Paediatr Croat 2010; 54 (Supl 1): 10-17

Pregled
Review

UPDATE MANAGEMENT OF PEDIATRIC BURNS: FLUID RESUSCUTATION, METABOLIC CARE, INFECTION CONTROL AND SURGICAL TREATMENT OF THE BURNS WOUND

ZDRAVKO ROJE[1], ŽELJKA ROJE[2], MIHAJLO LOJPUR[3], JULIJE MEŠTROVIĆ[4]

Children, especially younger ones, form a risky group among burned patients. The number of burned victim among children has been decreased in most of European countries including Croatia. The burn care for burned children must take place in specialized burn unit and burn centre for children. The complex pathophysiology of severe burn injury still make it one of the most demanding therapeutic challenges. Scald burns secondary to household accidents predominate in most cases, constituting 70% of all thermal injuries in infant, toddlers, and pre-school children. Recent advances in the area of pathophysiology of burn shock and burn infection, intensive care, surgical treatment and skin substitutes have a strong reflection on the four main levels of modern treatment of burned children: fluid replacement, metabolic care, prevention of infection, and burn wound surgery. However, delivery of these outcomes in not easy and requires a multidisciplinary effort team of surgeons, paediatricians, physiatrists, physical and occupational therapists, nurses, anesthesiologists, social workers, psychiatrists, and psychologists with great experience in burn care, need to work together as a unit. Unless such coordinated multidisciplinary aftercare is provided, it is not possible to achieve the highest quality outcome after serious burn injuries.

Descriptors: PAEDIATRIC BURNS, FLUID RESUSCITATION, METABOLIC REQUIREMENTS, INFECTION CONTROL AND SURGICAL TREATMENT

Introduction

Children, especially younger ones, form a risky group among burned patients. A severe burn injury is not only a life threatening problem for injured children, but may have serious physical, psychosocial and financial effects on them, their families and the society. Although the number of burned victims among children has been decreasing in most of the European countries, including Croatia, during the last two decades, the complex pathophysiology of the severe burn injury still makes it one of the most demanding therapeutic challenges. Recent advances in intensive care, and knowledge of the pathophysiology of burns shock have a strong impact on the four mainstays of supportive treatment in burned children: *fluid replacement, metabolic care, prevention of infection and early excision and grafting.* Serious burns among have three distinct characteristics. In the first place a great incidence of burn shock is presented, which may be lethal to patients with large burns during the first week post injury. Next, burn wound infection and sepses occur often and commonly kill patients who survive the burn shock during the few post burn weeks. Finally, there is a great incidence of post burn deformities in patient who survive the healing process of their wound, which depend on the appearance of contractions and hypertrophic scaring. In recent years, there has been an important shift in the understanding of and approach to *fluid resuscitation*, fuelled largely by the increasing recognition that modern crystalloid resuscitation frequently provides substantial volumes of fluid, often exceeding the amount predicted by current formulas, resulting in edema related complications "*Fluid creep*" (1). The today consensus is to perform resuscitation on the individual bases what is dramatically reduces the mortality rate from burn shock. *The hypermetabolic stress* is responsible for severe catabolism, immune dysfunction, and profound physiologic perturbations affects in every burned child with burns greater than 40% of its total body surface area (TBSA). This response is pervasive and prolonged and cannot be completely abolished despite the pharmacology and non-pharmacology interventions (2). After the initial resuscitation, *burn wound infection and sepsis* still present a leading causes of morbidity and mortality, and remains one of the most challenging concerns for future investigation. Most superficial burns will heal with *topical*

[1]Division for Plastic Surgery and Burns,
Department of Surgery,
University Hospital Centre Split
[2]Division of Surgery,
University Hospital Dubrava, Zagreb
[3]Department for Anesthesiology and Intensive Care Unit, University Hospital Centre Split
[4]Paediatric Clinic, University Hospital Centre Split

Address:
Zdravko Roje, MD, DS
Division for Plastic Surgery and Burns
Department of Surgery
University Hospital Centre Split
10000 Split, Spinčićeva 1
E-mail: zdravko.roje@st.t-com.hr
zdroje@kbsplit.hr

antimicrobials and/or dressing within approximately two weeks. However, deep burns either require early excision and grafting or may be left to heal without surgical intervention but with the aggressive use of dressing. The choice of dressing is nevertheless still controversial. The control of *burn sepsis and nutritional requirements* is dramatically improved with early surgical excision of deep burns and immediate grafting of all excided surfaces.

