INHALATION OF TOXIC PRODUCTS FROM FIRES*

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Approximately 8,000 fire fatalities occur annually in the United States, and about 80% of these fatalities are attributed to inhalation of toxic combustion products. It is further estimated that 200,000 fire injuries occur annually and that many of these invoke smoke inhalation. The recent MGM, Hilton, and Stouffer Hotel fires emphasize the role of smoke in unwanted fires even though these multiple fatality fires comprise a very small percentage of the total number of annual fire deaths.

As a first step to understanding these losses, a fire-fatality study was initiated in October 1971 in cooperation with the Maryland State Medical Examiner's Office in an effort to define the smoke inhalation hazard resulting from fires.[1] The objective of the program was twofold: to determine the specific cause of death by detailed autopsy study of fire victims and to determine the specific causes of fatality-producing fires by on-the-scene fire investigations.

In addition to this study, the Center for Fire Research has also been involved in such other major multiple death fires as the Tennessee jail fire.[2] The major findings and toxicological issues raised by these studies are summarized below. The Tennessee jail fire represents a nonresidential fire and is included because it highlights a number of smoke-inhalation problems related to the use of synthetic materials, and complements some of the findings of the Maryland study.

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Scope and Limitations of the Study

The fatality study was initiated in the State of Maryland because Maryland law requires that a medical examiner investigate all victims of violent death, which includes fire deaths. In addition, Maryland represents a cross-section of economic and social patterns in the United States likely to lead to fire accidents.

The fire-fatality study was generally limited to residential fires, although a few selected multiple fatality nonresidential fires were included. Residential fires were selected because review of various fire loss data showed that 72 to 84% of the fatalities occur in residential occupancies.[1] The study was further limited to deaths that occurred within six hours of the fire because of the difficulties involved in following victims that die after six hours.

Data reported in this study were obtained over a six-year period from September 1971 through December 1977. During this period 523 fire fatalities resulting from 392 fires were studied. Approximately 350 of these fires were residential. A few individuals who died in explosions or in automobile accidents with attendant fires are included in the data if fire was the major cause of death.

Residential Fire Problem

The results of 398 fire investigations in terms of initial materials ignited are shown in Table I. Ignition of mattresses and bedding account for 30% of the fires (120) and 24% of the victims (127). Sofas and upholstered chairs were the second largest category of items to ignite. These items account for 17% of the fires (68) and 20% of the deaths (106). As noted in the table, other flammable materials and liquids when combined account for 23% of the fires and 27% of the deaths.

Table II is a survey of the causes of these 398 fatal fires. As noted in the table, 47% of the fatal fires are due to smoking, which accounts for 45% of the fatalities. Smoking items such as cigarettes and matches were identified as the single largest cause of fatal fires. In fact, the predominant scenario leading to a fire death in the home is ignition of furnishings (upholstered furniture and mattresses) by cigarettes and other smoking items. An independent study of three data bases on fires and fire deaths not limited to private residences concluded that the most common fire death scenario, by far, is the residential-furnishings-smoking scenario,

TABLE I

DISTRIBUTION OF FATAL FIRES ACCORDING TO INITIAL ITEM IGNITED

Initial item ignited	*% of fires*	*% of victims*
Mattresses and bedding	30	24
Sofa and chair	17	20
Other flammable materials	13	16
Flammable liquids	10	11
Clothing	7	6
Electrical	3	3
Other	12	10
Unknown	8	10
Total	100	100

Total no. of fires 398
Total no. of fatalities 530

TABLE II

DISTRIBUTION OF FATAL FIRES ACCORDING TO CAUSE

Cause	*% of fires*	*% of victims*
Smoking	47	45
Electrical	7	7
Heating equipment	7	8
Stove	4	3
Matches	6	7
Flammable liquids	3	3
Cooking	3	4
Suicide	4	3
Arson and suspicious	5	7
Other	10	10
Unknown	4	3
Total	100	100

Total no. of fires 398
Total no. of fatalities 530

which alone accounted for 27% of the fire deaths.[3] The present study confirmed these findings. To understand the nature or cause of death in this study, each fatality was studied in detail.

