinin, urea, ALT, AST) additional samples for blood group and coagulation parameters investigation are taken. Urine samples are obtained immediately after urinary catheter insertion. In severely burned and artificially ventilated patients capillary, arterial and venous blood samples are obtained for acid base balance investigation.

4.11 **PRIMARY SURGICAL MANAGEMENT OF EXTENSIVE BURN WOUNDS**

Primary burn wound care should start immediately after the initial resuscitation procedures have been finished and substitution therapy was initiated. The wound care of extensive burns should be provided in clean rooms – special dressing rooms or operation theatres with assurance of asepsis and antisepsis. Manipulations with the fresh, extensive open wounds require good analgosedation of the patient, or general anaesthesia. Rooms for wound care procedures shall be heated to at least 30 °C to prevent heat loss of the patient. In ventilated, heavily sedated and anaesthetized patients the manipulation with the patient requires sufficient staffing. Minimal staffing include 1 surgeon, 2 scrub nurses, 1 anaesthetist, 1 anaesthesia nurse, 1 auxiliary person. Preceding the care of the patient the room should be heated to required temperature and all the instruments and other material should be readily available.

The order of particular procedures should be as follows:

4.11.1 **REMOVAL OF ALL CLOTHING AND FOREIGN BODIES FROM THE BODY SURFACE (IF NOT DONE BEFORE)**

The patient’s body shall be exposed and inspected in details looking for all the signs of injuries (not only burns) and pathological findings. All the observations shall be recorded in
patient’s file. Exposure of body parts should be done part by part and covered back thereafter to avoid heat loss

4.11.2 **OBTAINING INITIAL SAMPLES FOR BACTERIOLOGY EXAMINATION FROM ALL WOUND AREAS**

The obtained samples should be sent to microbiology laboratory as soon as possible.

**Cleansing and initial disinfection of skin surrounding the burn wounds followed by cleansing of the wounds located in different body areas part by part.**

In patients with heavily contaminated wounds scrubbing by mild disinfectant soap and water should be done first, thereafter cleansing of surrounding healthy skin followed by cleansing of the wound by mild, water-based, non-irritating disinfectant solutions such as Betadine, Octinesept, Prontoderm etc. should be provided. Alcoholic disinfectant solutions shall not be used for the wound areas. Most contaminated areas should be cleansed first, less contaminated later. It is necessary to change frequently the scrubbing sponges and/or gauzes together with the personnel’s gloves. The cleaned areas should be protected by wrapping them by sterile towels. Genital and perianal areas should be cleansed as the last ones.

4.11.3 **DEBRIDEMENT**

It includes removal of all the remnants of clothes, foreign bodies, including loose parts of devitalized epidermis from the burn wound area.

Management of blisters needs special attention. Remnants of epidermis from ruptured large blisters should be removed. Intact large blisters can be punctured and the blister content evacuated gently. Further blister management depends on the fact, which kind of open wound coverage will be used:

- If the wound will be covered by skin substitutes, it is necessary to remove all the devitalized epidermis including intact blisters as well.
- If skin substitutes for wound coverage are not available, then smaller intact blisters can be left in place, larger intact ones should be evacuated, and all the other loose epidermis should be removed from the wound surface completely.
- **In contaminated or already infected wounds all the devitalized loose tissues from the burn wounds surfaces shall be removed always in order to prevent occurrence or spread of infection.**
4.11.4 NECROTOMY, FASCIOTOMY

In cases of deep circumferential burns of extremities, trunk, or neck areas the necrotic skin desiccates, shrinks and prevents oedema expansion similarly as a tourniquet. At the same time blood vessels and nerves are compressed in their compartments which can result in compartment syndrome and ischemic damage to tissues. Increased compartmental pressure can be clinically detected by palpation and by assessment of sensitivity and blood flow at the acral areas. Increased compartment pressure can be measured by introduction of a needle or cannula in the compartment involved and subsequent measurement by direct arterial pressure measurement device. At the trunk area tough necrotic skin prevents respiratory movements which can lead to hypoventilation and respiratory dysfunction. In order to prevent development of deleterious consequences of these events the necrotic skin shall be incised – necrotomized in order to release excessive compartmental pressures and/or enable free respiratory movements. In deep burns necrotic areas are anaesthetic; therefore necrotomies can be provided with minimal analgesia. By typical necrotomy the necrotic skin and subcutaneous tissues are cut by surgical knife down to subcutaneous fat. Only in very deep burns with involvement of muscles and in high tension electrical burns the fascia should be incised as well. On extremities necrotomies are provided longitudinally laterally, or at both sides. On thoracic area necrotomies should be provided in chess like fashion enabling full expansion of the thoracic wall. Neck area should be necrotomized longitudinally bilaterally (see Fig. 6). Defects following necrotomies can be covered temporarily by any kinds of temporary skin substitutes (see also pp. 80-81).

4.11.5 TOPICAL THERAPY, DRESSING

Wound care procedure is finalized by selection of topical therapy method. There are 3 options:

- **Open method** with or without use of topical medications,
- **Semi – closed method** by application of medicated compresses,
- **Closed method by application of topical medications and/or wound covering materials, or temporary skin substitutes.**

More detailed description of these methods is on p. 81.
4.12 PREVENTION OF GASTRIC/DUODENAL STRESS ULCERS

Delayed or insufficient primary patient care, delayed start of substitution therapy, very deep and extensive burns and a range of other factors can cause prolongation of the shock period associated with a variety of resulting complications. Development of gastric or duodenal stress ulcerations used to be very common in these situations. Major threat from stress ulcers to the patient could be massive bleeding from the ulcers, or even their perforation into the abdominal cavity. Both of them can be life threatening. In order to decrease these risks preventive measure should be undertaken. The most effective prevention of stress ulceration is to maintain the original function of the gastrointestinal tract (GIT) by early enteral feeding. Principle condition of enteral feeding is good GIT function. The feeding should be initiated preferably not later than 6 hours post injury in conscious patients by oral administration of milk or enteral feeding preparations. In unconscious, ventilated and non-cooperating patients nasogastric, or nasoduodenal tube should be inserted and the feeding is provided by the tube. It there is initial gastroplagia, or there are bowel movement disturbances, enteral feeding cannot be provided and should be replaced by total parenteral nutrition. In these situations treatment for prevention of stress ulcers should be started by H2 blocker, or proton pump blocker drugs such as ranitidine, famotidine or omeprazol.

4.13 TETANUS PROPHYLAXIS ACCORDING TO NATIONAL REGULATORY REQUIREMENTS (SLOVAKIA)

Tetanus prophylaxis shall be provided as follows. In patients with small burns who have been immunized and since the last anatoxin dose did not elapse more than 10 years no other prophylaxis is necessary. In extensive burns which received their last anatoxin booster dose more than 5 years ago, a new booster dose of anatoxin shall be administered. In those patients, where since the last immunization more than 10 years elapsed a prophylactic dose of hyper immune human serum should be administered and simultaneously a new immunization by tetanus anatoxin shall be started (first anatoxin dose followed by second one after 6 weeks and third one after six months since the second dose).
4.14 OTHER NECESSARY PROCEDURES

4.14.1 ARTERIAL LINE INSERTION
It is advisable in all extensive burns and it is important for invasive continuous blood pressure monitoring and regular obtaining of samples arterial blood samples for acid-base balance investigation.

4.14.2 NASO-GASTRIC OR NASO-DUODENAL TUBE INSERTION
It shall be done in all severe and critical burn patients for assuring early start of enteral nutrition, or for decompression of the stomach in case of acute gastroplegia.

4.14.3 THERMAL COMFORT MAINTENANCE
Thermal comfort of the burned patients is of utmost importance. During emergency period the patients are threatened by massive heat loss during procedures, or where the ambient temperature is not high enough. Optimal ambient room temperature for such patients is between 30 and 33 °C. Particularly hypothermia is a big threat to patients during shock, as it can contribute to both circulatory and metabolic disturbances.

4.15 DIAGNOSIS

Initial diagnosis should be based on following components:

- Summary of vital signs and functions evaluation,
- Detailed diagnosis of the burn injury:
  - TBSAB (in % BSA),
  - BSAB superficial burns (i.e. I<sup>st</sup> degree and II<sup>nd</sup> a. degree) in %,
  - BSAB deep burns (i.e. II<sup>nd</sup> b. degree and III<sup>rd</sup> degree) in % BSA,
  - Localization of burns (description of body areas affected) (see also pp. 24-26),
- Concomitant injuries, if present (such as inhalation injury, intoxication, mechanical injuries etc.),
- Concomitant diseases – acute and chronic,
- Evaluation of key organs states and functions,
- Re-evaluation of burn severity and of burn classification.
4.16 DECISION ABOUT SUBSEQUENT CARE OF THE PATIENT

For decision about subsequent care of the patient see also p. 27, Tab. 4.

According to burn severity estimation and classification there are 3 modalities for subsequent care and treatment and the patients will be categorized into 3 following groups (Tab. 4):

- **Outpatient care** will be assigned to patients with small (minor) burn injuries. It will be provided by general practitioners, general surgeons, trauma surgeons, and plastic surgeons; in children by paediatricians or paediatric surgeons.

- **Hospital admission** and care in local hospital. Patients with moderate burns should be assigned for in-patient treatment in local and regional hospitals. In case when serious complications, or delayed healing will occur, transfer to higher level hospital facilities (such as burn centres) shall be indicated.

- **Transfer to burn centre.** All severe and critical burns shall be transferred to highly specialized treatment facilities, which are called burn centres. Transport logistics and guidelines are described below.

4.17 TRANSPORTATION OF BURN VICTIMS

4.17.1 MINOR BURNS

They do not require special precautions for transportation to physicians’ offices or hospitals. Usually either the nearest such facility, or a facility selected by the patient should be used. In cases where functional body areas (face, both hands, feet) are not affected, the patient can manage his/her transportation by itself.

4.17.2 EXTENSIVE BURNS

In exceptional cases, if the patients are conscious and alert and the hospital is close to the accident site, they can be transported by private cars. In all other situations (which are the most commons); emergency medical services shall be alerted and called. Immediately the emergency professionals will arrive to the accident site, they will start to provide all the emergency procedures which have been already described above (see also pp. 17-18). Initial patient’s stabilization is a basic condition for subsequent transportation (see p. 35).
4.17.3 **TIMING OF TRANSPORTATION, TRANSPORT WINDOWS**

Patients indicated for transfer to burn centres should be transported by emergency ambulance services, or emergency air rescue services. Selection of the transportation method will depend on anticipated transportation time and burn severity.

There are two transportation options:

1. **Primary transport** means transportation of the victim from the accident site to the nearest hospital, or directly to regional burn centre according to burn severity evaluation.

2. **Secondary transport** means transportation of the victim from the hospital facility where primary care was provided to higher level hospital facility, or specialized burn care facility (burn centre).

4.17.4 **PRIMARY TRANSPORT**

The most optimal modality is transportation immediately after emergency patient care at the accident site to the nearest burn centre in severe and critical burns (option 1). The best time frame for transportation is if the time from accident until arrival to final destination hospital facility will not exceed 2 hours. This is because during the first two hours post burns the symptoms of burn shock usually develop fully with a dominance of circulatory instability which can in worst scenarios progress to circulatory failure. In such cases the transportation will threaten the patient by deterioration of his/her circulatory instability. Therefore in cases, when emergency medical care was for various reasons delayed, or it would not be possible to finish the transportation within 2 hours from the accident, the patient shall be transported as fast as possible to the nearest hospital where appropriate primary care could be provided.

4.17.5 **SECONDARY TRANSPORT**

As soon as he/she will become stabilized (which takes usually at least 24 hours, but it should be no longer than 48 hours) the patient can be transported to the higher level hospital facility. It is not advisable to delay the transport for time longer than 48 hours in deep burns wound, as this delay could increase substantially both the probability and severity of infectious complications.
For the patient transportation there are 2 “ideal transport windows” to our disposition:

1st transport window: up to 2 hours post injury (from injury to arrival to destination)
2nd transport window: from 24 to 48 hours post injury (preferably not later)

Of course, the above mentioned criteria are not an absolute demand. Depending on the local situation and patient’s condition, the transportation indication and timing need to be clarified, negotiated and confirmed by the recipient hospital specialists on call.

4.17.6 PATIENT’S MANAGEMENT AND PREPARATION FOR TRANSPORTATION

Conditions for provision of transportation immediately after injury (up to 2 hours):

Transport shall be indicated by emergency physician. The decision should be followed by phone call to attending physician at the burn centre. By phone communication the following data should be submitted to the destination facility physician:

- Aetiology, time and circumstances of injury,
- Burn severity, vital signs, patient’s age, co-morbidities, and concomitant injuries,
- Time which elapsed since injury until arrival of rescuers,
- Injury site, location and distance from receiving facility,
- Estimated time and type of transportation (ground, air),
- Estimated arrival time.

4.17.7 PATIENTS CARE PRECEDING TRANSPORT SHALL INCLUDE:

- Insertion and assurance of 1 to 2 safe i.v. - lines by well-fixed large bore flexible cannulae (if not done at the scene), initiation and/or continuation of i.v. fluid substitution therapy. The fluid substitution shall continue during the entire duration of transportation as well.
- Vital signs monitoring during transportation.
- Endotracheal intubation with artificial ventilation is mandatory before transportation, particularly in case of patient transportation by air and if there is suspicion of inhalation injury or the TBSAB exceeds 50%. The ventilatory support shall continue during transportation of the victim.
- Primary emergency wound care should be limited to covering of the burned areas and the victim by sterile blankets, or dressing material. Detailed primary wound care of the victim should be delayed until arrival to the final destination facility.
Thermal comfort of the patient shall be assured by using body heaters, blankets and by heating of the transport vehicles, helicopters.