Epidemiology and organization of burn care in Croatia

Since the number of burns per year in the Republic of Croatia has not been exactly defined, neither the number of burns in children under the age of 18 has been known. The total number of hospitalized children has not been determined either, but we can say that children have been hospitalized in the Burn Unit (BU) of Surgical Clinics, Intensive Care Unit (ICU) of Paediatric Clinics and Paediatric Surgical Clinics, and in the Clinic for Children Diseases in Zagreb. According to our estimations, the number of hospitalized children in Croatian hospitals does not exceed 200 cases per year. The total mortality rate of burned children is also unknown. According to the most recent international data of cases history, paediatric burns account for about 40-45% of all burn related hospitalization (3). They are frequent worldwide, and have the same occurrence from country to country, but with some differences related to local customs. The most frequent etiologic factor of burns in children is in 70% of cases hot water and open flame in work accidents. Especially endangered are young children aged 4 to 5 years (4). In this group the most frequent causes are sudden contacts with burn agents such as contact with power circuit and parents' inattention. It is obvious that the education of parents as well as other the preventive measures, are the best way to reduce the incidence of burns in children.

The national burn centre (BC) for children must have a well developed program of care and complete treatment, specialized staff of physicians and nurses as well as very good technical equipment and instruments. In general hospitals and clinical centre burn unit (BU) with a very similar program of burn care must be established. Besides that, a BC should have an elaborated *National guideline for burn care* and a *Program for transport of children with burns to the BC*. In the Republic of Croatia, the *Croatia Burns Association (CBA)* has been finally founded in December 2008, as a *Section of Croatian Society for Plastic, Reconstructive and Aesthetic Surgery*. The basic goal of CBA is the organization and treatment of children burns according to the highest criteria of EBA (European Burns Association) and ISBI (International Society for Burns Injuries).

Patient and methods

The retrospective studies covered the period between January 2003 and December 2008 in the BU, Division of Plastic Surgery and Burns, University Clinical Hospital Centre Split. During the study period, we admitted 51 burned child with acute burns. The injuries had several different etiologies, including flame burns, hot liquid, electrical current, arc burns and hot solid. All patients were admitted to BU within six hours from the injury. The anatomical distribution included: head and neck area, trunk, and extremity, separately upper and lower extremity. There were only two children with respiratory burns and three with burns caused by electrical currents. The majority of burn victims received initial surgical debridement as they were admitted to BU. There were 39 (77%) partial thickness burns (PTB), and 12 (33%) full thickness burns (FTB) that were treated with early tangential excision and skin grafting.

Method

The data about the burns wound was collected from the medical documentation of burned children at the BU and from the therapy protocol of the Emergency Care Unit (ECU). The main anamnesis data included the demographic range, the mechanism of the injury, the site and the size of the burn and its associated injury. Additionally, we controlled the vital signs and EKG data, the laboratory results, and x-ray pictures. At the admittance we determined the fluid resuscitation, metabolic needs and nutrition. Depending on the burn severity all children with burn shock and multiple organ failure syndrome (MOF) have been treated in the Intensive Care Unit (ICU). The final result was analyzed according to the hospitalization and the outcome at the time of discharge.

Burn injury physiology

A thermal injury causes the coagulation necrosis of the skin and underlying subcutaneous tissues. A local tissue response to burning involves not only direct tissue coagulation but also microvascular reaction in surrounding dermis, in means of progressive vasoconstriction and thrombosis that may result in extension of burns (5). The systemic response was assured with the loss of skin barrier and the release of vasoactive mediators from the wound and from subsequent infection. Those changes involved hemodynamic, tissue inflammation, wound healing, immune response, hypermetabolic reaction and excessive catabolism. When burn size exceeds more than 25% of the body surface area (BSA), interstitial edema develops in distant organs and soft tissue secondary to an action of wound released mediators and hypoproteinemia. The treatment of burned children differs substantially from that of adults not only because of the smaller body surface areas and the different anatomical structures but also because of involved metabolic process, homeostasis factors, hormonal response, immunological profile, the degree of psychological maturation, and healing potential. All these combined factors make children very special patients - younger they are the more special they become.

After a successful resuscitation, a hypermetabolic response occurs with near doubling of the cardiac output and resting energy expenditure (6). Enhanced gluconeogenesis, insulin resistance, and increased protein catabolism go along with each severe burn injury. The etiology of these changes is not well know, but it is assumed to involve a combination of factors including a change in hypothalamus, and increased glucagon, cortisol and cateholamine secretion, GI barrier dysfunction and translocation of bacteria and their toxins through gut mucosa, manifested burn wound infection and sepsis, and appearance of hypothermia. The additional goal is to eliminate the unfavorable aspects of the hypermetabolic response, especially the muscle catabolism. For the elimination of hypermetabolism the referent BC used beta-adrenergic blockade, beta adrenergic supplementation, nonsteroidal anti-inflammatory agent, growth hormone, and insulin like growth factor-1. All of those medications are under active investigation. Current data seem inadequate to support the routine use of such therapies outside clinical trials (7).

