

the lung.<sup>49</sup> At very high concentrations, smoke deposits may physically clog the airways. This could occur even with biologically inert particles at concentrations in excess of 5 mg/L, and is more probable with irritant smoke particles that are likely to acutely inflame tissues. Apart from the toxic effects of these particles on the lung, they may also be important in increasing the thermal capacity of the smoke and increasing the likelihood of lung burns (see next section of this chapter).

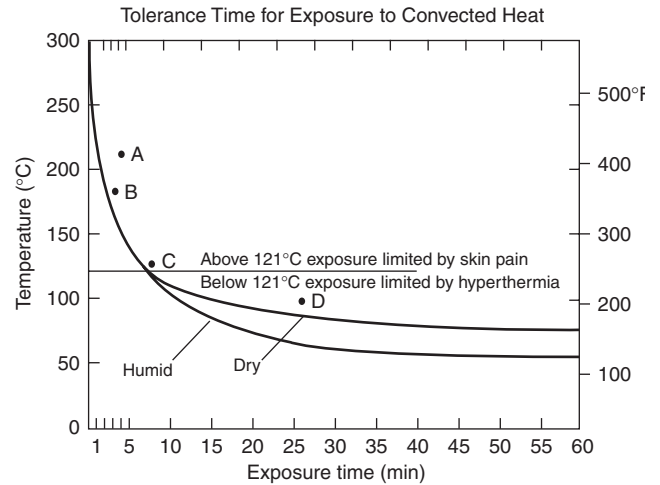
### The Exposure of Fire Victims to Heat

There are three basic ways in which exposure of fire victims to heat may lead to incapacitation and death: by (1) heat stroke, (2) body surface burns, and (3) respiratory tract burns.

#### Heat Stroke (Hyperthermia)

If a subject is exposed to a hot environment, especially if the humidity is high and the subject is active, there is a danger of incapacitation and death due to hyperthermia. The time to effect and the type of hyperthermia depend principally upon the heat flux to which the subject is exposed, and are greatly affected by factors such as the amount and type of clothing and degree of work performed. A detailed analysis of the parameters that determine heat transfer to subjects over a range of environmental conditions and levels of activity, and the protective effects of different types of clothing, is given by Berenson and Robertson,<sup>111</sup> and Simms and Hinkley.<sup>112</sup>

Simple hyperthermia involves prolonged exposure (approximately 15 minutes or more) to heated environments at ambient temperatures too low to cause burns. Under such conditions, where the air temperature is less than approximately 120°C for dry air or 80°C for saturated air, the main effect is a gradual increase in the body core temperature.<sup>113</sup> Increases above the normal core temperature of 37°C up to approximately 39°C are within the physiological range and can occur at normal ambient temperatures during hard exercise, but once 40°C is reached consciousness becomes blurred and the subject becomes seriously ill. Further increase causes irreversible damage, with temperatures above 42.5°C being fatal unless treated within minutes.<sup>111,114</sup> The time taken to reach such a state depends upon a number of variables including those mentioned. Figure 2-6.26, adapted from Block-



**Figure 2-6.26. Thermal tolerance for humans at rest, naked skin exposed, with low air movement (less than 30 m/min). Adapted from Blockley.<sup>113</sup> See text and Table 2-6.16 for discussion of data points A to D.<sup>112,115,116</sup>**

ley,<sup>80</sup> shows approximate tolerance times for unclothed subjects at rest, under conditions of low air movement (30 m/min). At temperatures below 120°C tolerance is limited by hyperthermia, whereas above this temperature pain followed by burns become important. The data points A to D (for clothed subjects) were taken from various authors, and are added for comparison. At temperatures below 120°C evaporative cooling from sweat is important, so that humidity has a considerable influence on tolerance time. Clothing, therefore, offers some immediate protection at temperatures above 120°C, but at lower temperatures may reduce tolerance time by impeding heat loss due to evaporative cooling. Details of the data points and authorship are given in Table 2-6.16.

Experiments conducted with pigs by Moritz et al.<sup>118</sup> confirm the basic signs of hyperthermia, with death occurring principally due to circulatory collapse associated with severe cardiac irregularities (ventricular tachycardia).