Selection of transport vehicles:

- If the surface transport time expected will not exceed 1 to 2 hours, the transportation can be realized immediately following emergency care by rescue ambulances.
- If the expected time for surface transportation will exceed 2 hours, air ambulance (helicopter) should be used.
- Selection of the mode of transportation should be considered taking into account the patient’s burn severity as well.

4.17.8 CONDITIONS FOR PROVISION OF DELAYED (OR SECONDARY) TRANSPORT TO BURN CENTRE

- Phone call and information exchange with the recipient facility.
- Continuation of patient monitoring, fluid replacement therapy and advanced life support measures during transportation.
- Primary hospital wound management including necrotomies/fasciotomies if indicated according to guidelines is described on the pages 40-44.
- Assurance of thermal comfort of the patient as above.

4.17.9 SUMMARY OF PRIMARY EMERGENCY CARE PROVISION IN HEALTH CARE ESTABLISHMENTS

Main tasks of the medical team by provision of primary care of the burned patients include:

In minor burns:
- Patient and his/her wound management,
- Pain management,
- Topical therapy and dressing applications,
- Tetanus prophylaxis,
- Instructions for home/ambulatory care.

In extensive burns:
- Re-evaluation of vital signs, initiation and/or continuation of advanced burn life support measures,
- Re-evaluation of burn severity assessment,
- Initiation and/or continuation in complex burn shock treatment measures,
- Primary surgical wound management,
- Diagnosis and decision of subsequent care of the patient,
- Management of patient’s transfer to higher level facility if indicated.

4.17.10 **SUBSEQUENT PATIENT CARE ACCORDING TO BURNS SEVERITY**

- **Minor/small burns** are assigned for outpatient care by family physicians, or by specialists such as surgeons, paediatricians, trauma surgeons etc.
- **Moderate burns** are treated in local or regional hospitals at surgical or trauma departments.
- **Severe and critical burns** require specialized treatment in burn facilities or burn centres.

If during outpatient treatment serious complications such as invasive burn wound or generalized infection, burn wound deepening, or significantly delayed healing occur the patients shall be referred to hospitals for their treatment.

There are also situations, where minor or moderate burns shall be referred to burn centres. These situations include severe complications such as rapidly spreading burn wound infection, worsening of co-morbidities, or in cases of unsuccessful treatment in other facilities.

It should be kept in mind, that the first few hours are very important and even decisive about the fate of severely burned patients. Each mistake, false decision, or delay, particularly in life support measures, initiation of substitution therapy, ventilatory and circulatory support, or management of transport modalities, can have a major impact on subsequent burn – related, or other kinds of complications, which can then worsen the prognosis and increase the mortality of such injuries.
5 MANAGEMENT OF MINOR BURNS

Minor burns represent 96% of all the burn injuries occurring annually among countries populations. Therefore their optimal management is of utmost importance and it can contribute to the overall quality of particular health care systems.

Principally even very small burn injuries where blister formation occurs, should be seen by physicians. If those injuries are neglected, the blisters usually rupture and expose open wound beds which in addition to pain can be easily contaminated and infected by microorganisms from the outer environment.

Most of the patients with minor burns are treated by family physicians, The others look for treatment in specialized physicians’ offices, mostly surgical, traumatological, paediatric, or even dermatological. As in other kinds of small injuries, when they are for the first time seen by physicians, basic medical principles should apply for their primary management and subsequent follow-up management until the wounds will heal completely.

5.1 PRIMARY MANAGEMENT

It is of utmost importance to treat a patient with his wound and not to treat “just a burned hand or foot”. The primary management, as well as the subsequent care, should be tailored individually to each patient: to his/her other health problems, his/her way of life, his/her profession etc. The sequence of events in primary care should be as follows.

1. Patient’s personal history, cause and circumstances of burn injury

The patient should be asked about acute and chronic diseases, allergies, previous injuries, surgeries, tetanus vaccination and medications which could interfere with treatment or healing of his/her burns. By asking about burn aetiology, questions pertaining burn source (scald, flame, contact, other), source temperature in thermal injuries and exposure time to source are important to know about. Next questions should pertain how and by who was provided the first aid. By knowing the source temperature, the time of exposure and how the first aid was provided, approximate estimation about the possible depth of the burn can be obtained.
2. **Burn wound inspection and evaluation**

Wound inspection includes wound localisation description, visual inspection of the wound itself, looking for blisters, eschars, and foreign bodies on the surface or embedded, and condition of the wound bed and appearance of wound margins. Capillary refill is checked on the wound bed for recognition of the burn depth. The extent of the wound is estimated according to the rule of palm (Fig. 2, p. 11). In smaller wounds the wound size is measured by tape. The appearance of the wound should correspond to the data about the injury mechanism and source given by the patient. If the wound is not fresh and the patient visits the doctor’s office after some days, the wound appearance is much different. The colour of the wound bed is usually not pink, or reddish, but can be darker, deeper areas are brownish, or even black. Signs of inflammation such as redness of the wound margins, or signs of wound infection such as purulent content of blisters, or purulent wound discharge can be present.

3. **Diagnosis**

Based on wound inspection and investigation and clinical findings which should be recorded in patient’s file an initial diagnosis should be set up. The diagnosis should include location of the burn, extent of burn in % or square cms and estimation of the depth of the burn. Presence of blisters, ruptured blisters, and eventual necrotic areas should be noticed and recorded as well. In non-fresh wounds signs of secondary changes should be observed and recorded.

4. **Microbiology samples**

It is advisable to obtain wound swabs for microbiology examination from all burn wounds deeper than 1st degree. Initial microbiology findings should serve as basic investigation in cases when burn wound infection will develop.

5. **Primary wound care**

Preceding primary wound care pain management should be considered. In patients with severe pain if cooling of the wound is not effective enough for pain relief, administration of analgesic medications is necessary according to physician’s choice. The wound care should be provided by aseptic techniques in order to prevent secondary wound contamination.

Primary wound care is provided by physician. Aseptic techniques in all phases of manipulations with the open wounds are mandatory. Its principles are as follows:
Preparatory phase
- sterile table with material and instruments preparation,
- the physician shall use sterile gloves, face mask and cap,
- using of aseptic non-touch techniques by all wound manipulations,
- obtaining initial wound surface smear samples for bacteriology

Wound care phase
- Scrubbing and cleansing of surrounding healthy skin by antiseptic soap solutions followed by disinfection by skin disinfecting preparations
- Gentle washing and cleansing of the wound area by water based disinfecting detergents followed by scrubbing with gentle preparations usable in open wounds (such as water based povidone iodine, Octenisept®, Prontoderm® etc.)
- Debridement of all the foreign bodies from the wound including remnants of devitalized epidermis
- Repeated wound rinses by sterile saline solution
- In dirty and heavily contaminated wounds debridements scrubbing and saline Rinses can be repeated several times until the wound will be clean
- Isolation of the debrided wound by sterile blankets
- Management of blisters: small size epidermal blisters can be left intact, larger size blisters shall be evacuated and epidermal remnants shall be left in place. If the wound was contaminated significantly, all the blisters shall be removed and the resulting wound bed shall be protected by temporary skin substitutes
- Application of topical antibacterial medications
- Application of burn wound dressing: Vaseline non-adherent mesh gauze or similar kind of medicated gauze should be used as wound contact layer of the dressing followed by gauze layer medicated by topical antibacterial agents (if not applied already directly on the wound) and covered by absorption layer (sterile cotton wool, or gauze) which is then fixed in place by elastic bandages
- If the burn wound is fresh (i.e. not older than up to 24 hours post burn) instead of mesh gauze contact layer temporary skin substitutes (see also p. 82) can be used. It should be kept in mind, that temporary skin substitutes shall not be used in full thickness burns, heavily contaminated or infected burns and in acute burns older than 24 hours.
- In selected cases the burned body areas shall be immobilized by tapes, special bandages or splints to assure uneventful healing of burned functional body areas over joints or at body parts with increased mobility of skin.

**Tetanus prophylaxis** shall be provided in patients according to regulatory requirements (see p. 43).

Following termination of the wound care procedures the patient should be instructed about any kind of home care procedures and precautions such as taking care that the dressing will not be displaced, or wetted from outside etc.

**Medications**

Preceding and during provision of wound management procedures administration of analgesic medications is recommended to the patient. Use of systemically administered antibiotics has certain rules. In fresh burns where primary wound care by physician was provided without any delay systemic antibiotic therapy is not indicated. Prophylactic **antibiotic administration** can be recommended **in patients with high risk** of infection development such as diabetics with poor diabetes compensation, immunocompromised patients, and patients using immunosuppressive drugs and/or treated by high doses of corticosteroid hormones etc. In patients with deeper burn areas, or patients who left their wounds untreated for several days and infection started to develop, before antibiotic administration samples from the wound for microbiological investigation should be taken and sent to the laboratory.

Analgesic drugs can be recommended and prescribed for the first few days post burn. Medications which have been the patient using before injury should be continued in unchanged doses and regimens.

### 5.2 FOLLOW – UP MANAGEMENT

First patient´s visit to physician´s office following primary care shall be scheduled following 48 hours. The dressing is removed from the wound, the wound is inspected by the physician and new topical treatment and dressing are applied. In case of uncomplicated further course the dressing should be changed regularly every other day until the wound has healed, or its area was reduced enabling home self-care by the patient. Normal healing time of uncomplicated superficial burns is from 5 to 14 days.

In patients with deeper burn areas or where the primary care was delayed, or the wound have been heavily contaminated from the beginning, wound infection can develop as the most
frequent complication. Clinical signs of wound infection include change of wound appearance, change of the character of wound discharge (from clear, serous to cloudy, purulent, accompanied by increased pain, redness of the wound margins and elevated body temperature. Unless there are signs of invasive or systemic infections (such as fever above 38.5 °C, increasing severe pain at the wound site, intensive redness of wound margins, purulent or foul smelling wound discharge, and lymphangitis with regional lymphadenopathy), the wound care should be provided more frequently with dressing changes every day, or twice a day and simultaneous use of topical antibacterial therapy. In cases where signs of spreading infection are present, systemic antibiotic therapy shall be started immediately and then continued according to bacteriology results.

If the spread of wound infection is rapid and its control not sufficient, the patient should be referred to the nearest hospital for hospital treatment.

5.3 Healing of Minor Burns and Complications

As it was mentioned above, superficial burns in case of uneventful treatment course will heal within 5 days to 14 days by epithelialisation without any permanent sequelae (scars). Deep dermal burns (II\textsuperscript{nd} b) are often threatened by infectious complications, which, if not treated early and efficiently will result to their conversion to full thickness burns, where surgical treatment by necrotic skin removal and skin grafting will become necessary. By uneventful treatment course the healing time of partial thickness burns extends from 3 weeks up to 6 weeks and they heal mostly by formation of scars. Smaller full thickness burns can heal by conservative treatment only occasionally by optimal conditions. Their healing time can extend to several weeks or even months, as in full thickness skin loss the skin cannot regenerate and healing can progress by epithelialisation from the wound margins only. Scars will always develop. Therefore in full thickness burns surgical treatment is recommended as a treatment of choice. By surgical treatment the necrotic burned skin is excised and replaced by split thickness skin grafts procured from healthy skin areas of the body (donor sites). Healing of the grafts occur by formation of vascular connections from the wound bed with the capillary network of the graft. Time to graft „take“ varies from 5 to 7 days. If formation of vascular connections will fail, the grafts will not take and they will necrotize.
6 MANAGEMENT OF EXTENSIVE BURNS

6.1 ACUTE PERIOD CARE

Emergency period of the burn disease will finish as soon as the general condition of the patients will become stabilized and they recover from the burn shock. During acute period continuation of comprehensive general patient care procedures including resuscitation and/or intensive care should be provided by intensive/resuscitation care departments (ICD) or intensive care units (ICU). Major threats to the burn patient in the acute period originate in the extensive open wounds (containing variable amounts of necrotic tissues) which persist open for relatively extended time periods. They include several complications which can affect practically any organ and/or system in the human body resulting from severe systemic impact of such an extensive injury to the human body.

6.1.1 REANIMATION AND INTENSIVE CARE

Comprehensive general patient care at ICD/ICU is managed by physicians – intensive care specialists in close cooperation with experienced burn surgeons, or plastic surgeons. Main goals of this treatment are listed below and consist of vital signs monitoring, continuation in maintaining fluids, electrolytes, energy, and thermal balances, infectious complications and other complications prevention and treatment, care of the extensive burn wounds, surgical interventions and intensive nursing care. This complex care needs a team approach meaning close cooperation of both medical and nursing specialists where the team leader is the senior burn or plastic surgeon. One of the world’s current leading burn specialist and professor of the University of Texas in Galveston, USA, and the Shriners Burn Institute doctor David Herndon calls this approach as “Total Burn Care”.

Main goals of the comprehensive general patient care at the ICU include:

- Vital signs and organs/systems functions monitoring/observations,
- Laboratory parameters investigations and monitoring,
- Organs/systems functions support,
- Assurance of thermal comfort of the patient,
- Monitoring and maintenance of fluids, electrolytes and acid-base balances,

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6.1.1.1  **VITAL SIGNS AND ORGANS/SYSTEMS FUNCTIONS MONITORING/OBSERVATIONS**

Regular observations and continual monitoring of vital signs and functions shall be provided.

