Handbook of Burns Volume 1
Preface

Severe burn injuries are maybe not the most common injuries occurring on a daily basis; however, it is estimated that within North America approximately 300,000–500,000 patients are hospitalized annually due to a burn-related injury and that worldwide approximately 500,000–1,000,000 people die due to a burn-related injury. Once a burn injury has occurred, it is one of the most severe forms of any injury, inducing a complex cascade of various responses including inflammatory, hypermetabolic, immune, as well as infectious responses. These responses interact with each other and are extremely complex and difficult to treat. Specialized centers, protocolized treatment, multicenter trials, and close collaborations improved morbidity and mortality after severe burn injury over the last two decades. However, a vast morbidity and mortality postburn still occurs and represent one of the major problems in burn treatment.

One of the major characteristics of burn injury that has been evolving over the last decade is that a burn injury is not treated and healed once the wounds are healed. This used to be a landmark that no longer exists. Various studies have indicated that a burn injury and its pathophysiologic sequelae persist for at least 5–10 years, not only in terms of scarring, infection, metabolism, and various other responses. Therefore, this leads to the importance of the current two volumes of these burn books. It has been speculated and hypothesized that early intervention and alleviation of these detrimental responses benefit in terms of clinical outcomes; therefore, the individual book chapters focus on the treatment and complexity of each of these responses to improve outcomes.

We reedited and worked over the two books to include novel aspects of burn. We now focus more on quality of life, on mental health, and on novel technologies. The up-to-date chapters provide evidence-based medicine and current state-of-the-art treatments for any practitioner dealing with acute burn wounds, chronic burn wounds, and all other types of burn wounds. The second volume will then delineate the importance for long-term treatment as it describes the reconstructive and alternative approaches of long-term treatment postburn.

This is unique and therefore will hopefully improve the outcome of burn patients by guiding various kinds of burn practitioners from nursing, physicians, occupational therapy, physical therapy, pharmacy, and so forth. The focus of each chapter is not only to give an overview but also to “summarize” current best treatments and to make it easy for each reader to easily access the treatment options and knowledge.

We hope that these books will raise as much enthusiasm as it has for its contributors.

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Contents

Part I  History, Prevention, Education, Quality, and Team Building

1  A History of Burn Care ................................................................. 3
   Leopoldo C. Cancio and Steven E. Wolf

2  Epidemiology and Prevention of Burns Throughout the World .......... 17
   Michael D. Peck and Jason Thomas Toppi

3  Prevention of Burn Injuries .......................................................... 59
   Joanne Banfield

4  Burns Associated with Wars and Disasters .................................... 71
   Leopoldo C. Cancio and Jonathan B. Lundy

5  Population-Based Research Using Administrative Data
to Evaluate Long-Term Outcomes in Burn Injury .......................... 85
   Stephanie Mason, Rae Spiwak, and Sarvesh Logsetty

6  Education in Burns ................................................................. 93
   Sebastian Q. Vrouwe and Shahriar Shahrokhi

7  Burn Care Teams ................................................................. 99
   Sarah Rehou and Marc G. Jeschke

8  Quality Improvement in Burn Care ............................................. 103
   Alan D. Rogers and Heinz Rode

9  Burn Centers and the Multidisciplinary Team,
   Centralized Burn Care, and Burn Care Quality Control Work ........ 115
   Folke Sjöberg, Ingrid Steinvall, and Moustafa Elmasry

Part II  Pre-hospital and Initial Management of Burns

10  The First Responders’ Role in Managing Burn Care .................... 125
    Ken Webb

11  Prehospital Management of Burn Injuries ................................. 147
    Folke Sjöberg

12  Transfer, Telemedicine and Transportation in Pre-hospital
    Burn Management .................................................................. 159
    Ryan E. Austin

13  Admission of Burn Patients to the Burn Center Including Burn
    Wound Evaluation ................................................................ 171
    Moustafa Elmasry, Ingrid Steinvall, Pia Olofsson, and Folke Sjöberg
14 Burn Size Estimation, Challenges, and Novel Technology ................. 181
   Herbert L. Haller, M. Giretzlehner, and Stefan Thumfart

15 Early Management of Burn Patients and Fluid Resuscitation .......... 199
   David G. Greenhalgh

16 Novel Resuscitation Strategies and Technology ....................... 211
   Chris Meador and George Kramer

Part III Critical Care and Acute Phase After Burn

17 Respiratory Management in Burn Care .................................. 219
   Kevin N. Foster

18 Pathophysiology of Burn Injuries ..................................... 229
   Marc G. Jeschke and Gerd G. Gauglitz

19 Organ Responses and Organ Support ................................... 247
   Craig R. Ainsworth, Julie A. Rizzo, and Kevin K. Chung

20 Critical Care in Burns .................................................. 255
   Luis R. Taveras, Marc G. Jeschke, and Steven E. Wolf

21 Nutrition Support for the Burn Patient ................................ 279
   Audra Clark, Jonathan Imran, Tarik Madni, and Steven E. Wolf

22 Anabolic and Anticatabolic Agents in Burns ............................ 287
   Roohi Vinaik, Eduardo I. Gus, and Marc G. Jeschke

23 Diagnosis and Treatment of Infections in Burns ....................... 299
   Kaitlin A. Pruskowski, Kevin S. Akers, and Kevin K. Chung

24 Perioperative Care of the Burned Patient ................................ 309
   Jamie L. Sparling, J. A. Jeevendra Martyn, and Erik S. Shank

25 Treatment and Prevention of Pain in Children and Adults with Burn Injuries .................................................. 323
   Stefan J. Friedrichsdorf

26 Psychological Factors During Acute Hospitalization:
   Delirium, Anxiety, and Acute Stress Disorder ......................... 339
   Shelley A. Wiechman

27 Nursing Management of the Burn Patient ................................ 347
   Judy Knighton

28 Rehabilitation Management During the Acute Phase ................... 385
   Matthew Godleski and Nisha Chopra Umraw

Part IV Specialized Burn Care

29 Pediatric Burns ......................................................... 395
   Robert L. Sheridan

30 Geriatric Burns .......................................................... 401
   Holly B. Cunningham, Kathleen S. Romanowski, and Herb A. Phelan

31 Burns in Patients with Special Comorbidities .......................... 415
   Kevin N. Foster
32  **Wound Healing** ................................................................. 423
    Eleanor Curtis and Nicole S. Gibran

33  **Outpatient Burn Management** ........................................... 435
    Charles J. Yowler and Tammy L. Coffee

34  **Surgical Management of Burn Patients** ................................. 443
    Jorge Leon-Villapalos

35  **Acute Management of Facial Burns, Acute Versus Long-Term, Surgical Versus Non-surgical Face Transplant** .................. 459
    Juan P. Barret and Julia Barret-Joly

36  **Hand Burns** ................................................................. 465
    Clifford C. Sheckter and Matthew B. Klein

37  **Treatment of Burns: Established and Novel Technologies** ........ 475
    Janos Cambiaso-Daniel, Stefanos Boukovalas, Alexis L. Boson,
    Ludwik K. Branski, and Lars-Peter Kamolz

38  **Scarring and Scar Management** .......................................... 489
    Gerd G. Gauglitz and Julian Poetschke

**Part V  Non-thermal Burns**

39  **Electrical Burn Injuries** .................................................. 505
    Jessica Shih and Marc G. Jeschke

40  **Chemical Burn: Diagnosis and Treatments** ............................ 511
    Ali Izadpanah

41  **Necrotizing Soft Tissue Infections** .................................... 517
    Helene Retrouvey and Shahriar Shahrokhi

42  **Frostbite** ................................................................. 529
    Christopher M. Nguyen, Rowan Chandler, Imran Ratanshi,
    and Sarvesh Logsetty

43  **Epidermal Necrolysis Spectrum from Basic Theory to Practice Essentials** ........ 549
    Neil Shear and Abrar Bukhari

**Part VI  Challenging Burn Cases Examples**

44  **Burn Reconstruction: The Role of Integra in the Dorsum Hand and Wrist Reconstruction** ............................... 561
    Anthony Papp

45  **Innovative Autologous Coverage for a 90% TBSA Full-Thickness Burns** ........ 565
    Isabelle Perreault and Patricia Bortoluzzi

46  **Delayed Management of Acute Burn Wounds in Rural Areas of Low-Income Countries: Global Burn Surgery** .................... 571
    Claudia C. Malic

47  **Levamisole: Adulterated Cocaine-Induced Soft Tissue Necrosis** ....... 575
    Sarvesh Logsetty and Shahriar Shahrokhi

48  **Outcome of an Extensive Cold Injury with a Burn Injury Component** ........ 577
    Claudia C. Malic, Marc G. Jeschke, and Shahriar Shahrokhi

**Index** ................................................................................. 579
A History of Burn Care

Leopoldo C. Cancio and Steven E. Wolf

1.1 “Black Sheep in the Surgical Wards”

If one uses the incontrovertible index of postburn mortality, it is evident that our ability to care for burn patients has improved markedly since World War II. This can be quantified by the lethal area 50% (that burn size which is lethal for 50% of a population), which in the immediate postwar era was approximately 40% of the total body surface area (TBSA) for young adults, whereas it increased to approximately 80% TBSA by the 1990s in the USA [1]. Furthermore, the mortality rate at the Galveston Shrine for children with 80% TBSA burns or greater (mean 70% full-thickness burn size) during 1982–1996 was only 33% [2]. What has been responsible for these improved outcomes in burn care? What practices were essential to this growth, and what are the major problems that remain unsolved? In this chapter, we will take as our focal point the fire disaster at the Cocoanut Grove Night Club which took place in Boston in 1942, less than a year after Pearl Harbor. The response to that disaster, and the monograph written in its aftermath, serves as a useful benchmark for the burn care advances which followed. To fully appreciate those advances, however, we must go back in time to an earlier era.

