Overview of Burns

Melvin A. Shiffman

1 Introduction

Burns can be one of the most devastating injuries, although ranging from first-degree burn that can be treated at home to third- and fourth-degree burns that can require extreme care both medically and surgically that can, hopefully, keep the patient alive but with major scars that may remain to be treated.

2 History

Cave paintings from more than 3500 years ago showed burns and their management [1]. The earliest Egyptian records on treating burns describe dressings prepared with milk from mothers of baby boys [2]. The 1500 BCE *Edwin Smith Papyrus* describes treatments using honey and the salve of resin [1]. Tea leaves were used by the Chinese as early as 600 BCE. Hippocrates described the use of pig fat and vinegar in 400 BCE, while Celsus used wine and myrrh in 100 CE [1]. Ambroise Paré was the first to describe different degrees of burns in the 1500s [3], and Guillaume Dupuytren developed the 6° classification of burns 1832 [4, 5].

James Syme established the first burn unit in Edinburgh in 1843 [6]. He argued that mixing burn patients with postoperative patients would make him "chargeable with the highest degree of culpable recklessness." This logic motivated the Edinburgh Royal Infirmary leadership to set aside the former high school janitor's house for burn patients. This experiment was relatively short-lived, however, since burn patients were transferred to one of the "sheds" in 1848 to make way for an increased number of mechanical trauma casualties from railway accidents. Glasgow Royal Infirmary had from 1833 to 1933 accumulated 100 years of experience with over 10,000 burn patients and in 1883 established a separate burn ward. In Dunbar's report on these patients, he commented that in the pre-antiseptic era, only the worst burns would come to the hospital; there were a biphasic mortality pattern (with the highest number of deaths between postburn hours 12–24), a high incidence of streptococcal wound infection, the infrequency of skin grafting, and a frustratingly high mortality rate of 20–30% despite the introduction of antisepsis.

During World War I, Henry D. Dakin and Alexis Carrel developed standards for the cleaning and disinfecting of burns and wounds using sodium hypochlorite solutions, which significantly reduced mortality [1]. In the 1940s, the importance of early excision and skin grafting was acknowledged, and around

M.A. Shiffman, M.D., J.D.
Private Practice, Tustin, CA, USA
e-mail: shiffmanmdjd@gmail.com

Recent Clinical Techniques, Results, and Research in Wounds (2017)
DOI 10.1007/15695_2017_20, © Springer International Publishing AG
Published Online: 24 October 2017

the same time, fluid resuscitation and formulas to guide it were developed. In the 1970s, researchers demonstrated the significance of the hypermetabolic state that follows large burns.

3 Types of Burns

1. Heat burns (thermal burns):
   Caused by fire, steam, hot objects, or hot liquids.
2. Cold temperature burns:
   Caused by skin exposure to wet, windy, or cold conditions.
3. Radiation burns:
   Caused by the sun, tanning booths, sunlamps, X-rays, or radiation therapy for cancer treatment.
4. Chemical burns:
   Caused by contact with household or industrial chemicals in a liquid, solid, or gas form.
5. Electrical burns:
   Caused by contact with electrical sources or by lightning.
6. Friction burns (abrasion and heat burns):
   Caused by contact with any hard surface such as roads ("road rash"), carpets, or gym floor surfaces.
7. Inhalation injuries:

Breathing in hot air or gases can injure the lungs.

4 Burn Degree [7, 8]

1. First-degree burns:
   Burns of the epidermis. Painful but no blisters and get better over 3–7 days
2. Second-degree burns:
   Superficial partial-thickness burns to the papillary layer. Pink, moist, and painful and forms blisters. Heal within 2–3 weeks without scarring
   Deep partial-thickness burns to the reticular layer. White, pink, or red but less painful and fairly dry. Slow capillary refill. Heal within 3–8 weeks with scarring
3. Third-degree burns (full-thickness burns):
   Injury to all the skin layers and subcutaneous layer. Leathery, no pain, and no capillary refill. Heal by epithelial migration from the periphery
4. Fourth-degree burns:
   Extend through the skin and subcutaneous tissues to injured muscle, ligaments, tendons, nerves, blood vessels, and bones. Black and necrotic

5 In this Article

5.1 Symptoms of Burns

The symptoms of burns depend on the cause and type of burn. They can include:

1. Red skin or pale and clammy skin.
2. Blisters with peeling.
3. Swelling.
4. Pain: The degree of pain is not related to the severity of the burn. The most serious burns can be painless.
5. Faintness.
6. Weakness.
7. Decreased alertness.
8. Dizziness.
9. Bluish lips and fingernails.
10. Muscle twitching.
11. Seizures.
12. White or charred skin.
13. Irregular heartbeat.
14. Shock.
15. Cardiac arrest.

6 Surface Area Assessment of Burns

Smart (1876) [9] noted that burn severity was determined by their size and depth as well as other bodily systems that were affected including airway.

6.1 Rule of Nines

The rule of nines was devised by Pulaski and Tennison in 1947 [10] and published by Wallace in 1951 [11].

