Chapter 4
Fire-Related Injury Mechanisms

Fredrik Huss

Abstract Understanding medical and biological factors affecting survivability in fires provide a solid ground for safety and preventive measures.

Most fire deaths are due to (smoke) inhalation injuries alone or in combination with burns. Smoke is a complex mixture of compounds that cause harm through Asphyxia, Acute irritation, Physical exposure, and Long-term effects. Not only the present incident and the acute setting are important, the following period also holds a substantial morbidity and mortality. Risk groups such as men, smokers, alcohol−/drug-impaired, physically disabled or cognitively impaired, and elderly have been identified. The global number of elderly people has increased significantly and is projected to continue increase rapidly.

While considering their own safety, emergency medical services need to quickly identify injured patients to promptly initiate life-saving interventions such as securing airways and providing O_2 counteracting the effects of inhaled toxic gases. Minor burns and smoke expositions can be treated ambulatory with routine wound care or a short period of O_2 inhalation. However, larger burns and inhalation injuries are often serious and need advanced treatment in hospital.

Different biological and/or medical factors that can explain, or identify, persons more vulnerable than others to fire, smoke, or heat incidences are reviewed in this chapter.

Given the short amount of time at hand to escape a fire situation, measures to extend this time must be taken; however, prevention is really the key. Environmental modifications, safety rules promotion, etc. need to be tailored to specific groups; children do not function as adults, psychiatric disabled differ from physically disabled, etc.

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1 Introduction

At 03:00 the fire alarm goes off. Mum hears the annoying noise distantly. It was only an hour ago she took the sleeping pill, the wine didn't do it for her this night. The divorce is really taking a toll on her, she can't sleep any longer. She smells smoke, her heart starts to pound. Her head is spinning, she can hear the children cry in their room. She's terrified. Her dad recently moved in to help her after the divorce. She's thankful for that even though she's afraid that he is showing signs of Alzheimer and just recently recuperated from his third heart attack. Dad! She cries, while swearing to herself that he, of course, removed his hearing aid for the night. She stumbles out of bed and heads for the nursery. In the hallway thick black smoke is lingering in the ceiling. The little one stands clinging to the crib wall, crying. She picks him up while screaming to the older child to run outside. With the little one in her arms she heads for her dad. The smoke is thicker now, she can barely see where she's going in the hallway. Her lungs ache, she coughs heavily and can feel how it's becoming heavier to breathe. Mum reaches for her asthma-spray just to realize she's in her pyjamas and the spray is still on the night stand…

Who in the family will make it out safely? This small, typical, family and situation show several intrinsic factors that have severe impacts on the chances for them to escape the dangerous situation.

Fire, heat, and hot substances have since the beginning of times been risks, to all species, and in all societies. Even the *homo erectus* (approximately 1.8 million years ago) was in danger, e.g., from lightning and wildfires. The risks increased even more when hominins learned the ability to control fire (suggested to happen approximately one million years ago [1]). In parallel with mankind's evolution, of course also measures to avoid and handle dangers have been developed and implemented.

As society, with time, has become increasingly more complex, so have also the risks.

Can be seen as obvious, but what exactly are the mechanisms behind people being injured or dying due to fire, heat, and hot substances? Which are the medical and/or biological explanations that make some people (more) vulnerable to fire, smoke, or heat (that also could explain the increased morbidity and mortality in certain groups)?

A multidisciplinary approach to reduce, and possibly avoid, injury and death due to fire, heat, and hot substances has previously mostly prevailed. However, it is evident that an interdisciplinary approach would be more fertile. Given the fact that most victims belong to one, or several, specific risk groups, it is necessary to take on the whole chain; pre-, per-, and post-incident. What can be done from the medical/health care sector to reduce the risks for, and effects of, a fire-related incidence for the more vulnerable persons?

2 Causes of Death in Residential Fires

Preventing situations that can put people in risk of fire and burns is of course a prerequisite in decreasing fire-related morbidity and mortality, e.g., how buildings are built regarding material, escape routes, etc. Once a fire incident is at hand, another prerequisite is that the person is made aware of the danger. It is not obvious that the person can embrace the situation and rationally seek to escape. Elderly, children, and physically, psychiatrically, or alcohol/drug-induced debilitated individuals often need longer time to react, if they react at all, to the threat. Even if/when the threat is acknowledged and the person, hopefully, reacts to escape, other, fire-related, factors come at play.

The majority of residential fire deaths (75–80%) are due to smoke inhalation alone or in combination with cutaneous burns. Since only 26% of deaths are attributable to burns, smoke inhalation is deemed as the main killer [2–5].

Asphyxiation, heat stroke (core hyperthermia), and distributive shock as a result of incidences with fire, heat, and hot substances are examples of immediate causes of death [6]. Occurring, but less common, are associated trauma or even already dead (homicide, suicide, or disease). The cause of death can seemingly be obvious, but there is often a road to the deadly point that is not routinely evaluated in autopsies or forensic examinations. In the scope of this book, these are factors that need to be considered. Why did the person end up in this situation, i.e., why did/could the individual not flee? Incapacitation plays a large role. Smoke, heat, physical condition, illnesses, etc. all add up to the situation preventing the individual from escaping the danger zone, thus exposing himself to further harm or, eventually, death.

2.1 Inhalation Injury

Inhalation injury is defined as inhalation and/or aspiration of hot gases, vapors, liquids, or toxic/chemical substances (residues after combustion). The risk is higher if the fire incident occurs in a closed room. Inhalation injuries can be seen in 2–20% of the cases with (flame) burns. The combination burns and inhalation injury increases morbidity and mortality significantly and increases the patient's need for fluid resuscitation in the early resuscitation phase. Since the heat exchange is very effective in the upper airways, the vast majority of thermal inhalation damage is limited to the upper airways (above the glottis) [7].

Although anamnesis or findings indicate that inhalation of hot or harmful substances has occurred, there are no clear clinical acute signs of an actual inhalation injury. This means that the acute diagnosis is only based on a number of probability factors (an adequately performed and assessed bronchoscopy may help but is not conclusive in the acute setting). These factors are partly based on

circumstances at the time of the injury itself and partly on symptoms of that the upper airways have been damaged. However, the main clinical problems resulting from an inhalation injury are mainly caused by effects at the alveolar level and these cannot be assessed initially, but always appear somewhat later in the process (5–7 days).

Signs of Inhalation Injury

- Fire accident (or similar) in a closed room.
- Soot in sputum.
- Burns to face/neck.
- Changed level of consciousness.
- Strained breathing.
- Hoarseness of voice.

If an inhalation injury is likely, or in anticipated major and early airway problems, immediate intubation (placement of a flexible plastic tube into the trachea to maintain an open airway) should be considered. Especially when there is:

- Deep burns to the neck and face.
- Burns in the mouth.
- Stridor, hoarseness, or swelling in the pharynx.
- Unconsciousness.
- Hypoxia/hypercapnea.

