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FIREFIGHTING AND HEART DISEASE

by
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Experimental and clinical evidence has accumulated in recent years supporting the view that certain strenuous occupations, such as firefighting, are capable of adversely affecting the cardiovascular apparatus to an appreciable degree. A fairly recent study of mortality by occupations among white male insurance policyholders shows that city firemen actually have the highest standardized relative index of mortality (125) for the principal cardiovascular renal diseases. While it has not always been possible to evaluate these disabilities equitably because of legislation based upon ancient views held in most states, sufficient evidence has been amassed in recent years suggesting the need for a wider acceptance of firefighting as an occupational factor in the production or aggravation of certain heart diseases.

Etiologic factors to be considered are environmental extremes (temperature and humidity), stresses and strains, trauma and shock, burns, and smokes and gases to which firemen are repeatedly subjected.

Stresses and Strains

Several cardiovascular diseases currently considered to be more or less independent clinical entities may be related to stress and strain in one way or another. Among these are hypertension, arteriosclerosis and certain collagen diseases.

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Expressed or repressed emotions or muscular effort may cause large rises in blood pressure. Hypertension may occur because one or more pressor mechanisms become unduly overactive in response to a systemic stressor. "Blast hypertension" has been found to persist for weeks in people who were in the vicinity of a major explosion such as the Texas City disaster.

Frost and Associates evaluated the effect of combined physical and mental stress on normal young healthy males. The "stress" in this study was the annual 500-mile Indianapolis Speedway Race. This is a competitive and dangerous contest in which the drivers race their cars around a two and one-half mile oval track at average speeds of between 120 and 125 miles per hour. There was evidence of increased pituitary adrenal stimulation during the stress period in that they showed marked decrease in the total number of circulating eosinophils after the race as well as an increased exertion of 17 ketosteroids of at least 50 percent following the stress as compared to the control period.

There is considerable clinical and experimental evidence that stress can also cause morphologic changes in the heart and that cardiac infarcts, hypertension and angina pectoris might be regarded as diseases of adaptation (stress). Hypertension, malignant nephrosclerosis and hyalinization with inflammatory arterial changes in the heart have been reproduced repeatedly by subjecting experimental animals to stress situations.

Numerous statistical surveys attest the fact that the incidence of arteriosclerosis appears to be definitely higher among individuals exposed to much stress and strain than in the population at large. The acceleration of arteriosclerosis with premature vascular breakdown is not uncommon as a result of the greater exposure of firemen and other individuals to hazardous physical tasks.

It is a well-known fact, though inadequately explained that hypertension and arteriosclerosis enjoy a synergistic relationship since the presence of one so frequently favors the earlier production of the other. Such an acceleration of normal atheromatous changes may result in premature changes in the coronary and other arteries, terminating in an anginal syndrome or myocardial infarction. Rupture of the capillaries in an arteriosclerotic intima immediately following sudden strain and trauma has been presented as evidence that they are possible precipitating factors in the development of coronary occlusion. The resulting hemorrhage may raise the plaque sufficiently to impede critically the coronary flow.

Forced or excessive muscular exercise so frequently experienced in firefighting and other strenuous occupations acts as the stressor agent and produces the alarm reaction of Selye. The goal of the circulation in exercise is to meet the enormous demands of the active muscles for oxygen and to help eliminate the carbon dioxide formed. Venous return is greatly increased with a markedly increased cardiac output. The output per beat (stroke volume) may reach 200 cc. and may be accompanied by a rise in blood pressure. It is obvious that the presence of underlying heart disease can be markedly affected by this change in cardiac dynamics which can disastrously affect a decreased cardiac reserve. This is especially true for those suffering from hypertension or coronary disease.

Numerous instances of myocardial infarction following great physical exertion are now on record. Anginal pain may be precipitated in patients with coronary sclerosis by induced anoxia. This phenomenon appears to be the consequence of both myocardial anoxia and the increased cardiac output and work due to lowered arterial blood saturation with oxygen.

Environmental Factors

A. Heat: Exposure to high temperatures with the accompanying increase in the rate of circulation through the lungs and skin may lead to a more rapid absorption of harmful chemical substances from the lungs (such as carbon monoxide) or through the skin (such as coal tar derivatives). Studies made in various laboratories and in some industries have shown that the amount of physical effort decreases as the temperature increases. Indeed, at very high temperatures, especially when associated with high humidity, physical work may become impossible. Heavy work in high temperatures adds materially to the burden already placed on the body. The circulation must compensate for this excess heat at a time when there is diminished heat loss due to the high temperature. Under these conditions, the body temperature tends to rise more rapidly and the strain on the circulation is great. The pulse rate increases while the stroke volume of the heart decreases markedly. When the heart attains its maximum rate, further work becomes impossible.

Yet, firemen and others engaged in emergency work are frequently forced to labor in such environments. In "weather sensitive" individuals even moderate changes in atmospheric conditions can act as stress agents and produce somatic changes. The possible effects on a person with underlying heart disease, such as coronary sclerosis, are obvious. Focal myocardial degeneration with renal changes similar to those seen in the "crush" syndrome have been found regularly during pathologic observations.

B. Cold: Exposure to extreme cold likewise causes stress which affects most tissues of the body. Hyalinosis of the heart with muscular hypertrophy have been produced regularly in rats exposed to cold. Even fibrinous pericarditis and fibrin deposits within the heart have been found.

Exposure to even moderate chilling causes an elevation in pulse rate and blood pressure often accompanied by various manifestations of vasospasm. In fact, continuous exposure to cold has been found to be a particularly effective type of stress in the experimental production of persistent hypertension, nephrosclerosis, cardiac hypertrophy and cardiovascular hyalinosis.

Trauma and Shock

Following extensive traumatic injuries and shock, numerous cardiovascular phenomena occur. These include a fall in arterial blood pressure and decreased stroke volume owing to insufficient blood return to the heart. Morphologic changes affecting all organs include capillary damage, petechiae and edema. Degeneration of the myocardium may occur and reveals characteristic changes consisting of granular appearance of the fibers with basophilia, liquefaction and uneven density. Cloudy swelling and fatty degeneration of heart muscle fibers are conspicuous in patients who die of traumatic shock. Reports of coronary thrombosis following surgical shock are now on record. Similar changes are frequently noted following shock and collapse, especially in the presence of underlying heart disease. Subendocardial infarctions may be found owing to acute coronary insufficiency. Cardiovascular changes may also result from electric shock. When immediately nonfatal, a rise in blood pressure and various arrhythmias have been noted. Severe electrocardiographic changes have been recorded during ordinary electroshock therapy. Shock associated with "live wires" to which firemen and others may be exposed can result in similar damage.

Burns

It has been recognized in recent years that extensive burns may also result in cardiovascular changes. Hemoconcentration and toxic absorption are important factors. Focal necrosis, calcilication and even the formation of granulomatous nodules have been observed in the hearts of rabbits. Some authors consider "serous myocarditis" with valvular edema to be quite characteristic of burns. It is often accompanied by panarteritis at a distance from the directly injured area, e.g., in the kidney. Less prominent degenerative lesions, such as cloudy swelling and fragmentation of the myocardial fibers have been noted in various species as well as in humans.

Smokes and Gases

Atmospheric concentrations of smokes and gases to which firemen are repeatedly subjected may cause acute attacks of anoxia. They result in an increase in blood pressure and other vasomotor phenomena. The physiologic action of these toxic substances is associated with an interference with oxidation-reduction processes in the body cells. It is interesting to note that other agents to which firemen are repeatedly subjected may act in a similar manner. They include marked exertion and exposure to extremes of temperature which have been discussed previously. In dogs exposed to acute anoxia, cardiac edema and hemorrhages occur as a result of capillary damage. A severe oxygen-want of this type causes a generalized increase in capillary permeability with a predominance of symptoms and findings in the lungs (hemorrhage and edema) rather than the heart. In the presence of incipient heart disease this may precipitate cardiac infarction or failure.

A combination of anoxia and emotional stimulus, such as may occur during smoke exposure, are simultaneously applicable to many firemen and it has been shown that they act synergistically in the production of morphologic changes. The importance of anoxia in relation to the size of the oxygen debt is illustrated by the observation that normal individuals breathing air containing low concentrations of oxygen have increased oxygen debts after work.

Hazards from the following smokes and gases are especially applicable to firemen. Intoxications from various burning or escaped chemical compounds may cause manifestations of stress as well as poisoning. In addition to carbon monoxide which is generated by burning materials even in the presence of an excess of air, other irritating and lethal gases form which are synergistic in action. These include carbon dioxide, hydrocyanic acid, ammonia, hydrogen sulfide and sulfer dioxide. They are formed in the presence of burning rubber, silk, wool and many other organic substances. The presence of carbon dioxide induces increased respirations and more rapid death. Escaped gases from refrigerating and other industrial systems may also produce toxic changes (e.g., ammonia, formaldehyde, carbon tetrachloride, methyl chloride and other refrigerants).

Carbon monoxide has an affinity for hemoglobin which is up to 300 times greater than for oxygen. This common gas produces an oxygen-want in tissues, which in the presence of underlying heart disease, may proceed to myocardial infarction. Chronic exposure to low concentrations for long periods may also produce permanent injury. Unfortunately, many of these cases are returned to duty following resuscitation and a careful search for cardiac damage is seldom performed.

Symptoms and signs specifically related to the cardiovascular system include fatigue, dizziness, palpitations, dyspnea on slight exertion and precordial pain. Early exposure to low concentrations causes a rise in the diastolic blood pressure and rapid heart rate. Eventually, the systolic blood pressure may become elevated. This depressant action may lead to circulatory collapse as a result of marked diminution in muscle tonus and a failure of the venous blood to return to adequate quantity to the right heart. In man and experimental animals, this sequence has been followed electrocardiographically to the very moment of collapse and two findings are almost always noted: (1) a progressive diminution in the height of all T waves, beginning at oxygen concentrations of around 14 percent and (2) a moderate depression of the ST segments. This objective evidence reflects the direct effect of anoxia upon the myocardium as well as changes due to the large outpouring of potassium from anoxemic tissues throughout the body.

SUMMARY

(1) Hypertension, coronary thrombosis, the anginal syndrome, and manifestations of accelerated atheromatous changes are especially prone to occur in firemen and related dangerous occupations because of certain mental and physical factors associated with these occupations.

(2) Adequate experimental and clinical evidence has been accumulated to show that the stresses and strains of firefighting, environmental extremes, trauma and shock, burns, and gases and smokes may act as predisposing factors in the causation of several cardiac disorders.

(3) An aggravation of preexisting heart disease may also occur in the presence of the above factors.

GLOSSARY

Angina Pectoris:	A disease - due most often to anoxia of the myocardium and precipitated by effort and excitement.
Angina:	Spasmodic choking or suffocation pain.
Anoxia:	Oxygen deficiency.
Arrhythmia:	Variation from normal heart beat rhythm.
Arteriosclerosis:	Disease of the arteries marked by the formation of fibrous nodes or plaques in the lining membranes of the arteries.
Atheromatus:	Arteriosclerosis with marked degenerative changes.
Basophilia:	A discolor from basic - staining cells.
Cardiovascular:	Pertaining to the heart and blood vessels.
Collagen:	An albuminoid, the main organic constituent of connective tissue and of the organic substance of the bones.
Coronary:	Applies to vessels, nerves, ligaments, etc.
Dyspnea:	Difficult or labored breathing.
Edema:	Presence of abnormally large amounts of fluid in tissue spaces.
Eosinophil:	A structure, cell or histologic element readily stained by dyes.
Etiology:	The study or theory of the causation of disease.
Fibrinous Pericarditis:	Chronic inflammation in which adhesions become replaced with fibrous bands.
Granulomatus:	Tissue tumor.
Hyalinization:	Producing starchy deposits.
Hypertension:	Abnormally high tension; especially high blood pressure.
Hypertrophy:	Enlargement of an organ or part.
Infarct:	An area of coagulation resulting from obstruction of circulation in the area.

Glossary (cont'd)

Intima:	Innermost of three coats of an artery.
Ketosteroid:	The 17 Ketosteroids have keytone (carbonyl) groups on the carbon atom. They are found in urine of normal men and women.
Morphologic:	Pertaining to the science of the forms and structures of organized beings.
Myocardial:	Relati[illegible]mary cardiac insufficiency.
Necrosis:	Death o[illegible]on of tissue.
Neophrosclerosis:	Scleros[illegible]rdening of the kidney.
Petechiae:	A small spot formed by the effusion of blood.
Renal:	Pertaining to the kidney.
Sclerosis:	An induration, or hardening.
Serous:	Pertaining to serum.
Somatic:	Pertaining to the body.
Syndrome:	A complex of systems.
Synergistic:	Acting with another medicine or agent.
Systemic:	Pertaining to the whole body.
Subendocardium:	Below the membrane heart lining.
Trauma:	A wound or injury.
Panarteritis:	Inflammation of tissues around an artery.
Precordial:	The region over the heart or stomach - lower part of thorax.
T Waves and TS Segments:	Electrocardiograph deflection upward and downward of ventricular processes.
Vascular:	Pertaining to or full of vessels.
Vasomotor:	Presiding over movement of walls of blood vessels.
Vasospasm:	Spasm in blood vessels with decrease in their caliber.

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Carbon Monoxide—the Oklahoma Study

The development of professional fire services in the United States has been accompanied by specific health outcomes among its workers. The 855,000 firefighters in this country (189,000 fulltime, 666,000 volunteers), who were responsible for extinguishing $2.3 billion dollars in losses in 1972, have been cited as having the highest job related death rate of any occupation in this country. Clinical, experimental and actuarial reports have also identified certain disability and mortality risks incurred by this sector of the working population.

Although the decline of the relative mortality ratio from 134 to 120 percent among insured firefighters has been consistent with mortality decreases in many occupations, the severity rate, indicating days of disability per million man-hours of work, has reached nearly one and a half times the average of all industries. Death and permanent disability figures have risen to 3.53 per million man-hours, which is the highest of all municipal employees and comparable to the hazards of mining.

The incidence of cardiovascular and respiratory disease among professional firefighters has for years been considered to be occupationally related.

What is common knowledge in the fire service, to those firefighters on disability retirement and to the widows of professional firefighters has scant substantiation in the scientific literature.

In 1974 the Oklahoma City Professional Firefighters (Local 1524, IAFF) and the University of Oklahoma entered into a research program (in an attempt) to elucidate some of the causal factors involved as well as to attempt to ascertain if, in fact, there was any way to practically quantify the various parameters involved. To date there have been three studies completed and reported upon. One has been published in the Journal of Occupational Medicine (August 1974) and hopefully the others will soon be published.

Before the individual projects are discussed it must be emphasized that these studies deal with a selected population in a defined geographical area. The results, significant as they are, must be considered directly applicable to this population only. This in no way is suggestive that they are invalid and without scientific merit but as will be seen in the second part of this presentation literally demand that a centralized and controlled research effort be made to extend the results to all members of the fire service regardless of their geographical location. These data can be considered as strong positive indicators of the occupational factors working to increase the mortality and morbidity, as well as exerting life-shortening influences on members of the fire service.

The first study had as its primary purpose to determine if an occupational group exposed repeatedly to sub-acute episodes of carbon monoxide inhalation had a significantly higher residual body burden of carbon monoxide in its various physiological forms. Carbon monoxide was chosen as an indicator because of its ubiquitiousness in the fire-smoke complex, its physiological reactivity and the ease with which it can be measured as carboxyhemoglobin.

During initial literature research it became obvious that repeated and/or continuous exposures at substantially lower concentrations than are usually considered detrimental to health can and do exert a detrimental effect on the body and can result in intracellular damage to heart and nervous tissues. In those cases where death was *not* rapid and *not* due to massive excursions with the agent the majority of the tissue slides reviewed had intracellular changes. Diffusely distributed focal myocardial injury and necrosis as well as leukocytic infiltration and punctate hemorrhages were the most common findings.

These data were considered of sufficient importance to include a modified enzyme battery to test the theory

that minimal intracellular damage was occurring in the test (firefighter) population and not in the control group. Each firefighter had a paired or control subject matched as colsely as was possible with the exception of exposure to the fire-smoke complex.

The conclusions drawn after the five month study of 36 firefighters were:

1. The non-smoking firefighter had already achieved the maximum allowable COH_B Saturation under NIOSH Guidelines.

2. The test group, as a whole, xceeded the COH_B content that would have been achieved if they labored at heavy work for 1440 minutes (24 hours) in an atmosphere of 42 MG/M^3 (50 PPM) CO.

3. As a group, the test population exhibited changes in enzymes that suggest heart damage resulting from repeated, chronic sub-acute exposures to CO.

4. For this group of firefighters and their controls, the observed differences could best be attributed to occupation.

The second study dealt with pulmonary function studies of 549 firefighters and 151 controls. The initial health profile of both test and control populations did not reveal marked diseases of the heart, lungs, liver, kidney, or nervious systems, but some health issues such as bronchitis, asthma and possible emphysema were discovered during the testing. The study plan was to determine if, after proper corrections for such factors as age, smoking and prior lung disease, the professional firefighter suffers a decrement in his lung functions.

Based on the comparison of the test results with the control results as well as statistical comparisons with accepted normals and population values the following conclusions were drawn:

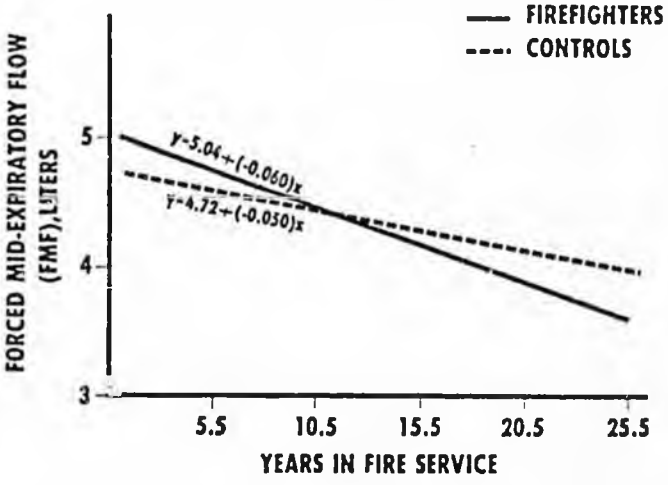
1. The vital capacity, forced vital capacity and flow rates decreased in firefighters as in controls but the firefighters mean values declined at a faster rate; even though the test population excelled the control population in height and weight.

2. Nonsmokers were superior to smokers in the flow rate mean values, which suggested smoking potentially impairs lung capacity. The mean values were found significant for the firefighters in both FEVI and the FMF, but only in the FMF for the controls. Smokers were at high risk through cigarette smoking and when the firefighting exposure was added this undoubtedly represented an increased potential health hazard.

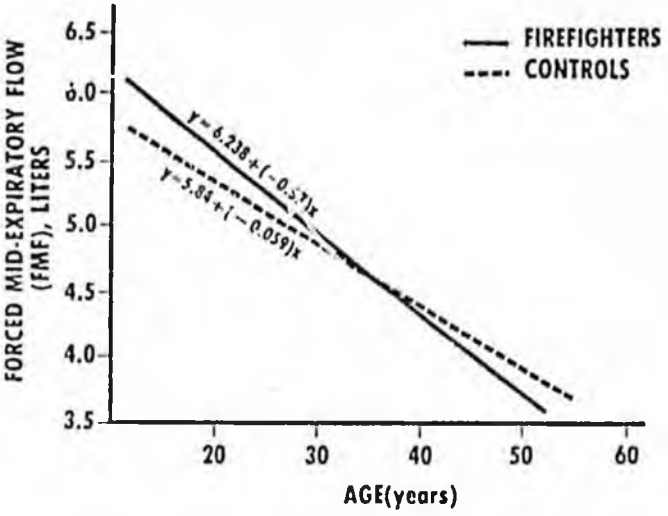
3. The observed differences between the test and control groups could best be attributed to occupational exposure as indicated by reduced volumes suggestive of airway obstructive and restrictive diseases especially when considered in light of the fact that the worst cases have been retired.

The third study is possibly of the most immediate significance in that, it addresses itself specifically to the health effects of firefighting as a profession. The population studied included all occupational categories having firefighting experience, all ranks and all classes of employment (active, retired, separated). The active group represented 23,354 unadjusted person-years of risk for the period 1930 to 1972. Five hundred fifty-six members were included in the separated population and 322 were in the pensioned group. This population provided a working population with sufficient history so that latent effects could be observed and large numbers examined.

FORCED MID-EXPIRATORY FLOW VERSUS YEARS IN FIRE SERVICE



FORCED MID-EXPIRATORY FLOW VERSUS AGE



Based upon the findings of this study the following conclusions were made:

1. The lack of routine medical evaluations to assess non-service related chronic or acute disabilities in terms of early identification of high risk personnel exerted a compensating force on the beneficial effects of pre-employment screening.

2. Significantly increased mortality from cardiovascu-

lar and respiratory disease was observed in the study population for all periods. Failure to identify a declining trend through significant changes in the standard mortality ratio was sufficient to reject any suggestion that improved mortality experiences necessarily followed improved services.

3. The course of mortality due to cardiovascular and respiratory disease indicated a significant shortening of the life span for this group. Although autopsy data from other causes of death were not available the inspection of such records would undoubtedly further increase the prevalence of cardiovascular and respiratory disease.

4. Individuals who have entered a stressful occupation such as the fire service at an older age have been occupationally associated with a disability experience. The inference that higher ages of accession somehow predisposed individual firefighters to premature and chronic disability was substantiated since the population of disability retired personnel demonstrated a markedly higher mean (average) entry age. Either through errors in prescreening or "negative" self-selection (a firefighter with a known pre-existing condition voluntarily elects to expose himself to the hazards of the fire service) *30* percent of all retired personnel were disabled.

These three studies, very briefly reported on here, reinforce (the theory) that there are (real) occupational factors involved in the fire service that result in increased mortality, higher numbers of disability retirements, and attendant life shortening. In addition to the conclusions reached, the investigators made several recommendations. The recommendations pertain to facts that are common knowledge to those who are familiar with the fire service but their acceptance is problematical.

By concerted efforts, politically, at the bargaining table and through the support of intensified research efforts the professional firefighter can bring about changes that will materially improve his health. These changes can be made through understanding and controlling of the occupational environment. The following recommendations are offered as avenues towards achieving these goals.

1. Establish a center of excellence where the health effects of the fire service can be evaluated and research efforts coordinated. Initial emphasis should be placed on the cardiovascular and respiratory diseases.

2. Establish a central data registry to collect occupational safety and health data for all professional firefighters. This will provide a data base to both researchers and bargainers.

3. Work for the acceptance of cardiovascular and respiratory disease as an absolute risk that can be caused or aggravated by agents in the firefighters occupational environment.

4. Work for the adoption of uniform entry requirements, preemployment physical exams, periodic physical examinations and adequate medical evaluations following occupational exposures such as smoke inhalation episodes.

5. Evaluate the possibility of establishing an entry age limit of less than 30. The disability experience of the ages above 30 have definite predictive value as a firefighter selection criterion. Findings are suggestive that a lower age limit coupled with more discriminate physical requirements should result in fewer disability retirements, longer service histories and extended post-separation survival time.

6. Urge or unilaterally adopt occupational safety and health administration recordkeeping requirements, utilizing ANSI Z16.2 standardized reporting methods.

7. The traditional labor/management emphasis of establishing special benefits for disabled workers should

COMPARATIVE CARDIOVASCULAR-RENAL MORTALITY AMONG FIREFIGHTERS 1930-1973

	STUDY PERIODS											
	1930-1940			1941-1951			1952-1962			1963-1973		
AGE	Expected Rate/ 1000	Observed Rate/ 1000	P Value	Expected Rate/ 1000	Observed Rate/ 1000	P Value	Expected Rate/ 1000	Observed Rate/ 1000	P Value	Expected Rate/ 1000	Observed Rate/ 1000	P Value
20-29	.2	—	—	.08	—	—	.1	—	—	.07	—	—
30-39	.5	3.0	.05	.3	3.6	.05	.6	.6	NS	.51	—	—
40-49	1.8	2.4	NS[a]	1.5	2.5	NS	2.3	4.0	NS	2.1	3.9	NS
50-59	5.1	23.4	.05	4.7	3.6	NS	6.5	10.3	NS	6.1	8.0	NS
60-69	12.7	12.7	NS	11.1	18.2	NS	17.1	15.2	NS	14.5	14.0	NS
70-	39.3	10.3	NS	28.3	21.3	NS	56.8	110.0	NS	41.5	64.3	NS
30-69	2.3	5.5	.01	3.2	4.6	NS	4.5	5.3	NS	6.4[b]	7.7	NS
30-70-	3.9	6.0	NS	4.1	5.2	NS	6.3	9.0	.05	9.1[c]	12.0	.05

a Not significant at the .05 level.
b 40-69.
c 40-70.

shift to effecting substantive improvements in the work environment. The development or improvement of any comprehensive safety and health program should not be solely oriented to mechanical compliance with standards and should be extended to include active participation by the entire fire service in safety and health analysis.

Feasible improvements in the work environment should yield benefits for employees such as a decrease in lost wages, lower insurance premiums, reduced medical expenses and a stronger retirement fund. The community and management should gain with higher productivity by a reduction in absenteeism and a generally healthier working class, decreased compensation benefits and lower administrative costs particularly in the areas of claims litigation and replacement personnel training.

AGE-SPECIFIC MORTALITY FOR FIREFIGHTERS

AGE	1930-1940			1941-1951		
	Expected Rate/1000	Observed Rate/1000	p < (.30-.001)	Expected Rate/1000	Observed Rate/1000	p < (.30-.001)
20-29	3.50	6.45	NS	2.40	1.25	NS
30-39	4.60	5.00	NS	7.80	10.83	.001
40-49	7.40	5.97	NS	5.60	7.43	NS
50-59	13.70	35.16	.01	12.70	8.46	.30
60-69	31.30	25.32	NS	26.90	22.73	NS
70-	87.40	10.31	NS	79.50	31.91	.10
20-69	7.80	9.80	.10	7.60	7.8	.02
20-70	10.80	9.80	.01	9.40	8.5	.01

NS figures not significant p < .3.

AGE-SPECIFIC MORTALITY FOR FIREFIGHTERS

AGE	1952-1962			1963-1973		
	Expected Rate/1000	Observed Rate/1000	p < (.30-.001)	Expected Rate/1000	Observed Rate/1000	p < (.30-.001)
20-29	1.60	.99	NS	1.80	.65	.30
30-39	2.50	2.39	NS	2.40	2.16	NS
40-49	5.60	7.91	NS	5.80	8.88	.10
50-59	12.70	17.51	.20	14.10	13.61	NS
60-69	30.50	26.52	NS	31.50	22.65	.10
70-	86.40	200.00	.001	92.90	102.83	NS
20-69	7.70	8.50	NS	9.10	8.30	.20
20-70	9.90	14.00	.001	12.70	12.40	.30

NS figures not significant p < .3.

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Report on Studies of Exposures To the Fire Environment

Thank you Dr. Conrad, and good morning ladies and gentlemen. I am very pleased to be here at the Third Redmond Fund Symposium on the Occupational Health and Safety Hazards of the Fire Service. I would like to take the opportunity this morning to discuss briefly the research we have been doing at the Johns Hopkins School of Public Health under a grant from the National Science Foundation through its RAND Program. Our studies have taken two main directions. First, we have been looking at acute and chronic effects of exposure to the fire environment on the fire fighters; and secondly, we have been looking at civilian casualties who are trapped in the fire environment. On behalf of Dr. Ted Radford and our team, I would like to express our very deep appreciation for the excellent cooperation we received from the IAFF, from the Baltimore Fire Department, and from the fire fighters themselves. Our efforts could not have proceeded without their generous assistance.

As a measure of *acute* exposure of the fire fighters, we have examined carbon monoxide in blood samples taken at the fire ground. A registered nurse, and an I.V. technician rode with the Fire Chief to fires which were considered to be fairly significant exposures. Over 500 blood samples were drawn on the fire ground as soon as feasible after the men left the fire itself. These samples were then taken to our laboratory and analyzed for the amount of carboxyhemoglobin in the blood. The results as shown in Figure 1 compare the cumulative frequencies of blood carboxyhemoglobin found in fire fighters and in controls. These are broken down into both smoking and non-smoking groups. The data show that while the majority of men have received exposure to carbon monoxide which is not significantly greater than that which resulted from cigarette smoking alone, there is a small percentage of men, both in the smoking and in the non-smoking group, who have received enough exposure to carbon monoxide to raise their carboxyhemoglobin level to 15-20% of the hemoglobin in their blood. We feel that this exposure is significant, especially in the light of previous studies that have been done by our group. These studies, which are reported elsewhere, were done on civilian fatalities in fires and show that there is an additive effect between the level of carbon monoxide absorbed by the blood and the degree of narrowing of the coronary arteries. In other words, these studies show that people who are compromised by coronary artery disease are significantly more sensitive to carbon monoxide in the atmosphere than are people who are not so compromised. In light of these studies then, we feel that our findings of carboxyhemoglobin levels of 15-20% in a very small percentage of fire fighters may, in fact, be evidence of significant exposure, especially if some compromise of the coronary artery circulation is present.

Figure 2 shows the levels of thiocyanate in the blood of the fire fighters. This is a measure of the cyanide gas which may be produced in a fire, and it must also be differentiated from that caused by cigarette smoking. This data does not show as clear a relationship to exposure as we have seen with carbon monoxide. Further studies are now being developed using new techniques for measuring cyanide in the blood and we hope to pin this relationship down more completely.

