

Incendiary Weapons



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Chapter 3. Thermal effects of incendiary weapons on the human body

I. Introduction

A vital consideration in arguments about military necessity *versus* humanitarian priorities is the medical aspect of the use of incendiary weapons. Such questions as the nature of the casualty effects, the problems of medical treatment, the prospects for the surviving casualties, and the likelihood and the time of death are all relevant to any assessment of the amount of suffering caused to victims of incendiary attack. From the viewpoint of the international laws of war, this amount of suffering has somehow to be weighed against the "military necessity" of the use of incendiary weapons, for it is presumably only in this way that it may be determined whether all or a part of this suffering is "unnecessary".

The traumatic effect of most weapons is to penetrate the skin and damage internal organs. Although the action of flame weapons is essentially to damage only the skin, fire may cause death in a number of ways so that the exact cause may be difficult to determine in a given case. In general, death by fire may be due to the following causes: (a) heatstroke, caused by the transfer of heat to the body sufficient to increase the body temperature above a critical level (approximately 43°C), (b) pulmonary damage due to inhalation of smoke and toxic fumes, (c) carbon monoxide poisoning, (d) deprivation of oxygen, and (e) severity of the burn wound in terms of depth and the extent of the body surface area affected.

The likelihood of death from burn wounds is also affected by such factors as the age, the general health and the nutritional status of the victim, and the potential risk of infection.

Heatstroke may be an important cause of immediate death from fire, but it is not a significant cause of later death. The same applies to carbon monoxide poisoning and the deprivation of oxygen, since these reactions are also reversible (although some long-term injury may result: see chapter 4). Major factors related to the death rate from burns, after initial survival, are the degree of pulmonary injury and the extent of the body surface area involved. The age of the patient and his general health and nutritional status are additional factors which may influence the prognosis. One analysis of the complications causing or contributing to death due to burns is shown in table 3.1.

Where toxic and respiratory complications are not present, the mortality from burns in individuals of given age and general health depends pri-

Table 3.1. Complications from burning which caused or contributed to death

Number of cases		Number of cases	
Non-bacterial complications		Bacterial complications	
Early shock and/or cardiac failure	1	Septicaemia	60
Early hyperkalaemia	18	Bronchopneumonia	56
Other shock	19	Pyelonephritis	16
Subtotal		Meningitis	2
		Peritonitis	2
		Enterocolitis	3
Respiratory complications		Gas gangrene	1
Carbon monoxide	2	Extra-renal abscess	1
Inhalation of smoke	3	Total bacterial complications	141
Glottic burns	4		
Obstructive tracheobronchitis	2		
Blast injury to lungs	1		
Congestive atelectasis	9		
Pulmonary oedema	2		
Inhalation of vomit	2		
Subtotal	25		
Acute cardiac complications			
Toxic myocarditis	1		
Cardiac arrest	4		
Subtotal	5		
Renal failure	24		
Hypokalaemia	4		
Pulmonary embolism	5		
Hepatic jaundice	2		
Acute dilatation of the stomach	3		
Paralytic ileus	1		
Agranulocytosis and thrombocytopenia	1		
Haemorrhage from acute duodenal ulcer	1		
Anoxic cerebral softening	1		
Subtotal	42	Total non-bacterial complications	91
		Total all cases	232

Source: Sevvit (1966).

marily upon the depth and extent of the burn, because of the physiological importance of the skin as a vital organ of the body. This is true whether death results from inadequately treated shock in the early stages of burn care, subsequent infections or other less frequent causes of death such as burn-induced gastric ulceration. Except for electrical burns, characterized by the deep destruction of muscles and other tissue but with limited skin damage, the causative agent is less important in determining the outlook for a burn patient.

For these reasons, the major thermal effects of incendiary weapons on the human body to be described in this chapter relate to burns of the skin, although reference is also made to the so-called "pulmonary burns", which have now been shown to be due more to the inhalation of smoke and fumes than to actual thermal burns of the lungs. The toxic effects of incendiary

weapons are described in chapter 4, where further reference is made to lung irritant effects.

It is sometimes useful to distinguish between *primary* burns, due to the direct effects of an incendiary agent on the body, and *secondary* burns, due to the combustion of clothes, equipment, houses, and so on. Primary burns caused by military incendiary agents are likely to be especially severe since they are the result of extensive technological efforts to increase both the heat of combustion and the burning time. Secondary burns resulting from incendiaries are in principle little different from burns experienced in civilian life, about which there is a voluminous medical literature. In this chapter, the general medical literature on burns is surveyed in order to convey some indication of the complexity and severity of the burn wound and the resulting demands on treatment facilities. More specific effects of the use of incendiary weapons are also described. The chapter concludes with a discussion of some additional factors which complicate medical treatment—and therefore the prognosis for the patient—in wartime conditions.

II. Pathological effects of burns

The skin as a vital organ

The significance of the physiological role of the skin is not commonly appreciated. The skin is not only an essential part of the body, but it also has considerable psychological significance to the individual. In both cases this is because the skin forms the interface between the individual and the outside physical and social world.

A living organism may be described as an "open system" maintaining itself in a dynamic equilibrium with its environment. This equilibrium, known as homeostasis,¹ is maintained by balancing many processes, including the ingestion of foodstuffs and the excretion of waste products, the inhalation of oxygen and the exhalation of carbon dioxide, and the imbibing of water in order to compensate for the loss of moisture by evaporation or excretion.

The skin plays a vital role in maintaining homeostasis, particularly in controlling the temperature of the body and in limiting evaporation of moisture from it. When food is metabolized in the body it produces heat enabling the body to maintain the normal temperature of about 37°C. The skin acts as an insulating layer, actively controlling the loss of heat to the surrounding air, and thereby diminishing the food intake needed simply to provide the body's "central heating".

¹ *Homeostasis*: The maintenance of a state of equilibrium within the body with respect to temperature, heart rate, blood pressure, water content, blood sugar level, and so on.

Preventing water from evaporating from the body is important for three main reasons. First, evaporation has a cooling effect. Without control by the skin the heat loss due to evaporation places excessive demands on the body's capacity to generate heat by metabolism. Second, water is essential for the transport of substances in the body. If water is lost by evaporation, the body fluids become more concentrated, and in an effort to restore the balance, water passes out of the cells into the fluids surrounding them, and may be lost from the body by further evaporation. The cells can only tolerate a minor water loss before they die. Third, the effect of the loss of water, whether due to the loss of circulating blood from damaged blood vessels or by evaporation from open wounds, is to decrease the flow of fluids through major organs such as the brain, heart and kidneys, threatening these major body systems with injury or death.

Further, the skin is a major means of contact with the environment, containing nerve endings which provide the brain with a continual feedback of information about pain, temperature, touch and pressure through proprioceptive sensors. If the skin is removed, these sensors are removed with it, depriving the brain of much of the information necessary for adaptive behaviour.

Although much of the skin is usually clothed for social and practical reasons, it has great psychological importance for the individual since, being the outside of the body, the skin is most accessible to observation and touch by other persons. Damage and deformity to the skin, particularly in such important areas as the head and hands, have far-reaching psychological consequences.

The skin is made up of several layers. The outer layer, the *epidermis*, is composed of non-living, hard-wearing cells and in some parts of the body, such as the soles of the feet, may be quite thick. As these outer cells are worn away, they are constantly replaced by cells from the underlying layer, called the *dermis*. This layer is composed of living cells and contains all the functional components of the skin—small blood capillaries, nerve endings, sweat glands and hair follicles. Dilation of these blood capillaries enables the body to increase the flow of blood at the surface of the body, where heat is more readily dissipated. Contraction of these vessels helps to conserve the heat of the body. Changes in the state of these blood vessels are responsible for changes in the colour of the skin when a person is too hot or too cold. Perspiration from the sweat glands is another means of dissipating heat from the body, while the hair is a means of conserving heat, albeit one which is largely vestigial in man.

Underneath the skin is a layer of adipose tissue, made up largely of cells containing large globules of fat, which also protects and insulates the body. The thickness of this layer varies considerably from one individual to another and in different parts of the body.

Stoll & Green (1959) showed that the receptors in the skin which are

effective in mediating the pain sensation have a threshold of approximately 43.2°C.

In a major series of experiments Moritz and his colleagues (Henriques & Moritz, 1947; Moritz & Henriques, 1947; Moritz, 1947) showed that the lower temperature limit for a cutaneous burn due to hot water is approximately 44°C.

At 44°C it took about three hours to raise the temperature of the underlying skin at the dermis-fat interface to 44°C, damaging the full thickness of the skin. At a temperature of 55°C, it took only 0.4 minutes to create the same effect. At 100°C it took only some 0.1 seconds to destroy the epidermis, but in that short time there was no significant temperature increase at the dermis-fat interface. That is, the physiological changes in the skin due to burns depended upon both the temperature and the time of contact with the burning agent: the hotter the agent, the shorter the contact time required to destroy the tissue.

It will be obvious from the description of the functions of the skin that the severity of a cutaneous burn wound depends not only upon the depth of the burn but also upon the extent of the body surface area affected, which determines the loss of fluids and heat and therefore the impact on the major physiological systems of the body.

The depth of burns (degree)

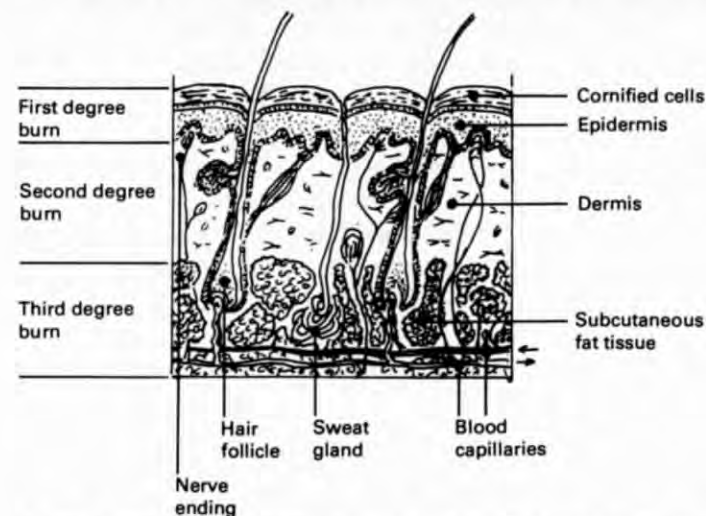
Burns which only injure the epidermis, such as mild sunburn, are known as *first degree* burns (see figure 3.1). They result in temporary erythema (redness), due to dilation of the capillaries, and oedema (swelling). They usually heal within a few days after sloughing off the epidermis, and leave no scar.

Burns which extend into the living layer of the skin, the dermis, are much more serious. It is common to distinguish between *second degree*, or partial thickness, burns, and *third degree*, or full thickness, burns. Second degree burns denote those in which necrosis² extends into the dermis, but with the survival of a sufficient foundation of such skin appendages as sweat glands and hair follicles to ensure that the skin regenerates without having to heal from the edges of the wounds. There is a wide variation in the severity of these burns. Some, such as severe sunburn with blistering, will heal within a week or two and leave no scars; others will heal within a month with reasonable care; and still others will not heal spontaneously unless specific treatment is given to prevent the destruction of surviving epithelial³ cells of the skin by secondary infection and to encourage these cells to grow. Third degree, or full thickness, burns are those in which all the dermis is destroyed.

² *Necrosis*: The death of one or more cells or a portion of a tissue or organ.

³ *Epithelium*: A thin layer of tissue without blood vessels which covers all the free surfaces of the body, including eyes, glands, respiratory passages, and so on.

Figure 3.1 Schematic outline of cross-section of the skin showing degrees of burn^a



^a First degree, or superficial burns, involve only the epidermis and heal rapidly. Second degree burns damage the upper part of the dermis but islets of living dermal tissue remain and proliferate to cover the area in about 14 days. Third degree, deep, or full thickness, burns extend through the dermis and may extend to underlying tissues. Some authors refer to burns which extend into underlying tissues as fourth and fifth degree burns. In general, third degree burns greater than 1-2 cm² in area require skin transplantation in order to heal.

In addition there may be destruction of the underlying fat, muscle, bone and other tissues. The terms *fourth* and *fifth degree* burns are sometimes used to describe such injuries, although in general this nomenclature is no longer favoured in the international literature, as specific deeper injuries of this sort are better recorded as such.

The depth of the burn is easily misjudged and depends upon the cause, the temperature, the length of exposure, and the thickness of the skin (the soles of the feet, the palms of the hands, and the back being thicker than elsewhere). Intense heat over a short period may produce a burn which *looks* very much like one produced by less heat over a longer period, although the latter may destroy more of the underlying dermal elements. Both first and superficial second degree burns blanch on pressure with an instrument such as a sterile microscope slide; the colour returns when the pressure is released. Third degree burns do not blanch or flush on pressure or release. The persistence of sensation, which is easily tested with a sterile needle, is often helpful in differentiating second and third degree burns, although this test is not completely reliable. Destruction of the full skin thickness kills the nerve endings, resulting in loss of feeling (Phillips & Constable, undated). Even experts may have difficulty

in estimating burn depth, and subsequent sepsis⁴ may invalidate initially correct judgements by converting a second degree burn to a full thickness injury. This is particularly likely to happen in malnourished anaemic patients, who also commonly have no access to adequate medical facilities. Deep second degree burns often heal with severe scarring.

Deep burns heal only after the dead tissue is removed either by unassisted or natural separation, which usually involves bacterial infection, or by repeated dressings (debridement) or surgical excision. The 'clean' burn wound may then heal by epithelial ingrowth from the edges, if it is less than 1–2 cm² in area, by the effective surgical closure of small wounds, or by the application of skin grafts from other parts of the body.

The cropping of skin grafts leaves painful donor sites, comparable to second degree burns, which usually heal in about two weeks with no more than minimal scarring. Under less than optimal conditions, however, sepsis may intervene and these donor sites, in turn, become areas of full thickness or third degree skin loss. This may account for the reluctance of surgeons to apply skin grafts to burns which otherwise appear to necessitate them when patients are in poor condition and medical facilities are minimal. At present, permanent skin grafts can only be taken from the recipient himself, identical twins excepted.

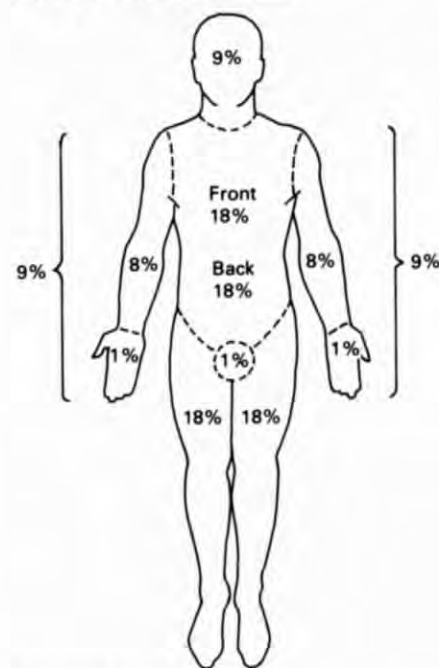
Second degree burns are characteristically extremely painful until they are essentially healed. Third degree burns, because of the destruction of the nerve endings in the dermis, are characteristically not strikingly painful during the first few days after the injury. With progressive healing and removal of the insensitive overlayer of dead tissue, the wounds become more and more sensitive until covered with grafts or by the ingrowth of epithelium. Daily changes of dressings are excruciatingly painful, equivalent to tearing off the outer, insensitive layer of the skin from the inner, sensitive layer.

The extent of burns

The second factor determining the severity of burns is the extent of the body surface area involved, which is significant for several reasons. The greater the area of skin destroyed, the greater the loss of moisture and heat by evaporation, which are major factors in the pathophysiology of the burn injury. Second, the greater the area involved, the greater the problem of coping with infection. Third, the greater the area of skin destroyed, the less skin is available for grafting—a procedure which is all the more necessary. As a result of these and other factors there is a direct relationship between the extent of a burn of a given depth and the chance of sur-

⁴ *Sepsis*: The localized or superficial presence of various pus-forming and other pathogenic organisms or their toxins which kill the tissues. The presence of microorganisms or their toxins in the circulating blood may give rise to systemic disease, a condition known as *septicaemia*.

Figure 3.2 The "rule of nines" as a method for estimating the approximate extent of burns in an adult



Source: After Berkow (1924).

vival. A convenient means of estimating the extent of body area burned was introduced by Berkow in 1924, and is often presented as the "rule of nines" (figure 3.2). The face or an arm make up approximately 9 per cent of the total body surface area, whereas a leg, the front (chest and abdomen together) or the back each make up about 18 per cent, a multiple of 9. The proportions are only approximate and are somewhat different for a child under five years of age (see table 3.2). The palm of the hand and fingers make up about 1 per cent of the body surface area, so that the extent of the burn may be measured approximately by the number of "hands" required to cover it.

The severity and mortality of burns

As a rough guide, third degree burns of less than 5 per cent of the body surface area may be regarded as light, while those of more than 10 per cent, or first and second degree burns of more than 30 per cent, are regarded as severe. In addition, any third degree burns affecting the face, hands,⁵ feet and genitalia are classified as severe, as well as circumferential

⁵ Burns of the hands account for approximately one-third and burns of the face for approximately one-quarter of all burns (Tubiana, Baux & Kenesi, 1967; Converse, 1967; Kovacic, Aaby, Hamit & Hardway, 1969). See table 3.10.

Table 3.2. Methods of calculating approximate extent of burns over body surface

Area of body surface	Surface area formulae		Rule of nines
	Adult or child over 5 yrs of age	Child under 5 yrs of age	
Head	6	15	9
Anterior trunk	18	18	9 × 2 = 18
Posterior trunk	20	20	9 × 2 = 18
Both upper arms	7.5	18	9 each = 18
Both forearms	6.0		
Both hands	4.5		
Both thighs, incl buttocks	19	13	9 each = 18
Both legs	13.0	10	9 each = 18
Both feet	6.0	6	

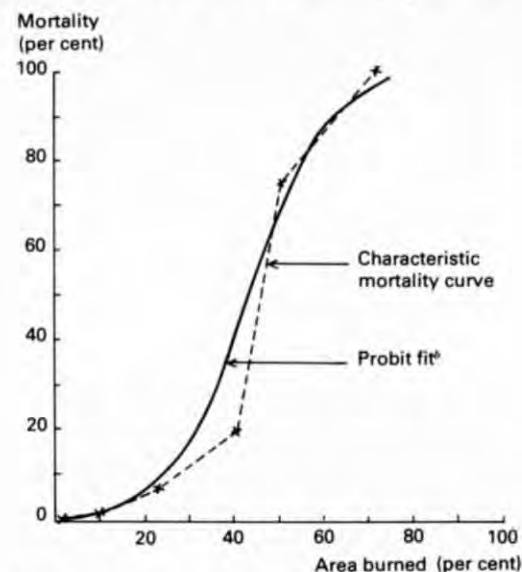
Source: Phillips and Constable (undated).

deep burns of the limbs which affect the circulation to the extremities. Arturson (1966) adds to the category of severe burns those which are complicated in the following ways: (a) soft tissue wounds and skeletal damage; (b) complications of the respiratory passage; (c) electrical burns; (d) simultaneous radiation injury, and (e) burn wounds due to white phosphorus, mustard gas or other chemicals.