Fluid resuscitation

Burn shock is a form of hypovolemic shock that arises as a result of the translocation of isotonic protein containing fluid from the vascular space into the interstitial space, resulting in edema (1, 7). Their basic clinical characteristics are the rapid and progressive decrease of plasma volume and the increase of haematocrit values, decreased minute volume of the heart, decreased tissue perfusion and the occurrence of hypoxia. Immediately after a large burn we can detect the systemic permeability of capillary leak, which increases with burn size, the delay in initiation of resuscitation, and the presence of an inhalation injury. If fluid resuscitation is appropriate, it usually takes only 24-48h to reduce the hypovolemia and stabilize the cardiovascular system and organ perfusion. Fluid resuscitation formulas are the only appropriate, and they will accurately predict the volume requirements of individual patients. The ideal hydration solution could re-establish the cardio-circulatory function without increasing the local burn and the general edema. The two most commonly used formulas for children resuscitation are different modification of the Parkland formula that originally prescribes 4 ml/kg/%burn/24h as *isotonic Ringer lactate (RL),* and Carvajal formula of Galveston regime that prescribes maintenance fluid (2000ml/m^2/ plus burn replacement fluid /5000 ml/m^2) in the first 24h given as *dextrose 5% in RL containing 12.5 g of albumin/L* (8). The Galveston regime causes least edema; it better recovers the minute volume of the heart and enables early excision of burn necrosis. One half of the calculated resuscitation fluid should be administered within the first 8h and the rest in the following 16h. For reanimation of children in the next 24h recommendation is to administer 3750 ml RL/m^2/BSA/24h and additional 1500 ml RL/m^2/24h as maintenance fluid (8).

As a general rule, burns of 15% BSA are not associated with an extensive capillary leak, and children with burns in this size range can be managed with fluid administered at 150% of a calculated maintenance rate. However, the total hydration status must be strictly monitored. Those who are able and willing to take fluid by mouth may be given fluid by mouth with additional fluid given IV at a maintenance rate. In order to prevent hypoglycaemia, which has been mostly observed in younger children, it is necessary to add additional *5% glucoses and 12.5 g albumin/L* into the resuscitation RL solution. Most resuscitation formulas recommended the administration of colloid when capillary integrity returns, generally after 24h, as colloid is more likely to remain in the intravascular compartment at that time. The earlier use of *colloid* seems to improve hemodynamic and decrease volume needs. Most of the clinical physicians use *5% albumin with isotonic crystalloids*, although *fresh-frozen plasma* can be given, what is much better for the correction of coagulopathy and the prevention of disseminated intravascular coagulation (DIC). Colloids are administered in a continuous infusion and the dose is determined in relation to the size of the BSA. At this time *enteral feeding* should also start, except in the case of major burns, and in children who have not been administered enough fluid resuscitation or have developed ileus due to gut hypoperfusion. It is often observed that inhalation injury, delay in resuscitation, and unusually deep burns result in higher fluid requirements. It has also been a common belief that younger children require higher resuscitation volumes per body mass than older children and adults. However, a urine output of 2 ml/kg/h is probably appropriate in infants, whose renal concentrating abilities are immature. But in toddlers and older children, whose renal concentrating abilities are more mature, this may result in an excessive volume administration. *Hypertonic replacement solutions* have been less used nowadays because of their very demanding technical application and, in addition to this; they can compromise the final outcome of the resuscitation. In case of large burns, *sodium bicarbonate* can be added to the RL solution, which than makes it moderately hypertonic (7, 8). The appropriate doses of bicarbonate are approximately 0.25 mEq/kg/h.

Fluid resuscitation should be *regularly monitored.* The best parameter is the urine output. Hypotension is one of the last indicators for the burn shock; therefore the pulse rate measurement is a much more significant sign than the blood pressure measurement. Normal sensorium, body temperature and the relevant peripheral capillary refill are additional clinical sings of good tissue perfusion. Increased burn edema and high urine output are usually basic signs of an over-resuscitation, while under-resuscitation is usually difficult to diagnose and categorize. Sometimes reanimation is not going as we expected and the fluid resuscitation quantity rises above 6 ml/kg/% of burned area/24h. In that case intravascular volume should be measured much more precisely, what demands placing a central venous catheter, or installing pulmonary artery catheter. The dopamine infusion dose of 5 μg/kg/min can significantly improve renal perfusion and increase urine output, without raising the fluid intake. Furthermore, early protein administration may also sometimes improve renal perfusion too. A major burn usually causes myocardial dysfunction, and a small-dose of cardiotonic, like Dobutamine should be administered (7). Pigmented urine indicates a high voltage electrical burn or very deep burns. This may lead to the renal tubule block and therefore the myoglobine must be urgently rinsed. Urine output must be over 2 ml/kg/h, and then particularly in the course of the next few hours, the crystalloid infusion should be prolonged (7). The administration of bicarbonates and diuretic manitol can also prevent the tubular block with myoglobin cylinders. Serum ionized calcium and serum magnesium should be monitored because a supplementation is commonly required during this period. Clinical trials supplementing endogenous antioxidant defenses in children with large burn, and high dose vitamin C and other agents during burn resuscitation may play an important role in the future (7). After 18-24h the integrity of the capillary membrane is usually re-established, what are the best indicator of successful reanimation. At that moment total fluid intake should be adequately decreased, because additional over-resuscitation can result in newly aggravated clinical sings of burn shock. The surgical management of the burn wound has an additional impact on fluid resuscitation, and on serum electrolytes. Hypernatremia can cause additional damage to the brain, and trigger intracranial bleeding. The application of silver sulphadiazine topical antibiotic (DERMAZIN), which is not soluble in water, may trigger an additional need for water that can be replaced enteraly. However, water-soluble topic antibiotic (5%-solution of silver nitrate) may lead to hyponatremia and brain edema. This is why a regular control of serum electrolytes is essential, particularly of sodium, calcium and magnesium (7).

