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Free Radical Production from Controlled Low-Energy Fires: Toxicity Considerations

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ABSTRACT: Most fire departments respond within the first 5 min of notification of a fire. If fire victims are found at that stage by the firefighters, then incapacitation or death has occurred during the initial low-energy phase where smoke is being produced. Studies have shown that during this initial low-energy phase of the fire, gases commonly thought responsible for incapacitation or death are frequently not present in concentrations adequate to cause this result. In the current study free radicals, measured by electron spin resonance spectroscopy, were trapped in concentrations that we consider incapacitating, thus providing an explanation for "incapacitation without cause." This finding points the way to the design of more efficient temporary protective equipment for those who are in a high fire hazard environment, such as airline passengers, and suggests the idea of establishing a thermodynamic marker for the relative toxicity of building materials.

KEYWORDS: forensic science, fires, free radicals

The results of a two-year study of gases produced in structural fires in the Dallas area caused us to initiate an investigation into the formation of free radicals during the low-energy phase of the fire[5] and into their possible role in the incapacitation of fire victims [*1*]. The first phase of a fire produces incapacitation, many times resulting in death. During this phase no gases have been found in concentrations capable of producing rapid unconciousness. Therefore, low-energy controlled fires were studied using numerous materials such as wood, paper, synthetic fabrics, and plastics (including poly[vinyl chloride]).

Background and Literature Survey

Friedman [*2*] has set forth the physical parameters to be considered regarding the variations of ignition and burning of solids. These factors include the pyrolysis gas concentration in the boundary layer, heat transfer from flame to surface, internal heat transfer, radiant loss from surface, and radiant flux from surroundings to surface.

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[5]"Low-energy fire" is defined in this text as a fire where a material is burning or decomposing (pyrolyzing) at a temperature below the combustion of wood and is producing minimal radiant heat.

Free radicals produced by combustion range in stability from explosives to species that are stable at room temperature. Several known bond-dissociation energies are listed in Table 1. The bond-dissociation energy for a C—C, C—H, or C—O bond is about 376.56 KJ·mol^{-1} (90 kcal·mol^{-1}). To provide sufficient energy for the homolytic dissociation of such bonds by thermal means requires temperatures of about 450 to 650°C (842 to 1202°F). This is the temperature range of the piloted ignition temperatures of various materials, ranging from certain plastics on the low end to wood on the upper end. The increased homolytic dissociation or organic material with increasing temperature has been shown very clearly by trapping free radicals throughout the temperature rise from 200°C (392°F) to approximately 500°C (932°F) during the pyrolysis of oil-distillation residues [*3*].

Most covalent bonds require high temperatures for dissociation, but under certain conditions the dissociation may occur at quite low temperatures. The free radical(s) formed may be stabilized by resonance or the reaction may involve the formation of a very stable molecule, such as nitrogen, which has a high heat of formation. As a consequence of the comparatively easy production of free-radical products $R\cdot$ from such compounds, they may be used to initiate the homolytic dissociation of other suitable compounds at relatively low temperatures [*4,5*].

Oxygen has the electronic configuration $(1s)^2(2s)^2(2p)^4$ and is a biradical, as demonstrated by its paramagnetic properties. Peroxy radicals formed during the process of combustion may abstract a hydrogen from a neighboring molecule to form the hydroperoxide:

$$RO_2\cdot + X - H \longrightarrow RO_2H{=}X\cdot$$

This reaction of oxygen with many types of free radicals is used in this text to indicate that a particular pulmonary mechanism proceeds through the formation of free-radical intermediates [*6*].

Animal studies concerning free-radical mechanisms and pulmonary toxicity indicate that changes in the lung surfactant (dipalmitoyl phosphatidylcholine) result in decreased oxygen tension and decreased oxygen uptake [*7,8*].

Membrane lipid peroxidation has been set forth as a primary event in toxicity of certain chemicals during the past few years. The definition of lipid peroxidation is the oxidative dete-

TABLE 1—*Bond dissociation energies.*

Bond	D, kcal·mol^{-1}
CH_3—H	101
—CH_2—H	92
=CH—H	120
=C—H	80
Cl—Cl	58
CH_3—Cl	81
CCl_3—Cl	68
CCl_3—Br	49
CCl_3—H	90
CCl_3—F	102
OH—H	117
OH—OH	52
HOO—H	90
CH_3—OH	90
CH=CH	230
CH_3—CH_3	83
CH_3—NH_2	80
NH_2—NH	60

rioration of polyunsaturated lipids and it involves the reaction of oxygen with polyunsaturated lipids to form lipid-free radicals and semistable hydroperoxides.