The extent of burn injury to the skin can be estimated using the "rule of nines" (Fig. 1). This allocates approximate percentages to the major anatomical areas relative to the total body surface area (TBSA). The adult head and neck are allotted 9% of TBSA, each upper limb 9%, each lower limb 18%, each anterior and posterior surfaces of the trunk 18%, and perineum and urogenital structures 1%. The area of an adult palm and fingers is approximately 1% of TBSA, and

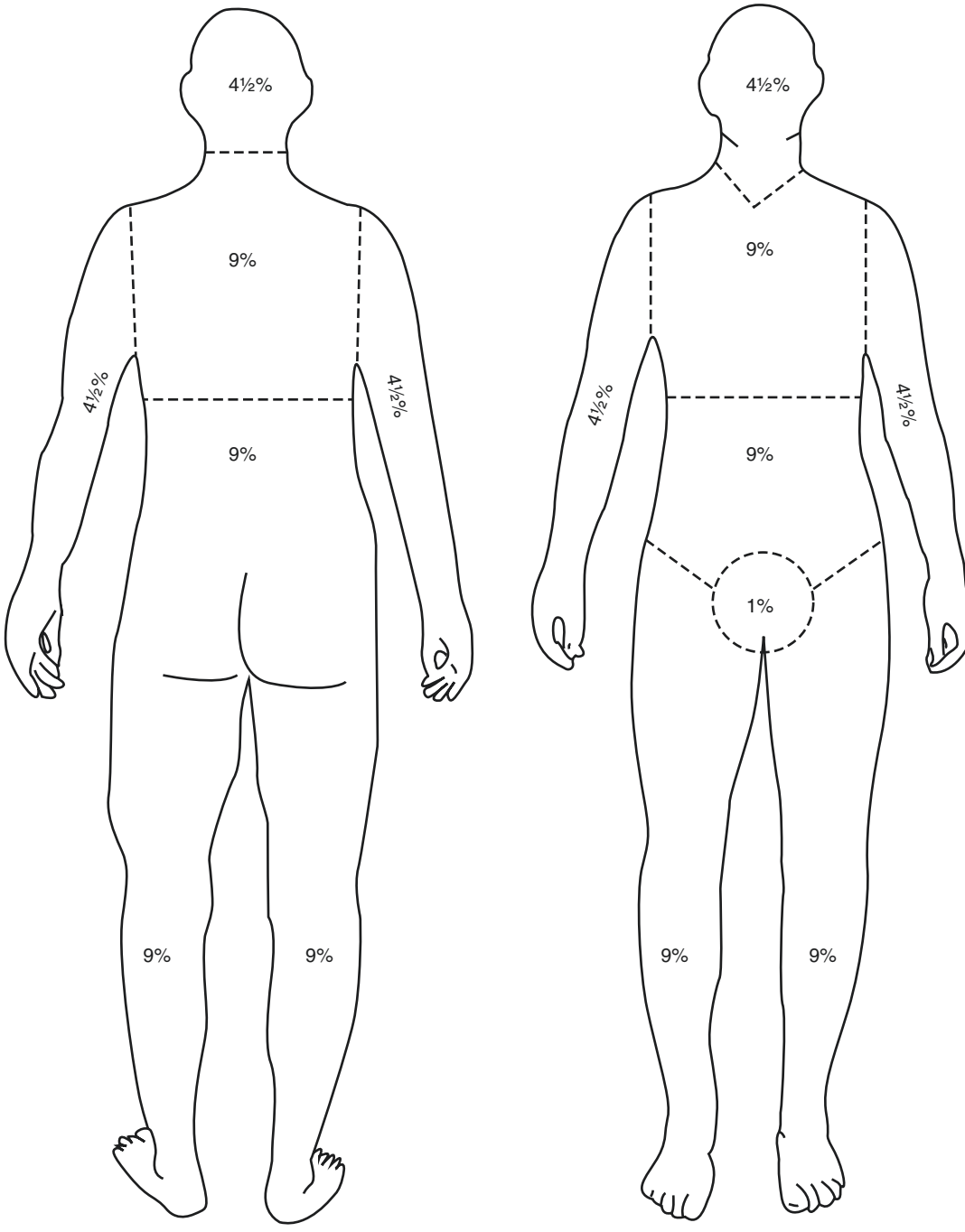

Fig. 1 Rule of nines

the total of multiple scattered areas of burn injury can be estimated using the open hand size as a guide.

6.2 Serial Halving

Serial halving is a prehospital assessment tool for burns. Using this method, the assessor decides if the area burned is greater than half of total body surface area (TBSA), between a quarter and half, between an eighth and a quarter, or less than an eighth. This can be useful in decisions about initial management and does not depend on knowledge of the proportion of the major anatomical areas relative to the TBSA.

Smith et al. (2005) [12] found no statistical difference between serial halving and the rule of nines as an initial assessment tool when determining dispositional. Serial halving has an inherent weakness when assessing certain sizes of burn. The rule of nines requires that the assessor knows and understands the proportionate areas of the body. The mathematics of percentages and fractions appeared to confuse some assessors. Simply stated, a burn from the wrist to the elbow is 50% of the arm, while a burn from the wrist to the mid-forearm is 25% of the arm. A burn from the feet to the nipple line is 75%.