First degree burns have no significant mortality. Superficial second degree burns, however extensive, are rarely fatal if some medical care, such as that sufficient to maintain an adequate fluid intake in a hot climate, is available. The percentage of deep second degree and third degree burns in a given individual can be combined for practical purposes in estimating probable mortality, although the mortality from the third degree burn will generally be greater. With *adequate* treatment, deep second and third degree burns of less than 15 per cent are unlikely to cause death, whereas burns of over 50 per cent often do (cf. Bull & Squire, 1949). With ideal treatment young adults with burns of up to 85 per cent of the body, including 70 per cent full thickness burn, have survived (Birke, Liljedahl & Nylén, 1970).

In general, there is a statistical relationship between the extent of the burn and the mortality, which is shown in figure 3.3. The sigmoid relationship has been noted by a number of authors, such as Clarkson & Lawrie (1946). It implies that even for relatively mild burns (e.g. 5–15 per cent in adults aged 15–44 years) a few individuals less able to withstand the physiological stresses resulting from burning will die. A similar relationship has been noted in the case of some other biological phenomena, such as the relationship between mortality and the dose of toxic drugs (cf. Gaddum, 1933). Acceptance of the idea of a sigmoid relationship be-

Figure 3.3. Characteristic relationship between mortality and severity of burn^a



^a The figure illustrates the typical relationship between the percentage of patients dying (the mortality) and the average area of the body surface burned, which is a measure of the severity of the burn. The data are averages drawn from various groups of patients between 15 and 44 years old (total sample=311 patients).

^b The real data approximate to an ideal curve indicated by the probit fit. It is assumed that the larger the number of data, and consequently the larger the number of points it is possible to plot, the more the actual data would approach the ideal sigmoid distribution.

Source: Bull & Squire (1949).

tween mortality and burn severity is of great clinical importance, since it suggests that in theory some probability of survival remains even for the most seriously burned patient.

This . . . is preferable to the old idea that a burn of a certain proportion of the body surface was necessarily fatal. If each burned patient is regarded as having a chance of recovery, every effort will be made to help by means of treatment (Bull & Squire, 1949, p. 171).

A mathematical method of analysis, known as probit analysis, was set out by Bliss (1938), among others, and developed by Finney (1947). The method was first applied to burn data by Bull & Squire (1949) and is now common practice.⁶ It enables estimates to be made of the severity of a burn likely to produce any level of mortality. For instance, in Bull & Squire's original data (figure 3.4) a 51 per cent burn in the 0–14 age group was likely to

⁶ Bull & Squire (1949) described the rationale for this method and constructed an actuarial table from their data for the probability of death (the mortality risk) for an individual of given age and area of burn. Bull & Fisher (1954) published revised estimates for calculating the mortality risk which have since been used as a standard of comparison with other studies.

Table 3.3. LA₅₀ for some large treatment series

Age (years)	Baxter & Shires (1968) saline	Bull & Fisher (1954)	Bull (1971) before AgNO ₃ , 1944-64	Bull (1971) after AgNO ₃ , 1965-70	Pruitt <i>et al.</i> (1964) before sulpha-mylon, 1950-60	Birke & Liljedahl (1968) dextran group A, 1954-59	Birke & Liljedahl (1968) plasma group B, 1960-66
under 15	36	49	48	64	49	45	75
15-44	27	46	47	56	56	43	64
45-64	23	27	29	40	29	36	54
over 65	7	10	10	17		23	30

Source: Birke & Liljedahl (1971).

produce death in 50 per cent of the patients treated. The great advantage of this method is that it enables an assessment of the effectiveness of treatment to be made by comparing the *actual mortality* with the *predicted mortality* from the probit analysis. Further, Bull & Squire suggested that

just as the toxicity of a drug is best expressed in terms of the dose needed to kill 50 per cent of a batch of experimental animals (usually known as the Lethal Dose of 50 per cent or L.D. 50), so we may assess the treatment of a series of burned patients in terms of the area of burn producing death in 50 per cent of cases (i.e. Lethal Area for 50 per cent, or L.A. 50) (Bull & Squire, 1949, pp. 166-167).

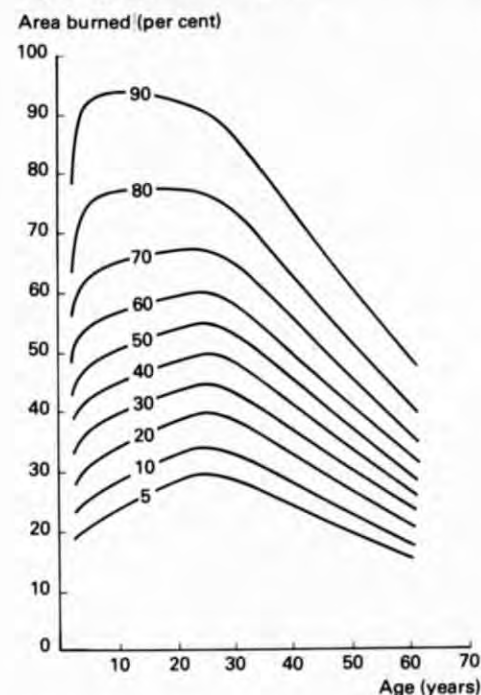
That is, the greater the LA₅₀, the more successful the means of treatment. Table 3.3 shows the results of a series of studies by different authors, some of which indicate progress in the treatment of burns as new methods were introduced.

The influence of age on mortality

The age of the patient is a major factor in the mortality of burns, with the elderly surviving burns less effectively (see figure 3.5). A *very rough* rule for adult patients is that if the age of the patient and the percentage of the body surface area burned added together exceed 100 then survival is unlikely. For example, a 50-year-old patient with a 40 per cent burn may hope to survive; an 80-year-old patient with a 25 per cent burn is unlikely to do so.

* In the mortality data provided by Bull & Squire (1949), all children within the age group 0-14 were classified together, and these data indicated a relatively good capacity to survive burn injuries (see figure 3.4). Other authors, by contrast, believe that very young children have less ability to survive burns than do young adults. Data provided by Pruitt, Tumbusch, Mason & Pearson (1964) confirm such a view, and these authors suggest a possible explanation.

The present data on the younger age groups (0-4 and 0-14) . . . would suggest that these younger groups are able to withstand burn injury of up to 30 per cent of the body surface virtually as well as the older groups. Beyond that level, how-

Figure 3.4. Typical mortality curves (deaths in per cent) for burned patients according to age and extent of burn^a

^a Data from 1100 patients treated at the US Army Surgical Research Unit, 1950-1960.

Source: Pruitt, Tumbusch, Mason & Pearson (1964).

ever, this group appears significantly less able to withstand comparable percentage area burns. The large area to weight ratio in children may explain this greater effect in terms of mortality of the larger burns in the younger group (Pruitt *et al.*, 1964, pp. 399-400).

The higher susceptibility to burns of very young children is also reported by Winterscheid & Merendino (1960) and Rittenbury, Madox, Schmidt, Ham & Haynes (1966).

Similarly it is well known that older patients tolerate a burn wound very poorly (Moyer, 1954). In one large series, no patient over 65 years with a burn of 25 per cent of the body surface area survived, and as many as 18 per cent of the older patients died with burns of only 4 per cent or less (Rittenbury *et al.*, 1966).

This means that the young adult of military age and standard of fitness is more able to withstand the physiological stresses of the severe burn than the young child or the elderly person. Because of the difference in the age distribution, it is clear that a sample of the general civilian population subjected to burns risks a greater proportion of deaths than a military sample of the same size.

The time taken to die

An important consideration in evaluating the suffering resulting from different weapons is the time taken to die. If a victim is going to die he will in general suffer less if he dies immediately than if he dies slowly. The implication of the legal expression that weapons should not "render death inevitable" is that weapons, where they do not kill immediately, should offer the wounded person a high probability of recovery.

So many factors are involved in different wounds that it is difficult to make meaningful comparisons. However, there are indications that, where adequate medical facilities are available, simple penetrating and blast wounds in general approach the above humanitarian criterion. For example, a study of 45 094 US Army casualties in Viet-Nam between 1965 and 1967 showed that 5387 (11.9 per cent) were killed in action, 623 (1.4 per cent) died of wounds, and 39 707 (88.1 per cent) survived. Of those who died of wounds in hospital, 62.3 per cent died within the first 24 hours, and it was concluded that a high proportion of these early deaths represented casualties which, in previous wars without highly effective helicopter evacuation, would have been classified as "killed in action". Of the wounded in action 43.3 per cent were returned to duty within the theatre of operations within 30 days and 54.2 per cent were evacuated for further treatment (Whelan, Burkhalter & Gomez, 1968). These figures indicate that, while conventional weapons are by no means "perfect", nevertheless, death tends to be relatively rapid or the chances of recovery relatively good.

There are indications that burn wounds, by contrast, conform less to such a humanitarian criterion. While there is little doubt that war burns have a high early mortality, in many cases death may not ensue for days or even weeks. Ironically, the better the treatment available, the more drawn-out the period of dying may be. That there is evidence to support such a conclusion may be seen from the following surveys.

Fifty-one girls of the Hijiya High School in Hiroshima, Japan, were outdoors in the school grounds, less than one kilometre from the explo-

Table 3.4. Day of death of 51 girls, at the Hijiya High School, Hiroshima, Japan, who received severe burns from the atomic bomb explosion

Day after the explosion	Daily death rate of the 51 girls
1	10
2	7
3	15
4	9
5	4
6	5
7	1

Source: Oughterson & Warren (1956).

Table 3.5. Average day of death from burns in a five-year survey (1963-1967) of hospitals in Melbourne, Australia^a

Hospital	Number of patients	Number of deaths	Average post-burn day of dying
1	195	16	17
2	123	10	11.4
3	119	19	18.8
4	144	20	20.9

^a The original data includes a further 111 patients at four other hospitals. Only five of these patients died.

Source: MacLeod (1970).

sion of the atomic bomb. They were all severely burned, and mortality within one week was 100 per cent. However, only 10 of the girls died on the first day; the largest number died on the third day (table 3.4). At the Kameyama Hospital, also in Hiroshima, peak mortality from burns occurred on the fourth day, although another peak in deaths occurred in the third and fourth weeks, when complications, including those associated with radiation injury, set in (Oughterson & Warren, 1956).

In a recent five-year survey of eight hospitals in Melbourne, Australia, it was found that on average death from burns occurred 10 to 20 days after the injury, depending on the quality of the hospital treatment (see table 3.5). The author concluded:

Table 3.6. Day of death for severely burned patients in Stockholm, Sweden^a

Case	Age years	Extent of body surface area burned per cent		Post-burn day of dying
		Total	Third degree	
1	22	100	98	11
2	38	95	90	7
3	34	95	75	20
4	60	85	80	32
5	57	80	75	9
6	47	75	70	13
7	44	75	70	10
8	75	70	70	9
9	64	65	60	6
10	58	65	60	14
11	74	60	50	19
12	74	45	45	15
13	79	45	40	38

^a The original data includes an additional 50 patients, none of whom died. All patients were treated with the modern warm dry air method.

Source: Birke & Liljedahl (1971).

... 20 years ago 75 % of deaths occurred from shock in the first three days, [whereas] only the most severely burnt now die at this stage ... although over recent years the interval before death has been prolonged, there has been little reduction in overall mortality (MacLeod, 1970, p. 776).

Birke & Liljedahl (1971) reported the time of death for a series of civilian burn patients in Sweden (table 3.6). In no case did death occur in less than six days, and even for patients with a very high probability of dying, death was in some cases drawn out to three weeks or one month. The (US) Brooke Army Medical Center (1972) reported that, for patients treated between 1965 and 1971, the average time taken to die was 14.9 days after the burn.

These indications are not conclusive, but they are suggestive of a significant difference between the time of death caused by conventional penetrating and blast weapons and that caused by incendiary weapons. It has not been possible to trace data in the public literature which would enable a clear statement to be made on this aspect of the problem, and further studies are warranted.

Pathophysiology of the surface burn wound

In this section the pathological effects of the burn wound at the physiological level are described briefly. It is important to understand that a surface burn is not simply a matter of damage to an unimportant area of the body: not only is the skin itself an organ of vital importance, as has been explained, but extensive burn wounds cause such extreme disturbances that the whole physiological balance of the body may be upset and major internal organs such as the kidneys, the heart and the digestive system threatened.

Two specialists on burns open a major article with the words:

Patients with extensive deep burns suffer the greatest trauma to which the body can be exposed and have profound metabolic disturbances that persist far into the course [of treatment] (Birke & Liljedahl, 1971, p. 5).

These general metabolic effects are the primary initial causes of death, and present the immediate challenge for treatment.

Physiological changes in the shock phase

The initial phase following a burn wound is usually known as the 'shock phase'.⁷ Shock here refers to *hypovolemic*, or *oligaemic* shock, a physiological syndrome resulting from a reduction in the volume of blood. It should be distinguished from the profound mental and nervous effects

consequent upon severe injury or emotional disturbance, resulting from the release of hormones such as adrenaline and histamine into the blood stream which in turn affect the nervous system; such shock is sometimes known as *neurogenic* shock. Both kinds of shock may be present in the burn casualty.

Hypovolemic shock is due to the loss of body fluids by direct flow or by evaporation from the burned surface. In a severe burn, loss of water by evaporation alone may amount to four to five litres per day (Roe, Kinney & Blair, 1964). The reduction in the volume of circulating blood has profound effects on the heart and on the peripheral blood flow. Moncrief (1966) showed that in the immediate post-burn period, the cardiac output of experimentally scalded dogs was reduced to 43 per cent of the pre-burn level, and then further declined to 20 per cent of the pre-burn level, unless the volume of lost fluids was replaced. In order to increase the blood flow to the heart the peripheral blood vessels are constricted, greatly reducing the blood supply to other organs and muscles. Beyond critical levels, the reduced blood flow is insufficient to maintain the supply of oxygen to the heart muscle, and the heart fails. Cardiac arrest is followed within a few minutes by death.

Changes in the electrolyte composition of the body fluids

The site of the burn wound, where the tissue is destroyed, is surrounded by an area of partial injury. In the dead tissue, circulation of blood ceases but in the surrounding area there is increased permeability of the capillaries, leading to an increased flow of water, electrolytes, proteins and other constituents from the blood into the wound, from where they may be lost. The flow of liquids is indicated by seepage from the wound, swelling of the tissue (oedema) or the formation of blisters.

It is thus not only the volume but also the composition of fluids lost that is important since the salts, proteins and other substances which they contain are essential for the normal functioning of the body. Replacement fluids (see below) must contain not only large quantities of water but also balanced amounts of electrolytes, the exact amount and concentration being carefully judged in each individual case. Normally water in the body flows into the cells from the extracellular fluid by osmosis—the flow through a semi-permeable membrane from a weaker to a stronger solution. When water is lost from the body fluids, the concentration of the solution outside the cells becomes greater than that inside, and water flows out, leading to the death of the cells. Any fluid leaked from the wound will contain some sodium ions, and the kidneys remove some sodium too, unless the concentration rises beyond the level with which the kidneys can cope (hypernatraemia).⁸ Thus the burned person may suffer losses of sodium

⁸ Hypernatraemia may also indicate increased secretion of aldosterone, a substance secreted by the adrenal glands which causes retention of sodium ions and the loss of potassium. Aldosterone is normally broken down by the liver or excreted in the urine.

⁷ The many uses of the term shock make it unsuitable for scientific use. However, the term is still widely used clinically and so it is used here. Simeone (1963) reviews the history of the term and attempts to combine semantic clarity with physiological complexity.

ions from the tissues, even though there may be a higher concentration in the (reduced volume of) body fluids.

The balance of potassium ions is affected somewhat differently. Potassium is normally found in a higher concentration in the intracellular fluid rather than in the extracellular fluid. When the cells are destroyed by heat (or by disease), additional potassium ions are released into the body fluids. Thus there may be an initial rise in the concentration of potassium ions (hyperkalaemia) following a burn. If the kidneys continue to function normally they will remove some of this additional potassium so that, as the tissues are reconstituted, there may later arise a condition of hypokalaemia, due to a shortage of potassium ions. In the first day or two of treatment it is usually unnecessary to replace lost potassium ions (Phillips & Constable, undated), but careful monitoring of electrolyte concentrations in the body fluids remains necessary for some time to avoid shortages later. (Table 3.1 records one death from hyperkalaemia, and four from hypokalaemia.)

Changes in blood protein levels

The major components of the blood are plasma—a fluid which contains dissolved salts, proteins (such as albumins, globulins and fibrinogen) and other compounds—red blood corpuscles, containing the red iron-rich protein haemoglobin, and white corpuscles, whose principal function is to protect the body against the invasion of microorganisms.

Extensive deep burns may result in the loss of as much as 20 per cent of the red corpuscles (Liljedahl, 1967). The red blood cells are broken down, a process known as haemolysis, releasing the haemoglobin into the plasma (a condition known as haemoglobinaemia), from which it may be extracted and be excreted in the urine (a condition known as haemoglobinuria). Removal of red corpuscles from the circulating blood may also result from local thrombosis—the formation of a clot of blood which occludes a blood vessel—in and around the area of burned tissue. Damage to the capillaries in non-burned areas as a result of thrombosis has been demonstrated experimentally by Arturson (1961) and by Birke and his colleagues (1960).

In addition to the loss of blood proteins by direct flow from the wound and by haemolysis, there are other large fluctuations in the levels of blood proteins, which appear to be related to changes at the site of the wound and to the greatly increased metabolic needs of the burned person. Since it is difficult to supply sufficient additional energy from external sources, the body turns to metabolizing its own proteins. The result is a severe initial decrease in the blood protein levels, which in severe burns may be reduced to one-half of normal levels (Liljedahl, 1967). Subsequently, muscle tissue is metabolized, and the patient's weight declines rapidly although the blood protein levels return to normal.

Changes in the kidney function

The kidneys have two major functions. They play a major role in maintaining the water balance of the body. Secondly, they extract a range of waste products from the blood and pass them into the urine which is then excreted. The kidneys consist of large numbers of tubules. In the first part of each tubule, known as the glomerulus, water and waste products are filtered out of the circulating blood. In the last part of the tubule some water is reabsorbed into the blood while the waste products and the remaining water are passed into the bladder as urine. These processes are regulated by hormones, such as those produced by the adrenal glands adjacent to the kidneys.

The decrease in the volume of the circulating blood, due to loss from the burn wound, haemolysis and thrombosis, induces constriction of the blood vessels which further decreases the flow of blood through the kidneys. In the initial postburn phase, the flow of fluids through the kidneys may decrease by 30–50 per cent. The amount of fluid filtered by the glomeruli is reduced by 40–50 per cent, while the resorption of water in the tubules increases as the body attempts to maintain the fluid balance. The higher concentration of waste products may block the filtering action of the tubules, hindering the excretion of waste products into the urine.

The frequency of confirmed kidney damage resulting from larger burn wounds varies between 9 and 12 per cent in various studies (Liljedahl, 1967). Electrical and extensive pulmonary burns are particularly liable to produce physiological disturbances sufficient to result in renal damage (Birke, Liljedahl & Linderholm, 1958; Birke & Liljedahl, 1966). Kidney damage in patients with extensive burns greatly complicates the prognosis, even where an artificial kidney is available. Mortality in these cases is high (Cameron, 1969).