Peroxidation of unsaturated membrane lipids has far greater biochemical effects than mere deterioration of lipids. The position of these lipids with proteins in membranes allows for further interaction between the lipids and proteins. During lipid peroxidation, and in particular during the termination reactions where two adjacent free radicals join to form new bonds, the result can alter the structure. If the molecule is an enzyme, the activity may be significantly altered. One may observe abnormal lipid-lipid, lipid-protein, or protein-protein cross-linking. It is therefore obvious that the biochemical consequences of lipid peroxidation are extremely important in the interpretation of toxicological events [*3, 9*].

Emission of airborne free radicals has been found to occur in the processing of polyethylene and polystyrene plastics [*10*]. Table 2 presents the data obtained, showing the detection of free radicals in air samples during injection molding, extruding, seam welding, and wire cutting of the plastics. The air samples were collected into a solution of phenyl-*N*-tert-butylnitrone (PBN). This compound is a spin trap which, when reacted with a free radical, produces a spin adduct (see Fig. 1). Figure 2 presents the electron spin resonance (ESR) spectrum of the spin adduct.

Arterial blood gases, residual lung volume (RV), deflation pressure volume (PV) curves, pulmonary pathology, and body weight changes were studied in rats exposed up to 14 days to either 0.75- or 1.0-ppm ozone. Arterial PO_2 and body weights decreased progressively with length of exposure, while $PaCO_2$ and RV increased [*11*]. The slope of the PV curve decreased in all groups exposed to ozone.

Pathological changes in the lung increased in severity with concentration and length of exposure. The present findings have shown that arterial blood gas measurements represent a sensitive index of altered lung function in rats, a species very sensitive to ozone exposure.

Nieman et al [*12*] demonstrated in dogs a marked decrease in the arterial PO_2 after smoke inhalation. This improved slowly but was still significantly reduced two hours after smoke insult. Decrease in PaO_2 were greater if the smoke exposure was repeated. In these studies, they also observed a marked increase in the minimum lung surface tension. An increase of 68 mean to 220 MN · m mean (6.8 mean to 22 dynes/cm mean) indicated that the lung surfactant was no longer active or that less surfactant was available to the surface. The findings in these studies indicated an instantaneous surfactant deficit, suggesting chemical or physical inactivation of preformed surfactant.

Studies on the effect of cigarette smoke on pulmonary surfactant indicate that the effect of this smoke is also instantaneous [*13-14*].

Nieman et al [*12*] found that by placing a filter between the smoke source and the endotra-

TABLE 2—*Relative concentrations of free radicals near production machinery* [10].

Process	Plastic	Processing Temperature,[a] °C	No. of Active Samples	Free Radicals Detected $\times 10^8$ Unpaired Spins, m^{-3}	
				Mean	Range
Injection molding	HD polyethylene	320	2	6.1	6.0-6.2
	LD polyethylene	180	2	3.6	1.8-5.4
	Polystyrene	180	2	1.5	0.7-2.4
	Acrylobutadiene-styrene	180	6	1.4	1.2-1.6
Extruding	LD polyethylene	180	4	1.2	1.0-1.30
Seam welding	LD polyethylene	200	5	0.5	0.3-0.6
Wire cutting	Polystyrene	500	6	2.9	1.0-4.6

[a]The temperature of the die, knife, or cutting device during normal operating conditions.

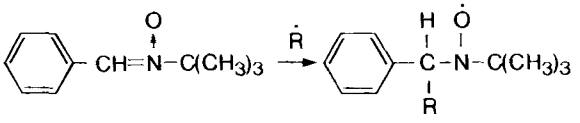

FIG. 1—*Spin adduct.*

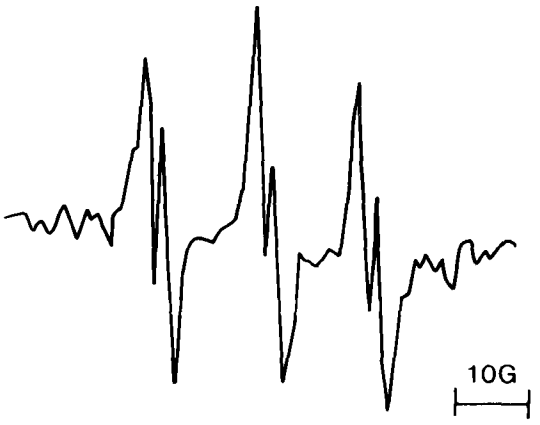

FIG. 2—*Electron spin resonance spectrum of the spin adduct.*

cheal tube there was complete physiologic protection and preservation of surfactant. They attributed this phenomenon to aerosol-type materials in the smoke. However, a filter would also trap free radicals within the smoke.

