Modelling Toxic and Physical Hazard in Fire

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ABSTRACT

Studies of incapacitation and lethality in laboratory animals and human fire victims, resulting from exposure to thermal decomposition products from many materials, indicate two main mechanisms of toxicity, narcosis (asphyxia) and irritancy. Narcotic effects are caused by the common asphyiant gases, CO, HCN, low O_2, and CO_2 and can be predicted to a reasonable degree with existing knowledge. Irritant effects are caused by a variety of combustion products some of which are unknown, but can be quantified from small scale rodent combustion toxicity tests in terms of the mass loss concentration of combustion products (RD_{50} and LC_{50} in mg.min/liter). A mathematical model is presented for estimating toxic and physical hazard in fire in terms of time to incapacitation or death. The model takes the concentration/time profiles of the above products, smoke optical density, temperature and radiant heat flux (derived from other mathematical models or large scale fire tests) and calculates time to incapacitation using a Fractional Effective Dose method based upon the known toxic effects of the combustion products, and of the physical hazards, in man, primates and rodents.

INTRODUCTION

Since the major toxic effects in fires are caused by a small number of common products [1,2,3,4], toxic and physical hazard in fires become predictable if two sets of information are available:

1. The time/concentration profiles of the important toxic fire products and physical hazards (carbon monoxide, hydrogen cyanide, carbon dioxide, oxygen vitiation, mass loss concentration of total fire products [for irritancy assessment], smoke optical density, radiant heat flux and air temperature).

2. The time/concentration/toxicity relationships of these parameters in man when they occur individually and in combination.

A mathematical model for predicting time to incapacitation or death for human fire victims has been developed based on the above criteria. The model, summarized here, is to be published in the SFPE Handbook of Fire Protection Engineering [5].

TOXIC AND PHYSICAL HAZARDS IN FIRES

Combustion products cause incapacitation and death in fires by two main toxic mechanisms - narcosis and irritancy [2]. Narcotic gases (CO, HCN, CO_2, low oxygen) cause loss of consciousness followed ultimately by death from asphyxiation. Irritant fire products cause immediate painful sensory stimulation of the eyes, nose, throat and lungs; they also cause lung inflammation and oedema which may lead to death due to impariment of respiration, usually a few hours after exposure. Visual obscuration by smoke

reduces escape efficiency or renders a victim uwilling to enter a smoke-filled escape route, while heat initially hinders or prevents escape due to skin pain and burns or hyperthermia, and may cause death either during or after exposure.

CONCENTRATION/TIME/DOSE RELATIONSHIPS

For sensory irritation and visual obscuration by smoke, the effects occur immediately on exposure and the hazard depends upon the concentration. This also applies to some extent to radiant heat, where incapacitation due to skin pain and burns occurs very rapidly at intensities above 0.25 w/cm^2. Therefore for these parameters tenability limits have been set above which it is considered that serious incapacitation is liklely. For all other toxic effects except sensory irritation and for convected heat, effects are related to the dose received rather than the exposure concentration. This can be quantified approximately as the product of concentration (c) and exposure time (t), and in general concentration and time are approximately equivalent so that $c \times t = k$ (equation 1) where k is a constant dose required for any given toxic effect. The time during the fire when the victim will have received an incapacitating or fatal ct dose of products can be calculated by integrating the area under the concentration/time curve for the toxic product in the fire until the integral is equal to the known incapacitating or lethal dose. In practice the ct product doses for small periods of time during the fire are divided by the ct product dose causing a toxic effect (such as lethality), and these Fractional Effective Doses are then summed during the exposure until the fraction reaches unity, when the toxic effect is predicted to occur. Thus (equation 2):

$$\text{Fractional Effective Dose} = \frac{\text{dose recived at time t (ct)}}{\text{effective ct dose to cause incapacitation or death}}$$

For some substances the denominator of the equation is a constant for any particular toxic effect, but for most fire gases the dose relationships deviate significantly from ideal behaviour, in that for high concentrations a lower dose is required to cause incapacitation than for low concentrations. For the model developed here the denominator is presented in the form of equations giving the required ct product doses predicted for man, which have been derived for each toxic gas [5], and are presented in the following sections.