Damage to the digestive system

During the initial phase of the burn injury the muscle wall of the intestines may be paralysed, suspending peristalsis, the normal waves of alternate contraction and relaxation which cause the contents of the alimentary tract to be propelled forward. This may be followed by acute retention of food in the stomach. Changes in the gastro-intestinal tract may cause ulcers, indicated by vomiting of blood and the passage of dark, tarry-coloured stools, due to the presence of blood chemically altered by the intestinal juices (melaena). Gastric and duodenal ulcers arise, in some cases in the surface, and in others, as deep, acute ulcers known as Curling's ulcers. These ulcers may result in dangerous haemorrhages and perforations. The pathogenesis of such ulcers is not fully ascertained although the most widely accepted hypotheses include the effects of histamine, steroids, shock, gastric acid, microemboli and endotoxins as possible causa-

tive agents (Friesen, 1950; Moncrief, Switzer & Teplitz, 1964; O'Neill, Pruitt & Moncrief, 1968).

Even in later phases of the burn injury, particularly where chronic infections of the wound develop, difficulties in digestion may create problems in maintaining an adequate nutritional state.

Changes in the hormone balance

The physiological stress due to thermal injury stimulates the production of a number of hormones, particularly those produced by the adrenal glands (Birke *et al.*, 1958; Feller, 1962).

Adrenaline, also known as epinephrine, is released by the medulla of the adrenal glands and stimulates the heart, inhibits the movements of muscle wall of the intestines, relaxes the bronchioles of the lungs and constricts or dilates the blood vessels. Noradrenaline secretion is also increased; this hormone possesses the excitatory function of adrenaline but not the inhibitory functions. The production of corticosteroid hormones by the adrenal glands also increases.

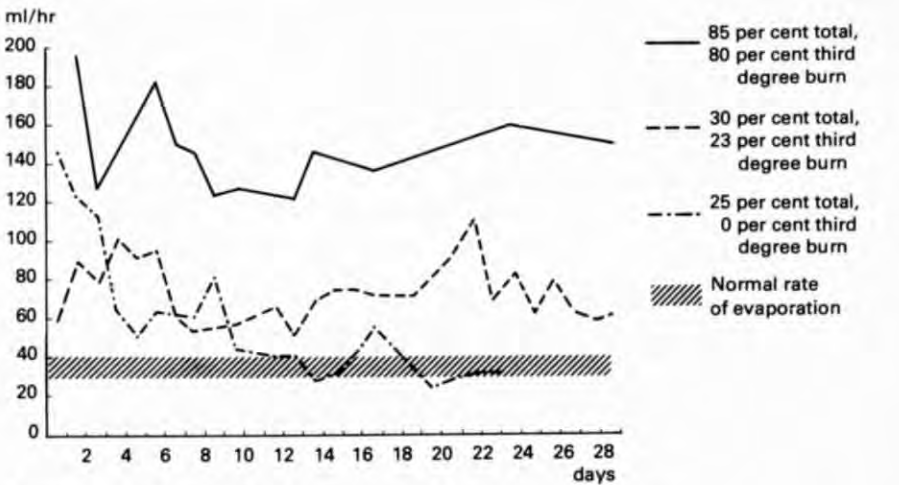
In some cases, however, the stress of thermal injury results in haemorrhage and necrosis of the adrenal glands, which in turn leads to a lack of adrenal hormones sufficient to cause death (Foley, Pruitt, Myers & Moncrief, 1967).

In the first hours after the wound occurs there is a large increase in the concentration of histamine in the blood. Histamine is a hormone which is liberated as a result of injury. It causes itching or pain, dilation of the blood vessels, reddening of the skin, lowered blood pressure, increased gastric secretion and may result in shock-like manifestations. The liberation of large quantities of histamine into the blood may be a possible contributing factor to gastric ulcers and to the general capillary damage evidenced in extensive burns (Liljedahl, 1967). Heparin has been used experimentally in the treatment of burns because of its strong anti-histamine effect. Heparin appears to decrease the size of the burn, shorten the healing time, and significantly relieve the pain (Saliba, 1970; Saliba & Griner, 1970), although its use cannot be prolonged since it interferes with the blood-clotting mechanism.

The problem of heat loss

One of the major functions of the skin is to prevent the evaporation of the body fluids and the loss of heat from the body. Apart from the threat of hypovolemic shock due to loss of fluids, the evaporation of each litre of water requires 580 kilocalories of heat (the latent heat of vaporization of water). Figure 3.5 shows that the rate of evaporation of water for varying areas of burn wound may amount to many litres per day, compared with the rate from the unburned body surface. In an attempt to maintain the normal temperature of the body, the severely burned person uses up large amounts

Figure 3.5. Rate of evaporation of water in three patients with 85, 30 and 25 per cent burn wounds compared with normal values, during first four weeks after injury



Source: Liljedahl (1971).

of energy. Some calculations of typical energy requirements are given in table 3.7. It will be seen that the severely burned person may use up to twice as much energy as normal just to compensate for the loss of heat by evaporation of water.

The increase in the basal metabolic rate as a result of burn wounds was reported by Cope, Nathanson, Rourke & Wilson (1943). Birke *et al.* (1958) showed that the basal metabolic rate may increase by as much as 100 per cent several weeks after the injury. This increased rate of metabolism

Table 3.7. Average daily rate of evaporation of water from burn wounds of varying extent and corresponding heat loss due to this evaporation, compared with the rate of water evaporation from a normal adult male

Area of third degree burn per cent	Loss of water by evaporation litres/day	Heat lost due to evaporation ^a kilocalories/day
20-40	1-2	580-1 160
40-60	3-4	1 740-2 320
60-90	5-7	2 900-4 060
Normal 70 kg male	approx. 0.85	approx. 490
Normal 70 kg male	basal heat production	approx. 1 700 kilocal./day
	moderate activity	approx. 2 700 kilocal./day

^a The latent heat of vaporization of water is 580 kilocalories per litre.

Source: Liljedahl (1971).

presents a major problem in treatment, since the severely burned person, who frequently develops complications in the digestive tract, has great difficulty assimilating sufficient quantities of nutrients to provide so much energy.

Where only simple medical facilities are available, it is a common experience that the severely burned person in fact uses up energy faster than he is able to replace it from food and as a result loses weight rapidly. Gestewitz (1968), for example, reports that for burn patients in North Viet-Nam, weight losses may be as much as 1 kg per day, and finally average a loss of 15 per cent of the normal body weight. This remarkable weight loss gave rise to the expression "napalm intoxication" and is very difficult to cope with except in the most advanced treatment centres.⁹ Roe *et al.* (1964) showed that slowing down the rate of evaporation decreased the heat loss and lowered the rate of metabolism; this finding led to the method reported by Liljedahl (1967) whereby warm, dry air is pumped into the room in which the patient is treated. This increases the rate of evaporation temporarily, but the wound rapidly dries, and after two days the rate of evaporation, and hence the increased rate of basal metabolism, declines considerably.

Pathophysiology of "pulmonary burns"

In addition to the surface burns described above, fire may cause death or injury by various forms of damage to the lungs and respiratory passages. These injuries are often referred to as "pulmonary burns". Such "burns" have now been recognized as one of the principal causes of death by fire (cf. Phillips & Cope, 1962; Shook, MacMillan & Altemeier, 1968).

It was at first assumed that the victim of pulmonary burns actually suffered burns of the respiratory tract and the lungs, due to the inhalation of flames and hot air. It is now realized that the term is something of a misnomer; thermal injury of the lower respiratory tract is rare, since the flames do not pass beyond the nose and throat, and the heat of the air is rapidly dissipated before reaching the lungs. Only where steam is inhaled does thermal damage to the lungs possibly occur, since steam has a thermal capacity 4000 times greater than that of dry air (Moritz, Henriques & McLean, 1945).

However, there is no doubt that pulmonary complications are a significant cause of death. In many cases, pulmonary complications arise as a direct sequel to burns of the face, nose, mouth and throat, or the inhala-

⁹ Horn (1969) gives a vivid account of such a problem in the People's Republic of China: "At a time when he most needed nourishment, the patient's appetite started to flag . . . When this news leaked out the chefs of Shanghai's famous restaurants put their heads together . . . and sent a stream of delicacies to the hospital . . ." (p. 108). Ironically, the answer is not tempting dishes but the intravenous administration of an emulsion of soybean oil and egg yolk (see below, page 169).

Table 3.8. Major cause of death in 38 patients who died as a result of inhalation injury^a

Autopsy finding	Number of patients who died
Pneumonia	15
Laryngotracheobronchitis	8
Burn wound sepsis	6
Respiratory tract burn	3
Pulmonary oedema	2
Atelectasis	1
Myocardial infarction	1
Cardiac tamponade	1
Gastrointestinal haemorrhage	1
Total	38

^a The data are gathered from the hospital records of the 2297 patients treated for thermal injuries at the US Army Institute for Surgical Research between 1956 and 1968. Sixty-six patients were identified with inhalation injury, and of these 38 died.

Source: DiVicenti, Pruitt & Reckler (1971).

tion of large amounts of smoke or fumes, particularly in a closed space. While the majority of such injuries occur in confined spaces, one study of 66 patients showed that eight patients sustained smoke inhalation injury from accidents that occurred in open areas (DiVicenti, Pruitt & Reckler, 1971). Mortality in these cases is very high. Of the 66 patients mentioned above, 38 died, a mortality of 57.6 per cent. This may be compared with an overall mortality of 9.6 per cent for thermal injuries treated at the same institution in the last year of the study. The major causes of death in these patients are shown in table 3.8.

However, pulmonary complications frequently accompany burn injuries even where immediate signs of inhalation injury are not obvious. A 20-year survey at the Massachusetts General Hospital showed that 88 per cent of burn patients developed respiratory complications (Phillips & Cope, 1962).¹⁰

Pulmonary complications take a number of forms, the most important of which are inhalation injuries due to smoke and toxic fumes, pneumonia and pulmonary oedema.

Inhalation injury resulting from smoke and toxic products of combustion is characterized by severe inflammation of the trachea and bronchi due to the chemical irritation of the mucous membrane lining these respiratory passages. This inflammation may lead to severe breathing difficulties. Clinical indications include the presence of carbon particles in the sputum, wheezing, hoarseness and coughing. Striking changes may be visible on a chest X-ray. The patient with inhalation injury is particularly susceptible to bacterial infection of the tracheobronchial tree (Pruitt *et al.*, 1970).

¹⁰ This institution did not use mafenide acetate cream, which may lead to additional respiratory complications. See page 176.

Such injuries are similar to those caused by the type of chemical warfare agent known as the lung irritant (see chapter 4).

Pulmonary oedema most commonly occurs in patients with large burns, especially children and older patients, patients with associated renal failure, and patients to whom inappropriate amounts of fluid were administered at the time of operations performed in their postburn period (Pruitt *et al.*, 1970). Mortality in these cases is high, although it may be accounted for by the large average size of the burns and other associated factors. A potentially fatal lung oedema may occur with considerable rapidity, even quite late in the recovery period (Walder *et al.*, 1967). This in turn may lead to a weakening of the heart and decreased blood circulation. Continuous monitoring of blood pressure and blood volume is required so that, if necessary, early measures can be taken to prevent decreased blood pressure leading to irreversible tissue damage of the heart and other organs.

Pneumonia is a disease of the lungs resulting from infection by bacteria. The burn wound itself acts as a medium for the culture of bacteria which may subsequently infect the lungs either through the blood or through the air. In one study of 70 burned patients who developed pneumonia it was found that 23 (33 per cent) were infected by airborne bacteria, while 47 (67 per cent) were infected through the blood. In a subsequent analysis of 113 patients who were treated with an antibiotic agent it was found that 74 (65 per cent) developed pneumonia from airborne bacteria and 39 (35 per cent) from bacteria in the blood. Nine (13 per cent) of the patients in the first group, and 25 (22 per cent) in the second group, died (Pruitt, DiVicenti, Mason, Foley & Flemma, 1970).

The problem of infection

In spite of many recent advances in medicine, the prevention and treatment of infection—entering the body through wounds from whatever cause—remains a problem of great magnitude. Where advanced medical facilities are available, enabling successful control of the hypovolemic shock phase, infection is generally the major cause of death following burns.

The deep burn is an ideal site for infection (Artz, 1964). The moisture and warmth of the body provide a near perfect medium for the growth of a range of bacteria, which find abundant nutritive material in the dead tissue and multiply rapidly. Thrombosis and oedema in the tissue decrease the flow of blood and thereby inhibit the patient's natural defence mechanisms against bacterial invasion. For these reasons, all deep burns become infected to a greater or lesser degree. A large burn wound may contain thousands of grams of highly infected tissue, and organisms from this tissue may enter the blood stream or be breathed in by the patient, causing generalized infection, or septicaemia. When infection reaches this stage



"The severity of a cutaneous burn wound depends not only upon the depth of the burn but also upon the extent of the body surface area affected, which determines the loss of body fluids and heat, and therefore the impact on the major physiological systems of the body." (p. 126)

"There is a direct relationship between the extent of a burn of a given depth and the chance of survival." (p. 128 ff)

"While there is little doubt that war burns have a high immediate mortality, in many cases death may not ensue for days or even weeks." (p. 134)



"The severely burned person, who frequently develops complications in the digestive tract, has great difficulty assimilating sufficient quantities of nutrients (p. 142) ... Since it is difficult to supply sufficient additional energy from external sources, the body turns to metabolizing its own proteins ... and the patient's weight declines rapidly ...' (p. 138)



"There is no doubt that pulmonary complications are a significant cause of death. In many cases, pulmonary complications arise as a direct sequel to burns of the face, nose, mouth and throat, or the inhalation of large amounts of smoke or fumes, particularly in a closed space ... However, pulmonary complications frequently accompany burn injuries even where immediate signs of inhalation injury are not obvious." (p. 142 ff)

"Where there are signs of upper respiratory tract obstruction, large amounts of liquid in the respiratory passages or shallow breathing, tracheotomy—the operation of making an opening directly in the trachea ('windpipe') so that oxygen may be administered by tube—may be required. This should only be done where it is clearly warranted by the observed symptoms, since tracheotomy may be itself a factor in pulmonary infection." (p. 171)





"Burns from napalm most typically occur on the unclothed areas of the body, particularly the hands, head and face, and feet and legs. Because of the thin layer of tissue over these areas napalm burns rapidly affect the underlying muscles, tendons and bones which greatly complicates the surgical problem. Reconstructive surgery to treat such conditions requires a long series of operations over several years and is unlikely to be available to ordinary people in most societies in wartime conditions." (p. 154)



"Hypertrophic scar is ... thickened, raised and grotesque ... Keloids ... may progressively extend beyond the site of the original trauma ... Skin cancer is much more prone to develop in scars caused by burns than in normal skin." (p. 146ff)

"All full thickness burns heal with some degree of scar ..." (p. 146)





"Contractures sufficient to cause serious functional disability frequently occur in the case of deep burn wounds of the hands and the head and neck . . . Burns in these locations are particularly common in the military context." (p. 147)

the chances of survival are not great. According to Moncrief & Teplitz (1964) over 80 per cent of late deaths following burns result from septicaemia.

Large burn wounds are particularly prone to infection because of the extent of necrotic tissue. Infection of clean mechanical wounds, for example those resulting from low velocity bullets, is relatively easy to contain. The bullet itself, or pieces of clothing or dirt carried into the wound, may infect it, but the area affected is generally restricted to the immediate vicinity of the bullet path. Dum-dum bullets and very high velocity bullets, on the other hand, cause an "explosive-type" wound, where tissue is damaged at a considerable distance from the point of contact with the projectile itself; and this type is much more liable to become infected due to the much greater volume of necrotic tissue acting as a culture medium for infective bacteria. Given that in general there is a direct relationship between the size of an open wound and the amounts of infective bacteria which grow, it is obvious that there are very large quantitative increases in the amounts of infection resulting from the entry wound of a bullet, which is less than one square centimetre in area, the exit wound of a high velocity bullet, which may be many square centimetres in area, and a burn wound which may, potentially, cover the total body area of approximately 1.5–2.0 square metres.

The nature of bacterial infection is highly dependent upon the circumstances and geographical location of the patient. In heavily populated and highly cultivated areas the likelihood of infection from grit and dirt in wounds is greater, a major factor to consider in combat conditions. A World War II study of the bacterial infection of comparable wounds showed that only 30 per cent of wounds incurred in the Libyan desert were infected with *Clostridium welchii*, whereas in France 80 per cent were so infected (McLennan, cited in Cope, 1953, p. 15).

The development and utilization of new antimicrobial agents during the past 30 years have resulted in changes in the types of organisms responsible for fatal infection of burn patients. Streptococcal and staphylococcal septicaemia predominated prior to the availability of antibacterial agents effective against these organisms. Since the late 1950s other organisms, predominantly *Pseudomonas aeruginosa*, have emerged as the principal offenders (Pruitt & Curreri, 1971).

Thus, the exact nature of the bacterial infection depends very much upon the circumstances in which a particular patient finds himself, including the type of treatment available. Whatever the cause of infection, however, burn patients tend to be particularly susceptible. This is due to the other effects of the burn on the body, including an initial loss of circulating antibodies and decreased ability of white blood corpuscles to kill bacteria, representing a decline in all of the body's normal defence mechanisms against infection (Liljedahl, 1967).

Disabilities resulting from burns

Severe burn wounds usually result in disability even after the wound has healed.

The disability following the healing of the burn is due to a number of factors. The most common ones are disabilities due to scar formation, to the development of keloids, to contractures in general, to lowered resistance to other diseases, to psychic trauma, to the development of unwanted emotional complexes, to inability to assume former roles in society, and the loss of strength and activity (Aldrich, 1943, p. 581).

Scarring occurs when the burn involves the full thickness of the skin and underlying tissues. All full thickness burns heal with some degree of scar. Skin grafts, if used, never perfectly match the adjacent skin and are surrounded with a rim of scar, especially if grafting is delayed or if infection prevents prompt healing of the burn. In a large burn these areas of scar are very extensive and conspicuous.

This scarred tissue may develop a number of abnormalities. Hypertrophic scar is defined as "a pink, white, or telangiectatic¹¹ scar which is hard, taut, unyielding, and more or less fixed to deep tissues" (Wells & Tsukifuji, 1952, p. 130). Although they may be thickened, raised and grotesque, they do not normally extend into undamaged skin, and if tension is reduced by releasing contracture, or if adequate skin grafts are applied, over the course of time they generally soften and become much thinner. Keloid is a term introduced by Alibert in 1806 and has been defined as "a movable, superficial lesion, presenting an exuberant, lobulated, overhanging profile, a glistening, inflamed or acrocyanotic¹² surface, a rubbery consistency, and characterized symptomatically by a prickly, burning itch" (*Ibid.*). Keloids are cumulative masses of scar tissue that may progressively extend beyond the site of the original trauma; characteristically, if keloids are excised, a larger keloid results. There is therefore an important surgical distinction between these two types of scar, although "difficulties in clinical interpretation can arise when one approaches the other in appearance or when the two extremes coexist in the same patient" (*Ibid.*). In addition there may be scarring which presents a raised or irregular surface, or a thickness greater than the surrounding normal skin, which is somewhat different again from keloid or hypertrophic scarring as defined above.