Whitener et al [*15*] performed serial pulmonary function measurements in 28 patients with thermal injury to investigate the pulmonary effects of smoke inhalation, small and large surface burns, and the combination of burns and smoke inhalation. The results of these clinical studies indicated that smoke inhalation caused severe airway obstruction 9 h after exposure.

Robinson et al [*16*], Moylan et al [*17*], and Agee et al [*18*] have described ventilation and perfusion abnormalities that were present in victims of smoke inhalation injury preceding clinically apparent hypoxemia. Robinson et al demonstrated that a moderate smoke inhalation injury, not associated with thermal cutaneous burns, produced a "chemical tracheobronchitis" concentrated in large and medium airways.

Thirty firefighters were studied by Unger et al [*19*] utilizing pulmonary function tests immediately after a severe smoke exposure. These firefighters were examined 1½ and 18 months later to evaluate acute and chronic changes in their spirometry. The results were compared with predicted values and with those from a group of closely matched control subjects. No significant differences were found between the acute postexposure spirometry values and those recorded at 6 weeks and 18 months later. A trend toward an increased rate of volume loss in the forced vital capacity (FVC) and forced expiratory volume (FEV) was noted. A significant decrement was found in FVC compared with predicted value, and in FVC and FEV compared with matched control subjects.

Experimental Procedures

Air samples were pumped with a Mine Safety Appliances (MSA) portable pump (Model TD) at a rate of 0.75 L/min through a glass impinger containing 20 mL of 0.05*M* PBN in toluene.

The length of time for sampling was 5 min and the control fire was maintained for 60 min, producing twelve samples per fire. Each sample was immediately transferred to a glass screw-top test tube, placed in an alcohol-dry ice bath, and transported to the laboratory.

The measurement for trapped free radicals was performed with a Varian Instrument Model E-4 electron paramagnetic resonance (EPR) spectrometer operating at a frequency of 9.15 GHz and a magnetic field modulation amplitude of 16 kT (1.6 gauss). The samples were initially bubbled with prepurified argon gas for 30 s and then sealed in the quartz EPR sample tube. The spectra were recorded at 25°C and the temperature was adjusted with a variable temperature accessory. The EPR signal of the trapped free radical (Fig. 3) was quantitated by comparing the integrated area of the signal to that obtained with a standard pollution of 2,2,6,6-tetramethylpiperdine *N*-oxide (TEMPO, Aldrich), in buffer.

Carbon monoxide was continuously analyzed by infrared spectrophotometry, utilizing a Miran 1A general-purpose gas analyzer produced by Foxboro Analytical Co. This infrared gas analyzer is a single-beam variable filter spectrophotometer, set at a wave length of 4.61 μm, with a path length of 20.25 m and 0.045 absorbance. The minimum detection limit was 0.2 ppm. The infrared gas analyzer was equipped with a strip chart recorder for recording the absorbance values of carbon monoxide as a function of time.

Carbon monoxide, hydrogen cyanide, and hydrogen chloride were also monitored at 5-min intervals by utilizing colorimetric detector tubes, No. 91229 MSA, No. 93262 MSA, and No. 91636 MSA, respectively.

The organic analyses were conducted by computer-interfaced gas chromatography/mass spectrometry (GC/MS) utilizing a Finnigan MAT 1000 series, OWA-automated GC/MS system. The gas chromatography was performed with a 1.8-m (6-ft) by 2-mm inside diameter column packed with 0.2% Carbowax 1500 on Carbopack C (60–80 mesh). The temperature was held at 60°C for 3 min, then raised 8°C/min to a final temperature of 160°C with a final time of 15 min.