6.3 Berkow Formula (Berkow's Table)

Berkow (1924) [13] described a method of estimating the extensiveness of lesions (burns and scalds) based on determining the percentage of total body surface affected by a burn. A method for the formula is derived from the rule of nines where certain body areas account for 9% each and the total body area is given a value of 99. The remaining 1% is the perineum. The age of the patient is taken into consideration when applying the Berkow formula. For example, the head of a 1-year-old child is proportionately larger than that of an adult; therefore, the 1-year-old's head would account for 19% of total body surface, while the head of an adult would account for 7%.

6.4 Lund and Browder Chart

A series of charts was produced by Lund and Browder in 1944 [14]. They are useful in the management of burns of children for estimating the total body surface area affected. It takes into consideration the age of the person, with decreasing percentage of body surface area (BSA) for the head and increasing percentage of BSA for the legs as the child ages. Children have smaller extremities but larger heads than adults.

More accurate assessment can be made using a Lund and Browder chart that maps the percentage total body surface area (TBSA) in a little more detail and includes some of the variations that occur with age, from birth to adulthood (Table 1).

The degree of burn can be drawn on Fig. 2.

7 Resuscitation Fluid

For calculation of resuscitation fluid, Jain (2014) [15] used the Berkow formula to determine the percent surface area burned.

A simplified version of the Parkland burn therapy fluid formula is described by

Table 1 Lund and Browder chart

Area	Age 0	Age 1	Age 5	Age 1	Age 15	Adult
1/2 of the head	9 1/2	8 1/2	6 1/2	5 1/2	4 1/2	3 1/2
1/2 of the thigh	2 3/4	3 1/4	4	4 1/2	4 1/2	4 3/4
1/2 of the lower leg	2 1/2	2 1/2	2 3/4	3	4 1/4	3 1/2

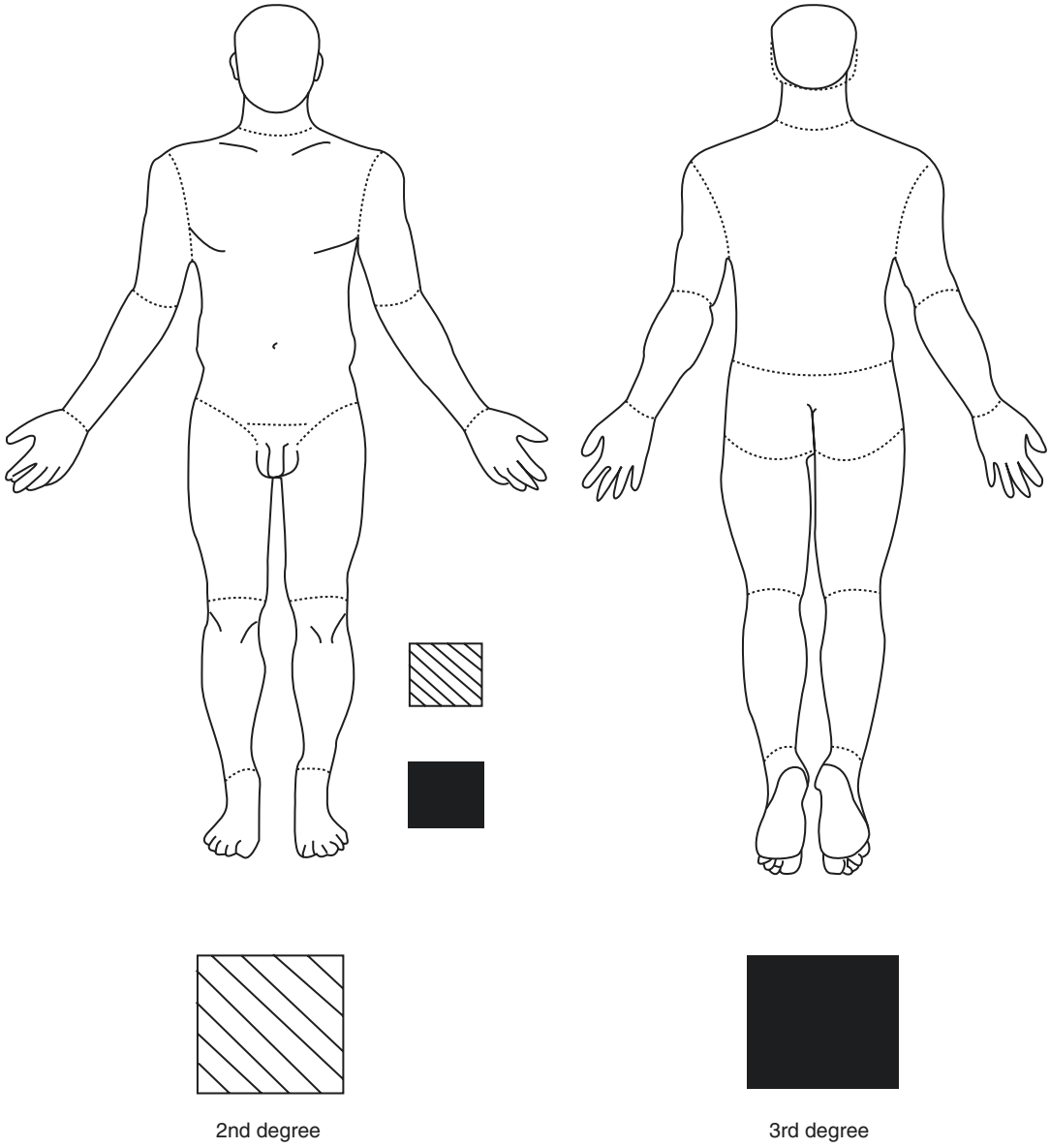

Fig. 2 Lund and Browder

Freshwater and Su (1979) [16]. The rule states that for the first 8 h after burn, the hourly rate of Ringer's lactate solution (in milliliters per hour) equals the patient's weight in pounds multiplied by the percent burn and divided by 9.