A second situation described by Moritz et al.<sup>118</sup> involves exposure to high temperatures for short periods (less than 15 minutes), and here hyperthermia is accompanied by cutaneous burns (in pigs at temperatures above 120°C). When deaths occurred soon after exposure to severe heat (within 30 minutes) the cause was considered to be due not to burns but to a rise in blood temperature. In

**Table 2-6.16 Reported Tolerance Times for Exposures to Hot Air**

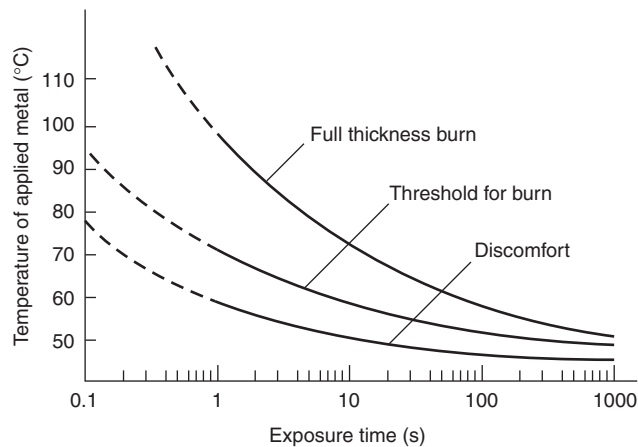
Temperature (°C)	Time (min)	Reference	Letter in Figure 2.6.26
<b>Dry air</b>			
110	25	112	D
180	3	112	B
205	4	115	A
126	7	116	C
<b>Humid air</b>			
32 at 100% RH	32	117	Men working

this situation the exposure duration was insufficient to raise the body core temperature greatly, but if the temperature of the blood in the heart reached 42.5°C, the animal died within a few minutes from circulatory collapse.

It therefore seems that a victim exposed for more than a few minutes to high temperatures and heat fluxes (exceeding 120°C) in a fire is likely to suffer burns and die either during or immediately after exposure, due principally to hyperthermia. Victims surviving the hyperthermia phase may die later due to burns of the upper respiratory tract, particularly the larynx, or due to the secondary effects of skin burns. A victim or fire fighter exposed to temperatures unlikely to cause burns (less than 120°C) may also suffer heat stroke after a prolonged exposure (exceeding 15 min), especially if the humidity is high and the person is working hard.

**Skin Burns**

According to Buettner,<sup>119</sup> pain from the application of heat to the skin occurs when the skin temperature at a depth of 0.1 mm reaches 44.8°C, which agrees with the finding of Lawrence and Bull<sup>120</sup> that discomfort was experienced when the interface between a hot handle and the skin of the hand reached 43°C. The sensation of pain is followed soon afterward by burns, causing incapacitation, severe injury, or death depending upon their severity. The time from the application of heat to the sensation of pain, and from pain to the occurrence of burns of various degrees of severity, depends upon the temperature, or more properly, the heat flux to which the skin is exposed. The effects of heating the skin are essentially the same whether the heat is supplied by conduction from a hot body, convection from air contact, or by direct radiation.<sup>119,121</sup> Curves for the relationship between time and effect have been published for conducted heat from a "hot handle"<sup>121</sup> and radiant heat. The relationship between time and effect is exponential. (See Figure 2-6.27.) Thus for conduction from heated metal at 60°C, pain occurs af-



**Figure 2-6.27.** The relation between time and the temperature of metal to cause thermal injury to skin (values below 1 s are extrapolated).<sup>121</sup>

ter 1 s and a burn after 10 s, while at 80°C pain occurs at 100 ms and a burn after 1 s contact.

Pain therefore occurs when the difference between the rate of supply of heat to the skin surface exceeds the rate at which heat is conducted away by an amount sufficient to raise the skin temperature to 44.8°C. The thermal inertia of human skin is similar to that of water<sup>119</sup> or wood<sup>121</sup> with a value of  $k\rho c$  for the surface (depth 0.1 mm) of 1.05 W/m·K. For the skin surface the rate of heat removal is not considered to be affected by blood supply<sup>119</sup> except for the fingertips, where blood flow may be sufficient to remove a significant amount of heat.<sup>119</sup> However, blood supply may have some effect on the occurrence of burns, especially to the deeper layers of the skin.<sup>121</sup> Obviously, rates of heating and the occurrence of pain and burns are greatly affected by the extent and type of clothing,<sup>112,118,122</sup> but only effects on naked skin are considered here. The temperature increase of the skin for the situation in which constant radiant heat is absorbed by the upper surface of the skin, or heat from a hot air current is applied to the skin, may be calculated as follows:<sup>119</sup>

$$T - T_0 = \frac{2Q\sqrt{t}}{\sqrt{\pi k\rho c}} \tag{25}$$

where

- $T$  = final temperature of skin at 0.1 mm depth
- $T_0$  = starting temperature of skin at 0.1 mm depth
- $Q$  = heat supply (W/m<sup>2</sup>)
- $k\rho c$  = 1.05 W/m·k
- $t$  = time (s)

**Conducted heat:** The effect of conducted heat is related to the temperature of the hot object and its thermal inertia, depending upon the interface temperature between the object and the body tissue at the skin surface,<sup>121,122</sup> as illustrated by the examples in Table 2-6.17.

