Chapter 10

A BRIEF HISTORY AND THE PATHOPHYSIOLOGY OF BURNS

WILLIAM G. CIOFFI, Jr., M.D.,* LORING W. RUE III, M.D.,** TERESA M. BUESCHER, M.D.,*** AND BASIL A. PRUITT, Jr., M.D., FACS****

*Major, United States Army; Chief, Burn Study Branch, United States Army Institute of Surgical Research
**Captain, United States Army; Burn Study Branch, United States Army Institute of Surgical Research
***Major, United States Army; Burn Study Branch, United States Army institute of Surgical Research
****Colonel, United States Army; Commander and Director; United States Army Institute of Surgical Research, Fort Sam Houston, San Antonio, Texas 78234-5012

A BRIEF HISTORY OF BURN CARE

Burns have been recognized as threats to our well-being since fire was discovered. The Ebers and Smith Papyri (about 1600–1500 B.C.) contain the earliest known accounts of the treatment of burns. They describe the agents that the ancient Egyptians used, which included various oil preparations, plant extracts, and amalgams of animal tissue, and emphasize their proper sequence of application. The recommendation in the Ebers papyrus that a warmed, oiled frog be rubbed on the burn wound may establish a certain ancestral claim for not only topical therapy but also the brief application of a biological dressing. More importantly, these early accounts stress the need for regular wound examination, because the application of these various treatment agents depended upon the wound's appearance.

The Greeks in the fifth century B.C. stressed the importance of avoiding suppuration in the burn wound, and thought that cleansing and proper care of the wound were of paramount importance. The Roman physician Cornelius Celsus referred to burn care in his book *De Medicina.* This work is also notable for the earliest reference to the surgical care of burns, specifically the excision of burn scars. As the ancient Egyptians had done, Greek and Roman physicians employed poultices of various animal tissues—and even animal excreta—to care for burn wounds.

When gunpowder was introduced to warfare, the incidence of burn wounds sustained during conflict greatly increased. This stimulated a number of surgeons such as Ambroise Paré to propose various treatment regimens for gunpowder burns and projectile wounds. The first systematic evaluation of burns was outlined in Guilhelmus Fabricius Hildanus's book De *Combustionibus* in **1607.** Hildanus, the father of German surgery, classified burns on the basis of their appearance; he was probably the first to ascribe the variations in wound appearance to the depth of the burn. He described the most superficial burns as red and blistered, deeper burns as withering but not charring the skin, and the deepest as those with eschar and charred skin.

Treating the Burn Wound

Modern care of burns involves either closed dressings or open exposure. In 1887, the Alabama surgeon W. P. Copeland first proposed an open technique of burn-wound treatment. He stressed a marked improvement in patient comfort, and many other physicians enthusiastically adopted this approach. The exposure method fell into disfavor because of the unavoidable problems of infection, and it was not until the late **1940s** that Wallace of Edinburgh reintroduced the notion of open wound care, which is used by many centers today.

Local burn-wound care was greatly advanced when topical antimicrobial agents were introduced. These limited microbial proliferation and invasion through the burned tissue, and significantly reduced mortality due to invasive burn wound sepsis. In **1965,** Moyer described using a solution of 0.5% silver nitrate applied as soaks, contemporaneously, at the U.S. Army Institute of Surgical Research, Moncrief described using topical mafenide acetate (Sulfamylon) burn cream. Subsequently, *Fox* introduced silver sulfadiazine in **1969.** The effectiveness of each of these three agents has been documented through extensive clinical use, and together they comprise the therapeutic armamentarium available for topical antimicrobial therapy of burn wounds.

Developments in Fluid Resuscitation Therapy

Baron Dupuytren was the first physician to describe the importance of fluid therapy in burns, and made the first statistical study of burn patients in 1828, when he reported the mortality of fifty patients treated at the Hotel Uieu based upon their age, sex, and extent of burn. In **1854,** Buhl made the association between the hemoconcentration that occurred in burn patients and that in cholera patients, due to their fluid loss. Modern concepts of fluid resuscitation did not appear until the beginning of the twentieth century, when the Sicilian physician Tomasoli, in **1897,** and the Italian physician Parascandolo, in **1901,** described treating severe burns with saline infusions. The first modern scientific evaluation of fluid management was conducted by researchers at Yale University under the direction of Dr. Frank Underhill. This study was based on about twenty patients who were burned in the **1921** Rialto Theater fire in New Haven, Connecticut. These investigators demonstrated that burn shock was the consequence of fluid and electrolyte shifts that occurred after the thermal injury, a concept that served as the

basis for resuscitative fluid therapy following a thermal injury.

Subsequent studies by Cope and Moore, performed while treating patients who were burned in the **1942** Coconut Grove nightclub disaster in Boston, provided further information on the fluid, electrolyte, and protein losses that developed after a thermal burn They demonstrated that the fluid losses that caused hypovolemia not only occurred through the burn wound itself, but also were a consequence of internal fluid shifts throughout the body. They concluded that large volumes of salt-containing solutions were important for adequate fluid resuscitation and proposed a "burn-budget" formula for estimating the fluid needs of patients with extensive burns, relating the patient's colloid-containing fluid needs to body weight.