The GC/MS was equipped with a Tekmar Model LSC-2 liquid sample concentrator with a 25-mL sampler and Tenax-Silica gel trap column. The purge rate was 40 mL/min, with a purge

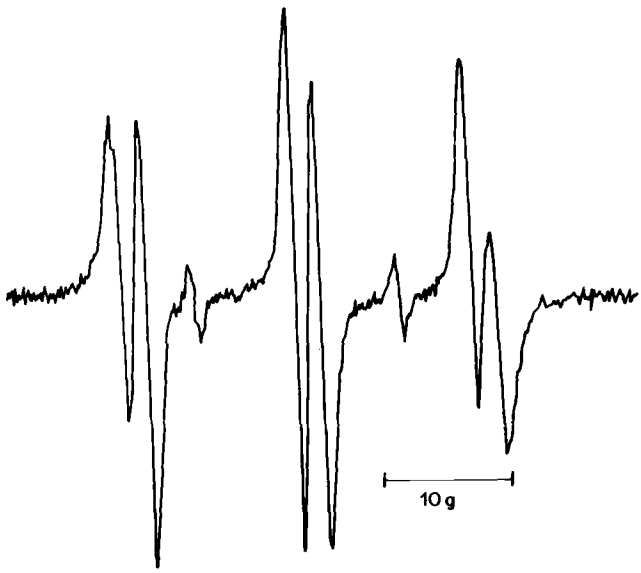

FIG. 3—*EPR signal of the trapped free radical.*

time of 10 min at 30°C. The desorb time was 4 min at 180°C and the bake time was 3 min at 225°C. All gas samples for analysis were handled with a 30-mL Hamilton gas tight syringe.

The test area was a jail cell located in the Dallas County Jail prior to occupancy. The doors were of glass rather than bars, providing a unique test chamber. The interior of the cell was all tile, with exception of the concrete floor.

The test cell (Figs. 4 and 5) were 3.4 m (11 ft 2 in.) deep, 1.7 m (5 ft 8 in.) wide, and 3.5 m (11 ft 7 in.) high, producing a volume of 21 m^3 (733 ft^3). Displacing a portion of this volume was a bunk for bedding and sitting purposes that was 2.2 m (7 ft 2 in.) long, 0.7 m (2 ft 3 in.) wide, and 0.4 m (1 ft 3 in.) high, for a volume of 0.6 m^3 (20.2 ft^3), thus reducing the total volume to approximately 20 m^3 (713 ft^3). Figure 5 shows the bunk with a partition behind it and a floor-to-ceiling extended wall that contained the plumbing. These additional building design factors reduced the cell volume by 1.2 m^3 (42.7 ft^3), giving a total cell air volume of about 19 m^3 (670 ft^3).

Air flow within the cell was from air-conditioning ducts located above the door of the cell, 51 mm (2 in.) below the ceiling. Air exhaust from the cell was through a 51-mm (2-in.) space below the door. Cell air volume exchange rates could be controlled by eliminating various air handling systems within the building. The stages for test systems employed for obtaining the data discussed below was with the system completely off, producing zero air exchanges per hour and with half the air-conditioning system running, producing two air exchanges per hour. The latter rate was an engineering estimate.

Fuel loading for each test was 2 kg (4 ½ lbs) of combined wood, paper, clothing (cotton and polyester), and other synthetic materials, including poly(vinyl chloride). The materials chosen for combustion were those items that the Dallas County Sheriff would issue prisoners. In addi-

FIG. 4—*Test cells in Dallas County Jail.*

FIG. 5—*Close-up of test cell showing built-in bunk and plumbing area.*

tion, items were included representing materials that prisoners would have access to within the prison area or could possibly obtain from visitors.

The fuel materials were placed in a metal container and ignited by a small quantity of gasoline. The container was located on the bunk area, 0.4 m (1 ft 3 in.) above the floor and 51 mm (2 in.) from the rear partition, allowing a distance of 2.13 m (7 ft) between the fire source and the front door. All gas measurements were made at a height of 1.5 m (5 ft) and were obtained at a distance of 0.3 m (1 ft) from the door. Air temperature was monitored at heights of 1.8 and 3.4 m (6 and 11 ft).

The overall fire test protocol was developed following recommendations set forth in ASTM Guide for Room Fire Experiments (E 603).

A total of six tests each were conducted under each condition. Data variances of carbon monoxide production were within 10 ppm for each test. Data variances for HCN were within ± 2 ppm and for HCl, ± 1 ppm.

Results

Figures 6 and 7 set forth the significant gases produced in these tests.

Zero Air Exchanges per Hour

The most significant concentrations were obtained, and obtained in the shortest period of time, with zero air exchanges per hour (Fig. 6).