It is generally agreed that the incidence of such abnormal scarring depends upon the severity of the initial injury, the inadequacy of early treatment, the incidence of infection, and delays in healing due to mal-

nutrition or absence of skin-grafting and other factors (Block & Tsuzuki, 1948; Wells & Tsukifuji, 1952). Such factors may explain the high incidence of keloids following the incendiary and atomic bomb attacks on Japan, although some authors suggest that racial characteristics may also be involved.

A secondary factor is the known disposition for keloid formation to occur among the Japanese and other dark-skinned people as a racial characteristic. Many spectacular keloids, for example, were formed after the healing of burns produced in the incendiary bomb attacks on Tokyo (US Department of Defense, 1962, p. 570).

Keloids, which are extremely rare in the patient populations typical of most of the present-day medical literature, have been noted in Vietnamese burn victims by Liljedahl (1967) and Constable (1973).

Wells & Tsukifuji (1952) noted a tendency for abnormal scars to disappear over a period of years unless provoked by infection, foreign bodies, such as retained fragments, contractures or abnormal skin tension.

Contractures are due to the restriction of underlying muscles and joints by superimposed scar or inadequate grafts. Contracture bands of fibroblastic cells are formed in the granulation tissue that builds up in the burned areas. These contracture bands may be very dense and can produce many untoward results. Contractures sufficient to cause serious functional disability frequently occur in the case of deep burn wounds of the hands and the head and neck, where they may cause hideous deformity. Burns in these locations are particularly common in the military context (table 3.9).

Disabilities such as contractures and keloids may not develop until weeks or months after the wound has healed.

If such disabilities interfere with the movements of the body, it is impossible for the individual to assume all of his former functions and duties. Frequently, contractures can occur around orifices of the body preventing or interfering with natural functions such as defecation, urination, sexual intercourse and even eating. Late disabilities are prone to produce a lack of social adjustment, thus having a direct bearing on the emotional stability of the patient (Aldrich, 1943, p. 582).

Such complications demand a long series of reconstructive surgical operations coupled with physio- and occupational therapy in order to produce a functional result. This process, spread over many months or even years, is both painful and stressing to the patient. It places great demands on hospital facilities. As a result adequate treatment may only be available to those able to call upon considerable economic resources and is least likely to be available to those most prone to such complications.

Further, it is a well established fact that skin cancer is much more liable to develop in scars caused by burns than in normal skin (Bang, 1925; Johnson, 1926; Treves & Pack, 1930; Lawrence, 1952). There is evidence that this is even more likely to occur where burn wounds have not been

¹¹ *Telangiectasis* (from the Greek *telos*, end; *aggeion*, vessel; *ektasis*, extension) is the dilation of groups of capillaries, forming raised, dark red, wart-like growths; *acrocyanotic* (from the Greek *akron*, extremity; *kyonosis*, dark blue colour) means discoloured blue or dark purple at the finger tips or other extremities, due to the presence of abnormal amounts of reduced haemoglobin in the blood.

Table 3.10. Location of military burn wounds compared with locations of wounds from penetrating and blast weapons: US forces in Viet-Nam, February–November 1967^a

Wounding agent	Wound location (per cent)						Number of wounds per agent	Number of patients
	Head and neck	Thorax	Abdomen	Upper extremities	Lower extremities	Genitalia		
Burns ^b	23.2	13.3	7.1	36.1	7.6	7.6	224	118
Small arms	10.2	12.8	9.8	25.8	38.6	2.6	1 093	882
Mortar	16.3	15.6	6.9	25.6	32.1	3.3	1 728	1 170
Mine	17.7	10.0	6.8	26.3	35.8	3.2	1 060	660
Punji stake ^c	0.0	0.0	1.0	9.0	85.0	3.0	65	65
Native ^d	11.8	11.8	3.0	34.0	36.0	3.0	68	44
Other ^e	27.5	19.4	5.5	26.4	35.6	5.6	1 339	1 014
All wounds	15.7	12.2	7.1	26.3	34.8	3.9	5 577	3 953
Normal per cent body surface	9.0	18.0	18.0	18.0	36.0	1.0	–	–

^a The 3 954 patients in this survey were wounded in a total of 5 577 body regions, that is, an average of 1.4 per patient. The data do not include *killed in action*, because this category is not brought to hospitals.

^b Of the 118 burns patients, 68 (57.7 per cent) were victims of accidents rather than of hostile action.

^c A fire-hardened, sharpened bamboo stake, concealed in foliage, which readily penetrates combat boots and heavy clothing, inflicting lower extremities.

^d "Native" refers to injuries resulting from such weapons as arrows and spikes.

^e The large "other" category includes 398 non-battle injured patients; the remainder include patients wounded by "unspecified mechanisms such as knifings, phosphorus grenades, home-made bombs, blast injuries, blunt trauma, or otherwise unclassified type incidents"

Source: Kovacic, Aaby, Hamit & Hardaway (1969).

grafted at an early stage, when it may well be that, instead of healing, the wound becomes ulcerous for a period of months or years before becoming cancerous. Lawrence (1952) records 11 such cases, with periods of ulceration ranging from 4 months to 61 years. The latent period before the onset of cancer may be many years and is inversely correlated to the age of the person at the time of burn injury. For example, a person burned in childhood may suffer from a cancerous growth of the scar when he is 40 or 50 years old. This kind of complication is now rare where modern treatment facilities are available, but may be more frequent where such facilities are not available, for example in underdeveloped countries, or in industrialized countries following mass destruction.

Additional long-term pathophysiological problems result from the effects of shock and infection which may affect the kidneys, liver, heart and lungs, resulting in a permanent weakening of the individual's ability to resist disease, and facing the victim of serious burns with a further intermediate and long-term hazard. Renal complications, such as nephritis, may have a permanent influence on the health of the patient and his life expectancy. Prolonged infection which injures the heart may force the patient into a sedentary life.

Psychological effects of burns

The immediate psychic impact of the burn wound may be followed by intermediate and long-term psychological effects on the patient.

A number of factors contribute to a very severe psychological burden on the burned person (Hamburg, Artz, Reiss, Amspacher & Chambers, 1953; Hamburg, Hamburg & de Goza, 1953). The event itself, due to the relatively long time involved in inflicting a burn compared with, say, a bullet wound, is likely to have been particularly traumatic. Extensive second degree burns, where the nerve endings are bared but not destroyed, are excruciatingly painful, and may give rise to profound mental and physical disturbances.

The problems of combating pain and infection may themselves create psychiatric problems for the patient. Easing pain by the administration of analgesics such as morphine may create drug-dependency, and withdrawal from these drugs may cause additional pain and distress. Topical treatment with silver nitrate¹² to prevent infection causes the healthy skin to turn black; though this is temporary it may be an additional source of anxiety for the patient and relatives.

Particularly in the case of a child, the patient who is isolated and receiving only occasional visits by masked and robed hospital staff must also cope with resulting emotional problems.

With some trepidation we have permitted nurses on our Children's Burn Service to work unmasked. The psychological effect of masked attendants is undesirable, and thus far we have observed no evidence of a deleterious effect on the wounds by having the nurses unmasked (Phillips & Constable, undated).

The psychological problem of isolation is exacerbated by the more sophisticated treatment facilities and is most extreme where the patient is completely isolated and observed by television cameras and remote measuring instruments. The emotional strain of having to confront alone the sight of one's own naked and burned body lying on a bare plastic bed, and the stench of one's own rotting flesh, observed only by the cold eye of the television camera, is difficult for the uninitiated to envisage.¹³

In many underdeveloped countries it is common for the patient to be attended by his family, thereby gaining considerably in psychological reassurance and emotional support—factors which may be overlooked in advanced industrial societies. On the other hand, the limited methods of treatment available to the burn patient in most underdeveloped countries greatly diminish the prospects of recovery, thereby multiplying the attendant emotional strain that this implies.

The sum of the psychiatric effects upon the severely burned may be

¹² See page 176.

¹³ The emotional strain on the attending personnel is also enormous and even experienced medical personnel have been known to ask for transfers from burn units.

overwhelming. At best, the patient with extensive burns will be seriously scarred in spite of all efforts at reconstructive surgery. Many of these disfigured patients, even in cases where function has been restored, will psychologically withdraw and never make an adequate social readjustment.

It is an unfortunate fact that little can be done to ease the psychological problem since it is to a large degree a function of the emotional shock elicited among the social contacts of the patient by his severe disfigurement. Burned persons may find themselves socially stigmatized, as in the case of the survivors of the atomic bomb attacks on Japan, whose keloid deformities resulting from burns rather than radiation mark them as *hiba-kusha*, a name connoting disease, defect and disgrace (Lifton, 1967).

III. Casualty effects of incendiary weapons

In this section a description of the known effects of incendiary weapons is given. In this connection, it should be borne in mind that the specific effects of incendiary weapons depend upon the circumstances of use, the quantity and types of agent used, and factors in the target population such as age, general health and nutritional status, degree of protection, and medical facilities available.

Metal incendiary burns

The metal incendiaries are characterized by high burning temperatures and the tendency to splatter small particles of molten or burning metal on the victim. These small particles may cause small but deep burns. Such burns are rare in civilian life; they normally occur only among workers in munitions factories and among persons subjected to incendiary bombing (Artz & Moncrief, 1969).

Magnesium burns produce ulcers which are small at first but which gradually enlarge to form extensive lesions. According to Wilson & Egeberg (1942) the deeper part of the lesion is usually quite irregular. Tissue destruction may be increased by the formation of small bubbles of hydrogen in the wound (US Army, 1968). The outer layers of the skin must be scraped under local anaesthesia soon after injury in order to remove the magnesium. However, depending on the size of the particles, magnesium may form a rapid or a slow burning ember. If the larger slow-burning particles penetrate deeper than the outer layers of the skin they must be thoroughly excised surgically (Artz & Moncrief, 1969).

Burning thermite produces particles of iron which at the temperature of combustion are molten. Thus, while not actually burning, the drops of molten iron are capable of causing small, deep burns until they cool and solidify. These particles must then be surgically removed. In practice the

heat and flame from thermite close enough to shed drops of molten iron on a victim would probably have greater clinical effects, causing severe burns or death from hyperthermia.

Napalm burns

The thickened oil incendiary agents which are collectively grouped under the name "napalm" are intended to cause burns by means of direct adhesion of the burning agent to the body and by instigating combustion of clothes, houses, vegetation and so on. Burning hydrocarbon fuels are characterized by the production of thick, black smoke (carbon particles), considerable heat and a tendency to produce carbon monoxide unless there is very good ventilation. Thus the casualty effects of napalm are due to a variety of causes, the significance of a particular cause depending upon the circumstances of use, such as the quantity of the incendiary agent used, the amount of oxygen available, and so on.

These factors are illustrated in a pilot experiment by Moritz and his colleagues (Moritz, Henriques, Dutra & Weisiger, 1947). They concluded that exposure to burning hydrocarbon fuel in an enclosed space with good ventilation may lead to death within two minutes at 600°C and five to six minutes at 250°C. Death may be due to two mechanisms. First, systemic hyperthermia (heatstroke) results from the overheating of the blood during its circulation through the superficial network of subcutaneous blood vessels. Second, respiration is prevented by severe burning of the mouth and pharynx, followed by an obstructive oedema of the pharynx, trachea and lungs. Where there is bad ventilation, the hydrocarbon fuel will burn more slowly and may even go out. This results in a lower temperature than where there is good ventilation and there is therefore less likelihood of death from heatstroke. On the other hand, the bad ventilation increases the danger from carbon monoxide and other toxic fumes.

According to a US Army Field Manual, the casualty effects of napalm are as follows:

Personnel casualties result from a number of flame effects. First of course is contact with the burning fuel. Thickened fuel sticks to and burns hot on the target. It is difficult to extinguish, thus deterring the individual from accomplishing his combat mission. Casualties are also caused by the extreme heat (1200°F–1400°F). Inhalation of flame, hot vapours, carbon monoxide, and unburned hydrocarbons can also produce casualties. Personnel in a pillbox or other closed space will suffer due to the withdrawal of oxygen from the air. The shock effect of flame on the individual is great (US Army, 1960, pp. 3–4).

A Swedish handbook states:

In addition to an increase in the number of burn wounds, war conditions also produce an increase in the proportion of burn wounds with associated injuries (soft tissue wounds, fractures, etc.) and burns with a special aetiology. As an example of the latter can be mentioned the typically very deep wounds caused by

phosphorus and napalm. These agents also add the risk of systemic poisoning from white phosphorus and carbon monoxide, respectively (Arturson, 1966, p. 329; trans. from Swedish).

More specifically, the various effects of napalm may be summarized as follows:

1. *Heatstroke* (hyperthermia). Some evidence suggests that immediate death from napalm attack may be less frequent than has otherwise been supposed, since the short duration of the fireball (about 6–10 seconds) is too short to cause heatstroke. However, the air temperature close to the burning napalm rises to some 800–1200°C and persons exposed to this temperature for more than a very few minutes may die rapidly from heatstroke.

2. *Pulmonary burns*. Those close to the fire may suffer severe burning of the nose, mouth and pharynx within a short time as a result of breathing in the hot air and fumes. The physical damage to the tissues, combined with the oedema which this produces, obstructs respiration and may cause rapid death.

The dense black smoke from burning napalm and other incomplete products of combustion may cause *delayed* pulmonary oedema, even in persons not apparently burned by the immediate fireball or by direct contact of napalm on the body. As noted above, such pulmonary complications have come to be recognized as a major cause of death in peacetime experience, and very likely contribute at least as much to deaths from napalm attack.

3. *Carbon monoxide poisoning*. Hydrocarbon fuels, such as napalm, produce carbon monoxide when they burn. Where a victim is confined in a badly ventilated space containing carbon monoxide the proportion of carboxyhaemoglobin in the blood may reach a lethal concentration (66 per cent; see chapter 4). This circumstance is likely to occur relatively infrequently in the case of attack by napalm from the air, but relatively frequently where napalm is projected by flamethrowers into closed spaces such as pillboxes. The effects of carbon monoxide and other toxic substances are discussed further in chapter 4.

4. *Oxygen starvation*. Burning napalm uses up large quantities of oxygen, and in confined areas there is a possibility that the level of oxygen may sink below that necessary to support life (see chapter 4).

5. *Shock*. Shock due to the loss of body fluids is a function of the extent of the body surface area burned. According to Do Xuan Hop (1967), even when napalm burns do not exceed 10 per cent of the body surface area of the persons affected, shock conditions are nevertheless usual and serious. This may be due to the fact that hypovolemic shock is complicated by neurogenic shock as a result of the intense pain or fear, inhalation injuries and the toxic effects of carbon monoxide, benzene and phosphorus.

Gestewitz (1968) reports that 71.4 per cent of early deaths are due to irreversible shock or acute carbon monoxide poisoning.

6. *Burn wounds*. Napalm is spread in clumps of burning jelly, which may burn for up to some 10–15 minutes at temperatures exceeding 800°C, depending on the composition (see chapter 2). Beneath the clump of napalm, the temperature is relatively low, unless slag has been added to increase the downward heat flow. Unless the victim inadvertently spreads the napalm in an attempt to remove or extinguish it, napalm burns are typically round, with an area of inflammation and swelling around the burn and a less damaged area in the middle. According to Hashimoto (1971) it has been demonstrated that the tissues beneath the burned area remain above the normal temperature for five or six minutes after the fire is quenched. This causes further tissue damage and local thrombosis and necrosis spreads rapidly in the first hours and days, the wound becoming easily infected.

Because of the high burning temperature and extended burning time of thickened gasoline fuels, burn wounds resulting from them are typically deep and extensive. According to Do Xuan Hop (1967) and Hashimoto (1971) some 75 per cent of napalm burns extend into the subcutaneous tissue and involve muscles and bones in a further 10–15 per cent. The reports of Gestewitz (1968) and Hashimoto (1971) suggest that some two-thirds of the victims have total areas of burn of up to 25 per cent of the body surface area and one-third have burns of more than 25 per cent.

7. *Physiological complications*. The physiological complications of the burn injury as outlined above (pages 136–142) also occur in the case of napalm burns. The characteristic weight loss in the patient may be as much as 1 kg per day. In the Democratic Republic of Viet-Nam burn injuries have been reported as incurring weight losses averaging 15 per cent of normal weight (Gestewitz, 1968). This is due both to evaporation of water from the burned area and mobilization of the body's fat and proteins to compensate for the heat loss resulting from the evaporation. Such changes are likely to be particularly difficult to compensate for in patients who are initially undernourished and anaemic.

These metabolic changes are frequently complicated by injury to the kidneys and liver, gastric ulcers, decreases in the blood protein levels, disturbances of the hormone balance, psychiatric disturbances, and so on, as described above. Such developments are particularly likely where only limited medical facilities are available, as in many of the combat situations where napalm weapons have been used.

8. *Infection*. The area of dead tissue caused by the burn is easily infected. Sepsis of the wound may extend the depth of the necrotic tissue, converting a less serious burn into a full thickness injury. Due to the general decline in the patient's physiological and nutritional status as a result of the burn, infection is particularly liable to develop rapidly from local sepsis to generalised septicaemia, threatening the life of the patient. As noted

above, exceptional medical resources are required to prevent and treat burn wound sepsis, resources which are unlikely to be immediately available in an embattled area.

It may be concluded that the extensive tactical use of napalm in the Pacific theatre in World War II, in Korea, in Indo-China and elsewhere has resulted in a high mortality amongst the persons affected. Yet there is too little published information to enable an accurate assessment of the mortality to be made. Some authors have gone so far as to describe napalm as an "all-or-nothing" weapon (Dudley, Knight, McNeur & Rosengarten, 1968).

The common offensive agent is napalm (jellied petroleum) dropped from aircraft. However, this is an all-or-nothing weapon and just as it was not usual to be called upon to treat bayonet wounds in World War I or II (Taylor, 1953) it is rare to see napalm burns; in 3 months we did not encounter a single instance. (Dudley, Knight, McNeur & Rosengarten, 1968, p. 334)

Other authors, such as Do Xuan Hop (1967) and Gestewitz (1968), report on the basis of studies carried out in the Democratic Republic of Viet-Nam that 35 per cent of the persons affected died immediately, that is, within 15 to 20 minutes. (Unfortunately, neither author gives details as to the circumstances under which the victims were affected.)

Those who survive the initial infliction of serious burns are faced with a great variety of potentially fatal complications over a period of weeks or months. Do Xuan Hop (1967) reports that in addition to an average of 35 per cent immediate mortality, a further 21.8 per cent of victims die in hospital—a total of 56.8 per cent. After an investigation in the Democratic Republic of Viet-Nam, Gestewitz (1968) reported that 62 per cent of affected persons die before the wounds heal. Dreyfus (1971) says: "I do not have definitive statistics, but it seems that only about 30 per cent of those wounded by napalm and not killed outright can be saved" (p. 198).