8 Indications for Referral to a Burns Unit [17]

1. All complex injuries should be referred.
2. A burn injury is more likely to be complex if associated with:

   (a) Extremes of age—under 5 or over 60 years
3. Site of injury:
   (a) Face, hands, or perineum
   (b) Feet (dermal or full-thickness loss)
   (c) Any flexure, particularly the neck or axilla
   (d) Circumferential dermal or full-thickness burn of limb, torso, or neck
4. Inhalational injury:
   (a) Any substantial injury, excluding pure carbon monoxide poisoning
5. Mechanism of injury:
   (a) Chemical injury >5% of total body surface area
   (b) Exposure to ionizing radiation
   (c) High-pressure steam injury
   (d) High-tension electrical injury
   (e) Hydrofluoric acid burn >1% of total body surface area
   (f) Suspicion of non-accidental injury
6. Large size (dermal or full-thickness loss):
   (a) Pediatric (<16 years old) >5% of total body surface area
   (b) Adult (≥16 years) >10% of total body surface area
7. Coexisting conditions:
   (a) Any serious medical conditions (cardiac dysfunction, immunosuppression, pregnancy)
   (b) Any associated injuries (fractures, head injuries, crush injuries)

9 Mortality in Burns

Schjerning (1884) [18] advanced the idea of the relation of mortality with burn size in 1884. He found that death always followed if two thirds of the body was burned, was to be expected if 50% of the body was burned, and generally occurred if a third of the body was burned.

9.1 Baux Score

The Baux score is a system used to predict the chance of mortality due to severe burns. The score is an index which takes into account the correlative and causal relationship between mortality and factors including advancing age, burn size and the presence of inhalational injury. Studies have shown that the Baux score is highly correlative with length of stay in hospital due to burns and final outcome.

9.1.1 Original Method

The original Baux score was proposed by Baux in 1961 [19]. Two factors were used. The first was the total body surface area affected by burning (usually estimated using the Wallace rule of nines or calculated using a Lund and Browder chart) and the second being the age of the patient. According to Roberts et al. (2012) [20], the Baux score continues to provide an indication of the risk of mortality.

9.1.2 Modified Baux Score

The modified score takes into account the effect of inhalation injury. It was found that inhalation injury resulted in an increase of around 17 on the Baux score, and this addition means that a patient with inhalation injury would have their score calculated by *body area affected* + *age of patient* + 17 [21]. Recent analysis of mortality in burn units worldwide has shown that for well-performing units the LD50 (the point at which 50% of patients would be expected to die) for major burns has significantly improved and the best units have a modified Baux score of 130–140. This means that all burns in children (except 100% TBSA full-thickness burns) should be considered survivable injuries and actively treated [22].

9.2 The Belgian Outcome in Burn Injury (BOBI)

The Belgian Outcome in Burn Injury (BOBI) prediction model consists of a 0–10 point score based on three major predictors for mortality: increasing age, total burned surface area, and the presence of inhalation injury [23]. This is an easy-to-use prediction model, which proved to be accurate in distinct populations with severe burn injury.

9.3 Charlson Comorbidity Index (CCI)

Heng et al. [24] reviewed the revised Baux score, Belgian Outcome in Burn Injury (BOBI) score, Abbreviated Burn Severity Index (ABSI), APACHE II score, Sequential Organ Failure Assessment (SOFA) score, and updated Charlson Comorbidity Index (CCI). Only the revised Baux score and the updated CCI were independently associated with shorter time to death. The data suggested that the revised Baux score and the updated Charlson Comorbidity Index (CCI) are independently associated with inpatient mortality in patients admitted to intensive care with burn injuries affecting ≥15% total burn surface area (TBSA). This emphasizes the importance of comorbidities in the prognosis of patients with severe burn injuries.

The Charlson Comorbidity Index (CCI) assesses the comorbidity risk associated to a series of conditions in order to offer medical specialists an informed decision-making process in terms of specific screenings or medical procedures [25]. The index accounts for the patient age and 16 conditions**.** This instrument is used to categorize comorbidities of patients and uses the International Classification of Diseases (ICD) diagnosis codes [26].

9.3.1 Charlson Comorbidity Index Scoring System

The CCI index predicts the 10-year mortality for patients presenting one or more of the conditions in the model. This is an index used in decision-making when a medical professional is presented with a treatment solution but needs to take into account the short- and long-term benefits of the treatment in a patient with other comorbidity conditions and should assess their long-term risk. Comorbidity is the term given to the presence of one or more additional conditions existing simultaneously, independently or not (with or without a causal effect), with a disease considered primary. It also suggests the effect of one or more additional conditions on the primary disease.