A wide variety of therapies for burns have been described since ancient times [3], but the idea of collecting burn patients in a special place is relatively new and emerged in Scotland during the nineteenth century. James Syme established the first burn unit in Edinburgh in 1843. 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 [4].

Another Scottish hospital, the Glasgow Royal Infirmary, had by 1933 accumulated 100 years of experience with over 10,000 burn patients, having established a separate burn ward midway through that period in 1883. In Dunbar’s report on these patients, he commented:

Burn cases have until recently been looked upon as black sheep in surgical wards, and have been almost entirely treated by junior members of the staff, who have not had any great clinical experience from which to judge their results (…) In the pre-antiseptic era only the worst burns would come to the hospital. The state of the hospitals was well known to the public, who also knew that a burn of slight or moderate severity had a better chance of recovery at home.

He documented the steady rise in the number of admissions to this hospital, a biphasic mortality pattern (with the highest number of deaths between postburn hours 12 and 24), the 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 [5].

1.2 Toxemia, Plasmarrhea, or Infection?

Against this background, the founders of modern burn care must be credited with considerable clinical courage and intellectual foresight. Although the era of growth which they introduced is often dated to World War II, its roots were in earlier fire disasters and in World War I. This period featured a debate about the cause of postburn death and accordingly the appropriate treatment. A prevailing theory attributed...
death to the release of toxic substances from the burn wound: “The reaction of the body to a burn strongly resembles the clinical state described by the term ‘toxaemia,’ which implies the presence in the circulation of some toxic agent. The more serious cases usually present early in the course a clinical picture commonly described by such terms as shock or exhaustion” [6]. Treatments were widely employed to prevent this from happening. The most important such treatment was tannic acid, popularized by Davidson in 1925 [6]. Tanning of eschar, or of animal leather, involves collagen cross-linking and the formation of lipid-protein complexes in the remaining dermis. This generates a brown, supple, leather-like eschar [7]. Davidson asserted ambitiously that tannic acid not only lessens toxemia but also provides analgesia, prevents loss of body fluid, limits infection, decreases scar formation, and generates a scaffold for healing [6].

By contrast, in 1930 Frank Underhill published seminal observations on the pathophysiology of burn shock based on experience gained following the Rialto Theater fire of 1921 in New Haven, CN. These included the concepts of “anhydremia” and “hemoconcentration.” Here is his description:

> When loss of water from the blood becomes great, the circulatory deficiency becomes magnified. The thick, sticky blood...finds great difficulty in passing through the capillaries...the blood is quickly robbed of its oxygen by the tissues...the tissues in general suffer from inadequate oxygenation...the heart pumps only a portion of its normal volume at each stroke [8].

Underhill then points out that his thinking on this process began during World War I, when he noted that inhalation of chemical warfare agents (chlorine, phosgene, and chloropicrin) produced both massive pulmonary edema and hemoconcentration. Applying this concept to thermally injured skin led to our basic understanding of burn shock: “fluid rushes to the burned skin with great rapidity and is lost to the body...or the part affected becomes edematous with great celerity.” The fluid lost is similar to plasma—implying increased capillary permeability—whereas in cholera it is a dilute salt solution. Measurement of the blood hemoglobin percentage is proposed as an index of resuscitation, and resuscitation aimed at preventing hemoconcentration is required for 24–36 h postburn. Intravenous sodium chloride solutions should be used, supplemented by oral, rectal, and subdermal solutions [8].

In 1931, Alfred Blalock reported laboratory confirmation of Underhill’s theory. Dogs underwent burns to one third of the body surface area, limited to one side of the body. After death, animals were sagittally bisected, and the difference in weight between the halves was estimated to be the amount of fluid lost into the tissues as a consequence of injury. This weight difference was on average 3.34% of the initial total body weight, indicating a loss of approximately one half of the circulating plasma volume. He also noted that the fluids collected in the subcutaneous tissues had a protein concentration similar to that of plasma and that the blood hemoglobin content increased markedly [9].

But plasma loss was an incomplete description of the biphasic death pattern documented for burn patients. Shortly thereafter, Aldrich introduced the treatment of burn wounds with gentian violet, a coal-tar derivative which kills Gram-positive organisms. He argued against the toxemia theory and attributed postburn toxic symptoms not to the eschar but to streptococcal wound infection. Early use of gentian violet would prevent this, whereas tannic acid did not. He distinguished this delayed infectious process from “primary shock,” downplaying the latter’s importance: “it is sufficient to say that if it is combated early and adequately, with heat, rest, fluids, and stimulants, it can be overcome in the majority” [10].

### 1.3 The Guinea Pig Club

The transformation of burn care required not only the above observations but also an institutional commitment. In 1916, Sir Harold Gillies returned from service in France to lead the first plastic and oral-maxillofacial surgery service in the UK at Cambridge Military Hospital, Aldershot, later moving to Queen’s Hospital in Sidcup, Kent. Gillies and team treated over 11,000 casualties with facial injuries by the end of the war, including burns [11, 12]. The Spanish Civil War (1936–1939) convinced the British leadership that the next war would involve air combat and asked Gillies to establish plastic surgery units around London [12]. At that time, there were only four plastic surgeons in the country, including Gillies’ cousin, Sir Archibald McIndoe, who had joined Gillies in 1930 after training at the Mayo Clinic [13]. During 1939, McIndoe established the burn unit for the Royal Air Force at East Grinstead, UK, which persists to this day. Beginning in summer 1940, approximately 400 RAF personnel (mainly fighter pilots) were seriously burned during the Battle of Britain, revealing both aircraft design limitations and the intensity of aerial combat. The focus of the new unit was on the reconstruction and rehabilitation of these patients. McIndoe assembled a team of nurses, anesthetists, microbiologists, orderlies, and others to undertake this journey into the unknown:

Historically there was little to guide one in this field apart from the general principles of repair perfected by British, Continental and American surgeons. There had until then been no substantial series of cases published and none in which a rational plan of repair had been proposed. At most, individual cases appeared...in which only too often the end result seemed to convert the pathetic into the ridiculous [13].

Soon, four more units were established in the UK, which together with East Grinstead served the hundreds of
casualties who followed from operations such as Royal Air Force’s strategic bombing campaign.

McIndoe’s work underscores several important points about burn care. First, the impetus for a breakthrough in the organization and delivery of burn care was the catastrophic nature of modern warfare, the large number of casualties therefrom, and both a national and an individual commitment to care for these casualties. Second, the experimental nature of burn care was recognized, and a scientific approach based on clinical evidence was espoused. Among the East Grinstead unit’s contributions were the condemnation of tannic acid as coagulation therapy for acute burn wounds; perfection and description of a methodology for burn wound reconstruction; and, in collaboration with Leonard Colebrook (see below), early experience with penicillin therapy for Gram-positive infections. Third, the East Grinstead unit became a hub for new UK burn units, as well as a training center for scores of surgeons and nurses in the principles of the emerging specialty. Fourth, the psychological and social needs of the patients were highlighted. At East Grinstead, this was embodied in the “Guinea Pig Club,” a social network for burn survivors whose membership totaled 649 people (Fig. 1.1). The longevity of both the needs of burn survivors, and the strength of this network, is exemplified that the last issue of The Guinea Pig magazine was published in 2003 [13]. Clearly, none of these steps—the scientific approach to improving burn care, the emphasis on clinical expertise on the part of all members of the multidisciplinary team, and the creation of a mechanism for effective psychosocial support—would have been possible without the concentration of patients at a center dedicated to overcoming a seemingly insurmountable problem.

The origin of infection control in burn patients, however, belongs not to McIndoe but to Leonard Colebrook, a physician, bacteriologist, and colleague of Alexander Fleming [14]. In an era dominated by multidrug-resistant Gram-negative and methicillin-resistant Staphylococcus aureus infections, it is important to recall the major role played by Streptococcus pyogenes infections before the introduction of antibiotics. Colebrook confirmed Domagk’s 1935 “startling

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**Fig. 1.1** Members of the “Guinea Pig Club” of war-injured burn survivors, nurses from the burn unit at East Grinstead, UK, and Sir Archibald McIndoe celebrating around the piano. This photograph graphically depicts the value of peer support in the recovery of the whole patient. 
Source: East Grinstead Museum, East Grinstead, UK
success” on the efficacy of the sulfanilamide parent drug, Prontosil, using a murine model of streptococcal peritonitis, and reported lifesaving treatment of 38 patients with puerperal fever [15, 16]. Turning his attention to burns at the Glasgow Royal Infirmary, he studied dressings impregnated with sulfanilamide and penicillin creams [14, 17] and the use of serum and plasma for burn shock resuscitation [18]. (The problem of Gram-negative burn wound infection remained to be recognized and solved at another time, since “coliform bacilli, B. proteus and Ps. pyocyanea, when present in the wounds, were apparently not affected” by these drugs.) [17]. He then established a new burn unit at the Birmingham Accident Hospital [19]. In contrast to the toxemia theory, Colebrook and others proposed that burn wounds became infected with bacteria and that strict infection control practices could prevent infection by reducing transfer of these organisms; these concepts were incorporated into both the design and practices of the new burn unit [20, 21].