TOXIC AND PHYSICAL HAZARD ASSESSMENT MODEL BASED ON FRACTIONAL INCAPACITATING DOSES OF KNOWN TOXIC PRODUCTS AND PHYSICAL HAZARDS

NARCOSIS

In order to determine when a victim is likely to become incapacitated by loss of consciousness due to the effects of narcotic gases, it is neccessary to calculate the fractional incapacitating doses of each gas (CO, HCN, hypoxia, CO_2) individually, and the interactions between them, for each successive minute of the fire. The principle of Fractional Effective Dose can be applied to all the gases causing narcosis and has been validated in rodent experiments for combinations of CO and HCN [6].

CARBON MONOXIDE

Toxicity from carbon monoxide depends upon the dose accumulated in the blood as carboxyhaemoglobin (COHb). The Fractional Dose of CO available each minute during the fire is therefore best represented by the ratio of the COHb concentration at time t with the COHb concentration known to cause incapacitation or death, rather than by simple ct product ratios. The carboxyhaemoglobin concentration resulting from any given exposure to CO (for periods of up to one hour), can be calculated approximately using the Stewart equation [7].

$$\%COHb = (3.317 \times 10^{-5})(ppm\ CO^{1.036})(RMV)(t) \qquad \text{(equation 3)}$$

where: CO = CO concentration (ppm)
RMV = volume of air breathed each minute (l/min)
t = exposure time (min)

Using this equation it is now possible to derive an expression for the fractional incapacitating dose of CO for a 70 kg man engaged in light activity over periods of up to 1 hour. The numerator is the dose in the form of COHb acquired each minute for a given CO concentration, and the denominator is the dose causing incapacitation [8]. When the summed fractional doses during an exposure reach unity, loss of consciousness is predicted.

$$F_{Ico} = \frac{K(CO^{1.036})\ (t)}{D} \qquad \text{(equation 4)}$$

where: F_{Ico} = fraction of incapacitating dose
t^{Ico} = exposure time (min)(= 1 in this case)
K = 0.00082925 for 25 l/min RMV (light activity)
D = COHb concentration at incapacitation (30% for light activity)

HYDROGEN CYANIDE

Time to incapacitation by hydrogen cyanide depends partly on rate of uptake and partly on dose [9]. Below 80 ppm HCN only minor effects should occur over periods of up to 1 hour. From 80-180 ppm time to incapacitation (loss of consciousness) will be between 2 and 30 minutes according approximately to the relationship:

$$\text{Time to incapacitation } (t_{Icn}) \text{ (min)} = (185 - \text{ppm HCN})/\ 4.4 \qquad \text{(equation 5)}$$

For concentrations above approximately 180 ppm incapacitation will occur rapidly (0-2 minutes).

Since toxicity by HCN does not follow ideal behaviour the denominator of the fractional dose equation is not a constant, but depends partly upon the exposure concentration. A Fractional Incapacitating Dose equation for HCN based on ct product data has been derived from Equation 5 as follows:

$$F_{Icn} = \frac{1}{(185 - \text{ppm HCN})/\ 4.4} \qquad \text{(equation 6)}$$

LOW OXYGEN HYPOXIA

The time taken to achieve a blood oxygen concentration causing loss of consciousness depends on a 'dose' of hypoxia acquired as the blood oxygen concentration decreases in a subject exposed to a hypoxic atmosphere [10]. The relationship between oxygen concentration and time to incapacitation is shown in Figure 1. Hypoxia was induced by rapid decompression, which has similar effects to those of reduced oxygen concentrations at sea level. For input into the model an exponetial equation derived from these data of time to loss of consciousness is given by:

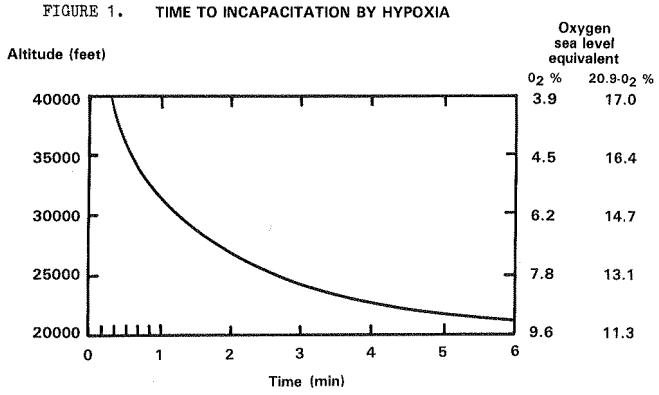
$$(t_{Io}) \text{ min} = e^{(8.13 - 0.54\ (20.9 - \%O_2))} \qquad \text{(equation 7)}$$

where $20.9 - \%O_2 = \%O_2$ Vit (% oxygen vitiation)