Study of Fire Victims

Results of the detailed autopsy study are presented under the following three major categories: toxicological analysis (carboxyhemoglobin, alcohol, and cyanide), heavy metal analysis of trachea and bronchi, and pathology (heart study).

Toxicology. Samples of blood, urine, bile, liver, kidney, and spleen were collected from fire victims for toxicological analyses. Blood samples were analyzed for alcohol, drugs, carboxyhemoglobin, and hydrogen cyanide. The major chemical asphyxiants that can contribute to the inhalation problem are carbon monoxide and hydrogen cyanide gases. There is no doubt about the role of carbon monoxide in fires, but whether carbon monoxide interacts with other toxic agents (hydrogen cyanide gas, soot, heavy metal, etc.) at low levels to cause incapacitation and then death remains unanswered.

Carbon monoxide. Carbon monoxide is produced in all fires and is often considered the primary toxicant. Table III shows the distribution of the blood carboxyhemoglobin saturation levels found in victims in this study. For the purposes of this study, a 50% carboxyhemoglobin level was considered lethal and, as noted in Table IV, 60% of the victims fall into this category. In those fatalities in which the carboxyhemoglobin was less than 50%, other factors, such as cardiovascular disease or other toxicants such as hydrogen cyanide and alcohol were considered. Table IV tabulates the cause of fatalities. Preexisting heart disease plus carbon monoxide poisoning or a possible combination of toxicants including alcohol may account for the remaining "unexplained" category.

Alcohol involvement. Blood alcohol was found in 40% of the fatalities, and it is the only drug that appears to be involved in fatal fires to any significant degree. In fact, it is a major problem in fatal fires both from the standpoint of the fatal outcome as well as the initiation of the fire. Table V shows that elevated blood alcohol levels were found in 211 of the 530 victims and that 85% of these victims had blood alcohol levels that met the legal definition of intoxication ($\geq 0.1\%$). The alcohol problem is further highlighted when one considers that approximately half of the fires were caused by "careless smoking" and that in two thirds of these fires alcohol was involved. The preponderance of the alcohol cases involved men 20 years of age and older. The role of alcohol will be discussed further in the heart study.

Hydrogen cyanide. The toxicological significance of hydrogen cyanide

TABLE III

DISTRIBUTIONS OF FIRE VICTIMS ACCORDING TO BLOOD CO SATURATION LEVELS

COHb (%)	*No. of victims*	*%*
0–9	48	9
10–19	42	8
20–29	37	7
30–39	38	7
40–49	43	8
50–59	58	11
60–69	79	15
70–79	111	21
> 80	74	14
Total	530	100

in fire victims is difficult to assess whether from the literature or from this study. Part of this difficulty is due to lack of inhalation toxicity data from controlled experiments on animals. It has become a major concern in fire fatalities because it is known to result from the thermal degradation of some polymers containing nitrogen, it has a relatively high toxicity, and there is strong public reaction to this toxicant.

For a detailed discussion of the mechanism of action of cyanide on biological systems the reader is referred to Goodman and Gilman.[4] In summary, hydrogen cyanide reacts readily with trivalent iron of cytochrome oxidase to form a cyanide complex that inhibits cellular respiration, an action referred to as cytotoxic hypoxia. In low concentrations, cyanide stimulates respiration while it depresses the brain electrical activity. What does not appear to be well known is the environmental cyanide level that will produce a given biological or blood level and the corresponding physiological effects in humans or animals. A great deal of the difficulty appears to be due to the lack of a good technique to measure hydrogen cyanide in postmortem samples and the effects of storage on cyanide levels.

To determine the significance of blood cyanide levels in fire victims, a determination of background cyanide values in nonfire-related victims was made.[5] In this study normal blood cyanide values were determined in 64 random postmortem nonfire deaths, 22 impact injury airplane crash victims, and 32 living subjects with variable smoking histories. The results are shown in Table VI. These 118 cases had a mean ranging from 0.05 to

TABLE IV

CAUSES OF FIRE FATALITIES (530 CASES)

Cause of death	%	
CO alone	60	(COHb > 50%)
CO + cardiovascular disease	20	
Burns	11	
Unexplained	9	
	100	

Note: Alcohol was involved in 40% of fatalities.