1.4 Burns and Sulfa Drugs at Pearl Harbor

In the USA, the attack on Pearl Harbor on December 7, 1941 served a function similar to that of the Battle of Britain by energizing burn care research. Fortuitously, the USA, anticipating the likelihood of war, had already made two major national commitments to supporting medical research of military relevance. The first such effort was the creation, by the National Research Council’s (NRC) Division of Medical Sciences, of Advisory Committees to the Surgeons General in April 1940 [22]. Critical among these for the burn care in the USA were the Committee on Chemotherapeutic and Other Agents and the Committee on Surgery (which included, among others, Subcommittees on Surgical Infections and on Burns).

The second such effort was the creation by the federal government of the Committee on Medical Research (CMR) of the Office of Scientific Research and Development (OSRD) in June 1941 [23, 24]. The purpose of the CMR was to identify problems of military medical importance and to fund university research to solve these problems. These two activities (the NRC Advisory Committees and the CMR) were collocated at NRC headquarters, and the NRC advised the CMR on how best to expend federal funds [22]. In brief, by the time of Pearl Harbor, the USA had the framework in place for academic, military, and federal collaboration in pursuit of solutions for combat casualty care.

For the NRC and the CMR, Pearl Harbor highlighted the importance of burns in modern warfare. About 60% of over 500 casualties admitted to the Pearl Harbor Naval Hospital were thermally injured (Fig. 1.2). Many of these wounds were contaminated by fuel oil or complicated by fragment injuries. Care was variable and included some sort of topical tanning agent, delayed debridement, infusion of available intravenous fluids, and treatment of fractures [25]. “At the Naval Hospital, ordinary flit guns were used to spray tannic acid solution upon the burned surfaces,” indicating the persistence of the toxemia theory in clinical care. On the other hand, both plasma and saline solution were used for fluid resuscitation, and sulfa drugs were given to patients with infected wounds—indicating a conglomeration of the competing theories of burn pathophysiology. In response to Pearl Harbor, the NRC rapidly dispatched Perrin Long, the chairman of the Committee on Chemotherapeutic and Other Agents, and surgeon I.S. Ravdin to Hawai‘i, in order to evaluate the use of sulfa drugs and other aspects of care.

Fig. 1.2 Aboard the USS Solace hospital ship, caring for wounded from the attack on Pearl Harbor, December 7, 1941. The Solace dispatched small boat crews to rescue casualties: “they boarded the burning Arizona, while its crew was abandoning ship, and they rescued the burned and injured casualties found on its deck, some very close to the flames” [131]. Pearl Harbor alerted the US Government of the urgent need for burn research. Source: US Navy [132]
They submitted their report to the War Department on January 18, 1942, emphasizing the lifesaving characteristics of sulfa drug use and the value of plasma for resuscitation:

> We have been impressed again and again with the incalculable value of sulfonamide therapy in the care of many of the casualties... We believe that it is highly important that physicians—both civilian and military—become familiar with the general and specific considerations which govern the oral and local use of the sulfonamides in the treatment of wounds and burns.... [25, 26]

Despite this impression and the fact that the sulfa drugs were the only antibiotics available in significant quantities in 1941, their indications and limitations were unknown. Accordingly, the Subcommittee on Surgical Infections, chaired by Frank Meleney, defined this question as a major objective at its initial meeting in June 1940 [22]. Wound study units were set up at eight US hospitals, and a multi-center trial was conducted of both local and systemic sulfa use. Meleney, in his report on this study, lamented that

> The original plan was altered to a considerable extent by the reports which came back from Pearl Harbor. Observers who saw the casualties there were profoundly impressed by the low incidence of wound infection, which they believed to be due to the copious application of sulfanilamide to the wounds. Our original plan called for observation on control cases without drugs and other controls receiving treatment with local bacteriostatic agents other than the sulfonamides. But, said the Pearl Harbor observers: “You cannot withhold from these patients the benefit of the sulfonamide drugs.” [27]

By the end of 1942, 1500 patients (with soft tissue injuries, fractures, and burns) had been enrolled. In his report on this study, Meleney concluded that neither local nor systemic sulfonamides were effective at controlling local wound infection and that inadequate surgical treatment predisposed to infection. The antibiotics were effective at preventing systemic sepsis, but were not a panacea [27]. An awareness of these limitations and emerging experience with *Staphylococcus* and *Clostridium* resistance to sulfa drugs [22] set the stage for research on penicillin.

### 1.5 Penicillin and the Burn Projects

Although Alexander Fleming discovered penicillin in 1929, its clinical utility was not appreciated until 10 years later, when Howard W. Florey, Ernest Chain, and others (the “Oxford Team”) performed murine and human experiments demonstrating the new drug’s lifesaving potential against *Streptococcus*, *Staphylococcus*, and *Clostridium* infections [28, 29]. Since British pharmaceutical firms were overwhelmed with wartime production of other drugs, Florey went to the USA in summer 1941 to obtain support for large-scale manufacturing, ultimately meeting with and convincing the chairman of the CMR, Alfred N. Richards [24]. Once a method of mass-production of the drug had been developed, the CMR turned in January 1942 to the Committee on Chemotherapeutic and Other Agents, headed by Perrin Long, for help in organizing clinical trials [30]. Long appointed Champ Lyons at the Massachusetts General Hospital (MGH), Chester Keefer, and colleagues to accomplish this [30]. This was the origin of one of the two burn-related research programs in place at the MGH at the time of the Cocoanut Grove fire in 1942 [31].

The second MGH research program dealt specifically with thermal injury [31]. On January 7, 1942, the NRC sponsored a pivotal conference on burns, chaired by I.S. Ravdin [32, 33]. The conference proceedings recommended plasma, topical tannic acid, and oral sulfadiazine. Henry Harkins presented the available formulas for resuscitation of burn patients. His own method (the “Method of Harkins”) was based on hemoconcentration: give 100 cc of plasma for each point that the hematocrit exceeds 45. For wartime, when lab facilities are unavailable, he recommended the “First Aid Method”: slowly give 500 cc of plasma for each 10% of the total body surface area (TBSA) burned [34, 35]. The latter is the first formula based on TBSA. The NRC report from the conference advocated 1000 cc of plasma for each 10% TBSA over the first 24 h, in divided doses [32].

The Subcommittee on Burns was organized under Allen Whipple in July 1942 [22] and was charged with determining the best therapies for acute burns and whether tanning was appropriate. The wound study units of the Subcommittee on Surgical Infections found that tanned burns had a high wound infection rate, and the Subcommittee on Burns soon recommended against use or further procurement of tannic acid in October 1942—less than 1 year after it was liberally used at Pearl Harbor. Nevertheless, “it cannot be said that unanimous agreement was ever attained on the choice of the best local agent” [22]. Another early contribution by Whipple was stating for the first time the importance of well-organized “burn teams”:

> By burn team we mean a group made up of a general surgeon, interested in problems of infection and wound healing, a physician or technician, thoroughly trained in problems of fluid, protein and electrolyte imbalance, a general plastic surgeon...with experience in skin grafting large granulating areas, a group of trained nurses and orderlies, able and willing to stand the stress and strain of caring for severely burned patients. [36]

### 1.6 The Cocoanut Grove Fire of 1942 and Beyond

The fire at the Cocoanut Grove (CG) nightclub in Boston, MA on November 28, 1942 was one of the worst civilian fire disasters in the US history, killing 492 of the estimated 1000
occupants [37]. Oliver Cope, editing the monograph published on the MGH’s response, felt that they were well prepared in large part because of the war:

Had such a catastrophe taken place before Pearl Harbor, the hospital would have been swamped. As it was, the injured found the staff prepared, for the war had made us catastrophe minded. (...) A plan of therapy for burns, suited to use in a catastrophe, was developed and decided upon. When the victims of the Cocoanut Grove fire arrived, the treatment was ready and it was applied to all. [31]

Specific preparations for war that were already in place at the time of this fire included organization of personnel, publication of a disaster manual, preparation of sterile supplies for 200 operations, acquisition of wooden i.v. poles and of sawhorses to support stretchers, establishment of a blood bank, and training of Red Cross volunteers and of Harvard students as orderlies [38].

The CG monograph contains the first detailed description of a scientific approach to multidisciplinary burn care [37]. As such, it serves as an invaluable point of departure for understanding subsequent changes and current practice.

1.6.1 Burn Center Concept

Although the MGH did not have a dedicated burn unit in 1942, the 39 CG patients were all hospitalized on a single ward. “In a disaster of this type, where the injuries were all of the same kind, the importance of concentration of casualties in one group in one ward or floor where they can be under concentrated medical treatment and where isolation procedures can be set up if needed, was clearly demonstrated” [39]. The first permanent unit in the USA was established in Richmond, VA by Everett Evans, who had become chairman of the NRC Committee on Burns [40]. In 1947, the Army Wound Study Unit was moved from Halloran General Hospital to Fort Sam Houston, Texas by Edwin Pulaski—an Army surgeon who had trained under Meleney—and was renamed the Surgical Research Unit (SRU) [41]. At that unit, patients with infected burns and other wounds were treated on a special ward at the US Army’s Brooke General Hospital. Two years later, growing concerns about the possibility of nuclear war with the Soviet Union, and recognition that such a war would generate thousands of burn survivors, refocused the SRU on the treatment of burns, and the second US burn unit was formally established [40].