From this is derived the expression for fractional incapacitating dose:

$$F_{Io} = \frac{1}{e^{8.13 - 0.54\ (20.9 - \%O_2)}} \qquad \text{(equation 8)}$$

FIGURE 1. TIME TO INCAPACITATION BY HYPOXIA



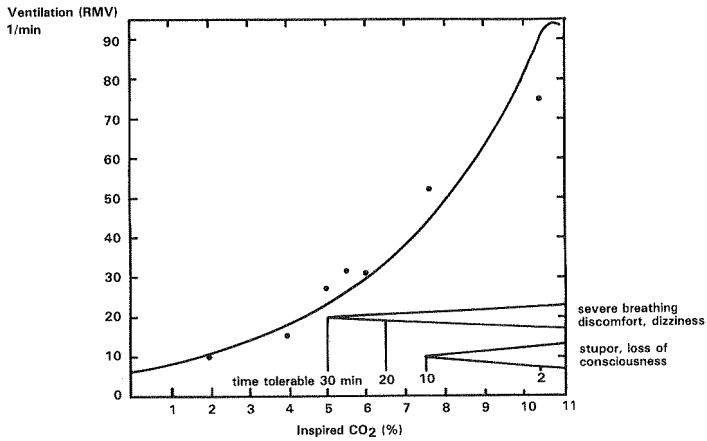
CARBON DIOXIDE

Carbon dioxide has two important effects: it greatly increases the respiratory minute volume (RMV), which will increase the rate of uptake of other toxic gases; and it is itself a narcotic [11].

It is therefore necessary to calculate a factor to allow for the effect of the increased RMV caused by carbon dioxide on the rate of uptake of other toxic gases. Figure 2 shows the relationship between carbon dioxide concentration and ventilation (RMV) constructed from data derived from a number of human exposure experiments [5]. The figure also gives an indication of the toxic effects of exposures to various concentrations for various times. The expression for the effect on ventilation derived from these data is is as follows:

$$\text{Multiplication Factor } CO_2 = e^{\frac{0.2496 \times \%CO_2 + 1.9086}{6.8}} \qquad \text{(equation 9)}$$

FIGURE 2. THE VENTILATORY RESPONSE TO CARBON DIOXIDE



It is also necessary to calculate the fractional incapacitating dose of carbon dioxide. From the data shown in the figure time to unconsciousness by carbon dioxide is given approximately by:

$$t_{Ico2} = e(6.1623 - 0.5189 \times \%CO_2) \qquad \text{(equation 10)}$$

From this is derived the fractional incapacitating dose expression:

$$F_{Ico2} = \frac{1}{e^{(6.1623 - 0.5189 \times \%CO_2)}} \qquad \text{(equation 12)}$$

INTERACTIONS BETWEEN NARCOTIC GASES

The data on the concentration/time/dose relationships of the dangerous and lethal narcotic effects in man of individual fire gases are adequate for the construction of a usable incapacitation model, but little information is currently available on effects of interactions between combinations of these gases on time to incapacitation in fires. The best that can be done currently is to suggest likely degrees of interaction based on physiological data from individual gases, and on the limited available data for gas combinations. Interactions between the different narcotic gases are discussed in detail in Purser [5]. From the available evidence, the following interactive relationships are used for the hazard model:

1. CO and HCN are considered to be directly additive [12,6].
2. CO_2 increases the rate of uptake of CO and HCN in proportion to its effect on the RMV.
3. The narcotic effect of low oxygen hypoxia is considered to be directly additive to the combined effects of CO and HCN, but not increased by CO_2 induced hyperventilation [5].
4. The narcotic effect of CO_2 is considered to act independently of the effect of the other gases.