The age groups and each condition are awarded a specific number of points, some conditions weighing more than others, based on the adjusted risk of mortality (Table 2). The more points given, the more likely the predicted adverse outcome. The index then sums the points and offers a 10-year survival/mortality prognosis.

Table 2 Charlson Comorbidity Index scoring system

Score	Condition
1	Over 40 years of age: Divided into four age groups of different risk—under 50, between 50 and 59, between 60 and 69, and 70 or over Myocardial infarction: Patient or family Congestive heart failure Peripheral vascular disease peripheral artery occlusive disease and peripheral obliterative arteriopathy, including aortic aneurysm Cerebrovascular disease Dementia Chronic pulmonary disease Connective tissue disease Peptic ulcer disease Mild liver disease Diabetes without end organ damage (excludes diet controlled alone)
2	Hemiplegia Moderate or severe renal disease Diabetes with end organ damage or brittle diabetes Tumor without metastasis except over 5 years Leukemia Lymphoma
3	Moderate or severe liver disease
6	AIDS (not just HIV positive)

9.3.2 Calculating the Charlson Probability

This is the method through which the CCI score is transformed into a survival/mortality percentage: taking into account that C is the score result obtained by adding the points. For example, at a score of 6, the 10-year survival is 2.25%.

The list of comorbidities has been modified to 17 categories by Deyo et al. (1992) [27]. The list of specific ICD diagnosis codes that are used to identify different categories of comorbidity has been modified by Romano et al. (1993) [28] and updated from ICD-9-CM to work with ICD-10 coding by Quan et al. (2005) [29]. The original weights developed for use with the index have also been modified by Schneeweiss et al. (2003) [30].

There have been several variations to the index such as the Charlson-Deyo, Charlson-Romano, Charlson-Manitoba, and Charlson-D'Hoores comorbidity indices.

10 Treatment of Burns

The treatment of burns is dependent on the type and extent of the burns. A first-degree burn can be treated at home. The only symptom may be pain, and acetaminophen or ibuprofen may be used. The wounds are best treated with cool water for 5 min or longer to decrease the discomfort. If the patient still complains, lidocaine with aloe vera gel or cream may be used. At times, an antibiotic ointment with loose gauze can be applied especially on the knees, ankles, feet, spine, shoulders, elbows, and forearms. These will heal in 7–10 days.

With second-degree burns, there will be blisters and soreness. Pain medication such as acetaminophen or ibuprofen may be used. Running cool water on the region for about 15 min may help. Some blisters may burst and a wet or weeping appears. This can be treated with an antibiotic cream. Over time, thick, soft, scab-like fibrinous exudate may develop over the wound keeping the area clean, and bandaging it properly is required to prevent infection. Colloid (dextran or plasma) may be necessary. The wound heals in 3 weeks or longer. Rarely is a skin graft necessary.

Third-degree burns require hospitalization, and treatment is dependent on the extent of the area burned. There may not be significant pain except with dressing changes. If there is less than 15% of the body involved, fluid requirements are minimal and can be met with oral salt-soda solution containing 3 Gm of sodium chloride and 1.5 Gm of sodium bicarbonate [31].

In more extensive burns, the first 24 h should have colloid (whole blood), 0.5 mL per Kg body weight for each percentage of body surface burned, and electrolytes 1.5 mL per Kg body weight for each percentage of body surface burned. About 2000 mL of glucose should be administered. More than 50% burn is calculated as a 50% burn.

Extensively burned patients should have one half of the fluids in the first 8 h, and more colloids may be necessary. In the second 24 h, one half of the first 24 h of electrolytes and colloids should be infused with 2000 mL of glucose in water [31]**.** The rate of fluid administration depends on the blood pressure (if falling) and the urine output under 30 mL/h. If the urine output is more than 50 mL/h, the rate of administered should be slowed. The rate of infusion is slowed if the patient has respiratory involvement. The serum sodium level should not exceed 140 meq/L. From the 3rd to the 12th day, electrolyte imbalances occur and should be followed at least daily. When diuresis occurs, electrolyte-free water should be given in order to maintain the serum sodium at about 135 meq/L. Maintain the hematocrit at about 45%.

In extensively burned patients, the wounds should be cleaned thoroughly and debris and dead tissue removed. An occlusive dressing may be applied, but this may be conducive to infection because of the warmth and moisture. Changing dressings daily is painful, and morphine is usually required. The risks of general anesthesia may be necessary if the patient still cannot tolerate the dressing changes. Exposure of the area, if possible, allows the wound to be examined frequently for early detection of infection.

11 Complications

11.1 Metabolic Disturbances with Hypermetabolism

A burn of over 20% of the body is conducive to metabolic disturbances with hypermetabolism, increased gluconeogenesis, insulin resistance, increase in endogenous lipolysis, and loss of lean body mass [32, 33].

The nutritional requirements are particularly increased for glucose and proteins.

There is a maximal oxidation rate for glucose of 5 mg/kg/min for adults and children that should not be exceeded to avoid the development of fatty liver [34]. Protein requirements are increased to 1.5–2 g/kg/day.