The US Army Burn Center at the SRU (later renamed as the US Army Institute of Surgical Research, USAISR) was at the forefront of many of the advances in burn care described below. Also critical for improving care in the US was the unit’s commitment to training surgeons, many of whom became directors of civilian burn centers [42–44]. Designation of the unit as the single destination for all the US military burn casualties, as well as for civilians in the region, provided the number of patients needed both to maintain clinical competence and to support the research mission during war and peace. Another major factor in the development of burn care in the USA was the decision in 1962 by the Shriners Hospitals for Crippled Children privately to fund the construction and operation of three pediatric burn units—in Cincinnati, Ohio; Boston, Massachusetts; and Galveston, Texas. These units opened during 1966–1968, and like the US Army Burn Center, became centers of excellence in care, teaching, and research [45, 46].

1.6.2 Shock and Resuscitation

The MGH used a version of the NRC First Aid Formula for resuscitation of the CG casualties. All but 10 patients were given plasma intravenously (Fig. 1.3):

The initial dosage of plasma was determined on the basis of the surface area of the burns. For each 10 percent of the body surface involved, it was planned to give 500 cc in the first 24 hours. Because the plasma delivered by the Blood Bank during the first 36 hours was diluted with an equal volume of physiologic saline solution, the patient was to receive 1000 cc of fluid for each 10 percent burned. The plasma dosage was modified subsequently on the basis of repeated hematocrit and serum protein determinations. [47]

Cope and Moore, in a follow-on paper in 1947, described a refinement of the NRC formula called the Surface Area Formula: 75 mL of plasma and 75 mL of isotonic crystalloid solution per TBSA, with one-half given over the first 8 h and one-half over the second 16 h. The urine output was to be used as the primary index of resuscitation [33].

Fig. 1.3 A survivor of the Cocoanut Grove nightclub fire in November 1942 receives an infusion of plasma. Ongoing preparations for war enabled Boston hospitals to respond more effectively to this civilian disaster. Source: Boston Public Library, Leslie Jones Collection [133]
Subsequent revisions of this basic concept included the following formulas:

- **Evans Formula**: incorporation of body weight; colloid 1 mL/kg/TBSA and crystalloid 1 mL/kg/TBSA [48]
- **Brooke Formula**: decrease in colloid content to 0.5 mL/kg/TBSA, with crystalloid 1.5 mL/kg/TBSA; replacement of plasma with 5% albumin because of hepatitis risk [49]
- **Parkland Formula**: elimination of colloid during the first 24 h; increase in crystalloid to 4 mL/kg/TBSA [50]
- **Modified Brooke Formula**: elimination of colloid during first 24 h; crystalloid 2 mL/kg/TBSA [51]

Despite their differences, employment of these formulas reduced early deaths due to burn shock to about 13% of post-burn deaths, and made acute renal failure due to burn shock distinctly unusual. Today, the hazards of “fluid creep” mandate a continued search for an approach to resuscitation that decreases the rate of edema formation [52, 53].

### 1.6.3 Wound Care and Infection

By the time of the CG fire, tannic acid had fallen into disfavor: “A bland, protective ointment dressing is indicated in the treatment of skin burns since the chemical agents currently recommended are believed to be injurious to otherwise viable epithelium and delay wound healing” [54]. Attention turned to use of i.v. antibiotics for the prevention of infection. Hemolytic streptococcal infection responded to sulfa drugs: “an effective blood level of sulfonamide offers the most certain control of systemic infection due to the hemolytic streptococcus” [55]. Meanwhile, Champ Lyons, the surgeon in charge of penicillin research at MGH, received enough of the experimental drug from Chester Keefer to treat 13 CG patients. The doses given were too low and the experience was inconclusive, although he did not observe toxic side effects [55]. From there, Lyons undertook larger studies of penicillin at Bushnell General Hospital, Brigham City, Utah (April 1943) and at the new Wound Study Unit at Halloran General Hospital, Staten Island, New York (June 1943) [56, 57]; the latter was the forerunner of the US Army SRU. These studies constituted the first large-scale studies of penicillin, documented efficacy against staphylococcal and streptococcal combat wound infections [57], and convinced the Army of the need for large-scale production of the drug [24]. Lyons next obtained a commission as an Army Major in August 1943, deploying to the North African theater to facilitate the introduction of penicillin into battlefield care under Edward Churchill [56].

Penicillin, however, was only a partial answer to the problem of late postburn death. In 1954, the SRU noted that effective fluid resuscitation now kept many patients with greater than 50% TBSA burns alive past the 2-day mark, only to succumb at a later date [58]. The conquest of hemolytic *Streptococcus* now revealed the role of Gram-negative organisms, and the presence of positive blood cultures, particularly in patients with large full-thickness burns, pointed at bacteremia of burn wound origin [58]. The natural history of this “burn wound sepsis” was not clear, however, until a model of invasive *Pseudomonas* burn wound infection in rats was conceived and characterized by Walker and Mason at the SRU [59–61]. At that time, however, no effective topic or intravenous therapy had been identified.

Pruitt and colleagues at the SRU achieved a dramatic improvement in postburn mortality in 1964, with the introduction into clinical care of a topical antimicrobial effective against Gram-negative burn wound infection, mafenide acetate (Sulfamylon) cream (Fig. 1.4) [62]. This drug had been first synthesized in the 1930s and evaluated by Domagk, but abandoned, interestingly, because of lack of efficacy against *Streptococcus* [63]. It was rediscovered by US Army researchers at Edgewood Arsenal, who demonstrated efficacy in an otherwise lethal caprine model of *Clostridium perfringens* infection following extremity blast injury [64]. Because it penetrates deeply, it appeared particularly effective in wounds with devitalized tissue, a feature which also made it attractive for the treatment of full-thickness burns. Lindberg and colleagues at the SRU had similar success in the Walker–Mason *Pseudomonas* model [65]. In thermally injured patients, death from invasive burn wound infection declined from 59% (pre-mafenide) to 10% (post-mafenide) [62]. Meanwhile, Moyer and Monofo confirmed the effectiveness of 0.5% silver nitrate soaks in preventing burn wound infection [66]. Charles Fox subsequently developed silver sulfadiazine to combine the advantages of a sulfonamide with the silver ion [67]. Silver sulfadiazine, the recently developed silver-impregnated fabrics [68], and mafenide acetate are the commonly employed antimicrobials used in burn care today.

### 1.6.4 Burn Surgery

Surgeons accustomed to early excision of the burn wound should bear in mind that at the time of the CG fire, burn surgery was performed after the separation of eschar: “The first graft was applied on the twenty-third day...and the last at four months to several small areas” [69]. Originally, the surgical treatment of burn wounds, if performed, was limited to contracture release and reconstruction after the wound had healed by scar formation. In patients with larger wounds or burns of functional areas, this was wholly unsatisfactory. The creation of burn units committed to care for these patients led to the development of more effective techniques for wound closure. Artz noted that one should “wait until natural
sequestration has occurred and a good granulating barrier has formed beneath the eschar...After removing the eschar... skin grafting should be performed as soon as the granulating surface is properly prepared” [70]. Debridement to the point of bleeding or pain during daily immersion hydrotherapy (Hubbard tanks) was used to facilitate separation of the eschar [71]. Then, cadaver cutaneous allografts (homografts) were often used to prepare the granulating wound bed for autografting [72].

In patients with larger (>50% TBSA) burns and in the absence of topical antimicrobials, this cautious approach did not prevent death from invasive burn wound infection, leading some to propose a more radical solution: that of primary excision of the burn wound. Surgeons at the SRU suggested that a “heroic” practice of early excision, starting postburn day 4, should be considered for patients with large burns. This would reduce the “large pabulum” of dead tissue available for microbial proliferation; immediate coverage with a combination of autograft and cadaver allograft would further protect the wound [61]. Several authors during the 1950s and 1960s demonstrated the feasibility of this approach, but not an improvement in mortality [73].

In 1968, Janzekovic described the technique of tangential primary excision of the burn wound with immediate grafting; operating in postwar Yugoslavia, she recalled that “a barber’s razor sharpened on a strap was the pearl among our instruments” [74, 75]. In a retrospective study, Tompkins et al. reported an improvement in mortality over the course of 1974–1984 which they attributed to excision [76]. William F. McManus and colleagues at the Army Burn Center compared patients who underwent excision with those who did not during 1983–1985, noting that an improvement in mortality could not be attributed to excision because preexisting organ failure precluded surgery in many unexcised patients. However, only six of the 93 patients (6.5%) who died in this study had invasive bacterial burn wound infection, whereas 54 of the 93 (58%) had pneumonia—indicating a shift from wound to non-wound infections [77].

In McManus’ study, excision was performed in a mean of 13 days postburn. By contrast, David Herndon et al. at Galveston implemented a method of excision within 48–72 h of admission, which relied on widely meshed (4:1) autograft covered by allograft. In a small study of children during 1977–1981, these authors noted a decrease in length of stay but not in mortality with this technique [78]. During 1982–1985, adults were randomized to undergo early excision vs. excision after eschar separation 3 weeks later. Young adults without inhalation injury and with burns >30% TBSA showed an improvement in mortality [79]. A recent meta-analysis found a decrease in mortality but an increase in blood use in early excision patients without inhalation injury [80].