A skin temperature of 43°C causes pain and some cellular damage, while a temperature of 60°C coagulates tissue protein. A brass block heated to 60°C will produce a partial thickness skin burn within 10 s, pain within 1 s and a full thickness burn after approximately 100 s.<sup>121</sup> The time/temperature relationships for these effects of conducted heat are shown in Figure 2-6.27.

**Convected heat:** For a victim attempting to escape from a fire, the most important sources of heat exposure are ra-

**Table 2-6.17** Theoretical Contact Temperatures between Skin at 35°C and a Selection of Hot Bodies at 100°C<sup>121</sup>

Material of Hot Body	Contact Temperature (°C)
Mild steel	98
Glass	82
Wood	65
Cork	46

diation from hot areas and convection from hot gases. Pain and the likelihood of skin burns occur at air temperatures above approximately 120°C. The rate of heat transfer from hot air to the skin depends upon the rate of ventilation, humidity, and the protective value of clothing as well as air temperature. The effects of these parameters are described by Berenson and Robertson,<sup>111</sup> and Simms and Hinkley.<sup>112</sup> However, for unprotected areas such as the head, data on naked skin are relevant, and the data shown in Figure 2-6.26 for temperatures above 120°C provide limits for tolerance to the painful effects of contact with hot air.

Apart from the problem of hyperthermia, dry air has been tolerated by humans as shown in Table 2-6.16. Moritz et al.<sup>118</sup> state that dry air at 300°C injured unprotected skin within 30 s in pigs and dogs. Pigs also suffered burns at 150°C after 100 s and after 400 s at 100°C. However, it was considered that humans would be more resistant to burns, especially at temperatures below 120°C, due to the protective effect of sweating. Air with a high level of humidity not only reduces or prevents heat loss through sweat, but also delivers more heat to the skin. Thus Moritz et al.<sup>123</sup> found that steam at 100°C destroyed the epidermis of dogs within a few seconds.

Figure 2-6.26 shows curves for tolerance time of convected heat for both dry and humid air. A search for the original data used to produce these curves has not been successful, but upon careful consideration it seems likely that the humid curve must represent air that was humid (perhaps saturated) at room temperature, which was then heated subsequently, and was therefore nowhere near saturated with water vapor at higher temperatures. This must be the case because the capacity of air for water vapor increases dramatically at temperatures above 60°C, so that the amount of deliverable latent heat also increases. In practice, 60°C has been found to be the highest temperature at which 100 percent water-vapor saturated air can be breathed. Since all fires produce a considerable amount of water from combustion, it is possible that the presence of water vapor may be an important neglected hazard in fires.

**Radiant heat:** For radiant heat, clothing also greatly influences tolerance times, but again, data on naked skin are relevant to exposure of unprotected areas such as the head. Figure 2-6.28,<sup>111</sup> shows the relationship between time to skin pain and radiant heat flux. Data points A to E, taken from a number of authors (detailed in Table 2-6.18) have been added for comparison. Points B through E agree with the curve presented by Berenson, but data from one source (Perkins et al.)<sup>124</sup> (points labeled A) deviate somewhat from the rest. From Perkins's data (which were produced by experiments where thermal injury was caused by exposing subjects to radiant heat from a searchlight), the heat fluxes for erythema (reddening of the skin said to coincide with pain<sup>121</sup>) appear rather higher than the heat flux limits for pain supplied by the other authors. This is possibly due to differences in the wavelength, and thus degree of penetration, of the radiation.<sup>119</sup> The searchlight data do, however, show the relationship between time to erythema, time to partial skin burn, and time to

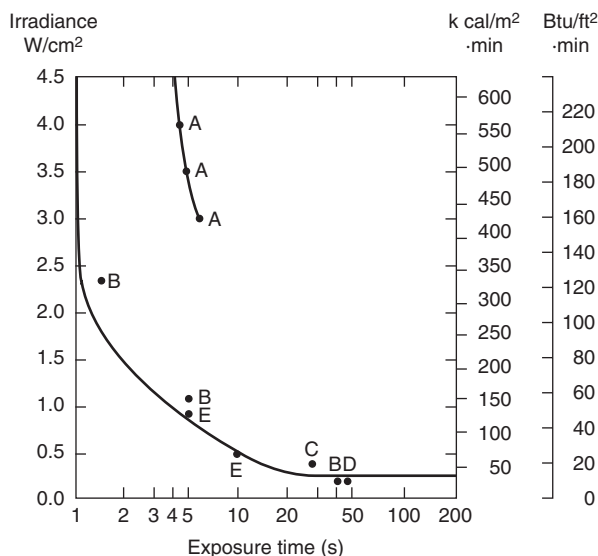


Figure 2-6.28. Time to severe skin pain from radiant heat. Adapted from Berenson and Robertson.<sup>111</sup> See text and Table 2-6.18 for discussion of data points A to E.<sup>56,112,115,118,124,125</sup>