Our studies of *chronic* exposures of fire fighters are now underway. We are looking at the long-term chronic effects of fire fighting on the heart and the lung. This study has three main parts to it and requires voluntary

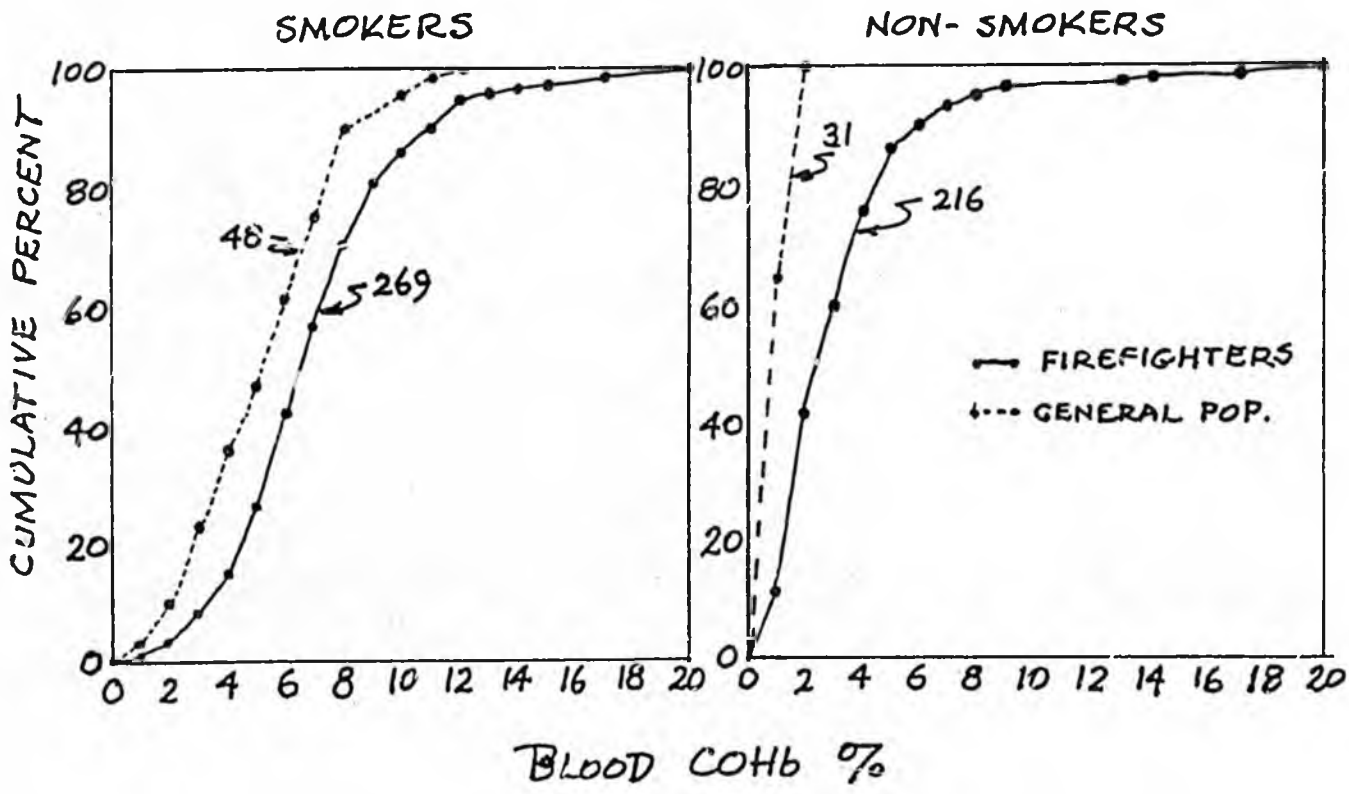


FIGURE 1—Cumulative Frequences of Blood COHb—Firefighters and Controls

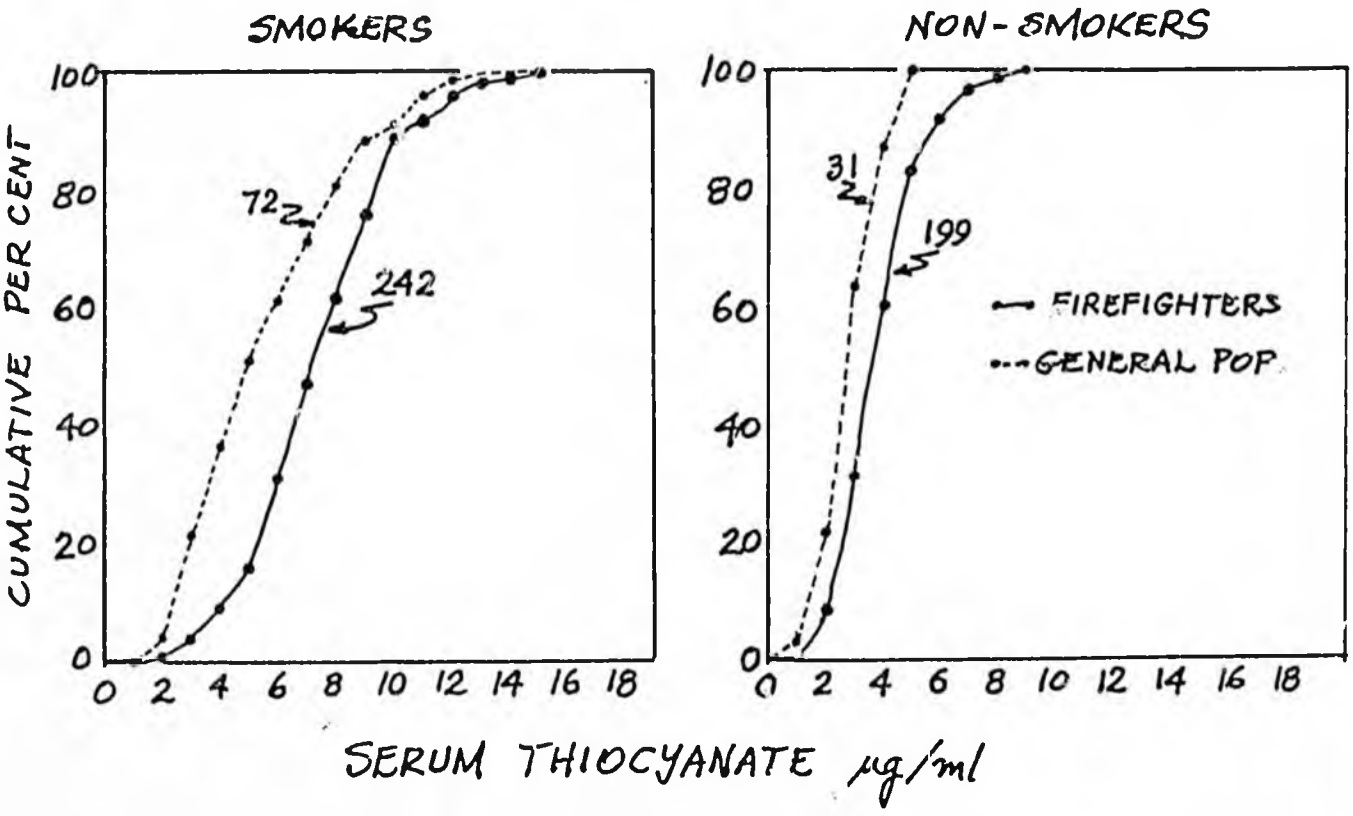


FIGURE 2—Cumulative Frequencies Serum SCN—Firefighters and Controls

participation of the men at each individual fire house. All information is, of course, held in the strictest confidence. These investigations are in the early phases, and no data is available at this time. The first part of the study consists of a respiratory questionnaire which is adapted from the British Chronic Respiratory Disease Questionnaire and in addition records some historical information about other past medical diseases; the second part of the chronic study consists of pulmonary function tests, particularly the forced expiratory volume in one second (FEV), and the forced vital capacity (FVC); the third part of this chronic study looks at cardiovascular effects of chronic exposure as measured by blood pressures and resting EKG's. As will be discussed later in this meeting by Dr. Barnard, we will take a sample of our normal participants and offer them maximum stress testing as well. The results of these studies will then be compared with the expected rates of disease and disability found in the general population. We also intend to make a correlation with "levels of exposure". We are using measures of exposure which are for the most parts subjective, consisting of the number of beatings taken at the fire ground, the amount of black sputum which is brought up after the fire, the number of times oxygen is taken, and the number of times the patients have been hospitalized for exposure or smoke inhalation as well as age and years of service. The final phase of this particular portion of our project will consist of a mortality study of the fire fighters themselves, in which we hope to compare the mortality experience of fire fighters to that of the general population.

The second major thrust of our investigations has concerned civilian casualties who are caught in fires. We obtained all hospital and autopsy records on any patients for whom a fire card was made out by the Baltimore Fire Department for a 14 month period from January, 1973 to March, 1974. Figure 3 will show that during that time there were 414 injuries, 58 of which ended fatally, and 356 of which were taken to the hospital for treatment. Figure 4 will show that looking at the fatalities (58) we had 6 who were pronounced dead at the scene of the fire; 25 who received some resuscitative measure en route to the hospital, but to no avail and were pronounced dead upon arrival at the hospital, and the remaining 27 patients who were kept at the hospital and treated for varying lengths of time prior to their fatal outcome. Our preliminary data shows

FIGURE 3

Injured persons transported from scene of fire by Baltimore City Fire Department

(January 1973-March 1974)	414
Fatalities	58
Casualties	356

FIGURE 4—Fatalities (Preliminary Data)

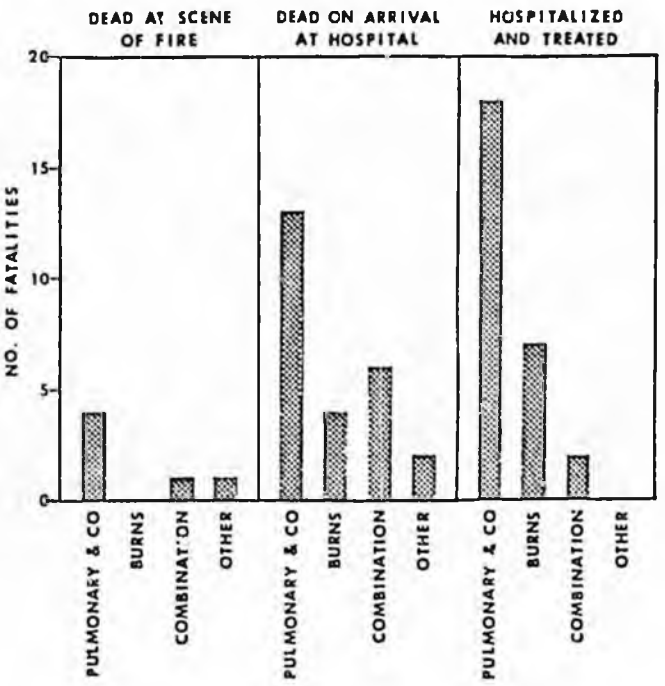
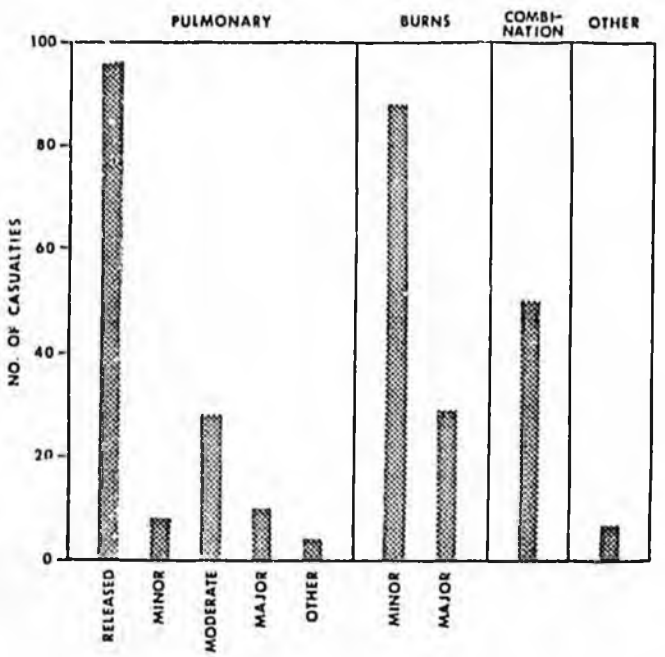


FIGURE 5—Casualties (Preliminary Data)



that in each of these three categories the major cause of death was "pulmonary problems and carbon monoxide." We hope to be able to separate these causes of death when our computer data is completed. Figure 5 shows a breakdown of the casualties who were taken to the hospital and treated and survived and again, we see that pulmonary problems were the major cause of disability. In both pulmonary and burn categories, however, the majority of people were treated and released and less than half had what could be considered moderate or major injuries from the fire exposure. As this data is computerized, we will be able to determine both the exact causes of death and mechanisms of disability of these casualties, and we will be able to take a close look at the pre-disposing diseases and the circumstances which existed prior to the fire, such as heart disease, diabetes, alcoholism, smoking and other pertinent problems.

I have presented a brief overview of our efforts, and some of our preliminary findings. As our investigations proceed and as our data becomes more refined, we hope to be able to make some more definitive statements about the types of problems which arise in the fire fighting population due to specific hazards faced in fighting fires, and add our findings to the excellent work being done at other centers.

We also hope to be able to add to the understanding of the causes of death and disability in casualties with suggestions to the medical community for more efficient and effective treatment. Thank you very much.

HAZARDOUS MATERIALS
IN THE
FAIRBANKS NORTH STAR BOROUGH

NAME		CLASS	NO.
1-PROPANOL		flam. liq.	1274
2-ETHOXYETHANOL		comb. liq.	1171
2-PROPANOL		flam. liq.	1274
ACETIC ACID	GLACIAL	corrosive	2790
ACETONE		flam. liq.	1090
ACETYLENE		flam. gas	1001
ACID	ACETIC	corrosive	2790
ACID	HYDROCHLORIC	corrosive	1789
ACID	HYDROCHLORIC anhyd.	nonfl.gas	1050
ACID	NITRIC 70%	oxidizer	2031
ACID	*NITRIC fuming	oxidizer	2032
ACID	NITRIC other than fum.	corrosive	1760
ACID	n.o.s.	corrosive	1760
ACID	PERACETIC	org. per.	2131
ACID	SULFURIC	corrosive	1830
ACID	SULFURIC, fuming (20%)	corrosive	1831
ACID	SULFURIC, tech.	corrosive	1830
ACRYLONITRILE		flam. liq.	1093
ADIPYL CHLORIDE		corrosive	
ALCOHOL	denatured	flam. liq.	1095
ALCOHOL	n.o.s.	flam. liq.	1987
ALCOHOL	poisonous, n.o.s.	flam. liq.	1986
ALCOHOL	WOOD	flam. liq.	1230
ALLYL ISOTHIOCYNATE	tear gas	irritant	1545
ALUMINUM CHLORIDE	crystal		1726
ALUMINUM PERCHLORATE		oxidizer	----
AMMONIA	*ANHYDROUS	nonfl.gas	1005
AMMONIUM BIFLUORIDE	solid	corrosive	1727
AMMONIUM BIFLUORIDE	solution	corrosive	2817
AMMONIUM NITRATE	FERTILIZER	oxidizer	2067
AMMONIUM NITRATE	FUEL OIL MIXTURE	high exp.	----
AMMONIUM PERCHLORATE		oxidizer	1442
ANTI-FREEZE	dry or liquid	flam. liq.	1142
ANTIMONY PENTACHLORIDE		corrosive	1771
ARGON	compressed	nonfl.gas	1006
ARSENIC TRICHLORIDE		poison B	1560
ASPHALT		flam. sol.	1993
AVIATION FUEL	turbine engine	flam. liq.	1863
BENZINE		flam. liq.	1115

* = major evacuation required in spill

BLASTING AGENTS	n.o.s.	blast. ag.	----
BOMBS	SMOKE	corrosive	2028
BORON TRIFLUORIDE	*	nonfl.gas	1008
BROMINE	*	corrosive	1744
CALCIUM CHLORITE		oxidizer	1453
CALCIUM HYPOCHLORITE		oxidizer	1748
CARBON DIOXIDE		nonfl.gas	1013
CARBON DIOXIDE	liquified	nonfl.gas	2187
CARBON REMOVER		flam. liq.	1132
CHLORINE	*	nonfl.gas	1017
CHROMATE 84D/47	PRIMER	flam. liq.	1263
CLEANING COMPOUND	160Z	comb. liq.	1142
CLEANING COMPOUND	liquid corrosive	corrosive	1760
CYANIDE		poison B	1588
CYCLOPROPANE		flam. gas	1027
DINITROBENZENE		poison B	1597
DIOXANE		flam. liq.	1165
ETHANOL		flam. liq.	1170
ETHYL MERCAPTAIN		flam. liq.	2363
ETHYLENE		flam. gas	1962
ETHYLENE GLYCOL		comb. liq.	1153
ETIOLOGIC AGENTS	n.o.s.	etiol.ag.	2814
EXPLOSIVES A		expl. A	----
EXPLOSIVES B		expl. B	----
EXPLOSIVES C		expl. C	----
FERTILIZER	AM. SOL.	nonfl.gas	1043
FLAMMABLE GAS	n.o.s.	flam. gas	1954
FLAMMABLE LIQUID	corrosive n.o.s.	flam. liq.	2924
FUEL	AVIATION turbine eng.	flam. liq.	1863
FUEL OIL		comb. liq.	1993
GAS	compressed or liquid	nonfl.gas	1956
GAS	FLAMMABLE n.o.s.	flam. gas	1954
GAS	liquified nonflam.	nonfl.gas	1058
GAS	liquified nonflam.	nonfl.gas	1056
GAS	LIQUIFIED PETROLEUM	flam. gas	1075
GAS	PROPANE	flam. gas	1075
GAS	RARE, mixed with o^2	nonfl.gas	1980
GAS	REFRIGERANT n.o.s.	nonfl.gas	1078
GASOLINE	blended	flam. liq.	1203
GRENADE	TEAR GAS	irritant	2017
HELIUM	compressed	nonfl.gas	1046
HEXANES		flam. liq.	1206
HYDRAZINE		corrosive	2030
HYDRAZINE		flam. liq.	2029
HYDROCHLORIC ACID	*anhydrous	nonfl.gas	1050
HYDROCHLORIC ACID	solution	corrosive	1789

HYDROGEN FLUORIDE	* =	corrosive	1052
HYDROGEN PEROXIDE		oxidizer	2014
HYPOCHLORITE	solution	corrosive	1791
INSECTICIDE	dry n.o.s.	poison B	1615
INSECTICIDE	dry n.o.s.	poison B	2588
INSECTICIDE	gas n.o.s.	nonfl. gas	1968
INSECTICIDE	gas poisonous n.o.s.	poison A	1967
INSECTICIDE	liquid n.o.s.	flam. liq.	1993
INSECTICIDE	liquid poisonous n.	poison B	2902
ISOPROPANOL		flam. liq.	1219
KEROSENE	including jet fuel	comb. liq.	1223
KETONE	METHYL ETHYL	org. per.	2563
LIME	SODA	corrosive	1907
MAGNESIUM	ALLOY	flam. sol.	1869
MAGNESIUM PERCHLORATE	ANHYDROUS	oxidizer	1475
MAGNESIUM TURNINGS		flam. sol.	1869
MALATHION		ORM-A	2783
MERCAPTAIN	ETHYL	flam. liq.	2363
MERCAPTAIN	n.o.s.	flam. liq.	1228
MERCURY WASTES		poison B	2025
METHANE	liquid	flam. liq.	1972
METHANOL		flam. liq.	1230
METHYL ETHYL ETHER		flam. liq.	1039
METHYL ETHYL KETONE	PEROXIDE	org. per.	2563
METHYL-ISO-BUTYL KETONE		org. per.	2126
N-BUTYL ALCOHOL		flam. liq.	1120
NAPTHA		flam. liq.	2553
NAPTHA	DISTILLATE	flam. liq.	1268
NAPTHA	PETROLEUM	flam. liq.	1255
NAPTHA	SOLVENT	flam. liq.	1256
NEON	compressed	nonfl.gas	1065
NITRIC ACID	40% or less	corrosive	1760
NITRIC ACID	70%	oxidizer	2031
NITRIC ACID	*fuming	oxidizer	2032
NITRIC ACID	*red fuming	oxidizer	2032
NITROGEN	compressed	nonfl.gas	1066
NITROGEN	cryogenic liquid	nonfl.gas	1977
OCTYL TRICHLOROSILANE		corrosive	1801
OIL	PETROLEUM	flam. liq.	1270
ORGANIC PEROXIDE MIXTURE			2756
OXIDIZERS	corrosive liquid n.	oxidizer	9193
OXIDIZERS	corrosive solid n.o.s.	oxidizer	9194
OXYGEN	compressed	nonfl.gas	1072
OXYGEN	liquid	nonfl.gas	1073
PAINT		flam. liq.	1263
PAINT REMOVER		comb. liq.	1142

PAINT STRIPPER		corrosive	1760
PCB		ORM-E	2315
PERACETIC ACID		org. per.	2131
PESTICIDE	liquid n.o.s.	poison B	1996
PESTICIDE	liquid poisonous	poison A	1995
PESTICIDE	liquid poisonous	poison A	2902
PESTICIDE	liquid poisonous	poison A	2903
PESTICIDE	solid n.o.s.	poison B	2588
PETROLEUM	CRUDE OIL	flam. liq.	1267
PETROLEUM	OIL	flam. liq.	1270
PETROLEUM	SPIRITS	flam. liq.	1271
PHOSPHORUS	AMORPHOUS RED	flam. sol.	1338
PHOSPHORUS	WHITE dry	flam. sol.	1381
PHOSPHORUS	WHITE wet	flam. sol.	1381
PHOSPHORUS	WHITE, dry	flam. sol.	1381
PHOSPHORUS	WHITE, in water	flam. sol.	1381
PHOSPHORUS	YELLOW, dry	flam. sol.	1381
PHOSPHORUS	YELLOW, in water	flam. sol.	1381
PHOSPHORUS OXYCHLORIDE		corrosive	1810
PHOSPHORUS PENTACHLORIDE		corrosive	1806
PHOSPHORUS PENTASULFIDE		flam. sol.	1340
PHOSPHORUS TRICHLORIDE	*	corrosive	1809
POISON	corrosive liquid	poison B	2927
POISON	corrosive solids	poison B	2928
POISON	flammable liquid	poison B	2929
POISON	flammable solids	poison B	2930
POLISH	FURNITURE	flam. liq.	1142
POLISH	METAL	flam. liq.	1142
POLISH	STOVE	flam. liq.	1142
POTASSIUM HYDROXIDE		corrosive	1814
POTASSIUM METAL		flam. sol.	2257
POWDER	SMOKELESS small arms	flam. sol.	1325
PRIMER COAT	metal	flam. liq.	1263
PROPANE	GAS	flam. gas.	1978
PROPANOL			1274
RADIOACTIVE DEVICE	n.o.s.	radioact.	2911
RADIOACTIVE MATERIAL	fissile, n.o.s.	radioact.	2918
RADIOACTIVE MATERIAL	L.S.A.	radioact.	2912
RADIOACTIVE MATERIAL	L.S.A. solid	radioact.	2914
RADIOACTIVE MATERIAL	limited qty. n.o.s.	radioact.	2910
RADIOACTIVE MATERIAL	n.o.s.	radioact.	9181
ROCKET	SOLID PROPELLANT	expl. B	----
RODENTICIDE	n.o.s.	poison B	1681
SALTPETER			1486
SHELLAC		flam. liq.	1263
SMOKELESS POWDER	small arms	expl. B	1325

SODA LIME	=	corrosive	190?
SODIUM		flam. sol.	1429
SODIUM BROMATE		oxidizer	1494
SODIUM CYANIDE		poison B	1689
SODIUM HYDROXIDE		corrosive	1823
SODIUM METAL		flam. sol.	1429
SODIUM PERCHLORATE		oxidizer	1502
STYRENE		flam. liq.	2055
SULFURIC ACID		corrosive	1830
SULFURIC ACID	fuming (20%)	corrosive	1831
SULFURIC ACID	tech.	corrosive	1830
TAR	liquid	flam. liq.	1999
TEAR GAS		irritant	1693
TOLUENE	technical	corrosive	1294
TRICHLOROETHANE		ORM-A	1710
TRIFLOUROBROMOMETHANE			1009
TURPENTINE		flam. liq.	1299
WATER REACTIVE SOLIDS	n.o.s.	flam. sol.	2813
WAX	liquid	comb. liq.	1993
XYLENE	(XYLOL)	flam. liq.	1307
XYLOL	(XYLENE)	flam. liq.	1307

OCCUPATIONAL CANCER AND THE FIRE FIGHTER

DEPARTMENT OF RESEARCH
HEALTH AND SAFETY DIVISION

INTERNATIONAL ASSOCIATION OF FIRE FIGHTERS,
AFL-CIO-CLC

THE RELATIONSHIP BETWEEN OCCUPATION AND CANCER

In 1775, Percivall Pott, an English surgeon, observed the first documented case of occupational cancer. He attributed the development of cancer of the scrotum in chimney sweeps to their exposure and contact with soot. While Pott discovered the relationship between occupational exposure and cancer more than 200 years ago, coke-oven workers in the steel industry still die of lung cancer at 10 times the rate of other steelworkers because of their exposure to the same kinds of substances that Pott had shown caused cancer in chimney sweeps.

Scientists at the International Agency for Research on Cancer have estimated, based on studies from around the world, that up to 6% of all cancers can be directly related to exposure at the workplace. The National Institutes of Health scientists have concluded that at least 20% of all cancers will be related to workplace exposure. The World Health Organization has estimated that between 75% and 85% of all cancers are caused by environmental exposures.

In the United States, there are about 45,000 chemicals currently in production. Obviously, some of these chemicals are capable of inducing chronic health effects in humans. The occurrence of chronic diseases has an extraordinary large impact on health in the United States and Canada. NIOSH has estimated that the United States has at least 100,000 deaths a year that are directly related to occupational exposures. In addition, probably more than 400,000 new cases of occupationally related diseases are occurring annually.

The cost of chronic diseases, such as cancer, is also staggering. The General Accounting Office has estimated the cost of cancer at $15 billion per year. This estimate was based on the cost of treatment and the loss of earning power and productivity. If social costs (i.e., the costs of pyschosocial deteriorations brought on by a disease but which are not reflected in economic cost analysis) are included then the price tag for cancer may rise to as much as $150 billion annually. The ever increasing cost for medical care only means that these figures are bound to rise even more in the coming years.

There is a wide range of opinion among scientists regarding how much exposure a person can have to a carcinogen to cause cancer. Some believe that a single asbestos fiber could cause a cancerous growth to begin in the lungs. Others believe that exposure to vinyl chloride will not cause cancer until it reacts and uses up all of a non-cellular substance that is produced and secreted in the body. Thus, a worker may be able to be exposed to a certain threshold level of vinyl chloride without using up all of this noncellular substance.

Although both opinions may be valid, there are no known scientific methods for determining threshold levels for carcinogens, even

If such thresholds do exist. The American Conference of Governmental Industrial Hygienists has developed threshold limit values for over 400 substances. These values are based on information gathered from industrial experience as well as human and animal studies and represents what is believed to be a level that all workers can be exposed to day after day without adverse health effects.

SELECTED KNOWN OR SUSPECTED INDUSTRIAL CARCINOGENIC AGENTS

Substance	*Target Organ*	*Route of Entry*
Acrylonitrile	lung, colon	inhalation, skin
4-Aminobiphenyl	bladder	inhalation, oral
Arsenic Compounds	skin, lung	oral, inhalation
Asbestos	lung and chest cavity gastrointestinal tract	inhalation, oral
Auramine	bladder	oral, inhalation, skin
Benzene	bone marrow	inhalation, skin
Benzidine	bladder	inhalation, oral, skin
Beryllium Compounds	lung	inhalation
Bis(chloromethyl)ether	lung	inhalation
Cadmium Compounds	prostate, lung	inhalation, oral
Carbon Tetrachloride	liver	inhalation, skin
Chromium Compounds	lung	inhalation
Coke Oven Emissions	lung, urinary tract	inhalation
3,3'Dichlorobenzidine	liver, bladder	skin
Dimethyl Sulfate	respiratory	inhalation, skin
Hematite	lung	inhalation
Isopropyl Alcohol	paranasal sinuses	inhalation
4,4'-Methlylene Bis(2-Chloroaniline)	bladder	skin, inhalation
2-Naphthylamine	bladder	inhalation, oral
Nickel	nasal cavity, lung	inhalation
Polychlorinated Biphenyls	skin	skin
Soots, Tars & Mineral Oils	lung, skin, bladder	inhalation, skin
Thorium Dioxide	liver	inhalation
Vinyl Chloride	liver, brain, lung	inhalation, skin

To complicate the picture, we are constantly exposed to carcinogenic agents whether in the workplace, in the air we breathe, the food we eat and the water we drink. Since these carcinogenic agents which are present in the environment and those in the workplace may attack (even though they are different chemicals) the same cells, it is possible that many persons may have received doses much greater than the threshold presumed for any single carcinogen. This is especially true for heavy cigarette smokers. As a result, even a small exposure at the workplace could result in an increased risk of cancer.

The National Institute for Occupational Safety and Health has maintained that "exposure to any known or suspected carcinogen must be reduced to the lowest level possible by whatever means available." There is one agreement among scientists: *cancer cannot be caused if exposure to carcinogenic agents does not occur.* Although exposure to carcinogenic agents cannot be totally eliminated, the situation is far from hopeless. Not everyone will get cancer. Among those who do, many forms of cancer are curable. In addition, the risk of exposure leading to the development of cancer can be greatly reduced if proper precautions are taken such as not smoking. For fire fighters, this also means utilizing self-contained breathing apparatus (SCBA) and personal protective clothing during any emergency situation where exposure to a carcinogen may be possible.

FIRE FIGHTING AND CANCER

Epidemiological studies of fire fighters which indicate a direct correlation between exposure to a carcinogenic agent and the onset of cancer are rare. Unlike many other occupations, fire fighters are constantly entering uncontrolled environments. In many instances, fire fighters are not aware of the potential toxic and carcinogenic substances that they may be exposed to.

For example, in April 1980, a fire broke out at a chemical dump site in Elizabeth, NJ. The state, which had taken over the site two years earlier, had inventoried and removed 10,000 of the approximately 50,000 drums of chemical waste present at the site. While the fire was in progress, no one was aware of what those remaining 40,000 drums contained. Fire fighters fought the fire for almost two days; mostly without respiratory equipment because none was available. The IAFF immediately requested NIOSH to conduct a Health Hazard Evaluation and they responded by being at the scene the next day. NIOSH found that a high percentage of fire fighters were experiencing some symptoms at the time of their medical screening 7 to 10 days after the fire. Nose and throat irritation were the most common symptoms along with acute respiratory problems such as coughing, wheezing and shortness of breath. There was also a prevalence of skin dematitis due to chemical contacts. However, the full health effects of this fire on the exposed fire fighters will remain unknown.

Without a controlled environment, it is extremely difficult to perform an epidemiological study to determine the effect of exposure to carcinogenic agents by fire fighters. In addition, there are two other considerations that must be addressed: the "healthy worker effect" and the "dead worker effect."

The "healthy worker effect" is simply that the healthiest worker are those that are employed. The physical demands of fire fighting means that only those that can frequently meet stringent employment standards in the first place are hired. Thus, the initial population is not indicative of the population at large and can significantly alter your findings in studies which seek to determine the incidence of cancer among a specific population.

Previous studies, such as Abrams' dissertation on *Occupational Mortality Among Professional Firefighters* (1974), have shown that fire fighters live approximately 10 years less than the population in general. Since cancers can take up to 40 years to develop, in many cases the fire fighter may have died from other causes such as line-of-duty or heart disease before being diagnosed as having cancer. This "dead worker effect" could result in a finding that the incidence of cancer does not increase for fire fighters even though the exact opposite may be true.

The proliferation of synthetic substances into the marketplace has added a new dimension to fire fighting. Fire fighters are increasingly exposed to known and suspected carcinogenic agents whether at a residential, hardware store, drug store, dry cleaning establishment, pesticide warehouse or chemical manufacturing plant fire. The more than 30,000 hazardous waste sites and the transportation of such hazardous substances poses still more new and significant potential health risks for fire fighters.

Fire fighters, unlike other workers, are often exposed simultaneously to multiple known or suspected carcinogens. This presents another difficulty because there is little experimental data on the synergistic effects of carcinogens. The fact that smoking greatly increases the risk of lung cancer does indicate that multiple exposures to carcinogens may indeed have such synergistic effects.

Although the length and level of exposure for fire fighters may differ from the epidemiologic studies that have been performed for workers in controlled settings, it is still apparent that fire fighters are exposed to the same type of substances that have been known to cause cancer in asbestos, textile, steel, rubber industry and other workers.

Practically every emergency situation encountered by a fire fighter has the potential for exposure to carcinogenic agents. However, fire fighters can also be exposed to carcinogenic agents when the protective clothing they wear is exposed to high heat or burns. Fire fighters can even be exposed to carcinogens through the fire extinguishing agents they utilize.

Asbestos is still commonly used as a flame resistant fabric, especially in proximity fire fighting clothing and fire blankets. Manufacturer advertisement of the availability of clothing and blankets made out of asbestos fabric is another indication of its acceptance within the fire service. Asbestos fibers can separate due to flexing and abrasion and be inhaled as a carcinogenic agent by the fire fighter.

MOCA (4,4-methylene bis(2-chloroaniline)) is primarily used in the production of solid elastomeric parts. Thus, insulation in fire fighter boots and helmets and personal flotation devices can contain MOCA. MOCA can also be found at fires involving polyurethane foams found in furniture cushions, mattresses, automobile seats and safety padded dashboards, home appliance components, jet engine turbine blades and radar systems. MOCA has been shown to be associated with liver and lung cancer in rats.

Another carcinogenic agent, carbon tetrachloride, was once used in fire extinguishers and was recommended and widely used for electrical fires. There have been several reports showing liver cancer in humans as being associated with exposure to carbon tetrachloride.

Carbon tetrachloride is still utilized as a metal degreaser, refrigerant and grain fumigant.

The list of potential carcinogenic agents that fire fighters can be exposed to is almost as long as the list of all known or suspected carcinogens. Among the more common substances to which fire fighters are potentially exposed include asbestos, creosote, polychlorinated biphenyls, plastics and pesticides. Another new danger is the cancer hazard caused by radiation exposure.

Asbestos

Asbestos is a mineral that appears in a fibrous and fluffy form when separated from rock into fibers of differing length. These fibers are resistant to heat, acid, corrosion and possess the ability to absorb and filte

Asbestos has been widely used in many industries as insulation and fireproofing. Currently, there are more than 3,000 products, mostly in the construction industry, that are made using asbestos. There are four major types of asbestos that are commonly encountered by fire fighters: amosite, anthophyllite, chrysotile and crocidolite.

TYPES AND USES OF ASBESTOS

Asbestos Fiber	*Fiber Color*	*Common Uses*
amosite	brown, grey, green or yellow	cement pipe, cement sheet, roofing products, thermal insulation.
anthophyllite	brown, grey, green or yellow	cement pipe, packing and gaskets, plastics, paper
chrysotile	white, grey, green or yellowish	cement pipe, cement sheet, flooring products, roofing products, packing and gaskets, thermal insulation, electrical insulation, paper friction products, coatings and compounds, plastics, textiles
crocidolite	blue	cement pipe, packing and gaskets, plastics, paper

Fire can destroy the surrounding material in which asbestos fibers are embedded, thus releasing them into the environment. The fibers themselves do not burn. During overhaul operations, fire fighters tear apart burned structures searching for fire extension. This proces

releases asbestos fibers from the torn insulation and construction materials into the air. Fans used to vent the fire scene during overhaul may cause these fibers to spread even more.

Asbestos is dangerous when the fibers are released into the air and inhaled or swallowed. Asbestos fibers are microscopically small; each fiber is hundreds of times smaller than a human hair. The fibers are so fine that they can float in the air indefinitely without settling. These small fibers can easily enter the lungs where they can remain for life. There are two types of diseases that can result from breathing or swallowing asbestos fibers: asbestosis and several forms of cancer.

Asbestos exposure is known to increase occurrence of lung and wind pipe cancer, cancer of the large intestine and is chiefly linked to occurrences of mesothelioma (a rare cancer of the chest and abdominal lining). It is also known that smoking considerably increases the risk of persons who work around asbestos. Despite the high occurrences, not everyone exposed to asbestos will get cancer.

Asbestos exposure will not produce any immediate adverse health effects. Such adverse health symptoms may not occur for 20, 30 or 40 years after exposure. Symptoms of asbestos-related diseases, once they become apparent, include shortness of breath, coughing, blood in the fluid coughed up from your lungs, pain in the chest or abdomen, difficulty in swallowing and rapid large weight loss.