The minority of victims whose lives are saved are faced with varying degrees of physical disability, characterized by ugly scars and contractions. Burns from napalm most typically occur on the unclothed areas of the body, particularly the hands, head and face, and feet and legs. Because of the thin layer of tissue over these areas, napalm burns rapidly affect the underlying muscles, tendons and bones which greatly complicates the surgical problem. Reconstructive surgery to treat such conditions requires a long series of operations over several years and is unlikely to be available to ordinary people in most societies in wartime conditions. Thus, in the Democratic Republic of Viet-Nam,

a limited number of gravely burned persons can be treated in a general hospital, especially those in Hanoi, but the majority of victims are treated in the village maternity infirmaries and the district hospitals where skin grafting is not possible. Instead of grafting, wounds are left to heal by slow skin extension from the wound periphery. (Dreyfus, 1971, p. 198)

In these conditions, a wound which might heal in two months where grafting is carried out might take a year or more to heal without grafting. A child whose entire scalp was an open wound one year after the burn took place was observed by Constable (personal communication) in a South Viet-Name hospital. Do Xuan Hop (1967) reported that with the treatment facilities available in North Viet-Nam at that time, 67.1 per cent of wounds in survivors healed within 3 months, 19.8 per cent between 3 and 6 months, and 13.1 per cent took longer than 6 months to heal.¹⁴

Dreyfus (1971) sums up the situation of the surviving casualty of napalm attack as follows:

Poor grafting also leaves serious after-effects. Retractable skin and contraction of scars form huge welts which will need further treatment. Keloid and hypertrophic scars will form to limit and inhibit the normal elasticity of the skin, which in turn inhibits the normal movements of the member. These scars are prone to pyodermic and microdermic infections. The new skin is extremely fragile, and sclero-atrophied skin will always be susceptible to minor infections that a normal skin would easily combat.

Lastly, concerning the medical effects of napalm recovery, there is the spectre of secondary cancers. Old burn scars show a frequency of skin cancer out of proportion to such appearance in normal skin. This cancer consists of spinocellular epithelioma with a negative prognosis because of the rapid invasion by the malignant cells of the related ganglion areas.

Though some of the victims may partially recuperate after long and costly treatment, nothing much can be done for the majority of napalm-burned persons. (Dreyfus, 1971, pp. 198-99)

Compared with single penetrating weapons such as low velocity bullets, napalm must be regarded as an exceptionally cruel weapon. There is also fragmentary evidence that a higher proportion of casualties die from napalm burns compared with projectile injuries, that the advent of death is more prolonged, and that rehabilitation of the survivor is more difficult. If this is so, then napalm may also be regarded as more "effective" from a military point of view. Since no comparative study has appeared in the public literature any judgement as to whether the excessive cruelty can be justified by the claim of military necessity is likely to be subjective.

White phosphorus burns

White phosphorus has been used extensively as a smoke, incendiary and anti-personnel weapon in small arms ammunition, grenades, artillery shells, rockets, bombs, and antipersonnel mines.

¹⁴ Of 3977 admissions due to burns and scalds (all causes) amongst British troops during World War I, 76.87 per cent were hospitalized for less than 3 months, 6.61 per cent for between 3 and 6 months, and 1.97 per cent for more than 6 months (Mitchell & Smith, 1931). It seems likely that the figures reported by Do Xuan Hop reflect both the depth of napalm burns compared with many other burns, and the limited medical facilities available in the Democratic Republic of Viet-Nam, particularly for long-term surgical treatment.

White phosphorus burns have certain characteristics. Because the particles of white phosphorus are usually distributed by an explosive charge, these particles may penetrate deeply into the skin. Secondly, the phosphorus may continue to burn for hours, and in some cases days, until it is neutralized by some means. For these reasons, phosphorus burns tend to be made up of small, deep lesions.

During World War II there were several reports of deaths from the toxic action of white phosphorus contained in tracer bullets (Rabinowitch, 1943; Cope, 1953), which made up 2 per cent of all missile wounds (C. G. Rob, in Porritt, 1953). Porritt (1953) reported:

Wounds due to incendiary or tracer bullets need special management because the phosphorus they contain causes chemical destruction of the tissues, early and progressive shock, and a risk of fatal hepatic and renal damage if not removed early (Porritt, 1953, p. 29).

The 30-lb incendiary bombs dropped by the British on Germany contained white phosphorus, and Bauer (cited in Bond, 1946) reported that "not only laymen but also doctors called every burn a phosphorus burn". However, Bauer states that "no cases have been reported in which organic damage resulting from the absorption of phosphorus through the skin could be proved".

Thirty years ago any toxicity of phosphorus in burns would probably not have been observed since severely burned patients usually died. Since World War II, the treatment of burns has improved considerably, with the result that the death rate in the best modern conditions has decreased. With this decline in mortality the severity of white phosphorus burns compared with wounds from other burning substances has become apparent from a number of recent studies. Because of its importance the question of the toxicity of white phosphorus weapons is discussed in depth in chapter 4, while this section is restricted to a description of the burn wounds resulting from these weapons.

Many authors distinguish between thermal burns, due to heat and flame, and chemical burns, due to the action of a chemical substance on the skin. This terminology is ambiguous, since the production of heat and flame is the result of a chemical reaction. Nevertheless, for clinical purposes it is useful to make the distinction since, in general, flames must be extinguished by the exclusion of oxygen, whereas chemical substances must be neutralized or diluted.

In the case of white phosphorus burns initial injury is largely "thermal" rather than "chemical", but elemental phosphorus, or phosphorus compounds produced in the wound, may be responsible for subsequent complications. For this reason, most authors classify white phosphorus burns as chemical rather than purely thermal (for example, Curreri, Asch & Pruitt, 1970).

The principal difference between thermal and chemical burns, including white phosphorus burns, is the length of time during which tissue destruction continues, since the chemical agent continues to cause damage until inactivated by reaction with the tissue, neutralizing agents or dilution with water (Curreri, Asch & Pruitt, 1970). White phosphorus tends to burn its way through the skin and, even days after the original injury, spontaneously igniting particles may be found deep in the wound (Jones, Peters & Gasior, 1968). That is, white phosphorus burns are usually of second or third degree. These burns are most frequently localized on the arm, hand, thigh, lower leg and head. Because phosphorus is lipid soluble it spreads rapidly through the fatty tissue underlying the dermis (Summerlin, Walder & Moncrief, 1967) and the phosphorus-derived acids produced in the wound may cause further tissue damage (Rabinowitch, 1943).

The most frequent complications are contractures of the joints with resultant functional loss. Injuries to the head lead to a significantly high incidence of eye complications. Other complications such as inflammation of the cartilage of the ears, gangrene of the fingers and pneumonia can be observed in 10 per cent or more of the patients (Curreri *et al.*, 1970).

The estimation of burn depth by clinical observation following chemical injury is difficult. The severe full thickness chemical burn may appear deceptively superficial with only a greyish-brown discolouration of intact skin during the first few days (Curreri *et al.*, 1970).

Surveying the surgical literature resulting from World War II, Cope (1953) reported cases of wounds caused by explosive bullets which deposited phosphorus in deeper tissues. Healing of these wounds was slower than normal (Cope, 1953, p. 308).

During World War II phosphorus was used in bombs as well as in bullets and shells. Cope (1953) records the case of two children injured while playing with an unexploded phosphorus bomb:

The two children, aged 7 and 8 were accidentally burned while playing with a phosphorus bomb on July 27, 1943 . . . Child A had extensive second degree burns on the backs of the legs and thighs and a patch of full thickness destruction on the back of the left thigh about 4 in. square. Child B was more severely burned and had lost the skin from the greater part of the back of the right leg from the gluteal region to the ankle and on the left side from the gluteal region of the knee. She also had splash burns on the face and right hand and arm . . . The rate of healing was slow in both cases and multiple grafting operations were done but it was not until December of the same year that epithelialisation was complete. The scars which arose from these burns were in both cases keloidal and unstable. Active contraction took place necessitating re-excision and Thiersch grafting in one case and X-ray therapy in the other (Rooksdown House Centre Hospital account, cited in Cope, 1953, p. 310).

The longer time required to heal phosphorus burns was reported by Obermer (1943) and Sinilo (1961). This has been recently confirmed by Currier *et al.* (1970), who analysed 111 cases of white phosphorus and other

chemical burn patients admitted to the US Army Institute of Surgical Research during the period from 1950 to 1968.

The mean total body surface area of the burns was 19.5 per cent as compared with 28.5 per cent for all burn patients admitted during the same period, although the mean area of third degree (full thickness) burns was 10.5 per cent for both groups. The lower total burn area in the chemical burns was reflected in a lower mortality: 5.4 per cent compared with 19.3 per cent for the non-chemical burn admissions. However, longer hospitalization was required for complete skin healing of the chemical burns than for the overall group: an average of 104 hospital days compared with 74, or an increase of 40.5 per cent.

An average of 5.8 surgical operations per patient was required, 78 per cent involving homo- or autografting of third degree burn sites. Table 3.10 shows the 227 complications that were recognized in the 111 patients, the most frequent being contractures of joints (52 cases), and complications of the eye and eyelids (36 cases). Table 3.11 shows the distribution of the contractures which resulted.

Systemic complications such as pneumonia (10 cases), upper gastrointestinal bleeding (seven cases), septicaemia (five cases), acute renal failure (four cases), jaundice (three cases) and urinary tract infections (three cases) further complicated treatment. Systemic complications resulting from phosphorus burns may be due to hypovolemic shock, or to the toxic effects of phosphorus absorbed in the body fluids through the burn wound, to toxic effects of substances, such as copper sulphate, used to treat the wound, or to a combination of these factors.

White phosphorus burns from exploding munitions are often associated with other injuries such as lacerations, fragment wounds, traumatic amputation of one or more digits, and fractures of the long bones. Table 3.12 shows the distribution of such associated injuries in the US Army study.

In some cases particles of phosphorus may penetrate deep into soft tissue or into the chest or abdominal cavities. There is usually a need for radical debridement because of the depth of the penetration and it is extremely difficult to remove all the particles. US Army recommendations in Viet-Nam state that re-debridement should be planned after 6-24 hours, by which time the continued burning of previously undetected particles will have created more clearly circumscribed areas of burned tissue, enabling the particles to be located more readily (Whelan, Burkhalter & Gomez, 1968).

There is no doubt that burns due to white phosphorus weapons are extremely serious. Not only do they share the characteristics of thermal burns but the wounds take longer to heal. By continuing to burn in the wound, phosphorus presents serious problems of treatment and results in deep wounds which may require extensive surgical excision. In addition, the known toxicity of white phosphorus is a hazard which may result in complications such as kidney and liver damage, although published

Table 3.10. Classification of 227 complications following white phosphorus and other chemical burns in a total of 111 patients^a

Complication	Number of complications	Per cent
Contracture of joints ^b	52 ^b	22.9 ^b
Ectropion	18	7.9
Chondritis of ears	17	7.5
Gangrene, one or more fingers	17	7.5
Cataract	11	4.8
Pneumonia	10	4.4
Ankylosis, one or more inter-phalangeal joints	7	3.0
Upper gastrointestinal bleeding	7	3.0
Corneal ulcer	7	3.0
Cellulitis or lymphangitis	6	2.6
Cutaneous abscess	6	2.6
Septicaemia	5	2.2
Acute renal failure	4	1.8
Infected fragment wounds	4	1.8
Osteomyelitis	3	1.3
Jaundice	3	1.3
Urinary tract infection	3	1.3
Others	47	20.7
Total	227	100.0

^a Ninety-six of the 111 patients had wounds caused by white phosphorus, 5 by concentrated sulphuric acid, 3 by lye, 3 by mustard gas and 4 by other causes. Sixty-four of the injuries were sustained in the Republic of Viet-Nam. Fifty-nine of the 111 cases were due to hostile action, and 52 to accidental causes (of which 36 involved exploding phosphorus grenades, shells, bombs or booby traps).

^b See table 2.11. below.

Source: Curreri, Asch & Pruitt (1970).

Table 3.11. Contractures resulting in functional disability as a result of white phosphorus and other chemical burns^a

Location	Number of cases
Hand	11
Fingers	10
Neck	9
Elbow	7
Axilla	5
Wrist	3
Mouth	3
Ankle	2
Knee	1
Perineum	1
Total	52

^a See footnote a, table 3.10 for explanation.

Source: Curreri, Asch & Pruitt (1970).

Table 3.12. Mechanical injuries associated with white phosphorus and other chemical burns in a total of 111 patients^a

Type of injury	Number of injuries
Multiple lacerations or superficial fragment wounds	17
Amputation of one or more fingers	11
Fracture, open	7
Fracture, closed	5
Major nerve or artery injury	5
Penetrating wound of the cornea	4
Penetrating wound of the abdomen	3
Perforation of tympanic membrane (unilateral or bilateral)	3
Penetrating wound of the chest	2
Loss of teeth	2
Total	59

^a See footnote a, table 3.10 for explanation.

Source: Curreri, Asch & Pruitt (1970).

research does not indicate clearly the extent to which this hazard is a major factor in battlefield casualties. The question is further examined in chapter 4.

Thermal radiation burns from nuclear weapons

No account of thermal effects of modern weapons would be complete without reference to the thermal effects of nuclear weapons.

Depending upon the nature of the weapon, the fraction of the nuclear explosion yield emitted as thermal energy ranges from 30 to 40 per cent at an altitude below 30 000 metres. The distribution of energy may be roughly estimated as in table 3.13. This thermal radiation is emitted as a pulse of extremely high energy of short duration. For a 1-kiloton¹⁵ air burst, the effective duration of the thermal pulse is 0.3 seconds. For a 10-megaton air burst it may be as long as 30 seconds. Roughly speaking, assuming approximately one-third of the energy is emitted as thermal radiation, it may be estimated that for every kiloton exploded some 3.3×10^{11} calories are released in the form of thermal radiation (US Department of Defense, 1962).

It has been estimated that on the ground immediately below the Hiroshima bomb explosion, which had an effect of some 12.5 kilotons and took place at a height of 560 metres, solid materials were heated to about 3 000–4 000°C, and at 1 200 metres away temperatures exceeded 1 600°C. However, the thermal pulse from a nuclear explosion is so short that while some porous materials such as lightweight fabrics, paper and dry grass may ignite, wood may only char but not ignite. Surface temperatures are

¹⁵ A kiloton is the nuclear equivalent of 1 000 tons of TNT high explosive; a megaton is the equivalent of one million tons of TNT.

Table 3.13. Distribution of total energy released by a typical nuclear explosion in air at an altitude below 30 000 metres

Source	Per cent energy
Shock and blast	50
Thermal radiation	35
Initial ionizing radiation	5
Residual ionizing radiation	10

Source: US Department of Defense (1962).

highly dependent on the reflective properties of the material: light coloured material reflects the heat rather than absorbing it, whereas dark material absorbs the heat.

Nuclear explosions may cause burns both directly and indirectly:

One of the most serious consequences of the thermal radiation from a nuclear explosion is the production of "flash burns" resulting from the absorption of radiant energy by the skin of exposed individuals. In addition, because of the focussing action of the lens of the eye, thermal radiation can cause permanent damage to the eyes of persons who happen to be looking directly at the burst¹⁶ (US Department of Defense, 1962, p. 328).

It is estimated that 20–30 per cent of the fatal casualties in Hiroshima and Nagasaki were caused by flash burns.

In spite of the thousands of cases experienced in the Japanese explosions only the general features of flash burns have been described. These observations have since been supplemented with experimental studies using the skin of pigs which has many similar characteristics to human skin (Pearse, Payne & Hogg, 1949). In addition to being largely limited to exposed areas of the body, flash burns generally show a smaller depth of penetration than do flame burns because of the short exposure time (less than one second in the Japanese explosions). Severity of flash burns in Japan ranged from mild erythema (reddening) to charring of the outermost layers of the skin. Healing of burns in survivors was hampered by inadequate care, poor sanitation and general lack of proper facilities resulting from the general widespread destruction.

In addition to the primary effects of thermal radiation, nuclear weapons may ignite fires in two ways. First, thermal radiation may ignite combustible materials, typically paper, rubbish, curtains and dried grass, which in turn ignite more solid fuels such as wood, plastics and bitumen. Second, the blast wave can upset stoves and furnaces, cause electrical short-circuits and break gas mains. The outbreak of such secondary fires is diffi-

¹⁶ A recent report of a group of experts convened by the International Committee of the Red Cross points to the fact that laser weapons, which are at present being developed (Meyer-Arendt, 1968), may have similar effects on the eyes (ICRC, 1973a). An experimental study of flash-blindness is described by Chisum (1968).

Table 3.14. Comparative mortality due to heat, blast and ionizing radiation in first-day survivors in Hiroshima

	Casualties by major cause of injury			
	Heat	Blast	Radiation	Total
Number of survivors on first day	41 992	45 356	37 657	125 005
Per cent of total number of first-day survivors	33.6	36.3	30.1	100.0
Number of deaths on subsequent days	9 776	3 475	5 649	18 900
Per cent of all deaths	51.7	18.4	29.9	100.0

Source: Oughterson & Warren (1956).

cult to predict since it is dependent on the density of ignition points, humidity, wind and other prevailing conditions.

The secondary incendiary effects from a nuclear attack do not differ significantly from those due to a massive use of conventional incendiary and high explosive bombs.¹⁷

The incendiary effects of a nuclear explosion do not present any especially characteristic features. In principle, the same overall result as regards destruction by fire and blast might be achieved by the use of conventional incendiary and high-explosive bombs. It has been estimated, for example, that the fire damage to buildings and other structures suffered at Hiroshima could have been produced by about 1 000 tons of incendiary bombs distributed over the city. It can be seen, however, that since this damage was caused by a single nuclear bomb of only 20 kilotons energy yield, nuclear weapons are capable of causing tremendous destruction by fire, as well as by blast (US Department of Defense, 1962, p. 345).

According to Oughterson and Warren (1956), 45 000 people died on the first day at Hiroshima, while 22 000 died at Nagasaki:

Burns and blast effects were responsible for most of the effects on the first day, but since the effects were interrelated, it is impossible to know what proportion of deaths was due to each of them. However, all observers are agreed that heat was the major cause of death on the first day (Oughterson & Warren, 1956, p. 95).

According to the US Department of Defense (1962) about two-thirds of the casualties at Hiroshima and Nagasaki who died during the first 24 hours suffered from burns the effects of which were often augmented by other injuries, and there were many deaths from burns during the first week. It is estimated that roughly 50 per cent of all deaths were due to burns of one kind or another. Among the survivors after the first day, 65 per cent suffered from burns, 95 per cent of which were flash burns. The reason for the low proportion of flame burns among the survivors is that "most

¹⁷ It is estimated that 83 000 people died in the incendiary raids on Tokyo. However, rather little has been published on the effects of the incendiary raids on Japan, in contrast to the wealth of material on the atomic bomb attacks.

of those who suffered flame burns did not survive, since they were caught in burning buildings and could not escape" (US Department of Defense, 1962, p. 565).

In Hiroshima alone nearly 42 000 fairly serious burn cases were reported among the survivors of the first day. Of these, 23.3 per cent died subsequently, compared with 7.7 per cent of the 45 000 blast wounded survivors, and 15.0 per cent of the 38 000 ionizing radiation-affected survivors (table 3.14).

Thus there is good reason to regard the Hiroshima and Nagasaki bombs as largely, and perhaps even primarily, incendiary weapons, and only secondarily as blast and radiation weapons. Further, the exceptionally large casualty statistics which resulted indicate that incendiary weapons may be the cause of more serious injuries than either blast or radiation weapons. This conclusion is largely borne out by studies made of the casualty effects of the strategic bombing of German cities, described in the next section.