Free Radicals (Fig. 8)—These organic species were trapped, with sampling every 2 min for the first 10 min and every 12 min thereafter over a period of 1 h. The maximum concentration of 1200 ppm was reached in 4 min and was maintained until the fire was out. The temperature

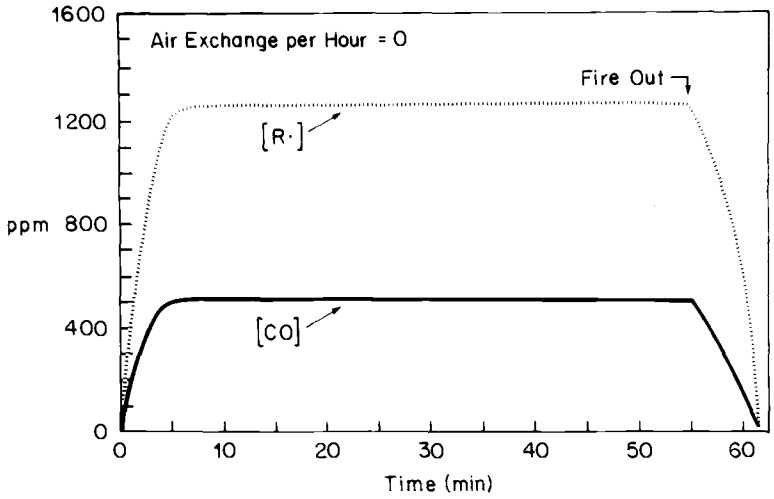

FIG. 6—*Gas production with zero air exchanges per hour.*

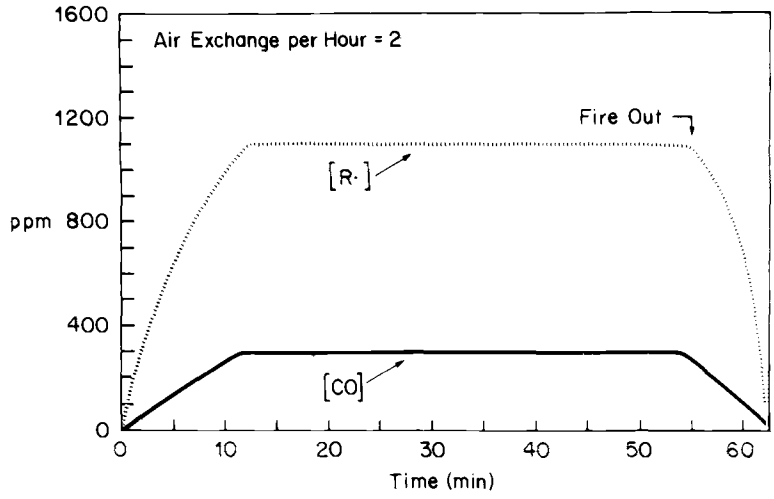

FIG. 7—*Gas production with two air exchanges per hour.*

reached a maximum of 37°C (98°F) at 1.8 m (6 ft) and 63°C (145°F) at 3.4 m (11 ft) after 45 min.

Carbon Monoxide—Carbon monoxide paralleled the free radical production, as would be expected from combustion kinetics. The maximum concentration of 500 ppm was obtained in 4 min. This concentration was maintained until the fire was out. The pressure differential between the inside and outside of the cell created by the temperature change caused a slight air flow from the cell. This caused the constant air concentrations to be maintained.

Hydrogen Cyanide—Cyanide air concentrations were monitored at 10-min intervals. During the first 10 min the air concentration reached 1 ppm and by 30 min it was 3 ppm. The maximum air concentration of 5 ppm was reached in 50 min.