Metabolic control and children feeding

The main targets during the *hypermetabolic response* in severe burned children includes mediators of the hypermetabolic response, acute phase proteins peak up i.e. cytokines an hormonal changes, changes in resting energy expenditures (REE), multiorgan dysfunction, whole body cathabolism, changes in glucose metabolism and appearance of sepsis. Unfortunately, there are many other factors leading to hypermetabolic response. The most important ones are the evaporative fluid loss, stress hormones (catecholamines, cortisol, and glucagon) and mediators of wound inflammation, and exotoxins from the burn wound. The clinical picture is characterized by fever, increased metabolic rate, increased minute ventilation, increased cardiac output, decreased after load, increased gluconeogenesis that is resistant to glucose infusion, and increased skeletal and visceral muscle catabolism (9). Future changes, like *burn sepsis* increase REE and protein cathabolism up to 40% compared with those with like-size burns who do not develop sepsis (7, 9). Early burn wound excision and skin closure significantly decreases the occurrence of sepsis. The accurate support of this physiology changes is essential mainly to prevent impaired wound healing, cellular dysfunction, and decreased resistance to infection. Both, metabolic exhaustion and manifested burn wound infection with sepsis are the major causes of children death. Child remains hypermetabolic response until wound closure is complete and for a variable period thereafter, usually 1 year post-burn and more (7, 9). *Thermoregulation* is also significantly disturbed, primarily due to evaporative fluid losses and inflammatory mediators and cytokines that increase the thermoregulatory set point and alter endocrine function (7). This results in *hypothermia*, and requires an additional energy source to recover the body temperature. It is principally recovered from decomposition of carbohydrates and fats, and in major burns the decomposition is 2.5 to 4.5 times greater. The treatment of *pain and anxiety* also support thermoregulation, and ensure early burn wound excision and skin closure. *Metabolic needs* with the burns more than 25% TBSA are slightly above normal, and with the burn more than 40% TBSA they are 1.4 to 2 times bigger than normal. *REE* is also increased to about 180% during the acute hospitalization, after that it gradually starts to decrease. When burn wounds are completely healed, they return to 150% of the normal range. Furthermore, changes within the *hormonal milieu* occur as well, which have to re-establish significantly disturbed homeostasis and ensure burn wound healing (7-9).

Assessment of energy and other substrate requirements

An accurate estimation of *metabolic and energy requirements* in burned children is of great importance, but it is difficult to adequately determine it because of the implication of many factors (7, 9). The well known, frequently used Currery formula (25 × body weight/kg/ + 40 × of percentage BSA burned) was proposed in the late 1970s, and presents a good standard. It is a simple prediction of daily metabolic needs based on early attainable information, and calorimetric measurements. The Toronto formula measured energy expenditure very closely to the Currery formula, with the addition of the factor of activity for 24h energy expenditure. Other formulas do not account for TBSA burned, and are based on calculation of REE, by the Harris-Benedict equation, which takes in account the patient's age, sex, height, and weight. The Harris-Benedict derived REE calculations are widely used at present days. However, they under-estimate by 23% the actual energy expenditure for second and third degree burns ranging between 10% and 75% TBSA, while the Currery formula over-estimates energy expenditure by 58% (9, 10).