Despite the limitations of the early studies, early excision is today performed in most of the US burn centers—controversy remains about the definition of “early” and the feasibility of performing radical, total excision at one operation, especially in adults. We now understand excision and

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**Fig. 1.4** Application of mafenide acetate cream (Sulfamylon) to a thermally injured patient at the US Army Surgical Research Unit in 1964. At the left, Dr. John L. Hunt; at the right, Dr. Basil A. Pruitt, Jr. The dramatic decrease in invasive Gram-negative burn wound infection, that followed the introduction of Sulfamylon, was the epitome of integrated laboratory and clinical research championed by Dr. Pruitt. Source: Collection of the US Army Institute of Surgical Research. Courtesy Mr. Glen Gueller
definitive closure of the burn wound as fundamental for patients with massive injuries; the “race” to achieve this before sepsis and other causes of organ failure supervene is the main effort; patients whose grafts fail repeatedly (“wound failure”) will not, in the authors’ experience, survive. To facilitate massive excision for patients with the largest wounds and limited donor sites, new methods of temporary and permanent closure have been sought. Burke and Yannas developed the first successful dermal regeneration template (Integra®), composed of a dermal analog (collagen and chondroitin-6-sulfate) and a temporary epidermal analog (Silastic) [81]. Cultured epidermal autografts provide material for wound closure for patients with the most extensive burns, although the cost is high and final take rates are variable [82–84]. The ultimate goal of an off-the-shelf bilaminar product for permanent wound closure, with a take rate similar to that of cutaneous autografts, has not yet been achieved.

1.6.5 Inhalation Injury and Pulmonary Care

Pulmonary problems were a significant cause of mortality after the CG fire, and options for diagnosis and treatment were limited. About 114 patients were brought to the MGH, some alive, some dead; it is clear that many of these fatalities died of carbon monoxide poisoning or early airway obstruction. Of the 39 patients who survived long enough to be admitted, seven died, all of whom had evidence of inhalation injury. The authors noted: “Although intubation and tracheotomy were not highly successful in our cases, we believe that they fulfill a definite function in relieving labored breathing and in facilitating the delivery of oxygen, and should be resorted to in patients with acute cyanosis and in those with severe upper respiratory lesions.” On the other hand, “the resuscitation of patients in acute attacks of edema was difficult and unsatisfactory” and “the pulmonary complications were bizarre and characterized by extreme variability, with areas of lung collapse and emphysema…” [85].

Subsequent improvements in inhalation injury care required the development of positive-pressure mechanical ventilators. Forrest Bird, V.R. Bennett, and J. Emerson built mechanical positive-pressure ventilators toward the end of WWII, all inspired by technology developed during the war to deliver oxygen to pilots flying at high altitudes [86]. The availability of these and similar machines, and the Scandinavian polio epidemic of 1952, spurred the creation of separate intensive care units (ICUs) within hospitals [87]. Today, in one model of burn care, burn units are separate from ICUs, and the two types of units are run by different personnel. At the US Army Burn Center and several other centers, by contrast, ICU beds have been located within the burn unit and have been directed by surgeon-intensivists—ensuring continuity of multidisciplinary care and clinical research.

Once accurate diagnosis of inhalation injury by bronchoscopy and xenon-133 lung scanning became available, it was apparent that smoke-injured patients had greatly increased risk of pneumonia and death [88]. Large animal models were developed, and the pathophysiology of the injury was defined [89, 90]. Unlike ARDS due to mechanical trauma or alveolar injury due to inhalation of chemical warfare agents, smoke inhalation injury was found to damage the small airways, with resultant ventilation–perfusion mismatch, bronchiolar obstruction, and pneumonia [91]. This process featured activation of the inflammatory cascade, which in animal models was amenable to modulation by various anti-inflammatory agents [92]. Practically, however, the most effective interventions to date have been those directly aimed at maintaining small airway patency and at avoiding injurious forms of mechanical ventilation. These include high-frequency percussive ventilation with the Volumetric Diffusive Respiration ventilator developed by Bird [93] and delivery of heparin by nebulization [94].

1.6.6 Nutrition and the “Universal Trauma Model”

Bradford Cannon described the nutritional management of the survivors of the Cocoanut Grove fire: “All patients were given a high protein and high vitamin diet…it was necessary to feed [one patient] by stomach tube with supplemental daily intravenous amogen, glucose, and vitamins” [69]. But it soon became apparent that survivors of major thermal injury evidenced a hypermetabolic, hypercatabolic state which lasted at least until the wounds were closed, and often resulted in severe loss of lean body mass. Burns thus epitomize what David Cuthbertson, summarizing work done with orthopedic injuries, identified as the biphasic response to injury: an initial “ebb” period (shock) was followed by a longer “flow” period (inflammation) [95]. Thus, burns constitute the “universal trauma model,” as described by Dr. Pruitt in the 1984 Scudder Oration on Trauma:

The burn patient in whom a local injury (the severity of which can be readily and reproducibly quantified) evokes a global systemic response (the magnitude and duration of which are proportional to the extent of injury) meets the criteria for a useful clinical model (…) Among all trauma patients, the burn patient should perhaps be regarded as a metabolic caricature, since the metabolic rate in patients with burns of more than 50 percent of the body surface exceeds that encountered in any other group of patients.

Cope and colleagues reported measurements of metabolic rate of up to 180% of normal in the early postburn period, ruled out thyrotoxicosis as an etiology, and recognized a relationship between wound size and metabolic rate [96]. Wilmore and colleagues identified the role of catecholamines
as mediators of the postburn hypermetabolic state (Fig. 1.5) [97]. Wilmore et al. also demonstrated the feasibility of providing massive amounts of calories by a combination of intravenous and enteral alimentation [98]. Curreri published the first burn-specific formula for estimating caloric requirements: calories/day = 25 (wt in kg) + 40 (TBSA) [99]. Provision of adequate calories and nitrogen failed to arrest hypermetabolism and reduced, but did not eliminate, erosion of lean body mass in these patients. Three approaches have recently been taken to address this problem with modest success: use of anabolic steroids such as oxandrolone [100]; blockade of catecholamines with propranolol [101]; and insulin [102], insulin-like growth factor [103, 104], or human growth hormone [105, 106].

1.6.7 Rehabilitation

As postburn mortality decreased, the problems of burn survivors, particularly those with deep and extensive injuries, became paramount [107–109]. The scientific study of rehabilitation of the thermally injured patient is a relatively young field. The CG monograph briefly states:

Six patients who received severe burns to the dorsum of the hands and wrists were referred to the Physical Therapy Department either while in the hospital or at the time of discharge to be treated as out-patients...In all cases surface healing was complete before beginning treatment...The first patient...was referred to this department 51 days after the fire.... [110]

This method, which conceives of rehabilitation as a “phase” which begins after resuscitation and reconstructive surgery phases, may be acceptable in patients with minor injuries. But it soon became apparent that wound healing is so prolonged in patients with major thermal injuries that these three phases must be conducted concurrently rather than sequentially, to avoid the catastrophic effects of chronic bed rest, extremity immobilization, and contracture formation [108]. In the 1950s, Moncrief began rehabilitation soon after admission and resumed it 8–10 days after skin grafting [111, 112]. The advent of heat-malleable plastic (thermoplastic) material made it possible to fabricate increasingly complex and effective positioning devices [113]. This was followed by the introduction of pressure to treat hypertrophic scars and the development of customized pressure garments [114]. Others reduced or eliminated the delay between skin grafting and ambulation, without deleterious effects on graft take [115, 116]. New frontiers for physical, occupational, and neuropsychiatric rehabilitation of burn patients include the following:

- Optimizing pain control; use of novel techniques such as virtual reality [117]
- Documentation of long-term outcomes [118, 119]
- Definition of barriers of return to work and community [120, 121]
- Diagnosis and treatment of posttraumatic stress disorder [122]
- Management of scar formation [123]
1.7 Conclusions

This review indicates that the advances in burn care achieved since WWII were not accidental, but depended on integrated laboratory and clinical research; generous national funding; centers of excellence focused on comprehensive burn care; highly skilled multidisciplinary clinical teams; and committed leadership. Reflecting on recent progress in the 1976 American Burn Association presidential address, Colonel Basil Pruitt noted the importance of a tight working relationship between clinicians and basic scientists, working together to solve problems of clinical significance [124]. This paradigm should be strengthened and expanded, since we have entered an era in which the number of large burns has declined nationwide [125]. As a result, we are challenged with the need for multicenter trials if we are to continue to make progress. Fortunately, the creation of the American Burn Association (ABA) Multicenter Trials Group and federal funding have created the framework and the opportunity for such collaboration. In a manner reminiscent of events in the UK and the USA during WWII, the recent conflicts in Iraq and Afghanistan [126] and the attacks of September 11, 2001 [127] have highlighted the importance of thermal injury as a national problem. The following multicenter trials have been funded by the Department of Defense (at a total cost to date of US $25.7 million) and carried out by the ABA research network during 2008–18 (ABA, personal communication, February 1, 2018):

1.7.1 Completed Studies

• Scar contractures and rehabilitation treatment time [128]
• Restrictive vs. traditional transfusion triggers [129]
• Military and civilian outcomes [130]

1.7.2 Ongoing Studies

• High-volume hemofiltration in burn patients with septic shock and mild acute kidney injury
• Rapid polymerase chain reaction (PCR) test for Staphylococcus aureus infection
• Community-based exercise for adults
• Enteral glutamine effect on infections and mortality
• Inhalation injury scoring
• Effects of propranolol in adults
• Resuscitation of burn shock with albumin
• Resuscitation of burn shock with the Burn Navigator decision support system

The spirit of collaboration and inquiry embodied by these projects is the surest guarantee that they will continue to bear fruit in the years to come.