Table 2-6.18 Data on the Effects of Exposure to Radiant Heat

Reference Source	Heat Flux W/cm <sup>2</sup>	Time to Effect(s)			Letter in Figure 2-6.28
		Erythema (or pain)	Burn	Full Burn	
Perkins et al. <sup>124</sup>	15	1	2.5	4	
	10	2	4	6	
	5	4	7	>15	
	4	4.5	9	>15	A
	3.5	5	9.5	>15	A
Buettner <sup>119</sup>	3	6	10	>15	A
	2.35	1.6			B
	1.05	5			B
Veghte <sup>115</sup>	0.25	40			B
	0.42		Blisters 30		C
Simms and Hinkley <sup>112</sup>	0.126	Unbearable pain 600			
	0.252	30 to 60			D
Dinman <sup>125</sup>	0.24	Lower limit for pain after a long period			
	0.82	5			E
	0.48	10			E
Berenson and Robertson <sup>111</sup>	0.34	Limit for blood to carry away heat			
Babrauskas <sup>56</sup>	0.25	Tenability limit			

full thickness skin burn. The shape of the radiant heat tolerance curve suggests a fairly obvious tolerance limit for exposure to radiant heat of 0.25 W/cm<sup>2</sup> (2.5 kW/m<sup>2</sup>) which is that suggested by Babrauskas.<sup>56</sup>

**Consequences of body surface burns:** Apart from the immediate pain caused by exposure to heat and by skin burns, as well as the accompanying psychological shock and fear, incapacitation may result from body surface burns during or after a fire due to physiological shock. In this situation loss of body fluids into the burn results in circulatory failure and a fall in blood pressure, which may lead to collapse and even loss of consciousness.<sup>14,118</sup> The immediate effect of burns and the later chances of recovery depend upon a number of factors such as the site and extent of the burn, the depth of the burn, the age of the victim, and the treatment received.<sup>121</sup> While victims may continue to function for some time with severe burns, and survivals have occurred with up to 80 percent body surface area burns,<sup>121</sup> in general, if 35 percent or more of the body surface area is burned the chances of survival are low. Young adults generally have the best chance of survival, with a 50 percent chance of surviving a 50 percent body surface area burn, while children and old people are the most vulnerable, with a 50 percent chance of surviving a 20 percent body surface area burn in the elderly.<sup>14,121</sup> The depth of burn is classified on a scale of six degrees. First-, second-, and third-degree burns involve damage to the skin from which it can recover, while fourth-degree burns require skin grafts. Fifth- and sixth-degree burns involve destruction of muscle and/or bones, respectively.<sup>14</sup> Another scheme classifies burns as partial thickness skin burns, which will heal, or full thickness burns, which require grafts.<sup>121</sup>

If the victim survives the initial period of shock, death may occur over a period of up to a few weeks due to secondary effects on the brain, heart, lungs, liver, and kidneys.<sup>14</sup> The most common secondary effect and cause of death involves the lungs,<sup>14,126-128</sup> consisting of pulmonary edema resulting from effects on the circulatory system secondary to shock and metabolic acidosis. Post-exposure treatment to replace body fluids and control acidosis are important in improving the prognosis for survival. If the victim survives the respiratory distress resulting from edema during the first week after exposure, pneumonia may then develop as a further, possibly fatal complication.<sup>14,129-131</sup>

This fatal damage to the lungs may occur following body surface burns when there has been no inhalation of heat or toxic gases. In many fire victims, however, damage to the respiratory tract and lungs results from a combination of all three causes.<sup>14,129-131</sup>