Overfeeding is associated with hepatic steatosis, leading to hepatic dysfunction and increased CO_2 production exacerbating respiratory insufficiency. *Underfeeding* results in inanition and poor wound healing. Children managed with prompt wound excision and grafting have reduced energy expenditure, and multiple studies have shown that standard formulas do not accurately predict the energy requirements in burned children. The total energy expenditure can be roughly estimated by using expired gas indirect calorimetry to determine a resting REE, and then multiplying REE by a factor of 1.3 to 1.7 (11). Protein administration of 2.5 to 3.0 g/kg/day will adequately support the energy needs of patients. The route of nutrition support is ideally *enteral tube feeding* started during resuscitation. Not only does this provide immediate protein and calorie needs, but enteral feeding may better protect the gut barrier, possibly decreasing the prevalence of bacterial translocation. Some children, in the case of very large burns during the early resuscitation period or those with intervening sepsis, will not tolerate enteral feeding at goal rates. Therefore, supplemental *parenteral nutrition,* in addition to enteral feeding at low rates, is justified to ensure delivery of all needed nutrients. The use of *anabolic agents* is an area of active investigation as well, as the use of human growth hormone. Available data does not seem sufficient to support their routine use (12).

Infection control

Infection of a burn wound and burn sepsis is the major problem in the treatment of burned children, and nowadays it is the leading cause of death. Host defense is extremely altered in both cellular and humoral components of the immune system. In burn wound 70 to 90% of infections are caused by auto contamination either from digestive or the respiratory tract, and in 65% of cases infection develops after cross contamination with highly resistant bacteria. *The American Burn Association* recently published the general criteria for burn sepsis and wound infection. They distinguish various types of burn wound infection like *colonization, wound infection, invasive infection, cellulites, and necrotizing fasciitis*. The main source of infection is the wound invasive and non-invasive infection, translocation of bacteria from the ischemic bowel, respiratory tract infection, and bloodstream and urinary tract infection (10).

Because the dominant commensal skin organisms are gram-positive cocci, organisms such as *Streptococcus and Staphylococcus aureus* tend to be early colonizers and the reason for manifested infection of the burn wound. Over time, especially if topical agents that act against gram-positive organisms are used, gram-negative organisms become dominant. One of the most common gram-negative pathogens is *Pseudomonas aeruginosa*, which tends to leave fluorescent yellow/green exudates on the burn wound. If gram-negative pathogens are controlled, then yeast (*Candida*) may appear next. Finally, more resistant bacteria and *fungi* will invade a burn wound. In BU, MRSA has emerged as a major cause of burn wound infection too. The multi-resistant gram-negative organisms of the *Acinectobacter and Aspergillus spp, and Herpes simplex* viruses are also being seen more often. The important clinical sings of a burn wound infection include redness and swelling, like in streptococcal cellulites, discoloration, and premature separation of burn eschara. Because of that, it is necessary to perform the daily monitoring of the burn wound and to recognize of the local sings of manifested infection such as conversion of a *partial-thickness burns (PTB) to full-thickness wound (FTB)*, worsening cellulites of surrounding normal tissue, eschara separation, and additional tissue necrosis. In the course of burn wound infection, gram-positive and gram-negative *burn sepsis* and *systemic inflammatory response syndrome (SIRS)* present a very important issue and must be treated on time and in the proper way (13).

The mainstays in treatment of burn wound infection include early reanimation and fluid resuscitation, early enteral feeding and higher calorie replacement diet, application of topical antibiotics in deep dermal burns, meticulous daily wound debridement, early tangential excision and skin grafting, hydrocolloid dressing in superficial dermal burns, and selective administration of antibiotics that depending on the results of antimicrobial analyses. The efficiency of many immune-protective measures in use (convalescent plasma, specific tetravalent Pseudomonas immune globulins, and recombinant granulocyte colony stimulating factors) has not been proved by large controlled studies. Selective bowel decontamination with Azytromicin has been shown to decrease burn wound colonization with gram-negative bacteria. Routine antibiotic prophylaxis is not recommended any more, whereas shot-term preoperative prophylaxis is still used in many centers (13).

Surgical management of burn wound and use of skin substitute

Early identification, excision, and closure of FTB change the nature history of the burn injury and are the leading subject of recent progress in burn care. The objective of this phase of care is to remove all necrotic tissue and to achieve immediately closure. In case of burns more than 25% TBSA early excision and wound coverage significantly decreases the mortality rate (14). When the excision is performed before the development of wound colonization and infection, it circumvents the development of burn sepsis and SIRS. Accurate knowledge of the probability that a wound will heal is central to the operative planning. The examination by an experienced burn surgeon remains the most reliable method, despite the many modern devices to measure the burn depth or burn blood flow (Sh-59.60). The changes in the wound appearance over the first few days after the burn injury make daily examinations a particularly useful method in surgical planning.