TABLE V

BLOOD ALCOHOL CONCENTRATIONS: LEVELS FOR 211 FATAL FIRE VICTIMS

Blood alcohol concentrations (%)	*% of victims*
0.01–0.04	7
0.05–0.09	8
0.10–0.14	15
0.15–0.19	23
0.20–0.24	17
0.25–0.29	16
0.30–0.34	4
0.35–0.39	7
>0.4	3
	100

Note: 40% of the 530 fire victims had a positive blood alcohol level.

TABLE VI

NORMAL BLOOD CYANIDE CONCENTRATIONS (μg./ml.)

	No.	*Mean*	*Range*	*SD*
Random postmortem	64	0.05	0.00–0.22	0.04
Airplane crash	22	0.06	0.04–0.19	0.03
Living	32	0.07	0.02–0.21	0.05

0.07 μg./ml. and an upper limit of 0.22 μg./ml. For the purposes of this study, 0 to 0.25 μg./ml. was considered normal. The higher background blood cyanide levels within the range were attributed to smoking cigarettes.

Cyanide concentrations were determined in the last 272 fire fatalities during the period of this study. A distribution of the results of these analyses is shown in Table VII. Normal concentrations would not be expected to elicit any biological effect, although concentrations exceeding 0.26 μg./ml. indicate the presence of cyanide produced from burning building materials. Concentrations that are possibly toxic (1.01 to 2.00 μg./ml.) were found in 24% of the cases and probable toxic concentrations (➤2.00 μg/ml.) were found in 10% of cases. Sublethal concentrations of hydrogen cyanide may cause confusion, incapacitation, or may interact with other toxic substances to produce death. An analysis of the number of cases with elevated blood cyanide on a yearly basis is shown in Table VIII to see if there is a changing pattern as a function of the introduction of more synthetic furnishings.

A correlation of blood cyanide concentrations in the fatalities with carboxyhemoglobin saturation levels show that potentially dangerous cyanide levels were associated with high carbon monoxide levels. When cyanide content was high, carbon monoxide content was high. No low carbon monoxide levels were associated with high cyanide levels. Death could not be explained by cyanide levels not otherwise explained. This is further confirmed by a correlation between carboxyhemoglobin and cyanide levels in 10 victims of the Tennessee jail fire shown in Table IX. However, cyanide and other toxicants could play an intermediate role leading to early incapacitation which prevented escape and as a consequence the victim may have been exposed to high levels of carbon monoxide. This is an unanswered issue in the fatality study.

Pathology of fire casualties. Postmortem examinations were performed at the Maryland State Medical Examiner's Facility within six hours of the fire. This examination included an autopsy which involved the determination of burn injuries, soot in the respiratory tract, pulmonary edema, pulmonary congestion, and cardiovascular disease. As expected, the most pertinent pathologic changes were in the respiratory and cardiovascular systems. This is not surprising because the major insult in the early phases of a fire is to the respiratory system.

Respiratory complications are significant because these can diminish both the transfer of oxygen to the blood and the removal of carbon diox-

ide. Not only do chemicals such as carbon monoxide and hydrogen cyanide deprive the system of oxygen, but deposits of soot and such pulmonary irritants as aldehydes and such acid gases as hydrogen chloride (HCl) contribute to anoxia. Pulmonary irritants cause edema and separation of mucosa from the epithelium of the respiratory tract. The soot particles, debris, and fluids introduced into the airways can cause varying degrees of obstruction, thus interfering with the victim's ability to inhale oxygen.

Heavy metal analysis. As a result of finding significant soot deposits in the respiratory tract of some fire victims, it was decided to determine the nature of these deposits. For this part of the study, soot was scraped from the trachea and bronchi and analyzed for heavy metals by atomic absorption spectroscopy. The results of some of these analyses are shown in Table X. Heavy metals in the body tissues of fire victims are thought to come predominantly from the heavy metals incorporated during formulation of synthetic polymers and paints. For example, antimony oxide is frequently added to polyvinylchloride to reduce its flammability. These metal compounds are vaporized during the fire and find their way into fire victims.