Table 3 Calculation of caloric requirements in the patient with burn wounds can be assessed utilizing either the Curreri or Toronto formulas [35]

Curreri formula = 25 × body weight (kg) + 40 × % TBSA burned
Toronto formula = −4343 + 10.5 × % TBSA + 0.23 × previous 24 h caloric intake +0.84 × Harris-Benedict equation +114 × previous 24 h maximal temperature—4.5 × days postburn injury

Table 4 Derivation of IBW using the Devine formula and adjusted body weight (ABW)

Devine formula: Men—ideal body weight (in kilograms) = 50 + 2.3 kg per inch over 5 ft Women—ideal body weight (in kilograms) = 45.5 + 2.3 kg per inch over 5 ft
IBW (male) = 50 + (2.3 × (height [in inches]—60))
IBW (female) = 45.5 + (2.3 × (height [in inches]—60))
ABW = 0.25 × (actual body weight—IBW) + IBW

Enteral nutrition calculator may be used to calculate the appropriate tube feeding rate and supplemental protein required to meet a burn patient's nutritional needs. It provides the results of calculations using both the Curreri and Toronto formulas (Table 3). Protein requirements in the burn patient are estimated at 2 g/kg/day.

11.1.1 Determination of Ideal and Adjusted Body Weight

If the patient is greater than 20% above ideal body weight (IBW), the adjusted body weight should be used in the calculation of enteral nutrition rates for surgical and trauma patients. Derivation of IBW using the Devine formula and adjusted body weight (ABW) are shown in Table 4.

11.1.2 Trace Elements

Trace elements are lost in large amounts with the exudative losses in adults and children [36, 37]. The losses cause trace element deficiencies in patients with major burns, if not compensated (particularly selenium, zinc, and copper). Early substitution, with doses that represent six to ten times the recommended parenteral doses, is associated with improved wound healing and reduction of infective complications.

11.1.3 Further Treatment

Wolf et al. (2006) [38] found that propranolol, by attenuating the sympathetic response and its nonselective metabolic beta-2 blocking actions, strongly reduces catabolism and reduces cardiac workload in children and adults. Anabolic agents, such as insulin and oxandrolone, accelerate wound healing and shorten hospital stay. Oxandrolone started within 7 days of injury has been shown to improve long-term outcome in both adults and children in prospective randomized trials. Recombinant growth hormone (GH) probably has its place in the treatment of children (but not in adult burns patients) as children with burn injuries have a proven GH deficit.

The hypermetabolic state that may persist for years after a major burn can result in a decrease in bone density and a loss of muscle mass [39].

11.2 Multiple Organ Failure

Infective complications lead to multiorgan failure (MOF), poor outcome, and even death. Sheridan et al. (1998) [40] reported that burn patients dying in MOF did not have a high rate of positive blood cultures in the later phase of their care but succumbed during so-called "sterile" conditions. This finding was important as it underlined that infection is not always the immediate cause of death for this patient group. Some patients also develop sepsis, having had MOF of a different etiology. This led to the idea that burn injury and the inflammatory reaction secondary to it were important and significant contributors to the development of MOF in burn injury. Organ failure occurs in the lung (ARDS) and kidneys [41–43]. Mortality rates of patients with increasing number of organs failing are less than previously documented because of aggressive surgical approaches used in larger burn injuries and to a reduction in do not resuscitate (DNR) orders in some TBSA% ranges.

11.3 Delirium

Antipsychotic drugs are most effective in all types of delirium such as haloperidol

(0.5−10 mg), the dose to be reduced when symptoms improve, and alternatively or additionally olanzapine (5–10 mg) [44–46]. However, in delirium caused by alcohol or sedative hypnotic withdrawal, benzodiazepines are the treatment of choice, complemented in time by clonidine (600–1200 μg/day). Levomepromazine is not advocated to treat delirium in ICU patients.

11.4 Other Complications

A number of complications may occur, with infections being the most common [47]. In order of frequency, potential complications include pneumonia, cellulitis, urinary tract infections, and respiratory failure. Risk factors for infection include burns of more than 30% TBSA, full-thickness burns, extremes of age (young or old) or burns involving the legs, or perineum [48]. Anemia secondary to full-thickness burns of greater than 10% TBSA is common. Electrical burns may lead to compartment syndrome or rhabdomyolysis due to muscle breakdown. Pneumonia occurs commonly in those with inhalation injuries [49]. Blood clotting in the veins of the legs is estimated to occur in 6–25% of people. Keloids may form subsequent to a burn, particularly in those who are Hispanic, Asian, or African-American. Following a burn, children may have significant psychological trauma and experience post-traumatic stress disorder [50]. Scarring may also result in a disturbance in body image. In the developing parts of the world, significant burns may result in social isolation, extreme poverty, and child abandonment [51].

11.5 Prognosis

Deaths from burn injury increased with advancing age and burn size and the presence of inhalation injury. For patients under age 60 and with a TBSA between 0.1 and 19.9, the presence of inhalation injury increased the likelihood of death by nearly 24 times [52].