Mass casualties from incendiary attack

The air attacks on German cities during World War II led to the deaths of between 500 000 and 800 000 people.¹⁸ Several reports were prepared on the causes of death in these raids, during which the British Royal Air Force dropped 196 335 tons of incendiary bombs and 234 312 tons of high explosive bombs (Harris, 1947) and the US Army Air Force a further 80 000 tons of bombs (US Strategic Bombing Survey, 1945) on German population centres—a total of 510 747 tons, or approximately one ton for every fatality.

The Medical Branch of the Morale Division of the US Strategic Bombing Survey published a report in December 1945 on *The Effect of Bombing on Health and Medical Care in Germany* which included a chapter on the nature of air raid casualties by Captain Franz K. Bauer. Bauer concluded in part:

... It can be stated that there is a definite relationship between the type of bomb dropped, and the type of death or injury to be expected. An incendiary raid was expected to cause more dead than wounded, through the effects of heat and carbon monoxide; in bombings with high explosives, mechanical injuries outnumbered deaths.

... In all the cities visited carbon monoxide poisoning was regarded as the primary cause of death or injury, sometimes reaching to as much as 80 per cent of all incendiary raid casualties. Air blast was found to be a relatively infrequent cause of death and affected only people within a radius of 30 meters from the explosion of a bomb (Bauer, cited in Bond, 1946, p. 113).

Bauer's report contains considerable interesting detail. It became apparent to leading German pathologists that the air war was leading to

¹⁸ See chapter I.

Table 3.15. Most prominent causes of death as a result of the incendiary raids on Hamburg, July and August 1943

Causes of death from external injury

1. Burial under rubble and debris and injury from flying fragments
2. Secondary injuries through explosions (drowning, scalding, chemical burns, poisoning from the by-products of exploded bombs)
3. Burns
4. Tetanus secondary to burns where no serum was given prophylactically

Causes of death from internal injury

1. Carbon monoxide poisoning in air raid shelters and occurring during rescue work
2. Effect of heat through conduction and radiation in the presence of very high temperatures
3. Overheating over a prolonged period of time through temperatures which, normally, can be tolerated for short periods only
4. Dust inhalation; blocking of the upper respiratory passages and inhalation, with damage of the small bronchi and alveoli
5. Carbon monoxide poisoning from bursting gas mains
6. Sudden heart death through fright and exhaustion in cardiac patients
7. Blast injuries in which external injuries may be absent or which may be masked by external injuries

Source: Conclusions of a meeting of experts called by the inspector of sanitary and medical matters of the Luftwaffe held in Jüterborg, December 1943; reported by Captain Franz K. Bauer, US Army Medical Corps, "The nature of air raid casualties", in US Strategic Bombing Survey, Morale Division, Medical Branch, *The Effect of Bombing on Health and Medical Care in Germany* December 1945; cited by Bond (1946).

causes of death which were not at first obvious. In 1943 measures were taken to ensure correct diagnoses. All persons with unclear diagnoses were subjected to autopsy: between 20 000 and 30 000 autopsies were carried out during the war. Physicians from the Luftwaffe were appointed to supervise commissions whose task was to study the causes of death. Prior to the establishment of these commissions a meeting of experts was called in Jüterborg in December 1943 to discuss the effects of the raids on Hamburg in July and August 1943. Their conclusions as to the most prominent causes of death at that time are shown in table 3.16. Several theories regarding deaths from anoxia, "carbon dioxide poisoning", "gas poisoning" (that is, "phosphorus gas") or from high frequency waves subsequent to explosions were disproven.

Additional studies after 1943 led to more understanding of the causes of death. In addition to mechanical injuries, only heat and carbon monoxide proved to be of statistical importance. In large-scale fires, deaths resulting from heat and carbon monoxide poisoning outnumbered those from mechanical causes.

The effects of heat were of two kinds in large-scale incendiary attacks such as those on Hamburg:

- (1) The effect of direct heat of short duration through conduction or radiation with production of burns proper.
- (2) The effect of high temperatures over long periods of time which did not immediately lead to protein coagulation, but which caused a syndrome identical to that of heatstroke. (Bauer, cited by Bond, 1946, p. 115)

Heatstroke depends on a number of factors such as the humidity of the air, the cessation of sweat production and the amount of heat to which the body has been exposed. In humid air heatstroke may occur at a temperature of 60°C and is not necessarily associated with subjective complaints.

This accounts for the many bodies which were found dead in rooms from which escape would have been possible, and which were in a position not suggestive of agony before death occurred (Bauer, cited by Bond, 1946, p. 115).

According to Bauer, police engineers in Hamburg estimated that temperatures in the burning city blocks rose as high as 800°C.

Literally hundreds of people were seen leaving shelters after the heat became intense. They ran across the street and were seen to collapse very slowly like people who were thoroughly exhausted. They could not get up (Dr. Helmuth Baniecki of Hamburg) . . .

Most of these people were not burnt to ashes when recovered, but dry and shrunken, resembling mummies. In many the intense heat had caused the skin to burst and retract over typical areas such as the elbow, the knee, the scalp and the orbit. Baniecki thought that the cause of death in these cases was shock. In approximately 80 autopsies he found all organs shrunken, showing venous stasis with increased permeability of the small blood vessels. . . .

Professor Rose, chief consultant to the Air Ministry, summarized the effects of heat. Besides immediate contact with flames, he wrote, the effect of heat through hot air as well as radiation of hot gases and from objects is important. This accounts for the severe heat changes in women who do not wear more than stockings on their legs, or not even stockings. In many cases, when stockings were worn, they were not even singed, although the skin and underlying structures were severely damaged. Radiation heat of over 225°C can inflame clothes and [hair]. Besides this local effect of heat, overburdening of the heat-regulating mechanisms of the body is important . . .

During escape from overheated shelters through burning city blocks, the danger was chiefly from radiated heat. The inhalation of hot air can cause severe damage to the respiratory passages such as ulcerous necroses of the mucous membranes. Whether this is a separate entity or the changes a part of the whole picture which leads to death is as yet unsolved. It should be kept in mind that the inhalation of dangerous gases or by-products of fire must be considered . . .

The degree of temperatures produced in incendiary raids gave rise to a question from the office of Professor Karl Brandt, commissar for sanitary and health matters for Germany. Professor Shuetz, a physiologist of Muenster, answered from the Institute of Aviation Medicine: The question concerned is the effect on the human body of overheating to 41°C for eight hours. The answer is: in the tissues increased temperature up to 50°C is followed by death of all cells, with subsequent vacuolization; higher temperatures are followed by shrinking and falling apart of the cells. According to Ludwig Aschoff, human cells die at 50°C, vesicles form in the tissues at 51°C, and haemolysis occurs at 60°C. Ganglionic cells are destroyed at 43°C. Animals die exposed to temperatures of from 60°C to 100°C in overheated rooms, usually in convulsions, after a few minutes to half an hour (Bauer, in Bond, 1946, p. 116).

Bauer gives the following description of the victims of the Hamburg attacks on 27–28 July 1943, taken from an account of Professor Graeff,

consulting pathologist to the *Wehrkreis X* (military defence area) in Hamburg:

Many of the bodies were lying in the street half-clothed or nude. The only covering that they always had on was their shoes. The victims' hair was often burned, but preserved. A few hours after the start of the raid the corpses had a peculiar aspect; they seemed blown up, lying on their stomachs. The buttocks were enlarged and the male sex organs were swollen to the size of a child's head. Occasionally the skin was broken and indurated in many places and in the majority of cases was a waxen colour. The face was pale. This picture lasted only a few hours; after this time the bodies shrank to small objects, with a hard brownish black skin and charring of different parts and frequently to ashes and complete disappearance . . .

In the shelters bodies assumed various aspects corresponding to the circumstances under which death had set in. Nowhere were bodies found naked or without clothing as they were in the streets. The clothes, however, often showed burned out holes which exposed the skin. Bodies were frequently found lying in a thick, greasy black mass, which was without a doubt melted fat tissue. The fat coagulated on the floors as the temperature decreased. The head hair as a rule was unchanged or only slightly singed. The bodies were not bloated except for a few which were found floating in water which had seeped into the shelters from broken mains. All were shrunken so that the clothes appeared to be too large. Those bodies were *Bombenbrandschrumpfleichen* ("incendiary-bomb-shrunken bodies"). These were not always in one piece. Sleeves and trouser legs were frequently burned off and with them the limbs were burned to the bones. Frequently such bodies burned to a crisp weeks after death—apparently after oxygen had become available. In the same rooms with such bodies were found other more or less preserved or shrunken corpses and also some which had fallen to ashes and could hardly be recognised. Many basements contained only bits of ashes and in these cases the number of casualties could only be estimated (Bauer, in Bond, 1946, p. 119).

While Bauer makes no reference to death by asphyxiation due to the withdrawal of oxygen from the air, many survivors stated that the air "just didn't come anymore" and breathing became very difficult. In the shelters levels of oxygen fell so low that matches or candles would not burn, and people lay on the floor where, because of a higher concentration of oxygen, breathing was easier. Outside, in the city streets, the firestorm drew in a mass of fresh air with sufficient force to blow people to the ground.

According to Bauer, carbon monoxide as a major cause of death was a possibility which few had expected before the war. However, carbon monoxide deaths assumed such proportions that the high command of the Luftwaffe issued an order to examining commissions to procure statistical evidence of carbon monoxide poisoning. The question of carbon monoxide poisoning is examined in chapter 4.

IV. The medical treatment of war burns

In this section an outline of some of the problems of the medical treatment of victims of burns is presented. Such information is necessary for an adequate understanding of the humanitarian issues involved in the use of fire

as a weapon of war, since prospects for casualties caused by incendiary weapons are particularly dependent upon the quality of the medical treatment available to survivors.

The treatment procedures outlined below explain what can be done for the victim of burns with the best facilities currently known to medical science. Even with such facilities, the treatment of the severely burned is long, tedious and exceptionally painful—a severe emotional burden on the patient and his family, and indeed, in many cases, on the medical staff as well. The complexity of a severe burn wound is such that exceptional medical resources are required to give the patient a reasonable chance of recovering a sufficient degree of functional ability to be able to perform normally in society. Even where an adequate functional result is obtained, scars and disfigurement often present a psychological and social barrier to normal social life.

The nature of many recent conflicts has meant that the medical equipment and expertise available to the conflicting parties has been highly variable. Many of the facilities required to treat severe burns are not available or are destroyed in combat. Such conditions are tantamount to increasing the "effectiveness" of the weapons employed, since a greater proportion of the victims will die or be permanently disabled. To this end, efforts may be made to reduce the medical support available to an adversary. Suffering will thereby be considerably increased. Due to inadequate treatment by skin grafting and associated surgical measures, a third degree burn wound may never heal and the patient will have to bear a permanently open wound as a continual source of infection, subsequently liable to become cancerous. By contrast, a wound caused by a low-velocity bullet or fragment may in general be treated by relatively simple means and the prognosis for the patient is not so vitally dependent upon the quality of medical treatment available, except when his major organs are damaged.

Because of wide variations in the facilities available no precise course of treatment can be described. The following outline refers to the best treatment possible but it should be borne in mind that such facilities are not available to a high proportion of the victims of incendiary attack.

First aid

It may be assumed that some form of first aid is available to the majority of war wounded, although it may be the only treatment available. The major points to note in first aid are as follows:

1. Where possible, flames should be "suffocated" by excluding air, for example, by covering them with a thick cloth. Where napalm is burning on the skin, great care should be taken to exclude the air by covering it without spreading the sticky jelly. Once the flames are extinguished, simple napalm will not ignite spontaneously. However, it may contain white phosphorus in order to reignite it in air, or sodium to ignite it in water. Phosphorus

fragments should be bathed in water or covered with a wet cloth until they can be removed.

2. To protect the sore from infection and further trauma, it should preferably be covered with large, sterile, absorbent dressings or, failing that, the patient may be wrapped entirely in a sheet, blanket or coat. It is important to keep the patient warm.

3. Particularly if he is unconscious, the patient may have breathing difficulties. The respiratory channels may be blocked by blood, saliva, vomit or the tongue. It is easiest to open the air passages with the patient lying on his side with the neck bent backwards. If the patient does not breathe spontaneously, artificial respiration by the "mouth-to-mouth" method or, if available, the administration of oxygen, may be called for.

4. In the event of great pain, which may contribute to neurogenic shock, analgesics such as morphine may be slowly (over a period of one to two minutes) injected intravenously for adults. Codein may be given to children for the same purpose.

Hospital treatment of burns

The hospital treatment of burn wounds is a specialized field of considerable complexity, enabling a certain variation in treatment methods (e.g. Liljedahl, 1967; Phillips & Constable, undated; Pruitt & Curreri, 1971; NATO, 1958). A brief outline of basic therapeutic measures is given to indicate the extent of the requirements for the adequate treatment of burn wounds.

The initial therapeutic objectives are:

- (a) the prevention of shock,
- (b) the maintenance of adequate oxygen supply in the event of respiratory complications, and
- (c) the prevention of infection.

Subsequently attention may be given to the surgical treatment of the wound, to psychiatric care and finally to rehabilitation.

The prevention and treatment of shock

The first two to three days after the infliction of the burn injury normally bring fluid loss and oedema, leading to hypovolemic shock which is brought on by too little fluid in the body. Replacement or substitution of lost fluids is the obvious treatment.

The administration of fluid volume in shock should have as its goal: (1) an adequate blood volume based on the central venous pressure measurements; and, (2) a normal red cell mass, adequate protein and colloid, the replacement of deficiencies in extracellular fluid, the correction of electrolyte and pH abnormalities in the blood and supply of caloric requirements. No one fluid is adequate, but a combination of different fluids, tailor-made to keep a normal blood composition, is needed. Protein and other colloids are essential to maintain water in the blood and prevent edema. Whole blood colloids and electrolytes are all needed in proper amount and proportion. (Hardaway, 1969, p. 653)

Table 3.16. Typical replacement fluids for a severely burned adult during the first 24-hour period

Colloid (blood plasma or substitute)	0.5–1.5 ml × kg body weight × per cent body burned
Electrolyte (e.g. Ringer's lactate) solution	0.5–1.5 ml × kg body weight × per cent body burned
Metabolic requirement (5 per cent sugar solution)	1 000–2 000 ml

Sources: Phillips & Constable (undated); NATO (1958).

It is usual to administer fluids intravenously where burns exceed 10 per cent of a child's body surface area or 15 per cent of an adult's. These fluids have three main components. To compensate for the fluids lost from the wound, replacement fluids must contain electrolytes, especially sodium ions, and plasma or a colloidal solution made up of plasma proteins or a plasma substitute such as dextran.¹⁹ The most preferred electrolyte solution is Ringer's lactate,²⁰ a solution of electrolytes resembling the electrolyte composition of the blood. Finally, because of the difficulties of administering food by mouth in sufficient quantities (due to the danger of the food getting into the lungs and causing serious complications), solutions of sugar (for example, dextrose) and fat emulsions²¹ must be administered intravenously.

Typical replacement fluids for an adult during the first 24-hour period are shown in table 3.16. These proportions are calculated on the basis of body weight in kilogrammes. Because the ratio of body surface area to body weight is different in children, Phillips & Constable (undated) recommend a formula based on the area of the body surface of the child. For each square metre of the body surface area of the child, whether burned or unburned, they suggest 90 ml plasma and 10 ml saline per percentage of the burn.²² In addition 1 500 ml water (by mouth) or 1 500 ml of a 5 per cent concentration of dextrose (intravenously) should be given for each square metre of the body surface area, whether burned or not.

In general an adult requires about 150 ml of electrolyte and colloid solu-

¹⁹ Dextran is a water-soluble, high molecular weight (average 75 000) glucose polymer produced by the action of *Leuconostoc mesenteroides* on sucrose. It is used as a 6 per cent solution in sodium chloride in the treatment of shock.

²⁰ Ringer's lactate solution is made up of 5.7–6.3 per cent sodium chloride, 2.9–3.3 per cent sodium lactate, 0.27–0.33 per cent potassium chloride and 0.18–0.22 per cent calcium chloride.

²¹ Fat emulsions were introduced by Wretling (Schubert & Wretling, 1961; Wretling, 1962). A recent composition tried successfully by the US Army Brooke Medical Center contains soybean oil, egg yolk, phosphatides and glycerol, and provides one Calorie/ml. Single 500 ml units of the 10 per cent soybean emulsion were administered to patients over a 4-hour period through a forearm vein (Wilmore, Moylan & Pruitt, 1972).

²² For example, a nine-year-old child, with a body surface area of 1.0 m² and a 30 per cent burn, would receive 1.0 × 30 × 90 = 2 700 ml plasma and 1.0 × 30 × 10 = 300 ml saline in the first 24 hours.

tion per percentage of the burn. Phillips & Constable (undated) recommend a ratio of five parts colloid to one part electrolyte, but the ratio depends upon the individual needs of the patient, the method of treatment and the availability of plasma or plasma substitutes. Due to shortages of plasma in the United States as a result of the war in Viet-Nam, many patients have had to be treated entirely with electrolyte solution (Ringer's lactate). Where electrolyte solution alone is used, no formula can be given and the amount is determined by urinary output and venous pressure as measured with a central venous catheter.

In many cases fluid therapy can be reduced after the first 24 hours, usually reducing the electrolyte and colloid solutions to one-half of the initial amount. After the first few days the infusion of sugar solution or fat emulsion for basal metabolism is required. Careful observation and measurement is necessary to compensate for individual differences. The most valuable indication of an adequate rate of intravenous infusion is the urinary output, which should be maintained at 30–50 ml per hour in adults and 15–25 ml per hour in children. A catheter is inserted into the bladder in order to monitor the flow of urine accurately every hour. Where the flow of urine is inadequate a 12.5 per cent solution of mannitol is sometimes recommended in order to initiate or maintain diuresis and prevent renal damage associated with haemoglobinuria.

After the initial shock phase, lasting two to three days and characterised by loss of fluid and oedema, the burn wounds, if they do not become infected, dry out and the oedema begins to subside. This is known as the resorption phase and lasts from approximately the third to the eighth day.

Moderate burns usually require only three days of intravenous infusion of fluids. For serious burns, infusions may be reduced successively, but the quantity and composition of the fluids may require considerable variation, depending on the tendency to lung oedema, pneumonia and sepsis. A new shock phase may occur due to heart damage or a combination of heart damage and fluid deficiency. Digitalis²³ may be required where there are indications of heart failure. Urine flow and blood potassium and sodium levels require continual monitoring.

Maintenance of respiration

Maintenance of the individual's ability to absorb oxygen is of primary importance. Administration of 95 per cent warmed and humidified oxygen, by mask or nasal tube, may be indicated if:

(a) there are breathing difficulties (dyspnoea), a bluish or purple colouration of the skin and mucous membrane, indicating oxygen deficiency (cyanosis), or sounds in the chest detectable with a stethoscope (rales);

(b) there are second and/or third degree flame burns about the nose or mouth;

(c) there is evidence that the patient was overcome by smoke or is restless, confused or panicky;

(d) the patient has a cherry-red colour or was close to a smouldering fire when rescued, indicating the possibility of carbon monoxide intoxication;

(e) there is a history of heart disease; or

(f) the burn is spread over more than 50 per cent of the body area (Phillips & Constable, undated).