**Basic vital functions monitoring include:**

- **Consciousness** of patients can be either described verbally (clear, oriented, disoriented by part, disoriented completely, obscured, unconscious) or rated using **Glasgow Coma Scale** (GCS). Best GCS score is 15, worst one is 1.

- **Cardiac and circulatory functions are monitored continually by multiple channels monitoring devices.** The parameters most frequently monitored include ECG, blood pressure by direct (continuous) or indirect methods, arterial/capillary blood oxygen saturation (SaO₂), pulse rate and quality, and central venous pressure. In very severe and critical cases other functions such as cardiac output and pulmonary artery wedge pressure should be monitored as well.

- **Respiratory functions** can be observed clinically, or monitored. In patients undergoing artificial ventilation respiratory parameters monitors are integral parts of artificial ventilation devices.

- **Body core temperature** is monitored continuously by temperature probe insertion into rectum, or it can be included in tips of urinary catheters.

**Other parameters for observations and/or monitoring include:**

- **Urine output** should be measured each hour by insertion of permanent urinary catheter. The catheter system should be closed with urine derivation into a sterile calibrated container. In urine samples obtained urine specific gravity should be measured at the bedside. Twice a day laboratory examination of urine should be provided.

- **Peripheral circulation** is usually evaluated clinically by palpation of peripheral pulses where appropriate, by observation of colour of non-burned skin, and by palpation and checking the peripheral healthy body parts (hands, toes) temperature. At
the same time capillary refill time can be observed as well by finger pressure and subsequent release.

6.1.1.2 LABORATORY PARAMETERS INVESTIGATIONS AND MONITORING

Blood samples for basic laboratory parameters monitoring should be obtained from patients at least once a day, selected parameters should be investigated more frequently.

Basic laboratory parameters should include:

- **Hematological parameters** should include blood count (erythrocytes, leucocytes, and platelets), hemoglobin, hematocrit, and selected coagulation parameters (fibrinogen, fibrin degradation products, prothrombin time etc.).
- **Blood group** should be examined in cases where blood transfusions are expected.
- **Acid-base** balance is examined from both arterial and venous blood samples.
- **Biochemistry investigations**: serum electrolytes (Na, K, Ca, Mg, Cl), glucose levels, bilirubin, hepatic enzymes (ALT, AST).
- **Inflammation and infection indicators**: serum levels of C-reactive protein (CRP), procalcitonine (PCT), presepsine.
- **Microbiology investigations**: at admission and subsequently in regular intervals (at least twice a week) swabs from burn wounds surfaces shall be obtained and sent to microbiology laboratory. In cases of sudden wound discharge changes, or signs of systemic infection, wound swabs and blood samples for cultures should be obtained and sent as soon as possible to the laboratory.

6.1.1.3 ORGANS/SYSTEMS SUPPORT

During the course of acute period complications from any organs or systems can occur. It is of utmost importance to observe and diagnose initial symptoms of organs or systems dysfunctions in order to slow or stop deterioration of the impaired functions and prevent them from progressing into organs/systems failure. If the diagnoses of organs/systems dysfunctions have been confirmed, appropriate therapeutic interventions should be started as soon as it is possible. Any delays in effective treatment can contribute to deterioration of the patient’s condition.
Most frequently affected organs and systems include the following ones:

- **Respiratory system** (inhalation injury, pulmonary oedema, adult respiratory distress syndrome – ARDS, in infants – IRDS, lung atelectasis, pneumonia etc.),
- **Genitourinary system** (renal dysfunction, renal failure, catheter related infections),
- **Gastrointestinal system** (abdominal compartment syndrome, stress ulcers – gastric, duodenal; acute gastroplaegia, paralytic ileus, gastrointestinal haemorrhages or perforations of stress ulcers with peritonitis, hepatic dysfunction, hepatic failure, acalculous cholecystitis etc.),
- **Cardiovascular system** (heart failure, refractory hypotension, ischemic complications etc.),
- **Central nervous system** (dysfunctions present themselves as disturbances of consciousness which can progress into coma states, failure occurs if GCS drops to 6 or below),
- **Endocrine system** (dysfunction or failure of suprarenal glands, thyroid gland dysfunctions, burn stress pseudo diabetes etc.),
- **Immune system** (disturbances in both humeral and cellular immunity, severe immunosuppression followed by infectious complications, etc.),
- **Hematopoetic system** (anaemia, coagulation disorders, leucopenia, thrombocytopenia, disseminated intravascular coagulation etc.),
- **Metabolism and nutrition** (hyper catabolism, malnutrition, burn cachexia etc.).

These problems usually do not affect an isolated organ/system only, but usually two or several organs or systems are becoming affected. As skin is also one of the human body organs, extensive burns cause its dysfunction and/or failure. Therefore skin dysfunction and failure shall be considered as one organ dysfunction/failure. If two or more organs are simultaneously affected by dysfunction or failure, the patients will become high risk patients suffering from multiple organ dysfunction syndromes (MODS), or multiple system organ failure (MSOF). MSOF is the most severe life threatening complication of extensive burn injuries. In case of failure of 3 or more organs/systems the expected mortality is 100%, although exceptions still can exist.
6.1.1.4 **MONITORING AND MAINTENANCE OF FLUIDS, ELECTROLYTES AND ACID-BASE BALANCES**

In all intensive care patients the amounts of all fluids intake and output should be measured and summarize at least once a day, or even in shorter time intervals (from 1 hour up to 12 hours). In addition to really measured figures immeasurable fluid losses (such as through the burn wound and by respiration) shall be taken into account when calculating fluid needs of the patients. In case of laboratory findings of increased or decreased plasmatic values of electrolytes, appropriate compensations should be provided. The same applies for maintenance of the acid-base balance of the patients.

6.1.1.5 **ASSURANCE OF THERMAL COMFORT OF PATIENTS**

Severely burned patients are very prone to body heat loss, particularly during therapeutic procedures such as dressing changes, or operations. Therefore the ambient temperature in patient rooms, dressing rooms and operation theatres should be maintained at a level which would not cause patients’ discomfort. Optimal ambient room temperature for such patients should be kept between 30 and 33 °C. If this cannot be achieved, heating blankets or other patient heating devices shall be used.

6.1.1.6 **METABOLIC AND NUTRITIONAL SUPPORT**

All severe burn injuries are accompanied by severe hyper metabolic state of the patients. Their metabolic rates can exceed twice the normal rates of other surgical or trauma patients and cause excessive loss of lean body mass during treatment. Due to these facts appropriate nutritional support of such patients is of utmost importance. Several formulas are used for energy requirement calculations. The best way of patients feeding is the natural one – oral (by conscious patients), or enteral (ventilated and unconscious patients), as it usually keeps the gastrointestinal tract functional. In case of malfunctioning of gastric or enteral passage, or intolerance of enteral feeding, total parenteral nutrition shall be instituted until regular gastrointestinal passage functions will resume.

Adequacy of nutritional support can be monitored by regular patient body weight checks, by nitrogen balance calculations, and by regular serum proteins and serum albumin investigations. There are also some other clinical signs of malnutrition such as delayed wound healing, abnormalities of immune function and susceptibility to infection. Inadequate nutritional support will lead to significant body weight loss. Critical body weight loss of more
than 20% of pre-injury value will increase the occurrence of serious complications and shall be prevented.

6.1.1.7 REHABILITATION

The range of rehabilitation procedures in acute period is quite limited initially. The reasons include shock treatment, non-cooperative patients while in coma and/or ventilated artificially, need of body parts immobilisation following surgical procedures, or painful burn wound and donor site areas. Nevertheless, provision of selected procedures is possible by qualified physical and respiratory therapists such as positioning and splinting of extremities, isometric exercises, gentle range of motion exercises, respiratory therapy in both ventilated and non-ventilated patients etc. In case the procedures provided would cause pain, analgesic medications should be administered. After skin graft will take and progress of wound healing is optimal, the range and intensity of rehabilitation procedures can be increased gradually.

6.1.1.8 PSYCHOLOGICAL SUPPORT

It is important to provide this kind of support both for the patients and for their relatives by psychologist familiar with the burn problem.

6.1.1.9 NURSING CARE

It is an integral part of burn patient’s treatment particularly in patients who are unconscious or heavily sedated during artificial ventilation. The care starts with resuscitation of the patient at admission, continues through emergency, acute and rehabilitation periods and finishes with dismissal of the patient from the hospital care. Nurses are equal partners of physicians and together with other specialists create a well-educated, skilled, and motivated total burn care team.

Very important part of nursing care is prevention of pressure sores in unconscious and artificially ventilated patients. Turning and position changes in these patients are necessary to be provided at least each 2 hours, which is physically and technically very difficult. Due to these reasons special beds (air fluidized beds) which can prevent development and assist in treatment of pressure sores and which would enable better wound areas ventilation have been developed and manufactured. These beds consist of a container filled with 600-700 kg of fine synthetic ceramic sand particles coated by silicone to prevent their sticking to each other. Specially constructed turbines push pre-heated air through the sand particles causing an effect of “fluidization” of the particles. This will result in aeration and permanent movement of the
sand particles as it would be like a fluid fill of the bed (Fig. 7). This way the patient’s body weight is evenly distributed to any square cm of his body in contact with the bed. That prevents increased pressure to body parts with bony prominences which are the body areas most exposed to development of pressure sores. The advantages of these beds include prevention of pressure sores, prevention of wounds maceration, better graft take and optimal thermal comfort of the patients. Their disadvantages are high initial and maintenance costs.

6.1.2 COMPLICATIONS, THEIR PREVENTION AND MANAGEMENT

6.1.2.1 INFECTION

Infection represents a major threat to burn patients. Complications related to infection and its consequences are responsible for 80% of all the mortality causes. The most common origin of infection is the extensive open burn wound area exposed to uncontrolled invasion of microorganisms from outer environment into the human body. It is by great part facilitated by loss of the major part of skin barrier and immunity functions. At the beginning the infection spread starts in the burn wound areas and continues to spread into other organs and systems. The most severe generalized infection originating in the burn wounds presents itself as burn wound sepsis.

Pathophysiology

The burn wound is never sterile, as the normal healthy human skin is usually contaminated by various kinds of microorganisms which persist there without causing any harm to the human body. Violation of both barrier and other skin functions by the burn injury and presence of necrotic tissues in the extensive and open wounds create an ideal environment for growth and multiplication of all kinds of microorganisms. The mean bacterial cell generation time is 20 minutes therefore a single cell can generate within 24 hours more than 10 billion cells. Immunosuppressed status of the patients allows the microorganism to spread rapidly.

Development of burn wound infection, if not controlled, is gradual and progresses by time from the lowest grade - contamination to the highest one – sepsis.

I. Contamination

The thermal insult itself is sufficient to damage or kill skin cells, but does not kill all the bacteria, or spores present in injured skin. Due to this fact the burn wound is considered as primarily contaminated from the very beginning.
II. Colonization

Microorganisms contaminating the burn wound have ideal environment for multiplication and growth, which initially does not show any clinical signs. But they can be detected by taking swab cultures, or even better by quantitative counts of biopsy specimens. Bacterial counts below $10^5$ bacteria per 1 g of tissue represent colonization.

III. Local infection

If bacterial counts will reach or exceed $10^5$ **bacteria per 1 g of tissue**, local infection occurs. It is usually accompanied already by clinical signs such as change in appearance of wound discharge (from serous to cloudy, purulent), pain, redness of wound margins etc.

IV. Locally invasive infection

If local infection is not controlled, it spreads causing more intensive clinical symptoms such as elevated body temperature, tachycardia, more severe inflammatory symptoms, lymphangitis and lymphadenitis. Due to death of previously viable tissue parts the burn wounds can deepen from superficial or partial thickness wounds to deep dermal and/or full thickness wounds. In wound biopsy specimens bacterial invasion along capillaries into healthy tissues can be detected.

V. Generalized infection – sepsis

By uncontrolled invasion of microorganisms into surrounding tissues they can invade into the blood stream as well and in case where the host defence system of the body fails, uncontrolled spread of infection by blood stream to distant body parts occurs. This state is called **septicaemia which can progress to sepsis**. Clinical signs of sepsis include very high (above 39 °C) or low (below 37 °C) body temperature, the patients are very sick, sometimes confused. Other signs are severe tachycardia and unstable circulation, which can progress in shock state (septic shock), or respiratory dysfunction which can progress in respiratory failure requiring ventilatory support. Some clinical symptoms of sepsis can be different depending on its origin, if it was caused by gram positive or gram negative bacteria or by fungi.

In addition to severe clinical symptoms the diagnosis of sepsis needs to be confirmed by blood cultures and laboratory investigations – leucocytes total and differential counts (very high or very low values, prevalence of neutrophils), thrombocytes counts, serum
procalcitonine (PCT) level, serum presepsine level, serum C-reactive protein (CRP) level.

Sepsis, if not controlled adequately, progresses in septic shock, multiple organ and system dysfunction (MOSD) and finally into multiple organ and system failure (MOF) which is the most frequent cause of death due to infection.