Another specific formula for determining fluid requirements after a burn injury was reported by Evans in **1952.** This formula, based upon the surface area of the burn and the weight of the patient, resulted from a combined research effort between the Medical College of Virginia and the U.S. Army Medical Research and Development Command. The Brooke formula, proposed in **1953,** was a modification of the Evans formula. These early formulae recommended administering a volume of colloid-containing fluid that equalled or exceeded the amount of the crystalloid fluid component. It remained for Curtis P. Artz and associates at the U.S. Army Institute of Surgical Research to demonstrate the relative importance of crystalloid fluid and recommend a 3:1 ratio of crystalloid to colloid-containing fluid as the Brooke formula. In recent years, additional resuscitation regimens, such as the Parkland formula, the modified Brooke formula, and the hypertonic salt formula have been recommended to avoid the complications of either over- or under-resuscitation that were associated with the early formulae.

Advances in Skin Grafting

the first century A.D., first **mentioned a surgical approach to burn injury, it was** not until the **1870s** that George Pollock performed the first free skin graft to treat a chronic burn wound. Wilms first excised a burn wound in the early twentieth century; he used skin grafting for later closure of the excised areas. The usual approach to the closure of bum wounds was *(a)* local debridement, to permit an adequate bed of granulating tissue to *(b)* subsequent skin grafting. Dr. John Stage Davis, of The Johns Hopkins University, first employed the pinch-graft technique in **1914.** This technique employed the application of many small (approximately **1** thickness grafts, which were applied to a wound bed of granulation tissue, with the expectation that these grafts would eventually expand to cover the wound. Subsequently, surgeons observed that broad, thin sheets of split-thickness graft provided not only more **rapid closure** of **the wound** but also **were less disfig**uring cosmetically than the pinch-graft technique.

Blair first popularized the use of a long, thin knife to obtain these skin grafts. Padgett, Hood, and Reese improved on this and developed drum-type dermatomes. Dr. Harry Brown, while he was a prisoner of war in the Philippines during World War II, conceived the notion of an electric dermatome, and this instrument has greatly facilitated the harvesting **of** split-thickness skin grafts. In the early 1960s, Tanner developed a meshing device that permits up to a ninefold expansion of split-thickness skin grafts and reduces the need for multiple reharvesting of the few available donor sites on patients with extensive burns.

In 1970, Janzekovic described tangential excision, used to remove only the eschar and to produce a wound bed of bleeding dermis or viable subcutaneous fat, which would be immediately covered by split-thickness skin grafts. Although no prospective randomized studies have documented statistically that excising the burn wound improves the survival of extensively burned patients, tangential excision has been widely employed, and is now considered integral to bum-wound care.

Wound closure becomes a problem when the extent of the full-thickness burns exceeds the area of available donor sites. A variety of biological dressings and skin substitutes have been used to overcome such wound-to-donor-site disproportion and to provide temporary coverage of the burns until the donor sites can be reharvested. Allograft skin, first used by the French surgeon Riverdin in **1869,** is the standard of biological dressings, but its storage requirements, limited shelf life, limited availability, and the possible transmission of disease from cadaver to recipient restrict its use. Other biological dressings have been used to avoid or overcome these limitations and to **provide temporary coverage when autografting is not** possible. In **1682** Canady reported on the use of water-lizard skin for wound closure and, since then, the membranous lining of eggs, and the skins of chickens, guinea pigs, rabbits, and pigs, and amniotic membranes have been used in the treatment of burn wounds. Porcine cutaneous xenograft, the most commonly used **biological dressing,** achieves **biological** union when infiltrated by the host's fibroblastic tissue and, if it is left in place long enough, it undergoes necrotic slough but not a true rejection reaction.

Burn Research and Modern Treatment

Studies that clinicians and investigators at the U.S. Army Institute of Surgical Research have performed over the past four decades have yielded many additional improvements in burn care, including:

- **documenting the pathophysiology** of **tissue** injury due to electric current, which has led to improved care of patients with high-voltage electrical injury
- demonstrating the complications of using copper sulfate to treat white phosphorus burns, which has led to abandoning that treatment modality
- identifying the stereotypical gastrointestinal-mucosal response to burn injury that defined the central role of intraluminal acid in the pathogenesis of Curling's ulcers; antacid prophylaxis has essentially eliminated that cause of life-threatening upper-gastrointestinal hemorrhage in burn patients
- developing accurate diagnostic techniques that have permitted the early identification **of** inhalation injury in burn patients and the **prompt initiation** of therapy
- identifying the neurohormonal changes that orchestrate postburn hypermetabolism, in an extensive program of metabolic studies conducted by Soroff, Wilmore, Mason, and others, which has prompted the development of effective metabolic-support regimens
- revealing the immunosuppressive effects of burn injury on all limbs of the immune system, which serve as the basis for present evaluations of immunomodulation therapy

THE **PATHOPHYSIOLOGY OF BURNS**

Assessing the Extent **of** Thermal **Burns**

Various parts of the body account for roughly 9% (or a multiple) **of** the total body surface, and burn size can be estimated using the Rule of Nines: **9%** of the body surface for the head and each upper extremity, 18% for each lower extremity, the anterior trunk, and the posterior trunk, and 1% for the perineum (Figure 10–1). The patient's palm represents approximately 1% **of** the body **surface area, and that relationship can** be used to estimate the size of small, irregular burns. **A** Lund-Browder diagram, which takes into consideration age-related differences in the proportions **of** body parts, allows a more accurate burn-size estimate, and is used by most burn centers (Figure 10-2). The patient's weight must be either measured or estimated.