The toxicological significance of these metals in early death (within six hours) delayed death or slow recovery is not known. We suggest that short-term acute effects of these metals are not as important as the immediate effects of carbon monoxide, hydrogen cyanide, and pulmonary irritants because many victims die from these effects without the presence of metals.

For example, in one study of antimony toxicity in rats,[6] the most significant postmortem findings were cardiac lesions with myocardial congestion. A single exposure of dogs and cats for one hour to 40 to 45 ppm. antimony hydride (SbH_3) caused death within 24 hours. The animals had pulmonary congestion and edema.[7] A review of antimony and its compounds has been prepared.[8] Which compounds of antimony are the most prevalent in the fire environment and which of these are the most toxic have not been established. However, based on fire-retardant chemistry, one would expect the halides of antimony to be the predominant products. The toxicity of such other heavy metals as lead is well known. In a new study, attempts are being made to determine the possible effects of chronic exposures to heavy metals by following the recovery of survivors of the same fires.

The effects of pulmonary irritants, such as hydrochloric acid, fre-

TABLE VII

DISTRIBUTION OF BLOOD CYANIDE CONCENTRATIONS IN 272 FIRE FATALITIES

Cyanide concentration (μg./ml.)	*%*
0.00–0.25	31
0.26–1.00	35
1.01–2.00	24
>2.01	10
	100

TABLE VIII

FIRE FATALITIES IN MARYLAND: AVERAGE CYANIDE VALUES (μg./ml.)

Year	*Mean*	*SD*	*No.*
1975	1.12	0.87	89
1976	0.74	0.84	73
1977	1.05	1.04	83
1978*	1.05	0.85	34
Total	0.99	-	279

* Refers to only part of year.

TABLE IX

CORRELATION OF BLOOD CARBOXYHEMOGLOBIN WITH BLOOD HYDROGEN CYANIDE

Case no.	*Age*	*Cyanide (μg./ml.)*	*Carboxyhemoglobin (% saturation)*
13	37	0.30	61
14	26	0.93	45
16	25	0.42	64
17	45	0.05	59
18	20	0.43	58
21	59	1.64	58
23	22	0.59	60
24	19	1.06	58
25	18	1.83	77
42	18	0.35	76

TABLE X

RANGE OF HEAVY METAL CONCENTRATIONS IN INTERNAL SOOT SAMPLES FROM FIRE VICTIMS (NONSEPARATED, 94 CASES)

Metal	*No. of cases found*	*Range (μg./g. of sample)*
Sb	13	0-1479
Cd	66	0-121
Pb	50	0-767
Mn	8	0-28
Cu	76	0-376
Bi*	0	0
Be*	0	0
Cr†	0	0
Zn†	20	42-444

* Bi and Be, 74 cases analyzed
† Cr and Zn, 20 cases analyzed

quently result in delayed deaths, eight to 12 hours after exposure. Again, these deaths were not included in the study because it was limited to deaths within six hours of the fire. In addition to the delayed deaths due to acid gases, the effects and treatment of nonfatal victims have not been systematically studied to determine the extent of the problem and the best treatment for these victims.

Heart study. This part of the study explored the relationship between any preexisting heart disease, blood carbon monoxide levels, alcohol, and lethality. The premise was that individuals whose myocardial oxygen supply is already reduced by preexisting heart disease would be expected to succumb to lower levels of carbon monoxide. Further reduction in oxygen supply to the heart from preferential replacement of oxygen by carbon monoxide would then lead to heart failure. Knelson[9] has demonstrated that deleterious myocardial effects can occur at 3 to 5% carboxyhemoglobin in patients with angina pectoris.