Summary Box

• There has been a doubling in burn survival since World War II in young persons, measured as the lethal dose 50% (LA50%).
• Integrated laboratory and clinical research and multidisciplinary teamwork have been the foundations of improved outcomes.
• Advances in care responsible for this improvement in survival included these areas: fluid resuscitation, infection control, topical and surgical wound care, inhalation injury care, and nutritional and metabolic support.
• In addition to continued research in these areas, new frontiers are being addressed in wound healing, rehabilitation, and psychosocial recovery.
• We are now challenged by a decrease in the number of patients with big burns in most developed countries. Thus, further progress in burn care will depend in part on multicenter trials.
• The American Burn Association (ABA), for example, has built a successful framework for multicenter burn research.

References

2.1 Introduction

Injury is the physical damage that results when a human body is suddenly subjected to energy in amounts that exceed the threshold of physiologic tolerance [1]. Injury is a significant public health problem—injuries caused 8.5% of all deaths worldwide in 2015 [2]. Injuries are the fourth leading cause of death in men throughout the world (11% of total deaths) after cardiovascular, infectious, and neoplastic diseases. Although progress is being made against many illnesses, the incidence of injuries is decreasing at a rate slower than the reduction in illness in high-income countries (HIC). In low- and middle-income countries (LMIC), both death and disability from injuries are increasing very rapidly. In LMIC of the Americas, Europe, and the Eastern Mediterranean Region, the cause of more than 30% of disability-adjusted life years (DALYs, the loss due to either death or disability of the equivalent of 1 year of good health) among men aged 15–44 years in 2004 was from injury [3].

Injury is a burden on the young, taking more productive life-years than cancer or heart disease. Road traffic collisions are among the leading causes of DALYs lost in LMIC [3, 4]. In 2004, burns under 20% were approximately 6% of all unintentional injuries in children less than 15 years of age [3]. A community-based cross-sectional survey in Egypt found that burn injuries were the second most common type of injury (after falls) in children less than the age of 18 years [5].

Injuries are also the most common cause of DALYs lost worldwide: in 2004, injuries accounted for 17% of DALYs lost in adults aged 15–59 years [3].

Burns are an important mechanism of injury. Unintentional injuries include not only burns but traffic collisions, drownings, poisonings, and falls. Intentional injuries result from homicide, suicide, legal interventions, and conflicts; burns and fires can be the mechanism for assault or self-harm. Without question, burns contribute a significant proportion of the morbidity and mortality attributed to injuries throughout the world.

A burn is an injury to the skin or other organic tissue primarily caused by thermal or other acute trauma, according to the International Society of Burn Injuries. A burn occurs when some or all of the cells in the skin or other tissues are destroyed by hot liquids (scalds), hot solids (contact burns), or flames (flame burns). Injuries to the skin or other organic tissues due to radiation, radioactivity, electricity, friction, or contact with chemicals are also identified as burns.

In 2015, incidence of burns severe enough to require hospital outpatient presentation or an admission to hospital was 31 million people [6, 7]. Burns covering less than 20% of the body surface area occur to 153 per 100,000 population of children aged 0–15 years, making these burns the fifth most

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common cause of nonfatal childhood injuries after intracranial injury, open wounds, poisoning, and forearm fractures [3]. Five percent of disabilities at all ages in Nepal are due to burns and scalds [8].

Low- and middle-income countries represent a disproportionately high level of burn injury incidence and mortality. Data from WHO estimates that 265,000 deaths result from fire-related incidents per year globally [4]. This burden of disease is significantly higher in LMIC, with over 96% of fatal fire-related burn injuries occurring in these countries [4]. High-income countries have made significant progress in reducing the incidence of burn injuries and burn severity, lowering rates of burn deaths, and length of hospital stay through a combination of prevention and care strategies [9–13]. However, many of these improvements have been incompletely applied in LMIC [4].

When confronted with the narrative of a burn survivor, one first pictures agonizing open wounds, followed by resolution into undeniably obvious burn scars. But the thickened, noncompliant skin tells only part of the story. Much of the impact of burns is emotional, psychological, and spiritual. Studies of recovery from burn injury in the United States show clearly that the ability to adjust following injury is less dependent on the physical characteristics of the burn (such as burn size, burn depth, or location) and more on preinjury adjustment. Coping skills, family and community support, and general psychological health have more impact on recovery from burns than the nature of the burn itself [14].

In HIC, this means that burn survivors from struggling family backgrounds are likely to have problems reassimilating into school and community life. In LMIC, the consequences are direr, with isolation from or even abandonment by the family, social segregation, unemployment, and extreme poverty. Although burn victims from affluent families in LIC have a chance of recuperation, the vast majority of burn survivors will start from living situations that deny them the opportunity to recover from even a small burn.

Additionally, the sequelae of nonfatal burn injuries are often severe enough to cause permanent disability. In the Global Childhood Unintentional Injury Surveillance pilot study conducted among children (0–12 years of age) in Bangladesh, Colombia, Egypt, and Pakistan, 17% of survivors had long-term (greater than 6 weeks) temporary disability and 8% had permanent disability [15]. The incidence of long-term temporary disability was highest in children surviving burns and traffic injuries. Only near-drowning victims had a higher rate of permanent disability. Permanent disability was eight times more common in burn survivors than in those children recovering from falls.

Thus the wisdom of one of the founding fathers of burn care in India, Dr. M.H. Keswani: “The challenge of burns lies not in the successful treatment of a 100% burn, but in the 100% prevention of all burn injuries” [16].

### 2.2 Epidemiology

Collection of data specific to burn etiologies has been challenging. The most efficient approach is to join modules specific for injury causation with existing data collection systems. As an example, software was developed to employ the comprehensive categorization of multiple facets of injury events as described by the International Classification of External Causes of Injury (ICECI). This software was then used by registrars collecting and entering data into the National Burn Repository (NBR) of the American Burn Association (ABA). Use of this tool significantly augmented the quality and quantity of etiologic data entered into the existing data repository [17]. A global registry has recently been developed through collaboration among the World Health Organization (WHO), Global Alliance for Clean Cookstoves, and United States Centers for Disease Control (CDC). A process evaluation performed in 30 countries showed good user acceptance and the potential to prioritize the selection, development, and testing of primary prevention interventions throughout the world [18]. Fortunately, although burns and fires throughout the world in 2015 accounted for 238,000 deaths, the vast majority of burn injuries are not fatal [19]. Globally, 2016 data show 337 burns per 100,000 outpatient presentations and 32 burns per 100,000 inpatient admissions [19]. In 2008 there were 410,149 nonfatal burn injuries in the United States, giving an age-adjusted rate of 136 per 100,000 each year [20]. A higher estimate comes from data collected from the National Hospital Ambulatory Medical Care Survey during the period of 1993 to 2004 in the United States, in which the average annual emergency department visit rate for treatment of burns was 220 per 100,000 population [21]. The vast majority of these burn patients were treated and released from the emergency department; only 5% were hospitalized or transferred. In comparison, only 45% of those with nonfatal firearm injuries and near-drowning effects were treated and released, suggesting that the severity of most burns requiring medical treatment is low compared to other types of injury [22].

More severe burns also tend to be less common among hospitalized patients. A European study by Brusselaers et al., which reviewed 76 papers encompassing data on a total of 186,500 patients, found an annual incidence of 0.2–2.9 per 10,000 inhabitants for burn injuries requiring admission to a specialized burn service [23]. Dokter and associates reported an incidence approaching 1 per 100,000 person-years in the Netherlands for burns of 20% total body surface area (TBSA) or greater [24]. Other studies from high-income regions, including Australia, Singapore, and the United States, have reported rates of severe burn injuries (20% TBSA or greater) of less than 20% of burn injuries admitted to hospital [25–27].
Epidemiologic studies from LIC lend insight into the true impact burns have in communities. A cross-sectional survey of nearly 1400 households in Tigray, Ethiopia, revealed that 1.2% of the population is burned each year. Over 80% of these burns occurred at home, and 90% healed without any complications. Only 1% of the burn victims died [28]. A population-based survey of over 170,000 households representing nearly 350,000 children and 470,000 adults during 2003 in Bangladesh showed that the overall incidence of nonfatal burn injuries was 166 per 100,000 and that about 173,000 Bangladeshi children suffer moderate to severe burns each year; this calculates to an annual rate of 288 burns per 100,000 children. Similar to the study results from Ethiopia, 90% of the burns occurred at home. The rate of permanent disability due to burns in childhood was 5.7 per 100,000, and the mortality rate was 0.6 per 100,000 [29–31].

Other studies confirm the relative infrequency with which burn patients require hospitalization. In a study of patients treated for burns at emergency departments in North Carolina, 4% were admitted and only 4% were transferred to burn centers [32]. Based on the incidence of burns treated at emergency departments, and the proportion of those patients requiring admission, it appears that anywhere from 5 to 16 burn patients per 100,000 population require admission for the treatment of their injuries. In Pennsylvania in 1994, hospital discharge records showed the rate of hospitalizations for the treatment of burn injuries was 26.3 per 100,000 [33].