The **severity** of thermal injury in terms of both the casualty's physiological response and survival depends upon *(a)* the size and depth of the injury, *(b)* the casualty's age, and *(c)* the presence or absence of inhalation injury. The LA_{50} (that is, the lethal area or extent of burn that is associated with a 50% mortality) is **53%** for ages 0–14 years, 76% for ages 15-40 years, and 44% for those older than 40.[1] Depending upon the age of the patient and the extent of the burn, mortality may increase by as much as 20% if inhalation injury is present.* (These survival figures are based upon peacetime data with all patients receiving full care at a tertiary referral center that is utilizing essentially un

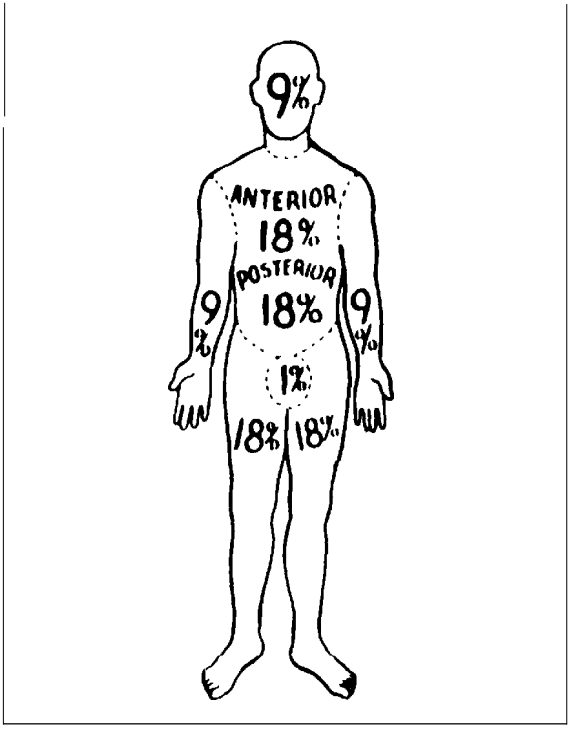
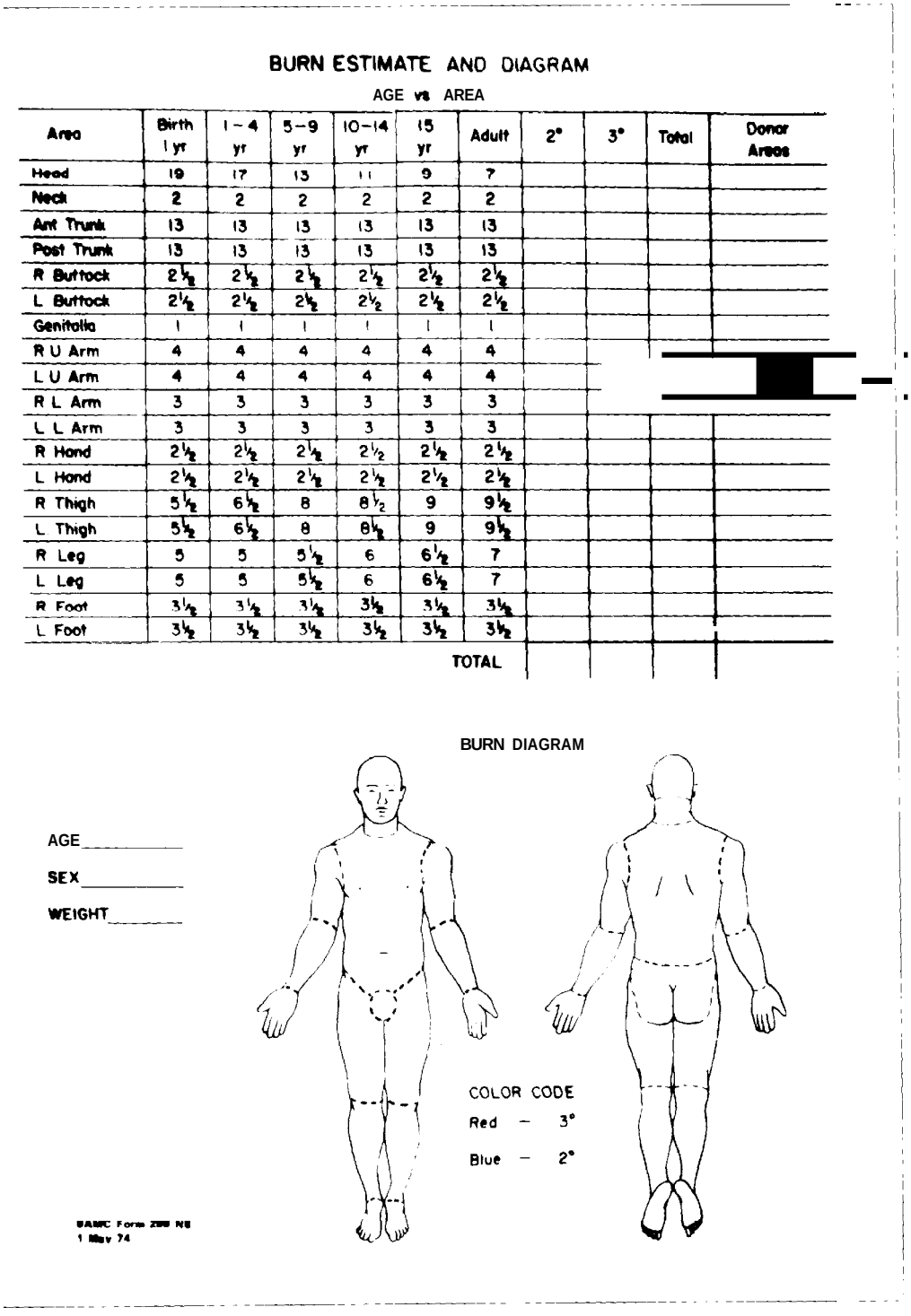