Clinical Entities of Inhalation Injuries

Three clinical entities of inhalation injury exist (and may exist simultaneously):

- Upper airway injury (above glottis).
- Lower airway injury (below glottis).
- Inhalation of toxic gases:

Upper Airway Injury (Above Glottis)

The airways cranially of larynx/glottis, that is, the uvula, pharynx, and epiglottis, can be damaged and thus also swell considerably. This always entails the risk of acute total upper airway obstruction. The patient has stridor, hoarseness, and/or changed pitch. The time for any possible total occlusion of the airways varies and can be unpredictable. Generous indication of intubation exists. Inhalation of hot gases (but also hot liquids or chemicals) is usually the pathogenesis.

Lower Airway Injury (Below Glottis)

Epithelial damage, increased secretion, inflammation, atelectasis, and obstruction occur when toxic substances reach beyond the larynx and the lung parenchyma is damaged. Due to the heat exchange in the glottic area, thermal damage is very rarely seen below the glottis, but they do occur (especially after explosions/explosive combustion). The presence of hot steam also increases the risk of thermal damage in the lower respiratory tract. The patient exhibits dyspnea, cough, wheezing voice, and abundant secretions. Bronchoscopy can reveal an image of edema, bleeding, and soot. Lower airway damage significantly increases mortality.

Inhalation of Toxic Gases

Results in more systemic effects than upper/lower airway damage, vide infra.

2.2 Smoke

Smoke is a complex, heterogeneous, mixture of many different compounds (gases and particles). Depending on the fuel (what's burning), temperature, time since ignition, oxygen supply, and more, the composition varies greatly, also over time.

Four major separate harmful pathways for smoke can be identified [5, 8–10] (Table 4.1):

- Asphyxia.
- Acute irritation.
- Physical exposure (heat, vision).
- Long-term effects.

2.3 Asphyxia

Since all cells of the body demand O_2 for their metabolism, i.e., to live and function, an adequate O_2 delivery is essential and is maintained by the cardiorespiratory systems. The air contains normally 21% O_2 and when inhaled O_2 diffuses, in the alveoli of the lungs, to the capillary blood where it binds to hemoglobin (>98%) or dissolves directly in the plasma (<2%). The heart pumps the blood into the systemic circulation and further eventually to the microcirculation of cells and tissues. There, the O_2 is released from the hemoglobin and diffuses into the cells where it enters the electron transport chain in the mitochondria to enable production of aerobic energy. Normally, the O_2 delivery to the cells is a demand-driven process.

Table 4.1 Table of selected important fire gases

	Type of component	Examples of sources	Principal risks
Asphyxiants			
CO_2, CO	Inorganic gas	All fires	Acute: asphyxia
HCN	Inorganic gas	Nitrogen containing fuel, e.g., nylon	Acute: asphyxia
Irritants			
NO, NO_2, (NO_X)	Inorganic gas	Nitrogen containing fuel, e.g., nylon	Acute: irritation Long term: lung damage
NH_3	Inorganic gas	Nitrogen containing fuel, e.g., nylon	Acute: irritation Long term: lung damage
HCl	Inorganic gas	Chlorine containing fuel, e.g., PVC	Acute: irritation Long term: lung damage
HF	Inorganic gas	Fluorine containing fuel, e.g., PTFE, PVDF	Acute: irritation Long term: lung damage
HBr	Inorganic gas	Bromine containing fuel, e.g., Br-flame retarded material	Acute: irritation Long term: lung damage
SO_2	Inorganic gas	Sulphur containing fuel, e.g., wool	Acute: irritation Long term: lung damage
Isocyanates	Volatile organic gas	Nitrogen containing fuel, e.g., mineral wool	Acute: irritation. Long term: asthma, cancer
Phenol	Volatile organic gas	Generally in many fires	Acute: irritation
Styren	Volatile organic gas	Polystyrene fuel	Acute: irritation
Benzene	Volatile organic gas	All fires	Long term: cancer
PAH	Semi-volatile/ condensed phase organics	All fires, aromatic fuels	Long term: cancer
Dioxins/ furans	Semi-volatile/ condensed phase organics	Chlorine or bromine containing fuels	Long term: cancer, immunotoxicity, etc.
Obscuring			
Soot particles	Particles	All fires	Acute: visual obscuration Long term: depositions in the lungs

After Blomqvist [8]

If the cells' O_2 demands cannot be met due to inability to acquire, transport, or deliver sufficient amounts through breathing/circulation asphyxia is present, cells and tissues become hypoxic and start to die.

Gases developed in fires can cause asphyxia in many ways [11], to mention a few:

- Displacement of O_2 in the inhaled air (less O_2 inhaled).
- Affect the diffusion of O_2 from lung to blood or blood to cells through.
  - (Soot) particles/debris depositions in the alveoli

  - Thermal injury to the mucosa.
  - Shifting of the dissociation curve of hemoglobin.
- Impair hemoglobin O_2 carriage (e.g., CO has 210 times higher affinity than O_2 to hemoglobin and thus blocks the bindings sites for O_2 leading to less O_2 being transported).
- Elicit metabolic acidosis and/or systemic ischemia.

Interference with the mitochondria's electron transport chain.

2.4 Acute Irritation

Mucous membranes in, e.g., eyes, mouth, nose, and respiratory system, react to chemical irritants in different ways, e.g., by increased secretion, bronchospasm, and cell damage. Some effects are acute and can result in, e.g., visual impairment or difficulties breathing rendering the person unable to escape the danger zone, whereas other effects are more long-term (lung damage, pneumonia, pneumonitis, lung edema) that may result in death some time later. The irritative effect that occurs directly is more related to the concentration than the dose of the irritant. However, in larger doses more long-term effects can be seen. Still, the critical doses and concentrations for humans are not easy to establish [8].

2.5 Physical Exposure (Heat)

The heat exchange in the upper airways is very effective. Thus, thermal inhalation injury is mostly limited to the upper airways (above the glottis). There, burns and resulting edema may quickly impair the airways' patency.

In escaping a fire scenario, the heat can also be incapacitating in the sense that the person is prevented to reach escape routes because of oppressing temperatures. The person may find himself trapped in a room because of fear of, or actual, injurious heat and succumbs due to additive harmful factors such as harmful gases.

2.6 Physical Exposure (Vision)

A person's vision may be impaired in a fire scenario not only by irritant gases. The aerosol of solid and liquid particles and gases that make up the smoke simply makes it difficult to see where one is going due to obscuration and darkening (light extinction) of the surroundings [8].

2.7 Long-Term Effects

Even though the acute effects of different smoke constituents probably pose the more important aspects regarding the context of this book. One also needs to consider more long-term effects. Several of the constituent gases, particles, and debris involved in fires can cause direct systemic and airway and lung tissue damage as well as trigger development of, e.g., cancer, immunotoxicity, and asthma later on. Deposition of solid particles, particularly those in the nm-range, are of concern regarding health effects long time after the fire incident [8].