One of the early and striking findings was the amount of heart disease, expressed in terms of coronary artery stenosis, in the fire victims and, in particular, in the youngest age group. Table XI depicts the maximum stenosis found in the coronary arteries of the victims subdivided into age groups. Of the 41 victims in the young age group (20 to 39 years), eight had more than 90% stenosis in at least one segment of their major coronary arteries. In addition to this unexpected amount of heart disease in the young, of the overall total 40% had at least one focus of 90% or more

TABLE XI

CORONARY ARTERIAL NARROWING IN FIRE VICTIMS*

Age	*0-24%*	*25-49%*	*50-74%*	*75-89%*	*90-100%*	*Total*
20-39	16	5	6	6	8	41
40-49	6	1	3	2	9	21
50-59	4	1	5	5	11	26
60-69	2	1	3	3	11	20
70 +	1	0	2	0	8	11
Total	29	8	19	16	47	119
	(24.4%)	(6.7%)	(16.0%)	(13.4%)	(39.4%)	(100%)

* Maximum observed in any one coronary branch.

stenosis and more than half had greater than 75% narrowing.

The relationship between blood carbon monoxide levels and coronary artery stenosis was examined. No constant relationship between the two factors was found. More victims with significant heart disease achieved a higher than 50% carbon monoxide saturation than died with lower levels. Further to explore the relationship between blood carbon monoxide content and coronary artery stenosis, men and women were studied separately because of the different pattern of atherosclerosis in the sexes. Of the 85 men studied, 44 had significant heart disease and of these victims 13 succumbed to lower than 50% carbon monoxide levels. More striking, however, was the finding that 31 of those with significant heart disease achieved greater than 50% carboxyhemoglobin levels before death, suggesting that those with cardiovascular disease do not succumb at a lower carboxyhemoglobin level.

It could be suggested that preexisting heart disease contributes to inability to escape from the fire but does not necessarily contribute to early death. This incapacitated group would then continue to inhale and to increase their amount of carbon monoxide. This cardiac-based incapacity would also explain the high incidence of heart disease in our series because the heart disease would "select" them to be fatalities. Another possibility is that coronary narrowing is compensated by anastomotic circulation around the occluded areas. However, before accepting these explanations, the role of alcohol was examined.

Alcohol has long been known as one of the major factors in house fires,

TABLE XII

ALCOHOL LEVELS, CORONARY DISEASE, AND CARBON MONOXIDE LEVELS IN MALE FIRE VICTIMS IN HEART STUDY

Alcohol levels	*Range 0–0.14% mean 0.05%*	*Range 0.15–0.24% mean 0.21%*
No. of males	36	37
Mean age (years)	45	45
Mean heart score	32	30
No. with heart score >30	21 (Mean COHb 63%)	16 (Mean COHb 62%)
No. with heart score >30	15 (Mean COHb 51%)	21 (Mean COHb 64%)

and various theories have been propounded regarding the interaction of carbon monoxide and alcohol in the death of fire victims. Blood alcohol determinations were done in all fire victims, so this was a factor that could readily be considered in analysis of fire deaths. Of the total of 119 victims, 92 (including 67 of the 85 men) had measurable amounts of alcohol in their blood at the time of death. There is no evidence to suggest that the presence of alcohol by itself has any effect on the level of carbon monoxide associated with death, and, although both are central nervous system depressants, there is no apparent additive effect.

Because of the relatively small group, only men were sufficient in number to warrant further analysis; they were divided into sober and inebriated. The sober individuals were taken as those with blood alcohol levels in the range of 0 to 0.14% and the inebriated in the range 0.15 to 0.29%. Those with blood alcohol levels of 0.30 and higher are excluded because, at this level, coma may be expected from the alcohol alone. Using this arbitrary division, we have two groups, one comprising 36 sober and the second 37 inebriated individuals. Of the sober individuals, approximately half of those with significant heart disease died with a low level of carbon monoxide while the remainder achieved over 50%. In the inebriated group, 18 of 21 individuals with significant heart disease had a greater than 50% carbon monoxide level. When these values are averaged out for the two groups we find that the sober individuals with heart disease had a mean carbon monoxide level of 51% while the inebriated victims had a mean level of 64%. Those without significant heart disease in both groups have similar carbon monoxide levels, 63% and 62% in the sober and inebriated respectively, as shown in Table XII.

Based on the above analysis, it would appear that those with significant heart disease live longer in a fire when under the influence of alcohol than when sober. There are several possible explanations, the simplest being that the sample is too small for the results to be valid. Another possibility is that inebriated individuals with heart disease survive longer because alcohol acts as a dilator of atherosclerotic coronary arteries in men. A third possibility is that coronary narrowing is compensated by anastomotic circulation around the occluded areas.