Global data are even more elusive, but an estimate of the frequency with which children are hospitalized throughout the world for treatment of burns is a rate of 8 per 100,000 [34]. In a rural community survey in Ethiopia, burns were the second most common injury to children under 15 years of age. The annual incidence of burns severe enough to restrict activity for one or more days was 80 per 1000 children [35]. Burns were therefore the leading cause of admission for injury to pediatric hospitals in Ethiopia and ranked third as a source of outpatient visits [36, 37].

In the United States, emergency department visits for burn injury decreased between 1993 and 2004 [20]. The absolute number of burn injuries in the United States may be declining, or the severity of those injuries decreasing, or both [38]. Fortunately, a similar trend is being observed overseas. For example, the number of burn patients admitted annually to the Burn Unit of Lok Nayak Hospital and Maulana Azad Medical College, New Delhi, India, from 1993 to 2007 has declined from 1276 to 724 [39].

In parallel with the decline in emergency department visits and hospitalizations for burns, mortality due to fire and flames has declined across the world. The Global Burden of Disease (GBD) 2015 project showed nearly a 10% decline in burn deaths over the previous decade [2]. The two decades from 1982 to 2002 have witnessed a decrease in fire and burn mortality in many countries. During this period of time, for instance, fire and burn mortality in Australian men declined from 1.5 to 0.7 per 100,000. Similarly the fire death rate in Brazilian women went from 1.1 to 0.5 per 100,000. Other countries observing reduction in fire and burn mortality from 1982 to 2002 include Canada, France, Mexico, Panama, Thailand, the United Kingdom, and Venezuela. In the United States, the age-adjusted death rate from fire and burns has dropped from 2.99 per 100,000 in 1981 to 1.2 per 100,000 in 2006 [22].

Yet not all countries have experienced a simple linear decline in the incidence of burn deaths during the last three decades. Significant political and economic upheaval in the nations that used to belong to the Union of Soviet Socialist Republics (USSR) has left its mark on trends in fire and burn deaths. Following a gentle decline through the early 1980s, fire and burn deaths began to rise before and just after the dissolution of the USSR in 1991. By the late 1990s, as capitalism and democracy began to replace communism, death rates again began to decline [40]. National variations in injury-related mortality may be related to individual factors, such as alcohol consumption and risk-taking behavior, as well as alterations in social, political, and environmental factors [41].

2.2.1 The Inequitable Distribution of Burns

As noted by Mock and associates in an editorial in the Bulletin of the World Health Organization, injuries and violence cause disability and death to tens of millions of people across the globe each year, and this burden is unfairly borne primarily by those in LMIC where prevention programs are uncommon and the quality of acute care is inconsistent [42, 43]. Burn injuries are dramatic examples of the inequity of injury.

The majority of burn deaths (90%) occur in lower middle- or low-income countries. Slightly more than 7% occur in HMIC. Only 3% of burn deaths across the world occur in HIC (Fig. 2.1). The rate of child injury death from fire and flames is nearly 2 1/3 times higher in low SDI (sociodemographic index) countries than in high SDI (Table 2.2; Global Health Data Exchange, http://ghdx.healthdata.org/gbd-results-tool, Accessed 12 Sept 2017). In absolute numbers, the proportion of childhood deaths due to fire and flames in LIC is four times that in HIC (Table 2.2; WHO Health Statistics and Information Systems, http://www.who.int/healthinfo/global_burden_disease/estimates/en/index2.html, Accessed 12 Sept 2017). In HIC, although the death rate in children from fire and flames is only 4% of the overall rate of death from unintentional injuries of all kinds, it is over 9% the death rate of all unintentional injuries in LIC.
Even in HIC, burn injuries disproportionately occur to racial and ethnic minorities in which socioeconomic status—more than cultural or educational factors—account for most of the increased susceptibility to burns. In the Republic of Korea (South Korea), for instance, the severity of burn injury is highest in the lowest socioeconomic groups [44]. As another example, the proportion of African-American infants requiring hospitalization at US burn centers for treatment is double the proportion of African Americans in the general population [45]. Similarly, the standardized mortality ratio for fire deaths in 1981–1982 among aboriginals in Manitoba was 4.3 times that of the population of the entire province [46]. Indeed, in many aboriginal communities in North America and Greenland, the third most common cause of unintentional fatal injury is house fires [47, 48].

At the time of burn injury, all patients—young and old—experience shock, horror, pain, and anxiety. The events that follow the injury may confuse the victims and lead them to believe (sometimes correctly) that their death is imminent. Because few burn victims in LMIC receive appropriate first aid or immediate acute care, the medical mismanagement of the burn is likely to lead the survivor to the hopeless conclusion that little or nothing can be done to soothe the pain and relieve the suffering. As a result, burn survivors become emotionally overwhelmed and typically withdraw. They lose interest in food and activity and retreat to dark corners where they may lay motionless for hours. Unfortunately, this lack of activity compounds the speed with which the healing burn wound causes wound contractures to occur, and heightens the survivor’s disability. For these reasons, the distribution of burn morbidity is also imbalanced. The prevalence of moderate and severe disability due to unintentional injuries in people under age 60 is 35.4 million in LMIC, 12.5 times higher than in HIC [3].

Differences in burn mechanism are also noted across income distributions. Flame burns are the most frequent cause of burn injury in adults in HIC, with a higher %TBSA associated with this type of burn. Over 80% of cases in the United States are caused by flame (43%), scald (35%), or contact injuries (8.9%) [25]. Scald injuries are more common in elderly patients; this is consistent with data from Australia, New Zealand, England, Wales, South Asia, and the East Mediterranean [26, 49, 50]. In LMIC however, the incidence of workplace burns, particularly those involving electricity, is higher than in other regions, likely due to differences in safety regulations and poorer infrastructure.

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Fig. 2.1 Low sociodemographic index (SDI) countries disproportionately suffer the impact of fire deaths and burn injuries throughout the world, according to statistics from the Global Health Data Exchange project. (Blue bars represent data about men and red bars about women.) (Global Health Data Exchange. Accessed 12 Sept 2017, at http://ghdx.healthdata.org/gbd-results-tool)

### Table 2.1 Global Burden of Disease Study 2015 (GBD 2015) Results

<table>
<thead>
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<th>Drowning</th>
<th>Fire and burns</th>
<th>Falls</th>
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2.3 Cost of Fires and Burns

Those costs that result as a consequence of fires and burns are incurred as a result of the fire exposure to property, individuals, and/or environment. Approximately 15–34% of the total fire expenditures are associated with cost as a consequence for HIC such as the United States, Canada, the United Kingdom, Australia, and Denmark [51]. These estimated costs include the following [51]:

- Property loss
- Fatalities and injuries
- Health care costs
- Loss of business (e.g., production, market share, public goodwill)
- Loss of wages/employment
- Environmental costs
- Heritage and cultural costs
- Removal of debris

Direct medical costs include costs associated with emergency department care, inpatient admissions, rehabilitation, medications, and investigative tests. Also included in direct costs are outpatient visits, medical home health care, medical devices, and ambulance transfers. Most of these costs are borne either by the patient’s health care insurance plan or by the regional or federal authority, although the amount of reimbursement allowed according to government fee schedules is often less than the cost of treatment for major burns [52].

On the other hand, indirect costs are almost always paid by the patients and their families and can accumulate significantly over time. In Spain, direct health care costs of burn patients represent only 20% of the total cost of care [53]. Indirect medical costs include time spent by family members or other caregivers providing nonmedical care, either in the hospital or at home. Additionally, reduction of working time by both the patient and caregivers results in cumulative lost income. Temporary disability of the patient can evolve into permanent disability, which is particularly disabling for families without provisional plans for short- or long-term disability. Finally, mortality, particularly in the young, leads to a loss of many productive working years, which impacts not only the family but the community as a whole.

In 2015 in the United States, a country with a relatively low incidence of burns, $14.3 billion in property damage was reported, an increase of 23% over the previous year. There were 3280 civilian deaths and 15,700 injuries from residential fires (National Fire Protection Association, NFPA [54]). In England and Wales in 2004, the economic burden of fires was estimated at £2.5 billion and included property damage, casualties (395 deaths and 12,300 injuries), and business disruptions [55]. In Australia, the estimated cost of fires in 2005 was AUD$12.5 million, with an estimate of 100 deaths and 3000 injuries annually [51].

Survivors of moderate to severe burn injuries may suffer a disability that will prevent or diminish employment and income. In 2015, 40,867 total years of potential life were lost in the United States alone because of burn fatalities, including 3483 by suicide and 2699 by homicide [56]. Cost-utility analysis of 898 patients treated at the Burn Center of Valencia, Spain, from 1997 to 2001 revealed that the mean cost per quality-adjusted life-year was US$686.

A mass casualty incident with a large number of patients with burn injuries will create a significant financial hardship for those hospitals assigned to treat the multiple patients with injuries because a significant proportion of these patients will be under- or uninsured. Typical routes of financial support for the care of these disaster victims may be insufficient to cover the cost of care [57]. Expected sources of cost include the additional burden of care of patients in excess of usual operating census. Most burn centers run at a capacity of around 80%, which means that sudden expansion to surge capacity (150% of designated burn beds) would tax human resources and significantly expand expenditures on personnel. Additionally, it may be difficult if not impossible to off-load the burden of mass casualty victims to other hospitals because transportation resources may be compromised by the disaster. Moreover, out-of-state hospitals are reluctant to accept the care of patients in the absence of guarantees by federal or state medical reimbursement programs [58].