On this basis a Fractional Incapacitating Dose equation for narcosis has been developed as follows:

$$\text{Total } F_{IN} = (F_{Ico} + F_{Icn}) \times VCO_2 + F_{Io} \text{ or } F_{Ico2} \qquad \text{(equation 12)}$$

Where;

F_{IN} = fraction of an incapacitating dose of all narcotic gases
F_{Ico} = fraction of an incapacitating dose of CO
F_{Icn} = fraction of an incapacitating dose of HCN

VCO_2 = multiplication factor for CO_2 induced hyperventilation
F_{Io} = fraction of an incapacitating dose of low oxygen hypoxia
F_{Ico2} = fraction of an incapacitating dose of CO_2

WORKED EXAMPLE

The following worked example uses data obtained from a large scale fire test (a single armchair room burn shown in Figures 3 and 5) [13]. The histograms in Figure 3 show the average concentrations of narcotic gases each minute for the first 6 minutes of the fire, which are shown numerically in Table I.

TABLE I. **Average concentrations of narcotic gases each minute during the first six minutes of the single armchair room burn**

Time (min)	1	2	3	4	5	6
CO ppm	0	0	500	2000	3500	6000
HCN ppm	0	0	0	75	125	175
CO_2 %	0	0	1.5	3.5	6	8
O_2 %	20.9	20.9	19	17.5	15	12

FIGURE 3. NARCOTIC GASES DURING EARLY STAGES OF SINGLE ARMCHAIR ROOM BURN

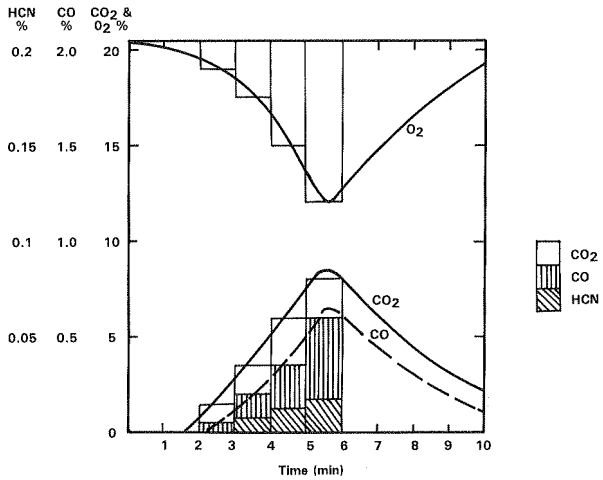


FIGURE 3. Average concentrations of narcotic gases (histograms) from gas profiles (curves) measured during the first tem minutes of a single armchair (polystyrene with polyurethane cushions and covers) room burn [13]. Since HCN was not measured, but was likely to have been present as an important toxic product, possible concentrations have been suggested for inclusion in the model. Using the equations given above, the fractional incapacitating dose for each narcotic gas is calculated for each minute and these data are shown in Table II. The fractional doses for CO and HCN, are added together and multiplied by the carbon dioxide hyperventilation factor VCO_2. To this is added the fractional dose of low oxygen hypoxia to give a total fractional dose for narcosis for each minute. The running total summed each minute exceeds unity during the fifth minute giving a figure of 1.204, indicating the onset of incapacitation (loss of consciousness). Alternatively narcosis may occur due to the effects of carbon dioxide, but the cumulative dose of this gas is only 0.065 during the fifth minute, which is insufficient to have any narcotic effect.

TABLE II. Fractions of an incapacitating dose of narcotic gases calculated for armchair burn

Time (min)	1	2	3	4	5
F_{Ico}	0	0	0.017	0.073	0.130
+ F_{Icn}	0	0	0.000	0.040	0.073
x VCO_2	0	0	1.442	2.376	4.434
=	0	0	0.025	0.268	0.900
+ F_{Io}	0	0	0.001	0.002	0.008
= Total	0	0	0.026	0.270	0.908
Running total (F_{IN})	0	0	0.026	0.298	1.204
or: F_{ICO_2}	0	0	0.005	0.013	0.047
Running total (F_{IN})	0	0	0.005	0.018	0.065

PHYSICAL HAZARD FACTORS AND IRRITANCY

RADIANT HEAT

As shown in Figure 4, derived from various literature sources [principally 14] there is a fairly obvious intensity limit for tolerance of radiant heat at 0.25 w/cm^2 (2.5 kw/m2). Below this intensity radiant heat can be tolerated for at least several minutes, but above this intensity for a few seconds only. The curves of radiant heat flux, and of the other physical parameters for the first 10 minutes of the armchair fire are shown in Figure 5. The tenability limit is

exceeded for approximately one minute during the sixth minute of the fire, and it is predicted that some degree of pain and skin burns might be sustained during that minute due solely to the effects of radiant heat.