3 Medical and Biological Factors Affecting Mortality

The global number of elderly people (>65 years) has increased. This is a result of advances in living conditions, social care, and medicine and is projected to continue increase rapidly from 0.7 billion people today and reach 1.6 billion in 2050 [12].

In 2019, the global death rate per 100 k population, all ages, regarding burns was 1.44. For people ≥65 years, the rate was more than 3-fold higher, ≥75 years more than 5-fold, and for ≥85 years more than 9-fold.[13].

The risk of injury and death in residential fires (at least in HMIC) has previously been shown increased in risk groups such as men, smokers, alcohol−/drug impaired, physically disabled or cognitively impaired patients, and elderly [14–19]. Cultural and sociodemographic differences along with behavioral factors aid in explaining this increased risk [19–22].

Can possible biological and/or medical factors be found that explain or identify one person being more vulnerable than another to fire, smoke, or heat incidences?

3.1 Carbon Monoxide

The major toxic gas that causes most deaths in residential fires is CO [8]. An inorganic color- and odorless, non-irritable gas, slightly lighter than air, present in basically all fires. The major acute effect is, due to CO's higher affinity to the heme proteins of hemoglobin, that CO competes out the O_2, thus less O_2 can be transported to cells and tissues. Furthermore, CO has high affinity also for other hemeproteins and thus binds to, e.g., myoglobin in myocardium and skeletal muscle [23, 24]. In the myocardium, the effect is cardiac dysfunction. The resulting tissue hypoxia increases the vascular permeability leading to an accumulation of interstitial fluid (edema). Depending on affected organ, the edema gives symptoms such as neurological/unconsciousness (brain), respiratory failure (lungs), arrythmias/heart failure (heart), and renal failure (kidneys). However, also several more delayed effects as neurological and other sequlaes like cognitive deficiency, anxiety,

stubborn headache, sleeping disorder, balance problem, dizziness, impaired vision, neuropathies, mood disorders, muscular weakness, hearing loss and/or tinnitus, stroke, and autoimmune connective tissue diseases can be noted [25–30].

Persons with cardiovascular diseases are postulated to be more vulnerable to CO with incapacitation and death at lower and sublethal concentrations [3, 25, 31–35]. In 541 fire deaths, investigated by Birky et al., 60% of the victims died of CO alone (50% CO-hemoglobin set as lethal concentration), 20% due to CO along with pre-existing cardiovascular disease, and 11% of burns alone [4]. Regional abnormalities of heart wall movement, diagnosed with echocardiography, suggesting preexisting asymptomatic atherosclerosis leading to local ischemia when exposed to CO were found in an older subgroup of patients treated with hyperbaric oxygen for severe CO poisoning [36]. Satran et al. also found preexisting hypertension and male sex-predicted CO-induced myocardial injury [36]. Other studies on people with exertional angina pectoris who were exposed to (low concentrations) CO have established a relation between CO-hemoglobin concentration and time to angina [37].

Healthy adult humans can tolerate 10–15% CO-hemoglobin without symptoms, whereas individuals with coronary artery diseases can develop angina rapidly during physical effort already at CO levels of 2%. A deadly fire in a care home for elderly where 14/19 residents died was investigated by Purser [38]. He could show that the residents with cardiovascular diseases trended towards increased susceptibility to CO poisoning, and for heart disease alone, there was a statistically significant increase.

The treatment is to counteract the O_2 displacement by increasing the amount of O_2 inhaled, thus the increased amount of O_2 can compete with CO at the binding sites on hemoglobin and reduce CO-hemoglobin's half-time from about 4–5 h (room air) to approximately 1 h. Hence, (100%) O_2 should promptly be provided, preferably via a tight-fitting mask. In certain situations, hyperbaric O_2 therapy (100% O_2 under increased atmospheric pressure – commonly 2–3 atmospheres) can be used and will decrease CO-hemoglobin half-time to about 20 min. However, the indications and (side) effects of HBO treatment are debated.

3.2 Carbon Dioxide

Carbon dioxide, CO_2, is a color- and odorless, inorganic, non-irritable gas, heavier than air (1.5 times), present in basically all fires. CO_2 is well-tolerated in low concentrations, but in higher (>5%) acts mostly as a simple asphyxiant, by displacing O_2, but also as a toxicant [39].

In higher concentrations, hypercapnia and respiratory acidosis develop which affect the parasympathetic nervous system, thus depressing the respiration and circulation. At even higher concentrations, neurological symptoms as convulsions, coma, and death appear. In >30% CO_2, almost instant unconsciousness followed by cardiac arrest within minutes occurs. Thus, in situations with very high CO_2 concentrations, the toxic effect may well be the lethal cause and not the asphyxiating [39].

However, in residential fires, it is unlikely that the concentrations of CO_2 will rise fast enough to be the lethal gas since other, more fast-acting, gases will reach lethal concentrations sooner.

Symptoms present in CO_2 intoxication thusly vary depending on the concentration and tachy−/bradypnoea, tachy−/bradycardia, arrhythmias, convulsions, and cardiac stress can be noted. This is probably the reason for that hypercapnia due to CO_2 intoxication is more dangerous for people with cardiovascular deceases [40].

Another important mechanism of CO_2 is its effect of increasing the respiratory rate, thus increasing uptake of other (toxic) combustion products [3]. Especially elderly and people with cardio−/respiratory conditions are more vulnerable in fire incidents [41].

3.3 Oxygen Depletion

The three necessary constituents in a fire (the fire triangle) are fuel, energy, and an oxidizer [8]. As the process of combustion proceeds, the constituents are consumed. For O_2, this means that the atmosphere becomes O_2-depleted which, of course, even worsens the effects of the asphyxiant gases.

Oxygen deprivation alone can incapacitate a person at around 10% O_2 concentration and be lethal at <7%. However, without extreme heat, such as in a flash fire (room temperature high enough to autoignite most flammable materials and the room is engulfed in flames), this situation is unlikely. Furthermore, the temperature involved in a flash fire would probably, in itself, be lethal before the O_2 deprivation affects the person [42].

3.4 Hydrogen Cyanide

Hydrogen cyanide, HCN, is an inorganic colorless gas (boiling point = 25.6 °C) released when nitrogen containing fuel burns with mostly asphyxiating effects. To most people, it has a marked bitter almond odor; however, approximately one-third of the population lack the ability to smell HCN, possibly genetically controlled. HCN is approximately 35 times more toxic than CO, and basically all organs of the body, especially those sensitive to low O_2 levels, brain, respiratory, and cardiovascular systems, can be affected. Inhalation of HCN can be rapidly fatal [8]. Even though its role in fire deaths is somewhat debated, it is regarded as being an important asphyxiating gas that needs prompt treatment. HCN shows a molecular similarity to O_2 in the sense that it binds (reversibly) to cytochrome a3 and interferes with the oxidative metabolism at the cellular level (intracellular hypoxia occurs within the mitochondria), thus turning the metabolism into anaerobic, producing lactate that leads to systemic acidosis. HCN needs to be promptly treated with, e.g., hydroxocobalamine, but since there are no specific tests or symptoms the

treatment indication is the suspicion of exposure and/or acidosis. Levels of HCN can be measured in blood, but the interpretation of the results is difficult since HCN is both produced and degraded in blood and body tissues, even postmortally [43, 44].