### Thermal Damage to the Respiratory Tract

Thermal (as opposed to chemical) burns to the respiratory tract never occur in the absence of burns to the skin of the face.<sup>81,131</sup> Heat damage to the respiratory tract is even more dependent upon the humidity of inhaled hot gases than are skin burns. As a result of the low thermal capacity of dry air and the large surface area of the airways, which are lined with a wet surface and good blood supply, thermal burns are not induced by dry air below the top of the trachea. However, steam at around 100°C is capable of causing severe burns to the entire respiratory tract down to the deep lung, due to its higher thermal capacity and the latent heat released during condensation. These effects of inhaled hot gases are demonstrated by the

work of Moritz et al.<sup>118</sup> in which anesthetized dogs and pigs breathed hot air, flame from a burner, or steam, supplied through a cannula to the larynx. Dry air at 350°C and flame from a blast burner at 500°C caused damage to the larynx and trachea, but had no effect on the lung, while steam at 100°C caused burns at all levels. In these experiments the most important site of damage was the larynx, and death resulted from obstructive edema of the laryngopharynx within a few hours of exposure. This work was taken further by Zikria<sup>131</sup> using steam burns in dogs, induced by a 15 s application of steam at 100°C via an endotracheal tube. The animals survived the initial effects and a number of phases of reaction were observed. The first phase consisted of necrosis and edema in the tracheobronchial airway, and early lung parenchymal edema within one hour. This was followed by increasing parenchymal edema, sloughing of the mucosa, and collapse of lung segments. The next phase after 24 hours consisted of bronchopneumonia behind respiratory tract obstructions.

All these features occur in fire victims, but it is difficult to separate the effects of thermal inhalation burns from edema and inflammation due to burns caused by irritant chemical smoke products, or edema secondary to body surface burns, all of which may be involved.<sup>14,129-131</sup> Thus fire victims with facial burns subjected to endoscopy have been found to have burns well into the respiratory tract.<sup>81</sup> If these lesions are caused by heat, it would imply that fire atmospheres resemble steam rather than dry air in terms of their thermal capacity. However, it is possible that such lesions are caused by chemical smoke irritants, which have been shown to produce fatal pulmonary edema and inflammation in the absence of heat.<sup>80</sup> Unfortunately, data on the thermal capacity and latent heat of actual fire atmospheres are not readily available, although it may be possible to calculate probable values from a knowledge of fire atmosphere temperature and composition.

The situation is therefore complicated, but from a fire engineering standpoint a number of basic points may be useful.

1. Thermal burns to the respiratory tract will not occur unless the air temperature and/or humidity are sufficient to cause facial skin burns.
2. Dry air at around 300°C may cause burns at the larynx after a few minutes. This may result in life-threatening obstructive edema of the larynx within an hour if not treated, although damage to the deeper structures of the lung is unlikely. It is possible that such laryngeal burns may occur at lower temperatures down to approximately 120°C depending upon the duration of exposure, and breathing dry air at these temperatures would be painful. Laryngeal burns followed by obstructive edema are common findings in fire victims, and are important causes of incapacitation and death during and immediately after fires.<sup>14,129-131</sup>
3. Humid air, steam, or smoke with a high thermal capacity or latent heat (due to vapor content or suspended liquid or solid particles) may be dangerous at temperatures of around 100°C, causing burns throughout the respiratory tract. It may be possible to predict the likely effects of hot-smoke atmospheres if thermal capacity or latent heat were measured.

4. In practice, fire victims may be affected by the inhalation of chemically irritant smoke, by hot humid gases, and by the secondary effects on the lung of body surface burns, all of which may combine to cause fatal respiratory tract lesions during the hours, days, or weeks following the fire exposure. However, these effects are probably less likely to be fatal during exposure to the fire atmosphere over periods of less than 30 minutes.
5. Heat flux and temperature tenability limits designed to protect victims from incapacitation by skin burns should be adequate to protect them from burns to the respiratory tract.

**Model of the Prediction of Time to Incapacitation by Exposure to Heat in Fires**

There are three basic ways in which exposure to heat may lead to incapacitation: through heat stroke (hyperthermia), skin pain and burns, or respiratory tract burns. Thermal burns to the respiratory tract from air containing less than 10% by volume water vapor do not occur in the absence of burns to facial skin. Therefore, tenability limits with regard to skin pain and burns are normally lower than for thermal burns to the respiratory tract. Thermal burns to the respiratory tract may occur upon inhalation of air above only 60°C when saturated with water vapor, as may occur when water is used for fire extinguishment.