Although the longer the exposure to asbestos the more apt one is to get cancer, studies have shown that a two- or three-month exposure can cause the onset of mesothelioma. Fire fighters who have worked for long periods without utilizing SCBA, such as during overhaul, could have had a similar exposure experience as those short-term asbestos workers.

Creosote

Creosote (creosotum, creosote oil, black oil) is a complex mixture of organic chemicals that appears in the form of a thick, tarry liquid or semi-solid substance. The main uses of creosote are as a wood preservative and in pitch for roofing. Creosote itself is a carcinogenic agent found in soot, tars and mineral oils. The 1775 study by Pott of chimney sweeps in England who developed scrotal cancer was due to a creosote-like substance.

Creosote has been used as an antiseptic, disinfectant, germicide, constituent of fuel oil and a therapeutic agent. Fire fighters may encounter creosote on the waterfront, from burning wharves, dock pilings and other wood on or near the water. Other common sources include utility poles, and/or other woods treated with creosote to prevent decay, mildew or other type of corrosion. Creosote in wood can be recognized through its tarry smell and dark or even black color.

Creosote, which burns at 122° F (50° C), also gives off a carcinogen called benzo(a)pyrene and many other related compounds known as polycyclic aromatic hydrocarbons (PAH). Exposure from creosote can occur by inhaling fumes as it burns and/or by skin absorption. Creosote is associated with cancer of the skin, forearms, prostate, testicles and penis. Exposure to benzo(a)pyrene has been associated with cancers of the mouth, throat, windpipe and lung.

After exposure to creosote, the skin may become reddish, burn, itch, turn a grayish or bronze color in areas, blister, ulcerate or even turn gangrenous. The fire fighter's eyes may be injured, producing an inflammation of the mucous membrane lining the inner surface of the eye or permanently scarring the cornea. Other acute health effects include salivation, vomiting, dizziness, headache, hypothermia, a bluish discoloration of the skin due to the lack of sufficient oxygen in the blood, convulsions, weak pulse, breathing difficulties and a skin rash.

There are no special tests to detect exposure to creosote. However, if there is a rash or other abnormalities on the skin, then a physician should be consulted to determine whether a biopsy is required.

Polychlorinated biphenyls

Polychlorinated biphenyls (PCBs) are a group of heavy, oily, liquid organic chemicals. PCBs are synthetic chemicals produced during a chemical reaction using chlorine and certain petroleum derivatives. There are more than 200 members of this chemical group known as PCBs, but the most common forms are chlorodiphenyl made up of either 42% chlorine or 54% chlorine. PCBs range in appearance from a straw-colored, oily liquid to a white or yellowish waxy solid depending on the amount chlorinated. PCBs from a capacitor or transformer that has exploded may be black in color. PCBs are flame resistant, but they do begin to give off vapors at 122° F (50° C). At high temperatures such as encountered in a fire, liquid PCBs give off toxic vapors.

PCBs, which are chemically inert, nonflammable, resistant to heat and pressure, and electrically nonconducting, are extremely attractive for industrial uses. PCBs are found wherever there are transformers or capacitors. These can range from electrical transformers in buildings and at utility company facilities to capacitors in television sets, fluorescent lights and home air conditioners. Any transformer or capacitor containing an oily liquid or a white or yellowish solid is likely to contain PCBs. PCBs are also used as an additive for extreme pressure lubricants (e.g., hydraulic systems, vacuum pumps and gas transmission turbines), as a coating for investment casting molds in foundries and in carbonless copying papers.

Although PCB production was restricted in 1971 and banned by the Environmental Protection Agency in 1977, equipment using PCBs is still in widespread use. Fire fighters should assume that any capacitor and any fluid-filled transformer contains PCBs or PCB-contaminated fluid. At high temperatures, PCBs also form other extremely hazardous substances such as dioxins (used in Agent Orange) and polychlorinated dibenzofurans.

PCBs have been marketed commercially since 1929 under trade names such as Abestol, Acroclor, Chlorextol, Clorhen, Kanechlor, Inerteen, No-Flamol, Phenoclor, Pyranol and the familiar Askarel. Fire fighters encountering PCBs in a transformer will usually see it labeled as Askarel or Acroclor

PCBs enter the body through inhalation of air contaminated with vapors, mists or particulates containing PCBs. They can also enter through the skin or eye contact with materials containing PCBs and/or by swallowing food or other materials contaminated with PCBs. For example, PCBs can readily penetrate the neoprene vapor barrier commonly used in fire fighter protective clothing. Once absorbed into the body, they tend to settle in the liver and fat cells.

PCBs are suspected to be associated with liver and pancreas cancer in humans. PCB exposure has also been associated with decreased sperm count, impotence and other reproductive problems; damage to the nervous system causing tremors; and liver damage.

Exposure to PCBs may produce irritation to the eyes, nose and throat as well as water retention and swelling, jaundice (if liver damage has occurred), vomiting, weight loss, loss of appetite, abdominal pains and fatigue. Exposure to PCB fumes may cause the onset of chloracne, a severe and painful skin rash.

Plastics

Plastics are long chains of organic molecules made through a linkage process known as polymerization. During the last two decades, the rapid proliferation of plastic products entering the marketplace has added a new dimension to fire fighting. The presence of plastics can probably be expected at every fire emergency, because of the variety of products that are made such as furniture, electric wire insulation, office equipment and kitchen gadgets. It is estimated that there are over 30 billion pounds of plastic made annually in the United States alone. Of these plastics, about 13 billion pounds is polyethylene and 6 billion pounds is polyvinylchloride.

For a fire fighter, the problems with plastics begin when they are heated. As plastic heats up, it begins to break down into different chemical elements. These elements which are given off as fumes may be odorless and colorless and are sometimes toxic and carcinogenic. Toxic fumes begin to be given off long before the plastic actually

catches fire. These fumes, as dramatized in the MGM Grand Hotel fire, when inhaled can cause death far from the actual fire site.

Polyvinylchloride (PVC) is a mixture of vinyl chloride and a variety of other additives. The particular mixture will depend upon the manufacturer and the intended purpose. Likewise, the fumes that are given off will depend upon the type of PVC and the temperature. As PVC heats up, vinyl chloride may be released. When PVC burns, benzene, hydrogen chloride, phosgene, carbon monoxide and carbon dioxide are also given off.

PRODUCTS OF SEVERAL COMMON PLASTICS

Plastic	*Typical Products*
ABS	piping, luggage automobile dashboards, calculator housings, refrigeration liners, margarine tubs
Phenolics	circuit breakers, distributor caps, automobile steering wheels, fuse blocks, pot handles
Polycarbonate	helmets (football/fire fighter/baseball), power tool housings, battery cases, safety glasses, molded products
Polyethylene	milk bottles, seats, waste baskets, disposal syringes, pallets, shipping pails, trash bags, packaging lids, communication cables, bowls, garment bags, wire/cable coatings
Polypropylene	auto fender skirts, battery cases, carpet packing, dishwasher tubs, door liners, radio/tv/phonograph housings
Polystyrene	foam and nonfoam cups, interior doors, margarine tubs, appliances, shutters
Polysulfonate	coffee makers, camera bodies, electrical connectors, battery cases
Polyurethane	cushioning for furniture, mattresses and bed pillows, carpet pads, building insulation, refrigerator and freezer insulation, structural portions of chairs, tables, cabinets, picture frames, decorative beams and wall panels, swimming pools, sporting goods
Polyvinylchloride	phonographic records, bottles, piping, siding, wall covering, flooring, upholstery, chemical wire coating

The raw material of PVC, vinyl chloride is one of the top fifty chemicals produced in the United States. Vinyl chloride and benzene are known or suspected carcinogenic agents. Vinyl chloride has been associated with cancers of the liver, brain, lung, blood and nervous system. Benzene has been associated with increasing the risk of leukemia. In addition to being a combustion by-product of PVC, polystyrene, polyurethane and other plastics, benzene is used as a constituent in motor fuels, as a solvent for fats, inks, oils, paint, plastics and rubber, in photogravure printing and as a chemical intermediate.

The degradation of the plastic polyurethane produces hydrogen cyanide gas and urethane, a probable human carcinogen. Polyurethane also produces acrylonitrile, which has been associated with increased incidences of respiratory and colon cancers. Acrylonitrile is also used in the manufacturing of synthetic fibers, acrylonitrile-butadiene-styrene (ABS) plastics, nitrile rubbers, chemicals and adhesives. In addition, acrylonitrile has been used as a pesticide.

ACUTE SYMPTOMS OF PLASTIC EXPOSURE

Carcinogen	*Immediate Symptoms*
acrylonitrile	Irritation of the eyes. Repeated and lengthy exposure may produce skin irritation. Blistering may occur after prolonged contact with the skin. May also produce nausea, vomiting, headaches, sneezing, and light-headedness and weakness.
benzene	Irritation to the skin, eyes and upper respiratory tract. May result in blistering in or beneath the skin, redness of the skin, and a dry, scaly, cracked rash. Exposure to extremely high concentrations results in central nervous system depression, headache, dizziness, nausea, convulsions and coma may occur. Continuing exposure causes changes in blood and in bone marrow.
urethane	Irritation of the eyes, respiratory tract and skin; may be severe enough to produce bronchitis and fluid in the lungs. May create an asthmatic reaction. Exposure over a long period of time may produce a decrease in breathing capacity.
vinyl chloride	Symptoms resemble mild alcohol intoxication. Lightheadness, some nausea, vomiting and dulling of seeing and hearing responses may develop with a very high level of exposure. Liver damage may occur, also eye damage.

Probably the most dangerous period of exposure to the by-products of plastic combustion for the fire fighter is during overhaul. This occurs because fire fighters frequently remove their respiratory protection leaving them exposed to the fumes that may linger for hours, especially in confined spaces.

Pesticides

Pesticides are found everywhere, in grocery stores, residential homes, drug stores, hardware stores, garden and flower shops, as well as agricultural sites. Pesticides known or suspected of being carcinogenic include chlordane, heptachlor, dieldrin, DDT, kepone, lindane, mirex, toxaphene and so forth.

Chlordane was used as an insecticide on preplanting soil, fire ants and harvester ants prior to being banned in the mid-1970's.

Heptachlor is used as an insecticide in seed treatment, preplanting soil application, dipping tops of plants and roots for control of insects, flies and mosquitoes. It is also used on household plants and on agricultural crops and fruits.

Kepone was first introduced in 1958 and has been used as an insecticide against leaf-eating insects, ants, cockroaches and as a larvicide against flies. In the late 1970's the production and use of kepone was stopped in the United States. Research studies have shown an increased incidence of hepatocellular cancers in rats and mice.

Lindane is the accepted common name for a group of gamma isomers of hexachlorocyclohexane. Lindane is primarily used for insecticidal treatment of hardwood logs and lumber, seed grains and livestock. Secondary uses of lindane include its application as an insecticide on several dozen fruits and vegetable crops. Exposure to lindane in humans have shown increased incidence of leukemia and lung tumors.

Mirex has been used extensively to control the fire ant, especially in the southeastern region of the United States. Mirex has also been used to treat other species of ants and yellow jackets. The use of mirex as a pesticide was discontinued in the late 1970's. In animal studies, mirex has caused an excess of liver tumors.

Toxaphene is one of the most popularly used pesticides. The primary use of toxaphene is to control cotton insect pests. It is also used to control insect pests on livestock, poultry and a few field crops (soybeans, peanuts). In the United States, the southeast and delta states are responsible for most of the toxaphene used. In animal studies, toxaphene has been shown to produce liver cancers in mice.

Pesticides can affect the body if inhaled, if they come in contact with the eyes or skin or if they are swallowed. Like PCBs, pesticides

may penetrate neoprene vapor barriers commonly utilized in fire fighter protective clothing. Mild poisoning after exposure can cause symptoms such as dizziness, nausea, abdominal pain and vomiting. Moderate poisoning can show the same symptoms as mild poisoning followed by severe irritability, convulsive seizures and coma. In severe cases, the convulsions may be continuous with rapid heart beat, labored breathing, unconsciousness and eventually death.

Radiation

Although we have always been exposed to minute amounts of radiation during our daily lives, exposure to high levels of radiation is a relatively new danger brought about by the use of atomic energy for peaceful uses.

While radiation is a form of energy rather than a chemical or metal, particles of radioactive substances can be found in dust or smoke. Radiation is emitted, transmitted or absorbed in a wave or energetic particle form. The most hazardous form of radiation is ionizing radiation which severely damages the body's cells and tissues.

Ionizing radiation is produced naturally through the decay of radioactive elements or artificially through X-ray machines and other devices. Fire fighters can encounter ionizing radiation when responding to emergencies at factories that produce drugs, fire alarms, X-ray tubes, electronic tubes or in medical offices, hospitals, television repair shops, petroleum refineries and scientific research laboratories.

Radiation is unique because of its ability to directly enter the body through the skin much like sunlight going through a window. This direct route of exposure is an external hazard. Internal hazards are caused by radioactive materials entering our bodies through inhalation, ingestion and skin absorption. Generally, radioactive materials enter the body under occupational conditions primarily through inhalation. However, a skin puncture or laceration could result in radioactive particles being implanted under the skin. In addition, contamination of a fire fighter's turnout, helmet, boot or gloves with radioactive materials can result in accidental ingestion of radioactive particles or dust. Experience has shown that workers exposed to radiation have high rates of occupational illnesses such as cancer, leukemia, sterility, cataracts and life span shortening. In addition, such exposure could also have teratogenic and mutagenic effects. Thus, infants born of mothers after exposure to the atomic bomb had an increased incidence of malformation and abnormality of the central nervous system.

The most common forms of ionizing radiation encountered are alpha, beta, gamma and X-rays. Alpha radiation cannot penetrate the skin, thus is not an external hazard. However, alpha-emitting materials can be inhaled into the body with serious consequences. Beta

radiation can travel into the tissues of the body, however, it usually cannot penetrate through a fire fighter's protective clothing. Exposure through inhalation is again the most severe hazard. Both gamma rays and X-rays are primarily external hazards, that is, they readily penetrate the skin surface. Gamma emitters can also pose serious hazards through inhalation or ingestion.

Radiation exposure represents one of the most severe cancer causing hazards. Like chemical carcinogens, radiation exposure is dose dependent with some risks even at the lowest measurable exposure level. Again, in a manner similar to exposure to chemical carcinogens, exposure to radiation may not produce any immediate adverse health effects. Radiation exposure can also be followed by a latency period that can last several decades.

RADIATION LATENCY PERIOD

Cancer Effect	*Time Elapsed From Initial Radiation*
Leukemia	5-30 years
Bone Cancer	5-30 years
Lung Cancer	10-50 years
Other Cancers	Variable years

GUIDELINES FOR DETERMINING OCCUPATIONALLY-RELATED CANCER

With the effort for presumptive cancer legislation just beginning, case by case determinations must be made for successful workers' compensation, disability or pension cases. In contrast to a traumatic injury that is readily apparent to everyone, the relationship between the profession of fire fighting and cancer may not always be clear cut. Cancers are usually slow to develop and may not appear until after an individual retires. In addition, it is frequently difficult to document exposures of an individual to carcinogenic agents.

Cancers develop very slowly in humans, usually manifesting themselves from 5 to 40 years after exposure to the cancer-causing agent. For example, cancers of the liver, lung or bladder may not appear until 30 years after exposure to asbestos, vinyl chloride or benzidine. This long period of latency is one of the major reasons why it is so difficult to determine the exact causes of cancers. It is also difficult to document occupational exposure to carcinogens because of the inability to pinpoint the specific agent or agents and the level of exposure that a worker may be exposed to.

Decisions in workers' compensation and other similar cases are generally based on an evaluation of the available information. When evidence is presented in an organized and logical fashion, when major issues are clearly identified and the causal factors are indicated, then the greater the likelihood of a favorable and equitable decision.

In such documented cases, the relationship between cancer and fire fighting has been constantly acknowledged. The most prominent example is the State of California's enactment of presumptive cancer legislation (Appendix II). This law presumes that cancer is occupationally related when it can shown that exposure to a carcinogen during employment took place which can be reasonably linked to the cancer.

Workers' compensation boards have already identified cancer in fire fighters which is employment-related. An occupational medical specialist, John B. Webster, M.D found that an Ohio fire fighter's leukemia was caused by his exposure to carcinogens. In San Francisco, the Retirement Board ruled a fire fighter's death from intestinal cancer was occupationally caused. Other studies have shown excessive incidences of buccal, pharyngeal, intestinal, rectal and colon cancer in fire fighters. A study in Toronto over the course of 25 years found that cancer increased steadily as a cause of death, from 15.4% in 1945-9 to 38.4% in 1967-70, among active fire fighters. A NIOSH study of mortality in Washington State over a twenty-year period found that fire fighters had a higher incidence of lymphatic leukemia and cancers of the lympatic and hematopoietic

tissues. Thus, the epidemiological work performed so far does suggest that cancer is an occupational disease afflicting fire fighters.

For jobs such as fire fighting, any stress may be an aggravating factor. Since most states hold that the employer accepts the worker "as is" such factors as age, sex, heredity and obesity can be excluded from the list of causative factors. This basically leaves those mechanical, chemical, physical or biological exposures in the working and nonworking environment to be considered. Although this is an easy and simple summation, aggravation cases frequently have multiple causes, many of which are either unknown or not understood.

FIRE FIGHTER MORTALITY REPORT

Prepared for Center for Fire Research Institute for Applied Technology
National Bureau of Standards

by International Association of
Fire Fighters
Washington, D.C.
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PART I
INTRODUCTION

Throughout the history of man, fire has served as both friend and foe. It is doubtful that without its use, civilization could have progressed to the state that it now enjoys. Under controlled circumstances, the benefits of fire are great; its scope is far reaching, ranging from cooking our food to providing the energy needed to run our cities. While the productive force of fire is unquestionable, such power is equally matched by fire's capacity to destroy. So great is its destructive potential that in recent history fire has claimed the lives of over 450 people in one fell swoop; it has been the sole factor responsible for the loss of billions of dollars worth of property each year; it has devastated millions of acres of valuable forest land. Yes, fire affects us all. It strikes indiscriminately; it transcends all social, economic and geographic boundaries. In the eyes of fire, we are all equal.

Due to the ravaging effects of fire, the fire fighters play one of the most vital roles in our society. They are the people who have consciously chosen to pit their minds and bodies against the unpredictable nature of fire. When all of man's natural instincts signal him to retreat, the fire fighter is the man who must advance and challenge. This situation obviously places him in a most perilous position. With each alarm, the demands of the job engender a constant threat of injury or death. And, unfortunately, this threat is becoming increasingly a reality for many a fire fighter.

Today the fire fighter has only slightly better than an even chance of escaping injury each year,[1] with many fire-related injuries resulting in permanent disabilities. Even more staggering are the statistics regarding the ratio of in-the-line of duty deaths.

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1 In the 1974 IAFF Death and Injury Survey, it showed there were 47.3 fire fighter injuries per 100 workers.

In the last ten years, fire fighters have averaged an annual ratio of 86 deaths per every 100,000 employees. While most occupations have experienced a downward spiral in their death rates over the last decade, the fire fighting profession has unfortunately not showed this trend: as their mortality rate has remained relatively constant[2] during this time period. Numerous facts and figures may be cited to underscore these inordinately high ratios, but let it suffice to say that fire fighting claims the dubious distinction for having decidedly the highest injury and mortality rate of any occupation in the country.[3]

While fire fighting is at the top of the death and injury scale, it is not equally balanced on the scales of health and safety protection. The occupational health and safety of the majority of workers in the U.S. are protected by Federal legislation, as their attendant OSHA problems receive increasing attention and research. But the fire fighter, who assumes one of society's greatest responsibilities, does not enjoy such coverage. Protection of fire fighters health and safety at the Federal, State and local level is almost nil with research into these problems to date being limited.

It is precisely the urgency of this situation that had prompted the International Association of Fire Fighters (IAFF) to undertake the Fire Fighter Mortality Study. For many years, the IAFF has annually compiled statistics on fire fighters' duty-connected deaths. However, this has done little more than provide us with grim mortality figures. Up until

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2 The IAFF Death & Injury Survey--1974 stated that in the previous decade fire fighters suffered an average annual death ratio of 86 deaths per 100,000 workers. With the exception of 1970, for which the ratio was 115 per 100,000, fire fighter deaths for any given year in this 10-year period remained constant to 86 deaths per 100,000, give or take 10 deaths.

3 IAFF Death & Injury Survey--1974 showed that for 1974 fire fighters suffered a higher death ratio than any other occupational grouping--84 deaths per 100,000 workers.

this time, data has not been gathered for the purpose of explaining the causes of the deaths, let alone the circumstances surrounding them.

In undertaking such a study for the first time, we have conducted an in-depth analysis of duty-connected deaths. The study involves scrutiny of the causes and circumstances of 101 fire fighters in-the-line of duty deaths. By extensively investigating each death, specific problem areas have been discerned. While this study lays the foundation for specified research into critical areas, solutions for some problems are immediately evident. It would be a grave mistake to view this study as the panacea for the problem of fire fighters in-the-line of duty deaths. Rather, this report should be perceived as a starting point, a springboard from which further research into the complexities of the problem may be launched. It might be well to view this report as a workbook offering to members of the fire service, legislators and other researchers' direction as to what steps might be taken to reduce dramatically the staggering mortality rate suffered by fire fighters.

STANDARDS AND RESEARCH METHODS

The intended scope of the study was to investigate all in-the-line of duty deaths that occurred in the time period between September 22, 1974, and December 31, 1975. In administering a study of this magnitude, it was necessary to set standards regarding the deaths to be investigated as well as the time during which the investigations would take place. Investigations were conducted when a fire fighter died while on duty or as a result of an on-duty activity or injury.

On-the-scene investigations were conducted into each fire fighter fatality. Since the most valuable sources of information were human testimony--the fire fighters who were at the site of the fatality--a question as to the timing of the investigation was raised. It was

originally felt that the investigator should move into the area as soon after the fatality as possible. However, it was found that the fire fighters who witnessed the fatality were not receptive to the investigator's inquiries when the interviews were conducted prior to the funeral. After a number of preliminary investigations, it was determined that the most ideal time appeared to be the first day the fire fighters were back on duty after the funeral. We found that by conducting the investigations at this time period the trauma of losing a fellow fire fighter had usually subsided, but the men still retained a vivid recollection of the circumstances surrounding the fatality. It should be noted that we attempted to meet this date on each case, but it was not always possible because of difficulties associated with late notification and scheduling. In these situations, the case was investigated at the earliest possible date.

Since we were concerned with obtaining as broad a sample as possible, there were no restrictions placed on the type of department or fire fighter. Full-time IAFF affiliates, non-IAFF departments and volunteer departments were all included in the study. It may also be noted that there were no restrictions based on rank. Fatalities of all fire service personnel were investigated.

One important area that required resolution prior to launching a study of this nature was the development of a system by which notification of fire fighter fatalities was received. Before the study began, all IAFF locals were informed of the study and instructed to contact the Project Administrator in the event of a fatality in their local. The IAFF Vice Presidents, who are located throughout the country, were most helpful in insuring that this system functioned properly. Since the majority of paid fire departments are affiliated with the IAFF, and the majority of

deaths occurred in paid departments, we were notified of most of the deaths by IAFF Vice Presidents and Local Officers. There is no doubt that this study would not have been as successful as it was had we not had the cooperation of these IAFF officials. This information network was supplemented by a news-clipping service which proved useful in informing us of non-IAFF and volunteer fire fighter in-the-line of duty deaths. Finally, other fire-service-related organizations cooperated in notifying us of fatalities.

As was noted earlier the most important data source came from the fire fighters who were at the scene of the fatality and this information was collected by means of personal on-the-scene interviews. In each case, the project administrator attempted to talk to every fire fighter who was on the fire ground. While in many instances a fire fighter may not have engaged in activities that directly related to the fatality, these interviews were nevertheless found to be important in terms of helping to provide a reasonably complete reenactment of the fire fighting operations, thus enhancing our overall understanding of the fatality. The general procedure involved conducting interviews with small groups of fire fighters from a particular company or a station house. While the group interviews were most common, certain circumstances dictated the need for individual interviews as well.

To supplement the oral accounts of the fatality, written reports of the incident were also gathered. These written data sources included such documents as fire alarm reports, company reports, officer reports, fire marshall reports, state compensation forms and newspaper articles. Since there are no uniform reports compiled by departments, the type and extent of the reports vary widely and thus information that could be

extracted from the given reports also varies widely. Finally, in every case where an autopsy was performed, an attempt was made to secure a copy of the autopsy report. Such reports were among the most valuable sources of documented information in those cases where fire fighters died of heart attacks.[4]

Upon completion of each investigation, a case portfolio was compiled, which included all information (notes, reports, articles, etc.) that had been gathered. From these portfolios, information was extracted and compiled into a detailed organized report of the entire incident. The data for these reports were processed into three sections. The first two sections were comprised of forms on which information could be standardized for the purpose of analysis. The first of these was a Personal & Department Information form that included such data as age, height, weight, type of shift worked, average work week, etc. The second was an Alarm & Fire Information form which included such details as date, day, time, type of fire, number of men responding, and other information of this nature. The third section contained the gist of the report: a written scenario of the fire fighter's activities from the time of the alarm up until the time of the fatality.

As was stated earlier, this study was conducted under the direction of the IAFF's Research Department. Additionally, a Joint-Project Team was formed to assist and give direction in analyzing the circumstances of the fatality. This committee consisted of representatives from the IAFF, the International Association of Fire Chiefs, the National Bureau of Standards, and the National Fire Prevention and Control Administration. Throughout

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4 As was the case with other written reports, the information on the autopsies also varied widely. Some autopsy reports were quite detailed and provided a good understanding of the cause of death and contributing factors, as other reports were much abbreviated and their utility was limited.

the term of the study, the Project Team was periodically convened to discuss each individual case. From these discussions, problem areas were identified and preliminary recommendations were offered for the abatement of future similar occurrences.

The presentation of the findings of this study reflects in many ways the manner in which the data were organized for individual cases. This report consists of four sections and, hopefully, by segmenting the report in this manner, the reader will be provided with the proper perspective from which he may view the problem of fire fighter fatalities. The first section presents general information pertaining to all of the fatalities examined. It is also hoped that these basic facts will provide the reader with background information that will aid him in understanding this report as it progresses to greater specificity. The other three sections present analysis of cases organized along lines of the cause of death. These general categories are (1) fire fighters killed while fighting fires (excluding heart attacks); (2) fire fighters killed in non-fire fighting situations (excluding heart attacks); and, (3) fire fighters who died of heart attacks in both fire fighting and non-fire fighting situations. In turn, each of these three sections will be broken down to present individual case summaries for specific causes of deaths under each section, followed by a presentation of the problem areas which will transcend the cause of death classifications.

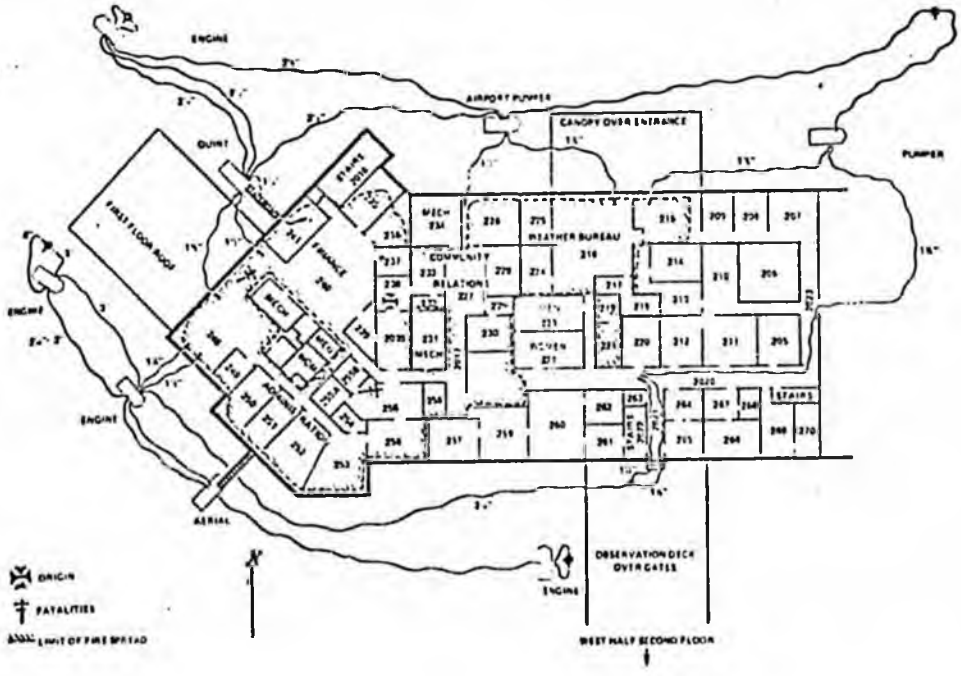
Before proceeding with this report, it should be noted that it is not my intent nor is it the intent of any of the other individuals associated with the IAFF Mortality Study to embarrass any specific fire fighter or fire department. We see no utility in pointing a finger on any one person

or department. Since the purpose of this study is purely one of research aimed at the far reaching goal of rendering fire fighting a less hazardous profession, all fire departments and individual fire fighters will remain anonymous in the course of the ensuing discussion.

CASE 48 & 49

VICTIMS - A 28 year old fire fighter with 2½ years total service.
- A 28 year old fire fighter with 2½ years total service.

SETTING - This fire occurred in mid-June 1975 at approximately 8:00A.M. The fire was on the second floor of an airport terminal. It did not involve the entire terminal, but only the west end which consisted of administrative offices. The structure was metal and glass walled with a metal deck roof



supported by steel trusses and beams. As is noted by the diagram there are no common hallways from east to west or north to south. There was a dropped ceiling on the second floor, with a 2 to 3 foot space between the ceiling and the roof. There were no fire stops in this space. The fire started in Room 238, from unknown causes, and quickly spread throughout much of this section of the building. The dark lines indicate the total fire spread.

SEQUENCE - The airport fire department, whose main responsibility is to protect against airplane fires, had jurisdiction on this structural fire. Fortunately the department was notified of the fire just as the shift was changing so they had double manpower. They responded with a pumper and 8 men. Upon arrival heavy smoke was emitting from the building and a mutual aid call to surrounding volunteer departments was made. The chief of the airport department was also notified of the situation. Prior to the chief and the mutual aid companies arriving a number of activities took place that directly related to the fatalities.

An effort was attempted at making entry to the second floor from the west stairs; however, heat had affected the lock and the door could not be

opened with a key. This effort was abandoned as the north wall was laddered and 1½" lines were directed into the building via the second floor window. Two fire fighters donned 30-minute breathing apparatus and went to open the double doors leading into the office area from the main terminal. These doors had already been opened by a maintenance man when the fire was first discovered; however, the fire fighters were not aware of this. Upon finding the doors open they entered the building.

It is uncertain why the men entered the building but most likely they wanted to survey the extent of the fire. They did not have a hose line with them. Either intentionally or accidentally while in the offices the two men became separated. The men entered the building at approximately 8:10 A.M., and their bodies were not found until after 9:30 A.M. Prior to the discovery of the downed men it was not realized by those on the fire ground that the two fire fighters were missing.

By referring to the diagram, the fronts from which the fire was attacked are pinpointed. Internally the fire was fought by three 1½" lines, and throughout most of the duration of this fire the Chief of the airport department was on one of these lines. The north wall had three 1½" lines in the windows, the southwest wall had two 1½" lines in the windows, and there were three 1½" lines that were directed in the vents on the roof. The aerial tower on the northwest wall had engine trouble when it arrived on the scene and could not be positioned properly to use the tower.

At 9:20 A.M. a nearby paid department responded with their aerial tower. They positioned themselves on the southwest wall, since at this point most of the fire was in that area. The aerial made a quick sweep knocking out the windows and then continued sweeping as it blackened the fire. After approximately 10 minutes of this the fire was extinguished with the exception of a few hot spots which were knocked out by hand lines.

Moments later the first man was discovered in room 255. A large ceiling fixture was lying over him but it is uncertain whether the fixture fell on him knocking him out or if it fell after he was already down. He had his complete protective gear on. The plexiglass on his face mask was broken out and there were jagged edges along the rim of the mask. He was lying on his left side; his proximity coat and pants and his right glove were partially burned. He was dead at the scene, a victim of smoke inhalation, with a 55% carbon monoxide level. His breathing apparatus was an older model and did not have a warning bell.

The second fire fighter was found 10 minutes later in the hall between rooms 248 and 240. The man's mask was off of his face and disconnected from the tank, which was empty. This indicates that he was conscious when his tank ran out and he removed the face piece. His protective clothing showed only slight signs of burns. He was dead on the scene - a victim of smoke inhalation, with a 61% carbon monoxide level. His breathing apparatus was equipped with a warning bell.

Case 85

VICTIM - A 57 year old fire fighter with a total of 26 years service; all served in an engine company.

SETTING - This fire occurred in mid-September 1975 at 9:20 A.M. The scene of the fire was a one-story wood frame house and a wood double garage. The fire started and for the most part was confined to the garage, with only minor burning in the house. Smoke was heavy and the fire was extremely hot.

SEQUENCE - Responding to the fire were four engine companies and a truck company, for a total manpower of 13 men. The fire building was located on the outskirts of town and the companies responded from different stations, thus the arrival on the scene was staggered. There was an approximately five minute gap between the arrival of the first engine and the other companies.