The overall *burn wound size* is the most important issue in determining the need for early surgery, as this correlates with the physiologic threat represented by the injury. Children with *small superficial burns* rarely develop burn sepsis. In small mixed-depth burns the burn surgeon must added time to allow the wound to fully evolve to a point at which it is easy to determine the burn depth during a physical examination. Burn wounds that heal spontaneously in 3 weeks or less are unlikely to become hypertrophic. A reasonable approach to that case is to treat them with topical antibiotic cream for 1 week to minimize desiccation and super-infection. Painful dressing changes are kept to a minimum, and painful debridement is not practiced. After 1 week the wound is usually clear, and if any deep areas of necrosis need to be excided and grafted it is then done. In this way all dermal burns, donor site and skin grafts are fully healed by 3 weeks, and children can return to their family and normal activities. In the case of *large burns* tangential excision must be performed as early as possible. All FTB have been excised and grafted during the first week post injury. As in small mixed depth wound, areas that heal in less than 3 weeks are left to do so. Superficial burns are daily debrided and covered with a biological dressing such as porcine xenograft, synthetic bilaminate - Biobrane, nanocrystaline silver dressing - Articoat, and hydrocolloid dressing - Aquacel Ag. When the wound is very large, >40% BSA, the treatment required a stages procedure. If the wounds involve >50% BSA it is not possible to achieve an immediate autograft closure. When autograft is exhausted, temporal biologic closure is achieved with human allograft or other temporal skin substitute. Wounds are later resurfaced with autograft when the donor sites have healed. In the case of electric burns, it is also better to perform prompt surgery excision, because of the appearance of distant necrosis with compartment syndrome.

The techniques of burn wound excision have evolved substantially and they included *tangential excision* and *excision to fascia*. Deep tissue necrosis should be managed with tangential (sequential, layered) excisions that optimize the finally appearance and function. Excision of the wound to fascia is used in the case of very large FTB or with very deep burns that extend into the fat or underlying tissue. Sheet skin grafts are used whenever possible, especially for covering the functional areas like hands or face. Usually, the amount of bleeding during these operations is in the range of 3.5-5% of the blood volume for every 1% of the body surface excise (14). Tangential excision of eschara has been very bloody and physiologically stressful, and blood loss during this operation should be minimized through the use of tourniquet, and tumescent infiltration of dilute epinephrine injection. The time in the operating room should be carefully controlled, again to avoid postoperative complications, extensive blood loss, and coagulopathy. A reasonable operating time limit, including general anaesthesia, is 2-3h. It is better to do several moderate operative procedures 1 or 2 days apart, than one massive procedure.

Skin substitutes

The covering of the excised surface can be performed with many different types of skin substitutes including fresh skin autografts, allografts (homografts), frozen or lyophilized human or animal skin, artificial tissues made of synthetic or biodegradable material and skin cells cultured in vivo. In the case of large excision split thickness autografts (STSG) may be limited in quantity, and a sandwich method with skin substitutes must be applied. In that particular case the wound is first covered with STSG, which have been previously meshed in ratio 2:1 or 4:1, and above them we put expanded homografts in ratio 2:1. Allografts are usually used with severe and critical burns where early excision of all deep burns must be performed, so that it is applied to save lives. If the burn was severe or critical, it is possible to use keratinocytes, which are in vitro-cultured epidermal autografts (CEA). A big problem with the usage of keratinocytes is the price of their production and fact that they are a very fragile and sensitive covering. Namely, if any friction affects is appearance after they have healed to the surface, vesicles filled with liquid rich in thromboxan and prostaglandins are formed, provoking an inflammatory reaction, which again inhibits re-healing of keratinocytes to the surface. As skin substitute have been applied Alloderm or Integra, which after having healed to the surface formed neodermis. The neodermis is then covered with CEA or very thin STSG (15).

Particularly critical are deep burns in *functional regions like neck, head, hands, face, feet and genitals*. Within the first 72h post-burn, an intensive monitoring of hand circulation is required because of possible compressive ischemia. In case of hand and finger compartment syndrome it is necessary to perform an urgent escharotomy and fasciotomy on the volar and dorsal side, and paramedially on fingers. The arm must be immobilized in a functional position, and elevated above the heart level in order to decrease edema. It is necessary to exercise the small hand joints two or more time per day. If deep dermal and subdermal burns are present, it is obligatory to perform an early tangenntial excision and sheet grafting. Skin grafts must be applied in proper way, on fingers longitudinally, and on the hand dorsum perpendiculary. The skin grafts usually healed from 7 to 10 days, and after that period it is necessary to initiate passive and active physical therapy in order to preserve the mobility of the small joints and thumb's opposition to other fingers (13).

Results

We have treated 51 burned children in the period from January 2003 up to December 2008. Boys were characteristically more often injured than girls, the ratio between male and female was 2.6:1. The youngest patient in our study was five months old, and the oldest one was 16 years old; main age was 7.2 years. The majority of patients were in the 1-7 year age category, with the remainder of patients ranging between 8-16 years of ages. As we expected, the age of the children directly impacted on the aetiology of the burn injury. Hot liquid burns or 21 (41.2%) cases dominated among all the burns. The dominating burns in the anatomical distribution have been the burns of the trunk and the limbs, 22 (43.1%). The size of the burns varied between 10 and 57% TBSA. There were 28 (54.9%) deep burns, and 23 (45.1%) superficial burns. Depending on the burn severity, there were 35 (68.6%) children without burn shock, 11 (21.6%) with burn shock, and 5 (9.8%) with burn shock and multiple organ failure. There were 17 (33.3%) cases with burn wound infection, and only 2 (3.9%) with burn sepsis.