Table 5 Mortality rate by burn group size (TBSA) [52]

% TBSA	Mortality rate (%)
0.1–9.9	0.6
10–19.9	2.7
10–29.9	8.4
30–39.9	16.8
40–49.9	26.8
50–59.9	36.7
60–69.9	44.7
70–70.9	55.0
80–89.9	71.4

Table 5 shows the proportion of patients in each category of total burn size who died and the case fatality rate. This clearly increased with burn size. The burn size associated with a 50% case fatality (LD50) appears to be approximately 65–70% TBSA [52].

Conclusions

Care of the patient with burns is determined by the total body surface area that is burned and the degree of the burn as well as the age of the patient and whether there is concomitant lung injury from the burn. Secondary problems such as medical disorders can add to the patient's risks of mortality.

Proper and knowledgeable care of the burn and the patient will make all the difference in the world.

References

1. Herndon D (2012) A brief history of acute burn care management. In: Herndon D (ed) Total burn care, 4th edn. Saunders Elsevier, Edinburgh, p 24
2. Pećanac M, Janjić Z, Komarcević A, Pajić M, Dobanovacki D, Misković SS (2013) Burns treatment in ancient times. Med Pregl 66(5–6):263–267
3. Song D, Neligan PC (2012) Plastic surgery, 3rd edn. Saunders, Edinburgh, p 393
4. Dupuytren G (1833) Clinical Lectures in Surgery, Delivered at Hotel-Dieu in 1832. In: Doane AS, transl. Boston, Carter, Hendee, p 234–236
5. Wylock P (2010) The life and times of Guillaume Dupuytren, 1777–1835. Brussels University Press, Brussels, p 60

6. Cancio LC, Wolf SC (2012) A history of burn care. In: Jeschke MG, Kamolz LP, Sjöberg F, Wolf SE (eds) Handbook of burns. Springer-Verlag, Wien, pp 1–17
7. Khan A, Solan M. Burns: types, treatments, and more. www.healthline.com/health/burns. Accessed 24 Sep 2016
8. Blahd WH Jr, O'Connor M. Burns and electric shock—topic overview. http://www.webmed.com/first-aid/tc/burns-topic-overview. Accessed 24 Sep 2016
9. Smart C (1876) On burns by gunpowder and scalds by steam. Lancet 2(2769):421–422
10. Wallace rule of nines. Wikipedia, the free encyclopedia. https://en.wikipedia.org/wiki/Wallace_rule_of_nines. Accessed 8 Oct 2016
11. Wallace AB (1951) The exposure treatment of burns. Lancet 1(6653):501–504
12. Smith JJ, Malyon AD, Scerri GV, Burge TS (2005) A comparison of serial halving and the rule of nines as a pre-hospital assessment tool in burns. Br J Plast Surg 58(7):957–967
13. Berkow SG (1924) A method of estimating the extensiveness of lesions (burns and scalds) based on surface area proportions. Arch Surg 8:138–148
14. Lund CC, Browder NC (1944) The estimation of areas of burns. Surg Gynecol Obstet 79:352–358
15. Jain V (2014) Review of preventive & social medicine (including biostatistics). Jaypee Brothers Medical Publishers, New Delhi
16. Freshwater MF, Su CT (1979) The second rule of nines: a guide for resuscitation of burn patients. Ann Plast Surg 2(4):298
17. Hettiaratchy S, Papini R (2004) ABC of burns: Initial management of a major burn: II—assessment and resuscitation. BMJ 329(7457):101–103
18. Schjerning (1884) About the death as a result of burning and scalding from the court physician standpoint. 42:24–66, 273–300
19. Baux S (1961) Contribution a l'etude du traitement local des brulures thermique etendues. Paris, France, Thesis
20. Roberts G, Lloyd M, Parker M, Martin R, Philp B, Shelley O, Dziewulski P (2012) The Baux score is dead. Long live the Baux score: a 27-year retrospective cohort study of mortality at a regional burns service. J Trauma Acute Care Surg 72(1):251–256
21. Granger J, Estrada C, Abramo T (2009) An evidence-based approach to pediatric burns. Pediatr Emergency Med Pract 6(1):1–18
22. Osler T, Glance LG, Hosmer DW (2010) Simplified estimates of the probability of death after burn injuries: extending and updating the Baux score. J Trauma 68(3):690–697
23. Brusselaers N, Monstrey S, Blot S (2010) Predictive value of the Belgian outcome in burn injury (BOBI) prediction model. Burns 36(8):1318–1320
24. Heng JS, Clancy O, Atkins J, Leon-Villapalos J, Williams AJ, Keays R, Hayes M, Takata M, Jones I, Vizcaychipi MP (2015) Revised Baux score and updated Charlson comorbidity index are independently associated with mortality in burns intensive care patients. Burns 41(7):1420–1427
25. Charlson ME, Pompei P, Ales KL, MacKenzie CR (1987) A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. J Chronic Dis 40(5):373–383
26. Frenkel WJ, Jongerius EJ, Mandjes-van Uitert MJ, van Munster BC, de Rooij SE (2014) Validation of the Charlson comorbidity index in acutely hospitalized elderly adults: a prospective cohort study. J Am Geriatr Soc 62(2):342–346
27. Deyo RA, Cherkin DC, Ciol MA (1992) Adapting a clinical comorbidity index for use with ICD-9-CM administrative databases. J Clin Epidemiol 45(6):613–619
28. Romano PS, Roos LL, Jollis JG (1993) Adapting a clinical comorbidity index for use with ICD-9-CM administrative data: differing perspectives. J Clin Epidemiol 46(10):1075–1079
29. Quan H, Sundararajan V, Halfon P, Fong A, Burnand B, Luthi JC, Saunders LD, Beck CA, Feasby TE, Ghali WA (2005) Coding algorithms for defining comorbidities in ICD-9-CM and ICD-10 administrative data. Med Care 43(11):1130–1139
30. Schneeweiss S, Wang PS, Avorn J, Glynn RJ (2003) Improved comorbidity adjustment for predicting mortality in Medicare populations. Health Serv Res 38(4):1103–1120
31. Artz CP, Soroff HS (1955) Modern concepts in the treatment of burns. J Am Med Assoc 159(5):411–417
32. Cunningham JJ (1990) Factors contributing to increased energy expenditure in thermal injury: a review of studies employing indirect calorimetry. JPEN J Parenter Enteral Nutr 14(6):649–656
33. Jeschke M, Norbury WB, Finnerty CC, Mlcak RP, Kulp GA, Branski LK, Gauglitz GG, Herndon B, Swick A, Herndon DN (2008) Age differences in inflammatory and hypermetabolic postburn responses. Pediatrics 121(3):497–507
34. Masters B, Aarabi S, Sidhwa F, Wood F (2012) High-carbohydrate, high-protein, low-fat versus low-carbohydrate, high-protein, high-fat enteral feeds for burns. Cochrane Database Syst Rev 1:CD006122
35. Burn Nutrition Calculator. http://www.surgicalcriticalcare.net/Resouces/burn_nutrition.php. Accessed 4 Oct 2016
36. Berger MM, Shenkin A (2007) Trace element requirements in critically ill burned patients. J Trace Elem Med Biol 21(Suppl 1):44–48
37. Berger MM, Baines M, Raffoul W, Benathan M, Chiolero RL, Reeves C, Revelly JP, Cayeux MC, Sénéchaud I, Shenkin A (2007) Trace element supplementation after major burns modulates antioxidant status and clinical course by way of increased tissue trace element concentrations. Am J Clin Nutr 85(5):1293–1300