Fig. 10-1. The Rule of Nines can be used to estimate burn size. The surface area **of** various anatomical regions represent 9% (or multiples) of the body surface.



BURN ESTIMATE AND DIAGRAM

AGE vs AREA

Area	Birth 1 yr	1–4 yr	5–9 yr	10–14 yr	15 yr	Adult	2°	3°	Total	Donor Areas
Head	19	17	13	11	9	7				
Neck	2	2	2	2	2	2				
Ant Trunk	13	13	13	13	13	13				
Post Trunk	13	13	13	13	13	13				
R Buttock	2½	2½	2½	2½	2½	2½				
L Buttock	2½	2½	2½	2½	2½	2½				
Genitalia	1	1	1	1	1	1				
R U Arm	4	4	4	4	4	4				
L U Arm	4	4	4	4	4	4				
R L Arm	3	3	3	3	3	3				
L L Arm	3	3	3	3	3	3				
R Hand	2½	2½	2½	2½	2½	2½				
L Hand	2½	2½	2½	2½	2½	2½				
R Thigh	5½	6½	8	8½	9	9½				
L Thigh	5½	6½	8	8½	9	9½				
R Leg	5	5	5½	6	6½	7				
L Leg	5	5	5½	6	6½	7				
R Foot	3½	3½	3½	3½	3½	3½				
L Foot	3½	3½	3½	3½	3½	3½				
						TOTAL				

BURN DIAGRAM

AGE_____

SEX_____

WEIGHT_____

COLOR CODE

Red – 3°

Blue – 2°

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Fig. 10-2. **A** Lund-Browder diagram permits more precise estimates of burn size, based on age-related changes in anatomical surface area.

limited resources. They may not pertain in wartime.)

During combat, available resources must be expended on those individuals with the greatest chance for survival. Casualties at the extremes of age (that is, those under 10 or over 60 years old) or those with 70% of their total body surface area burned (TBSAB) will, in general, do poorly, and should be allocated a lesser share of the resources. The care of those casualties with less than 20% TBSAB can safely be delayed pending either their evacuation to a higher-echelon medical treatment facility (MTF) or the availability of more resources?

Depth of Burns

The depth of thermal injuries may vary from involving only the superficial epidermal elements to the entire epidermis, the entire dermis, and even subcutaneous tissues (Figures 10-3, 10-4, 10-5, and Table 10-1). Superficial partial-thickness burns (that is, first-degree injuries) are erythematous, painful, involve only the superficial layer of the epidermis, and commonly heal within 3–5 days. Medium to deep partial-thickness burns (that is, second degree injuries) are also erythematous, quite painful, and frequently have bullae. Because some epidermal elements such as hair follicles and sweat glands remain uninjured, these burns, if protected from infections, will heal primarily, although the time required for their healing increases with the depth of the injury. Full-thickness burns (that is, third-degree injuries) are white or charred, leathery textured, insensate, and involve the entire thickness of the epidermis and dermis.