The tenability limit for exposure of skin to radiant heat is approximately 2.5 kW/m<sup>2</sup>, below which exposure can be tolerated for at least several minutes. Radiant heat at this level and above causes skin pain followed by burns within a few seconds, but lower fluxes can be tolerated for more than 5 min. For situations where occupants are required to pass under a hot smoke layer in order to escape, this radiant flux corresponds approximately to a hot layer temperature of 200°C. Above this threshold, time (minutes) to incapacitation due to radiant heat  $t_{\text{Irad}}$  at a radiant flux of  $q$  kW/m<sup>2</sup>, is given by Equation 26.<sup>132,133</sup>

$$t_{\text{Irad}} = \frac{133}{q^{1.33}} \quad (26)$$

The effects of heat on an occupant response may depend upon the situation. The threshold for pain occurs at approximately 1.333–1.667 (kW·m<sup>-2</sup>)<sup>4/3</sup> min, second degree burns at 4–12.17 (kW·m<sup>-2</sup>)<sup>4/3</sup> min and third degree burns at approximately 16.667 (kW·m<sup>-2</sup>)<sup>4/3</sup> min. Radiant heat tends to be directional in fires, so that the main problem tends to be local heating of particular areas of skin. The air temperature, and hence that of the air breathed and that in contact with other parts of the body, may be relatively low, even when the radiant flux is high. For this reason the main hazard is pain and burns to the skin, rather than hyperthermia. Skin temperature depends upon the relationship between the rate of heat supply to the skin surface and the removal of heat from inner layers by the blood. There is, therefore, a threshold radiant flux below which significant heating of the skin is prevented, but above which rapid heating of the skin occurs.

For exposures of up to two hours to convected heat from air containing less than 10% by volume of water vapor, the time (min) to incapacitation  $t_{\text{Iconv}}$  at a temperature

$T$  (°C) is calculated from Equation 27, which is derived from Figure 2-6.26.

$$t_{\text{Iconv}} = 5 \times 10^7 T^{-3.4} \quad (27)$$

As with toxic gases, the body of a fire victim may be regarded as acquiring a “dose” of heat over a period of time during exposure, with short exposure to a high radiant flux or temperature being more incapacitating than a longer exposure to a lower temperature or flux. The same fractional incapacitating dose model as with the toxic gases may be applied and, providing that the temperature in the fire is stable or increasing, the fractional dose of heat acquired during exposure can be calculated by summing the radiant and convected fractions using Equation 28:

$$\text{FED} = \sum_{t_1}^{t_2} \left( \frac{1}{t_{\text{Irad}}} + \frac{1}{t_{\text{Iconv}}} \right) \Delta t \quad (28)$$

*Note:*  $t_{\text{Irad}}$  will tend to zero as  $q$  tends to <2.5 kW/m<sup>2</sup>

Thermal tolerance data for unprotected skin of humans suggest a limit of about 120°C for convected heat, above which considerable pain is quickly incurred along with the production of burns within a few minutes. Depending upon the length of exposure, convective heat below this temperature may still result in incapacitation due to hyperthermia. Examples of tolerance times to different radiant fluxes and air temperatures are shown in Table 2-6.19. Conducted heat is physiologically important only when skin is in contact with hot surfaces, such as door handles. A 1-s contact with metal at 60°C can cause burns.

**Example of a Calculation of Time to Incapacitation for Physical Fire Parameters and Irritancy**

In a previous section the single armchair room burn shown in Figure 2-6.15 was used to illustrate how the model for prediction of asphyxiation could be applied to a practical fire scenario. To complete the incapacitation model it is necessary to include calculations for the effects of physical parameters (heat, smoke optical density), and mass loss concentration as an indication of irritancy. The curves for radiant heat, air temperature, smoke extinction

**Table 2-6.19 Limiting Conditions for Tenability Caused by Heat<sup>106</sup>**

Mode of Heat Transfer	Intensity	Tolerance Time
Radiation	<2.5 kW·m <sup>-2</sup>	>5 min
	2.5 kW·m <sup>-2</sup>	30 s
	10 kW·m <sup>-2</sup>	4 s
Convection	<60°C 100% saturated	>30 min
	100°C <10% H <sub>2</sub> O <sup>a</sup>	12 min
	120°C <10% H <sub>2</sub> O	7 min
	140°C <10% H <sub>2</sub> O	4 min
	160°C <10% H <sub>2</sub> O	2 min
	180°C <10% H <sub>2</sub> O	1 min

<sup>a</sup>v/v

coefficient, and mass loss during the first ten minutes of the armchair burn are shown in Figure 2-6.29.