Even though data are lacking regarding increased vulnerability to HCN in any risk group, it can be safe to establish that there is enough synergy among asphyxiant gases and people with cardiovascular diseases to view them more vulnerable. Synergy between HCN and CO has been shown in animal models [45, 46].

3.5 Asphyxiation Synergy

The common effect of all asphyxiants, or narcotic gases as they are sometimes called, is that they ultimately lower the O_2 concentration in tissues and organs, be it by simple ambient O_2 displacement (lowers inhaled O_2 concentration) or by systemic effects. Carbon monoxide is probably the most studied asphyxiant and preexisting cardiovascular diseases seem to increase the vulnerability through cardiac ischemia. Other asphyxiants probably produce similar effects and may also act in synergy.

The common, most basic, treatment is to provide a surplus of O_2 to be inhaled, be it through normo- or hyperbaric ways.

3.6 Irritants

Irritant gases are most often (volatile) inorganic gases with the common feature of acutely inducing irritation of, preferably, the airways, but also eyes, skin, and mucous membranes generally. The irritants have an important role regarding lethality in fire situations. The irritants may not be acutely lethal, but the rapid physiological and resulting behavioral effects reduce the possibility to escape and lead to incapacitation, allowing the accumulating irritating effects or the asphyxiants, or heat, to become lethal [8, 47, 48].

The principal effects of irritants are similar and HCl may be the most studied irritant in fire and smoke situations. However, most data are from animal models and the human interpretation needs to be careful [49].

Most irritants also elicit long-term effects such as lung damage, asthma, cancer, and immunotoxicity.

As with the asphyxiants, preexisting conditions like pulmonary diseases or asthma render people more vulnerable to the irritants since their physiological reserves are already limited.

Irritant gases can cause pain in eyes, skin, or respiratory tract, bronchospasm, hypersecretion, and impaired vision.

3.7 Nitrogen Dioxide

Nitrogen dioxide, NO_2, is an inorganic, reddish-brown gas with a pungent acid smell. It is released when nitrogen-containing fuel burns with acutely irritating and to some degree asphyxiating effect and can, in the long-term, cause lung damage.

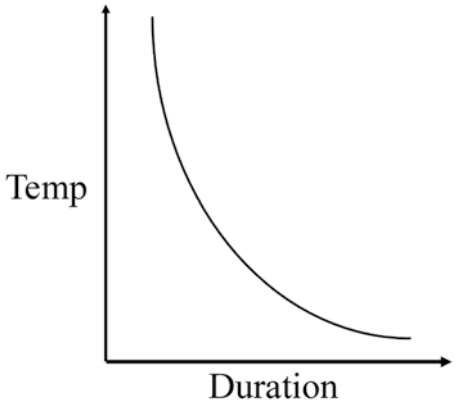
NO_2 is directly toxic to the respiratory tract and is easily absorbed through the lungs. NO_2 diffuses into the respiratory epithelial lining fluid and dissolves and chemically reacts. This reaction and its metabolites determine the health effects which include (acute and long-term) cardiovascular effects (triggering heart attacks), diffuse inflammation, bronchoconstriction, inflammation, reduced immune response, edema leading to bronchitis, pneumonia, or fulminant lung edema, and interstitial fibrosis [50].

Besides the asphyxiating effect of fluid accumulation in the lungs, the formation of methemoglobin blocks the O_2 transport in much the same way as CO [51].

3.8 Heat Exposure

Classic heatstroke can be caused by high surrounding temperatures [6]. Preexisting medical conditions and/or an inactive lifestyle with low aerobic capacity increase the vulnerability to heat (in)tolerance or heatstroke. Age itself seems not to be a decisive risk factor [52, 53]. In a fire incident, the rise in temperature is most often not that quick and other mechanisms of incapacitation, injury, and death occur before a classic heatstroke develops.

The amount of tissue damage inflicted by heat can be viewed as a simple relationship between how long the tissue is exposed to a certain temperature.



Considering that a prolonged exposition of the skin to 44 °C is enough to produce irreversible epithelial cell damage, it is easily understood that higher

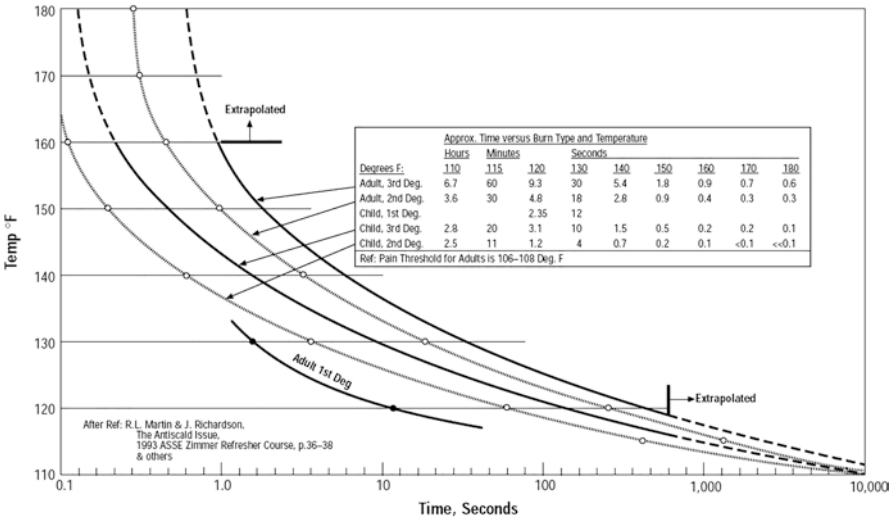


Fig. 4.1 Relationship temperature-time and suggested cut-offs for different burn depths in children and adults [55].

temperatures, such as those involved in a fire, can cause serious damage to the human body's deeper tissues rapidly [54] (Fig. 4.1).