Often, the depth of the injury cannot be ascertained immediately. Thermal injury results in concentric rings of varying degrees of tissue damage. A central zone of necrosis is surrounded by a zone of stasis, which, depending on the adequacy of the resuscitation, can either remain viable or proceed to cell death. This, in turn, is surrounded by a zone of hyperemia (Figure 10-6).[4]

Physiological Response to Thermal Injury

Thermal burns lead to alterations in the function of all organ systems. Their magnitude and duration are proportionate to the size of the burn, but reach a 50% 60% of the body is burned (Figure 10-7).[5]

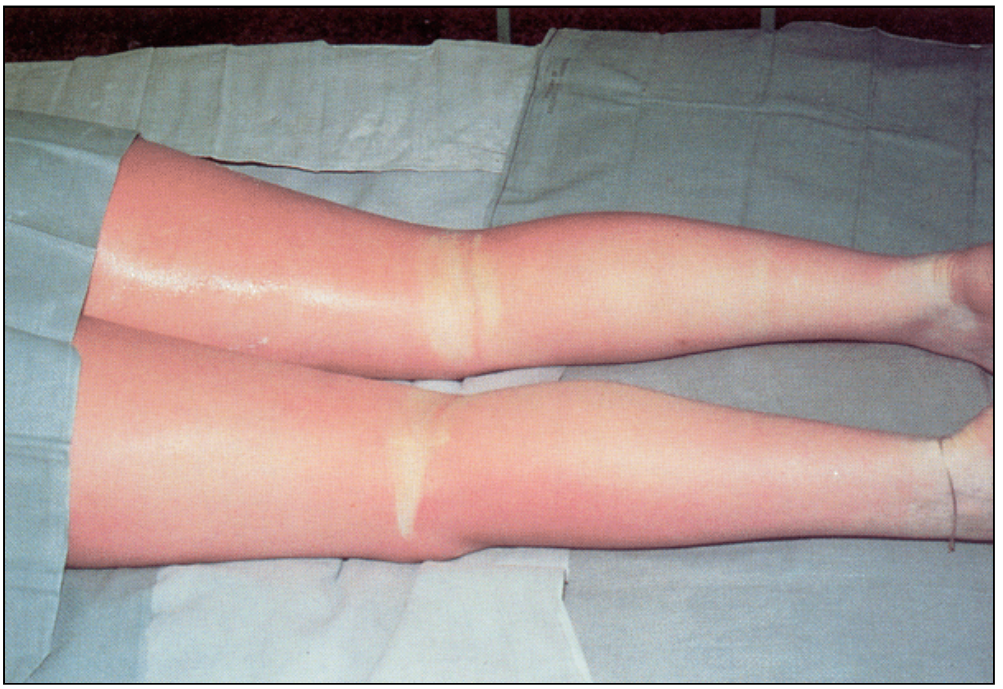

Fig. 10-3. A severe first-degree burn. The characteristic erythematous lesions (usually a sunburn) involve only the epidermis.

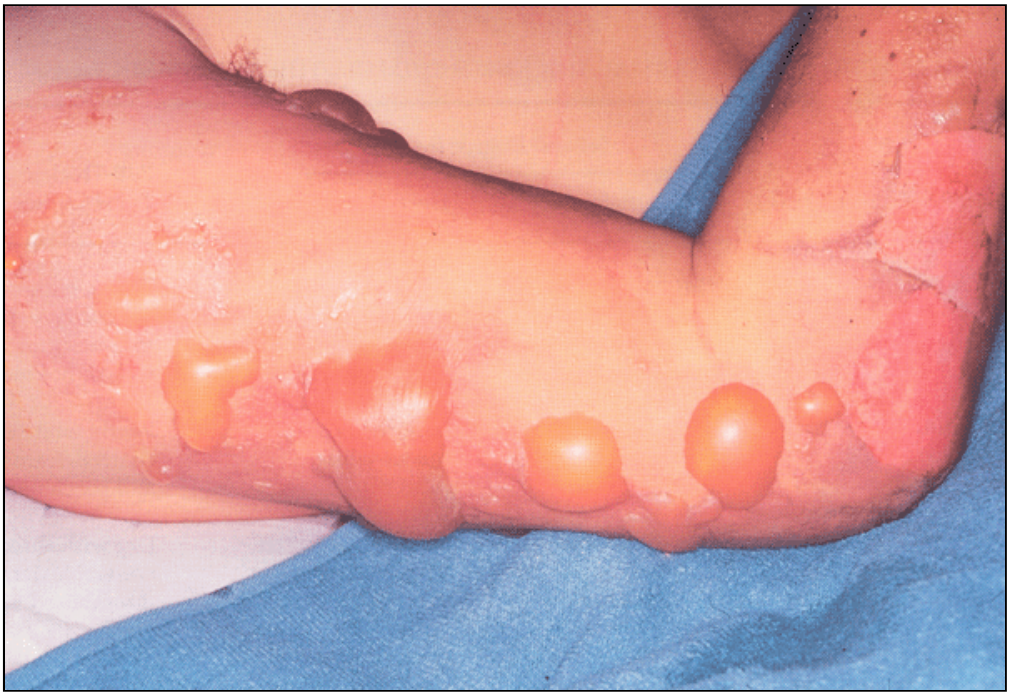

Fig. 10-4. **A** second-degree burn. Intact and debrided bullae characteristic of a superficial partial-thickness burn involving the entire epidermis and the upper layers of the dermis. These injuries should heal spontaneously in less than 21 days.