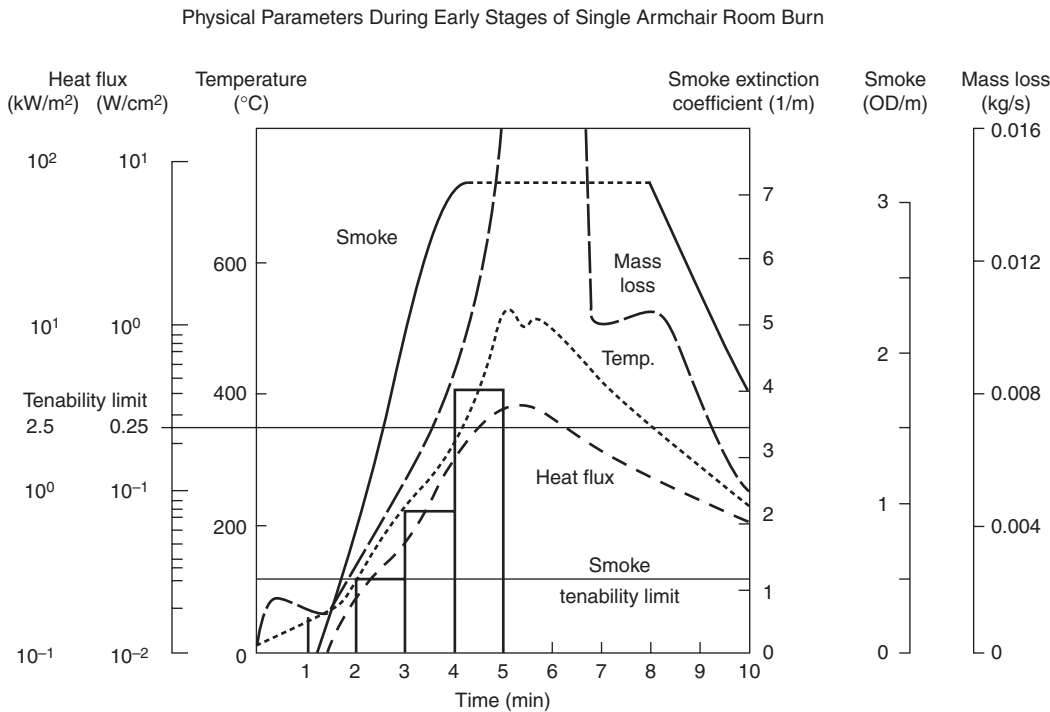
Of the physical factors likely to affect a victim during the fire exposure, the majority are basically concentration or intensity-related rather than dose-related, and for these factors tenability limits have been set (radiant heat, smoke optical density, and sensory irritancy). The other two factors, convected heat and lung irritancy, are primarily dose-related, but lung irritant effects are likely to be relatively minor until after exposure. This leaves the fractional incapacitating dose of convected heat to be calculated. The average temperatures per minute during the first minutes of the fire are shown as histograms in Figure 2-6.29 with Table 2-6.20 showing the fractional incapacitating dose calculation.

**Convected heat:** The effects of exposure to convected heat increase dramatically in this type of fire as shown in Figure 2-6.29. Incapacitation, mainly due to skin pain and burns, is predicted sometime during the fourth minute, when the air temperature is 220°C. The situation then rapidly worsens, and it would seem likely that severe and probably fatal burns or fatal hyperthermia would be sustained by any victim remaining in the fire during the fifth minute. Even if the victims were protected to some extent by clothing, they would sustain burns to the face and probably fatal burns to the larynx. The occurrence of lung

burns would depend on the thermal capacity (principally the latent heat) of the smoke.

**Radiant heat:** From Figure 2-6.29, it would seem that the effects of radiant heat would be relatively minor in this fire compared to the effects of convected heat. The radiant heat peaks at just above 3kW/m<sup>2</sup> during the sixth minute, and therefore just exceeds the tenability limit. Nevertheless, the radiation alone would probably be sufficient to cause some burns and seriously inhibit escape during the sixth minute, and there would almost certainly be some degree of additive effect with convected heat.

**Smoke:** From the point of view of its obscurational effects, incapacitation by smoke is concentration-related rather than dose-related. For this series of chair burns, Babrauskas sets a tenability limit of extinction coefficient 1.2/m (OD/m 0.52).<sup>56</sup> This would give approximately 2 m visibility, which should be adequate for escaping from a room, and could be used as a tenability limit for input into the model. Incapacitation due to visual obscuration would occur at the end of the second minute. The smoke curve is rising very steeply at this point, with an OD/m of 1 at the beginning of the third minute. Escape would therefore become extremely difficult, and certainly slow during the third minute, unless the victim was fa-



**Figure 2-6.29.** Profiles for heat (radiant flux and temperature), smoke and mass loss rate during the first 10 min of a single armchair (polystyrene, with polyurethane cushions and covers) room burn.<sup>56</sup> (Expanded detail from Figure 2-6.15.) Histogram shows average temperature each minute during the first five minutes.

**Table 2-6.20** Calculation of Fractional Incapacitating Accumulation of Convected Heat for the Single Armchair Room Burn Data Shown in Figure 2-6.29. (Calculated According to Equation 34.)