The engine was manned by three fire fighters who were immediately informed by neighbors of the possibility that people were still in the house. While one man hooked to the hydrant the other two men made their way towards the back door of the house with a 2½" line. The men entered the house and quickly blackened what little fire had spread to it and made a survey of the rooms. Finding no one in the house they returned to the yard and started attacking the fire in the garage. The men were exposed to extreme heat which was bottled up in the back yard between the garage and shrubs but fortunately the wind carried away a fair amount of the heavy smoke.

A second alarm was called by a responding Deputy Chief and within ten minutes, from the arrival of the first engine, sufficient manpower was on the scene to relieve the two men. The fire was quickly extinguished with the aid of two additional 2½" lines. After being relieved on the line one of the two men reported to the Deputy Chief that while in the backyard he was suffering chest pains and shortness of breath. The Chief ordered him to the hospital to be checked out.

At the hospital he was admitted for observations. After his fourth day in the hospital he was released. That same evening while at home he developed a high fever and was readmitted. His condition continued to deteriorate and approximately one month after returning to the hospital he expired. The cause of death was listed as pneumonia, brought on by smoke inhalation.

It is important to note that because the two fire fighters had reason to believe that people were in the house they did not take the time to don breathing apparatus. Also, of importance is the fact that twice in the month prior to this fire the victim suffered smoke inhalation while at the scene of a fire. In one case he was treated on the scene and in the other case he was hsopitalized overnight.

CASE 96 & 97

VICTIMS - A 48 year old fire captain with 24 years total service; the last 6 served in the capacity of captain.

- A 29 year old fire fighter with 3 years service, served in an engine company.

SETTING - This fire occurred in late December 1975 at 3:20 A.M. The fire building was a 22-story brick apartment building with eight apartments on each floor. A 120-foot long, 5 foot wide hallway ran the length of the floor with four apartments on each side of it. Three elevators rested in the center of the hallway and there were two stairway exits positioned 40 feet in from the east and west walls. There were fire doors on the stairway entrances that could only be opened from inside the hallway. The fire started on the 14th floor in the southeast apartment, its cause was undetermined.

SEQUENCE - Responding to the fire were two engines, truck, a squad and a Battalion Chief, for a total manpower of 18 men. Upon arrival only a small amount of white smoke was visible emerging from the fourteenth floor. The Battalion Chief and five other men took the elevator to the thirteenth floor, hooked up a 1½" line and climbed the east stairs to the fourteenth floor. As the men attempted to open the door with the aid of an axe and a pocket knife, the Battalion Chief tried to contact the truck company via his radio. Because of his position in the stairway he could not make contact and he and another man returned to the thirteenth floor.

When the men got the door open, the Captain and a fire fighter moved towards the apartments on the east side of the building. Another fire fighter entered the floor and moved to the west. The remaining two fire fighters stayed on the stairs helping occupants who were evacuating from the upper floors. All three men on the floor were equipped with 15-minute air tanks, and at this point they were most concerned with evacuating the floor of occupants.

The Captain and fire fighter moved back down the hallway moving west searching for residents. The fire apartment was #1418. At the other end of the building was apartment #1412 and its front door had been opened, possibly by the two fire fighters, in searching for occupants. The sliding balcony doors of the apartment were also opened and when the fire burned through either the door or ceiling of apartment #1418 a flash occurred, rolling down the length of the hallway and into apartment #1412.

It is deduced that at the time of the flash the Captain and fire fighter were standing a few steps inside or a few steps outside of apartment #1412. Either way, when the flash occurred the men escaped it by moving into the apartment across the hall. The Captain had no major burns and the fire fighter had only partial burns about the face. As the men retreated from the fire they made their way into the far northwest bedroom of the apartment. It is possible that they moved to this room because part of the front room was burning and this led them farthest from the fire.

It is probable that the men were just about out of air at the time of the flash. The third fire fighter who entered the fourteenth floor with the two men was making his way towards the west exit, because his tank had run out. As he saw the flash rolling down the hall he dove through the door and escaped the major brunt of the fire. The men from the truck company were just at the door and they all scrambled down the stairway to safety.

It was approximately an hour and a half after the flashover that the two men were discovered. The fire had grown to a 5 alarm fire and at the time the bodies were found the fire had been knocked out and the department was in the process of overhauling. Up to the point of discovery there was no indication that any of the personnel on the fire ground realized the two men were missing.

The Captain was found just inside the door of the far northwest bedroom with his empty tank on, but his mask off. The fire fighter had both his empty tank and mask off and was lying up against the far wall in the same room. Both men were dead at the scene and the cause of death was listed as carbon monoxide inhalation.

CASE 53 & 54

VICTIM(S) - A 30 year old fire fighter with five years total service, assigned to the ambulance/rescue squad.

- A 27 year old volunteer fire fighter with seven years total service, the last two served in this particular department, assigned to the ambulance/rescue squad.

SETTING - This fatal accident occurred in late June at approximately 3:00 P.M. The fire department that responded to this accident was part full-time/part volunteer with one man of each responding to the accident in the rescue/ambulance unit. The accident scene was at a rendering plant and occurred in a basement below the scale area. Sludge (80% water, 20% hide, meat and fat) regularly drained into this basement and was then pumped by a float activated sump pump to a skimmer tank on the ground floor, and the fat and materials are skimmed from the water which is then pumped into the sewer.

SEQUENCE - Throughout the day the sludge built up, and in the afternoon an employee entered the basement and unplugged the sump pump so that the material could be pumped to the skimmer. When he left the basement the sludge level was approximately one foot. After 15 minutes, when it appeared that the skimmer was going to overflow, the employee again entered the basement and closed off the pump. As he was leaving, he climbed two stairs, collapsed and fell back into the sludge which was at this time around the three foot level. Three employees were sent to the basement to assist the man and when they got to the foot of the stairs they all collapsed also.

Meanwhile the Fire Department had been called reporting that a man had fallen down the stairs and they responded with their rescue/ambulance unit. Prior to their arrival the plant supervisor descended to the middle of the basement stairway and observed one of the men at the bottom of the stairs in convulsions. He immediately left, shut off all electricity and reported to the two responding fire fighters that he thought the men had been electrocuted. They checked to be sure the electricity was off and then entered the basement. When the first fire fighter reached the bottom

of the stairs he leaned over to grab the shoulder of one of the men, who was partially submerged in the sludge. As he did this he immediately collapsed and fell over the man. His partner thought that he had slipped so he leaned over to grab him and also collapsed. The plant supervisor immediately put another call in to the Fire Department and they responded with all available manpower.

In rescuing the six downed men (two fire fighters, four industrial workers) all of the fire fighters wore breathing apparatus. The rescue operations were hampered by the fact that the sludge made the men extremely slippery and ro[illegible]s were needed to pull the men out. The two fire fighters were the first taken from the basement. One died en route to the hospital and the other died several days later, never regaining consciousness. The four industrial workers were all pronounced dead on the scene. The toxic fumes that the sludge created and which caused the fatalities, are believed to be methane and carbon monoxide.

It should be noted, before they pulled the last of the six men out a mutual aid call was made and the total manpower on the scene was over 70 men. The vast majority of these men were treated for dizziness, faintness, shortness of breath, chest heaviness. coughing, choking, nausea, vomiting and occular irritation.

PART V

FIRE FIGHTERS KILLED BY HEART ATTACK

Perhaps one of the most alarming statistics in the finding of this study was the high incidence of fatality caused by heart attack. Heart attacks were the highest single cause of death, constituting 44.5 percent of the overall total number of fatalities. In many ways this figure only represents the tip of the iceberg for heart attack deaths. Since standards of manageability had to be introduced in the study we did not have an opportunity to investigate fatal heart attacks occurring to off duty, vacationing, or recently retired fire fighters. Had we done this the above figure would have, most surely, been multiplied. As was clearly shown in the preceding two sections there is a definite need for protection and research concerning the safety aspects of fire fighting. The high fatal heart attack rate shows that equal protection and research is needed for the health aspects of the job.

In presenting an analysis of the heart attack problem this section is divided into a number of specific subsections: First, there are case summaries for each of the 45 cases. In doing this it is hoped that the reader will be provided with a general background on the victim, the setting in which the attack occurred, and the activities of the given fire fighter at the time of the attack. Due to the high number of fatalities, and the fact that in most cases there is not a definite relationship between the attack and a specific sequence of circumstances, the summaries

as compared to the others have been abbreviated. Second, there will be a discussion of the aspects of the individual that contributed to the heart attack. Third, there will be a discussion of the aspects of the job that contributed to the heart attack. And, finally, there will be a presentation of other general problems in the area of fire fighters' heart attacks. For the readers benefit a glossary of heart terms is presented at the end of this report.

Before proceeding, I should like to state that in the analysis of the heart attack cases I had the assistance of Dr. Robert F. Dyer, Director of the Washington, D.C., Police and Fire Clinic, who is also a cardiologist. Dr. Dyer, who reviewed and analyzed those cases for which we had autopsy reports, contributed to the study with helpful professional observations and his cooperation was most appreciated.

HEART ATTACK CASE SUMMARIES

CASE 3

VICTIM -
- 46 years old, 23 years total service, last 12 years in engine company.
- 5'10", 175 lbs., non-smoker, no previous history of heart attacks.

SETTING -
- Early October 1974, 10:00 A.M.
- Box alarm fire.
- 3-Story brick structure, fire fighter's assistance required in evacuating occupants.

ACTIVITIES -
- The fire fighter's main duties were to hook the engine to the hydrant and operate the pumper.
- The hydrant was tight and required a considerable exertion of energy to open it.
- He collapsed after being on the scene for 25 minutes.
- He was not revived at the scene and was pronounced dead at the hospital.

CAUSE OF DEATH -
- Atherosclerosis of the coronary arteries of the heart. (Autopsy)

CASE 4

VICTIM - . 41 years old, 16 years total service, last 6 years served as Battalion Chief.
. 6'1", 186 lbs., smoker, no history of prior heart trouble.

SETTING - . Early October 1974, 1:00 P.M.
. 4 Alarm fire.
. 1-Story brick structure, warehouse used to store paper.

ACTIVITIES - . The Battalion Chief was in the fire building for the first 45 minutes, directing different engine company operations.
. While in the building he did not have a breathing apparatus and was exposed to heavy smoke and heat.
. For an hour after this he was outside of the building directing varying operations.
. Several times during this time period he climbed and descended a 30-foot embankment.
. After being on the scene for close to two hours he collapsed. Although vital signs were present he could not be revived at the scene and was pronounced Dead on Arrival (DOA) at the hospital.

CAUSE OF DEATH - . (1) Severe atherosclerotic coronary artery disease (2) Recent hemorrhage into atherosclerotic plaque of right coronary artery (3) Ischemic heart disease. (Autopsy)

CASE 7

VICTIM - . 46 years old, Lieutenant, 23 years total service.
. 6'3", 215 lbs., no history of prior heart trouble, smoker.

SETTING - . Late November 1974, 7:00 A.M.
. Box alarm with mutual aid.
. 2-Story brick residential structure, which was extremely involved with fire upon arrival.

ACTIVITIES - . Upon arrival the victim was untangling 1½" preconnected line and when the truck pulled away his feet became entangled in the line - he was knocked down and dragged 10 feet.
. He refused to go to the hospital, stating he was all right; however, for most of the fire he stayed by the engine holding his chest.
. Several times the Chief asked if he would like to go to the hospital but he refused.
. For 5 minutes he manned a 1½" line.
. After an hour on the scene as he was helping move a line into the building he collapsed.
. Cardio-pulmonary Resuscitation (CPR) did not revive him on the scene and he died shortly after arriving at the hospital.

CAUSE OF DEATH - . Severe generalized atherosclerosis. (Autopsy)

CASE 8

VICTIM - . 46 years old, Captain, 21 years service.
. 5'11", 189 lbs., smoker, no history of heart trouble, suffered smoke inhalation 10 days before fatal attack.

SETTING - . Late October 1974, 7:30 A.M.
. Box alarm
. 1-Story wood frame building - basement fire.

ACTIVITIES - . Initially the Captain was the officer in charge of the five men who responded.
. He helped lay lines and then directed operations.
. Ten minutes into the fire the Chief ordered him on to a 1½" line at a basement window.
. He stayed on the line for approximately a half an hour during which time he was exposed to considerable smoke.
. He assisted in overhaul operations for approximately an hour.
. He developed an upset stomach and considerable coughing so the Chief ordered him to the hospital for a checkup.
. In the hospital his condition deteriorated and he expired at 2:30 P.M.

CAUSE OF DEATH - . Arteriosclerotic heart disease. (Autopsy)

CASE 9

VICTIM - . 51 years old, 25 years service, the last 2 years served as a Lieutenant.
. 5'11", 197 lbs., non-smoker, no history of prior heart trouble.

SETTING - . Late September 1974, 6:30 A.M.
. Box alarm.
. 2½-Story, 2 family wood frame structure - attic fire.

ACTIVITIES - . He helped set up a 35-foot aluminum ladder.
. He assisted an engine company in their initial attack on the fire - at this time he was exposed to considerable smoke and heat.
. At 7:10 A.M. he went to the second floor and assisted in salvage operations which consisted of laying covers, removing the ceiling, vacuuming water, and carrying debris bags down the stairs and outside.
. Once during this time he complained of chest and arm pains but after resting a few minutes he told the other fire fighters he was all right.
. After suffering pains a second time he collapsed as he exited the building.

. He was not given oxygen or resusitation until the ambulance arrived - 10 minutes after he collapsed.
. He was not revived and died shortly after arriving at the hospital.

CAUSE OF DEATH - . Coronary sclerotic hypertensive heart disease with acute thromobotic occlusion of left coronary artery. (Autopsy)

CASE 11

VICTIM - . 61 years old, Captain, 5 years service with the given fire department, with a total of 40 years service in fire fighting.
. 5'9", 220 lbs., smoker, had a previous heart attack.

SETTING - . Mid-November 1974, 9:20 A.M.
. Box alarm.
. 1-Story wood frame - fully involved with fire.

ACTIVITIES - . After dropping off fire fighters at the scene, the Captain drove a block to the hydrant. When stepping out of the cab he collapsed.
. Oxygen and heart massage could not revive him at the scene and he was DOA at the hospital.

CAUSE OF DEATH - . (1) Extreme right coronary atherosclerosis (2) old posterior sepal and left ventricular myocardial infarct with fibrosis. (Autopsy)

CASE 12

VICTIM - . 50 years old, 21 years total service, the last 18 served on an engine company.
. 5'7", 180 lbs., smoker, no history of prior heart trouble.

SETTING - . Early January 1975, 1:00 P.M.
. Box alarm.
. 3-Story brick apartment building, fire limited to second floor apartment.

ACTIVITIES - . Victim hooked up lines from two engines into his engine and remained with pumper to operate it.
. After 20 minutes, the fire was knocked down and the victim entered the building to assist in overhaul.
. After being in the building for a few minutes and exposed to moderate smoke the victim collapsed.
. Oxygen and heart massage could not revive him at the scene and he was DOA at the hospital.

CAUSE OF DEATH - . Acute coronary thrombosis.

CASE 14

VICTIM - . 44 years old, 12 years total service.
. 6'4", 200 lbs., smoker.

SETTING - . Mid-January 1975, 1:00 P.M.

ACTIVITIES - . There is a relationship between the fatality and a fire occurring 7 months before.
. At that fire the fire fighter was exposed to tremendous amounts of smoke as he directed a 1½" line on a fire in a bean-bag chair (a vinyl bag stuffed with bits of styrofoam).
. After that fire he suffered chest pains and was admitted to the hospital where he remained in intensive care for 3 days.
. In the time between this fire and his death he often complained of respiratory problems and chest pains.
. He was seeing his doctor regularly for these problems.
. On the day of his death he suffered severe pains while on duty; he was taken to the hospital and died shortly thereafter.

CAUSE OF DEATH/ DIAGNOSIS - . (1) Arteriosclerotic heart disease (A. Occlusion left coronary arter; B. very recent myocardial infarction of interventricular septum and posterior wall of left ventricle) (2) pulmonary congestion and edema (3) pulmonary emphysema. (Autopsy)

CASE 15

VICTIM - . 52 years old, 22 years total service, the last 3½ served as Captain.
. 5'10", 160 lbs., non-smoker. Previous heart attacks: 1969 - out of work 7 months, 1973 - out of work 5 months - returned both times with doctor's written permission.

SETTING - . Mid-November 1974, 3:30 A.M.
. Box alarm.
. 2-Story brick structure, suspected arson.

ACTIVITIES - . First 1½ hours of fighting the fire was external. The Captain was stationed on a 1½" line which he directed into the second floor window.
. The Captain then scaled a ladder with another fire fighter to the second floor - heat and smoke were moderate.
. After being on the second floor for 20 minutes the Captain collapsed.
. The resuscitator could not revive him at the scene and at the hospital doctors treated him for 15 minutes after which time he was pronounced dead.

CAUSE OF DEATH - . Ruptured left ventricle due to myocardial infarction. (Autopsy)

CASE 16

VICTIM - . 47 years old, 15 years total service.
. 5'11", 200 lbs., smoker, no history of prior heart trouble.

SETTING - . Late December 1974, 12:30 P.M.
. Box alarm.
. A 2-story brick building that was being converted to a factory.

ACTIVITIES - . The fire was extinguished by a sprinkler system in the building.
. The victim entered the building to open up the second floor windows to help ventilation.
. Shortly after entering the building the fire fighter collapsed.
. CPR and oxygen were administered and the man was rushed to the hospital but was pronounced DOA.

CAUSE
OF DEATH - . Acute myocardial infarction due to arteriosclerotic heart disease. (Autopsy)

CASE 17

VICTIM - . 54 years old, 23 years service, all served on an engine company.
. 6', 175 lbs., non-smoker, no history of prior heart trouble, had passed a physical one week before his attack.

SETTING - . Mid-December 1974, 11:30 P.M.
. Box alarm plus 2 additional companies.
. A 4-story brick apartment building - the fire was confined to a fourth floor apartment.

ACTIVITIES - . The victim ran along the side of the pumper directing it into position on the fire ground.
. Assisted in carrying a 2½" line up an aerial ladder.
. After carrying the line half way up the ladder he returned to the pumper to check the pressure.
. As he was at the pumper he collapsed.
. With the aid of an inhalator and heart massage he was revived and rushed to the hospital. He remained alive for close to 5 hours, but his blood pressure slowly dropped and as doctors were unable to raise it he expired.

CAUSE
OF DEATH - . Heart attack.

CASE 18

VICTIM -
- 58 years old, 28 years service, all served on a truck company.
- 5'5", 155 lbs., non-smoker, autopsy showed signs of previous heart attack.

SETTING -
- Early February 1975, 1:30 P.M.
- Box alarm.
- 2-Story brick auto parking garage.

ACTIVITIES -
- Victim hooked a line to a hydrant and opened it up.
- For 20 minutes he manned a 2½" line from outside the building.
- He attempted to make entry into the building but the smoke was too heavy.
- Helped break down 2½" lines to 1½" lines.
- He and another fire fighter then entered the ground floor with 2½" lines.
- They positioned themselves at the base of an elevator where there were embers from the fire above.
- Lines on the roof forced a considerable amount of smoke down the elevator shaft, which the men were subjected to since they were not wearing breathing apparatus.
- They remained at the shaft for 25 minutes at which time the fire was extinguished.
- The victim felt dizzy but did not leave the area - 5 minutes later he collapsed.
- Oxygen and CPR were administered. Once at the hospital doctors treated the fire fighter for 15 minutes at which time he expired.

CAUSE OF DEATH -
- Acute coronary insufficiency due to coronary atherosclerosis. (Authopsy)

CASE 19

VICTIM -
- 51 years old, Deputy Chief, 20 years total service.
- 5'10", 210 lbs., non-smoker, suffered a severe heart attack in 1959 - lost 3 years work.

SETTING -
- Mid-December 1974, 11:00 P.M.
- Responded from home on a mutual aid call to a 2-story wood frame structure fire.
- 30° temperature.

ACTIVITY -
- For the first hour the victim directed a 1½" line into a second story window.
- During overhaul the victim went to the second floor and started pulling plaster board from the wall. After doing this for a few minutes he collapsed.

- CPR and oxygen were administered. He did not regain consciousness but he was breathing.
- It took approximately 10 minutes to get him to an ambulance because they had to lower him in a basket stretcher from the second floor.
- He was pronounced DOA at the hospital.

CAUSE OF DEATH -
- Coronary occlusion due to arteriosclerotic heart disease. (Autopsy)

CASE 23

VICTIM -
- 57 years old, 28 years total service.
- 5'6", 181 lbs., smoker, no history of prior heart trouble, family history of heart problems.

SETTING -
- Late February 1975, 5:30 P.M.
- 9 alarms.
- 4-Story brick building abandoned.

ACTIVITIES -
- The victim's company responded on the initial alaram and he dropped them off near the fire ground and drove the engine a block up to a hydrant.
- He connected the soft suction and broke the hydrant.
- Most of his time was consumed with checking and watching the pressure pumper.
- He did help stretch a supplemental line to another engine.
- After being on the scene for 45 minutes his shift relief came.
- About this time he suffered chest pains and informed a nearby police officer of such.
- He was placed in an ambulance and taken to the hospital. En route he refused oxygen.
- As he was entering the hospital he took two gasps and went into deep unconsciousness.
- Doctors treated him for over an hour but this was to no avail and he expired.

CAUSE OF DEATH -
- Occlusion of left coronary artery by fresh hemorrhage and rupture of an atherosclerotic plaque. (Autopsy)

CASE 24

VICTIM -
- 44 years old, a total of 10 years service.
- 5'9", 155 lbs., smoker, suffered a heart attack one month prior to fatality.

SETTING - . Non-fire situation.
. Fire fighter was under considerable stress because of a fear that he would not be able to pass an EMT test. His psychological state changed, reflected by nervousness, tension and a fear that if he did not pass the test he would lose his job (an unfounded fear).
. He suffered his first heart attack while attending a one-week class which was designed to help him pass the state EMT exam.
. While recuperating from this attack he suffered a second attack and expired.

CAUSE
OF DEATH - . Acute myocardial infarction.

CASE 25

VICTIM - . 51 years old, 20 years service, the last 2 years served as a Lieutenant and an instructor in the fire academy.
. 5'10", 207 lbs., non-smoker, no history of prior heart trouble.

SETTING - . Non-fire situation.
. While sitting at his desk attending to routine office matters and talking on the telephone he suffered a heart attack and collapsed.
. Fire department EMTs performed emergency care on the victim who was placed on a M1lCPR which allowed the hospital to monitor the man's condition.
. For 40 minutes the EMTs followed the hospital's instructions but were unfortunately unable to defibrillate the victim's arrhythmia and he expired.

CAUSE
OF DEATH - . Rupture of coronary arteriosclerotic plaque due to coronary arteriosclerosis. (Autopsy)

CASE 27

VICTIM - . 57 years old, 20 years total service, the last 6 served as a Captain.
. 5'10", 185 lbs., non-smoker, no history of prior heart trouble.

SETTING - . Mid-February 1975, 3:30 A.M.
. Box alarm.
. A 2-story wood frame structure.
. Temperature in the teens, considerable snow on the ground.
. The man was awakened from a sound sleep by the alarm.

ACTIVITIES -
- The Captain and a fire fighter pulled a hose a half block to the area of a hydrant.
- Due to the snow the hydrant was not visible and the Captain dug four holes with his hands until he located it and hooked up.
- The hydrant was tight and it required both men to open it.
- He returned to the scene and directed the fire fighting operations. Shortly after returning he collapsed.
- Mouth-to-mouth resuscitation on the scene, and oxygen and CPR administered en route to the hospital did not revive him.
- Ten minutes after arrival at the hospital he was pronounced dead.

CAUSE OF DEATH -
- Coronary occlusion due to coronary atherosclerosis.

CASE 28

VICTIM -
- 62 years old, 30 years total service, the last 8 years served as a Captain.
- 5'11", 160 lbs., non-smoker, no history of prior heart trouble.

SETTING -
- Late March 1975, 5:00 A.M.
- Box alarm.
- Wood garage fire.

ACTIVITIES -
- Pulled 1½" preconnect line and line to hydrant.
- Hosed down blackened area during overhaul.
- During overhaul the Captain did not feel well and sat in the cab of the engine for 30 minutes.
- As they were returning to the station the driver asked the Captain twice if he wanted to go to the hospital. He responded negatively and the second time ordered him to return to the station.
- The Captain collapsed in the cab as it was backing into the station.
- Mouth-to-mouth resuscitation was administered. When the Battalion Chief arrived oxygen was given and between 10 and 15 minutes later the Captain was placed on the inhalator.
- At the hospital he was treated for 2 hours but this was to no avail and he expired.

CAUSE OF DEATH -
- Acute myocardial infarction due to arteriosclerotic heart disease.

Coupled with physical examinations, physical fitness programs should be instituted in all fire departments. An exercise program will help keep the circulatory system regular and strengthen the myocardium. On top of all of this, since fire fighters are called upon to exert considerable energy at differing intervals on the fire ground a physical fitness program will help keep the man's body conditioned.

In instituting a physical fitness program it is essential that doctors test each individual fire fighter carefully to measure the reserve and capability of his cardio-vascular system. By doing this the fire fighter will not surpass the limits of exercise that his heart can endure. If the fire fighter goes beyond these limits the exercise may be detrimental to the individual's heart as was the situation in case 95.

In reviewing the heart attack cases, very few of the departments had required physical examinations or regulated physical fitness programs. It is believed that if such programs were instituted in fire departments throughout the country it would go a long way towards cutting the incidence of heart attacks among fire fighters.

ASPECTS OF THE JOB

It is important that fire fighters maintain top condition of their body because due to aspects of their job they are already working against overwhelming odds in combating heart disease. There are conditions that fire fighters face in-the-line of duty that are conducive to heart disease. In general terms, these conditions are smoke, stress and over-exertion.

SMOKE

In recent years the medical community has shown that there is a relationship between smoke inhalation and heart disease. Carbon monoxide

has been the most commonly thought of toxic fume affecting the heart; however, in recent years with the increased use of plastic-based materials in construction and household products, fire fighters have been faced with even more deadly fumes given off by burning plastic. The most common of these plastics is polyvinyl chloride, which with its thermal degradation results in the formation of at least 75 identifiably potential toxic compounds. The inhalation of carbon monoxide and other toxic fumes contribute to the development, and irritation in existing conditions, of arteriosclerosis. Carboxyhemoglobin (carbon monoxide in the blood) and other toxic elements affect blood circulation resulting in an inadequate supply of oxygen to the heart, which in turn results in damage to the myocardium. Finally, it is believed that the effects of carbon monoxide and other toxic fumes are cumulative and contribute to the deterioration of the cardio-vascular system.

For the heart attack cases investigated over the duration of this study it is assumed that all of the victims were exposed to carbon monoxide and other toxic fumes in their lives as fire fighters. The average years of experience was 22 years, with the experience limits being 8 and 40 years. One would be naive to believe that even a fire fighter with only 8 years experience was not exposed to toxic fumes to at least some degree or another.

There were 4 cases in which the autopsies showed the presence of carboxyhemoglobin. The cases and levels were: Case 4 - 10%, Case 9 - 16.5%, Case 18 - 10% and Case 30 - 2%. It is quite possible that in these cases the inhalation of carbon monoxide at the fire, and the resulting levels of carboxyhemoglobin in the blood, brought on the heart attack. However, it should be noted that if the victims had not already developed arteriosclerosis it is doubtful that the carbon monoxide inhaled in this fire alone would have resulted

in a fatal heart attack. There were 9 additional cases (Case 16, 34, 35, 41, 42, 44, 52, 80 and 86) in which the victims most likely inhaled considerable amounts of carbon monoxide, but unfortunately this could not be proven since autopsies were not performed, or if they were, carboxyhemoglobin levels were not tested for.

It is assumed that during their careers as fire fighters most of the victims were exposed to and inhaled toxic fumes other than carbon monoxide. There are two cases in which there is a direct relationship between the inhalation of fumes, given off by burning plastic and the fatal heart attack. In Case 14 the fire fighter was subjected to fumes from a burning bean bag chair (vinyl bag stuffed with bits of styrofoam) and in Case 56 the fire fighter inhaled fumes from a burning plastic curtain. Both fire fighters showed delayed reactions - of several hours - to the fumes. Shortly after these incidents both men developed respiratory problems followed by heart disease. One man died 6 months after the fire and the other died 2½ years after. While in neither case could the attending physician state with any certainty that there was a relationship between the inhalation of the burning plastic fumes and the fatal heart attack, these two cases strongly suggest that such a relationship may exist.

The solution to the problem of toxic fume inhalation is simple: in all fire situations it should be mandatory that fire fighters wear self-contained breathing apparatus. In turn, fire departments have the responsibility to provide a sufficient number of breathing apparati be available. There were some cases (Case 18, 28, 43) where fire fighters died of heart attacks and breathing apparatus were not available to them while at the fire. To carry this recommendation one step further fire fighters should also be

required to wear their breathing apparatus during overhaul operations. In many cases, although smoke is no longer present, carbon monoxide and other toxic fumes which are invisible and odorless might still be in the atmosphere. A special hazard that occurs during overhaul is that concrete retains a great amount of heat and releases fumes throughout the operations. Recent tests showed "highly toxic concentrations of hydrogen-chloride to be present in concrete for as long as one hour after the fire has been extinguished."[7] There were three heart attack cases (Case 12, 19 and 81) in which the fire fighter either took off his mask during overhaul or entered the building for the first time, and without a mask, during overhaul operations. Unfortunately, since autopsies were not performed in any of these cases it is impossible to determine if any fumes in the atmosphere had an effect in the heart attack.

STRESS

While the above discussion pointed out ways in-which to eliminate the dangerous effect of toxic fumes on the heart -- for combating the effects of stress on the heart the solutions are not as easily found. For that matter the relationship between stress and heart disease has not been fully developed. There have been recent studies that strongly suggest that in the profession of fire fighting stress may contribute to the development of ischemic and arteriosclerotic heart disease.[8] Stress (measured by increased heart rates) occur under a number of given circumstances: At the time of the alarm, responding to the alarm, particularly severe fires and working in particularly adverse circumstances. Given the generally lengthy years of experience for the heart attack victims it is safe to assume that they all faced at one time or another the above situations and most likely they all suffered some degree of stress.

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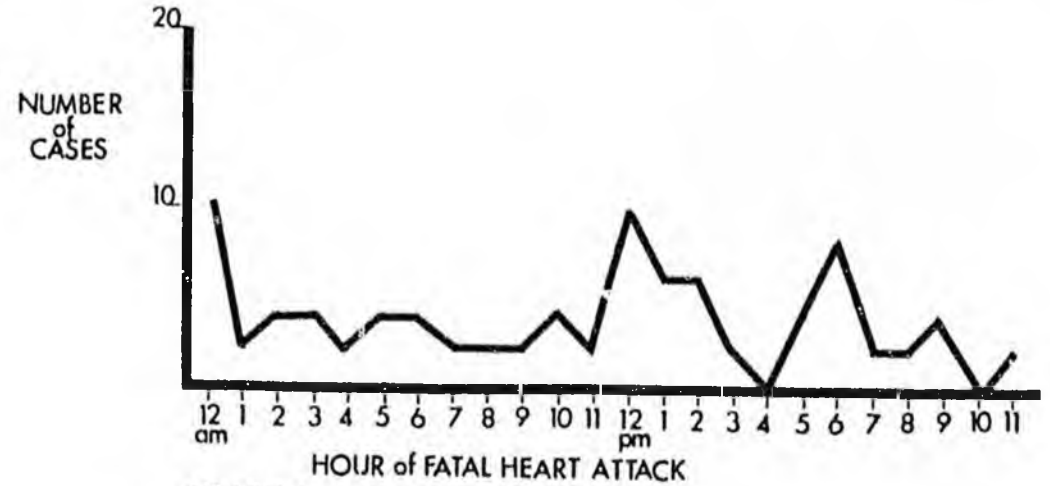
7 .Kobt. Dyer, M. D. & Victor Esch, M. D., "Polyvinyl Chloride Toxicity in Fires" JAMA, Jan. 26, 1976, page 390.

8 .R.James Barnard, PhD., Henry W. Duncan, B.S., "Heart Rate and ECG Response of Fire Fighters," Journal of Occupational Medicine, April, 1975, Vol.17, No. 4, pp. 247-250.

Reviewing the specific circumstances at the time of fatality there were 34 of the 45 cases which occurred at fires. Of these 45, 14 were multiple alarm fires and it is hypothesized that these situations were more stressful than the others. There were four fire situation cases in which, due to the individual's immediate circumstances or activities, he most likely suffered high rates of stress. In Case 4, the Battalion Chief was supervising numerous fire fighters attacking a multiple alarm fire in a warehouse; in Case 27, the Captain feverishly dug through snow in an effort to find a hydrant; in Case 36 the Assistant Chief was active in evacuating occupants from a fire in a high rise apartment; and in Case 80 the fire fighter was responsible for keeping a stairway/hallway clear so occupants could escape from the tenement fire.