**Treatment**

Basic principles of treatment of septic complications should include:

- Immediate start of systemic antibiotic therapy in highest tolerable doses according to causative agents.
- Fluid replacement therapy aimed at restoration of circulatory stability. If the patient’s response is inadequate then adrenergic medications for circulatory and heart functions support should be administered.
- Improvement of tissues oxygenation is necessary by oxygen therapy and/or by intubation and ventilatory support of the patient.
- Detection and eradication of source of infection such as early excision of necrotic tissues, removal of potentially infected intravascular or urinary catheters, eradication of other infection sources (like infected thrombi, endocarditis etc.) should be of utmost importance.
- Nutritional and energy support by early enteral, or parenteral nutrition
- Support of immune functions by administration of immunoglobulins, fresh frozen plasma and immunomodulators.
- In case of adrenocortical dysfunction short-term administration of corticosteroid hormones should be necessary.

**Other potential sources of infection**

In addition to burn wound as the main infection source other sources can include:

- bronchopneumonia,
- vascular catheter infections, purulent thrombophlebitis,
- chondritis,
- sinusitis,
- urinary tract infections,
- intraabdominal infections enteritis, enterocolitis, acute cholecystitis,
- infected intravascular thrombi,
- bacterial endocarditis,
- purulent joint infections.

### 6.1.2.2 Inhalation Injury

It is defined as acute damage to the respiratory tract caused by inhalation of hot gases or vapours, or of toxic products released from different burning materials. Occurrence of inhalation injuries does not need to be accompanied by body surface cutaneous burns.

**Pathophysiology**

Inhaled hot air cools down in upper parts of respiratory tract which are limited by the level of tracheal carina. Heat accumulation capacity of heated water vapours is 4000 times higher than that of dry air and this can cause damage to the lower parts of the tract. Around 80% of deaths in house fires are caused by inhalation of toxic products of burning materials; the most dangerous of these products include carbon monoxide (CO) and hydrogen cyanide (HCN). Binding capacity of haemoglobin to CO is 200 times higher than to oxygen. This means, than lethal intoxication by CO can occur at its concentration in the air as low as 0.2%.

There are 3 types of inhalation injuries:

- Upper airway injury characterized by oropharyngeal and mucosal damage accompanied by massive swelling and major threat of their rapid obstruction,
- Bronchial and pulmonary injury,
- CO intoxication alone or combined with intoxication by other toxic products of fires.

The three inhalation injury types can be also combined.

Respiratory mucosal damage can vary from minimal changes such as erythema through respiratory epithelium damage up to complete mucosal necrosis. These changes are usually accompanied by oedema which can lead to obstruction of parts of the bronchial tree, or alveolar atelectasis. Delayed consequences of initial damage to lower respiratory tract are usually caused by secondary infection of the damaged bronchial and lung tissues and/or secondary pulmonary fibrosis.

**Diagnosis**

It is evident, that inhalation injuries combined with cutaneous burns increase significantly both the morbidity and the mortality of such injuries. In fatal injuries they shorten the average
survival time. In order to reduce the consequences and improve the outcome of inhalation injuries early detection, diagnosis and treatment of such injuries is important.

Inhalation injury should be suspected if:

- **Flame or explosion burns** occurred in enclosed spaces, or inhalation of overheated water vapours occurred.
- **There are clinical signs of hot gases or smoke inhalation present** such as burns of the respiratory areas (nostrils, around the mouth, Fig. 8), singed nasal vibrissae, hoarseness, dry cough, carbonaceous sputum, respiratory distress or dysfunction, impaired sensorium, disorientation, or consciousness loss in absence of craniocerebral injuries.
- **COHb** levels in blood exceed 20%. It should be kept in mind that normal pulse oximetry oxygen saturation levels can be falsely normal by severe CO poisoning!

Diagnostic tools:

- **Laryngoscopy** with inspection of upper airways for presence of smoke particles or mucosal damage,
- **Fiberoptic bronchoscopy** is one of the most reliable objective diagnostic tools,
- **Xenon$^{133}$ lung scan**, eventually together with bronchoscopy. The scan needs special equipment which is not routinely available,
- **Pulmonary function testing**: not possible in unconscious and ventilated patients.

**Clinical course of inhalation injury**

There are four phases:

1. **Latent (subclinical phase)**

   In case of massive inhalation injury this phase is absent as there are clinical signs present immediately following injury.

   Duration of this phase is several days (2 to 5 days). In mild injuries the patient can recover during this time, in more severe ones it progresses to next phase.

2. **Early respiratory symptoms**

   There are symptoms of irritation, hoarseness, productive cough with carbonaceous sputum. In cases where oedema of the larynx starts to develop stridor is its first symptom. Oxygenation is usually not compromised in this phase.
3. **Adult (ARDS) or infant (IRDS) respiratory distress syndrome**

First symptoms include increased respiratory frequency (tachypnea) accompanied by hypoxia which can be leading to progressive respiratory distress and respiratory failure. Arterial blood oxygen saturation will be decreasing and accompanied by symptoms of hypercapnia and respiratory acidosis. Interstitial pulmonary oedema will present itself on x-ray as a “snow storm” picture.

4. **Late infectious complications**

They can develop slowly during several days or even weeks and present themselves under clinical picture of bronchopneumonia. Its development is facilitated by damage to natural pulmonary defence mechanisms such as pulmonary mucosal destruction, impaired motility of ciliary epithelium, surfactant deficiency or loss, and interstitial oedema.

6.1.2.3 **OTHER COMPLICATIONS**

**Acute phase** other complications are usually caused by initial barotrauma (injuries caused by explosion), or complications associated by iatrogenic manipulations (central venous cannulation) and artificial ventilation such as pneumothorax, atelectasis, pulmonary oedema, pneumomediastinum or subcutaneous emphysema.

**Late complications** in addition to infection (see above) usually include airway epithelium erosions, tracheal rings erosions, tracheomalatia, bronchiecathasis or bronchial stenosis, tracheooesophageal fistula, erosion of blood vessels with subsequent hemorrhage (very often of innominate artery), pulmonary obstructive disease, or fibrosis.

**Treatment**

Initial therapy should aim to prevent airways obstruction by preventive intubation, oxygen administration and initiation of artificial ventilation by moisturized and oxygen enriched air. The patient shall be admitted to intensive therapy department and his/her vital signs and function shall be monitored. Routine preventive administration of corticosteroids and antibiotics is not recommended. Simultaneously all the other therapeutic measures such as fluid replacement therapy, intensive wound care and nursing care shall be provided.

6.1.3 **BURN WOUND CARE**

It is a very important integral part of comprehensive burn care. Wound care shall be provided from the very beginning phase of the burn trauma through all its phases and will continue
even after all the burn wounds have been healed. Before the patients will be discharged from the hospital they need to be instructed about the after care and are advised to come regularly for follow-up examinations and treatment recommendations. Burn wound care is included in the holistic approach to the patients and their wounds and requires good knowledge of the burn wound pathophysiology and its healing mechanisms. Of utmost importance is individual approach to each patient depending on patient’s and his/her wounds conditions and qualified decisions about necessity of surgical procedures, use of topical agents or devices (coverage/closure materials), coverage/closure and bandaging/splinting techniques and pain management during all these mostly very painful procedures.

6.1.3.1 WOUND HEALING

Growth, regeneration and reparation are physiological processes which lead to tissue repair. Normal postnatal healing includes processes of both regeneration and reparation. The healing process itself requires a coordinated cooperation of different cellular activities such as phagocytosis, chemotaxis, mitogenesis, angiogenesis and synthesis of collagen and extracellular matrix. Most of the events participating in the healing process play active roles by the process of skin graft take as well.

Pathophysiology

There are four basic normal (physiological) healing types described in clinical practice:

- Primary healing (i.e. healing by first intention),
- Delayed primary healing,
- Secondary healing (i.e. healing by secondary intention),
- Healing of superficial wounds.

Healing of skin grafts is a special type of healing.

Besides physiological healing there are also pathological healing types where several pathologic conditions, systemic diseases (such as diabetes, collagenosis, malnutrition etc.), congenital anomalies, or medicaments (corticosteroids, immunosuppressive drugs etc.) would influence the normal healing process usually by its retardation or switch to chronic non-healing wound.

John Hunt in 1987 defined healing as a continual sequence of signals and responses, where epithelial, endothelial and inflammatory cells, thrombocytes and fibroblasts come together
temporarily outside of their normal domains, interact together, restore their original discipline and finally return to their original roles and activities.

**Importance of the character of the original injury**

For example, clean surgical incision with meticulous primary wound closure in anatomical layers initiates a minimal tissue response which results in rapid healing within a couple of days and with minimal scarring. On the other side, extensive deep burns with large amounts of necrotic cells and tissues trigger a massive both localized and generalized reaction in the human body which result in prolonged healing, threat of major complications and in cases where the patients manage to survive, the end results are massive scars with permanent both esthetic and functional impairments.

**Final result of the healing process**

From the final results viewpoint there are two basic types of healing:

**Regeneration**

It means complete restoration of shape, appearance and function of the injured part or body area. The consequences are no permanent scars and/or long-term functional impairments. Unfortunately, in humans this can occur only in superficial wounds (abrasions, superficial burns) affecting the epidermis and papillary dermis only.

**Reparation**

It includes incomplete restoration of shape, appearance and function resulting in scarring of the injured body areas and/or parts.

**Components of the wound healing process (Hunt 1990)**

Although wound healing is a continuous sequence of events, it consists of several components which interlock to each other. The components are as follows (Tab. 8):

- Coagulation and inflammation,
- Angiogenesis,
- Fibroplasia,
- Matrix deposition,
- Epithelialization,
- Contraction,
- Remodelation.
Coagulation

Damage to capillary endothelium at the site of injury causes loss of its anticoagulation properties. This is followed by adhesion and activation of platelets (thrombocytes). Activated platelets initiate two important processes:

- **Coagulation** which causes formation of thrombocytes clot and initiation of blood clot formation,
- **Platelet degranulation** resulting in release of platelet derived growth factor (PDGF) and other cytokines.

Blood clot (coagulum) is composed of polymerized fibrin and blood cells. Growth factors and cytokines released from the thrombocytes attract and activate fibroblasts and macrophages to the injury site. Fibrin fibers serve as guides for cells migration to the wound. In addition to fibrin also fibronectin (a binding protein) is contained in the coagulum.

Inflammation

It is initiated by tissue injury and is triggered immediately after it occurs. The inflammatory process will reach its peak one to two days post injury and thereafter is gradually replaced by reparatory processes. Early vascular changes enable exudation of protein rich plasma and migration of leukocytes into injured tissues. During next several days inflammatory cells migrate gradually to the injury site and will take over control of the healing processes. First line cells which appear there are granulocytes (neutrophil leukocytes) capable of releasing different intracellular substances and killing bacteria. They are followed by macrophages which are the key cells of the healing process. Macrophages activated by platelet and leucocytes derived products release different intracellular products such as cytokines and growth factors. By phagocytosis they are capable to ingest bacteria, devitalized cells and tissues. During next few days lymphocytes as the third line inflammatory cells infiltrate the injury site. They produce lymphokines which can modulate the healing process as well, although their role is not understood completely. Endothelial cells represent a selective permeability barrier between the blood stream and tissues. They can also produce and release products which inhibit coagulation. At the end of this phase a variety of different cells capable to eliminate dead tissues and cells, to combat bacteria and to modulate the process of healing have infiltrated the injury site. After they clean the injury site from debris, necrotic tissues and bacteria, their cellular products activate other cells which will take over reparatory processes including angiogenesis, fibroplasia and matrix deposition.
Tab. 8 Survey of the wound healing process

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Cells</th>
<th>Cell products</th>
<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 h</td>
<td>Coagulation</td>
<td>Thrombocyte</td>
<td>PDGF, TGF beta, Tr. factor IV</td>
<td>Aggregation of Tr., degranulation, clot formation, ↑ permeability, cells attraction</td>
</tr>
<tr>
<td>0-48 h</td>
<td>Early inflammation</td>
<td>Neutrophils (N)</td>
<td>Enzymes, free radicals</td>
<td>neutrophil margination, diapedesis, phagocytosis, killing bacteria</td>
</tr>
<tr>
<td>48-72h</td>
<td>Late inflammation</td>
<td>Macrophages (MF)</td>
<td>Growth factors (GF), cytokines</td>
<td>Phagocytosis, triggering and regulation of processes</td>
</tr>
<tr>
<td></td>
<td>Angiogenesis</td>
<td>Endothelial cells, Thrombocytes Fibroblasts</td>
<td>Endothelial GF, TGFβ, PDGF, bFGF, TNFα</td>
<td>Endothelial cells proliferation, formation of new capillaries</td>
</tr>
<tr>
<td>72h&lt;</td>
<td>Fibroplasia</td>
<td>Fibroblasts (F)</td>
<td>Growth factors, Collagens, GAG Matrix proteins</td>
<td>Wound filling by granulation tissue and connective tissue, creation of space for ingrowth of capillaries, fibroplasia</td>
</tr>
<tr>
<td></td>
<td>Matrix deposition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Epithelization</td>
<td>Keratinocytes</td>
<td>bFGF, KGF, EGF, keratin</td>
<td>Mitosis &amp; Epithelial cells migration from the wound edges</td>
</tr>
<tr>
<td>5&lt; days</td>
<td>Contraction</td>
<td>Contractile fibroblasts</td>
<td>??</td>
<td>Collagen fibers contraction, wound size reduction, scar formation and hypertrophy</td>
</tr>
<tr>
<td>3&lt; weeks</td>
<td>Remodelation</td>
<td>Fibroblasts</td>
<td>Colagenases</td>
<td>Scar atrophy, vascularity reduction, scar softening</td>
</tr>
</tbody>
</table>

**Abbreviations:** Thrombocytes (Tr.), PDGF – platelet-derived growth factor, TGF – transforming growth factor, GAG – glycosaminoglycans, GF – growth factor, bFGF – basic fibroblast growth factor, EGF – epidermal growth factor, KGF – keratinocyte growth factor, TNF – tumor necrosis factor

**Angiogenesis**

Endothelial cells start to proliferate from severed ends of capillaries forming buds which are recanalized gradually. This process is stimulated and regulated by angiogenetic factors released from cells.