Where the patient is breathing well on reaching the hospital, oxygen may not be necessary even where there are burns around the nose and mouth or some possibility of limited carbon monoxide poisoning. The best current measure of the patient's need of oxygen is the oxygen and carbon dioxide concentration in the blood, which can be monitored by methods which are available in most large hospitals. There is some danger of oxygen toxicity from excessive or prolonged use at high concentrations.

Where there are signs of upper respiratory tract obstruction, large amounts of liquid in the respiratory passages or shallow breathing, tracheotomy—the operation of making an opening directly in the “wind-pipe” so that oxygen can be administered by tube—may be required. This should only be done when it is clearly warranted by the observed symptoms, since tracheotomy may itself be a factor in pulmonary infection (Foley, Moncrief & Mason, 1968). In circumferential deep burns of the chest it may on rare occasions be necessary to incise burn eschar to permit adequate chest expansion.

Treatment of white phosphorus burns

Irrigation with a plentiful supply of water, immersion in water or placing water-soaked pads on the burn surface stops the combustion of phosphorus instantaneously (Norberg & Rosenqvist, 1944). Often a combination of these methods must be used, since if the surface is allowed to dry the phosphorus reignites. Although warm water is sometimes recommended it may be dangerous since it can liquefy phosphorus particles (Mendelson in Curreri *et al.*, 1970) and speed up absorption making phosphorus penetrate deeper into the tissue. Cold water makes phosphorus solidify, relieves some of the severe pain, and slows the blood circulation beneath and around the wound, thus minimizing oedema and resulting tissue damage. Water also dilutes acids formed by phosphorus combustion. Physiologic saline or weak sodium bicarbonate solution is a better choice but seldom available in large quantities in an emergency.

The standard means of initial treatment has been to irrigate the phosphorus wound with a solution of copper sulphate, usually at 5 per cent

²³ Digitalis is a powerful heart stimulant obtained from the dried leaves of *Digitalis purpurea* (foxglove) plants.

strength (cf. NATO, 1958; *Voennyi Vestnik* 48, May 1968, pp. 95-101), which reacts with the outer layer of the phosphorus particles to form a dark coating of copper phosphide which excludes air, thereby preventing further combustion of the phosphorus and helping to identify the particles.

In practice, this treatment has been disappointing (see below); moreover, copper sulphate is itself toxic and can be absorbed from the wound (Summerlin, Walder, & Moncrief, 1967). Whelan, Burkhalter & Gomez (1968) report that this treatment unequivocally caused the death of at least one soldier with a 10 per cent burn. This man collapsed with renal failure after treatment with copper sulphate for suspected phosphorus burns—a suspicion which turned out to be unfounded. Five of seven patients treated at renal units at the Clark Air Base in the Philippines and the Third Field Hospital in Saigon died; the two survivors had not been treated with copper sulphate at any time.

Since copper sulphate poisoning may be very similar to phosphorus poisoning, the possibility arises that symptoms attributed to phosphorus in fact arise from the copper sulphate used in treatment. Accordingly this question is further examined in chapter 4.

Since other methods are available for detecting and inactivating phosphorus, many recent writers conclude that there now seems to be no reason to place the burden of copper toxicity on patients already threatened with phosphorus toxicity and other stresses of the burn wound (Whelan *et al.*, 1968). Copper sulphate is not very adequate as a means of excluding air and preventing combustion (Norberg & Rosenqvist, 1944; Ben-Hur, Galidi, Appelbaum & Neuman, 1972). Norberg & Rosenqvist (1944) detected the luminosity of phosphorus in the wound four hours after vigorous surgical treatment with scalpels and forceps, combined with the application of pads soaked in 2 per cent copper sulphate, or 22 per cent copper sulphate dissolved in glycerine. Copper sulphate was no more effective than irrigation with water (Norberg & Rosenqvist, 1944; Curreri *et al.*, 1970).

To inactivate any residual phosphorus resistant to mechanical treatment, oxidizing agents such as hydrogen peroxide have been suggested (Summerlin *et al.*, 1967). Animal experiments have been conducted which show that a 1 per cent solution of potassium permanganate in a 5 per cent sodium bicarbonate solution neutralizes the phosphorus and removes all nonoxidized elemental phosphorus (Norberg & Rosenqvist, 1944). Residual phosphorus can be detected by means of fluorescence caused by ultraviolet light irradiation (Frye & Cucuel, 1969). Whelan *et al.* (1968) report that lithium iodate-isopropanol is also being investigated as a possible means of treatment.

Other debridement compositions have been proposed for treatment of burns caused by various liquefied white phosphorus mixtures. These mixtures are considered to be more troublesome than solid white phosphorus to remove from the wound surface. The compositions recommended are

suspensions of copper sulphate, various oils and organic solvents, or solutions of glycerine, copper sulphate, starch and detergents (Rabinowitch, 1943; Godding & Notton, 1942). The use of copper sulphate and organic solvents must be considered unwise because of the deleterious effects of copper salts and the risk of speeding up absorption of white phosphorus with the use of lipid solvents.

In the face of certain technical possibilities for "improving" white phosphorus incendiary agents, which would make proper debridement more time-consuming, the aim of the treatment must be fast removal of phosphorus from the wound. This must be done with non-toxic solutions which do not interfere with other surgical wounds or cause systemic poisoning or further damage to the tissues.

Animal experiments by Ben-Hur *et al.* (1972) (see also chapter 4) showed that, within the experimental conditions, a solution of 5 per cent sodium bicarbonate and 1 per cent hydroxy-ethyl-cellulose in 5 per cent copper sulphate solution prevents both systemic phosphorus and copper poisoning.

Early mortality in severe chemical burns is usually related to hypotension and acute tubular necrosis as a result of underestimation of the burn area and the depth of tissue necrosis (Curreri *et al.*, 1970). This underestimation, in combination with the possibility of phosphorus absorption, makes fluid resuscitation and careful monitoring of urinary output necessary, with scrupulous attention to serum sodium concentration and serum osmolarity.

The following general treatment schedule is recommended by the authors cited:

1. Remove contaminated clothes and irrigate with large amounts of cold water, or immerse the affected parts of the body in cold water. Care must be taken to maintain normal body temperature.

2. All visible white phosphorus particles should be removed with the aid of scalpel, forceps and pads. Physiologic saline or sodium bicarbonate (5 per cent solution in water) should be used if available. This treatment is extremely painful and is best done under anaesthesia.

3. Intense mechanical treatment under irrigation or immersion with a 1 per cent potassium permanganate in 5 per cent sodium bicarbonate solution should be performed until luminosity or fluorescence ceases, indicating that all the white phosphorus has been neutralized.

4. Pads soaked with the permanganate solution should be left on the wound for some hours.

5. In case of ocular injury irrigation should consist of water or physiological saline for at least half an hour. No pads should be placed on the eye since it is important to maintain its mobility.

6. Further treatment continues as for thermal burns.

It must be emphasized that this treatment is to be carried out without delay if the risk of phosphorus poisoning is to be avoided. Thus, correct treatment in battlefield conditions demands substantial medical facilities.

The prevention and treatment of infection includes the following three measures: (a) the reduction of sources of infection by asepsis in the patient's environment, (b) the treatment of the burn wound itself to prevent infection (sepsis) of the wound, and (c) prophylactic and curative measures to prevent generalized infection of the body system (septicaemia) resulting from microorganisms and toxins in the circulating blood.

Asepsis. The need for complex and thorough asepsis is particularly important in the treatment of burn wounds. Measures include not only usual precautions of sterile clothing, scrubbing, masks, gloves, and so on for medical personnel, but preferably treatment of the patient in an isolated sterile chamber, well ventilated by filtered air.

A further means of reducing the threat of bacterial infection is ultraviolet irradiation. Usually this requires the provision of an ante-room, provided with ultraviolet lamps, through which all staff must pass before they reach the patient's isolation room. Considerable success has been reported in reducing infection by such means (Hart, 1936; Hart, Postlethwait, Brown, Smith & Johnson, 1968).

A noteworthy improvement in reducing infection was achieved by Colebrook and his colleagues at the Birmingham Accident Hospital, England, towards the end of World War II. Dressings were changed in special aseptic chambers devised by Bourdillon, ventilated by about 305 cubic metres per minute of warmed, twice-filtered air. This rapid air flow carried with it out of the chamber particles and bacteria liberated from the patient's dressings which might otherwise have contaminated the new dressings (Bourdillon & Colebrook, 1946). This approach has been developed in recent years. Some promising results have been obtained with a plastic ventilated isolator. The isolator is a large, transparent box-like construction in which the patient lies. The patient can thus both be readily observed and have the opportunity to see out, which may be psychologically beneficial in contrast to isolation rooms, where the patient is observed by television cameras but may suffer from lack of social contact. The isolator is equipped with glove ports and transfer pouches on both sides of the patient, making it possible to treat the patient but protect him from contamination by contact or from the air (Haynes & Hench, 1965; Lowbury, 1967; Levitan, Seidler, Strong & Herman, 1968).

Another method has been developed where patients are treated in isolated rooms through which filtered warm dry air is pumped continuously (Liljedahl, 1971). This air is warmed to 32–36°C and its humidity is controlled. Medical personnel must pass through an ultraviolet irradiation chamber before entering the patient's room, and the patient is mainly observed via television cameras and other remote means. In this method, not only is the patient protected from bacteria in the air, but the warm, dry

Table 3.17. Decline in mortality following treatment of burn wounds with mafenide acetate

Per cent of body burned	Mortality	
	Without topical therapy (1962–1963)	With topical therapy (1964–1970)
0–30	4.3	2.0
30–40	44.4	12.6
40–50	61.1	23.1
50–60	78.3	41.0
60–100	89.1	79.5

Source: Pruitt & Curreri (1971).

air rapidly dries out the open wound which forms a protective crust in a few hours. This has enabled an *open* method of treatment to be developed, in contrast to the traditional closed method of covering the patient with dressings. The patient lies on a special bed enabling him to be turned without being moved from the bed. The bed is covered with a sheet of aluminized fibre, which does not adhere to the wound and in addition provides a certain protection against infection. Use of this method avoids the continual changes of dressings which are a major source of excruciating pain to the patient. Very few of these patients develop *Pseudomonas* infection which has been a major clinical problem (Liljedahl, 1967, 1971; Coriell, Blake-more & McGarrity, 1968).

Medicinal treatment of the wound. There are two reasons why such measures as those above are not sufficient. First, even with the most elaborate facilities, some cases of infection occur. Second, facilities such as those described above are so elaborate and so costly that they are simply not available in most areas of the world, or are not available on a scale applicable to large numbers of battle casualties. The development of surface means of treatment, such as antibiotic creams which can be put on the wound, is therefore of considerable importance.

During World War I a local application of picric acid was used to treat burn wounds. Davidson (1925) introduced the use of tannic acid in Detroit, a method which remained common until World War II. Burn casualties at Pearl Harbour and at the Coconut Grove night club fire in Boston²⁴ were treated with intravenous injection of sulphonamides (Evans & Hoover 1943; Cope, 1943). Later, sulphonamide-penicillin and penicillin creams were used for local application, before applying a dressing.

More recently a group at the US Army Institute of Surgical Research

²⁴ On 20 November 1942, 491 people died in a fire at a Boston, Massachusetts nightclub. The Massachusetts General Hospital received 114 dead and injured. A classic series of papers on these cases was published in the *Annals of Surgery* 117 (6) 1943.

has developed an antibiotic cream, containing mafenide acetate, known as Sulphamylon, which has resulted in a considerable decrease in mortality (table 3.17) (Lindberg, Moncrief, Switzer, Order & Mills, 1965; Moncrief, Lindberg & Switzer, 1966).²⁵ This treatment is now widely used. Mafenide is a methylated sulpha compound which, in the form of the acetate salt, is suspended in a soluble base. This suspension is regarded as critically important in order to permit continued absorption into the eschar and maintenance of effective concentrations of the medicament at the level of the interface between the viable and the non-viable tissue—the characteristic site of bacterial proliferation (Pruitt & Curreri, 1971). Mafenide acetate cream is a bacteriostatic rather than a bactericide; that is, it does not sterilize the wound by destroying the bacteria but it prevents them spreading and infecting non-burned tissue (Moncrief *et al.*, 1966). Mafenide acetate cream is spread over the entire wound with a sterile tongue blade or a sterile gloved hand, following daily cleansing, wound debridement, physiotherapy and hydrotherapy carried out in a special tank (Hubbard tank) in the ward. Twelve hours later the cream may be reapplied to areas where it has been removed by contact with the bed clothes.

A number of side effects have been noted with the use of mafenide acetate cream. The cream causes some initial pain and discomfort to the patient. Hypersensitivity reactions occur in some 7 per cent of patients (Pruitt & Curreri, 1971). More serious is the development of respiratory complications due to inhibition of the enzyme carbonic anhydrase, which is responsible for the normal process of excretion of carbon dioxide from the blood into the air to be exhaled from the lungs.²⁶ Mafenide may also delay epithelialization.

A second means of topical treatment which has been widely used in recent years is the application of thick sterile dressings soaked in a solution of 0.5 per cent silver nitrate (Monafo & Moyer, 1965; Polk 1966; Polk, Monafo & Moyer, 1969). Hypersensitivity reactions have not been attributed to the use of this agent and no development of resistant organisms has been noted. It is most effective when applied within 24 hours of the injury, and is active against the entire spectrum of wound bacteria, although some cases of clostridial myositis have been noted (Monafo, Brentano & Gravens, 1966). On the other hand, silver nitrate does not penetrate as deeply as

mafenide and requires the application of thick occlusive dressings, and on contact it discolours not only the patient's unburned skin but bedclothes and attending personnel as well. A number of side effects have been reported, including deficits of electrolytes (sodium, potassium and calcium), methaemoglobinaemia (a form of blood poisoning), and a restriction of the movement of joints, due largely to the dressings (Turnberg & Luce, 1968; Polk, 1966).

The Chinese news agency Hsinhua reported on 8 August 1973 that the teaching hospital of the Nantung Medical College in eastern China's Kiangsu province has treated extensive burns with a herb, *Ilex chinensis sims* (a species of holly), for over three years with good effect. The herb had been used by practitioners of traditional Chinese medicine for many years. Topical treatment of the wound with a solution or cream containing the herb had an antibacterial effect, confirmed by bacterial cultures from 60 patients on 240 occasions, and had the advantage of rapid formation of crusts on the wounds, little exudation, slight infection, rapid healing and few complications. By preventing a large amount of exudation, fluid requirements were also reduced. In addition, the herb has the advantage of being abundant, inexpensive and easy to use.

Other recent topical treatments include gentamicin sulphate (Stone, 1966), sulphadiazine silver cream (Fox, Roppole & Stanford, 1969) and various silver creams (Butcher, Margraf & Gravens, 1969).

Burn wounds may also be infected by fungi, such as *Candida*, and by viruses, such as *Herpesvirus hominus*. Systemic antifungal agents have not been particularly effective in combating fungal infection and the infected area may have to be removed surgically. However, topical application of nystatin (Mycostatin) is being evaluated in the laboratory (Pruitt & Curreri 1971). Several patients have died due to *H. hominus* and post mortem examination has shown systemic lesions in the oesophagus, lung, gastrointestinal tract and liver. Since no adequate means of treatment is presently available it is fortunate that most viral burn wound infections do not spread in this way. Topical application of idoxuridine is being investigated as a therapeutic measure (Pruitt & Curreri, 1971).

General prophylactic measures. In addition to direct treatment of the wound, measures to prevent general bodily infection by bacteria and their toxins may be required. Three general approaches are available. The first is to replace the deficit in the body's natural means of defence by administration of replacements of gammaglobulin. The second approach is the administration of antibiotic drugs such as penicillin. The third approach is the administration of a vaccine.

Liljedahl (1967) recommended that the prophylactic administration of 20–40 ml of a 12 per cent solution of human gammaglobulin be administered between the third and the eighth to tenth day after the burn injury. No definite clinical confirmation of its effectiveness had been re-

²⁵ Arthur D. Mason, Jr., and Robert B. Lindberg won the 1972 US Army Research and Development Achievement Award for their discovery and development of Sulphamylon cream (*US Army Medical Department Newsletter* 4 (1), 1972).

²⁶ "Alveolar-arterial carbon dioxide gradients of significance have been measured in patients with extensive burns who have elevated serum levels of mafenide acetate and its primary breakdown product *p*-carboxybenzene sulfonamide. In such a situation supervening pulmonary complications, further interfering with carbon dioxide excretion, may cause rapid change from respiratory alkalosis to acidosis. The mafenide acetate cream should then be removed from the burn wound and buffering carried out as necessary" (Pruitt & Curreri, 1971, p. 464).

corded, although Birke *et al.* (1964) previously demonstrated the considerable initial loss of gammaglobulin and a rate of breakdown five to six times the normal rate in burn-injured patients. In the four years of consistent treatment with gammaglobulin, mortality due to infection decreased considerably, although other factors were also involved.

As in many aspects of the treatment of burns there is some variation in medical opinion regarding the use of antibiotics. Many authorities agree on the value of prophylactic penicillin doses of from 300 000 to one million units a day for all patients with significant burns (Phillips & Constable, undated). This applies particularly to patients with pulmonary complications. However, antibiotics are no substitute for meticulous wound care. In particular there is a danger that general antibiotic treatment may change the bacterial flora of the wound so that if septicaemia develops later it will be from an organism resistant to normal antibiotics. There is a trend away from prophylactic antibiotic treatment in many burn units (Liljedahl, 1967), and the reservation of antibiotics for the treatment of infections due to specific, identified organisms is recommended (Phillips & Constable, undated).

Alexander & Fisher (1970) report the development of an antigen vaccine against *Pseudomonas* bacteria, a major cause of death from burns.

Skin grafting

The first stage in the surgical treatment of the burn wound is the removal of dead tissue. In some cases dead tissue may be removed by scrubbing (Litvine, 1970) or washing with a jet of water (*US Army Research and Development News*, August 1972). In other cases surgical excision is required. While some authors (Jackson *et al.*, 1960) advocate early excisions, this procedure is perhaps to be preferred only in the case of clearly delineated deep burns covering less than 5 per cent of the body (Bäckdahl, Liljedahl & Troell, 1962). Other authors recommend excision after 20–25 days, when it can be carried out with considerably less loss of blood (Liljedahl, 1967).

The ultimate objective of all burn wound care is to replace the lost skin as soon as possible. Although the skin may regenerate itself, in a full thickness burn it does so only at the edges of the wound, where the old skin remains intact. In large third degree burns, the rate of regrowth of the skin may never be sufficient to cover the wound. For this reason it is necessary to resort to surgical measures to replace the skin, following cleansing and preparation of the wound.

In order to cover the large burn wound, pieces of skin are transplanted from other areas of the body, a procedure known as autografting. The most common technique is to remove "split-thickness" skin from a donor site with a special instrument. The split-thickness graft is a thin layer of skin

which includes the epidermis and part of the dermis, but leaves a sufficient depth of dermis to enable rapid degeneration. During this period the donor site is an additional source of pain and discomfort, and may itself become infected. For this reason surgeons working in primitive or combat conditions may prefer not to transplant skin, even though they may be technically competent to do so.