Time (min)	1	2	3	4	5	6
Average temp. (°C)	20	65	125	220	405	510
$F_{th}$	0	0.033	0.170	2.273	355	6236
Cumulative $F_{th}$			0.203	2.476		

Incapacitation occurs when  $F_{th} = 1$ , and is therefore predicted during the fourth minute of exposure.

miliar with the surroundings and able to find the exit in the dark.

**Irritancy:** As stated in the section of this chapter on irritancy, there are two factors to consider: the immediate incapacitation due to the painful effects of sensory irritation of the eyes and respiratory tract, adding to the obscurational effects of smoke and disrupting escape behavior, and the later inflammatory effects on the lung which may cause death after exposure.

The first consideration is whether the victim would be able to escape from the fire. In this context, sensory irritation is the most important. This is concentration-related; to predict the irritancy of the smoke, it is necessary to know the  $RD_{50}$  concentration of the atmosphere produced by the materials involved under the particular decomposition conditions existing in the fire. Most importantly, it is necessary to know the concentration/time profile of the fire products in terms of mass loss per liter of air (NAC mass loss). Although the mass loss curve for the armchair is shown in Figure 2-6.29, there are no data on the volume of air into which this mass was dispersed during the fire; so for the purposes of this example it will be necessary to make an estimate of possible mass loss concentration. Also, since the  $RD_{50}$  of the polyurethane and polystyrene components of the chair under flaming conditions are unknown, it will be necessary to use estimated values.

In the discussion of irritancy, a general tenability limit for severe sensory irritation was set at a concentration of 1 mg/L NAC mass loss, and an incapacitating dose for serious postexposure lung inflammation was set at 10 mg/L NAC mass loss for 30 minutes (a  $Ct$  product of 300 mg·min/L). From the general conditions, the smoke curve, and the CO concentration curve, it is estimated that the tenability limit for sensory irritancy would be exceeded during the third minute, greatly adding to the deleterious effects of smoke on vision and escape behavior.

With regard to lung irritation, it is estimated that the average mass loss concentration over the first five minutes of the fire would be approximately 10 mg/L. If so, this would represent a fractional incapacitating dose of 50 mg·min/L, which would probably be insufficient to cause significant lung damage after exposure, compared

to the more serious effects of heat exposure. However, if the average mass loss concentration over the first five minutes should reach 60 mg/L, serious effects on the lung would likely occur after and probably during exposure.

**Interactions:** In terms of physiological effects, it is likely that there would be some degree of interaction between asphyxia and several of these physical factors, but it is likely that most would be relatively minor during the fire, except for some possible enhancement of pulmonary irritation due to the hyperventilatory effect of  $CO_2$  during the fourth to sixth minute of the fire. A reasonable model can be used in which asphyxia, sensory irritancy, and the effects of heat and visual obscuration can be treated separately. Interactions may be more important at the behavioral level. The interaction between sensory irritation and visual obscuration has been mentioned and there is some experimental evidence for such an interaction in humans.<sup>86</sup> After exposure, as mentioned in the section on heat, the effects of skin burns, respiratory tract burns, and chemical irritation (and even possibly CO asphyxia) all combine to increase the probability of fatal pulmonary edema and inflammation.

**Summary:** From the analyses performed, the effects on a victim exposed to the conditions in the armchair room burn (Figure 2-6.15) are predicted as follows:

1. Toward the end of the second minute and beginning of the third minute, the smoke optical density and mass loss/liter would sufficiently exceed the tenability limits for visual obscuration and sensory irritancy to severely inhibit escape from the room.
2. During the fourth minute, the average temperature was 220°C, and sufficient heat would be accumulated in the skin surface to cause skin burns resulting in incapacitation.
3. During the fifth minute, a victim is likely to lose consciousness due to the combined effects of the accumulated doses of asphyxiant gases.
4. It is predicted that a victim escaping or rescued after the fourth minute would suffer severe postexposure effects due to skin burns, plus pulmonary edema and inflammation which might well be fatal (due to the combined effects of inhaled hot gases, chemical irritants, and the pulmonary secondary effects of skin burns). After the sixth minute, it is likely that a rescued victim would die at some time between a few minutes and one hour due to the effects of asphyxia and circulatory shock.

It is unlikely that an otherwise healthy adult would be able to escape from a fire such as this if he or she remained longer than three minutes after ignition. However, three minutes is a long time in which to leave a room, so that providing the victim is awake and aware of the fire, is not otherwise incapacitated, and does not stay after two minutes in an attempt to fight the fire or rescue belongings, it is likely that he or she would be able to escape without serious injury. In the next section, data on real fire victims is examined in an attempt to relate fire conditions to actual injury and death statistics.