It is assumed that all fire fighters experience a certain degree of stress when the alarm sounds. I would hypothesize, that sleeping fire fighters awakened by an alarm suffer a higher degree of stress. In Graph L the frequency of heart attack is distributed according to the hour it occurred. It is noted that between the hours of 12 midnight and 6:00 A.M., 15 heart attack deaths occurred. In 14 of these 15 cases the heart attack victim was awakened by an



GRAPH L

alarm. In a few of the cases, as was true for alarms occurring during other hours of the day, the alarm was not for the given fire fighter but the department had an alarm system that rang in all stations. One way in which to combat the stress brought on by the alarm would be to institute an alarm system that would only ring in the station requiring companies to respond.

It should be pointed out that much of the discussion of the relationship between heart disease and stress was not based on fact statements but rather hypothesis. Based on discussions I have had with fire fighters, as well as the existing research on the subject of stress and heart disease (as minimal as it is), I truly believe there is a relationship. But, this is one area that definitely requires further research if one is to make any conclusive statements concerning fire fighting and stress.

In stress, as was the case with toxic fume inhalation, it is doubtful that any single incident brought on the heart attack. In viewing the effect that stress has on the heart, its cumulative effect has to be realized. Stress experienced on a continuous and regular basis, as is the situation with fire fighters, contributes to the development process of heart disease.

OVER-EXERTION

Oxygen is the fuel that our body needs to produce energy. The greater the amount of energy needed to perform a task corresponds with the ability of the heart to pump at a rate such that it provides sufficient blood-oxygen to the body. Endurance is measured by the ability of the heart to maintain this rate over a period of time. In cases where victims have pre-existing arteriosclerotic heart disease the capacity of the heart to provide the proper blood-oxygen level is limited. Pushing the cardio-vascular system beyond its limits of capability may cause damage to the myocardium - a heart attack.

There is not an apparent relationship between over-exertion and the development of arteriosclerotic heart disease; however, studies have shown that sudden strenuous exercise (work) can produce an ischemic condition (the killing of body tissue) in the heart.[9] Due to the nature of the job fire fighters are often called upon to perform sudden strenuous work.

Given the generally high total years served by the heart attack victims it is possible that at least some of these fire fighters suffered at one degree or another an ischemic condition. It was found that if warm-up exercises are performed prior to performing strenuous work much of the damage is removed.

As was stated it is possible that if the individual has arteriosclerosis a single act of over-exertion may bring on the heart attack. In most cases, due to the presence of arteriosclerotic heart disease, the victim would have suffered a heart attack sooner or later, but the over-exerting activity may have brought the heart attack on a year, five years or even ten years before it would have normally occurred.

It is impossible to state with certainty that in any of the heart attack cases over-exertion brought about the heart attack. However, in the course of my investigations there were numerous incidents in which fire fighters reported that they felt that they were pushed to the point of over-exertion. In general these cases usually showed that there was less manpower on the scene compared to situations in which the question of over-exertion was not raised. It seems only logical that if you have two comparable fires and on one the initial response is ten men and on the other it is 20 men, in the former the

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9 .Barnard, R. S., Gardner, G. W., Diaco, N. V., "Cardiovascular Responses to Sudden Strenuous Exercise - Heart Rate, Blood Pressure, and ECG. Journal of Applied Physiology, 34:833, 1973.
.Barnard, R. J. , MacAlpin, R. N., Kaltus, A. A., Buckberg, G. D., "Ischemic Response to Sudden Strenuous Exercise in Healthy Men" Circulation 48:936, 1973.

men are going to have to perform more duties and exert more energy, for a longer period of time than compared to the latter. The cases most clearly representative of this problem were Cases 8, 18, 43 and 44. As a matter of fact, in Case 44 the victim himself pinpointed this as a problem. In this case the Battalion Chief, who was responsible for directing the overall fire fighting operations, assisted in pulling hose to the roof, remained on the roof helping the men to open it up and manned a 1½" line for awhile. All of this was done in addition to his supervising the fire fighting. When asked by another officer, while in the hospital before suffering his second and fatal heart attack, why he performed the extra duties, he responded, "I had to do it, I didn't have enough men."

Finally, it should be noted that due to the protective clothing fire fighters must wear to protect themselves from fire hazards, the fire fighter is forced to exert additional energy. In a recent study it was shown that "the energy cost of wearing fire fighting protective clothing and equipment (turnout coat, helmet, boots, and breathing apparatus) is roughly one third above the energy required in performing what is essentially a moderate work load."[10] Considering the above fact and that fire fighters are called upon to perform very strenuous duties (from pulling hose to carrying people from burning buildings) it is obvious that fire fighters must be in top physical condition to have and maintain the energy level needed to perform the job.

The point of the preceding section was to explain to the reader that there are aspects of fire fighting that are conducive to heart disease. As was pointed out most of these factors tend to have a cumulative effect

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10 .Davis, P. O., Sarta Marice, D. L., "Energy Cost of Wearing Firefighting Clothing and Equipment," 5th Annual Symposium, University of Maryland, Sports, Medicine & Physical Fitness Center."

and usually are not manifested until the fire fighter is in his middle years. It would be a grave mistake to single out any one aspect about the individual on the job in explaining the reason for the heart attack. These conditions must be dealt with in their entirety, for they are all inter-related, and all contribute to the development of heart disease. There are a few procedures that can be taken to remove the risks in the job (i.e., use of breathing apparatus) but for finding solutions to most of the other harmful aspects of the job further research is definitely needed. Given the fact that aspects of fire fighting can attribute to heart attacks it is essential that fire fighters maintain peak physical fitness. Most likely this alone will not remove the threat of heart attack to fire fighters but it will definitely give the individual an advantage to combating these risks in his profession.

OTHER PROBLEMS IN THE HEART ATTACK CASES

HEART ATTACK AFTER MEAL

In referring back to Graph L, the distribution of the time of attack, it is noted there is a high incidence of heart attack at 12:00 noon (5), 1 p.m. (3), 2 p.m. (3) and 6 p.m. (4). In most of the fatalities occurring during these hours the fire fighters had eaten a meal prior to the fire and heart attack; some of the meals were light, but most were large meals. Dr. Dyer has stated that several hours after eating a large meal the stomach and cardiovascular systems compete for blood circulation and oxygen. Should the fire fighter catch a fire under these circumstances, given all other factors he's exposed to and his physical condition, the fact that he has just eaten a large meal may precipitate an acute myocardial infarction. In an effort to remedy this situation it is recommended that healthwise, it would be better if fire fighters would eat several light meals during their tour of duty rather than

News Watch

Stats Show Fire Fighting Still Most Hazardous

The 1979 Annual Death and Injury Survey, recently released by the International Association of Fire Fighters (IAFF), shows that fire fighting is still the most hazardous profession in the public sector.

With 930 municipalities reporting, the statistics revealed that 70 fire fighters died in the line of duty, while 77 died as the result of occupational diseases. Though this is a decrease for the second consecutive year, the 10-year average remains 80 deaths per 100,000 fire fighters.

Lynn Ilon Photo by Walter Hoey

The IAFF survey reported more than 45,000 fire fighter injuries during 1979.

In 1979, the 68.6 deaths per 100,00 fire fighters were up slightly from 1978 and were nearly double the police officers total of 35.7 deaths per 100,000.

The 1979 statistics for fire fighter deaths from occupational diseases increased significantly to 77 from 61 in 1978. Heart disease was a contributing factor in 57% of the cases and was the leading cause of death. According to the report, "Combined, heart and lung disease constitutes 65% of all reported fire fighter deaths from occupational diseases."

Most of the fire fighter injuries, 31,403 of a reported 45,070, were suffered on the scene while engaged in emergency operations. "Sprains and strains lead the list of causes," the report said, "followed by cuts, inhalation of toxic gases, burns and over-exertion. The balance of 13,667 injuries came while responding, returning or during training and other work-related duties."

The injury figures showed an average of 44.2 injuries per 100 fire fighters, meaning that there is a greater than 40% probability that every fire fighter will be injured at least once during the year.

"In 1979, 338 fire fighters were forced to leave their departments or retire as a result of injury sustained on duty," the survey indicated, "and another 348 were forced to leave the department or retire as a result of occupational disease."

For additional information on the survey, contact *Mary Barber, International Association of Fire Fighters, 1750 New York Avenue, N.W., Washington, D. C. 20006, telephone (202) 872-8484.*

Volunteers Covered By Freedom Of Information

A recent ruling by the New York State Court of Appeals has found that volunteer fire departments are subject to the Freedom of Information Law in all respects.

The ruling, in the case of *Westchester-Reckland Newspapers v. Kimball*, cited the legislative declaration in section 84 of the law which states "it is incumbent upon the State and its localities to extend public accountability wherever and whenever feasible."

To further explain the ruling, the court said, "True, the legislature, in separately delineating the powers and duties of volunteer fire departments, for example, has nowhere included an obligation comparable to that spelled out in the Freedom of Information statute. . . . But absent a provision exempting volunteer fire departments from the reach of article 6—and there is none—we attach no significance to the fact that these or other particular agencies, regular or volunteer, are not expressly included."

The ruling by the New York Court of Appeals makes it clear that volunteer fire departments have the same obligations under the Freedom of Information Law as the governmental agencies which are subject to its provisions.

For more information, contact *Committee on Public Access to Records, NYS Department of State, 162 Washington Avenue, Albany, New York 12231, telephone (518) 474-2518.*

IAFC Participates in Career Assessment Project

The International Association of Fire Chiefs (IAFC) has been selected as the subcontractor in a project to develop a career assessment test battery for the National Fire Academy's Career Development Center.

The IAFC will be working with Research Applications, Incorporated, a Washington, D.C., consulting firm that specializes in performance and career assessment testing. The two organizations are charged with identifying assessment tests to aid Academy personnel in discovering the strengths and weaknesses of career and volunteer fire officers who desire advancement in the fire service.

The tests to be prepared will consist of criterion-referenced measures used to assess skills and competencies of personnel identified by NFPA standards 1021, 1031 and 1041. The tests will be administered to participants during a week-long assessment program at the National Fire Academy. The test will include performance measures, paper-and-pencil exams, and structured interviews.

A seven-member task force will provide the project staff with a resource group of fire officers who have expertise in management, testing and career development. The

DR. ROBERT F. DYER

EFFECTS UPON FIRE FIGHTERS AFTER EXPOSURE TO THE PRODUCTS OF DECOMPOSITION OF POLYVINYL CHLORIDE.

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I greatly appreciate the honor of addressing this group devoted to the study of the occupational hazards of firefighters. In general about 12,000 citizens of the U.S.A. are killed annually by fire. Another 300,000 citizens are scarred and injured each year. Eighty percent of people who die in a fire actually expire from the effects of smoke and toxic gases. I have been studying the clinical effects of inhalation of toxic fumes in my capacity as an internist with the Board of Police and Fire Surgeons since 1963, and especially the results of exposure of this occupational group to decomposition of the products of pyrolysis upon plastic since 1968. In 1970 the death of a firefighter from severe pulmonary hemorrhage and pulmonary edema due to chemical pneumonitis, secondary to the inhalation of chemicals from smoke at a fire was reported.[1]

A program was instituted at the Firemens Clinic in Washington, D.C. for immediate followup and care of firefighters exposed to toxic fumes from plastic decomposition. Clinical research into the effects upon humans after exposure to the breakdown products of polyvinyl chloride was concentrated among the fire fighters who were exposed either during a fire or during the overhaul period when the cleaning up was done. Thermal degradation of polyvinyl chloride was found to release hydrogen chloride as well as 74 other chemical products. Hydrogen chloride gas is an irritant to the mucous membranes of the eyes and the respiratory tract. Hydrogen chloride causes desquamation of the bronchial epithelium when exposed tissues are studied microscopically by the pathologist. Another product, benzene, may cause changes in the blood cells; carbon monoxide may cause death when released also.

FREQUENCY OF EXPOSURE

The 3,600 firefighters in Washington, D.C. are primarily assigned to the firefighting division. From 1970 to 1976 we evaluated 190 cases of firefighters exposed one or more times to toxic fumes from fires where polyvinyl chloride was identified. Consultation with the fire department offiicals and the safety officer produced orders that the self-contained mask would be worn by all firefighters during any fire where plastic was involved, as well as during the overhaul period, where exposure to hydrogen chloride on soot particles was still a danger.

CLINICAL SYNDROME EXPOSED

The typical signs and symptoms reported by firefighters exposed to the fumes from decomposing plastic are a choking cough, a pain in the anterior chest, pain in the back of the throat, severe frontal headache, shortness of breath, dizziness, and an irregular pulse. The presence of these characteristic findings was

universal among the fighters exposed, except for irregular pulse, which was unnoticed by many, and noted on physical examination or on electrocardiograms, by about 20% of the men. I was especially interested in this finding since I had studied the cardiovascular effects of occupational exposures on firefighters for many years as a fire surgeon, and had noted in the early 1960's that those men exposed to plastic at the fire ground had reported weakness, and were found to have hypertension when examined at their homes in the period of 10 to 18 hours after the fire. In retrospect these individuals may have suffered from premature heart beat syndromes just after the fire, causing scondary hypertension.

FORMAT FOR EVALUATION OF EXPOSED INDIVIDUALS

In cooperation with the fire officials we ordered all exposed firefighters to be examined at either the fireground by the attending fire surgeon, or at the Firemens Clinic, or at the local Emergency Room, when plastic was identified at the fireground. The medical surveillance program consisted of the following:

1. Comprehensive medical history (usually already on record at the Firemens Clinic); including past and present medical history, occupational history, past exposure to hepatotoxic, cardiotoxic or renaltoxic chemicals.
2. Physical examination by the fire surgeon, including skin inspection, eyes, ears, nose, and throat exams, lungs, heart and vascular system, abdomen, etc.
3. A 14 x 17 posterior and anterior view x-ray of the chest.
4. Pulmonary function test when available. Forced vital capacity (FVC) and forced expiratory volume, 1 second (FEV_1) are determined.
5. Clinical laboratory procedures consisting of: Complete blood count; a blood chemistry profile including bilirubin, total protein, lactic dehydrogenase (LDH), serum glutamic oxaloacetic transaminase (SGOT), alkaline phosphatase, cholesterol, blood sugar, blood urea nitrogen (BUN), and serum electrolytes, as well as arterial blood gases. A routine urinalysis, serology, and cytology studies on the sputa were done also.
6. Electrocardiogram, 12 lead type. If abnormal, or if clinically indicated, the patient was monitored by electrocardiogram for as long as 24 to 36 hours after exposure. In these situations, premature heart beat syndromes were studied.

If the firefighter was found to require observation beyond that done in the Clinic or Emergency Room, he was admitted for 24 to 72 hours of observation in the hospital by the fire surgeon. About 40 firefighters have required hospitalization.

PREMATURE VENTRICULAR BEATS (PREMATURE HEART BEATS)

Since adverse symptoms may not develop for as long as 12 hours to 24 hours after exposure to toxic fumes, it was noted that electrocardiogram monitors showed some firefighters in the exposed group had premature heart beats while in the hospital.[3] This might have gone unnoticed if the firefighter was not under constant surveillance. It is known that there are certain premature ventricular beats that are benign and others that are the premature heart beats that lead to sudden death syndrome. Because of the latter situation, especially in those firefighters in their 30's to 50's (years of age), with underlying coronary artery disease, the finding of premature beats is of great importance to fire surgeons. All instances of premature heart beats have converted spontaneously or responded to lidocaine therapy in this group of firefighters.

PREVENTION, AND TREATMENT

In addition to educating the firefighters at seminars and during their Training School lectures, and requiring the use of the self-contained breathing apparatus at all times when plastic is identified at the fireground, the following steps have been utilized: (1) Use of bronchial decongestants to improve tracheobronchial ciliary action after exposure. (2) Bronchodilators. (3) Oxygen administration, at 5 liters per minute by nasal canula, to those exposed. (4) High humidity oxygen by mask technique to those hospitalized. (5) Intravenous steroid therapy to those with marked toxicity when hospitalized. (6) Intravenous Lidocaine drip therapy to those with premature beats in excessive amounts while hospitalized.

(7) Bed rest for 8 hours at home or in hospital, after severe exposure. (8) Avoiding black coffee, cigarettes, and stimulants when premature heart beats are found. (9) Use of ammonia ampules for inhalation at fireground after inhalation of toxic fumes.[3]

AREAS OF FUTURE STUDY

Human toxicity from the degradation products of polyvinyl chloride as well as other plastics which result from man's technology are being studied clinically by fire surgeons in Washington, D.C. Chemists are verifying in their laboratories many of these phenomena in controlled experiments. Because of the insidious nature of the effect of toxic fumes on the firefighter, fire surgeons and emergency squad, as well as emergency room personnel should be educated as to the recognition and treatment of these conditions. The vulnerability of those firefighters with incipient coronary artery disease to these dangerous syndromes, e.g., premature heart beats leading to sudden death syndrome, should be understood by firefighters and fire surgeons alike. All atmospheres at the fireground should be suspect. An effective gas analyzer to detect concentrations of toxic gases present at the fireground should be found. Further definitive cardiology studies should continue to be performed on exposed firefighters. The term, "smoke inhalation" should be replaced by the term "inhalation of toxic combustion products" so that national recognition to the seriousness of this problem can be followed.

Bibliography

1. Dyer, R.F., and Esch. V. H., Polyvinyl Chloride Toxicity in Fires. J.A.M.A., Jan. 26, 1976, Vol. 235. No. 4. pp. 393-397.
2. Ibid.
3. Esch, V. H., personal communication.

PANEL NO. 2—CARDIAC DISEASE IN THE FIRE SERVICE

DR. R. JAMES BARNARD

Chairman

HEART DISEASE IN FIRE FIGHTERS

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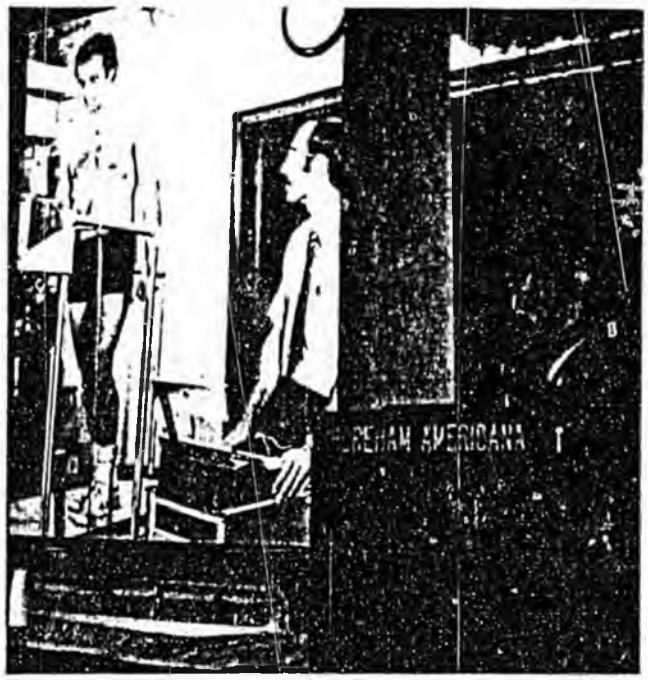
At the past three Redmond Symposia much attention was focused on the heart disease problem of fire fighters. The published proceedings from these meetings all pointed out the need to gain more information about the actual extent of the heart disease problem and how it might possibly be reduced. Since the last symposium in 1975, considerable research has been published on the etiology of coronary artery disease and some research has been done on the heart disease problem in fire fighters.

It must be realized that the term "heart disease" is a very general term which includes many abnormalities of the heart. Problems including valve dysfunction, aortic stenosis, arrhythmia, etc. can be readily detected through routine medical examinations. Ischemic heart disease, however, is more difficult to detect and unfortunately is probably the most common heart disease in fire fighters.

The term "ischemic" technically means a reduced blood supply. However "ischemic" heart disease has become a more general term which means an inadequate oxygen supply to the heart muscle. Since the heart is continually beating it requires a continual supply of oxygen. If the heart becomes too ischemic a myocardial infarction or heart attack occurs which means that some of the heart cells have died because of a lack of oxygen. Ischemia is very common in our society and is generally caused by coronary artery disease (atherosclerosis) which is due to the accumulation of smooth muscle cells and lipids (cholesterol and triglycerides) in the coronary arteries which reduces blood flow and oxygen supply to the heart muscle. Ischemia may also be caused by other factors which reduce oxygen delivery to the heart muscle including anemia and carbon monoxide inhalation. Factors which excessively elevate oxygen demands of the heart may also cause ischemia. Aortic stenosis, severe hypertension, or excessive amounts of adrenalin may greatly elevate myocardial oxygen demands.

In order to gain some insight into the ischemic heart disease problem in fire fighters we studied a random sample of 90 Los Angeles City Fire Fighters, 40-59 years of age (1). The testing consisted or near-maximal electrocardiographic (ECG) stress testing and coronary artery disease risk factor (cholesterol, hypertension, smoking) analysis. The results showed



that 10% of the men had ECG changes indicating the presence of ischemic heart disease. This percentage is higher than that found in other groups: Los Angeles insurance executives 8% (2), Indian State Policemen 5% (3), aircraft pilots and controllers 4% (4). When the risk factors for coronary artery disease were examined the men were found not to be at high risk. Only one fire fighter had all three risk factors elevated and 47 had no abnormal risk factors. The results of this study prompted us to conclude that "Since fire fighters are a medically-selected population with low risk factors for coronary heart disease, the observed incidence of ischemic stress tests is surprising and suggests that ischemic heart disease may be job associated."

Follow-up studies were conducted on the 9 men with ischemic stress tests (5). All of these men had cholesterol values within the normal range and 3 had values below 200mg% which are very low. One individual was hypertensive and two were smokers at the time of testing. Thus, even these men had low risk factors for coronary heart disease.

Six of the men elected to undergo cardiac catheterization and angiography. One patient had severe coronary artery disease in three vessels and subsequently underwent aorto-coronary bypass surgery. This man never experienced chest pains and had no idea that he was at high risk for sudden death. Another patient had obstructive (50%) coronary disease in one vessel while the other four men no visible signs of coronary obstruction. men with "normal" coronaries, however, show signs of abnormal cardiac function du atrial pacing. One man had cardiac enla ment, hypokinesis, ischemic ECG and abno lactate metabolism. Another had abnormal tate metabolism and ischemic ECG. A t man had moderate cardiac enlargement anterior wall hypokinesis. The fourth ischemic ECG changes with angina but ot wise normal cardiac function. All four of t men had pressures which were within no limits.

The results of this study show that some fighters have "ischemic" heart disease whic not due to coronary artery disease. Altho these men may not be at high risk for sud death they should not be continually exp to factors which may have caused or aggravate the problem. This type of isch heart disease may be due to job related fac such as carbon monoxide and other noxi fumes which limit oxygen supply or by adre lin which greatly increases oxygen demand the heart.

In 1976 the International Association of Fi Fighters published their Fire Fighter Mortal Study (6). The results showed that of the 1 on-duty deaths which were investigated 45 we caused by heart attack. The mean age of t men was 51.3 years, the youngest being

years. The mean years of service was 22. In over one-third of the cases, fire fighters experienced symptoms of heart trouble before the actual attack. Proper medical management may have saved some of these men. Fire fighters who experience angina (chest pains) should not be involved in fire fighting. Seven of the men had suffered prior heart attacks and probably should not have been working as fire fighters. Fire fighting can be a strenuous occupation which can tax the heart to maximal limits and is not an activity for people with known heart disease (7).

Indeed much progress has been made during the past two years. The other three speakers on the panel will expand on some of the concepts which I have introduced and will describe some of their work with fire fighters.

REFERENCES

1. Barnard, R. J., Gardner, G. W., Diaco, N. V., and Kattus, A. A. Near-maximal ECG stress testing and coronary artery disease risk factor analysis in Los Angeles City fire fighters. J. Occup. Med. 17:693, 1975.
2. Kattus, A. A., Jorgensen, C. R., Worden, R. E. and Alvaro, A. B. S-J segment depression with near-maximal exercise in detection of preclinical coronary heart disease. Circulation 44:585, 1971.
3. McHenry, R. L., Fisch, C., Jordan, J. V. V. and Corya, B. R. Cardiac arrhythmias observed during maximal treadmill exercise testing in clinically normal men. Am. J. Cardiol. 29:331, 1972.
4. Spangler, R. D., Horman, M. J. Miller, S. W. et al. A submaximal exercise electrocardiographic test as a method of detecting occult ischemic heart disease. Am. Heart, J. 80:752, 1970.
5. Barnard, R. J., Gardner, G. W., and Diaco, N. V. "Ischemic" heart disease in fire fighters with normal coronary arteries. J. Occup. Med 18:818. 1976.
6. Fire Fighter Mortality Report. Int. Assoc. Fire Fighters, Washington, D.C. 1976.
7. Barnard, R. J. and Duncan H. W. Heart rate and electrocardiographic response of fire fighters. J. Occup. Med. 17:247, 1975.

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CORONARY HEART DISEASE: CAUSE AND PREVENTION

HEART ATTACKS—OUR MAJOR CAUSE OF DEATH

Heart attacks, due to coronary heart disease, still remain the leading cause of death in the United States. There are over 1,000,000 heart attacks a year and more than half of them result in death. Coronary heart disease far exceeds cancer as the cause of death in the United States and, for persons in the age group 35-64 years, nearly one death in three is caused by coronary heart disease. Two-thirds of deaths due to coronary heart disease with heart attacks occur outside the hospital, the majority occurring so suddenly (within one hour of the onset of symptoms) that effective medical treatment is unavailable. One-half of all persons who

life. Healthful living has no bad side effects. In addition, correcting the risk factors and improving our life style has numerous benefits above and beyond the control of occlusive vascular disease. Improvement in our stamina, improvement in physical appearance, better energy, and a definitely increased sense of well being are important beneficial spin-offs of healthful living. For those individuals in the emergency services who are responsible for the public's protection, these spin-offs may be just as important and just as significant as the improvement attained in their cardiovascular health.

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HEART DISEASE IN FIRE FIGHTERS

Today I wish to explore with you the relationship between a fire fighters work and the appearance of job-related coronary heart disease.

My experience began on this subject when I was first requested to consult on job-related heart cases in Colorado (which has an "unusual strain" rule) following my fellowship in cardiology at the University of Colorado Medical Center. When I later went to Boston as a fellow in environmental health at the Harvard School of Public Health, Dr. John Peters, who is well known for his studies documenting lung function impairment in fire fighters,[1, 2] asked me to design a project to evaluate carbon monoxide exposure and other coronary heart risk factors in Boston fire fighters.

The Boston fire fighters, particularly Chiefs Buchanan and Stapleton, cooperated completely and were extremely helpful. Of potential fire stations to study in Boston, I chose the second busiest in New England, which had an average of 12.63 calls per 24 hours. From my on-the-site experience with the engine and ladder companies at this station, it was not unusual to go out to one, then two or even three fires in succession before returning to the fire station. These companies also provided a superb balance mix of young, middle-aged, and older fire fighters to observe.

Prior to starting, we were aware of the reports of Mastromatteo in Canada and Day in Kansas City which reviewed frequency of coronary heart disease in fire fighters.[3, 4] However, as pointed out by Sammons in your last conference in St. Louis,[5] statistical analyses were not in depth in these reports, and the conclusions in Day's report were based upon including disabled fire fighters retired due to impairment from heart disease in the active groups

studied, perhaps skewing the results. We had also reviewed Gordon's interesting "Project Monoxide" study which strongly suggested that carbon monoxide has a toxic effect on heart muscle.[6] As well, the information was available from the studies of Astrup and Thomsen showing that low levels of carbon monoxide enhance and accelerate the appearance of arteriosclerosis (hardening and thickening of the walls of arteries) in animals.[7, 8] Aronow had just reported that men with angina pectoris (or chest pain related to coronary heart disease) had an earlier onset of their chest pain during treadmill exercise when exposed to carbon monoxide.[9] Goldsmith's group had shown a higher frequency of deaths in Los Angeles due to acute heart attack in those districts in the city on days of higher ambient carbon monoxide,[10] plus Ayres had demonstated in the laboratory that the diseased human heart becomes even more impaired in function when exposed to low levels of carbon monoxide.[11]

Before studying our 31 fire fighters in Boston, each was oriented to the project, completed a medical interview and screening exam, and had blood drawn for cholesterol, triglycerides, and glucose, plus a resting electrocardiogram was recorded. With the average age of 41.9 years (Table 1), coronary risk ratios were calculated based upon Framingham risk tables, and a risk ratio of 1.49 to 1 was obtained.[12]

BOSTON FIRE FIGHTERS	
Age range:	24-58, $\bar{X}$=41.9, SD=11.9
Coronary risk ratio (Framingham):	1.49/1.00
Work study	A. Holter electrocardiograms B. Ambient and expired breath CO C. Urinary catecholamines

While this suggests that fire fighters possess excess cardiac risk before approaching work, numbers were not sufficient by sign testing in this small sample to account for significance.

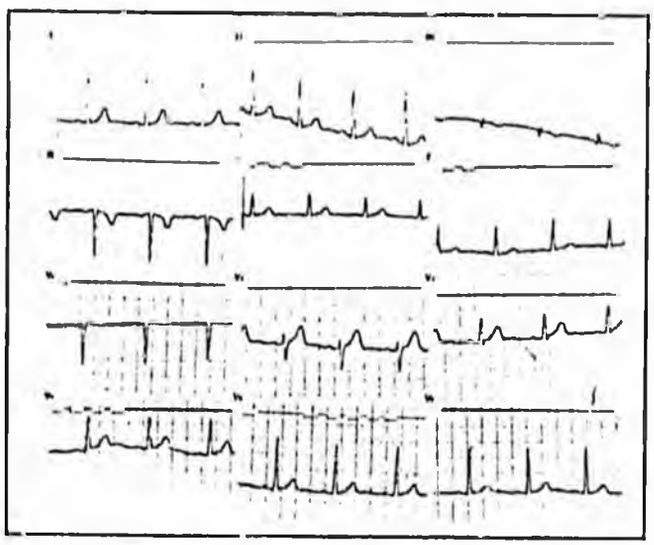
The on-the-job work portion of our study involved monitoring three areas: 1) Holter (portable continuous) electrocardiographic tape recordings were made over a work shift and played back for analysis; 2) ambient carbon monoxide was measured with a portable monitoring device worn on the belt and expired breath samples were obtained for carbon monoxide upon leaving fires; 3) urine was collected during the work shift for measurement of adrenalin-like compounds (catecholamines) which might reflect job stress.

The results of the portable continuous electrocardiogram (Table 2) show heart rates ranged from an average low of 70.3 beats per minute at rest to as high as 200 beats per minute during a fire call with the average high rate of 148.9 beats per minute. Satisfactory tracings for analysis were obtained on 28 of 31 fire fighters.

HOLTER ELECTROCARDIOGRAMS BOSTON FIRE FIGHTERS AT WORK 31 subjects, 28 satisfactory recordings		
Rate ranges:	mean low	70.3 beats/minute
	mean high	148.9 beats/minute
	maximum	200 beats/minute
Premature ventricular beats		8/28 (29%)
"Ischemia" (ST depression 1.0mm. or greater)		6/28 (21%)
T wave changes		2/28 (7%)

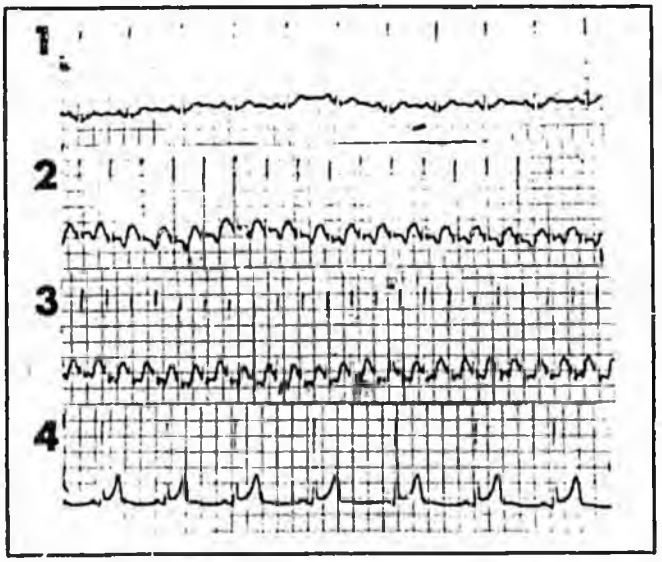
Irregular heart beats characterized as premature ventricular beats were seen in 8 of the tracings or 29%. Of significance, however, are the electrocardiographic changes described as "ischemic" in 6 of the tracings or 21% which cannot be considered normal. In their laboratory, Barnard and his colleagues have shown that stress treadmill tests designed to duplicate "normal" acute workloads cause similar changes.[13, 14] In our group, 2 or 7% also showed evidence of T wave changes alone, which less significantly reflect cardiac stress.