A variety of features characteristic of burns lead to prolonged and expensive hospital stays. In addition to pain management and wound care, burn patients require attention to nutritional deficiencies, to the consequences of suppression of the immune system, and to rehabilitation therapy. In the United States, the average hospital charge for care of a child (age 5–16 years) with extensive third-degree burns requiring...
skin grafting is over US$140,000 [45]. In one state alone in 1994, hospital charges for the treatment of burn injuries totaled over $93 million [33]. Yet in spite of this lavish medical care, many burned children leave hospitals in the United States with permanent physical and psychological scars.

During the decade from 1999 to 2008, patients at burn centers in the United States stayed a mean of 10 days in the hospital. The dominant predictors of hospital stay in burn patients are burn size and burn depth [59, 60]. In the time period 2006–2015 involving 69,015 survivors and 2421 nonsurvivors in the 2016 National Burn Repository of the American Burn Association, mean total charges for survivors were $94,130; for patients with burns smaller than 20% TBSA (whose survival rate is >99%), the mean total charges were $60,505. In contrast, mean total charges were $309,656 for nonsurvivors. Mean daily charges for survivors were $8179. In contrast, mean daily charges for nonsurvivors were $24,809. The mean daily hospital charges ranged by age from $7402/day for children under 5 years to $9938/day for the elderly over 60 years (ABA NBR [61]). In the last decade, charges for hospitalization have changed. Compared with data from the 2006 ABA National Burn Repository (ABA NBR [62]), mean charges for survivors rose from $57,575, an increase of 35% when adjusted for inflation. This may be because hospital charges per day for survivors increased from $4336 to $8179, a rise of 56% when adjusted for inflation.

Loss from burns and fires includes not only health care expenditures and property damage, but destruction of human resources as well. In 2006, nearly 70,000 years of potential life were lost in the United States because of burn fatalities [20]. The indirect costs of such loss of productive years of life arise from the absence of useful employees from the workplace and lack of wage earners in families.

**2.3.1 Cost by Age**

In some cases, where charges may be viewed as a surrogate for intensity of care, certain trends are apparent. The presence of comorbid medical conditions typical in the elderly increases the need for more complex services and longer hospitalizations. Whereas the mean hospital charge per day for survivors was only $2900 for children aged 1–5 years, it was $4700 for elderly adults 60 years of age or older. Elderly patients admitted to a New York City burn center from 2000 to 2004 for the treatment of scald burns incurred mean hospital charges of $113,000 per patient, even though the burns were relatively small (mean 7% TBSA) [63]. In contrast, mean hospital charges per day for fatal cases in US burn centers from 1999 to 2008 were $8850 for children aged 1–5 years, compared to only $9400 for elderly adults, suggesting that more intense utilization of resources used in attempts to salvage dying children [45].

At hospitals contributing to the ABA National Burn Repository, charges for survivors ranged from a mean of $60,505 for patients with burns less than 20% TBSA to $845,616 for patients with burns greater than 80% TBSA. For nonsurvivors, charges ranged from $224,807 for patients with less than 20% TBSA burns to $182,428 for patients with greater than 80% TBSA burns. In general, hospital charges are less for nonsurvivors than for survivors, for all burn sizes greater than 30% TBSA, because the shortened hospital stay of nonsurvivors reduces cost of care [61].

The majority of patients who seek medical care for their burn injuries can be treated and released from emergency departments. For example, in 2010 only 6.4% of burn patients in the United States were hospitalized [56]. The average cost (medical cost and work loss) of treating outpatients is much less ($5394) than that for treating inpatients ($65,022), but because of the vast difference in patient numbers (380,397 vs. 25,823, respectively), the total cost of treating outpatients each year in the United States is more than the total cost of treating inpatients ($2.1 Bn vs. $1.7 Bn). In addition, the proportion of total cost as work loss is far greater in outpatients ($1.4 Bn vs. $761 Mn).

As the proportion of the US population above 60 years of age grows, shifts in expenditures for burn care will occur. From 1999 to 2008 in the United States, the percentage of patients admitted to burn centers who used Medicaid for health insurance stayed the same at nearly 13% of all patients. However, with the aging of the population, the percentage of Medicare-insured patients rose in the same time period from 9 to 12%. During that time period, the proportion of Workers Compensation patients sank from 13 to 8%, reflecting the departure of working adults into retirement [45].

Children are also particularly impacted by thermal injuries and smoke inhalation. Fire and burn injuries resulted in the deaths of 1461 children in the United States in 1985. Children treated for burns totaled 440,000, of which nearly 24,000 were hospitalized. The society losses from these childhood burn injuries and deaths were estimated at approximately $3.5 billion [64]. The Multicenter Benchmarking Study at the Shriners Hospitals for Children-Boston estimated the cost of hospitalization for a cohort of 230 pediatric burn patients from between 2001 and 2009 [65]. The average number of hospitalizations was two per patient, typically over a 3- to 4-year period of time. The median cost of hospitalization in 2006 USD was $16,331.

Fortunately, the majority of young children have small burns requiring short hospitalizations. Seventy-five percent of children between the ages of 1 and 5 years in the United States from 1999 to 2008 were burned over less than 10% of their body surface area. These young children with small burns spent an average of only 3.6 days in the hospital [45]. In addition to children being healthier than their older counterparts prior to injury, children are more likely to be injured...
by hot liquids than by flames, and there are significant cost differences between the two burn mechanisms.

Using the Healthcare Cost and Utilization Project Kids’ Inpatient Database for 2000 in the United States, retrospective data analysis of pediatric burn–associated hospitalizations was performed. This analysis permitted an estimate that 10,000 children younger than 18 years were hospitalized for burn injuries during that year and that the charges for these hospitalizations totaled over $211 million. The mean length of stay was 6.6 days, and only 10% of admissions lasted longer than 14 days. Because of the predominance of short lengths of stay, mean charges were only $21,840 per patient, and only 10% of patients accumulated charges in excess of $47,000. More than half of admissions were children younger than 2 years, and males outnumbered females at all ages. Children under 2 years were more likely to suffer from scald burns, whereas older children were more likely burned by fire or flame.

### 2.3.2 Cost by Mechanism

Fire and flames are responsible for the bulk of the cost of burns. In 2008, fire departments in the United States responded to nearly 1.5 million fires. There were 16,705 fire injuries, 3320 fire deaths, and nearly $15.5 billion direct property losses. A fire death occurred every 158 min in the United States in 2008. The majority of the lost years of life are due to fire and flames (68,272), with only 1218 years of life lost due to scalds or contact burns.

In 2010, the combined costs (medical costs and work loss) of burn care for all fatal burns approached $26 Bn. Of those costs, nearly 99% were attributed to deaths by fire and flames, compared with only $36 Mn for scald and contact burns.

The hospital charges per day in Pennsylvania in 1994 for the treatment of flame burns from conflagrations were $4102, compared to $2187 for scald burns. This difference reflects the difference in depth of burn (flame burns are more likely to be third degree in depth than are scald burns) and the subsequent additional intensity of resources needed to treat third-degree flame burns and smoke inhalation injury, which include intensive care, surgery, blood transfusions, and antibiotics.

Data from LMIC regarding cost of burn treatment are scarce, but there are studies that corroborate the US experience that flame burns are expensive. For example, the cost of care for patients injured by kerosene stoves is high in LMIC. In 2003 in Cape Town, South Africa, the mean total cost per patient was US$6410. Extrapolating these costs to South Africa nationwide gives an estimated annual expense of US$26,250,000, which is more than 50 times the amount expended annually for kerosene in South Africa.

Nonetheless, because of the frequency with which scald burns occur, the cost of care for scalds is significant. Annual charges for treatment of scald burns in US children less than 14 years old is approximately $2.1 billion. Sixty percent of these charges are for children under the age of 5 years. Again, indirect costs are difficult to quantify, but are no doubt significant because each day a child is hospitalized or home ill with burn injuries, is a day that one of the parents or caregivers has to miss work. In addition, the cost of burn wound dressings is frequently not covered by most insurance policies, leaving the parents responsible for purchasing supplies out-of-pocket.

The cost of care for electrical injuries is typically much higher than the cost of care for flame or scald burns. In Ankara, Turkey, the mean total cost of electrical burns between 2005 and 2008 was US$22,501, compared with US$15,250 for other types of burn injury.

### 2.4 Limitations of Data

The majority of uncertainty in estimates of death in the Global Burden of Disease reports is associated with the assessment of systematic errors in primary data. That is, information about prevalence, incidence, and mortality from injuries is generally fragmented, partial, incomparable, and diagnostically uncertain. To estimate uncertainty for regional mortality, a simulation approach was used to create uncertainty ranges that take into account uncertainty in the expected number of total deaths, uncertainty in the diagnosis of underlying cause of injury, and uncertainty arising from miscoding of cause, among others. Based on these estimates, the range of uncertainty for fire deaths is 3000 to 5000 deaths lower or higher than the estimates for fire deaths in East Asia, the Pacific, Europe, and Central Asia. Even more uncertain are the estimates in South Asia and sub-Saharan Africa, where the range of uncertainty surrounding the stated estimates is 10,000 to 14,000 deaths lower or higher. Thus the real number of fire deaths each year may be almost 30,000 higher than the estimate of 310,000. Sources of uncertainty for estimating burden of injury in the GBD reports include:

- Incomplete information
- Biases in information