Several, mostly mathematical, models have been used to estimate what temperature skin and tissue reach during a burn/heat incident [56–58]. There are a plethora of factors affecting the tissue response to heat; e.g., skin thickness varies depending on the body area and between persons due to age, gender, and more; blood perfusion of the tissue varies; skin is affected differently whether the heat source is radiation, flame, scald, or contact; type of clothing worn; moisture on the skin (sweat); air humidity; etc. To be exposed to a fire/heat incidence elicits several physiological responses and reactions that further complicate the prediction of tissue damage. For example, a decrease in heart function with decreased cardiac output and hypotension, e.g., due to effects of inhaled gases, lessens the blood perfusion to the skin, which is followed by both the heat exchange (by the flowing blood) being decreased, and thus more heat energy absorbed, giving deeper burns. A sustained hypotension with the sustained lessened blood flow in the skin creates a cellular environment lacking enough nutrients and O_2, leading to that cells that have been subjected to reversible damage from the heat insult may well succumb due to the cellular environment.

From these complicated conditions, it can be perceived as contradictory as to when and how tissue damage occurs. An ambient temperature of 90 °C can well be tolerated for 45 min or longer without resulting in cutaneous burns (compared to being in a sauna) [59]. Others indicate that tolerance to temperatures <120 °C is limited by hyperthermia, but at >120 °C pain is followed by cutaneous burns [10].

Increased ambient temperature (⩾31 °C) during exposure to CO was studied in healthy adults, indicating synergistic effects of the heat and gas probably leading to more rapid incapacitation of a person.

Considering mortality and morbidity by fire, heat, and hot substances, one frequently focuses on the incident itself and the acute setting. However, one must not forget the period that follows the insult.

3.9 (Cutaneous) Burns

Some of the main functions of the skin are:

- To be a barrier against the environment (both "physically" and against pathologic microorganisms).
- Temperature regulation.
- Fluid regulation.
- To be an interface to the surroundings (sensation, looks).

After having been exposed to fire, heat, and hot substances, it is common that the person has acquired cutaneous burns. The burnt skin then loses the above mentioned functions.

The burn trauma is a condition with many faces ranging from minor superficial blisters/wounds that heal uneventfully spontaneously to massive full-thickness burns covering large areas of the body necessitating advanced intensive care and surgery. Treatment of significant burns is often considered one of the costliest forms of hospital care.

The burn trauma itself induces a massive inflammatory reaction both locally and systemically and affects basically all organs and systems in the body. The open wounds, along with different (blood-, respiratory-, gastric-, urinary-, etc.) catheters needed for the (intensive) care, are gateways for pathological microbes easily leading to infections and sepsis with high morbidity and mortality.

The depth, surface extension of the burn wounds, age, and the presence of an inhalation injury are the major denominators determining the severity, treatment, and outcome of the burn trauma [60, 61].

The modern terms used are:

- Superficial burns (epidermal, previous 1st degree).
  - Only the epidermis is damaged.
  - Intense redness and pain.
  - Equivalent to a sunburn.
  - Intact sensation and capillary refill.
  - Heals in few days without scars.

- Superficial dermal burn (previous 2nd degree, a).
  - The entire epidermis and superficial parts of the dermis are damaged.
  - Often blisters, moist surface, redness, and painful wound surface.
  - Intact sensation and capillary refill.
  - Usually heals within 2 weeks with proper wound care and without scars, but with possible changes in pigmentation.
- Deep dermal burn (previous 2nd degree, b).
  - The entire epidermis and deep parts of the dermis (hair follicles, sweat glands, and other adnexae) are damaged.
  - Marbled, dark red-white, dry, or moist wound surface.
  - Usually no blistering.
  - Diminished sensation and capillary refill.
  - Surgery with skin transplantation is usually required.
- Full-thickness burn (previously 3rd degree).
  - The entire skin (epidermis and dermis) and sometimes also deeper tissues are damaged.
  - Pale-brown or black color, leathery texture.
  - Dry surface.
  - No sensation or capillary refill.
  - Surgery with skin transplantation is required for healing.

Burns affecting >20% of the total body surface area (%TBSA) are usually considered major, or significant. Full-thickness burns are more serious than superficial. However, also larger superficial burns cause systemic (inflammatory) reactions that can be detrimental.

To predict the mortality in burn patients, professor Serge Baux presented, in 1961, the Baux-score: mortality rate = age + %TBSA [62]. However, advances in burn care and the appreciation of an inhalation injury's deleterious effect have rendered the original Baux-score too pessimistic. Thus, in 2010, Osler et al. revised the Baux-score to mortality rate = age + %TBSA + plus 17 points if inhalation injury is present [63]. Today, the better burn centers regularly successfully treat burns with Baux-score > 130–140. This indicates that the question today is not to be whether the patient survives, especially not in younger age groups, but more to what kind of life the patient can be rehabilitated back to.

Looking at global in-hospital mortality due to fire, heat, and hot substances, one must carefully evaluate published data because of an obvious risk of selection bias since patients with a poor prognosis will probably not be referred to, and treated, in hospitals in resource-constrained areas.

4 On the Scene

Of all the destructive mechanisms involved in a fire, carbon monoxide and hydrogen cyanide may be the most sinister. Mortality is highly increased when these 2 gases are involved. Emergency medical services (ambulance etc.) need to quickly identify these patients and promptly initiate life-saving interventions such as securing airway and spinal control and provide O_2 and intravenous fluid resuscitation [9].

The patients may not present with classic signs of toxic exposure; thus, triage may be difficult. Textbook signs of cherry red skin (CO) or smell of bitter almond (CN) are mostly only found in the books. Due to the varying time of exposure and concentrations of toxic gases, a broad variety of signs and symptoms may be present: e.g., nausea, confusion, headache, loss of consciousness, cardiac arrest, etc. Thus, also potential risk factors and environmental findings that could indicate inhalation injuries, like fire incident in an enclosed space, should be sought after [9]. Routine examinations with, e.g., pulse oximetry are unreliable and more specific laboratory data are not available on the scene. EMS therefore should broadly initiate treatment. Equally important, though, is the safety of the EMS and others working on the scene. Irritant and toxic substances released in the fire may also affect them and proper protective equipment is necessary.

A cornerstone in the field work on site is to provide supportive and safe care while acknowledging, in order of priority, safety, patient extrication and decontamination, airway management, getting intravenous access, burn treatment, prevent hypothermia, and patient dispersion [9].

4.1 Treatment

Minor scalds, burns, and smoke expositions can preferably be treated in ERs or GP's offices. Routine wound care or a short period of O_2 inhalation is often sufficient. However, larger burns and inhalation injuries are most often serious and need advanced treatment in hospital.

4.2 Isolated Inhalation Injuries

The medical issues with an (isolated) inhalation injury can be summarized as toxic effects (local and systemical), increased secretion, and obstructivity. If the patient cannot maintain a patent airway or the oxygen exchange, intubation and connection to a ventilator is necessary. The patient is treated in the intensive care unit. Symptomatic treatment with O_2, bronchodilators, and bronchial toilet is often sufficient. Complications as pneumonia and sepsis are treated with systemic antibiotics and other necessary organ supportive treatment.