In the next series of illustrations I would like to show you actual examples of these changes. The first depicts a normal resting electrocardiogram in a 24-year old fire fighter (Figure 1) which is in turn followed by four



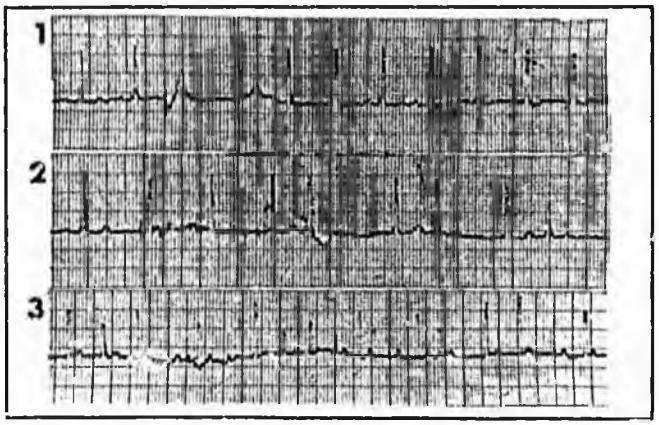
strips taken from his tape recorded electro-

cardiograph at work (Figure 2). Strip #1 was taken at rest, while strips #2 and #3 were taken from a work stress period of 30 to 40 minutes in an apartment house fire where ambient recorded carbon monoxide reached 220 ppm and an expired breath carbon monoxide taken immediately after estimated a blood carbon monoxide (carboxyhemoglobin) of 16.5%.



Of pertinence is that he was a non-smoker not wearing a self contained breathing apparatus and noticed only modest fatigue and a mild transient headache afterward. While his exercise strips #2 and #3 show heart rates of 142 and 186 per minute respectively with ST changes compatible with ischemia or inadequate coronary circulation, his post-exercise strip #4 revealed ST segment elevation which raises the question if some degree of permanent cardiac damage occurred.

The irregular heart beats described as premature ventricular are shown in a 54-year-old fire fighter in the next illustration (Figure 3)



of work related electrocardiographic changes. Since these consist of bursts of irregular beats with some sinus node irregularity as well, these should be considered relatively hazardous compared to simply an occasional irregular beat.

Measurement of the ambient carbon monoxide levels detected elevations as high as 4140 ppm. This exposure occurred in a non-smoker who was fortunately wearing a self contained breathing apparatus throughout his exposure and had an expired breath level of 29 ppm (for an estimated blood carbon monoxide level of 6.5%). While his heart rate increased with exercise, irregular heart beats or changes of ischemia were not seen. This confirms the impression of Radford in Baltimore that continuous use of self contained breathing apparatus can provide good protection.[15]

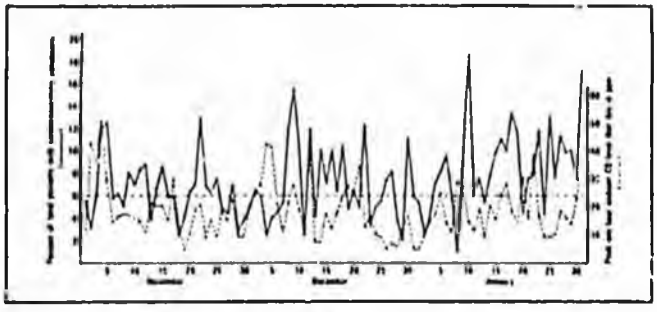
To assess work stress, urinary measurements of adrenalin-like compounds (catecholamines) were performed. Disappointingly only 4 of 31 (13%) were elevated. That these elevations occurred was important, of course, but factors which may have resulted in fewer elevations than expected were: 1) since this was the second busiest fire station in New England, perhaps these fire fighters were more conditioned and relaxed under conditions that may have been more stressful to a fire fighter with a lesser frequency of call experience; 2) the adrenalin-like compounds can be divided into categories of norepinephrine and epinephrine, and since norepinephrine more closely reflects exercise stress than epinephrine, perhaps norepinephrine should have been measured;[16] 3) although the urine was collected under proper conditions of acidification, some breakdown of urinary adrenalin-like compounds could have occurred during the transportation from the night shift at the fire station to the clinical laboratory measuring the levels.

Next, I would like to show you the results of research that has been performed with my colleagues, Drs. James Chandler and Peter Mogielnicki at the University of Colorado Medical Center. We studied the association of high ambient peaks of carbon monoxide in Denver with the frequency rate of presenting complaints of chest pain (not related to trauma) and shortness of breath seen in the Emergency Department at Colorado General Hospital. A study reported by Stewart on banked blood in all major cities in the United States showed that blood carbon monoxide levels in nonsmokers in Denver equalled those of Los Angeles as being the highest.[17] One hour maximum carbon monoxide levels above 35 ppm are not

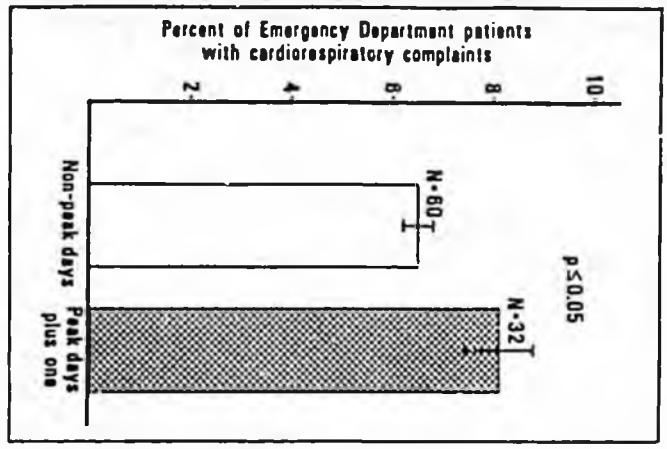
uncommon in Denver and sometimes exceed 50 ppm. Since Denver has very little heavy industry and natural gas is burned for heating rather than fuel oil or coal, sulfur dioxide and other sulfur compounds provide no major pollution.

As well, the November through January temperature inversion season which results in the highest carbon monoxide levels, occurs at a different time of the year than the late spring and early summer elevations of ozone and oxides of nitrogen. Therefore, Denver presents a rather unique environmental situation which allows one to assess relatively high levels of carbon monoxide, minimally contaminated in effect by other pollutants, on a large urban population.

Under these conditions, we matched the daily frequency of patients present with nontraumatic chest pain and shortness of breath in the Emergency Department over the three month November through January period. 8556 patient encounters were reviewed for a daily mean of 93. Matching the daily frequency rate of chest pain and shortness of breath with the ambient carbon monoxide levels recorded at a nearby 24-hour monitoring station (Figure 4),



we found a low level trend of association between those presenting complaints and the ambient carbon monoxide levels. The low level correlations were consistent whether the carbon monoxide levels were expressed as one hour maximums, 24-hour means, or two day moving averages (varying in significance from p less than 0.02 to 0.05). This effect tended to persist for one day after ambient carbon monoxide peaks returned to safer levels (Figure 5), which is not surprising since the half life of carbon monoxide in the blood (at Denver's altitude) is four hours (and five times a half life or 20 hours would be necessary to reach a negligible level).



The only exception in our association between ambient carbon monoxide and presenting nontraumatic chest pain and shortness of breath was the period of December 24 to 29, during which ambient carbon monoxide was low and the frequency of complaints was high. This was likely related to factors in the Christmas holiday season, and if subtracted would increase the significance of our results.

The daily mean one-hour maximum carbon monoxide during the November through January study period was 18.71 ppm with a significant (p less than 0.001) fall to a mean of 12.60 ppm during the two month period following (Table 3). This indicates that we had correctly selected a high ambient pollution period for study.

CO one hour maximum

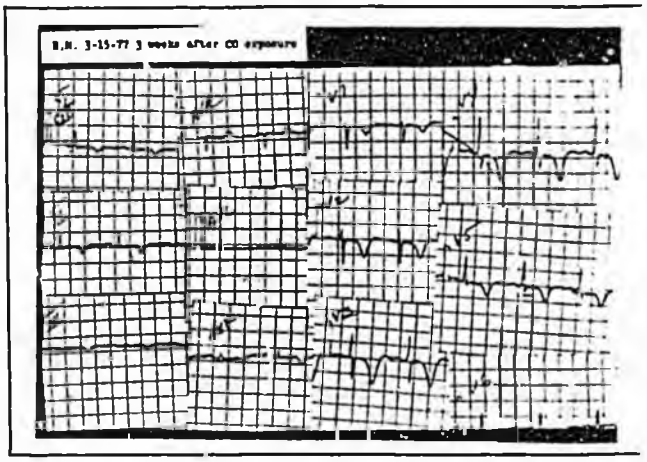
	Inversion season	Early spring
	Nov-Dec-Jan	Feb-March
Mean	18.71	12.60
SEM	1.01	0.69

Significance testing for difference $p<0.001$

Recently, I consulted on another research project in Denver at the National Asthma Center under an Environmental Protection Agency contract to assess the effects of low levels of blood carbon monoxide on heart and lung function. The principal investigators on this project, Drs. Phillip Weiser and Jerry Cropp, exercised ten normal young men to maximal capacity on a treadmill, four times each, twice with carbon monoxide and twice with filtered air. The carbon monoxide was given in a double blind fashion, that is, a technician determined whether the air breathed was filtered or contained a standard amount of carbon monoxide,

and neither the physician or subject knew which was being given at a particular time. Since pure carbon monoxide, unlike smoke, is an odorless and tasteless gas, this is easy to do. The outcome of these studies confirmed and extended that reported by Horvath, namely, that low blood levels of carbon monoxide (about 4.5%) significantly decrease treadmill exercise performance as measured by oxygen consumption and work performed.[18] As well, we found by a non-invasive measurement technique called systolic time intervals, that demands on left ventricular performance (the heart's ability to pump) are significantly increased by exercise during exposure to carbon monoxide.

Next there are anecdotal cases of carbon monoxide toxicity which were encountered in our emergency room, which I'd like to relate to you. The first concerns a 36-year old man who had a blood carbon monoxide level of 41% after discovering that the blow hose he was maneuvering to pump insulation into an attic was pumping in carbon monoxide from the truck's exhaust as well. After he experience no heart problems before exposure, he has had a dangerously excessive number of multiform irregular hearts beats (premature ventricular beats) in spite of taking medication to control them. He has had normal catheter x-ray studies of his coronary arteries (coronary angiograms), and now continues to have frequent irregular heart beats one year since his exposure. Another patient, a 64-year old woman was in a state of near collapse due to carbon monoxide inhalation from a defective space heater in her trailer. When first seen in our emergency room, her blood carbon monoxide level was 36% and her electrocardiogram was normal (Figure 6). She improved rapidly on mask oxygen and was re ferred to the medicine clinic for evaluation o diabetes because her blood sugar was elevated For routine medical screening her electrocar diogram was repeated three weeks later in th medical clinic (Figure 7) which revealed dif



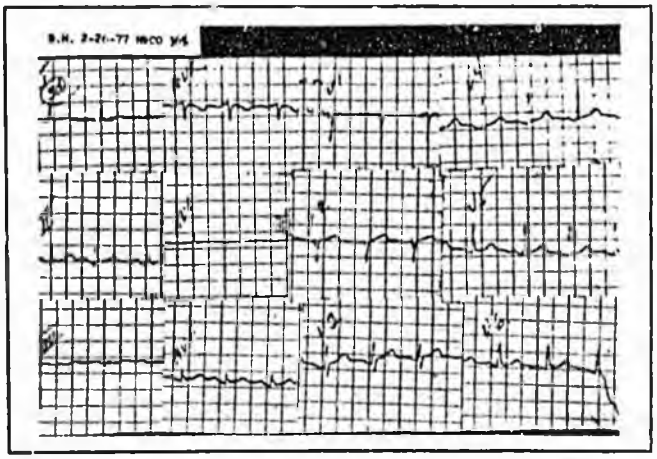
fuse T wave changes compatible with pericar ditis. An echocardiogram confirmed this show ing a small pericardial effusion (fluid formc in the sac around the heart, as an inflammato reaction). Both of these cases, plus others in th medical literature are warnings that signi cant blood levels of carbon monoxide inhal tions may have a delayed effect on the hea and should be seen by the physician in two o three return visits.

Dividing levels of carbon monoxide exposu into three categories, there are different grad tions of effect on the heart.[19-21]

1. Low level of carbon monoxide in the blo (2 to 12%) can occur in mild smoke inhalati and normal cigarette smoking. These can b associated over the years with the chronic ge eration of arteriosclerosis (hardening and thic ening of the arteries) as well as the acute pr cipitation of symptoms in those already wi chronically partially clogged arteriosclerot vessels.

2. Modest to moderate blood levels of carb monoxide (12 to 25%) have been associat with the precipitation of acute coronary even in individuals with probably less severe unde lying coronary arteriosclerosis.

3. Moderate to severe blood levels of carb monoxide (26% and above) can induce acu or delayed toxic inflammation of the hea muscle (myocarditis) as carbon monoxide b comes bound to myoglobin in heart muscle t



sue or the surrounding membrane of the heart (pericarditis), and sometimes result in irregularities of the heart beat, any of which may take months to resolve.

Naturally, any time you want to calculate back to the persons's peak blood carbon monoxide levels, you need to count back in time from the minute the blood is drawn to the time the person was removed from exposure, and multiply times the fraction of half lives. Again, the half life of blood carbon monoxide on room air is about four hours, on nasal prong oxygen about 45 minutes, and on mask oxygen about 30 minutes. This means that if the person was directly transported in an ambulance on continuous mask oxygen from the point of exposure and if his blood level tests at 16%, that its peak 30 minutes earlier was approximately twice that or 32%.

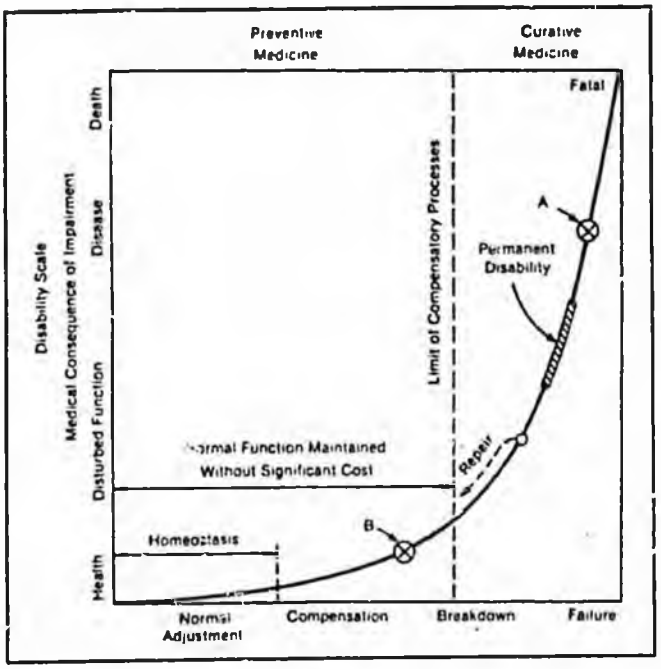
Other risk factors, of course, are associated, both job related and non-job related, with the development of coronary heart disease. These include the non-job related risk factors of cigarette smoking (self pollution with carbon monoxide), high blood pressure, high cholesterol, physical inactivity, diabetes, family history of heart disease and others shown by the Framingham reports of Drs. Kannel and Dawber and other epidemiologic studies.[12] Such a fire fighter oriented epidemiologic study of risk factors perhaps oriented through the fire fighters health insurance or pension statistics could provide helpful substantial information concerning all the work-related risk factors associated with coronary heart disease such as frequency of fire calls, levels of carbon monoxide exposure, use of self-contained breathing apparatus, and exposure to chemicals such as PVC products.

Consequently, a program to lower the coronary heart disease risk among fire fighters might include the following:

1. Intensive health screening at hiring designed to screen out those of highest risk to develop heart and lung diseases (the same principle as not putting asthmatics to work in a dusty coal mine).
2. Continuous and intensive education to discourage any form of smoking among fire fighters (as incompatible with the honor of the fire fighting profession as a member of Alcoholics Anonymous drinking alcohol).
3. Encourage the continuous use and further development of self-containing pure air breathing systems during smoke exposure (smartness should be associated with the proper use of breathing systems, instead of being a "smoker-eater").
4. Stimulate interest in improving physical fitness by jogging, or controlling high blood pressure by medications and treatment with regular follow-up and other methods to reduce heart and lung risk.
5. Active rehabilitation should help a fire fighter who develops a heart or lung problem which is treatable and salvageable to enable those to return to work who can and thereby minimize disability and pension costs.
6. Initiate an active prospective risk factor study, similar to that sponsored by the rubber workers union, through your health insurance and pension statistics, chaired by a blue ribbon committee of physician-advisors, to determine the specific excessive health risks of fire fighters including cancer, heart and lung diseases and thereby be able to plan to reduce them.

Therefore, to answer the question, "Is heart disease in fire fighters related to their work?" I reply with a qualified "yes." Since heart disease in fire fighters is not simply coronary heart disease, but often myocarditis or pericarditis, since each fire fighter's risk varies considerably in smoke exposure frequency and the use of protective breathing equipment, and since there are non-job related factors associated with heart disease such as cigarette smoking and high cholesterol, each case may have many contributing risks of different weight.

Acquiring more knowledge by such an ongoing risk factor study not only helps determine the cause of disease, but should result in a healthier fire fighting force. The cost-effect of a preventive approach to disability can be well demonstrated (Figure 8). Thereby monies which are now going to increasingly high health insurance and pension costs can be diverted to directly enhance wages and the fire fighter's standard of living.



General References

1. Sidor, R.: Peters, J.M.: Prevalence rates of chronic non-specific respiratory disease in fire fighters. *Am Review Resp Dis* 109: 76, 1974.
2. Sidor, R.; Peters, J.M.: Fire fighting and pulmonary function: an epidemiologic study. *Am Review Resp Dis* 109:83, 1974.
3. Mastromatteo, F.: Mortality in city firemen. *AMA Archives Industrial Health* 20:1, 1959.
4. Day, H.W.: Heart disease and its relation to fire service. *International Fire Fighter* 33:2, 1950.
5. Sammons, J.: Carbon monoxide—the Oklahoma study. Proceedings of the Third Symposium on Occupational Health and Hazards of the Fire Service. Feb. 24-27, 1975, IAFF, pp. 40-43.
6. Gordon, G.S.; Rogers, R.L.: Project Monoxide. A project report of the John P. Redmond Fund of the International Association of Fire Fighters, Washington, D.C., 1969.
7. Astrup, P.; Kjeedsen, K.; Wanstrup, J.: Enhancing influence of carbon monoxide on the development of atheromatosis in cholesterol-fed rabbits. *J Atherosclerosis Res* 7:343, 1967.
8. Thomsen, H.K.: Carbon monoxide induced arteriosclerosis in primates. *Atherosclerosis* 20:233, 1974.
9. Aronow, W.S.; Isbell, M.W.: Carbon monoxide effect on exercise-induced angina pectoris. *Ann Int Med* 79:392, 1973.
10. Cohen, S.I.; Deane, M.; Goldsmith, J.R.: Carbon monoxide and survival from myocardial infarction. *Arch Environmental Health* 19:510, 1969.
11. Ayres, S.M.; Gianelli, S., Jr.; Mueller, H.: Myocardial and systemic responses to carboxyhemoglobin. *Ann NY Acad Sci* 174:283, 1970.
12. Kannel, W.B.: Serum cholesterol, lipoproteins, and the risk of coronary heart disease: the Framingham Study. *Ann Int Med* 74:1, 1971.
13. Barnard, B.J.; Gardener, G.W.; Diaco, N.V.; Kattsu, A.A.: Near maximal ECG stress testing and coronary artery disease risk factor analysis in Los Angeles fire fighters. *J Occupational Med* 17:693. 1975.
14. Barnard, R.J.; Gardner, G.W.; Diaco, N.V.: "Ischemic" heart disease in firefighters with normal coronary arteries. *J Occupational Med* 18:818. 1976.
15. Radford, E.P.; Levine, M.S.: Occupational exposure to carbon monoxide in Baltimore fire fighters. *J Occupational Med* 18:628, 1976.
16. Howley, E.T.: The effect of different intensities of exercise on the excretion of epinephrine and norepinephrine. *Med Science* Sports 8:219, 1976.
17. Stewart, R.D.; Baretta, E.D.; Platte, L.R., et al: Carboxyhemoglobin levels in American blood donors. *J Amer Med Assoc* (JAMA) 229:1187, 1974.
18. Horvath, S.M.; Raven, R.B.; Dahms, T.E., et al: Maximal aerobic capacity at different levels of carboxyhemoglobin. *J Appl Physiol* 38:300, 1975.
19. Shafer, N.; Smilay, M.G.; MacMillan, F.P.: Primary myocardial disease in man resulting from acute carbon monoxide poisoning. *Am J Med* 316, 1965.
20. Anderson, R.F.; Allensworth, D.C.; DeGroot, W.J.: Myocardial toxicity from carbon monoxide poisoning. *Ann Int Med* 67:1172, 1967.
21. Scharf, S.M.; Thomas, M.D.; Sargent, R.K.: Transmural myocardial infarction after exposure to carbon monoxide in caronary artery disease. *New Engl J. Med* 291:85, 1974.

References related to research from Dr. Kurt (not listed in text)

1. Kurt, T.L.; Genton, E.; Chidsey, C.A., III; Beck, P.; Sussman, K.E.: Carbohydrate metabolism and acute myocardial infarction: Responses of glucose and insulin, catecholamine excretion, growth hormone, and cortisol secretion. *Chest* 64:21, 1973.
2. Kurt, T.L.; Peters, J.M.: Epidemiology of cardiac risks in fire fighters with Holter electrocardiograms and carbon monoxide sampling. *Circulation* 51:II-204, 1975.
3. Kurt, T.L.; Mogielnicki, R.P.; Chandler, J.M.: Cardiorespiratory complaints and ambient carbon monoxide. *CVD Epidemiology Newsletter* 22:22. 1977.
4. Kurt, T.L.; Weiser, P.C., Morrill, G.C.; Cropp G.J.A.: Effects of carbon monoxide on maximal aerobic working capacity at 1600 meters. *J Appl Physiol* (in press).

DR. HOWARD A. ANDERSEN

THE EFFECTS OF SMOKE AND TOXIC GASES ON THE RESPIRATORY TRACT

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My first encounter with burns of any magnitude was in World War II when I was aboard a destroyer that was hit and was sunk by enemy action on D-Day at 6:30 a.m., the first day of the Allied landings at Normandy. We were hit amid-ship in the fire and engine rooms. Boilers exploded releasing hot steam and fuel tanks burst releasing oil that burned. Over half of our crew were burned, injured, dead or missing and I was a busy physician for the next two days aboard a rescue ship.

My next experience was a very personal one on Okinawa. I was attached to the Sixth Marine Division and was assigned to the latrine detail. Neither my corpsmen nor I had had experience with this, but according to the Navy Manual, a very acceptable way of cleaning latrines was by daily burning using diesel oil. After liberal splashing of presumed diesel oil in a 2-holer my first day, I dropped some burning paper in and inquisitively peered into the hole. Whoosh! It exploded in my face giving me an even higher forehead than I now have. This was not as bad, however, as a few weeks later when I had 12 men who were burned. Immediately after breakfast when a 12-holer was fully occupied, someone dropped a lighted cigarette into the latrine! It still hurts to think of it. At least it was only my face that was burned!

The respiratory tract from the nose to the pharynx, larynx, trachea, bronchi, and alveoli or air sacs is vulnerable to injury from inhaled smoke which may contain irritants and asphyxiants. Any or all of these respiratory structures can be injured. Smoke is not, of course, a single substance, but may be composed of hot steam and many irritant and toxic gases such as ammonia, chlorine, hydrochloric acid, nitrogen dioxide, phosgene, sulfur dioxide, and possibly many others. One must also remember that when anything burns, carbon monoxide is produced. Though this gas is not a local irritant it may be lethal. The amount of harm resulting from inhalation of these gases depends on several factors but particularly on the concentration of the toxic substance and the duration of exposure.

Tracheobronchial Effects

The respiratory tract from the nose to the larynx downward to the trachea, bronchi, and respiratory bronchioles (very small airways) is lined by ciliated epithelium and goblet cells attached to a basement membrane. This mucosa is composed of only one cellular layer in thickness. The cilia are tiny hairs which have a rhythmic beat in unison waving mucus and other material toward the larynx. Beneath the basement membrane is the submucosa with a network of connective tissue, bronchial vessels, nerve fibers, elastic tissue, and secretory glands. Muscle and cartilage comprise the other tissues in the trachea and bronchi. Goblet cells

in the mucous membrane and secretory glands in the submucosa both secrete mucus which keeps the membrane moist.

Smoke with irritant fumes and gases causes an acute tracheobronchitis with red and inflamed mucosa or lining which may swell and thicken. This causes hoarseness, cough and wheezing. In some instances the entire mucous membrane down to the cartilage may be damaged. The cilia are particularly susceptible to irritants and are the first structures to be affected. When healthy they beat approximately 25 times per second waving as a field of wheat waves in a breeze and washing secretions or particles toward the larynx at approximately the speed of 1.6 mm. per minute. Their action may be stopped for approximately 20 minutes by inhalation of smoke from one cigarette. With heavy cigarette smoking cilia may be destroyed permanently. The same sort of effect is one of the first results of inhalation of smoke from other types of fires.

When damage goes deeper ciliary and goblet cells may be destroyed and there is infiltration of the submucosa or connective tissue of the lining of the trachea and bronchi with inflammatory cells and fluid causing edema or swelling of the membrane. This may cause cough, shortness of breath, and wheezing because the air ways are narrowed. These symptoms occur at different times after injury probably depending on the dosage and the substance involved. When hot steam is inhaled, having 4000 times the heat carrying capacity of air, these symptoms may occur as early as 30 minutes later. In the Coconut Grove fire in Boston doctors noted they occurred about 3 hours after the burn, approximately the same amount of time that the swelling occurred in the external burns. Usually, however, it takes one or two days for these symptoms to appear.

Additional obstruction may occur with spasm of the bronchi which results from a reflex mechanism transmitted via nerve endings from the mucosa to the muscles surrounding the bronchi. This may aggravate the wheezing, especially on exhalation, similar to a person with asthma.

Pulmonary Effects

As bronchi extend further into the lung, they continue to branch and become smaller and smaller. Terminal airways are 0.5 mm. (1/50 of an inch) or less in diameter. It does not, therefore, require much swelling of the mucosa to produce obstruction predisposing to collapse and airlessness of the lung (atelectasis) beyond the obstruction. "Micro-atelectasis" is a term that has been applied to this phenomenon when small bronchi and lung segments are involved. When airways of larger size are blocked by this swelling, bacteria may invade the lung beyond the effected bronchus causing pneumonia.

With continued branching the small airways become the respiratory bronchioles. The alveolar duct and the alveoli or air sacs branch off these. Within the alveoli are alveolar macrophages which normally provide one of the defense mechanisms of the lung against infection. They consume and inactivate bacteria and help avoid infection. It has been shown that after smoke inhalation alveolar macrophages have a reduced capacity for bacterial inactivation and that the bacteria within the macrophages are capable of multiplying. This phenomenon also predisposes to pneumonia.

Alveoli also may be the source of pulmonary edema or extravasation of fluid into them from actual damage to the alveolar lining cells or injury to the pulmonary capillaries. Reflex neurogenic factors affecting the tone of the pulmonary vessels have also been implicated as a cause of this transudation of fluid across the alveolocapillary membrane into the alveolus. Accumulation of fluid may be so extensive as to cause marked respiratory embarrassment and even death.

When atelectasis, pneumonia or pulmonary edema occur, gas transport is disturbed so that oxygen is not absorbed properly and hypoxia is likely to result.

Detection of Respiratory Effects

One should suspect the presence of damage to the respiratory tract, even after minor burns, when there is a history of prolonged smoke inhalation in an enclosed area, when there are burns of the face, head, neck, or membranes of the nose and mouth, and when there are symptoms of cough, hoarseness, shortness of breath and wheezing.

One can detect the presence of tracheobronchial injuries by bronchoscopic examination. It is a simple procedure to slip a flexible bronchoscope into the trachea and bronchi and visibly assess the amount of damage. Lung scans using

Xenon 133 consist of injection of this radioactive substance into a vein of the arm or leg from which it is carried directly to the lungs. When the circulation through the vessels of the lungs is normal and when there is a normal amount of oxygen and other gas exchange in the lungs, the gas traverses the membrane between the alveolar air and the blood very readily. When alveoli are blocked off, however, or when the membrane is thickened, diffusion of the gas across the membrane is slowed. A normal scan demonstrates complete and equal clearing of Xenon 133 within 90 seconds. In inhalation injury there may be delayed clearing. In one series of 50 patients, 15 had delayed clearing and the mortality rate in these patients was considerably increased.

Examination of the lungs by x-ray may also be helpful and should be done. In the first day or two, however, it may be disappointingly normal even when there is moderately severe damage to the bronchi or to the lungs. Measurement of pulmonary volumes such as vital capacity, total capacity, residual volume, maximum breathing capacity, and forced vital capacity is seldom helpful, however.

Measurement of arterial blood gases (oxygen and carbon dioxide) may be helpful, but one should not be lulled into a sense of false security if they are normal. Determination for carboxyhemoglobin in the blood should be performed to evaluate the amount of carbon monoxide absorbed.

In every study that has been done, the presence of an inhalation injury to the tracheobronchial tree or the lungs increases markedly the mortality rate. It is important, therefore, to remember to evaluate the respiratory tract in a burned person. The possibility of survival is definitely influenced by involvement of the tracheobronchial tree and the lungs, particularly if such involvement is ignored. It is particularly important that fire fighters do everything possible to protect their lungs whenever they enter smoke and whenever there is a possibility of inhalation of toxic gases.

DR. VICTOR H. ESCH

TOXICITY IN THE FIRE FIGHTER'S ENVIRONMENT

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I wish to express my sincere thanks and appreciation to the trustees of the Redmond Fund, Dr. Atwood and the International Association of Fire Fighters for the opportunity to participate in the Fourth Symposium on the Health and Hazards of the Fire Service.

During the past 10 years there has been an explosive proliferation both in the manufacture and use of plastic and synthetic materials. Polyvinyl chloride production alone exceeded 44 billion pounds last year.

These products now flooding the marketplace are finding increased use in the manufacturing of household furniture, plastic pipe, buildings,

wall coverings, automobiles, buses, subway cars, aircraft and the covering of telephone and electrical conduit.

Accordingly there has been an increasing concern about the toxicity of the fumes and smoke produced by the pyrolysis or combustion of these materials . . . unfortunately, the toxicological aspect has lagged so far behind the knowledge of the physical properties (such as flame spread, melting point, etc.) that the toxic hazards cannot be described adequately except in generalities.

Environmental hazards, not long ago synonymous with DDT and "silent springs" are casting longer, darker shadows over the health of all people. An incredible series of disasters implementing new chemicals in the market place are sending growing numbers of patients exposed to these agents to physicians who are often unaware of either the magnitude or the precise nature of the chemical hazard.

Today there are almost too many potential hazards around for individual physicians to become familiar with: polycyclic aromatic hydrocarbons, inorganic micorparticles, metals, halogenated ethers, aromatic amines and biphenyls, to name a few.

TRIS, KEPONE, VINYL CHLORIDE, BENZENE, PBB's and PCB's have now become household names due to recent disclosures by the American press.

The proliferation and sophistication of petrochemical products available to the building industry today far surpasses the ability of the scientific community to assess critically all of their toxic properties in a fire situation.

As experienced fire surgeons, we were shocked some six years ago by the untimely death of a 33 year old firefighter who died of acute pulmonary edema 24 hours after inhaling the fumes from a burning PVC-coated wire. Repeated inquiries at that time to the petrochemical and plastics industries as to the harmful effects of their products on humans were—in a word—unproductive. Few epidemiologic studies are mounted unless there is a suspicion, a guess or a hint that leads to these studies. Following a series of fires involving electrical insulation, our suspicions were confirmed and a five-year investigative study was launched. Our paper, published by the Journal of the American Medical Association was the result of that five-year study. We found that firefighters exposed to the fumes from burning or thermally degrading polyvinyl chloride rapidly became incapacitated secondary to the development of chest pain, severe headaches, shortness of breath and in many instances became comatose or disoriented. To date over 200 firefighters have required treatment at the hospital following exposure to these fumes. Dr. Dyer has reported a number of cardiac arrythmias developing after various periods of exposure.

In a fire situation PVC will soften and decompose on heating and produce hydrogen chloride, benzene and other decomposition products. Phosgene has also been listed by some investigators.

Hydrogen chloride vapors are more dehydrating and corrosive than aqueous hydrochloric acid and vapors. Hydrochloric acid mist will cause throat irritations when the concentration reaches 35 ppm. Further, concentrations above this level causes anosmia (loss of sense of smell) due to severe irritaion of the olefactory or first cranial nerve. The firefighter is usually unaware of this sensory loss and is unable to detect the presence of the corrosive gas until considerable quantities have been inhaled and pulmonary symptoms begin to develop.

We feel that concentrations over 50 ppm are not tolerable for any length of time. At levels well below 1,500 ppm hydrochloric acid fumes can be fatal in a few minutes. The physiological effect of the corrosive action of hydrogen chloride inhalation is often delayed and in our experience may develop as late as 12 to 24 hours after exposure. Benzene inhalation will also result in either acute or chronic toxic symptomatology depending upon the concentration of the vapor and the length of exposure. Acute poisoning will develop following inhalation of vapors in the 3,000 ppm range after exposure to the fumes for several minutes.

Electricians are particularly vunerable to death or severe injury if trapped in a confined area when a PVC electrical fire develops. We have observed a number of electricians either killed or injured in this manner since the onset of our investigation. Our findings have been confirmed by numerous other investigators and other PVC fatalities have since been recorded. There is now mounting evidence that perma-

nent lung damage will result from exposure to the corrosive fumes.