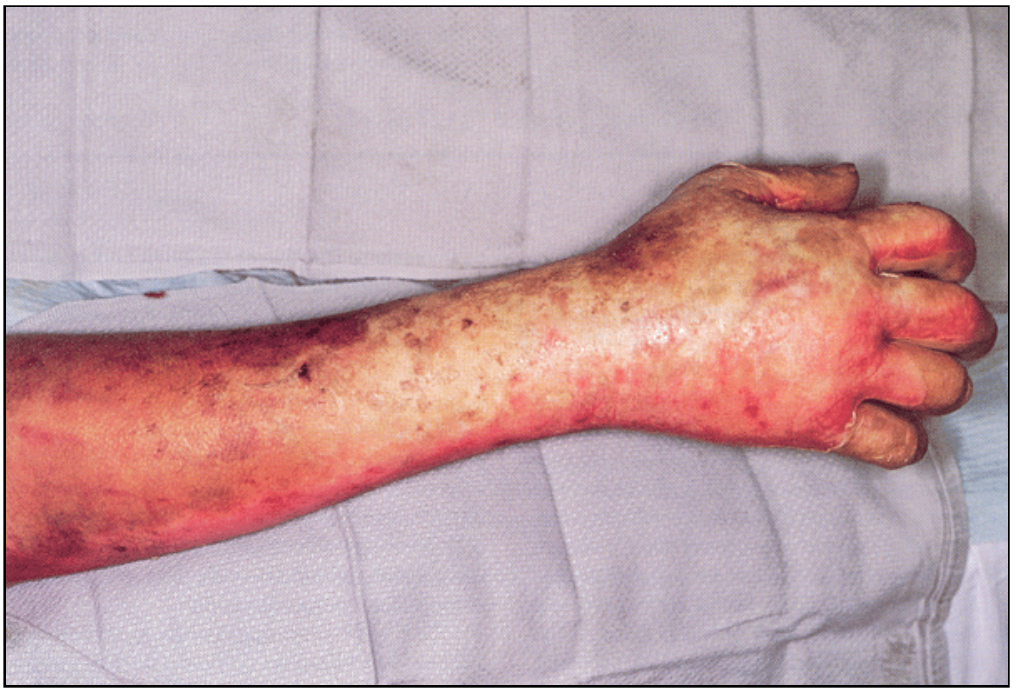

Fig. 10-5. **A** third-degree burn. The mixed deep-partial and full-thickness injury will require excision and grafting **for** optimal cosmetic and functional results.

TABLE 10-1

CLINICAL CHARACTERISTICS OF PARTIAL-THICKNESS AND FULL-THICKNESS BURN WOUNDS

	First-Degree Burns	Second-Degree Burns		Full-Thickness or Third-Degree Burns
		Superficial	Deep Dermal	
Cause	Sun, minor flash	Hot liquids, flashes of flame, brief exposure to dilute chemicals	Hot liquids, brief exposure to flame, longer exposure to dilute chemicals	Flame, high-voltage electricity, exposure to concentrated chemicals or hot metal
Color	Pink or light red	Bright red or mottled red	Dark red or yellow-white	Pearly white or charred, translucent and parchmentlike
Surface	Dry or small blisters	Variably sized bullae, moist and weeping	Large bullae, often ruptured, slightly moist	Dry with shreds of nonviable dermis, thrombosed vessels visible
Texture	Soft with minimal edema and later superficial exfoliation	Thickened by edema, but pliable	Moderate edema with decreased pliability	Inelastic and leathery
Sensation	Hypersensitive	Hypersensitive	Decreased pinprick sensation, intact deep pressure sensation	Skin insensate deep, deep pressure sensation present
Healing	3–6 days	10–21 days	>21 days	Grafting required

Typically, the physiological response to thermal injury is biphasic.[6] The initial period of hypofunction manifests as *(a)* hypotension, *(b)* low cardiac output, *(c)* metabolic acidosis, *(d)* ileus, and *(e)* hypoventilation. Serum glucose increases, oxygen consumption decreases, and the patient is unable to thermoregulate. This ebb phase, which typically extends for a variable portion of the first 24 hours, responds to fluid resuscitation. The flow phase, resuscitation, is characterized by gradual increases in *(a)* cardiac output, *(b)* heart rate, *(c)* oxygen consumption, and *(d)* temperature increase to supranormal levels. This hypermetabolic hyperdynamic response peaks 10–14 days after the injury and then slowly recedes toward normal as the burn heals naturally or is surgically closed by applying a skin graft.

Circulatory System. The alteration in capillary permeability that accompanies thermal injury is proportionate to the size of the burn and is greatest in the burned tissue itself.[7] The coagulation and complement cascades, white-cell products, histamine, serotonins, leukotrienes, and prostaglandins alter transvascular-pressure relationships and vascular integrity, and edema and volume depletion result.