Discussion and Conclusions

The fire-fatality study defines the principal scenario that leads to the loss of human life due to fire. This scenario involves ignition of upholstered furniture or bedding by cigarettes or items of smoking in a private residence. In addition, alcohol is frequently involved in this scenario. The predominant cause of death is carbon monoxide, although heart disease was also found in a significant number of these fatalities.

The results of the other biological measurements raise a number of questions regarding fire fatalities, smoke inhalation, and materials burned. Some of these questions are: the significance of various levels of blood cyanide, the relative toxicologic significance of hydrogen cyanide and carbon monoxide in fire, the significance of antimony and other metals found in the respiratory tract, the contribution of heavy soot deposits in the respiratory tract to a state of asphyxia, and the significance of such sensory and pulmonary irritants as HCl and aldehydes.

Hydrogen cyanide was found in the blood of a substantial percentage of the victims. The role of hydrogen cyanide in contributing to fatalities is not well understood because all victims who had toxic levels of hydrogen cyanide also had carbon monoxide saturation of 50% or greater. It can be stated that cyanide does not explain deaths not already explainable by other causes, such as carbon monoxide. However, hydrogen cyanide may contribute to early incapacitation, although this has not been proved. Soot deposits that include concentrations of potentially toxic metals were found in a significant number of the fatalities. These metals include antimony, lead, and zinc. The effects of these metals and other products in the soot on injuries or on delayed deaths may be substantial and are basically unknown.

The significance of the above factors in contributing to fire fatalities or to the quality of life of survivors is poorly understood, as is the diagnosis and treatment of such victims.

The role of alcohol in the fire deaths appears to be most significant in contributing to starting a fatal fire but may not contribute directly to a fatality, that is, alcohol does not appear to cause deaths at a lower carboxyhemoglobin level than in the case of those who have not consumed alcohol. However, the victim may have been incapacitated by the alcohol itself. Preexisting heart disease also appears in a significant number of the fatalities and may contribute to incapacitation, hence failure to escape. However, those with heart disease do not appear to die at lower carboxyhemoglobin values than those who do not have heart disease.

SUMMARY

A detailed fire-fatality study determined the primary cause of death and the specific cause of fatality-producing fires. The study showed that: the predominant cause of death is carbon monoxide, a high percentage of the victims have elevated blood alcohol levels, a significant number of the fatalities had preexisting cardiovascular disease, and the predominant fatal fire scenario is cigarette ignition of upholstered furniture or bedding and has alcohol as a contributory factor.

Issues raised by the study and left unanswered include: the relative significance of hydrogen cyanide and carbon monoxide, the significance of antimony and other heavy metals found in the respiratory tract of victims as they relate to death and injury, the significance of heavy soot deposits in the respiratory tract, and the significance of such sensory and pulmonary irritants as hydrochloric acid from vinyl materials and aldehydes.

Questions and Answers

DR. JAMES BEALL (U.S. Department of Energy): You touched on an important point about animals and humans being different with relation to the respiratory tract, breathing, and so forth. What this generally indicates is that after inhalation exposures, rodents' noses reveal more signs of toxicity and, in humans, the lungs do so. Those are very general, sweeping statements.

If you have not done so, I would like to encourage you, in your future studies, to do several tissue sections through the nose of your rodents to look for changes there. If you have done it, I would like to hear about it.

DR. BIRKY: Some of that was done at the University of Pittsburgh. We have not done a lot of that because we are primarily looking for the in-

capacitating or lethal effects in the first tier screening of these materials.

MISS MARGARET CONOMOS (New Jersey State Health Department): Would you comment on what percentage of the fire victims were fire fighters?

DR. BIRKY: None of those victims were fire fighters. Other studies have been done on fire fighters, both of long-term lung compliance and of lethality in fire fighters. Those studies show that the primary cause of death in fire fighters is cardiovascular disease. The limited studies do not suggest that fire fighters develop reduced lung capacity over a period of time or that they develop respiratory diseases any more frequently than does the normal civilian population.

DR. MERRIL EISENBUD (Institute of Environmental Medicine, New York University): I have a related question. Did those earlier statistics, shown in the first slides, include industrial fires?