**Fibroplasia**

It is initiated by inflammation and starts by principal connective tissue cells (fibroblasts) migration along newly formed capillaries. Fibroblasts synthesize collagen and extracellular matrix proteins. A mixture of newly formed capillaries collagen and extracellular matrix gradually fills the injury site where tissue loss occurred and is called granulation tissue.
Matrix deposition

Main components of intercellular matrix include collagen fibres network with gaps filled by intercellular matrix. Collagen fibers assure mechanical strength of the newly formed tissues, the newly synthesized matrix fills the gaps between the fibers and the capillaries and enables migration of cells in the newly formed tissues. Elastic fibers do not regenerate, therefore the final scars are hard and not elastic initially.

Epithelialization

It represents the final stage of the wound healing process and triggers signals for cessation of new tissue formation. Migration of epithelial cells is triggered by filling the tissue defect by granulation tissue. The cells start to migrate from healthy skin surrounding the wound. The proliferating cells move by sliding from the wound edges over the top of the granulations and by excreting binding proteins (fibronectin) attach and fix themselves to the wound bed. Other new cells then slide over the fixed cells and attach again to the wound bed. This process continues from the wound edges to the center of the wound until the new epithelium will cover the entire wound area. Sources of the cells are mitotically active zones at the wound edges, or other epithelial remnants (islands) which survived at the wound bed in skin appendages such as hair follicles, sebaceous and sweat glands. Epithelialization is enhanced by moist wound environment and by addition of some wound healing factors like TGFβ, EGF, PDGF, IGF-1 (insulin-like GF) etc. The new epithelial coverage is very fragile initially, as it is very thin and does not contain anchoring fibers which fix the epithelium to papillary dermis in normal skin. Papillary dermis is absent in scars, therefore this newly formed fragile epithelial cover has got the name „scar epithelium“.

Contraction

Newly formed collagen in the scar is organized such as it would attract the wound periphery to its center by forces stimulated by resident fibrous tissue cells – fibroblasts. These fibroblasts possess some contractile features resembling to those of muscle cells (myocytes), therefore they have got the name „myofibroblasts“. The final result of scar contraction is called contracture which can lead to deformations of the body parts and limitations of joints movements.
Remodelation

Following complete wound closure and scar formation the healing process does not stop immediately, but continues by simultaneously ongoing processes of new collagen formation (collagen synthesis) and collagen degradation (collagen lysis). If these two processes are balanced, the resulting scar is going to become softer by time and its appearance improves. If there is predominance of collagen synthesis, the scar becomes thicker, prominent, red and tender. This kind of scars is called „hypertrophic scarring“ and is quite common particularly following prolonged healing of deep burns. On the other side, if there is prevalence of collagen lysis, the healed wounds can reopen again with subsequent retardation of healing.

The process of healing is not the same in all kinds of wounds. For example superficial wounds (superficial burns, abrasions, shallow lacerations) involving the epidermis and papillary dermis only heal by epithelialization only without fibroplasia and without any permanent scarring. Deep structure wounds (closed bone fractures, ligament tears, muscle tears etc.) will heal by new bone formation (ossification) or fibroplasia without any epithelialization. Healing of extensive, deep, open wounds (such as extensive burns, tissue losses etc.) involve all the components of the wound healing process and result in massive scar formation and compromised body part functions.

Growth factors and cytokines

Proliferation and many other activities of cells are in most of the situations regulated by extracellular agents – growth factors and cytokines. Growth factors can be either products of different endocrine glands (hormones), but they can be produced and excreted by different cell types as well. Cytokines are products of different cell types such as platelets, fibroblasts, endothelial, epithelial or other cells. They have a wide range of biological activities and mostly serve as intercellular messengers enabling cell to cell communications or serve as regulators or modulators of different cell functions. The effects of these agents are dependent on their capability of binding to appropriate receptors which are parts of target cells cellular membranes. Interaction of growth factors/cytokines with corresponding receptors transmits the extracellular signals into the target cells.

SKIN GRAFTS AND THEIR HEALING

Free skin grafts are parts of skin with different thickness which are detached (cut off, procured) from one body part (donor site) and transferred (transplanted) to other body part (recipient site) in order to replace the skin which was lost or severely damaged. Skin grafts are
composed of epidermis and parts of dermis. According to thickness of the dermis contained in the grafts, two types of skin grafts are recognized:

- **Full thickness grafts** contain epidermis and the entire dermis,
- **Partial thickness (or split thickness) grafts** contain parts of dermis with different thickness – thin grafts contain up to one third thick dermis, medium thickness grafts contain one half and thick split thickness grafts contain up to three quarters thick dermis.

The dermal component is a very important part of the grafts, as it contains collagen fibres which add mechanical strength and blood vessels (capillaries) assuring nutrition of the skin.

**According to the skin source there are three types of skin grafts:**

- **Autografts** – they are grafts procured and transplanted at the same individual. By autografting there are no immunological barriers and survival of the transplanted skin is permanent.
- **Allografts** – they are grafts procured from one individual and transplanted to other genetically different individual of the same species (like human to human, or pig to pig etc.). As immunological systems of different individuals are different allergenic tissue can heal in only temporarily and later will be rejected.
- **Xenografts** – they are grafts transplanted between different species such as animal tissue to humans, or mice to rats etc. Xenogenic tissue is not able to revascularize in recipients due to primary rejection.

**Healing of autografts** (graft take) occurs by revascularization which means establishing vascular connections from the recipient site vascular network with the grafted skin dermal vascular network. This will require 5 to 7 days’ time and good permanent contact of the graft with the wound bed. Following revascularization fibroblasts at the transplantation site will synthesize collagen which will anchor the graft securely to the recipient site (Tab. 9).

Graft take can be compromised by insufficient immobilization, poor vascularity of the wound bed and by presence of infection.

**Summary**

Wound healing is a unique process where a great range of biological events and mechanisms participate. Description of the process of healing itself can never be exhaustive enough as new events, factors and mechanisms contributing to this process are being to be discovered day by day. The most important issue would be to acquire enough knowledge of basic wound healing
and graft take principles to an extent which will then enable their application in everyday clinical practices for benefit of the patients.

**Tab. 9 Skin graft revascularization**

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Clinical signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>24 hours</td>
<td>Absorption of intercellular fluid by graft capillaries, fibro elastic bond between the wound bed and graft by fibrin, fibronectin and integrins</td>
<td>Pink colour of graft, it can be easily lifted from the wound bed</td>
</tr>
<tr>
<td>48 hours</td>
<td>So called “plasmatic imbibition”, capillaries filled by plasma like fluid</td>
<td>Grafts are pale, they adhere better to the wound bed</td>
</tr>
<tr>
<td>Days 2-5</td>
<td>Start of vessel ingrowth, venous connections establish first, initiation of fibroplasia</td>
<td>Livid colour of graft, slow capillary refill following compression</td>
</tr>
<tr>
<td>Days 6-7</td>
<td>Complete vascular connection, start of capillary circulation, progression of fibroplasia</td>
<td>Pink colour, good capillary refill</td>
</tr>
<tr>
<td>Day 7 &lt;</td>
<td>Improved graft adherence secured by part by newly formed collagen</td>
<td>Pink colour, detaching of grafts more difficult, capillary bleeding if grafts are detached</td>
</tr>
<tr>
<td>Day 14 &lt;</td>
<td>Firm fibrous bond of grafts, reduced blood flow</td>
<td>Firm adherence of grafts to wound bed, detaching hardly possible, natural colour</td>
</tr>
</tbody>
</table>

### 6.1.3.2 BURN WOUND CARE IN THE ACUTE PERIOD

Burn wounds represent extensive open wound areas which are devoid of skin barrier, they are painful, they discharge great amounts of fluids, proteins etc. and they contain great amounts of cell debris and necrotic tissues. This environment offers excellent conditions for surviving and growth of a great variety of microorganisms and for rapid development of burn wound infection threatening the lives of the burn victims.

Burn wound is **contaminated primarily** as a great variety of microorganisms reside normally on healthy skin surface. The thermal insult is capable of damaging or even destroying large areas of the skin along with its functions, but is usually not sufficient to kill all the residential microorganisms present there at the moment of injury. After the skin defensive barrier is damaged or lost, the burn wound surface offers ideal environment for development of infection originating in residential skin bacteria, bacteria residing in natural orifices of the human body such as anus, oral cavity, respiratory tract etc. **Secondary contamination** of the burn wound occurs by bacteria from the outer environment or by subsequent manipulations with the burn wound.
Characteristic properties of the burn wound can be described as follows:

- Open, extensive, painful, longer time persisting wound;
- Extensively damaged or destroyed mechanical and biological defence barrier of the human body;
- Uncontrolled losses of fluids, electrolytes, proteins through the open wound surface;
- Extensive evaporation of wound discharge water through large open wound area associated with continuous heat loss and disturbances of thermoregulation;
- Extensive open wound surface capable of absorbing various toxic products including topically applied medications and disinfectants as well;
- Excellent nutrient broth for growth and multiplication of microorganisms.

Such a wound, if we simplify the problem, can be compared to a huge sieve, blotting paper, oven and culture plate with all their consequences to the human body. The major threat to the patients is burn wound infection leading to great range of complications. It can be partially prevented by meticulous wound care and topical therapy.

Primary hospital management of the burn wound is described in part 4.11 (Page 40)

Subsequent wound care consists of:

- regular dressing changes with simultaneous assessment of the wound healing progress, debridement of the wounds and deciding upon indications for surgical debridement, wound coverage, closure and type of topical therapy,
- Application of topical medications, coverage materials, skin substitutes etc.,
- Regular sampling of swabs for bacteriology (at least twice a week),
- Positioning or splinting of the affected body parts.

Main goals of topical therapy of the burn wound

Main aim of topical therapy is to remove all the devitalized tissues from the burn wound as soon as it is possible, to cover or close the extensive open wound, to prevent or combat infection and to achieve regeneration, or at least reparation of the body surface and of the destroyed skin functions. As persistence of the large open wound during a prolonged period of time has deleterious effects to the human body and to the progress of the burn disease, early wound closure and healing cannot just reduce the total healing time, but they can also prevent the occurrence or worsening of a wide range of complications which increase
substantially morbidity and mortality of the burn patients, prolong their wound healing time and increase the probability of long-term sequelae of such injuries.

Other principal goals of topical therapy include:

**Superficial burns:**
- Reduction and elimination of wound pain and discharge from the burn wound,
- Burn wound infection prevention/treatment,
- Assurance of thermal comfort of the patients,
- Reduction of pain associated with wound manipulations and dressing changes,
- Assurance of optimal conditions for wound healing by epithelialisation.

**Deep burns** (in addition to previous ones):
- **Early removal of necrotic tissues** by surgical excision, or other methods,
- **Coverage** (temporary) or **closure** (permanent) of the open wound areas by skin substitutes, or skin grafts.

6.1.3.3 **TOPICAL ANTIBACTERIAL AGENTS**

The extensive deep burn wounds with most of the host defence mechanisms impaired or destroyed are exposed to outer environment practically without any defence against microorganisms. Vascularity of deep burn wounds is reduced considerably, or absent in necrotic areas. Systemically applied medications (like antibiotics) are distributed throughout the human body by blood stream; therefore they cannot reach the avascular parts of the body in efficient concentrations. This is the reason, why preventive systemic administration of antibiotics is not effective enough in preventing burn wound infection. The role of topical antibacterial agents which are applied directly on the wound surface and have to be in close contact with the burn wound is to deliver the medications directly to the areas most vulnerable for development of infection. This is why their use is indicated in all burns deeper than superficial partial thickness burns (i.e. II\textsuperscript{nd} a. degree). There is a wide choice of topical antibacterial agents from disinfectants, to topical antibiotics, other antimicrobials (such as silver – Ag) or wound healing devices with antibacterial properties.

The categories of these products are listed below.
Creams and ointments (mostly used)

- **Silver sulfadiazine cream 1%** (Dermazin®, Flammazine®, Silvaden®). It is a white water soluble cream containing a combination of chemotherapy agent – sulfadiazine and silver with wide spectrum antimicrobial activities against both gram positive and Gram negative bacteria in all kinds of burns.

- **Silver sulfadiazine cream with hyaluronan** (Ialugen plus®). It contains the same antibacterial agent combined with low molecular weight hyaluronic acid which assists in the wound healing process.

- **Mafenide acetate cream 11%** (Sulfamylon®). It contains sulphonamide acting topically against particularly gram negative bacteria (like Pseudomonas aeruginosa). It was developed for use in extensive deep burns.

- **Silver sulfadiazine cream with cerium nitrate** (Flammacerium®) contains also cerium nitrate which exhibits immunomodulatory action and helps to harden the necrotic tissues.

- **Neomycin + Bacitracin ointment** (Framykoin®, Baneocin®) contains topical antimicrobials for short term treatment of minor burns.