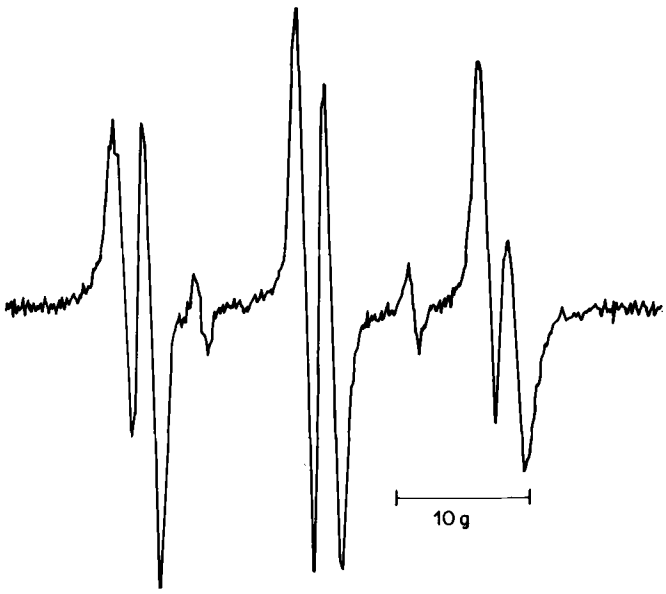

FIG. 8—*Spectrum of radical-PBN spin adducts.*

Hydrogen Chloride—Hydrogen chloride air concentrations were monitored at 10-min intervals. The maximum concentration of 2 ppm was reached within the first 10 min and was maintained throughout the burn.

Organics—Organics identified qualitatively included toluene, heptane, 3-methylcyclohexane, 3-methylhexane, vinyl chloride, and ethyl alcohol. The air concentrations were in the low parts-per-billion range and therefore considered irrelevant toxicologically. However, organics identified in smoke will become increasingly important for evaluating pyrolysis mechanisms in relationship to thermodynamic properties and pulmonary response (that is, toxicity).

Two Air Exchanges per Hour

Under the conditions of increased air flow (Fig. 7), the time required to reach maximum air concentrations increased, as would be expected.

Free Radicals (Fig. 8)—These organic species were trapped with sampling every 2 min for the first 10 min and every 12 min thereafter over a period of 1 h. The maximum concentration, 1000 ppm, was reached in 12 min and was maintained until the fire was out. The temperature reached a maximum of 22.4°C (98°F) at 1.8 m (6 ft) and 37.4°C (125°F) at 3.4 m (11 ft) after 45 min.

Carbon Monoxide—Carbon monoxide paralleled the free radical production, as would be expected from combustion kinetics. It reached a maximum of 300 ppm in 12 min, maintaining this level until the fire was out.

Hydrogen Cyanide—Cyanide air concentrations were monitored at 10-min intervals. It required 30 min to reach a maximum 1-ppm cyanide air concentration.

Hydrogen Chloride—Hydrogen chloride air concentrations were monitored at 10-min intervals. The maximum concentration of 1 ppm was reached in 50 min.

Organics—The following organics were identified qualitatively: toluene, heptane, 3-methylcyclohexane, vinyl chloride, and ethyl alcohol.

Discussion

The presence of free radicals in combustion products has long been recognized, but it has been assumed that these intermediates are so short-lived that they had no toxic importance.

Accurate data for heats of formation of gaseous free radicals have been as elusive as they are important. Over the past 20 years there has been a general and continuous revision in almost all free-radical heats of formation as methods for their measurement have become better understood and refined [*4,20-30*]. The thermodynamic interpretation is an increase toward greater molecular stability relative to bond-rupture processes, which is consistent with the data reported by Westerberg et al [*10*]. They reported that the quantitative measurements of the samples showed that the emission of free radicals into the work atmosphere was "related to the temperature, the plastic, and the processes used." This statement has a major impact in regard to understanding relative toxicities of materials. For example, Hinderer and O'Mara [*31*] have reported a study concerning quantities of materials required to produce a 50% mortality rate (LD_{50}) in mice. Table 3 summarizes their data, along with that of others [*32*]. On the surface, the toxicity difference between poly(vinyl chloride) (PVC) and acrylonitrile-butadiene-styrene (ABS) could be easily explained. The PVC required 0.14 g to produce 50% mortality, whereas ABS only required 0.005 g at 800°C. Since PVC begins a quantitative decomposition at about 270°C and ABS at 300°C, the difference in toxicities should be the cyanide produced by ABS. This may or may not be true. However, cyanide does not explain the LD_{50} values for polyethylene, 0.052 g; polyurethane, 0.038 g; polypropylene, 0.030 g; and nylon, 0.023 g. Considering the lower decomposition temperatures of these latter products, thermodynamics and free radicals may offer the explanation. The same study should be conducted at 200, 300, 400, 500, and 600°C. This latter temperature is approaching the autoignition temperature of wood, which is commonly accepted as a nontoxic material.