Cardiac output falls as a result of both decreased preload induced by these fluid shifts and the increased systemic vascular resistance caused by both hypovolemia and systemic catecholamine release. Blood flow preferentially shifts from the integument to the viscera, although perfusion may be significantly altered in all organ systems.[6]

The goal of resuscitation is to minimize these effects. With successful resuscitation, cardiac output will return to normal within 12–18 hours, and during the second day after the injury, it may increase up to two- and- one-half times normal and remain elevated

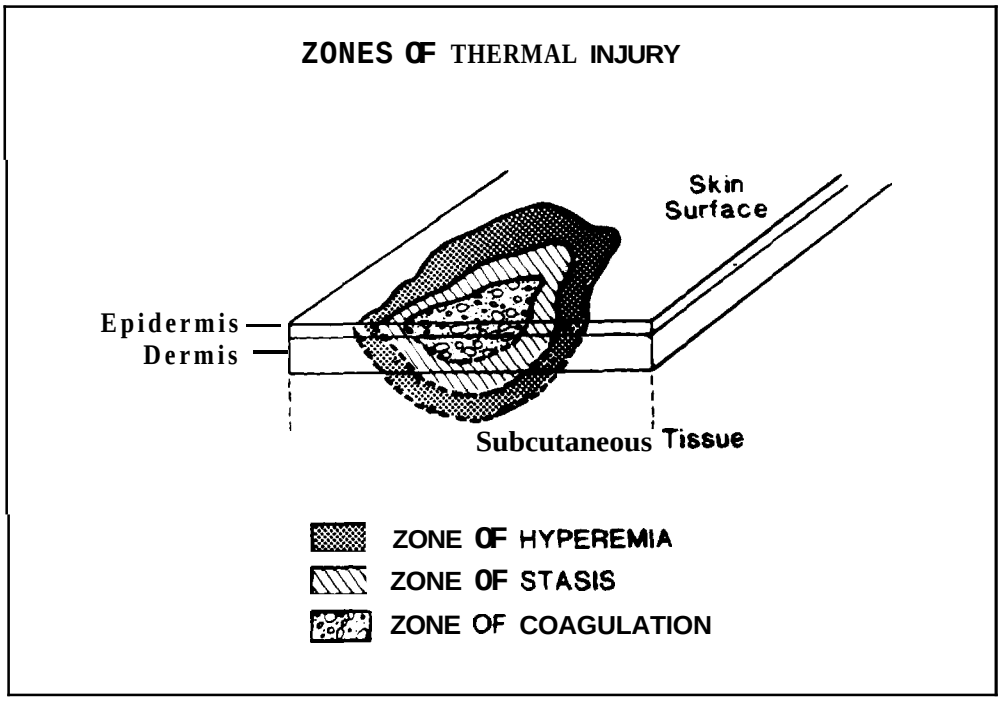

Fig. 10-6. Concentric zones of injury characteristic of the tissue damage caused by thermal energy. In full-thickness burns, the zone of coagulation involves the entire epidermis and dermis; in partial-thickness injury, only part of the dermis is involved.

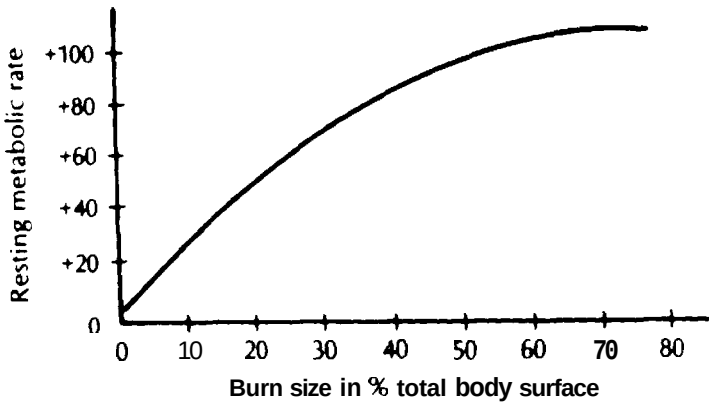

Fig. 10-7. The metabolic effects and all other physiological alterations caused by thermal injury are related to the extent of the burn, but usually plateau when burns cover greater than 60% of the body surface area.

until several months after the burn is closed (Figure 10-8).[5] After the casualty is resuscitated, the burn itself may receive a tenfold elevation in blood flow. This increase is due to the lack of autoregulation in injured vessels, and is also in reaction to both inflammatory mediators and the byproducts of anaerobic metabolism. Because the metabolism of burned tissue is largely anaerobic, local oxygen consumption does not increase similarly.

The red-cell mass decreases after the injury due to direct losses in the burn. Damaged cells are removed from the circulation during the first 4–5 days after the burn, which results in further net loss of red cells. Sampling for laboratory measurements can also cause iatrogenic losses of as much as 1–2 units of packed red cells weekly. Thrombocytosis and elevated fibrinogen, factor V, and factor VIII levels commonly occur, and a "normal" platelet or fibrinogen level may be an early sign of disseminated intravascular coagulation. In this setting, sepsis should be suspected, prompting medical officers to search for the source.