- **Mupirocin ointment** (Bactroban®) contains topical antibiotic effective against Gram positive bacteria with same indications as the previous one.

- **Fusidic acid ointment** (Fucidin®) is also effective against Gram positive bacteria.

Solutions (examples)

- **Furadantin 0.2% solution** contains topical chemotherapeutic agent and is used for soaking and wet compresses of mildly infected burn wounds.

- **Polyvinyl pyrolidone iodine** (Betadine®, Betaisodona®) is an iodine based wide spectrum skin disinfecting solution. It can be used also for wound treatment.

- **Silver nitrate 0.5% solution** (AgNO₃) is used for wetting gauze compresses and it has wide spectrum antibacterial action due to content of metallic silver ions.

- **Octenidyl dihydrochloride 0.1% solution** (Octenisept®) is a water-soluble skin and wound disinfecting agent. It acts against bacteria, fungi and some viruses as well.

- **Acetic acid 1% solution** is prepared under aseptic conditions by hospital pharmacy. It can enhance action of some antimicrobials particularly against Pseudomonas strains.
6.1.3.4 **TOPICAL NECROLYTIC AGENTS**

They include chemicals with ability to speed up demarcation and autolytic debridement of necrotic tissues and proteolytic enzymes which can dissolve necrotic tissues selectively.

**Chemical necrolysis**

Most commonly used chemicals with necrolytic action which do not harm healthy tissues are salicylic acid and benzoic acid. Depending on the necrotic layer thickness, they need some days or even one week until the necrotic tissues will slough off. They must not be applied to fresh burn wounds, as this can cause damage to healthy tissues. Their application is also contra indicated to massively infected wounds, as this can lead to progression of infection. They cannot be applied to surfaces exceeding 10% of the body surface area, as this can lead to systemic toxicity. Their disadvantages include systemic toxicity, slow action and their use possible in later phases of treatment only.

- **Benzoic acid 20% in white Vaseline base** is prepared in hospital pharmacies and applied directly to necrotic areas of the wound.
- **Salicylic acid 20%** is the same base is used the same way.
- **Salicylic acid 40%** is used the same way as well.

**Enzymatic necrolysis**

It is provided by application of proteolytic enzymes capable of dissolving the devitalized tissues. In the past there have been used some kinds of enzymes containing mostly trypsin with little efficacy.

- **Bromelain based enzyme mixture (NexoBrid\textsuperscript{TM})** is a very recently discovered enzymatic debriding agent which can completely change the philosophy of deep burns treatment. The debridement by NexoBrid\textsuperscript{TM} can be started within a few hours post injury, it is minimally invasive (can be done in the office, or at the bedside), very rapid (4 hours application time), selective and with negligible or no blood loss. It does not do any harm to viable tissues. One of the disadvantages is pain, but it can be controlled by analgesic medications.

6.1.3.5 **SURGICAL TREATMENT**

At the present time they represent the treatment of choice for almost all deep burns. One of the basic principles of surgery is:
“Remove the necrotic tissues from the body as soon as it is possible!”

In deep burn treatment it remained neglected for centuries until surgeons in early 60’s and 70’s of last century started by early deep burn wounds excisions and coverage. The pioneers of this method have been Donald Jackson from Birmingham in the UK and Zora Janzekovic from Maribor (former Yugoslavia, now Slovenia).

There are several techniques of surgical burn excision, the most commonly used are:

- **Formal (fascial) excision** of necrotic skin together with subcutaneous tissue down to the deep fascia (if it is healthy) or even going to deeper layers (Fig. 9).

- **Tangential excision** is excision of necrotic tissues by broad transplantation knives or dermatomes slice by slice until healthy tissue is reached (Fig. 10).

This kind of procedures in extensive burns can be done only in specialized burn centres with good technical and personal background. Adequate equipment, qualified and well trained surgical staff, and good anaesthesia and intensive care services are the basic conditions for providing such procedures. The operations are associated by large losses of blood. The excised wounds following haemostasis cannot be left exposed to outer environment, or covered by traditional dressings only.

### 6.1.3.6 Skin Substitutes

The excised burn wounds need to be either covered by some kind or temporary coverage which includes a wide choice of skin substitutes, or closed permanently by autologous skin grafts (Fig. 11), or combined methods of allogenic and autologous skin grafting (Fig. 12, Tab. 10).

Skin substitutes are commonly used as well in superficial burns for temporary coverage. Their advantages in these situations include diminution of pain, protection of the exposed deeper skin structures against the outer environment, elimination of pain during dressing changes, reduction of fluid losses through the open wound surfaces, assurance of optimal environment for the process of healing and reduction of dressing changes frequency.

Under certain conditions the burn wounds can be treated by exposure – open treatment method. This can be applied mostly to superficial burns in exposed body areas. All the extensive and deep burns and burns following surgical interventions (excision, coverage/closure) should be covered by appropriate dressing types – closed treatment method. The
dressings should be changed in regular intervals depending on the wound character, method of treatment, presence of infection and wound healing phase.

**Tab. 10 Skin Substitutes**

(Modified according to Pruitt, 1997)

<table>
<thead>
<tr>
<th>TEMPORARY</th>
<th>PERMANENT</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Biological</strong></td>
<td></td>
</tr>
<tr>
<td>Allograft</td>
<td>Autografts</td>
</tr>
<tr>
<td>Engrafts</td>
<td>Cultured autologous keratinocytes</td>
</tr>
<tr>
<td>Foetal membranes</td>
<td>Dermal composites seeded by autologous cultured cells</td>
</tr>
<tr>
<td>Acellular allergenic dermis</td>
<td></td>
</tr>
<tr>
<td>Allergenic cultured keratinocytes</td>
<td></td>
</tr>
<tr>
<td><strong>Synthetic and biosynthetic</strong></td>
<td></td>
</tr>
<tr>
<td>Synthetic bilaminates</td>
<td>Integra&lt;sup&gt;®&lt;/sup&gt;</td>
</tr>
<tr>
<td>Collagen based composites</td>
<td>Matriderm&lt;sup&gt;®&lt;/sup&gt;</td>
</tr>
<tr>
<td>Biobrane&lt;sup&gt;®&lt;/sup&gt;</td>
<td></td>
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<tr>
<td>Dermagraft TC&lt;sup&gt;®&lt;/sup&gt;</td>
<td></td>
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<tr>
<td>Apligraf&lt;sup&gt;®&lt;/sup&gt;</td>
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</tbody>
</table>

**6.1.3.7 DRESSING**

Wounds treated by closed method are covered by dressings. An ideal dressing should assure an environment enabling rapid course of the healing process in the wound resulting in early wound closure with acceptable aesthetic result.

The following conditions are required for optimal wound healing:

- Moist, but non – macerating environment,
- Infection control,
- Absence of toxic chemicals or fibres released from dressing materials,
- Assurance of optimal temperature for the healing process,
- Healing process will not be disturbed by frequent dressing changes,
- Optimal pH (slightly acidic),
- Good adherence to the wound surface,
- Absence or reduction of pain,
- Good capacity for wound discharge absorption,
- Good barrier functions,
- Transparency enabling visual control of the wound surface,
- Easy and painless application,
- Easy and painless removal,
- Easy storage.

As some of these requirements are quite controversial, an ideal dressing does not exist so far. There are several dressing types which are close to these criteria, but researchers still look for an ideal compromise.

**Classic dressing** shall have sterile components and should be composed of the following layers:

- **Contact layer** is the layer immediately contacting the wound surface. Its roles include assurance of free derivation of wound discharge from the wound surface and it should not stick firmly to the wound surface enabling removal by minimal or no pain. The most commonly used contact layer in the past was Vaseline gauze (tulle gras). It is still widely used in the present, but there are several kinds of new materials with added non-adherent and anti-bacterial properties.

- **Suction layer** – it is in direct contact with the previous one and its role is to derive the wound discharge to the next layer – absorption layer. It can be also impregnated by some kind of active substances, preferably by antibacterial activities.

- **Absorption layer** – it should have enough capacity to absorb large amounts of fluids in order to prevent their leakage to outer environment. It contains either cotton wool, or multiple gauze layers, or several layers of non-woven synthetic textile materials with good absorption capacity. In wounds with large amounts of discharge odours absorbing substances can be added as well.

- **Covering layer** – it should have barrier properties preventing leakage of fluids discharged from the wounds to outer layers of the bandages.

- **Fixation layer.** Fixation is provided by simple cotton, tube, or elastic bandages. They hold all the dressing layers together and prevent their displacement. In cases where immobilization of body parts is necessary, various kinds of splints can be used.

**Wound dressings following surgical procedures**

The deep burn wounds following surgical excisions are covered by temporary skin substitutes, or closed by skin autografts. Skin substitutes and/or grafts are covered by non-adherent contact layer followed by moistened gauze layer and topical antibacterial agents. Absorption
layer is usually represented by cotton gauze with cotton wool on its top. The dressing is fixed (compressed) by elastic bandages.

First dressing change with wound inspection is provided on day 2 after surgery when the outer layers of the dressing are changed leaving the contact layer on the wound. If the wound is healing without any complications, the following dressing changes should be done each 2\textsuperscript{nd} or 3\textsuperscript{rd} day. Revascularization of autografts occur 5 to 7 days post application. During this time the grafted area should be immobilized. If there are signs of local infection, the wounds should be re-dressed more frequently in order to remove excessive discharge. More firm – fibrous attachment of the grafts to the wound bed occur after two to three weeks.

6.1.4 SPECIAL BURNS CARE

6.1.4.1 PAEDIATRIC BURNS

Paediatric burns represent around one third of all burn injuries. The majority of them occur in age groups from 0 to 2 and from 2 to 4 years. Children’s organisms differ by several characteristics compared to adults and are not just reduced copies of them.

Physiologic differences of children’s bodies

Water content of children´s bodies is higher than of adults´ and varies between 50 to 60% of their body mass. One third of their total body water is in extracellular and two thirds in intracellular spaces. The younger the child is the higher is the rate of its extracellular water and the lower is its capacity to regulate it. Due to these reasons the lowest age groups of children (i.e. 0 to 4 years) are both dehydrated and over hydrated much easier, than older children and adults. They have also very limited physiological reserves as well. Physiologic circulatory volume is in children 80 ml per kg body mass, whereas in adults it is 60 ml per kg body mass. For example, burns of the same body surface area (20%) will cause in a child with 10 kg body mass reduction of as much as 60% of its circulatory volume, whereas in an adult with body mass of 70 kg only 25% of its circulatory volume.

Burn severity assessment in children is provided by the same system as in adults (see Tab. 4, p. 27) the only difference is, that the rule of 9 (Wallace-Pulaski) is not applicable for estimation of children’s´ body surface area burn percentage. The same applies for the Lund-Browder table, which needs to be modified for children according to their age (Fig. 5). Children’s skin is in average much thinner than that of adults, therefore the burn insult of the same intensity causes deeper burns in children.
Criteria for hospital admission for burned children are different as well and they are as follows:

- Children aged up to 3 years with TBSAB of 7% and more of BSA,
- Children aged more than 3 years with TBSAB of 10% and more of BSA,
- Children with deep burns affecting 2% and more of the BSA,
- Smaller TBSAB located in face, hands, feet and perineum,
- Electrical burns,
- Burns where inhalation injury is suspected,
- Burns in children with other severe co-morbidities.

Principles of first aid, emergency management and primary hospital management are the same as in adults. One need to keep in mind, that the start of fluid replacement therapy shall be done without any delay due to much higher sensitivity of children’s bodies to circulatory volume loss and subsequent hypovolaemia. On the other side, children are also much more sensitive to fluid overload; therefore the titration of fluid substitution according to patients’ response shall be more precise in children than in adults. Randomized controlled studies have shown that early and adequate provision of fluid resuscitation can reduce significantly the occurrence of serious complications as well as the mortality in paediatric burns.

Wound care, topical therapy and surgical treatment are provided by the same methods as in adult burns.

6.1.4.2 ELECTRICAL INJURIES

Pathophysiology

There are two current types – direct current and alternating current. Injuries caused by direct current are quite rare and cause mostly non-extensive local tissue damages. Alternating current injury, particularly of low voltage is life threatening. Currents with low intensities (from 30 to 200 milliamperes; mA) can cause ventricular fibrillation of the heart. If the intensity exceeds 5 Amperes (A) heart standstill can occur. Myocardial damage can be detected by ECG examination. Typical symptoms are ST segment and T wave changes. Initial myocardial damage caused by current pass can progress even to heart failure or myocardial infarction. Current intensities from 15 to 20 mA can cause very strong tetanic muscle contractions leading eventually to bone fractures. The contractures can also prevent the victims to release from contacting the current source (live wire). Current pass through deep
tissue structures can cause thrombosis of blood vessels with subsequent ischemic damage and necrosis of tissues or organs affected. Extensive muscle tissue necrosis is accompanied by release of myoglobin into circulation (myoglobinaemia) which can cause renal function impairment or failure. Ischemic damages in extremities can progress to gangrenes requiring amputations. If the current will pass through nerves, this causes nerve damage resulting in paresis. Current pass through brain structures causes loss of consciousness, its pass through spinal cord causes immediate or delayed paralysis. Quite frequent complications in the eyes include delayed lens cataracts.