Chlorine gas is an extremely stable free radical. The Cl—Cl bond dissociation energy is 58 kcal/mol (Table 1) and the free radical Cl can be initiated by sunlight or equivalent energy. The signs and symptoms of sublethal chlorine gas poisoning are like those of sublethal smoke inhalation [*15-19*]. Table 4 sets forth the chlorine exposure thresholds and limits. If one views chlorine gas toxicity as a free-radical mechanism, the interpretation of free radicals produced in this study would be that incapacitation would potentially occur after the concentration exceeded 1000 ppm. This would be consistent with smoke inhalation studies, where pulmonary effects, surfactant effects, and oxygen uptake diminishment were demonstrated [*3,7-19*]. This would also be consistent with medical examiners' toxicology reports of fire victims having blood carboxyhemoglobin levels below what is considered lethal.

The surface-active material to be found in the alveolar lining of the lung is a mixture of phospholipids, neutral lipids, and proteins. The surfactant works by lowering the surface tension

TABLE 3—*Material required to produce a 50% mortality rate in mice when the sample is combusted at 800°C (1472°F).*

Material	Amount Required, g
Polyacrylonitrile	0.160
PVC	0.14
Polystyrene	0.12
Polyethylene	0.052
Polyurethane	0.038
Polypropylene	0.030
Nylon	0.023
ABS	0.005

TABLE 4—*Chlorine exposure thresholds and limits.*

Cl_2 Concentration, ppm	Effect of Limit
0.03-3.5	range of reported odor thresholds
1	threshold limit value, OSHA time-weighted average (TWA); permissible level, 8-h workday
1-3	slight irritation; work possible without interruption
3	permissible level for 15 min; 60-min emergency exposure limit (EEL)
3-6	stinging or burning of eyes, nose, throat; lacrimation, sneezing, coughing
4	suggested 30-min EEL
5	severe irritation of eyes, nose, respiratory tract; intolerable after a few minutes; suggested 15-min EEL
7	suggested 5-min EEL
14-21	dangerous for 30 to 60 min, respiratory distress after 30 min
35-50	lethal in 60 to 90 min
430	lethal after 30 min
1000	immediate incapacitation followed shortly by death

and enhancing surface elasticity of the surface layers, thus assisting alveolar expansion and contraction.

The hydration of the polar head groups provides the driving force for spreading at the air-liquid interface. The peroxidation of the unsaturated lipid within the lung surfactant system initiated by free radicals within smoke can alter the polarity, hydration, and spacial arrangement of the lipid itself. The result is an immediate alteration of the surface tension. The primary lipid expected to be affected is phosphatidyl glycerol (PG), and minor molecular alterations would be expected to have major changes. If both PG and DPPC underwent lipid peroxidation reactions terminated by a radical-radical reaction, the product DPPC-PG would have devastating pulmonary effects. Since DPPC already has a 43°C melting point, the lipid-lipid product would have an immediate drying effect on the lung surface, thus producing lung surface tension, or O_2-reduced tension, resulting in pulmonary diminishment of oxygen uptake. Other radical reaction termination combinations, resulting in similar effects, would be PG-PG and DPPC-DPPC. Microscopically, the lung tissue may or may not show atelectasis in acute exposures. Studies are presently being conducted on lungs from animals exposed in monitored smoke environments and from victims of smoke inhalation investigated by the Dallas County Medical Examiner.

Based on the above evidence and data, and with continuing studies with animals underway, the proposed model for reaction in victims by smoke is a two-phase process. The first phase is rapid oxygen-uptake diminishment because of the large amount of stable free radicals in the smoke and their reaction with lung surfactant. As soon as the available blood oxygen is used, unconsciousness (incapacitation) occurs. Next, in Phase 2 the victim is unrelentingly exposed to the smoke and its contents. If the fire burn rate increases, the CO air concentration would increase. This would account for some smoke inhalation victims having lethal quantities of blood carboxyhemoglobin. However, if the victim remains in a relatively cool environment with dense smoke, the cause of death would be asphyxia resulting from oxygen diminishment, with those gases such as CO, CN^-, and the like contributing to the death.

One must seriously consider this mechanism of incapacitation when explaining the survival of the passengers of the Air Canada fire. Each survivor maintained a cloth or tissue over the

nose and mouth throughout the event. One of the termination reactions for free radicals is collision with an object.

Acknowledgments

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The entire event was recorded on file (35-mm slides) by Dr. Patrick Bessant-Mathews, deputy chief medical examiner, Southwestern Institute of Forensic Sciences and Assistant Professor of Pathology, University of Texas Southwestern Medical School. Two of these photographs are included in this text.

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