CONVECTED HEAT

The curve for tolerance time of convected heat, also derived from various literature sources [principally 15] is shown in Figure 6, and from this is derived the expression for time to incapacitation:

$$t_{Ih}\ (\text{min}) = e^{5.1849 - 0.0273\ T^{\circ}C} \qquad \text{(equation 13)}$$

FIGURE 4. TIME TO SEVERE SKIN PAIN FOR EXPOSURE TO RADIANT HEAT

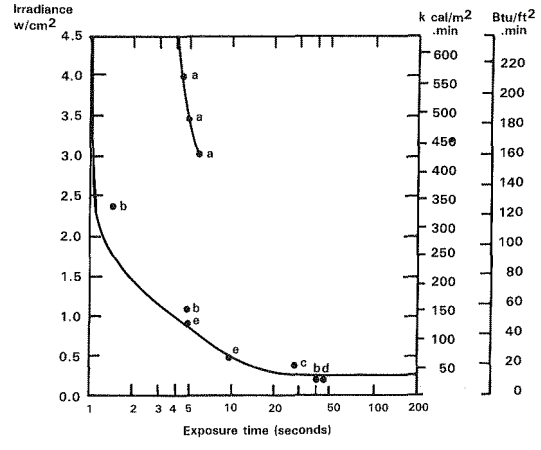
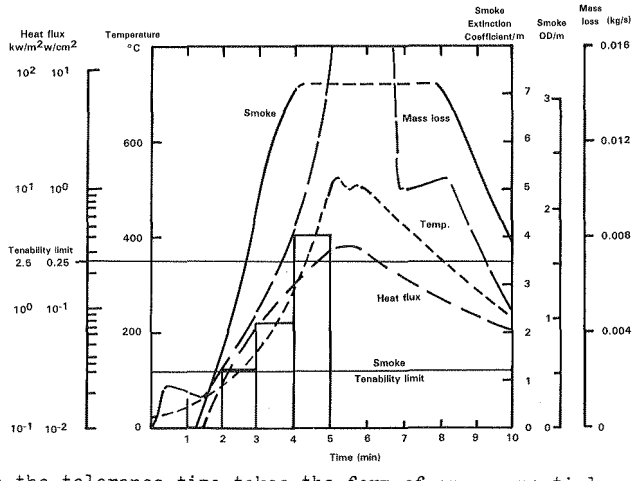


FIGURE 5. PHYSICAL PARAMETERS DURING EARLY STAGES OF SINGLE ARMCHAIR ROOM BURN

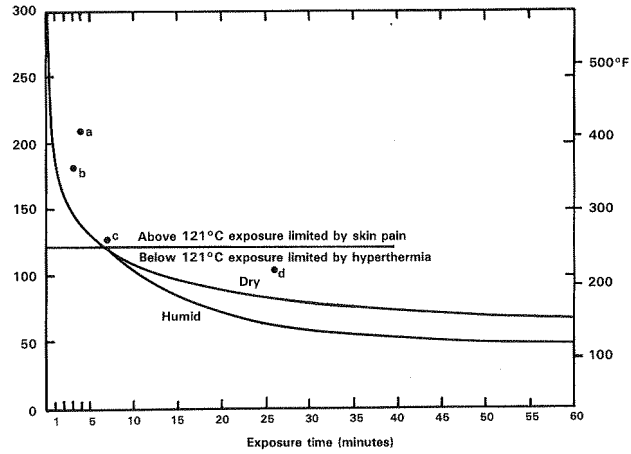


Since the tolerance time takes the form of an exponential curve, it is feasible to consider the victim as taking up a 'dose' of convected heat, much as a dose of a toxic product would be taken up. As with the narcotic gases it is therefore possible to use the concept of a fractional incapacitating dose of heat acquired each minute as follows:

$$F_{Ih} = \frac{1}{e^{5.1849 - 0.0273^{\circ}C}} \qquad \text{(equation 14)}$$

The average temperature each minute during the fire, and the fractional incapacitating dose of heat are shown in Table III. The cumulative fractional dose exceeds unity during the fourth minute (2.273) as the temperature exceeds 220°C, and then continues to increase dramatically during the fifth and sixth minutes. There will also be some degree of added effect from the radiant heat which would further increase the fractional dose. Incapacitation due to skin

FIGURE 6. TOLERANCE TIME FOR EXPOSURE TO CONVECTED HEAT



pain and burns is therefore predicted during the fourth minute, with severe and probably fatal burns of the skin and upper respiratory tract being a strong possibility, particularly after the fourth minute.