Functional and cosmetic considerations dictate priority to areas such as the hands, feet, joints and face, which should be covered before nonfunctional surfaces. In patients with extensive burns and limited donor sites, "meshed" autografts may be used (Tanner, Vandeput & Olley, 1964; Stone & Hobby, 1965). In this technique the skin is perforated by a series of small cuts so that it can be drawn out into a "lace" or "net" up to nine times the original area. The interstices of this mesh regenerate rapidly. However, the resulting skin is not cosmetically satisfactory and is thinner than normal; thus the technique is not recommended for the face or for joints, feet and other areas exposed to constant wear (Pruitt & Curreri, 1971).

It may take several months of skin-grafting operations to cover an extensive burn, since the patient will have little remaining skin to donate to the burned area. In such cases successive "crops" of skin must be transplanted from the same healthy areas. Because of this problem, several other materials have been used for temporarily covering the burn, including skin from other persons or animals, and synthetic materials.

The first clinical use of animal skin grafts was described in the seventeenth century and the transfer of temporary skin grafts from one person to another was first recorded in the nineteenth century (Davis, 1910). Skin grafts from other persons may be taken from cadavers, amputated parts or living volunteers, in that order of preference (Pruitt & Curreri, 1971). Such pieces of skin may be treated and kept for a short time under refrigeration. They will serve as acceptable dressings for up to two weeks.

Less satisfactory but also acceptable are grafts from dogs (Switzer, Moncrief & Mills, 1966) and pigs (Bramberg, Song & Mohn, 1965). Pig skin grafts treated by electron-beam irradiation may be stored under refrigeration until needed. These grafts are more difficult to apply and show less adhesiveness. They may have the advantage of being more readily available, at least in the case of pig skin, which can now be obtained commercially. Vishnevskii (1966) reports that frog skin has been used in the same way in the Democratic Republic of Viet-Nam.

A synthetic polymer burn covering has recently been described by Gregory, Schwoppe & Wise (1973).

The common uses of such temporary "physiological dressings" are given by Pruitt & Curreri (1971) as follows: (a) debridement of untidy wounds, (b) protection and "stimulation" of granulation tissue following eschar separation, (c) immediate coverage of excised burn wounds, (d)

coverage of eschar-free burns between autografting procedures in patients with large burns and limited donor sites, (e) as a "test material" to determine readiness of full-thickness burns to accept an autograft of the patient's own skin, and (f) provision of temporary coverage for any other surgical wound which cannot or should not be closed immediately.

The application of temporary grafts decreases the loss of water and heat by evaporation, and diminishes the exudation of proteins and blood. Wound pain is strikingly decreased, particularly in children, with the decrease in discomfort enhancing the use of joints, thus helping to ensure maximum functional results (Pruitt & Curreri, 1971; Shuck, Pruitt & Moncrief, 1969).

Reconstructive surgery and rehabilitation

Burned tissue of the hands, face and neck, which is particularly common in war burns casualties, is the most likely to result in disfigurement and functional impairment, the most difficult to reconstruct, and the most essential to normal social life. One of the leading specialists on the reconstruction of the face after burn deformities has summarized the situation as follows:

In the face and neck, contractures and scars, the result of tissue destruction from burns, distort the soft tissue structures and may result in severe facial disfigurement and functional impairment . . . The reconstructive surgical rehabilitation of the patient who bears scars and contractures following burns of the face and neck is a major task for the plastic surgeon. As important as the relief of functional disabilities is the reduction of the severity of facial disfigurement and its attendant sociological, psychological, and vocational implications (Converse, 1967, p. 323).

The initial period of treatment in the hospital has concentrated upon the ensurance of survival; facial disfigurement now becomes a major problem if contractures and hypertrophic scars mar the patient's facial features . . . The return of a patient to his home after a long period of hospitalization may be a traumatic experience. It is essential that the patient be forewarned of the reaction of members of the family, friends, and neighbours. A thorough understanding on the part of the patient's spouse and other members of his family is equally important. One of our patients recalls that when he returned home his two children recoiled, screaming, at the sight of their disfigured father. (*Ibid.*, p. 333)

In these circumstances it is understandable that there is often a clamour for early treatment. But the surgeon must resist the pressure placed upon him both by the patient and the family to undertake early reconstructive procedures, since time must be allowed for the maturation of the scar tissue and metabolic and immunological stabilization, after the severe strains which have been placed upon the normal physiological functions of the body (Converse, 1967). Mathews (1964) reports that the best results were obtained in repatriated prisoners of war who had had a forced delay of perhaps two years before reconstructive surgery could be undertaken.

A few other general points may be made about the problems of reconstructive surgery. Early skin grafting is generally recognized to reduce

the problem of hypertrophic scarring and contractions, but not to remove it. The most common type of graft in current practice, the split-thickness skin graft, contracts both during and after the period of healing. The younger the patient the greater the amount of contraction.

Hypertrophic scars are also more common in children:

The propensity of children to hypertrophic scars is well known and similar scars occur in donor areas, following the removal of split-thickness skin grafts of excessive thickness. In the child, the skin is relatively thinner because it is distended over a subcutaneous adipose layer which is thicker than in the adult and penetrates into the base of the dermis through larger and more numerous "columnae adiposae" (Converse, 1967, p. 332).

Hypertrophic scars can occur in various areas of the face. There are indications that certain individuals or racial types may be more predisposed than others to such scars, although the state of nutrition and general health are also determining factors. The most serious deformities occur in those patients in whom initial skin grafting has been delayed or unsuccessful.

After a waiting period to enable such scars to stabilize, the difficult, tedious, drawn-out and costly process of reconstructive surgery can begin.²⁷

For the patient, the numerous and repeated operations may represent a test for the most courageous . . . Because of the often protracted period of treatment and numerous operations, the importance of the psychological management of the severely burned patient is obvious; most of this management will rest upon the surgeon in whom the patient has placed his confidence and who must also act as his psychiatrist (Converse, 1967, p. 336).

One illustration of the length and tediousness of the treatment is the problem of preventing contractures of the neck after corrective surgery. A great advance in the treatment of such contracture was made by Cronin (1957), who put his patients into a moulded neck splint after skin grafting. This splint had to be worn continuously for five to six months. Deformities around the eyes are particularly common, due to the thin tissue of the eye lids. Further, the eyes are particularly liable to infection after burns. Residual scars present difficult problems in the final stages of rehabilitation. The removal of these scars, where possible, is psychologically beneficial to the patient.

²⁷ Dr John Constable, plastic surgeon at the Massachusetts General Hospital, Boston, Mass., estimated that the current cost of such a hospital stay in the United States may be as much as \$100 000. The running costs of treatment at the burn clinic at the Karolinska Hospital in Stockholm were Sw. cr. 1 018 000 (\$254 000) in 1972. The clinic reported 3 147 patient-days of treatment, that is, an average of 8-9 patients at any time, which gives a daily cost of Sw. cr. 323.5 (\$80) per patient. The treatment period ranged from a minimum of 1 month to 8-9 months, giving a treatment cost of Sw. cr. 9 705 (\$2 400) to Sw. cr. 87 345 (\$21 700). This includes staff salaries and medicines, but not overhead costs such as rent, electricity, heat, food, administration, and so on. Thus the total costs are even higher.

V. Additional military medical considerations

The foregoing section described some of the basic problems in the treatment of burns, based on the best methods available in ideal conditions. In combat conditions, the treatment of burns is complicated by associated mechanical injuries from blast and fragmentation weapons, and by limited or damaged facilities. The state of nutrition and of public health of the population affected by incendiary attack are significant factors in their susceptibility to injury.

It is common military practice to use incendiary weapons in combination with other weapons, such as machine-guns and fragmentation grenades; population centres may be bombed with a combination of high explosive and incendiary bombs. As a result, many victims may suffer from both burn wounds and blast and penetrating wounds. In some cases, such as an exploding white phosphorus munition, the particles of incendiary agent may themselves cause multiple penetrating wounds (Pruitt, 1970). This hazard may be increased as research continues into the development of "reactive fragments" (see chapter 2).

Experimental studies have demonstrated that combined injuries from thermal and mechanical trauma may cause a much greater rate of death than either form of injury alone. The mortality rate in mice following mechanical injury with a normal death rate of 10 per cent was increased eight-to-ten-fold if preceded by a sublethal burn injury 0-3 days earlier (Schildt, 1972).

Table 3.18 shows the proportion of burn cases suffering from additional injuries treated at the US Army Institute of Surgical Research between 1965 and 1969, which includes injuries incurred in both combat and noncombat conditions. In the majority of these cases the burns were inflicted through the ignition of fuel in combat vehicles rather than by incendiary weapons. Even so, it is apparent that burns resulting from hostile action are more serious than those from accidental events. A study of 1963 burn patients

Table 3.18. Burn patients with associated injuries admitted to the US Army Institute of Surgical Research, 1965-69

Year	Total burn admissions	Patients with associated injuries	
		Number	per cent of total
1965	174	20	11.5
1966	311	67	21.6
1967	389	96	24.7
1968	389	106	27.2
1969	301	80	26.6
Total	1 564	369	23.6

Source: Pruitt (1970).

treated at a US Army hospital at Yokohama, Japan, showed that hostile causes accounted for 43.2 per cent of the hospital admissions and 69.3 per cent of the deaths, while accidental causes accounted for 51.2 and 30.7 per cent, respectively (Allen, in DiVicenti *et al.*, 1971). The combat casualties, in most cases the trapped crews of armoured vehicles or helicopters in which the fuel exploded, were usually more severe and associated with mechanical injuries and inhalation injuries due to large amounts of smoke and carbonaceous products. It is likely that the combat use of napalm together with conventional munitions creates a somewhat similar pattern of injury.

The treatment of the burn wound by methods such as those outlined above is in general compatible with the treatment of associated mechanical injuries. Conversely, however, Pruitt (1970) concludes that the presence of burns in the multiple injury patient greatly increases the possibility of septic complications and necessitates modifications in many of the standard surgical techniques and principles of wound care.

A second factor of great importance in combat conditions is the general state of health and nutrition of the population at risk. The prognosis for a burned patient is highly dependent not only upon his age, as noted earlier, but upon his general physical condition. Persons with anaemia or a low state of nutrition have greatly reduced ability to survive a severe burn. In some wars a substantial part of the target population may suffer from food shortages, either as a result of their state of economic development or as a result of the destruction of food supplies or means of distribution.²⁸ A low state of nutrition not only increases the death rate from burns but also makes wounds more difficult to heal and more liable to hypertrophic scarring and other forms of disfigurement.

In wartime conditions, the possibilities for optimal treatment are often greatly diminished. Widespread use of incendiaries may result in a rate of burn casualties far greater than can be coped with by available medical facilities. Indeed, the experience of civilian air crashes shows that the burn treatment facilities over a wide area may be stretched to their limits by an influx of, say, some 50 seriously burned patients. In military medicine fine judgements may be required concerning how best to utilize limited resources (Wallace, 1969).

The problem of limited resources is highlighted in two types of warfare in which incendiaries have been widely used. The mass destruction of ad-

²⁸ Oughterson & Warren (1956) report that towards the end of the war in Japan "the inhabitants of the cities were seriously undernourished . . . Malnutrition was responsible for many deaths among injured people already weakened by undernourishment . . ." (p. 81). Dudley *et al.* (1968) say "our experience leads us to believe that the Southeast Asian civilian, perhaps because of marginal subnutrition, perhaps because of some undetected specific deficiency has inherently less ability to respond to massive trauma than either his fitter, better-fed military counterpart, or the Westerner" (p. 339).

vanced industrial cities by the combined use of incendiary and high explosive bombs not only creates many casualties, but also destroys a large proportion of the medical facilities which tend to be concentrated in these areas.²⁹ In the second type of combat situation, incendiaries have been used against guerilla liberation armies in areas which only possess the most rudimentary medical facilities.³⁰ In neither case is evacuation from the theatre of operations a possible solution to the problem of limited medical resources, and only simple forms of treatment can be attempted. By removing any hope of adequate treatment, the suffering of the burned person is magnified, since the problem of treating severe burn wounds becomes almost insurmountable. Victims will either be left to die, or if they recover from lesser burns, they will be left to cope with their own disabilities and disfigurements, including in some cases open wounds which never heal.

In order to make a complete humanitarian assessment of incendiary weapons it is necessary to consider all these aspects of the circumstances in which they are used.

²⁹ Oughterson & Warren (1956) report that "In Hiroshima most of the medical facilities were in the devastated area, and the larger part of them were extremely vulnerable to blast and fire; consequently casualties were heavy. Ninety percent of the 200 to 300 physicians were killed or injured . . . Almost every hospital in Hiroshima within 1 mile of the hypocenter was so severely damaged that it could not function as a hospital. Only 3 of the 45 civilian hospitals were usable . . ." (p. 71 ff). More recently, extensive damage to hospitals in North Viet-Nam has been well documented (US Senate Committee on the Judiciary, 1972). It is unclear from the published information on target selection whether these attacks are the result of imprecise "precision bombing" or precise attacks intended to undermine morale. The problem has in any case reached such proportions that the International Committee of the Red Cross (1973a) is seeking to strengthen the provisions in the Geneva Conventions designed to protect medical resources from attack.

³⁰ The medical services of the National Liberation Forces of Yugoslavia, for example, had to face the following problem during World War II: "Doctors had to flee as refugees or face death, imprisonment or deportation . . . The Germans seemed to have made a special point of destroying hospitals, or any other buildings capable of being used as such . . . the enemy on many occasions slaughtered all the patients and staff they captured before destroying the hospital. Under such circumstances the wastage of medical personnel, scanty to begin with, was enormous." (Hirst, 1945, p. 106).

More recently, a staff report prepared for the subcommittee on refugees of the US Senate Committee on the Judiciary (1970), described bombing raids in Laos with as many as 600 planes a day, dropping napalm, phosphorus, antipersonnel bombs and high explosive bombs and concluded that "with a very low standard of medical care generally, and with a near total absence of adequate government hospitals, the burden of war casualty treatment in Laos is specially heavy" (pp. 32-33). A report prepared by the General Accounting Office of the US Senate estimated that there were "about 36 Laotian doctors, of which 17 are in the military and 19 are administrators of the Royal Lao Government" (*Congressional Record*, 3 May 1972). According to Webb (1968), a former surgeon at the US John F. Kennedy Center for Special Warfare, the "denial of medical resources to insurgents" is an important facet of counter-guerilla warfare. It is noteworthy that the Draft Additional Protocols to the Four Geneva Conventions of 1949 contain the paragraph: "In no circumstances shall any person be punished for carrying out medical activities compatible with professional ethics, regardless of the person benefiting therefrom." (ICRC, 1973a).

VI. The question of suffering

The purpose of this chapter has been to describe the mode of action of incendiary weapons on the human body. This mode of action is complex and dependent upon the circumstances of the burn, the depth and extent, the parts of the body affected, the presence of other injuries, as well as upon the age and condition of the victim, and the quality of the medical resources available.

Military incendiary agents in general cause deep and extensive burns, since they have been developed to the levels necessary to ignite or damage materials, such as metals, much more durable than the human body. Further, their use is typically combined with explosive and fragmentation weapons mutually to enhance the effects.

The medical treatment of severe burns such as those caused by incendiary weapons is more costly, difficult, tedious and demanding both for the patient and the medical staff than is treatment of most other types of injury or sickness. Such treatment is not likely to be available to the majority of incendiary casualties, either in rural areas, or following strategic incendiary or nuclear attacks on cities leading to mass casualties.

The victim of a very serious burn does not necessarily die immediately. He may live for hours, days, or even weeks, depending upon the quality of the accessible medical treatment since even a large area of damage to the skin does not have the immediate impact of, say, a bullet wound in the heart or brain. Death follows in due course from infection, from the complex physiological effects of shock consequent upon loss of fluids, from respiratory complications, or from subsequent strain on other organs leading to heart, kidney or lung failure, or gastric or duodenal ulcers. Except where exceptional medical facilities are available on such a grand scale as are found in only some tens of specialist burn units around the world, the death of the victim of serious burns is rendered probable, if not inevitable, but not necessarily rapid.

The victim of deep, third degree burns may suffer little immediate pain since the pain receptors of the skin are themselves destroyed. The patient may even die without feeling much pain. For those who survive and for those with areas of less severe burn, pain is excruciating, intensifying over a long period during recovery. Deep burns require skin transplantation which itself creates painful donor sites on other areas of the body. In many parts of the world such essential surgery may not be undertaken either because surgical facilities are not available, or because the donor sites may themselves become a source of infection. In these circumstances, the burn wound may never heal, placing the patient under permanent threat of infection as well as under an enormous psychological burden.

The constitution of the World Health Organization (WHO) defines health as "a state of physical, mental and social well-being and not merely the

absence of disease or infirmity". Suffering may be defined by the converse as "a state of physical, mental and social distress, and not merely the presence of disease or infirmity". There can be no doubt that incendiary weapons cause excessive suffering in the fullest sense of this definition.

In view of these facts, the verdict of the Special Committee of the League of Nations disarmament conference in 1932 was that "the cruelty inherent in the uses of these appliances [causes] suffering that cannot be regarded as necessary from a military standpoint" (see chapter 1). It is difficult to see any reason to reverse this judgement.

Chapter 4. Toxic effects of incendiary weapons on the human body

I. Introduction

When materials are burnt or decomposed by heat, a variety of gaseous, liquid and solid substances are produced. Many of these substances, particularly certain gases and smoke made up of solid particles or droplets of acid, have toxic effects which in certain circumstances have proved to be the predominant cause of death and injury resulting from fire. Examples of toxic agents produced by or in fires include phosphine, hydrogen chloride, hydrogen cyanide, phosphoric acid, metal oxides and carbon monoxide. The toxic hazards of fire can thus no longer be overlooked either in the civilian or in the military context. This chapter reviews a number of the products of combustion or thermal decomposition of incendiary agents, and considers the toxicity of these products.

Toxic effects may also be produced by the direct action of certain incendiary agents on the human body. Recent clinical and experimental studies have shown that white phosphorus may be an agent of this kind. The available literature on the toxicity of white phosphorus burns has been extensively reviewed in appendix 4A.

In appendix 4B the toxicity of various incendiary agents and products of combustion are presented.

II. Incendiary agents as asphyxiants

Oxygen is essential both for human life and combustion. With the exception of the pyrotechnic incendiaries (which obtain oxygen from an oxidizing agent in the composition) incendiaries draw oxygen from the air. Where the supply of air is limited, as in a building, insufficient oxygen may be available to ensure complete combustion, while at the same time a variety of potentially lethal and toxic products may be generated.

In exceptional circumstances the amount of oxygen remaining in the confined space may be insufficient to support life, so that persons caught in the space become asphyxiated. Burning hydrocarbon fuels are themselves extinguished when the level of oxygen sinks below some 16 per cent, whereas human beings can continue breathing in an atmosphere with as little as 8–10 per cent oxygen. More frequently the diminished level of oxygen results in the production of carbon monoxide, carbon particles (black smoke) and other products of incomplete combustion. Where the hydrocarbon fuel is extinguished, toxic fumes of, for example, unburned

Incendiary Weapons



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