According to the **injury mechanism** there are **two types** of electrical injuries:

- **True (contact) electrical injuries** occur when the electric current passes through body parts which are acting as conductors. Current pass generates heat in tissues affected according to Ohm’s law. This mechanism is completely different from other kinds of thermal injuries caused by external heat sources. By external heat source burns the deepest tissue damage is the closest to the source. The depth of tissue damage decreases directly depending on the distance from the heat source.

- **Electrical arc burns.** They are caused when arcing occurs between live conductors and the earth. Electrical arc is of very high temperature (4000 °C) but of very short duration (thousands of a second). The depth of primary arc burns depends on tension (voltage) and intensity (amperage) of the current.

According to the **current source** there are electrical burns caused by:

- **Direct** current – they are much less frequent;

- **Alternating** current – they represent the majority of the injuries. The most frequent power lines for households have 230 Volts/50 Hz and 380 V/50 Hz for most of the enterprises.

According to the **current source tension** there are electrical burns caused by:

- **Low tension – up to 1000 Volts**

Major threat of low tension electrical injuries is cardiac arrest. If appropriate reanimation is not performed within less than 5 minutes following the arrest, the victims die. Burns caused by low voltage injuries are mostly not extensive, but they can be deep involving deeper structures while causing little damage to skin (only entry and exit wounds). There can occur also damages to the heart, tetanic muscle
contractures or respiratory paralysis. The majority of such injuries occur at homes or around homes, just minor part of them are work-related.

- **High tension – higher than 1000 Volts**
  They cause mostly extensive deep burns very often accompanied by ischaemic damages to deeper structures. High – tension electrical arc can ignite the victim’s clothes leading to combined electrical and thermal burns. High tension current causes mostly deep burns which can involve also muscles, bones, visceral organs etc. and are associated by a wide range of serious complications often leading to limb amputations and permanent sequelae.

- **Lightning** – here the tension can reach 1 million Volts
  Lightning is an electrical arch with extremely high tension and short duration. Lightning strike in addition to burns can cause consciousness loss, cardiac arrest and clothing ignition as well. Mortality is high, it is caused by cardiac arrest or ventricular fibrillation and it can kill the victim also without causing cutaneous burns. The burns caused by lightning are more superficial than those caused by high tension injuries. There are typical “lightning pictures” present at the skin surface (Fig. 16). It should be kept in mind that successful resuscitation following lightning strike can be provided also later as 5 minutes post cardiac and respiratory arrest.

**FIRST AID AND TREATMENT**

**First aid**
Main aim of the first aid in electrical injuries shall be to interrupt the current flow by turning off the current switch or the fuses, or pulling the live wires from victim’s body by any insulating tool. It is dangerous to do any manipulations with the victim unless the current flow is interrupted, as the rescuers could be caught into the electrical circuit with all its consequences. After the victim is secured at a safe place, resuscitation procedures shall be initiated and continued until emergency medical service arrives. If ventricular fibrillation is suspected, defibrillation shall be done.

**Emergency treatment**
Advanced life support procedures including vascular access, endotracheal intubation, cardiac massage and fluid replacement therapy shall be prioritized. Continuous patient monitoring is also of utmost importance. In deep burns of extremities evaluation of vascular functions along
with neurological examinations should be provided. Necrotomies/fasciotomies shall be started as soon as possible, but not later than 6 hours post injury. Primary early excision of necrotic tissues shall be provided as soon as possible, as leaving extensive deep necrotic tissue in place can predispose to life threatening anaerobic infections. If gangrenes of the extremities develop, emergency early amputations should be done.

6.1.4.3 CHEMICAL BURNS (CORROSIONS)

They are caused by many compounds with corrosive potential and compose only around 3% of all burns. These compounds cause tissue damage mostly by denaturation of biological proteins. Individuals are exposed to corrosions both in domestic and occupational environments. The severity of these injuries is determined by various factors such as:

- Basic character of the chemical compound (acids, alkali, other compounds) determines their mechanism of action;
- Quantity and concentration of the compound;
- Duration of exposure;
- Penetrating capacity;
- Rapidity and effectiveness of first aid measures;
- Individual disposition of the victims.

For example concentrated acids cause desiccation of tissues and exothermic reaction resulting in tissue coagulation whereas alkalis cause liquefaction of tissues enabling more deep penetration of the corrosive agent into tissues.

The resulting changes in tissues are similar to those present in burns caused by physical agents. Their severity classification according to severity factors is the same as well (see Tab. 4, p. 27).

**First aid**

In contrary to thermal burns where further tissue damage is arrested by elimination of the heat source, the chemical process continues until the corrosive agent is completely removed from the tissues by continuous irrigation or neutralized. Therefore it is of utmost importance to remove first the contaminated clothing from the victim and then provide copious irrigation by large amounts of water (shower) or diluted neutralizing agents. The duration of lavage shall be usually between 30 minutes and 2 hours, for the eyes even by longer time by continuous drips. Concentrated neutralizing agents shall not be used due to exothermic reactions which
can cause further thermal damage to injured tissues. There are just few commercially produced neutralizing agents such as diphoterin which can be used for all kinds of chemical agents and hexafluorin which is a specific inactivating agent for hydrofluoric acid burns. In more extensive exposure to some chemical agents signs of systemic toxicity can be present and need to be treated accordingly.

**Treatment**

Elimination of the corrosive compound from tissues is the most important issue before topical treatment can be initiated. The methods used are the same as in other kinds of burns and depend on the extent and depth of the injuries. Early surgical removal of contaminated tissues can prevent progress of systemic toxicity in indicated cases.

### 6.1.4.4 Frostbites (Cold-induced injuries)

They occur if during exposure to low environmental temperatures the local tissue temperatures fall below freezing point. Tissue damage occurs by formation of extracellular and/or intracellular ice crystals causing direct injury to cell membranes resulting in cellular dehydration and cell death. In addition to tissue changes capillary blood flow becomes severely compromised by initial complete flow cessation and subsequent reperfusion after re-warming of the tissues. This results later in endothelial damage and micro thrombi formation caused by reactive oxygen species, which can progress in progressive tissue death. The severity of tissue damage induced by cold is determined by:

- Temperature and humidity of outer environment. Dry cold penetration into tissues is lower than tissue exposure both to high humidity and low temperatures;
- Exposure time to below freezing point temperatures;
- Quality of first aid and of wounds management.

### Classification

**1st degree - congelatio erythematosa**

The affected skin is pale, hard, painful, or insensitive. After re-warming it becomes red, is painful and swelling occurs.

**2nd degree - congelatio bullosa**

Initially the skin is hard and whitish, after re-warming blisters are forming with increasing pain and oedema. If the blisters get ruptured, they can become infected easily.
3rd degree - congelatio necrotisans

The frozen part is pearl white, following re-warming it becomes first red, later bluish to black in colour. Severe pain develops during and immediately after re-warming. Dead tissue parts are insensitive to needle prick. The blisters are hemorrhagic, epidermis will slough off and devitalized dermal bed is getting exposed. Later the necrotic tissue desiccates and hardens. Several days later demarcation starts and facilitates recognition of vital tissues from necrotic ones. In acral deep frozen parts dry gangrenes develop by time (Fig. 17).

**Initial management**

Prevention is the best means. If first signs of frostbite are identified in remote outside environment, rubbing with snow is not more recommended and the affected body parts should be covered by additional dry clothing and shall be protected from further mechanical trauma. Field re-warming before transportation is not recommended, unless the transport conditions would be able to prevent the frozen and re-warmed parts from re-freezing. Rapid re-warming of frozen parts by immersing in a 37-39 °C water bath should be provided at final destination. The duration of re-warming is approximately 30 minutes. Topical wound care is similar to that in other kinds of thermal injuries. By systemic medications Ibuprofen, Pentoxifylline and some thrombolytic agents proved to be beneficial. Hyperbaric oxygen therapy showed to have beneficial effects as adjunctive therapy as well, but more randomized controlled studies should be needed to prove it. Aseptic techniques in wound management along with topical therapy should be provided.

**Treatment**

Principles of wound management are the same as in thermal burns. In partial thickness injuries conservative treatment is the gold standard for wound care. In full thickness frostbites surgical removal of necrotic tissues should be delayed until they become clearly demarcated. Wound closure is achieved by skin grafts or other plastic surgery techniques. If parts of extremities turn into gangrenes, amputations have to be performed.
7 REHABILITATION AND RESTITUTION PERIOD

Following closure of the major part of the burn wound the acute period is finished and rehabilitation and restitution period is started. With on-going wound closure the hyper catabolic state of the patient changes to anabolic one and he/she is going to regain gradually his/her original body weight. At the same time rehabilitation helps to regain the patient’s original skills preparing his/her for return back to family and work. There are a lot of new problems which need to be solved or overcome by close cooperation of the patient with the burn surgeon, rehabilitation therapists, nurses, psychologist and social workers.

The most frequent problems are as follows:

- Care of recently healed and or residual wounds and defects or re-opening of already closed wounds and/or donor sites;
- Restoration of joint movements and muscle strength;
- Appropriate nutrition necessary for anabolic processes and compensation of energy losses during exercises;
- Prevention of hypertrophic scars and contractures;
- Psychological support;
- Permanent sequelae.

7.1 CARE OF RECENTLY HEALED AND/OR RESIDUAL WOUNDS AND DEFECTS OR RE-OPENING OF ALREADY CLOSED WOUNDS AND/OR DONOR SITES

Following graft take and/or epithelialisation of the partial thickness burn wounds and donor sites there are always multiple residual small open wound areas where the healing is prolonged. This situation requires continuous both intensive residual wounds care and care of recently healed areas with thin, fragile epidermis and underlying scars which are very sensitive to secondary mechanical trauma and other factors from the outer environment. It is of utmost importance to protect the open wound areas from secondary infection which in worst scenarios can lead to re-opening of the already healed wounds and donor sites and even can cause systemic septic complications. The healed areas can be gently washed with mild soap and running water (shower). Local infection can be prevented by aseptic approach and
by use of disinfectants dissolved in water (such as povidone-iodine, chlorhexidine etc.) which do not irritate the freshly healed wound areas and which do not interfere with the epithelialisation process. The recently healed and skin grafted areas need also greasing by white Vaseline, or olive oil, or other commercially available hydrating and greasing substances. The residual open areas often need application of topical antibacterials or other kinds of topical devices such as hydrocolloids, hydrogels, alginates, impregnated mesh gauze etc. It is important as well to wear comfortable preferably cotton based soft clothing to avoid rubbing and friction of the fresh scars by clothing. Pressure garments for hypertrophic scar prevention can be used when the scars will be able to withstand pressure and mechanical forces.

7.2 **RESTORATION OF JOINT MOVEMENTS AND MUSCLE STRENGTH**

The primary goal of rehabilitation is to restore patient’s functional ability and range of motion and preparation for return home, eventually back to work. To achieve this goal a comprehensive approach including the following activities is of utmost importance:

- Positioning, splinting, casting, orthotics and prosthetics during the acute phase of burn treatment,
- Range of motion exercises with gradual increase of their intensity to regain the original muscle strength,
- Simultaneous scar management and care of healed areas,
- Training and performance of activities of daily living,
- Patients and their future home caregivers education,
- Psychological support of both the patients and family members.

In order to achieve optimal results of this quite difficult and lengthy process, active cooperation of the rehabilitation and psychology specialists’ team along with positive and active cooperation of the patients themselves is necessary.

7.3 **NUTRITIONAL SUPPORT OF ANABOLIC PROCESSES AND COMPENSATION OF ENERGY LOSSES DURING EXERCISES**

During acute phase the patients are in negative metabolic balance state and lose their body mass. This can be by part prevented by intensive realimentation preferably by enteral route.
In rehabilitation period they need increased nutritional support rich on energy proteins, vitamins and trace elements in order to replace energy needed for increased physical activities and exercises. Nutritional support is beneficial for better residual wounds healing as well.

7.4 PREVENTION OF HYPERTROPHIC SCARS AND CONTRACTURES

Burns which heal within less than 14 days post injury generally have no scarring. The longer the wounds remain open and the healing time is prolonged, the more intensive is the fibroplasia resulting in scar hypertrophy. All burns which are deeper than superficial partial thickness injuries are prone for development of hypertrophic scarring. Scar hypertrophy starts to be apparent clinically within 3 to 6 weeks following wound closure. Early signs of hypertrophy include gradual scar elevation and firmness together with its red colour which is accompanied by parestheses and itching of different intensity. This process can continue during several months. After scar hypertrophy will reach its peak, which is strictly individual in time and intensity, remodelation and atrophy of the scar masses will start which can last for several months or even years. Scars which will not undergo remodelation and remain in the phase of hypertrophy are called keloids. As it was mentioned above, the processes of hypertrophy and atrophy are individual and cannot be predicted. Unfortunately there are very few preventive and/or therapeutic measures which can so far influence or diminish the scar hypertrophy and which can speed up their remodelation process. Scar hypertrophy and contractures prevention and treatment will include:

**In acute phase** (if the wound is still open)

- Positioning and splinting in physiologic positions,
- Early excision and grafting of deep burns,
- Prevention and treatment of burn wound infection which leads to prolonged healing and more intensive scar formation,
- Early closure of residual open wound areas.

**Following wound healing/closure**

- Continuation in positioning and splinting,
- Passive, assisted and active rehabilitation procedures,
- Use of ointments and creams for care of the healed areas and scars,
- Continuous elastic pressure of scars by application of elastic bandages or custom – made pressure garments,
- Use of silicone products such as soft silicone plates or creams on hypertrophic areas,
- Taping of hypertrophic scar areas by hypoallergenic tapes,
- Intralesional application of long-lasting corticosteroids such as triamcinolone acetonide etc. (it is possible to limited area scars only).