4.3 Burn Injuries With/Without Inhalation Injury

More extensive burns should be treated in specialized burn units/centers. A burn trauma patient is a special type of trauma patient and should, in the acute phase, be treated according to Emergency Management of Severe Burns (EMSB) or Advanced Burn Life Support (ABLS), and Advanced Trauma Life Support (ATLS) [64–68].

The burn trauma itself elicits a massive systemic inflammatory response, leading to basically all organ systems and cascade systems becoming affected. (vide supra) The treatment involves securing a patent airway and organ-supportive measures. The cornerstone of the acute treatment is, besides ordinary intensive care (organ support), the fluid resuscitation, where the fluids that escape the bloodstream into the soft tissue due to the inflammatory reaction are compensated. This is to ensure that critical organs (kidneys, brain, heart, etc.) have enough circulation ("permissive hypovolemia") to maintain their primary functions. According to the Parkland formula, the patient is to be given 2–4 ml × body weight (kg) × %TBSA intravenous fluid the first 24 h after trauma [69]. Thus, for a 100 kg patient with 50% burns, the calculated required amount of resuscitation fluid for the first 24 h is 10–20 l! Still, the Parkland formula only calculates a predictive fluid amount and the individual volume should be adjusted according to the patient's circulatory status and urine production aiming for "permissive hypovolemia" [7, 70]. A concomitant inhalation injury usually increases this amount with >50% (i.e., >30 l in the example). A massive general swelling/edema thus follows that can lead to serious complications such as compartment syndrome in extremities, abdomen, eyes, or brain. The burned skin/tissue is no longer viable and should be removed within the first days. Surgically, the burned tissue is removed and resulting wounds covered with, preferably, split-thickness skin grafts from the patient's unburned areas. However, already at 20–30% TBSA (and above), the amount of unburned skin may not be sufficient to cover all wounds. In these cases, different techniques such as re-harvesting skin when donor sites have healed or in vitro culture of autologous epithelial grafts can be used.

Commonly, one estimates approximately 1 hospital day per % TBSA burned. The early hospital phase, though, is only the start of a long marathon. Months, and even years, follow with physiotherapy and rehabilitation.

5 Discussion

Globally, incidences with fire, heat, and hot substances compose a serious general public health problem. More than 300,000 deaths occur annually from fire incidents alone [71]. Add to that even more deaths from scalds, electrical−/, chemical−/, and contact burns. Furthermore, millions of persons involved in incidences with fire, heat, and hot substances suffer from long-term issues as disabilities or disfigurements, let alone issues as destroyed homes and belongings and other secondary personal and economic effects.

The absolute majority (>95%) of the global fire-related deaths and injuries occur in LMICs where health care systems often are not as developed [71]. Many fire/burn prevention strategies have been introduced over the years and results are seen. However, in the global sense, strategies that have been proven successful in HICs may not be transferable to LMICs as the epidemiology and trauma panorama is substantially different.

Through different modellings based on inter alia experimental data from human and animal studies, time to human incapacitation in fire situations can be calculated. Commonly, approximately 3–4 min since start of fire are mentioned as time to incapacitation, and around 5–6 min to death [10, 72, 73].

Obviously, different physical properties of the fire (fuel, time, etc.) and where the fire occurs (same room as person, further away, etc.) as well as different biological (age, gender, etc.) and physical properties (diseases, disabilities, etc.) of the person allow this time to vary greatly.

Fire incidents have always happened and will also continue to happen in the future. Given the short amount of time at hand, once there is a fire incident, to escape the threatening situation, one comes to the conclusion that, of course, measures need to be taken to extend this time period, but that prevention is really the key. However, it has become increasingly evident that there is no "magic bullet" when it comes to prevention. Common preventive measures like modification of the environment, safety rules promotion, and suggested changes in behavior pre- and peri-incident can be effective, but need to be individually tailored to specific (risk) groups both in how measures are taken and how information is given; children do not function as adults, psychiatric disabled persons differ from physically disabled, etc. [74–79].

References

1. Berna F, Goldberg P, Horwitz LK et al (2012) Microstratigraphic evidence of in situ fire in the Acheulean strata of Wonderwerk Cave, Northern Cape province, South Africa. Proc Natl Acad Sci U S A 109:E1215–E1220. https://doi.org/10.1073/pnas.1117620109
2. Alarie Y (1985) The toxicity of smoke from polymeric materials during thermal decomposition. Annu Rev Pharmacol Toxicol 25:325–347. https://doi.org/10.1146/annurev.pa.25.040185.001545
3. Shusterman DJ (1993) Clinical smoke inhalation injury: systemic effects. Occup Med Phila Pa 8:469–503
4. Birky MM, Clarke FB (1981) Inhalation of toxic products from fires. Bull N Y Acad Med 57:997–1013
5. Eggert E, Huss F (2017) Medical and biological factors affecting mortality in elderly residential fire victims: a narrative review of the literature. Scars Burns Heal 3:205951311770768. https://doi.org/10.1177/2059513117707686
6. Kenttämies A, Karkola K (2008) Death in sauna. J Forensic Sci 53:724–729. https://doi.org/10.1111/j.1556-4029.2008.00703.x
7. Burn Center, Uppsala University Hospital (2019) Brännskadekompendium (Burn manual). https://pdf-flip.se/Akademiska_Sjukhuset/Brannskadekompendium/
8. Blomqvist P (2005) Emissions from fires consequences for human safety and the environment. Lund University, Lund