Recent studies at Yale University School of Medicine have confirmed an earlier impression (JAMA Medical News 234:1211 (Dec. 22, 1975) that pulmonary damage due to inhalation of toxic combustion products (corrosive gases in particular) is often not detected by standard X-ray studies. Ventilation perfusion scans utilized by the Yale group demonstrated airway or parenchymal lung damage that was undetected by the previous chest x-rays.

Since thermal degradation of some polyvinyl chloride formulation can result in at least 75 different chemical compounds—it is quite likely, in my opinion, that repeated exposures over a period of years could result in the development of lung cancer in the firefighter—only time will tell. Meanwhile, any exposure to this material should be avoided.

Industry, to date, has a bad track record for truthful disclosure. The Plastic Pipe Institute report 5474 lauds the physical properties of PVC plumbing assemblies during actual fire endurance tests—there is no mention of the toxic properties when the material is thermally degraded.

The National Electrical Manufactures Association (NEMA) and the Society of Plastics Industry recently published a booklet—"Characteristics of Polyvinyl Chloride Conduct, Insulated Wire and Piping in Fire Situation." This report especially prepared for code groups, building inspectors, fire marshals and other officials states that "There are no documented cases of PVC conduit, insulated wire, or piping contributing unusual life hazards to a fire problem." Further, it states that the combustion products from plastics are "different"--but no more toxic than those from other organic material—(cotton, newspaper, douglas fir). We strongly disagree.

Finally on the last page is a thermometer showing a "flash ignition temperature" for PVC to be 735° F (well above the other organic materials listed). This is misleading since it is well known that thermal decomposition of PVC occurs at approximately 400° F or less with the quantitative release of HCL long before ignition occurs.

The 13th edition of the N.F.P.A. Handbook states that the "Toxicity of combustion and thermal decomposition products, *possibly because of long chemical names* of some plastics, have been cause for concern among fire fighters." This organization and the Society of Plastics Industry has recently released a "slide show" relating to plastics in fire situations.

Incredibly, the Society of Plastics Industry recently reported in a newsletter that they had not seen a single substantiated report of death, proven by autopsy related to PVC and HCL. Among other recent reports we would refer the ASPI to the February 1976, Fire Protection that lists 3 deaths from acute pulmonary edema from the inhalation of HCL. (proven by autopsy.).

Dr. Russell P. Sherwin, a University of California pathologist, stated that chronic low level exposure to smoke, chemicals, and other pollutants, can result in premature death. He calls the lung cells the "endangered species" and states that even low levels of pollutants can destroy lung tissue.

With the arrival of the Toxic Substances Control Act (TOSCA) there will be new emphasis on many of these problems and a new era of inter-agency cooperation within the Federal Government can be anticipated.

The engineering community is to be commended on the many technological advances in the art of combustion analysis and the physical properties of pyrolysis. In so doing, however, the technology of this phase of the problem has far out-paced our medical understanding of the patho-physiological relationship of humans actually exposed to fires and exotic toxic gases. At this time it is critical that the emphasis be re-directed to the biomedical aspects that will tell us what actually occurs with human exposure to these toxic combustion products.

Ralph Nader has stated that the FAA has dilly-dallied for years and still has not issued any standards in these crucial hazard areas. Synthetic materials used in aircraft seat cushions, carpets and the walls of aircraft interiors emit a deadly combination of cyanide and carbon monoxide fumes when subjected to intense heat or burning. These gases are so toxic that passengers are cut down in seconds before they can reach an emergency exit or other opening. A Boeing 707-300 series aircraft made a successful emergency landing near Paris in 1973 after a fire started near the rear lavoratory in a plastic waste basket. The in-flight cabin interior fire did not involve the aircraft's fuel but was fed by the interior's material. One-hundred

and twenty four people died from inhaling the toxic fumes and smoke from burning materials. During the same year 144 French teenagers were trapped, trampled, and asphyxiated when plastic synthetic decorations caught fire in a night club.

The danger from fire exposed electrical wiring systems on escape potential from buildings is now widely recognized. A Factory Mutual Research Report (September, 1976) identified HCL as the major toxic compound in the pyrolysis and combustion of PVC. Laboratory tests showed that the rates of production of HCL are more than twice as great for PVC conduit and non-metallic sheathed cable as for wiring systems where steel raceways are used.

The analysis of data indicated that the PVC conduit exposed to fire could adversely affect the human escape potential from buildings. Based on the same analysis, steel raceways would NOT adversely affect escape potential. Data also indicated that smoke levels exceeded acceptable limits for a PVC conduit wiring system than for other systems tested.

We ask a question at this point as to why the fire community allows this problem to exist in the electrical building codes?

Recently the District of Columbia Fire Marshall learned that 1000 polyethylene mattresses had been purchased for the D.C. jail. He ordered them removed thus eliminating a definite hazard. Since these mattresses are commonly used throughout the United States, and since smoking materials are readily available to prisoners and patients, it is inevitable that sooner or later a tragedy will occur in a jail, prison, or hospital.

Plastic and synthetic materials should not be used in high rise buildings, night clubs or other places of public assembly because of the "flash over" potential and the rapid build-up of toxic gases in a fire situation. Again, it would appear that continued indiscriminate use of those materials in these situations will result ultimately in a tragic loss of life.

The building industry is primarily interested in keeping the costs of construction down and providing adequate equipment to reduce fire losses. Life safety is relegated to a poor third for the sake of a profit. Thus, the building industry has encouraged the use of plastic pipe to avoid the plumber's wages since the pipe can be assembled on the job by unskilled laborers. Similarly, the use of PVC covered cable is cheaper than the safer metallic sheathed cabl There is a growing public awareness that tl existing regulatory agencies are not using t limited power that they have, while industry practicing "environmental blackmail" tactic threatening that stronger regulations woul exacerbate unemployment in some industrie Douglas Castle, Administrator of the Envi ronmental Protection Agency, stated that "We're a chemically oriented society . . . w know very little about those chemicals, an what they do, and about their passage throug the environment, and what risks might be asso ciated with them." He concluded that "the sin gle most demanding problem ahead of us— getting a handle on the use of toxics and making judgments about what is acceptable".

It is time for the fire community to legislat tougher safety building codes for fire protec tion and life and not back away from industria opposition.

Washington area fire chiefs have been suc cessful in forcing the Metro system to modify the use of flammable plastic and synthetic mate rials in buses and subway cars and are attempting to limit the indiscriminate use of PVC pipe & cable in buildings.

A fire in an electrical control panel is apparently one of those disorders to which technology invariably is prey. While many of the PVC fires in the District of Columbia involved large electrical installations, our experience indicates that sometimes limited quantities of PVC can also be deadly. For example; electrical wiring insulation, telephone cable, vinyl wall covering, vinyl ceiling covering, and vinyl covered furniture. Special caution should be used in fires involving a heavy plastic fire loads such as drug stores, department stores, record shops, etc. In all cases, self contained breathing apparatus must be used, both during the extinguishment and overhaul phase. It is most difficult to determine when the atmosphere is safe enough to remove the mask. Repeated testing has demonstrated no pattern of consistency in the gas distribution. For example, there might be a high concentration in one part of a room while a "safe" concentration may be recorded some 10 feet away.

We are in the process of developing an acid detector badge to be worn on the turnout coat. The badges are being improved as to criticality and further evaluation under actual fire conditions will be forthcoming.

Currently the suspected PVC fire atmospheres are tested at intervals for HCL using the Drager detector. Any reading over 50 ppm should be considered dangerous and every effort to remove the gas by ventilating fans should be employed.

The use of a water spray is somewhat unreliable and should not preclude the use of fans. Every fire officer should preplan his response district and be aware of any PVC fire potential and/or heavy PVC fire load.

Suspected plastic materials should be collected for analysis and future evidence should later pulmonary problems develop. Likewise, photographic evidence may prove to be valuable at a later date.

Despite consistent and persistent assurances from the plastics and petrochemical industries indicting carbon monoxide as the major cause of firefighter mortality and morbidity in plastics fires, recent studies by numerous investigators have refuted this claim. The evidence clearly shows that irritant "off gases" are responsible for lung and pulmonary tree damage.

It should again be noted that carbon monoxide is orderless, colorless, and tasteless and has *no* irritating effect on the air passages or lungs. While carbon monoxide does kill, it is an oversimplification of the problem to conclude routinely that a death occurs from "carbon monoxide poisoning" or "smoke inhalation." The terms "smoke inhalation" and "carbon monoxide poisoning" should be abandoned by the fire community in most instances. We would suggest the use of the term "toxic gas inhalation" which would tend to focus attention on the toxic agent.

Some investigators have attempted to correlate a high level carbon monoxide in the blood with the degree of pulmonary injury, i.e., a high COhg level would indicate a serious insult to the lungs and pulmonary tree. This may be true in "ordinary" house fires, but this is not a valid assumption when evaluating exposure to plastic fumes. We have noted that in many of the most serious injuries, carboxyhemoglobin levels may range anywhere from 15% to the low or mid 20's. In almost all cases we observe the rapid development of a metabolic acidosis following exposure to PVC fumes.

While many large cities and communities are served by expert medical examiners who are trained forensic pathologists, much of the country still labors under the outdated coroner system. In many instances, the "Coroner" is not a physician—rather he is a political appointee, local businessman or undertaker.

In the excitement and confusion following major fires the victims are rapidly removed from the fire scene, embalmed and buried with the blessing of the local coroner as "smoke inhalation." In most instances, to the delight of the insurance adjustors, local officials and building owners, the deaths are attributed to "smoke inhalation" and no serious effort is made to determine the nature of the toxic chemical compound causing death and/or whether codes were violated by the use of certain building materials or furnishings.

This practice should be condemned and the fire community should seek the establishment of a medical examiner system by legislation and continue to insist on better building codes, emphasizing life safety. Every fire death should be investigated by autopsy and microscopic examination of lung tissue and other organs. In addition, blood samples in specially prepared containers should be submitted for analysis to regional centers that can perform sophisticated analytical chemical studies.

All exposed firefighters should seek immediate medical assistance, including chest x-rays, prior to the development of any serious symptomatology to establish a baseline for serial X-Ray follow-up.

In conclusion, it was felt that corrections to old premises were in order and that our experiences should be recorded and shared candidly with the medical and firefighting profession. Industry should place more emphasis on life safety. The inherent dangers posed by the unrestricted use (for example) of PVC insulated wire and cable in ceiling plenums used for air handling should be controlled by law. The solution is to hold technology in abeyance until building codes can be revised and test protocols can be developed to screen materials that produce highly toxic products of combustion. The fire community should exert every effort to see that this is done.

Test on fire-fighters shows 'anxiety' increases heart rate; may lead to damage

The fact that heart attacks have been killers of fire-fighters for a number of years is not new. But, it has been until only recently that major steps have been made to determine the nature of the problem – and how the life of the fire-fighter might be extended through careful exercise, good diet habits and a change in work patterns.

A University of C[illegible]ornia at Los Angeles professor, R. James Barnard, Ph.D., has undertaken a number of studies involving fire-fighters. His most recent involved the actual monitoring of selected fire-fighters while on duty for their full 24-hour shift, and recording of changes in heart rhythm during every phase of their activity.

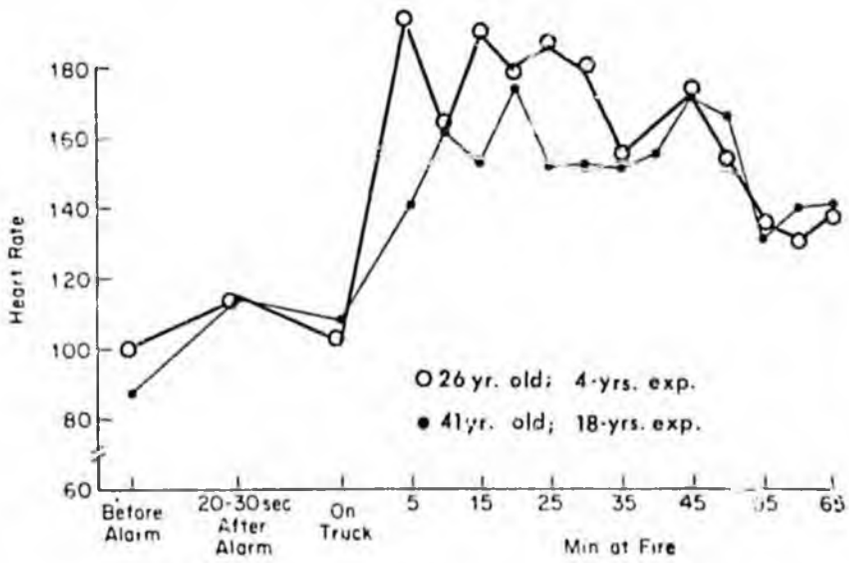
The subjects were 35 men from Los Angeles City and County fire departments. They ranged in age from 23 to 42 and were all considered to be in good health without any overt symptoms of heart disease. Fire-fighting experience ranged from one-half year to 19 years.

Prior to going on duty each shift, the men were connected to small Avionics Model 375 Mini Recorders. Electrodes were placed to provide a continuous ECG recording. The men kept a log book which included the exact time the recorder was started, time of all alarms and a description of their activities. The tapes were changed every four hours. Most of the men went to bed between 2300 and 2400 hours, with a new tape in the recorder.

TAPES ANALYZED

Tapes were transcribed to paper printouts and analyzed. The ECG was read before the alarm sounded, for 30 seconds after the alarm, and then every minute until the men returned to the station. To be included in the analysis, a man had to have responded to at least four alarms.

Data was obtained from 35 fire-fighters responding to a total of 189 alarms. Because of the wide range of heart rates recorded immediately before the alarm sounded, the data was expressed in beats per minute increase (beats/min) and was measured from 6 second rhythm strips.



Heart rate responses of two fire-fighters while involved in structural fire response

Fifteen to 30 seconds after the alarm, heart rate showed a mean increase of 47 beats/min. Range was 12 to 117 beats/min.

About one minute after the alarm, while on the truck, heart rate values were still 30 beats/min (range 1 to 80) above that before the alarm sounded.

While responding, no consistent increase in heart rate was observed for any one individual. In particular, the responses obtained from one man while sleeping showed marked variation. After two alarms, his heart rate increased approximately 50 beats/min but while in response to a third alarm his heart rate increased 104 beats/min.

PUTTING ON TURN-OUTS

Seven men were tested in the laboratory to find out what effect the physical activity involved with getting onto the fire truck ready for response would have. Moving a similar distance to that required in a fire station and then putting on a coat increased heart rate an average 36 beats/min (range 10 to 49). One minute after the start of this activity, the heart rate was increased only 9 beats/min.

Additional data was obtained from five men while fighting major structure fires. Two men, arriving at the fire scene in approximately 3.5 minutes after the alarm sounded, had heart rates of 150 beats/min. Another was recorded after having finished at one fire and then responding directly to anoth[illegible] upon receipt of a radio alarm. While on the truck going to the second fire he had a heart rate of 158 beats/min. For over 90 minutes in these two consecutive fires his heart rate was maintained above 160 beats/min. This included a 15 minute period of extremely high heart rate (average 188), during the initial stages of the second alarm when the roof on which he was working became surrounded by flames and there was danger of collapse.

HIGH ANXIETY SHOWN

The data obtained shows that for the most

Table 1. — Heart Rate and ECG Responses of a Less Experienced Fireman.*

Time	Before Alarm (beats/min)	15-30 sec After Alarm	On the Fire Truck
08:50	80	135†	140
09:45	80	120†	100
12:33	89	148†	128
13:46	120	155†	140
20:26	101	142	130

* Subject A, 24 yr old, fireman 1 yr

Table 2. — Heart Rate and ECG Responses of a More Experienced Fireman.*

Time	Before Alarm (beats/min)	15-30 sec After Alarm	On the Fire Truck
13:40	82	119†	106
15:11	73	142†	138
16:07	66	120†	126
17:41	81	132†	128
19:05	98	118	106
20:26	79	118	124
20:41	95	139	128

* Subject B, 43 yr old, fireman 15 yrs

Table 3. — Heart Rate and ECG Responses of a Fire Fighter While Sleeping.*

Time	Before Alarm (beats/min)	15-30 sec After Alarm	On the Fire Truck
23:30	52	100	86
02:24	45	149†	67
03:10	48	97	85

* Subject C, 36 yr old, fireman 11 yrs

part they were relatively inactive but at times are called upon to perform at or near their maximal heart rate for prolonged periods. The rate responses observed immediately after the alarm sounded as well as on the truck approaching a fire often indicated a state of high anxiety.

Why a man's heart rate might increase by only 15 to 20 beats/min in response to one alarm and then 70 to 80 in another could not be determined. The fire-fighters themselves recognized these different heart responses but could not explain them. Some men indicated they did not think they experienced anxiety when the alarm sounded, but their heart rate indicated otherwise. The data also showed that the rate of increase was about the same for the less experienced fire-fighter as the experienced men.

The method of receiving the alarm at the station also was analyzed.

One of the implications of the study was to show that mere receipt of the alarm, the ringing of a bell or sounding of a tone, produced anxiety and an increase in the heart rhythm. He concluded that unnecessary alarms should be eliminated in cities which transmit to all stations, not just that called for on the response. He called for further study into the health and safety aspects of fire-fighting.

Repeated exposure to states of high anxiety as well as inhaling pollutants such as carbon monoxide may be related to the high incidence of ischemic-stress tests previously observed in fire-fighters. He concluded, however, that his findings suggest that ischemic heart disease in fire-fighters may be job associated.

(Information compiled from a report in "Journal of Occupational Medicine/ Vol. 17, No. 4/April 1975," by R. James Barnard, Ph.D., and Henry W. Duncan, B.S.)

Muster 'finals' in Benecia on September 6-7

The first annual California Firemans Muster Assn. championships will be held in Benecia on September 6 and 7, hosted by the Benecia Volunteer Fire Dept.

Saturday's events start at 11 a.m. with gas pumping engines and hose wagons. Crowning of the fire queen will take place at 1:30 p.m. The bucket brigade finalists from other musters held this summer will complete for the state championship starting at 2 p.m. A dance will start at 9 p.m.

On Sunday, a parade will begin at 10 a.m., followed by hand pumping contests at noon, and hosecart races at 2 p.m.

Only fire equipment 1940 or prior is eligible to enter competition. All fire-fighters should register at the Benecia firehouse, 150 Military West, on arrival to obtain information on camping areas and guarded storage facilities.

CAFAA questions code change on door closers

The California State Fire Marshal has adopted the 1973 Uniform Building Code, but has proposed deleting the requirement for closing doors in fire rated corridors.

The California Automatic Fire Alarm Assn., has issued a bulletin calling this to attention. The bulletin stated: "Although integrity of fire rated walls is not within the scope of our Association's activities, we are calling this matter to your attention because codes should be progressive documents and this may be a step backwards."

The bulletin suggested interested parties "with convictions on Section 3304(h)" contact Fire Marshal Albert E. Hole in Sacramento.

'Half of fire-fighter's breathing apparatus fails in laboratory testing'

About half the respiratory devices worn by fire-fighters fail to work adequately when tested under conditions found in actual fires, according to a researcher from the Lawrence Livermore Laboratory (LLL).

Bruce Held, a respiratory protection specialist at the laboratory, presented his findings at the Third Annual Fire Protection Technology Seminar at Stanford Research Institute in Menlo Park, Calif.

Held said that breathing protection equipment used by the nation's fire departments often fails the most basic stress tests. In an 18-month LLL research program, Held tested the breathing devices for resistance to heat, cold and temperature shock often found in real-life fire-fighting situations.

Testing the devices in a special chamber which can simulate fire conditions, Held found that in two out of six breathing units, the facepiece lens popped out at 200 degrees F. Fire-fighters typically work in temperatures ranging from 200 to around 500 degrees F.

In another type of breathing protection device, Held found that at temperatures as low as 125 degrees, two out of four models failed. The facemask became loose, allowing outside air to flow in. In a fire-fighting situation, this would allow the fire-fighter to breathe smoke and other toxic gases.

STRAPS MELTED

In tests measuring the effect of radiant heat or the heat reflected from hot surfaces, Held found that in many models, the straps that hold the breathing devices on the fire-fighter's backs melt and break. In one model, the straps melted when exposed to the amount of heat reflected from a fire in a metal wastebasket.

"When the harness that holds the breathing device breaks," said Held, "the weight of the air tank pulls the face mask off the fire-fighter, allowing the heated air of the fire to sear his lungs. The real danger arises when a fire-fighter is trapped by a 'Flare-up' of the fire. In that case there is a good probability that the harness straps would melt in all models we tested."

Because fire-fighters commonly store their breathing protection devices on fire trucks where, in many areas of the country, they are exposed to severe winter weather, Held tested the devices for cold resistance.

He stored eight devices at −25 degrees F. for 24 hours. In one brand, the facepiece shattered, the valves froze, and the air leaked out of the cylinder. In another brand, the tubes connecting the air supply to the facemask became stiff and brittle.

In three other brands the air bottle leaked and the breathing tubes became rigid and difficult to use.

"Fire departments buy this protective equipment believing it will work under any conditions," Held said. "When the fire-fighters get to the scene of a fire and find their equipment doesn't work properly they may have to go without them.

"Fire-fighters have more job-related injuries and deaths than any other profession in the country. Many

The person found
as Fred Gonzales,
investigation.

of those injuries could be prevented if firemen were using dependable respirators. Since we have already protected a man walking on the moon, we could certainly develop adequate protection for fire-fighters."

Helds findings will be submitted to the National Institute for Occupational Safety and Health (NIOSH), the agency that certifies respiratory equipment as safe for use. Working with Held in his research are Gail Cardenas and Charles Harder.

"Historically, it was the Bureau of Mines that certified respiratory equipment," Held said. "Now that the responsibility lies with NIOSH, certification requirements should be upgraded to assure that respirators are safe to use during fires."

Once respirators are upgraded, the attitude of fire-fighters toward their own safety will still have to inprove, Held said.

NO MORE 'SMOKE EATERS'

"Among fire-fighters, it has always been considered manly to 'eat smoke' and go without breathing protection," Held said. "That may have been true in the days when fires burned mostly wood and paper. Now, with the development of synthetics, the situation is much more dangerous. Firemen have died from the gases produced by burning bean-bag chairs and plastic curtains.

"Industrial fires are likely to create phosgene and chlorine gas, deadly substances that have been used in chemical warfare. It is imperative that fire-fighters wear their respirators much more often than they do."

In future studies, the LLL researchers will test fire-fighters' respiratory equipment for its resistance to moisture and corrosion from combustion products. Their work is part of several fire safety and protection studies conducted at LLL for the Department of Energy.

The Lawrence Livermore Laboratory is operated by the University of California for the Department of Energy.

Funds for arson projects offered by factory mutual

A special fund of $100,000 to assist groups wishing to conduct programs to combat the growing arson threat has been established by the Factory Mutual System: Allendale Insurance, Arkwright-Boston Insurance, Philadelphia Manufacturer's Insurance and Protection Mutual Insurance. This group is the nation's largest insurer of industrial properties.

Harry Merrow, chairman of the Factory Mutual Committee to Combat Arson, has announced that the first grant from the fund has been awarded to The International Association of Arson Investigators, Inc.

In announcing the grant Merrow commented:

"The fund is intended to assist such groups as state advisory committees on arson prevention, associations of arson investigators or private industry groups formed for the specific purpose of fighting arson.

"Because of this growing threat to lives and property, more programs of education and training to combat it are essential," he said. "Factory Mutual's assistance plan is a step in that direction and its progress and results will be carefully monitored."

Organizations wishing to apply for grants should write to: Harry Merrow, Factory Mutual System, 1151 Boston-Providence Turnpike, Norwood, MA 02062.

Applications should indicate the scope of the organization activities and reasons for requesting funds.

The Case for Pressure-Demand Self-Contained Breathing Apparatu

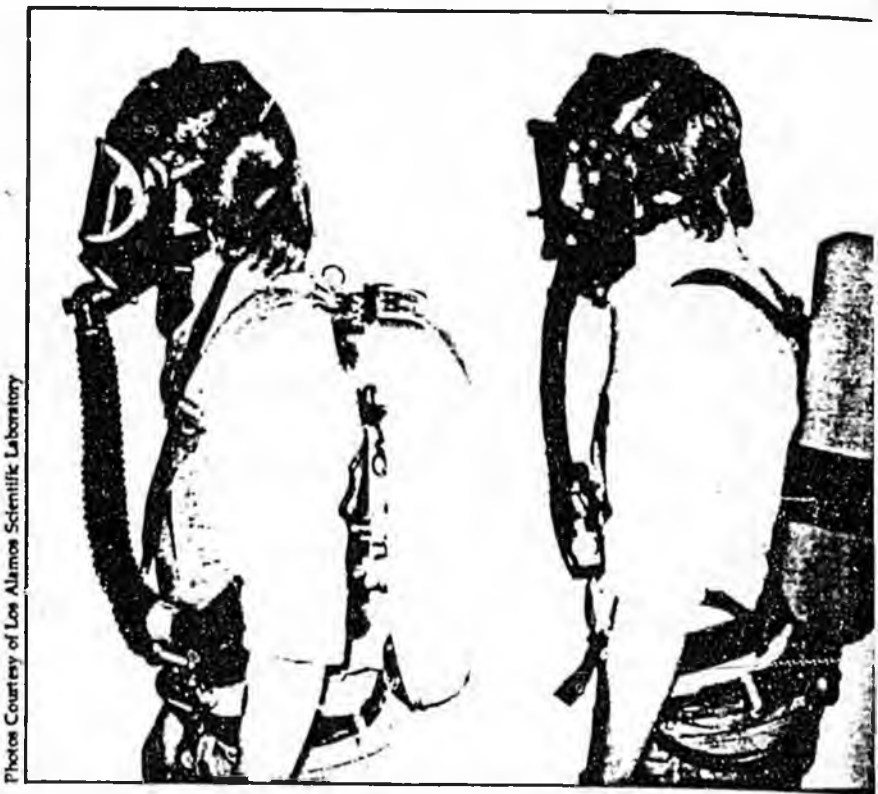
by
Thomas O. Davis and Bruce J. Held

This article deals with the controversy occurring within the fire service over pressure-demand (also referred to as safety pressure) versus demand self-contained breathing apparatus (SCBA) and explains why the authors believe that only pressure-demand devices should be used by the fire service. It is an appropriate place to start, as many fire service personnel are confused about what each type is and what issues are.

The fire service has one of the highest inhalation injury and death rates of any occupation in the United States.[1] In addition to the direct effect of the inhalation of toxic decomposition products involved in a fire, there is also the very real, but unmeasureable, factor that the inhalation of decomposition products plays in the high heart disease rate among fire fighters. Obviously, no one factor can be singled out as the major cause of the high heart disease and inhalation injury and death statistics. It is a complex problem that must be analyzed piece by piece so deficiencies can be corrected.

Future articles will concern problems found with SCBAs and their effects on the fire fighters who use them. However, in the context of currently available apparatus, it is necessary for the fire service to understand the problem areas. Then, not only can an intelligent selection of equipment be made, but training programs can be developed to work around the equipment deficiencies. The first major decision regarding the purchase of SCBAs is whether to buy pressure-demand or demand apparatus. To make this decision, the fire chief making the selection must know the operations, advantages and limitations of each type of SCBA.

This work was sponsored by the Division of Operational and Environmental Safety, Department of Energy.



Photos Courtesy of Los Alamos Scientific Laboratory

Fig. 1 A typical closed-circuit SCBA

Fig. 2 A typical open-circuit SCBA

Operational Characteristics

First, we must define the terms we will use. SCBA refers to both closed-circuit (Fig. 1) and open-circuit (Fig. 2) devices. In closed circuit devices the exhaled air from the facepiece is directed to the backpack (or front mounted pack), where carbon dioxide and water vapor are removed and oxygen added. Then the air returns to the facepiece. The airflow path is a complete loop, or a closed circuit, hence the classification of this type of device as a closed-circuit SCBA. By contrast, in open circuit devices, the airflow path is from a compressed air tank to the facepiece and then exhaled to the surrounding atmosphere. Therefore these units are referred to as open-circuit devices. The recommended type, and the most widely-used, for fire fighting is the open-circuit SCBA. In this section we will describe only open-circuit SCBAs.

Generally, in open-circuit both demand and pressure-demand, the air comes from compressed air. The air must be reduced from high pressures (2000 to 4500 psig) to a pressure suitable for use in a facepiece, three to five inches of water (or 0.1 to 0.2 psig), and the high-pressure air must be delivered to the facepiece when the user inhales. The SCBA regulator performs these functions. The exact mechanism is different for each manufacturer. Usually a series of valves, levers and expansion chambers reduce the air pressure. All regulators operate on the same mechanical principle: they admit air when the pressure in the diaphragm-operated chamber, as shown in Fig. 3, is low enough. The diaphragm is large, about three inches in diameter, so that slight changes in pressure from exhalation and inhalation will move the diaphragm, as indicated by the arrows in Fig. 3.

Breathing Apparatus
Part III

Operation of Self-Contained Breathing Apparatus Exposed to High Air Temperatures

by
Charles A. Harder, Bruce J. Held and Gail J. Cardenas

Introduction

Lawrence Livermore Laboratory (LLL) is operated by the University of California under a prime contract with the U.S. Department of Energy. Because of its size and the nature of its work, the Laboratory has a 40-member fire department that is sometimes called to fight fires that could involve highly toxic or radioactive materials. As the dangers of inhalation injury and death to LLL fire fighters are particularly acute, the Laboratory wants to provide them with the best possible protection. Thus, the Safety Science Group at LLL began a program to test various self-contained breathing apparatus (SCBA) under potential fire and emergency conditions.

We of the Safety Science Group knew the environment to which fire fighters and their equipment are exposed is the result of many variables such as high air temperatures, high humidity, water droplets in the air, fire-decomposition products, fire-extinguishing agents and radiant heat. Therefore, we felt fire fighters' protective gear should not only help them fight routine fires in safety and comfort, but should give them the protection they need for hazardous rescues or for such unforeseen emergencies as flare-ups. Ideally, we believe fire fighters' protective gear should give them more protection than they need.

We centered our studies on evaluating existing equipment so SCBA could be selected from devices that offer the best protection against conditions that could exist during a fire at the Laboratory. As a literature search revealed very little information on such evaluations, we developed test parameters and criteria to evaluate various makes and models of SCBA.

In this article, we report the results of tests conducted on various SCBA operated at air temperatures ranging from room temperature to 200°F (93°C). Our future articles will cover results from other tests in the program.

This work was performed under the auspices of the U.S. Department of Energy by the Lawrence Livermore Laboratory

Test Parameters and Equipment

Determining the air temperatures to which LLL fire fighters and their SCBA are exposed was complicated by conflicting reports from different investigators. Crempel and Burgess[1] monitored temperatures at 134 fires fought by the Boston Fire Department. For dry-bulb temperatures, they found the median maximum temperature to which fire fighters were exposed was about 92 to 125°F (33-37°C) and that maximum temperatures of more than 175°F (80°C) could be found in only one percent of all structural fires. Captain M.A. Myers reported that temperatures of 1100°F (594°C) were common six feet above the floor during fire tests in Los Angeles buildings.[2] When water was applied to the fire, fire fighters were exposed to temperatures in excess of 212°F (100°C) for short periods.

Another source, Dr. H. P. Utech, classifies the thermal conditions found in structural fire fighting into three categories:[3]

•**Routine** conditions are those in which one or two objects in a room, such as a chair, sofa or wastebasket, are burning; air temperatures range from 68 to 140°F (20-60°C), and fire fighter exposure is less than 5 minutes;

•**Ordinary** conditions are found at more serious fires, such as one next door to a flashed-over room, where air temperatures range from 140 to 572°F (60-300°C) and fire fighter exposure is from 10 to 20 minutes;

•**Emergency** conditions are severe and ur usual, such as being caught in a flashed-over room. Under emergency conditions, air temperatures range from 575 to 1800°F (300-1000°C) and the fire fighter is exposed for about 15 to 20 seconds while trying to escape.

The SCBA must function properly under all three conditions because failure could result in injury or death from fire-decomposition products, oxygen deficiency or lung burns.