Renal Function. Renal blood flow and glomerular filtration decrease soon after the burn occurs due to *(a)* hypovolemia, *(b)* decreased cardiac output, and *(c)* elevated systemic vascular resistance.[8] Initial oliguria is followed by a usually modest diuresis as the capillary leaks seal, plasma volume normalizes, and cardiac output increases after successful resuscitation. This diuresis becomes most evident as peripheral edema is mobilized. The increased renal blood flow and glo-

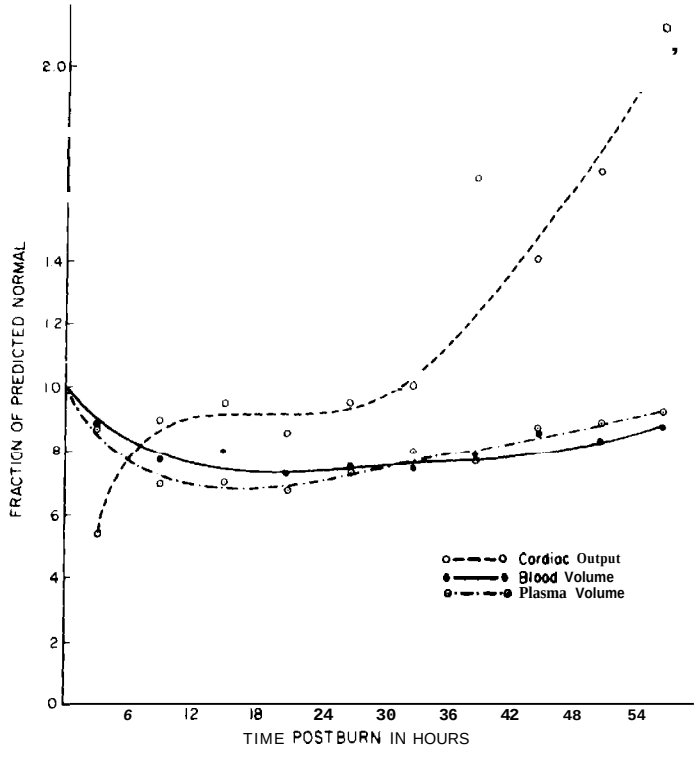

Fig. 10-8. The initially depressed cardiac output returns to normal 12–18 hours after the injury during fluid resuscitation, while blood and plasma volumes are still decreasing. After 24 hours cardiac output rises to supranormal levels, where it remains until wound closure is complete.

merular filtration rate associated with the persistently hyperdynamic circulation increase the rate of renal drug clearance. Consequently, increased dosages of drugs that are principally excreted by the kidneys are required.[8]

Gut Motility. Gut motility decreases in virtually all thermally injured patients whose burns cover more than 20% TBSAB, and manifests as ileus, which typically resolves 3–5 days after the casualty is burned.[9] Focal mucosal erosions may occur in the stomach and proximal small bowel because of splanchnic ischemia and progress to ulceration and perforation if their gastric-acid content is not adequately neutralized. Enteral alimentation, H_2 blockers, and antacids titrated to maintain gastric pH above 5 can virtually eliminate the life-threatening complications (bleeding and perforation) of stress ulcers.[10]

Pulmonary Function. Acute thermal injury alters pulmonary function in many ways. Elevation of pulmonary vascular resistance occurs to a proportionately greater extent than the increase in systemic vascular resistance and may protect against early pulmonary edema during resuscitation.[11] However, this increase in right-ventricular afterload may also alter right-ventricular cardiac performance. The lung parenchyma appears to be spared from diffuse capillary leakage because the ratios of pulmonary lymph-to-plasma protein are unchanged after a thermal injury.[12]

A small increase in minute ventilation occurs in response to the early hypovolemia. As resuscitation proceeds, minute ventilation continues to increase, and it peaks at levels proportionate to the size of the burn. The elevated minute ventilation is sustained by an increase in both tidal volume and respiratory rate. Infection, blood loss, fever, or using carbonicanhydrase inhibitors such as mafenide acetate may cause further increases in minute ventilation.

Endocrine System. Plasma levels of catecholamines, glucagon, and cortisol all increase, reaching maximal levels in patients with a 50–60% TBSAB, while insulin and thyroid hormone levels decrease.[13,14] Mild hyperglycemia and obligatory nitrogen wasting occur. The metabolic rate may increase by as much as 2.5-fold and remain elevated for months after the burn is closed.[15] Catecholamines are partially responsible for this hypermetabolism; their levels are proportionate to the size of the burn, and the hypermetabolic response is partially abrogated by beta adrenergic blockade. As cortisol levels rise, the normal diurnal variation is deranged, and the response to adrenocorticotropic hormone is altered.

Thermoregulation. Alterations in hypothalamic function result in a readjustment of the thermal regulatory set point, with the zone of thermal neutrality elevated to an ambient temperature between 31–33° C. If an extensively burned casualty is inadvertently cooled arid is unable to further increase his or her already maximally stimulated metabolic rate, both hypothermia and cardiovascular collapse may result.

Immune Response. Because the integrity of the skin is lost, this mechanical barrier to infection is impaired. Many immunological defects occur in burned casualties. Immunoglobulin levels are decreased and leukocyte chemotaxis, phagocytosis, and cytotoxic activity are impaired. The reticuloendothelial system's depressed bacterial clearance is thought to be secondary to decreases in opsonic function.[16,17] These changes, together with a nonperfused, bacterially colonized eschar overlying a wound full of proteinaceous fluid, place the patient at significant risk for infection.

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