9. Gold A, Perera TB (2020) EMS, asphyxiation and other gas and fire hazards. In: StatPearls. StatPearls Publishing, Treasure Island
10. Purser DA, McAllister JL (2016) Assessment of hazards to occupants from smoke, toxic gases, and heat. In: Hurley MJ, Gottuk D, Hall JR et al (eds) SFPE handbook of fire protection engineering. Springer, New York, pp 2308–2428
11. Prien T, Traber DL (1988) Toxic smoke compounds and inhalation injury—a review. Burns 14:451–460. https://doi.org/10.1016/S0305-4179(88)80005-6
12. World Population Prospects – Population Division – United Nations. https://population.un.org/wpp/Graphs/Probabilistic/POP/65plus/900. Accessed 4 Jan 2021
13. Institute for Health Metrics and Evaluation In: Inst Health Metr Eval. http://www.healthdata.org/institute-health-metrics-and-evaluation. Accessed 4 Jan 2021
14. Elder AT, Squires T, Busuttil A (1996) Fire fatalities in elderly people. Age Ageing 25:214–216. https://doi.org/10.1093/ageing/25.3.214
15. Marshall SW, Runyan CW, Bangdiwala SI et al (1998) Fatal residential fires: who dies and who survives? JAMA 279:1633. https://doi.org/10.1001/jama.279.20.1633
16. Beaulieu E, Smith J, Zheng A, Pike I (2020) The geographic and demographic distribution of residential fires, related injuries, and deaths in four Canadian provinces. Can J Public Health 111:107–116. https://doi.org/10.17269/s41997-019-00256-7
17. Svee A, Jonsson A, Sjöberg F, Huss F (2016) Burns in Sweden: temporal trends from 1987 to 2010. Ann Burns Fire Disasters 29:85–89. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5241198/
18. Jonsson A, Bonander C, Nilson F, Huss F (2017) The state of the residential fire fatality problem in Sweden: epidemiology, risk factors, and event typologies. J Saf Res 62:89–100. https://doi.org/10.1016/j.jsr.2017.06.008
19. Istre GR, McCoy MA, Osborn L et al (2001) Deaths and injuries from house fires. N Engl J Med 344:1911–1916. https://doi.org/10.1056/NEJM200106213442506
20. Miller I (2005) Human behaviour contributing to unintentional residential fire deaths 1997–2003. New Zealand Fire Service, Wellington
21. Nilson F, Bonander C (2020) Societal protection and population vulnerability: key factors in explaining community-level variation in fatal fires involving older adults in Sweden. Fire Technol 57(1):247–260. https://doi.org/10.1007/s10694-020-00997-9
22. Purcell LN, Bartley C, Purcell ME et al (2020) The effect of neighborhood Area Deprivation Index on residential burn injury severity. Burns S0305417920304733. https://doi.org/10.1016/j.burns.2020.07.014
23. Lim M, Jackson T, Anfinrud P (1995) Binding of CO to myoglobin from a heme pocket docking site to form nearly linear Fe-C-O. Science 269:962–966. https://doi.org/10.1126/science.7638619
24. Kinoshita H, Türkan H, Vucinic S et al (2020) Carbon monoxide poisoning. Toxicol Rep 7:169–173. https://doi.org/10.1016/j.toxrep.2020.01.005
25. Weaver LK (2020) Carbon monoxide poisoning. Undersea Hyperb Med J Undersea Hyperb Med Soc Inc 47:151–169
26. Mazo J, Mukhtar E, Mazo Y et al (2020) Delayed brain injury post carbon monoxide poisoning. Radiol Case Rep 15:1845–1848. https://doi.org/10.1016/j.radcr.2020.07.048
27. Garcia JH, Khanna S (2020) Severe vision loss and intracranial hypertension presenting as delayed sequelae of "Mild" carbon monoxide poisoning. J Neuroophthalmol. Publish ahead of print. https://doi.org/10.1097/WNO.0000000000001104
28. Huang C-C, Ho C-H, Chen Y-C et al (2020) Autoimmune connective tissue disease following carbon monoxide poisoning: a nationwide population-based cohort study. Clin Epidemiol 12:1287–1298. https://doi.org/10.2147/CLEP.S266396
29. Kwak K, Kim M, Choi W-J et al (2021) Association between carbon monoxide intoxication and incidence of ischemic stroke: a retrospective nested case-control study in South Korea. J Stroke Cerebrovasc Dis 30:105496. https://doi.org/10.1016/j.jstrokecerebrovasdis.2020.105496

30. Liu J, Si Z, Liu J et al (2020) Clinical and imaging prognosis in patients with delayed encephalopathy after acute carbon monoxide poisoning. Behav Neurol 2020:1–5. https://doi.org/10.1155/2020/1719360
31. Ernst A, Zibrak JD (1998) Carbon monoxide poisoning. N Engl J Med 339:1603–1608. https://doi.org/10.1056/NEJM199811263392206
32. Stefanidou M, Athanaselis S, Spiliopoulou C (2008) Health impacts of fire smoke inhalation. Inhal Toxicol 20:761–766. https://doi.org/10.1080/08958370801975311
33. Balraj EK (1984) Atherosclerotic coronary artery disease and "low" levels of carboxyhemoglobin; report of fatalities and discussion of pathophysiologic mechanisms of death. J Forensic Sci 29:1150–1159
34. Wright J (2002) Chronic and occult carbon monoxide poisoning: we don't know what we're missing. Emerg Med J 19:386–390. https://doi.org/10.1136/emj.19.5.386
35. Balzan MV, Cacciottolo JM, Mifsud S (1994) Unstable angina and exposure to carbon monoxide. Postgrad Med J 70:699–702. https://doi.org/10.1136/pgmj.70.828.699
36. Satran D, Henry CR, Adkinson C et al (2005) Cardiovascular manifestations of moderate to severe carbon monoxide poisoning. J Am Coll Cardiol 45:1513–1516. https://doi.org/10.1016/j.jacc.2005.01.044
37. Allred EN, Bleecker ER, Chaitman BR et al (1991) Effects of carbon monoxide on myocardial ischemia. Environ Health Perspect 91:89–132. https://doi.org/10.1289/ehp.919189
38. Purser DA (2017) Effects of pre-fire age and health status on vulnerability to incapacitation and death from exposure to carbon monoxide and smoke irritants in Rosepark fire incident victims: effects of pre-fire age and health status on fire victims. Fire Mater 41:555–569. https://doi.org/10.1002/fam.2393
39. Permentier K, Vercammen S, Soetaert S, Schellemans C (2017) Carbon dioxide poisoning: a literature review of an often forgotten cause of intoxication in the emergency department. Int J Emerg Med 10:14. https://doi.org/10.1186/s12245-017-0142-y
40. Langford NJ (2005) Carbon dioxide poisoning. Toxicol Rev 24:229–235. https://doi.org/10.2165/00139709-200524040-00003
41. Sharma G, Goodwin J (2006) Effect of aging on respiratory system physiology and immunology. Clin Interv Aging 1:253–260. https://doi.org/10.2147/ciia.2006.1.3.253
42. Alarie Y (2002) Toxicity of fire smoke. Crit Rev Toxicol 32:259–289. https://doi.org/10.1080/20024091064246
43. Huzar TF, George T, Cross JM (2013) Carbon monoxide and cyanide toxicity: etiology, pathophysiology and treatment in inhalation injury. Expert Rev Respir Med 7:159–170. https://doi.org/10.1586/ers.13.9
44. Barillo DJ, Goode R, Esch V (1994) Cyanide poisoning in victims of fire: analysis of 364 cases and review of the literature. J Burn Care Rehabil 15:46–57. https://doi.org/10.1097/00004630-199401000-00010
45. Levin BC, Paabo M, Gurman JL, Harris SE (1987) Effects of exposure to single or multiple combinations of the predominant toxic gases and low oxygen atmospheres produced in fires. Fundam Appl Toxicol Off J Soc Toxicol 9:236–250. https://doi.org/10.1016/0272-0590(87)90046-7
46. Norris JC, Moore SJ, Hume AS (1986) Synergistic lethality induced by the combination of carbon monoxide and cyanide. Toxicology 40:121–129. https://doi.org/10.1016/0300-483x(86)90073-9
47. Purser DA (2010) Hazards from smoke and irritants. In: Stec A, Hull R (eds) Fire toxicity. WP, Woodhead Publishing, Oxford, pp 51–117
48. (2001) International study of the sublethal effects of fire smoke on survivability and health (SEFS) : phase I final report. National Institute of Standards (NIST)
49. Hull T, Stec A, Paul K (2008) Hydrogen chloride in fires. Fire Saf Sci 9:665–676. https://doi.org/10.3801/IAFSS.FSS.9-665
50. US EPA National Center for Environmental Assessment RTPN Integrated Science Assessment (ISA) for Oxides of Nitrogen – Health Criteria (Final Report, Jan 2016). https://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=310879. Accessed 13 Jan 2021