These measures shall be used during the whole time of scar remodelation process which takes in average from 6 months to several years.

7.5 **PSYCHOLOGICAL SUPPORT**

Severe burn injury has a great influence on patient’s psychic. Burn survivors suffer of several psychological problems such as body image disturbance, traumatic stress, depression, anxiety, grief, pain, itching etc. Both appearance and physical limitations interfere with person’s social relations, their ability to work and perform familiar responsibilities. Approximately 30% of burns survivors experience moderate to severe long-term psycho-social problems. Due to these reasons psychological support of burn victims is of utmost importance starting as soon as immediately or few hours following injury. This support should pertain to both the patients and their families.

7.6 **PERMANENT SEQUELAE OF BURNS**

The most common permanent sequelae of burn injuries include:

- Scars,
- Contractures,
- Amputations,
- Functional sequelae,
- Deformities,
- Psychological disorders.

Permanent sequelae are determined primarily by the character of original burn injury. Most important factors which play decisive roles for occurrence and severity of these sequelae are burn extent, burn depth, localisation of deep burn injuries, timing and quality of primary care, subsequent treatment modalities and the patient’s individuality, occupation and family.
7.6.1 **SCARS**

Scars located at visible and aesthetically important body areas are always disturbing and making the patients to feel at least uncomfortable, or handicapped. Scars at functionally important areas of the face (Fig. 18) can lead also to deformations of these areas, particularly if they are located at the nose, lips, cheeks, forehead, around the eyes and at the ears. In addition to aesthetic impairments due to scar hypertrophy, the scars are often different in colour to normal skin, itchy and with impaired sensation. The same problems are with such scars on the hands, neck, feet etc.

7.6.2 **CONTRACTURES**

One of the scar properties is contraction which starts from its centre and thereafter pulls together the surrounding structures. End result of contraction is **contracture** which interferes with normal range of movement particularly around joints, or aesthetically important anatomical structures. Contractures which do not respond to conservative treatment such as positioning, splinting, pressure therapy and rehabilitation are indicated for secondary surgical reconstruction procedures (see later part 8, p. 95).

7.6.3 **AMPUTATIONS**

Amputation means surgical removal of a body part. It is permanent and dependant on the anatomical locations it leads to permanent loss of function. Function of amputated extremities or their parts such as hand, lower legs, fingers, nose, ear etc. can be by part replaced by prosthesis, epithesis, or surgical reconstruction. Prosthetic replacement of lower extremities can enable walking again, hand prostheses can be either aesthetic, or functional which enable some kind of grasp. Epithesis are used to replace lost parts of the face or fingers where surgical reconstruction would be difficult, or impossible.

7.6.4 **FUNCTIONAL SEQUELAE**

Their severity depends on their location. For example contractures of eyelids (ectropion, entropion, angular contractures) can lead to exposure and desiccation of the conjunctiva and cornea and need urgent reconstruction. Contractures around the mouth impair opening of the mouth which interferes with eating but also can make dental care, or intubation difficult or impossible. Contractures around joints at functionally important areas such as axilla, cubital fossa, popliteal fossa, wrist, fingers etc. (Fig. 19) cause severe functional impairments and
need to be reconstructed as well, particularly in growing children, where they can cause growth deformities.

### 7.6.5 Deformities

They are initiated by pulling forces of contracting collagen bundles in the newly forming scars. Deformities contribute to more or less disfiguring appearance of the burn victims which is a very handicapping condition. They are initiating factors of psychological disorders which make the victims anxious and depressive. They have also great impact to victims’ social life and overall quality of life.

### 7.6.6 Psychological Disorders

Long-term and/or permanent psychological sequelae of burn injuries are including:

- Body image disturbances,
- Depression,
- Social anxiety,
- Sleep disturbances,
- Grief,
- Post-traumatic stress disorder,
- Quality of life deterioration.

The process of psychological adaptation can be prolonged to several months or even years. Fortunately most of the burn survivors will resume their lives, productivity and social interactions fairly well. Those who will not will need continuous or intermittent therapeutic interventions for improving their quality of life.

In summary, permanent sequelae following healing of deep burns are always important and difficult problems accompanying the burn patients and their families for the rest of their lives. They require a comprehensive approach from the site of health care providers in order to offer to the patients and their families optimal solutions for continuing the life long after care, rehabilitation, psychological support, assistance with return to work and also options for surgical reconstructions or provision of prostheses and epiphysis.
8 PRINCIPLES OF RECONSTRUCTION

With increasing chances for survival of extensive and deep burn injuries in the last few decades, more and more attention is focused not just on the rapidity of extensive burn wounds closure, but on the quality of skin and scars in the healed areas as well. Although the main aim of reconstructive surgical procedures are restorations of shape and function of the injured body parts, complete renewal of shape and function (i.e. regeneration) cannot be achieved so far in most of the cases. The results of healing of majority of burns deeper than superficial partial thickness injuries are scars with subsequent hypertrophy, deformities, contractures and impairment of body parts appearance and functions. Reconstructive surgical procedures shall aim to improve shape and functions of body areas with unsightly scars, soft tissue and joint contractures. There are several techniques used for reconstructive procedures, they can be ranked in a „reconstructive ladder“ from the simplest procedure up to most difficult one on the top (Tab. 11).

In cases where the deformities are extensive and areas of healthy tissues which can be used as donor sites for both skin grafts and flaps are limited, planning of reconstructive surgical procedures should be meticulous and many local factors as well as patient´s individual wishes and imaginations should be considered. Aesthetically and functionally important body areas and areas where the pull of the scars can interfere with the child´s growth shall have priority.

Tab. 11 Reconstructive ladder

<table>
<thead>
<tr>
<th>Rank</th>
<th>Procedure</th>
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<tbody>
<tr>
<td>8</td>
<td>Microsurgical tissue transfers</td>
</tr>
<tr>
<td>7</td>
<td>Musculocutaneous, osteocutaneous flaps etc.</td>
</tr>
<tr>
<td>6</td>
<td>Fasciocutaneous flaps</td>
</tr>
<tr>
<td>5</td>
<td>Skin flaps (adjacent, distant etc.)</td>
</tr>
<tr>
<td>4</td>
<td>Free skin grafting (partial thickness, full thickness, dermis grafts)</td>
</tr>
<tr>
<td>3</td>
<td>Skin expansion using skin expanders</td>
</tr>
<tr>
<td>2</td>
<td>Dermabrasion</td>
</tr>
<tr>
<td>1</td>
<td>Simple scar excisions &amp; suture, local plasties</td>
</tr>
</tbody>
</table>
According to simplicity or difficulty of the surgical procedures they are ranked from the simplest to the most difficult ones as follows:

1. **Simple scar excisions & suture, local plasties**

They can be provided in cases where smaller scars are present and there is enough quantity of surrounding good and healthy skin which can be mobilized to enable primary wound closure following excision of scars. The most frequently used local plasty is so called Z-plasty (Fig. 20).

2. **Dermabrasion**

Dermabrasion means removal of scar surface using abrasive surgical tools or surgical lasers for correction of scar irregularities and colour. Healing following abrasion is by epithelialisation. One should be cautious not to remove thicker scar layers, as the scar epidermis is very thin and complete epidermis removal will lead to healing by secondary intention with new scar formation.

3. **Skin expansion**

It is provided by special inflatable balloons (expanders) of different shape made from medical grade silicone which are implanted adjacent to scars which are going to be reconstructed. The balloons are provided by valves which are used to fill the expanders by sterile saline in several sessions. Expansion of the balloons will cause expansion and growth of skin overlying the expanders. Their filling is provided gradually in several days or one week intervals by saline doses corresponding to 10% of the total content of the expanders. Following expansion of skin the adjacent scars are excised, the expanders are removed and the remaining defects are covered by the expanded skin which is of the same texture, colour and quality as the original skin where the scar has developed.

4. **Free skin grafting**

Free skin grafting is used to cover larger defects following hypertrophic or colloid scars excision. The donor sites should be selected carefully in order to match the texture and colour of the skin grafts to the recipient area. Mostly split thickness free skin grafts are utilized, as their donor sites can heal spontaneously. Full thickness skin can be used for special areas such as parts of the face, lower lids, neck, palms and soles. The problem in full thickness skin grafting is that the donor sites should be closed primarily or grafted by split thickness skin grafts. The thicker the skin grafts are the better quality of recipient site vascular supply is
required for optimal graft take. Otherwise the grafted skin will not heal in which means failure of the procedure.

5. Skin flaps

Flaps are areas of skin with subcutaneous tissue which have their independent blood supply. According to blood supply there are random flaps supplied by vascular network present in the nutrient pedicles of the flaps or axial flaps, where the flap is designed around the nutrient artery and vein present in the pedicle. There are just few areas of the body where axial flaps can be designed (such as inguinal flap, deltopectoral flap, frontal or supraorbital flap etc.). The advantages of a flap compared to free skin graft are, that the flap nutrition is assured by its own blood supply and its take is not as much dependent on the vascularity of the wound bed. Moreover, flaps can be used for coverage of exposed deep structures with blood supply insufficient for free skin grafts take such as exposed tendons, bones, nerves, joints, or cavities of the body.

According to distance of flap transfer there are local (or adjacent) flaps which are transposed or rotated to the adjacent areas, or distant flaps which are transferred to more distant body areas following previous flap preparation (such as tubulization of tube flaps, or using a carrier like forearm) for transfer.

6. Fasciocutaneous flaps

Deep muscular fascia is well vascularised and the overlying skin parts are supplied by perforator vessels. Inclusion of fascia into cutaneous flaps enhances their vascularity and improves probability of their survival following transfer. These flaps are pliable and suitable to cover even larger defects.

7. Musculocutaneous, osteocutaneous flaps etc.

In addition to fascia parts of muscles can be included as well in the flap design. Muscles can be used as flaps also without a skin component, eventually also pieces of bones to which the muscles attach can be incorporated in the flap design to replace defects of bones. Examples of such flaps are lower leg muscle or musculocutaneous flaps (from m. gastrocnemius or soleus), m. latissimus dorsi flap, m. gracilis flap etc.

8. Microsurgical tissue transfers

Axial pattern flaps in different body parts can be transferred to distant body parts also by microsurgical techniques, which mean preparation of axial flap together with its nutrient artery and vein and following transfer to recipient site anastomosing the nutrient vessels to
corresponding artery and vein at the recipient site. As these vessels are usually of quite small diameters (sometimes even less than 1 mm), microsurgical techniques of vessel anastomoses should be used. The major advantage of these operations is one-stage procedure. The disadvantages include prolonged operating time (several hours), need of an operating microscope, specially designed instruments and very fine suture material and that in case of failure the result is total flap loss.

Other reconstructive options

There are situations where there is not enough healthy autologous tissue which could be used for reconstructive procedures. In these cases use of allogenic materials can help to replace some of autologous defective or lost tissue parts. This applies particularly to the use of allogenic acellular dermis, or biosynthetic dermal templates combined with autologous thin split thickness skin grafts for heavily scarred areas, or allogenic bones for filling or replacing bone defects. There are also biosynthetic materials which can be used for such purposes. One of them which are used routinely in the last few decades is dermis replacement by biosynthetic templates such as Integra® or Matriderm®. Several other products from new and rapidly progressing techniques of tissue engineering and regenerative medicine are going to be used in the very near future for helping the burn victims to cope with their severe deformities.
9 CONCLUSIONS

Outcome from thermal injuries depends upon many variables which can include factors other than the severity of illness itself. These factors may include the victim’s personality, psychical state, social status, original professional engagements, family support, self-esteem and patient motivation. All these variables may finally be decisive and responsible for patient’s impairments secondary to burn injury and subsequent disabilities. Progress in comprehensive burn victims after care together with progress in methods and techniques of reconstructive surgery may assist the patients to overcome many difficult situations and help them to return to their families and society, which is finally the most important issue.
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13 APPENDIX OF FIGURES

Fig. 4 Lund – Browder chart for adults
Fig. 5 Lund – Browder chart for children
Fig. 6 Necrotomy, Fasciotomy

(Necrotomy: a) Lower legs b) Thorax, c) hand; Fasciotomy: d) Lower leg)
c) Hand

d) Lower leg
Fig. 7 Air fluidized bed
Fig. 8 Burns of respiratory areas
Fig. 9 Fascial excision

a) Lower legs

b) Thorax
Fig. 10 Tangential excison

Fig. 11 Meshed skin autografts
Fig. 12 „Sandwich“ skin grafting

Fig. 13 In vitro cultured keratinocyte sheet
Fig. 14 Integra artificial dermis

a) 3 weeks following necrectomy and application

b) Following removal of artificial epidermis (silicone membrane) ready to grafting
Fig. 15 Severe electrical burns

a) Localized hand injury

b) Extensive injury
Fig. 16 Lightning injury
(Lichtenberg figures)

Fig. 17 Deep frostbites
( forefoot gangrene)
Fig. 18 Hypertrophic face scars

Fig. 19 Flexion contractures of left hand II\textsuperscript{nd} and III\textsuperscript{rd} digit MP and PIP joints
b) Early healing following 8 days

Fig. 20 Reconstruction of contractures displayed on Fig. 19 by Z-plasty (index finger) and by full thickness skin graft (3rd finger)

Following scars remodelation