We have treated 34 (66.7%) patients in the conservative surgical manner, which included daily debridement of the burn wound and application of topical antiseptic (1% silver sulfadiazine cream or 0.75 to 1% povidin iodine solution). The group of 17 (33.3%) children was operated with the method of early tangential excision and immediately skin grafting. As skin covering we used autograts and all available skin substitute i.e. pigs xenografs, and human allografts The duration of hospitalization was divided in four groups: I group (<14 days) 7 (13.7%), II group (15-21 days) 24 (47.1%), III group (22-28 days) 15 (29.4%), and IV group (>29 days) 5 (9.8%).

Discussion

Remarkable advances in burn care have been made over recent decades, and it is recognized that the organized efforts of burn teams is required to continue enhancing the survival rates and the quality of life for burned children. Children with major burns are unique, representing one of the most severe models of trauma, and therefore they have need treatment in the best specialized facilities i.e. BU and BC in children's hospitals.

The main pathophisiology issues of the *burn syndrome* includes fluid replacement, metabolic care, prevention of infection, and early burn wound excision with immediately wounds coverage. Increased capillary permeability and reduced plasma colloid osmotic pressure after burn injury result in hypovolemia and development of edema in the burn and non-burn tissues. The replacement of the intravascular deficit with crystalloid fluid has been the mainstay of resuscitation for more decades in the past time. A progressive but as yet unexplained trend towards provision of resuscitation volumes well in excess of those predicted by the Parkland formula, associated with numerous edema-related complications, has been recently repeatedly observed. The correction of this phenomenon, called "*Fluid creep*" named by Parkland, will likely revolve around several strategies, which may include tighter control of fluid titration, re-emergence of colloids and hypertonic saline solution, and possibly the use of adjunctive markers of resuscitation other than urinary output (1). Severe burn injury is followed by a profound *hypermetabolic response* that persists up to 24 months after injury (11). It is mediated by up to 50-fold elevations in plasma catecholamines, cortisol, and inflammatory cells that lead to whole body catabolism, elevating REE, and multiple organ failure. Modulation of the response by early excision and grafting of burn wound, thermoregulation, early and continuous enteral feedings with high protein high carbohydrate diets, and possible pharmacologic treatment have markedly decreased morbidity and the mortality rate. Because burns destroy the barrier against invading bacteria, *topical antimicrobial agents* have been developed to minimize the proliferation of bacteria and other microorganisms (13). The topical therapy depends on the depth of burns. The superficial burns usually healed in the optimal time during 2 weeks. For deep burns, topical antimicrobial agents should be used to minimize microbial growth until the wound in grafter. Early excision of the burn eschar has been one of the most significant advanced in modern burn care. The advances in understanding of the pathophysiology of burn injury and the systemic inflammatory response fuelled by the burn wound, have led to earlier *excision and grafting* of the burn wound with improvement in morbidity and mortality rate (14). Efforts to control blood loss, and good operating planning and attention to functional areas, can lead to the safe excision and grafting of large burns. Prompt excision of major burns significantly improves the overall survival, speeds up closure, and reduces the infection rate. The immediate covering with autografts is the natural method of definitive closure of these wounds. However, when harvesting of *donor skin* is unavailable, or the wound is not ready for autografting, a *temporary closure* with a variety of *skin substitutes* can help reduce evaporative loss, prevent infection, and ameliorate pain and metabolic stress. Fresh cadaver allograft is the gold standard for such closure, but other skin substitutes, including frozen cadaver skin, xenografts, and several synthetic products, are also available. In the last time, burn surgeons are in position to close the burn wound with *permanent substitutes,* which are synthetic or laboratory derived autologous composite. Once the complete closure of the burn wound is achieved the treatment emphasis shifts from burn wound management to rehabilitation (15).

Conclusion

The treatment of children burns requires a complex supportive therapy and surgical care consisting of early tangential excision and immediate skin grafting that may be temporary or permanent. Advances in the understanding of burn pathophysiology have led to improvements in the critical care that appears in paediatric burns. Continued studies in fluid resuscitation, infection control, support in hypermetabolic response and nutrition, wounds healing and scars management are main variables which leads with update paediatric burn care. Unfortunately, treatment must be performed in specialized institutions with BU of Surgical Clinics or in National BC. These institutions employ a team of plastic surgeons, anesthesiologists and reanimatologists, burn nurses, physiotherapists, psychologists, nutritionists, clinical microbiologists and other specialist who are specialized in treating burns in children.