SMOKE

Visual obscuration by smoke is obviously concentration related, and a tenability limit of extinction coefficient 1.2/m (OD/m 0.5) has been set [13]. As Figure 5 shows, this is exceeded during the second minute of the armchair fire, and an extinction coefficient 2.4/m (OD/m 1.0), or approximately 1 m visibility is exceeded at the beginning of the third minute, the smoke curve rising very steeply at this point.

TABLE III. **Calculation of fractional incapacitating accumulation of convected heat for armchair burn**

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

SENSORY AND LUNG IRRITATION

The potency of combustion product atmospheres in terms of the concentrations causing sensory irritation and the doses causing lung irritation depend upon the materials involved and the decomposition conditions, and can at present be derived only empirically by animal experimentation [5]. However for the purposes of this example, as an approximate general figure based on mouse and primate data [5] it is suggested that a tenability limit concentration for sensory irritation in man for combustion products should be set at a mass loss

concentration of 1 mg/l, and a potentially fatal dose for lung inflammation is likely following a 30-minute exposure to a combustion product mass loss concentration of 10 mg/l (a ct product of 300 mg.min/l) [5,16].

For hazard estimation in the example fire, the first consideration is to decide whether the early appearance of sensory irritant smoke is likely to delay escape sufficiently for a victim to remain in the fire beyond the time when narcotic gases and heat are predicted to reach life threatening levels. In order to predict the irritancy of the smoke it is necessary to know the concentration/time profile of the fire products in terms of mass loss per liter of air. Although the mass loss curve for the armchair is shown in Figure 5, there are no data on the volume of air into which this mass was dispersed during the fire, but 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 second minute, greatly adding to the effects of smoke on vision and escape behaviour.

With regard to lung irritation it is estimated that the mass loss concentration in the smoke layer over the first five minutes of the fire would increase rapidly after the first minute to reach levels possibly approaching 100 mg/l by 5 minutes, with an average concentration over the 5 minutes of 10 to 20 mg/l. This would represent a fractional incapacitating dose of 50 to 100 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 sould reach 60 mg/l then serious effects on the lung would be likely after, and probably during exposure.

POSSIBLE GENERAL INTERACTIONS BETWEEN TOXIC AND PHYSICAL FIRE HAZARDS

It is likely that minor physiological interactions would occur between narcosis and several of the other factors, but it is felt that a reasonable model can be used in which narcosis, sensory irritancy and the effects of heat and visual obscuration can be treated separately [5]. At the behavioural level interactions may be more important and there is some experimental evidence for an interaction between sensory irritation and visual obscuration in man [17]. After exposure, the effects of skin burns, respiratory tract burns and chemical irritation (and even possibly CO narcosis) all combine to increase the probability of fatal pulmonary oedema and inflammation.

SUMMARY OF MODEL PREDICTIONS FOR ARMCHAIR FIRE

From the analyses performed, the effects on a victim exposed to the conditions in the armchair room burn (Figures 3 & 5) are predicted as follows:

1. Towards the end of the second minute and beginning of the third minute the smoke optical density and mass loss/liter would exceed the tenability limits for visual obscuration and sensory irritancy sufficiently to severely inhibit escape from the room.
2. During the fourth minute the average temperature was 220°C, and sufficent 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 narcotic gases.
4. It is predicted that a victim escaping or rescued after the fourth minute would suffer severe post-exposure effects due to skin burns, possible laryngeal burns with accompanying oedema and danger of obstructive asphyxia, and also pulmonay oedema 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 victim would die at some time between a few minutes and

1 hour due to the effects of narcosis, circulatory shock, and possibly hyperthermia.

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 that 3 minutes after ignition. However 3 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 2 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.

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