38. Wolf SE, Edelman LS, Kemalyan N, Donison L, Cross J, Underwood M, Spence RJ, Noppenberger D, Palmieri TL, Greenhalgh DG, Lawless M et al (2006) Effects of oxandrolone on outcome measures in the severely burned: a multicenter prospective randomized double-blind trial. J Burn Care Res 27(2):131–139
39. Rojas Y, Finnerty CC, Radhakrishnan RS, Herndon DN (2012) Burns: an update on current pharmacotherapy. Expert Opin Pharmacother 13(17):2485–2494
40. Sheridan RL, Ryan CM, Yin LM, Hurley J, Tompkins RG (1998) Death in the burn unit: sterile multiple organ failure. Burns 24(4):307–311
41. Cumming J, Purdue GF, Hunt JL, O'Keefe GE (2001) Objective estimates of the incidence and consequences of multiple organ dysfunction and sepsis after burn trauma. J Trauma 50(3):510–515
42. Fitzwater J, Purdue GF, Hunt JL, O'Keefe GE (2003) The risk factors and time course of sepsis and organ dysfunction after burn trauma. J Trauma 54(5):959–966
43. Steinvall I, Bak Z, Sjoberg F (2008) Acute respiratory distress syndrome is as important as inhalation injury for the development of respiratory dysfunction in major burns. Burns 34(4):441–451
44. Lonergan E, Britton AM, Luxenberg J, Wyller T (2007) Antipsychotics for delirium. Cochrane Database Syst Rev 2:CD005594
45. Devlin JW, Skrobik Y (2011) Antipsychotics for the prevention and treatment of delirium in the intensive care unit: what is their role? Harv Rev Psychiatry 19(2):59–67
46. Burns A, Gallagley A, Byrne J (2004) Delirium. J Neurol Neurosurg Psychiatry 75(3):362–367
47. Herndon D (2012) Epidemiological, demographic, and outcome characteristics of burn injury. In: Herndon D (ed) Total burn care, 4th edn. Saunders, Edinburgh, p 23
48. King C, Henretig FM, King BR, Loiselle J, Ruddy RM (2008) In: Wiley JF II (ed) Textbook of pediatric emergency procedures, 2nd edn. Lippincott Williams & Wilkins, Philadelphia, p 1077
49. Brunicardi FC (2010) Burns. In: Brunicardi FC, Andersen D, Billiar T, Dunn D, Hunter J, Matthews J, Raphael E, Pollock RE (eds) Schwartz's principles of surgery, 9th edn. McGraw-Hill, New York
50. Roberts MC (2009) Handbook of pediatric psychology, 4th edn. Guilford, New York, p 421
51. Peck MD (2011) Epidemiology of burns throughout the world. Part I: distribution and risk factors. Burns 37(7):1087–1100
52. American Burn Association. 2015 National Burn Repository: Report of Data from 2005–2014. p. 10