LITERATURE

1. Demling RH. The burn edema process: Current concept. J Burn Care Res 2005; 26: 207-27.
2. Jeschke MG, Mlcak RP, Finnerty CC et al. Burn size determines the inflammatory and hypermetabolic response. Crit Care 2007; 11 (4): 90.
3. Brigham PA, McLughlin E. Burn incidence and medical care use in the United States: Estimates, trends, and data sources. J Burn Care Rehabil 1996; 17: 95-107.
4. Tucker P. The burn victim: A review of psychosocial issues. Aust NZJ Psychiatry 1986; 20: 413-20.
5. Aggarwal SJ, Diller KR, Blake GK et al. Burn-induced alterations in vasoactive function of the peripheral cutaneous microcirculation. I Burn Care Rehabil 1994; 15: 1-12.
6. Sheridan RL, Ryan CM, Petras LM et al. Burns in children younger than two years of age: An experience with 200 consecutive admissions. Pediatrics 1997; 100: 721-3.
7. Sheridon R. Burns. Crit Care Med 2002; 30 (1): 2-32.
8. Derganc M, Brčić A. Treatment of burned child: Fluid replacement, nutrition prevention and treatment of infection. 12. Delčevi padiatrični dnevi Ljubljana: Zbornik 1991; 309-22.
9. Carlson DE, Cioffi WG, Mason AD et al. Resting energy expenditure in patients with thermal injuries. Surg Gynecol Obstet 1992; 174: 270-6.
10. Rose KJ, Herdon DN. Advance in the treatment of burn patients. Burns 1997; 23 (1): 19-26.
11. Murphy KD, Lee JO, Herndon DN. Burn wound care and support of the metabolic response to burn injury and surgical supportive therapy. In Berret-Nerin JP, Herndon DN: Principles and practice of burn surgery. Marcel Dekker, New York 2005; 91-315.
12. Jacobs DG, Jacobs DO, Kudsky KA, Moore FA, Oswanski MF et al. The EAST Practice Management Guidelines Work Group. Practice management guidelines for nutritional support of the trauma patient. J Trauma Injury Infection and Critical Care 2004; 57: 660-79.
13. Randi L, Rutan RL, Benjamin D. Special consideration of age: The pediatric burned patient. In Herdon DN: Total Burn Care, 3rd ed. Horndon DN. Sounders, New York: 2007; 0-56.

14. Mosier MJ, Gibran NS. Surgical excision of the burn wound. In Cartotto R: Burns, Clin Plast Surg 2009; 4 (36): 617-26.

15. Sheridan R. Closure of the excised burn wound: Autograts, Semipermanent skin substitutes, and permanent skin substitutes. In Cartotto R: Burns, Clin Plast Surg 2009; 4 (36): 643-52.

Sažetak

SUVREMENO LIJEČENJE OPEKLINA DJECE: NADOKNADA TEKUĆINE, METABOLIČKE POTREBE, KONTROLA INFEKCIJE I KIRURŠKI TRETMAN OPEKLINSKE RANE

Z. Roje, Ž. Roje, M. Lojpur, J. Meštrović

Djeca, osobito ona mlađe životne dobi spadaju u rizičnu skupinu opečenih pacijenata. Broj opečene djece se smanjuje u većini Europskih zemalja, pa tako i u Hrvatskoj. Opekline djece se moraju zbrinjavati u Jedinicama za opekline Županijskih bolnica ili u Centru za opekline djece. Složena patofiziologija opeklinske bolesti i dalje predstavlja veliki terapijski izazov. Najučestalije su i dalje opekline vrelom tekućinom koje obično nastanu u kući, a čine više od 70% svih opeklina kod novorođenih, male djece i predškolskog uzrasta. Posljednji napredak u razumijevanju problematike opeklinskog šoka i infekcije opeklinske rane, intenzivne skrbi, kirurškog tretmana i zamjenske kože, imaju znakoviti upliv na četiri glavne poluge suvremenog liječenja opeklina. To su: nadoknada tekućine, metaboličke potrebe, prevencija infekcije i kirurški tretman. Kao i uvijek, nije jednostavno ostvariti gore navedene ciljeve. To zahtijeva multidisciplinarni zajednički rad kirurga, anesteziologa, pedijatra, psihijatra, anesteziologa, psihologa, fizijatra, radnog i fizikalnog terapeuta, socijalnog radnika i opeklinskih sestara. Bez koordiniranog rada uistinu nije moguće postići visoku razinu liječenja teških opeklina.

Deskriptori: OPEKLINE DJECE, NADOKNADA TEKUĆINE, METABOLIČKE POTREBE, KONTROLA INFEKCIJE, KIRURŠKI TRETMAN