DR. BIRKY: No. This study was predominantly related to home fires. We have not studied industrial fires.

DR. NORMAN SCHELL (Nassau County Health Department): We have had some fires in school buildings and for the next two or three weeks the personnel and the students kept complaining about smoke irritation. Should we be looking for anything in particular in that type of air pollution?

DR. BIRKY: I am not sure how to answer that, but one of the reasons we think we underestimate the toxicity of fire products from materials such as polyvinylchloride is that fire fighters report severe respiratory difficulty after fighting a fire that has vinyl materials in it—at least they suspect that it is due to vinyl materials. But that seems to be more generally true of synthetic materials than of conventional materials.

It is these individuals that we are very concerned about because they are obviously the first exposed. We tell the fire fighting community that when they fight a fire involving synthetics and during clean up or overhaul, as they call it, they should make sure that they wear respiratory protective gear. Most of them do not, but this is changing slowly. Also, when a fire fighter complains of chest pains or sore throat he should be put under observation for 24 hours because we have had fatalities among fire fighters who just went back to the firehouse and died, without notice, without knowing why.

DR. EISENBUD: Pulmonary edema?

DR. BIRKY: I think that is correct.

DR. MAURICE E. SHILS (New York Academy of Medicine): Would you comment on the differences in causes of death among fires in homes,

tenements, apartments, or the Stouffer Hotel, for example, and what are the alcohol levels in tenement fires? Usually these start in one apartment and burn out many apartments, with deaths. Have any studies been done of alcohol levels in such fires as compared to that in Maryland which, I assume, involved a mixed population of homes and apartments?

DR. BIRKY: That is right; it is mixed. We have not sorted out the data in terms of private, single family residences versus multiple family residences. In fact, the statistics are probably too small to do that with any reliability. A study in Glasgow, almost identical to the Maryland one, began four or five years after we started ours. They found much higher blood levels of alcohol than we found here and, again, very strong correlation between the role of alcohol and these fire deaths. But I do not think I can answer your question about differences between residences specifically because we do not have the data broken down that way.

DR. EDWARD FERRAND (New York City Department of Environmental Protection): I agree that it is important and appropriate to discover what kills people in these fires, but I think it would be equally or maybe more important to do some work on the effects on people that survive. Are there any programs or any plans for programs to do this?

DR. BIRKY: Yes, as a matter of fact, there is. I agree with you, it is important to do such studies, including treatment.

We do have a small program at the University of Maryland on human treatment. There are some very serious problems concerning effects on humans that we have not been able to handle very well. One of those of course, is that it is very difficult for us to do a follow-up. The question is, what is the most appropriate treatment, and I throw that out for discussion. We would certainly like some insight into that. Follow-up studies are very expensive.

DR. FERRAND: Are there any investigations of the kinds of products of the fire? Some very irritating, destructive compounds are generated that may not cause immediate death but can destroy tissue and have long-term effects.

DR. BIRKY: Sure. I can give you selected examples of that. I have been in contact with an investigator who has been following up three women exposed to a relatively small fire. All three went their separate ways after the fire. All three have had tracheostomies supposedly as a result of that fire. I think it is probably related to the acid gases, primarily, but that is speculation on my part. In addition, gas sampling during fire fighting has been carried out in Boston and San Antonio.

DR. DONALD KENT (Life Extension Institute): Have you looked at any of the Navy statistics and their figures? They have some fairly long follow-ups after some shipboard fires, where sailors have been watched for years after they were involved in those fires.

DR. BIRKY: I am aware of those data, although I have not followed them closely. Of course, you are acquainted with the MGM Hotel fire, in which there were 83 fatalities. We have been up to our ears in those measurements and, again, there is still the problem of trying to identify the toxicant, because most of those victims were in upper floors of the structure above the fire and not on the fire floor.

DR. EISENBUD: Did you say that firemen do not show any chronic pulmonary stigmata?

DR. BIRKY: That is my understanding of the literature.

DR. EISENBUD: They should be an excellent cohort to study, because they must be exposed repeatedly.

DR. BIRKY: Yes, indeed.

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