51. Hoffman RS, Sauter D (1989) Methemoglobinemia resulting from smoke inhalation. Vet Hum Toxicol 31:168–170
52. Pandolf KB (1997) Aging and human heat tolerance. Exp Aging Res 23:69–105. https://doi.org/10.1080/03610739708254027
53. Kenney WL (1997) Thermoregulation at rest and during exercise in healthy older adults. Exerc Sport Sci Rev 25:41–76
54. Moritz AR, Henriques FC (1947) Studies of thermal injury: II. The relative importance of time and surface temperature in the causation of cutaneous burns. Am J Pathol 23:695–720
55. Bynum Jr D, Petri VJ et al (1998) Domestic hot water scald burn lawsuits – the who, what, when, why, where how. A Seminar and Technical Paper for the 25–28 Oct. 98 Annual ASPE Meeting at the Indianapolis Convention Center in Indianapolis, Indiana. Reprinted by Watts Regulator Company with permission of Dr. D. Bynum Jr. https://mail.inspectapedia.com/plumbing/DHW_Scald_Seminar_Reprint.pdf
56. Abraham JP, Plourde B, Vallez L et al (2015) Estimating the time and temperature relationship for causation of deep-partial thickness skin burns. Burns J Int Soc Burn Inj 41:1741–1747. https://doi.org/10.1016/j.burns.2015.06.002
57. Ng EYK, Tan HM, Ooi EH (2010) Prediction and parametric analysis of thermal profiles within heated human skin using the boundary element method. Philos Trans R Soc Math Phys Eng Sci 368:655–678. https://doi.org/10.1098/rsta.2009.0224
58. Majchrzak E, Jasiński M (2003) Numerical estimation of burn degree of skin tissue using the sensitivity analysis methods. Acta Bioeng Biomech 2003:93–108
59. Yeo TP (2004) Heat stroke: a comprehensive review. AACN Clin Issues 15:280–293. https://doi.org/10.1097/00044067-200404000-00013
60. Sheppard NN, Hemington-Gorse S, Shelley OP et al (2011) Prognostic scoring systems in burns: a review. Burns 37:1288–1295. https://doi.org/10.1016/j.burns.2011.07.017
61. Colohan SM (2010) Predicting prognosis in thermal burns with associated inhalational injury: a systematic review of prognostic factors in adult burn victims. J Burn Care Res 31:529–539. https://doi.org/10.1097/BCR.0b013e3181e4d680
62. Baux S (1961) Contribution a l'etude du traitement local des brulures thermiques etendues. Thesis
63. Osler T, Glance LG, Hosmer DW (2010) Simplified estimates of the probability of death after burn injuries: extending and updating the baux score. J Trauma 68:690–697. https://doi.org/10.1097/TA.0b013e3181c453b3
64. Stone CA, Pape SA (1999) Evolution of the Emergency Management of Severe Burns (EMSB) course in the UK. Burns 25:262–264. https://doi.org/10.1016/S0305-4179(98)00161-2
65. ABLS Program – American Burn Association. https://ameriburn.org/education/abls-program/. Accessed 9 Mar 2021
66. Kearns RD, Hubble MW, JamesH H et al (2015) Advanced burn life support for day-to-day burn injury management and disaster preparedness: stakeholder experiences and student perceptions following 56 advanced burn life support courses. J Burn Care Res 36:455–464. https://doi.org/10.1097/BCR.0000000000000155
67. Advanced Trauma Life Support In: Am Coll Surg. https://www.facs.org/quality-programs/trauma/atls. Accessed 9 Mar 2021
68. Sims JK (1979) Advanced trauma life support laboratory: pilot implementation and evaluation. J Am Coll Emerg Physicians 8:150–153. https://doi.org/10.1016/S0361-1124(79)80342-1
69. Baxter CR, Shires T (1968) Physiological response to crystalloid resuscitation of severe burns. Ann N Y Acad Sci 150:874–894. https://doi.org/10.1111/j.1749-6632.1968.tb14738.x
70. Saffle JR (2007) The phenomenon of "fluid creep" in acute burn resuscitation. J Burn Care Res 28:382–395. https://doi.org/10.1097/BCR.0B013E318053D3A1
71. Mock C, Peck M, Krug E, Haberal M (2009) Confronting the global burden of burns: a WHO plan and a challenge. Burns 35:615–617. https://doi.org/10.1016/j.burns.2008.08.016
72. Gann RG (2004) Estimating data for incapacitation of people by fire smoke. Fire Technol 40:201–207. https://doi.org/10.1023/B:FIRE.0000016843.38848.37

73. Purser D (1989) Modelling toxic and physical hazard in fire. Fire Saf Sci 2:391–400. https://doi.org/10.3801/IAFSS.FSS.2-391
74. Committee on Injury and Poison Prevention (2000) Reducing the number of deaths and injuries from residential fires. Pediatrics 105:1355–1357. https://doi.org/10.1542/peds.105.6.1355
75. Smith GA, Kistamgari S, Splaingard M (2020) Optimizing smoke alarm signals: testing the effectiveness of children's smoke alarms for sleeping adults. Inj Epidemiol 7:51. https://doi.org/10.1186/s40621-020-00279-6
76. Kegler SR, Dellinger AM, Ballesteros MF, Tsai J (2018) Decreasing residential fire death rates and the association with the prevalence of adult cigarette smoking — United States, 1999–2015. J Saf Res 67:197–201. https://doi.org/10.1016/j.jsr.2018.06.001
77. Shokouhi M, Nasiriani K, Cheraghi Z et al (2019) Preventive measures for fire-related injuries and their risk factors in residential buildings: a systematic review. J Inj Violence Res 11:1–14. https://doi.org/10.5249/jivr.v11i1.1057
78. Gielen AC, Frattaroli S, Pollack KM et al (2018) How the science of injury prevention contributes to advancing home fire safety in the USA: successes and opportunities. Inj Prev J Int Soc Child Adolesc Inj Prev 24:i7–i13. https://doi.org/10.1136/injuryprev-2017-042356
79. Istre GR, McCoy MA, Moore BJ et al (2014) Preventing deaths and injuries from house fires: an outcome evaluation of a community-based smoke alarm installation programme. Inj Prev J Int Soc Child Adolesc Inj Prev 20:97–102. https://doi.org/10.1136/injuryprev-2013-040823

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