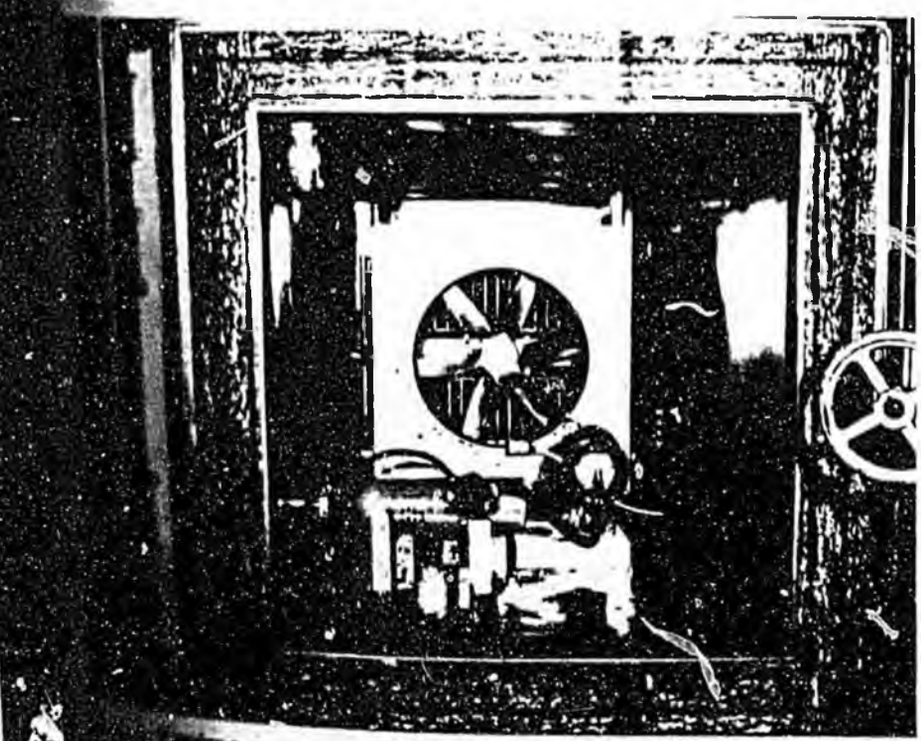
We chose a maximum temperature of 200°F (93°C) for two series of hot-air temperature tests for long exposures because it was the highest temperature obtainable in the only available environmental chamber large enough to hold an entire operating SCBA unit. This temperature also seemed reasonable, being only slightly higher than temperature used by Gempel and Burgess and not as high as Utech described for ordinary conditions with a 10- to 20-minutes exposure.

In the first series of hot-air temperature tests, we stored two units of each SCBA in the environmental chamber (Figure 1) for one-half hour at 150°F (65°C) and later at 200°F (93°C). Although the results of these tests were subjective, they identified problems that could be examined more thoroughly with instrumented tests. We also exposed five of each type of SCBA facepiece to the 200°F (93°C) air temperature in the chamber for 10 minutes and then hit the side of each facepiece lightly on the edge of the chamber to simulate a fire fighter accidentally bumping his facepiece while working.

In the second series of hot-air temperature tests, also using the environmental chamber, we dynamically tested two or three units (depending on availability) of each SCBA model. For these tests, we used mostly pressure-demand and some demand SCBA, conducting tests at 100, 125, 150, 175 and 200°F (38, 52, 65, 79 and 93°C) and at a relatively humidity of 85 to 95%. We mounted and sealed the facepiece for each SCBA on a dummy head and through this connected the entire SCBA to the LLL mechanical breathing machine[4] located outside the chamber. The breathing machine was set at a moderate to heavy work rate of 52.2 liters of air per minute to simulate a fire fighter at work, and we humidified the exhaled air to simulate human respiration. We measured facepiece pressure with a calibrated pressure transducer and monitored it on a strip-chart recorder. The air cylinder for the SCBA was turned on and off by means of a special clamp that attached to the valve with a handle that protruded through the chamber wall.

Each SCBA unit, including the facepiece, first was fully-inspected and tested for leaks. After setting up each unit in the environmental chamber and operating it for two minutes at room temperature (approximately 70°F (21°C) to ensure that it was operating properly and to measure its normal operating pressures, we turned the chamber on and set it at 100°F (38°C). We exposed the unit to that temperature for two hours and then operated the SCBA for two minutes. Next, we set the chamber at 125°F (52°C) and exposed the unit for 30 minutes before operating it for three minutes. We repeated this at 150°F (65°C). At 175°F (79°C), we operated the unit as soon as the chamber reached that temperature. We did not prolong exposure as a fire fighter wearing the unit would never be exposed long to temperatures that high. We followed the same procedure at 200°F (93°C). This time, however, we operated the unit until the cylinder ran out of air, noting the time it took. Also, we noted the time the low-pressure alarm on the unit sounded.

All the SCBA units tested were new, except for the Scott 6000- and 9000-series units.* The Scott units, although from existing supplies, first were fully-overhauled, inspected and tested by people certified by Scott for this work.



Environmental chamber used for temperature studies of SCBA.

Test Results

Subjective Tests**

We conducted the subjective tests to determine obvious problems and what instrumented tests should be run.

Two units of each of these SCBA models were tested:

- Scott Air Pak (6000 series)

*Reference to a company or product name does not imply approval or recommendation of the product by the University of California or the U.S. Department of Energy to the exclusion of others that may be suitable.

**This section on subjective test results has been abstracted

- Scott Air Pak (9000 series)
- Scott 4.5
- MSA Air Masks with Clearvue facepiece
- MSA Air Mask 401 with Hycar rubber Ultravue facepiece
- MSA Air Mask 401 with silicone Ultravue facepiece
- Survivair with neoprene facepiece.
- Survivair with silicone facepiece

The units were exposed to temperatures of 150°F (65°C) and 200°F (93°C) for 30 minutes and then were donned and operated by experienced SCBA users after each exposure. Any operating and visual problems were noted.

In general, the test subjects observed that black-colored facepieces, particularly those that had the greatest mass (and accordingly retained the most heat), were the most uncomfortable on contact with the face. Lighter weight and lighter colored (blue and yellow) silicone facepieces did not give this impression.

All the facepieces, except that on the Scott Air Pak (6000 series) were more flexible at these high temperatures and appeared to fit with less tension in the head harness. When exposed at 150°F (65°C) the Scott Air Pak (6000-series) facepiece lenses flattened out, causing the lens to separate from the facepiece.

At 150°F (65°C), the facepiece on the Survivair SCBA unit no longer fit firmly because the silicone headbands softened and slipped through the retaining buckles on the facepiece. The neoprene and Hycar rubber head harness materials did not exhibit any noticeable softening or slippage.

The breathing regulators on all the SCBA tested became increasingly noisy at higher temperatures and interfered with the user's ability to communicate. We believe this noise (a sort of honking) resulted from the softening and stretching of the breathing diaphragm housed in the regulator, the noisiest regulators being those with silicone breathing diaphragms.

The breathing tubes (most made of rubber compounds) of all devices tested softened and stretched considerably at 150 and 200°F (65 and 93°C), suggesting the airflow characteristics of the air-supply regulator could be reduced in volume. Such a reduction in airflow would increase

Table I

Effects of exposing SCBA facepieces to 200°F (93°C) air temperatures and then lightly bumping them.

Make and model of SCBA facepiece	No. of units tested	Effects of exposure and bumping
Scott 6000	5	Exposure alone caused all five lenses to flatten and separate from facepiece.
Scott 9000	5	No observable effects
Scott 4.5	5	One strap-retaining button broke on two of five units.
MSA Clearvue	5	No observable effects
MSA Ultravue with plastic ring and clips holding lens	5	Lens separated from facepiece on two of five units.
MSA Ultravue with plastic clamp ring holding lens	5	No observable effects
Survivair neoprene	5	No observable effects
Survivair silicone	5	No observable effects
Globe with Sierra facepiece	5	No observable effects
Biopak 45	1	No observable effects

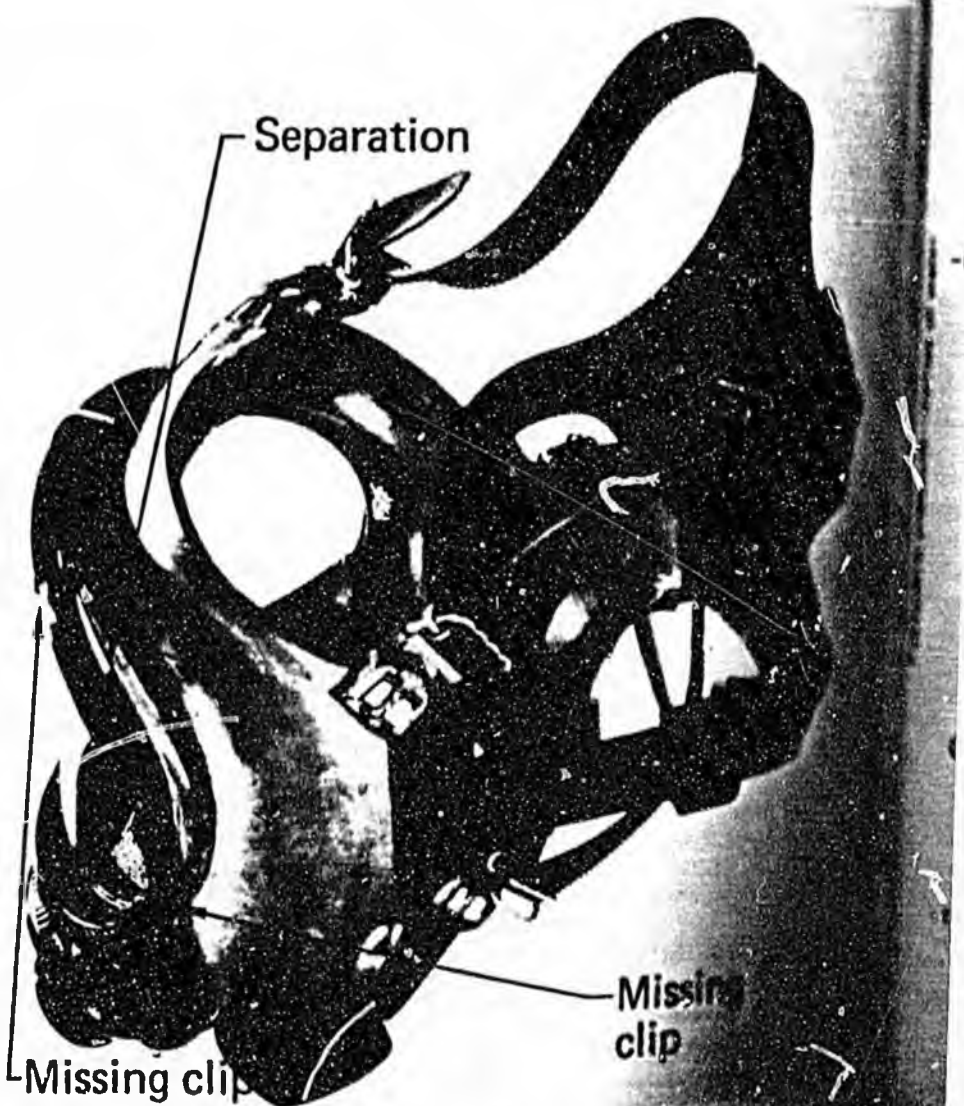


Figure 2. MSA Ultravue facepiece after heating and light [illegible] lens separated from body of facepiece. Though not directly [illegible] buttons holding the lens snapped off, allowing the lens to [illegible]

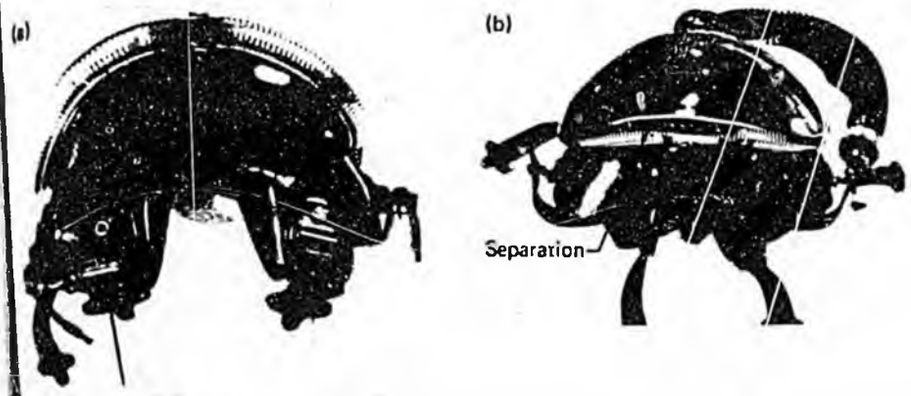


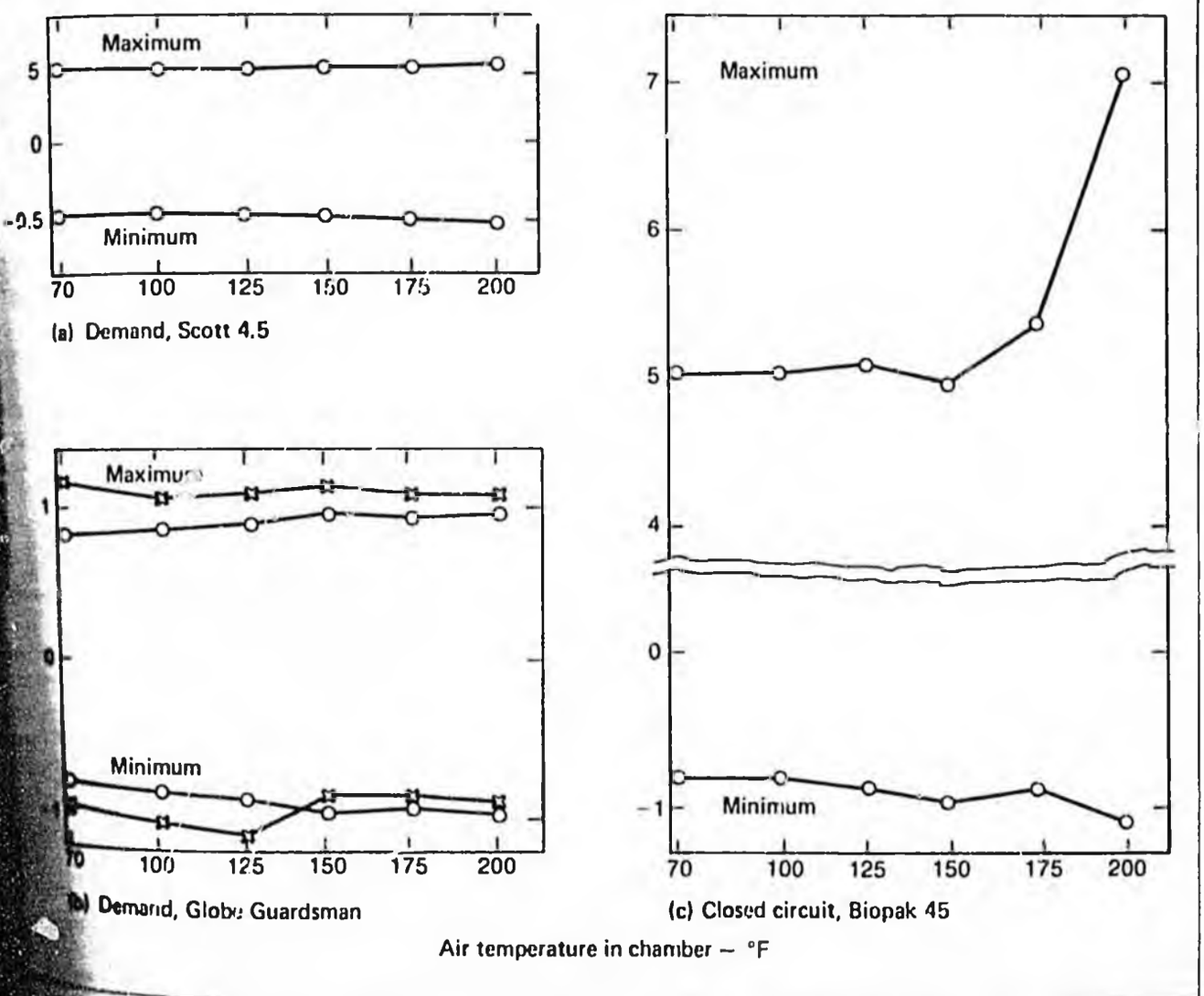
Figure 3. Old-style Scottoramic facepiece: (a) as purchased (note curvature of lens) and (b) after 10-min exposure to 200°F (lens has flattened).

the user's inhalation resistance and possibly cause leaking around the facepiece.

We conducted the facepiece tests to simulate a fire fighter moving around near a fire and accidentally bumping his facepiece while it was exposed to hot air. For this test, we exposed the various makes and models of facepieces to the 200°F (93°C) air of the environmental chamber for 10 minutes, bumped them lightly on the edge of the chamber as they were being removed and then examined each for damage. Table I shows the makes, models and number of each of the facepieces tested and the test results.

Two of the five MSA Ultravue facepieces, which have a lens held in place by plastic clips, failed when the lens separated from the facepiece after being lightly bumped (Figure 2). When the same facepiece was equipped with a plastic-ring lens holder, no problems were encountered.

The Scott 6000-series facepiece has a curved CR39 lens. At about 150°F (65°C), the curved lens tried to return to its original flat shape. Figure 3(a) shows the purchase condition of the facepiece and the curve of the lens, and Figure 3(b) shows the lens after it was exposed to 200°F



Facepiece pressures for demand and closed-circuit models. Curve (A) inhalation pressures measured at the [illegible] differ

93°C) for 10 minutes. The lens has flattened partially, distorting the shape of the facepiece, and has pulled free of the lens holder, allowing leakage. We did not bump the Scott 6000-series facepiece on the chamber edge because the heat exposure had already dislodged the lens from the facepiece. The lens is less likely to heat up and flatten when the facepiece is in place on a wearer's head and cooler air from the air cylinder is sweeping the inside of the facepiece. In this test series, the unit was not operating.

On two of the five Scott 4.5-series units, one of the plastic buttons holding the head harness to the facepiece snapped off when the unit was bumped.

Instrumented Tests

In the instrumented tests, we subjected the various makes and models of SCBA to air temperatures between 70°F (21°C) and 200°F (93°C) in the environmental chamber while they were operating on the breathing machine. We ran these tests on the following makes and models of SCBA:

- Scott 6000—pressure demand
  Old-style Scottoramic facepiece
  New-style Scottoramic facepiece
- Scott 9000—pressure demand
  Old-style Scottoramic facepiece
  New-style Scottoramic facepiece
- Scott 4.5—pressure demand
- Scott 4.5—demand
- Survivair—pressure demand
  Neoprene facepiece
  Silicone facepiece
- Globe Guardsman—demand
- Biopack 45—closed circuit

All units operated without failing in the 70°F (21°C) through 200°F (93°C) temperature range. However, the operating air pressure in the facepieces changed on some models. Figure 4 shows the facepiece pressures recorded in the pressure-demand units at the different test temperatures. The same information is shown in Figure 5 for a Scott 4.5 demand unit, two Globe demand units and the Biopak 45, a closed-circuit SCBA.

We tested the Scott 4.5 demand unit to see how it compared to the pressure-demand units. Our reasons for testing the Globe and Biopak units were because neither company made a pressure-demand unit at the time of the tests, and we thought some information on their high-temperature operation would be desirable. We did not test the Scott, MSA and Survivair demand units because we felt, aside from facepiece pressures, that they would not differ enough from the pressure-demand units to warrant testing.

The bell alarms on all units operated well and at the proper time while the units were exposed to even the highest temperature. The whistle alarm on the Scott 4.5, however, was very weak and barely audible on the pressure-demand units and undetectable on the one demand unit tested. Although the walls of the environmental chamber did reduce the sound of the alarms somewhat, the openings in the chamber allowed the testers to periodically listen for the alarms at one of the openings and tended to negate the effects of the chamber walls. We feel, however, that all alarms should sound loudly under any condition so the user and those nearby can hear them and get out of a dangerous atmosphere before their air is used up.

Table II summarizes the facepiece pressures we found at different air temperatures. At higher air temperatures inhalation pressures in the

Table II

Summary of facepiece pressures at different air temperatures (note the numerous negative pressures).

	Facepiece pressure at 70°F, in. water						Minimum inhalation pressure in facepiece, in. water			Temperature of minimum inhalation pressure, °F			Maximum exhalation pressure in facepiece, in. water			Temperature of maximum exhalation pressure, °F		
	Unit 1		Unit 2		Unit 3													
Make and model of SCBA	Inh.	Exh.	Inh.	Exh.	Inh.	Exh.	Unit 1	Unit 2	Unit 3	Unit 1	Unit 2	Unit 3	Unit 1	Unit 2	Unit 3	Unit 1	Unit 2	Unit 3
Pressure-demand units																		
Scott 6000 facepiece	1.2	2.6	0.5	2.6	—	—	0.9	0.25	—	200	200	—	2.8	2.8	—	200	175	—
Scott 6000 facepiece	-0.55	2.8	-0.4	2.5	—	—	-0.6	-0.5	—	100	100, 125	—	2.8	2.5	—	70	70	—
Scott 9000 facepiece	0.1	2.7	-0.46	2.3	—	—	-0.1	-0.5	—	200	150	—	2.7	2.3	—	175, 200	125	—
Scott 9000 facepiece	-0.25	2.4	0.08	2.4	—	—	-0.5	-0.1	—	175, 200	175	—	2.4	2.5	—	70	200	—
Scott 4.5	0.2	1.8	0.08	1.8	0.1	1.7	0.01	-0.06	0.05	200	200	200	1.9	1.8	2.1	200	70	200
Survivair facepiece	-0.1	2.6	-0.2	3.7	0.2	2.8	-0.15	-0.3	-0.06	200	200	200	2.8	4.6	2.9	100	200	200
Survivair facepiece	-0.04	1.7	0	1.8	-0.08	1.7	-0.2	-0.1	-0.27	200	200	200	1.7	1.6	1.6	150	200	—
Globe Guardsman	0.27	1.6	0.1	1.7	0.3	1.6	0.3	0.1	0.3	70	70	70	1.7	1.7	1.6	70-200	70, 175	200
Biopack 45	0.25	1.8	0.4	1.7	0.25	1.9	0.5	0.75	-0.1*	200	200	200	1.8	1.7	1.9	70	70, 100 125	70, 200
Demand units																		
Scott 4.5	-0.45	0.45	—	—	—	—	-0.5	—	—	200	—	—	0.5	—	—	200	—	—
Globe Guardsman	-0.92	1.1	-0.8	0.7	—	—	-1.08	-1.0	—	125	200	—	1.1	1.0	—	70	200	—
Biopack 45	-0.75	5.02	—	—	—	—	-1.0	—	—	200	—	—	6.9†	—	—	200	—	—

*The silicone headbands loosened as the temperature increased, thus permitting a leak between the facepiece and the face.
†The wearer would normally relieve the high exhalation pressure by cracking the face seal. This

facepiece are generally lower and exhalation pressures are higher. The only exceptions to this trend were the Survivair units, which maintained constant exhalation pressures regardless of temperature. Inhalation pressures also stayed constant with the neoprene Survivair facepiece but the inhalation pressures with two of three units having silicone facepieces increased with temperature. These increases probably resulted from the silicone becoming more flexible at higher temperatures and tending to seal to the face even better. The third silicone facepiece showed negative (subatmospheric) pressure on inhalation because the silicone head harness slipped (also noted in the subjective tests).

Besides Survivair, the only other pressure-demand units to maintain positive pressures in the facepiece on inhalation were Scott 6000-series units with the old-style facepiece and two of the three Scott 4.5-series units. The pressure in these two Scott 4.5 units approached zero (.008 and .048 inches of water) on inhalation and probably would go negative under a heavy breathing rate or with a leak between the facepiece and the face.

The negative pressures that showed up in the facepieces of pressure-demand units tested at a moderate to heavy work rate are disturbing because they indicate these units are not providing the protection they were assumed to provide. The facepiece leakage studies done at the Los Alamos Scientific Laboratory,[6] which showed a 10,000-plus protection factor for pressure-demand units, were conducted on men who were standing still and moving their heads slightly (a low work rate). At a moderate to heavy work rate, which is much more realistic for a fire fighter, some pressure-demand units showed negative facepiece pressures at ambient temperatures. At higher air temperatures, many units showed more negative pressures or went from positive to negative. This would be the worst possible time for such an occurrence as the fire fighter would be breathing at a moderately heavy to heavy rate while fighting a fire and would most likely encounter the highest concentrations of toxic materials when closest to the fire. At the same time, the high heat would cause increased negative pressure in the facepiece of a pressure-demand unit and increased leakage into the facepiece.

Results of the heat studies conducted and reported in this article will assist the LLL Fire Department in selection of SCBA. However, the results of other studies considering all parameters found in a fire environment will be necessary in order to select the most desirable unit for all conditions under which the fire department must operate. Perhaps even more important is the fact that, as these and other limitations become apparent and identifiable, the fire department training and use practices can make the necessary allowances to prevent the fire fighter from overtaxing himself beyond the capabilities of his equipment.

References

1. R. F. Gempel and W. A. Burgess, *Thermal Environment During Structural Fire Fighting*, prepared for National Fire Prevention and Control Administration, final report, NFPCA Grant 76010 (1977).

2. M. A. Myers, "Enduring the Heat" (letter to the Editor), *Fire Command*, 7 (1977).

3. H. P. Utech, Sc.D., *Protective Clothing Standards for Fire Fighters; Personal Protection—A Symposium*, U.S. Department of Health, Education and Welfare, National Center for Occupational Safety and Health, HEW Publication No. (NIOSH) 75-143, pp. 219-220 (1975).

4. G. O. Nelson, R. E., Johnsen, C. L. Lindeken and R. D. Taylor, "Respiratory Cartridge Efficiency Studies: III. A Mechanical Breathing Machine to Simulate Human Respiration," *Journal of the American Industrial Hygiene Association* 33, 745 (1972).

5. B. J. Held, C. P. Richards and R. D. Taylor, "Performance Reliability of Self-Contained Breathing Apparatus Exposed (Stored) to Various Temperatures," *Hazards Control Progress Report* No. 31, July-Dec. 1976, Lawrence Livermore Laboratory, Rept. UCRL-50007-76-2 (1977).

6. E. C. Hyatt and C. P. Richards, *A Study of Facepiece Leakage of Self-Contained Breathing Apparatus by DOP Man Tests*, Los Alamos Scientific Laboratory, Rept. LA-4927-PR (1972).

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Mr. Held has written numerous articles and papers on respirators and has been involved in research on self-contained breathing apparatus. Earlier this year, he wrote a two part article for this magazine on the history of breathing apparatus. He was the Chairman of the American National Standards Institute Z88.5 Committee on Practices for Respiratory Protection for the Fire Service.

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Toxicity of Off-Gases From Furnishings

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Product Safety Corp.

Abstract

Relative toxicity data are presented for the off-gases from various materials used in furnishings.

Introduction

The toxic hazard potential of the off-gases evolved from materials exposed to overheating is an important aspect of safety. For occupants of residences and institutions, toxic gases can prevent or hinder escape from fires, and can have effects ranging from incapacitation to death.

A toxicity screening test method developed by the authors has been used to evaluate a wide variety of materials for toxicity of off-gases under specified test conditions (1-11). A large selection of test conditions can be used, but the test conditions used for evaluating over 300 materials, including many furnishing materials, were a rising temperature program at 40°C/min from 200 to 800°C without forced air flow, an essentially closed system which provided for accumulation of the toxic gases produced over this temperature range. These particular test conditions are intended to simulate the pre-ignition stage of a fire, as well as the nonflaming aspects of the pre-flashover stage of a fire. It should be remembered that no single set of test conditions can simulate all the possible conditions which a material could encounter in an actual fire, and that these particular test conditions represent an effort to simulate a range of conditions in the critical early stages of a fire.

Toxicity test data obtained by this method on the off-gases from various materials used in furnishings are presented in this article.

Method

The toxicity screening test method has been described in detail in earlier articles (1-11), and will be only briefly summarized here. Four Swiss Webster male mice, 25 to 40 g body weight, are placed in a 4.2 liter hemispherical chamber and the off-gases from 1.00 g of material are admitted into the chamber. The test animals are observed for responses such as staggering, convulsions, collapse and death; and the times to the observation of each response in each animal are recorded. The test is terminated after 30 minutes.

The test conditions used to obtain the data in this paper were a rising temperature program at 40°C/min from 200 to 800°C without forced air flow.

Data and Discussion

Fabrics can be compared on the basis of generic type as shown in Table 1. The toxicity of the off-gases from blends of fibers was generally intermediate between that for the pure fibers and proportional to the content of the different fibers. In general, wool appeared to be more toxic than nylon; silk more than rayon; polyester more than cotton; cotton more than rayon; rayon more than polypropylene; and rayon more than nylon.

The toxicity test data on cushioning materials are presented in Table 2. There are differences between individual samples in each generic group, but the differences between generic types of foam plastics appeared

Continued on next page

Table 1. Relative Toxicity of Fibers and Fabrics by Generic Type

Material	Number of Samples	Time to Death min
Wool, 100%	4	7.65 ± 1.29
Wool, 85-90%/nylon	4	8.87 ± 1.01
Nylon, 100%	9	16.78 ± 3.49
Silk, 100%	2	9.18 ± 0.35
Silk, 70%/rayon, 30%	2	12.33 ± 0.58
Rayon, 100%	10	15.40 ± 2.41
Polyester, 100%	3	10.70 ± 2.25
Polyester, 65-87%/cotton	3	10.45 ± 0.26
Cotton, 59-70%/polyester	2	15.66 ± 1.17
Cotton, 100%	10	13.08 ± 2.14
Cotton, 100%	10	13.08 ± 2.14
Cotton, 82-86%/rayon	2	12.00 ± 0.16
Cotton, 52-75%/rayon	8	14.53 ± 2.06
Rayon, 54-75%/cotton	18	12.70 ± 2.49
Rayon, 100%	10	15.40 ± 2.41
Rayon, 100%	10	15.40 ± 2.41
Rayon, 56-73%/polypropylene	2	14.08 ± 2.16
Polypropylene, 100%	4	16.64 ± 2.76
Rayon, 100%	10	15.40 ± 2.41
Rayon, 52-72%/nylon	3	15.60 ± 3.86
Nylon, 57-62%/rayon	2	15.62 ± 0.15
Nylon, 100%	9	16.78 ± 3.49
Cotton, 100%	10	13.08 ± 2.14
Cotton, 100%, FR	6	14.19 ± 3.64

Table 2. Relative Toxicity of Off-Gases from Cushioning Materials

Material	Time to Death min
Flexible Foams	
polyether urethane, F1	20.86 ± 2.71
polyether urethane, F2	19.91 ± 1.41
polyether urethane, F3	20.49 ± 0.63
polyether urethane, F4	19.41 ± 0.55
polyether urethane, F5	19.31 ± 0.80
polyether urethane, F6	20.10 ± 1.06
polyether urethane, F7	20.73 ± 1.12
polyether urethane, F8	22.38 ± 3.84
polyester urethane, 3000	18.91 ± 0.75
polyester urethane, 3003	18.54 ± 1.45
polyester urethane, 3013	16.17 ± 0.61
polyurethane, 1	18.55 ± 0.01
polyurethane, 2	21.99 ± 1.82
polyurethane, 3	22.73 ± 1.07
polyimide, 1	12.71 ± 0.02
polyimide, 2	14.76 ± 0.95
polyester, Pneumacel	18.97 ± 0.44
polyethylene, Microfoam	20.42 ± 0.26
polyethylene	20.93 ± 0.47
polychloroprene	24.51 ± 1.46
polychloroprene, ALS	26.84 ± 1.27
polychloroprene, LS	26.50 ± 2.52
polychloroprene, LS200	27.29 ± 0.42
polychloroprene, Vonar	25.83 ± 2.16
polychloroprene	24.29 ± 0.69
polyphosphazene, 1	23.98 ± 1.52
polyphosphazene, 2	24.11 ± 2.58
polysiloxane, 3-6548	25.00 ± 0.57
Fiber Battings	
cotton batting, untreated	9.66 ± 1.29
10% boric acid, padded on	14.58 ± 1.06
8% boric acid, vapor phase	11.20 ± 1.25
polyester batting	9.44 ± 0.76
13.3% acrylic resin	10.15 ± 0.64
20.6% acrylic resin	10.54 ± 1.28
Miscellaneous Materials	
feathers/down 75/25	7.25 ± 0.12
wool, washed	7.06 ± 1.18
rubberized hair	8.18 ± 0.50
rubberized hair, FR	7.62 ± 0.83
excelsior	15.82 ± 3.11
sisal	12.59 ± 3.41
kapok	16.15 ± 0.47

Table 3. Relative Toxicity of Plastics by Generic Type

Material	Number of Samples	Time to Death min
Polyether sulfone	3	12.30 ± 2.08
Polyphenylene sulfide	4	13.21 ± 3.80
Polyaryl sulfone	2	13.48 ± 3.17
Polyimide flexible foam	2	13.74 ± 1.45
Wood	12	14.03 ± 1.48
Polyamide (nylon)	3	14.36 ± 1.71
Polyphenyl sulfone	1	15.46
Polyurethane rigid foam	7	15.49 ± 4.06
polymethyl methacrylate (PMMA)	1	15.58
Polyvinylidene fluoride	1	15.86
Cellulosic board	8	16.57 ± 3.54
Polyvinyl chloride (PVC)	2	16.60 ± 0.33
Acrylonitrile/butadiene/styrene (ABS)	3	17.13 ± 2.45
Polyethylene, including foam	5	17.31 ± 3.73
Acrylonitrile rubber (NBR)	3	19.13 ± 2.89
Polyphenylene oxide, modified	1	19.96
Polyurethane flexible foam	14	20.01 ± 1.74
Bisphenol A polycarbonate	3	20.40 ± 3.77
Polyvinyl fluoride	1	20.50
Ethylene/propylene/diene (EPDM)	2	20.69 ± 0.04
Chlorosulfonated polyethylene	2	20.88 ± 2.07
Polyisocyanurate rigid foam	2	21.68 ± 1.38
Polyisoprene (natural rubber)	1	22.13
Chlorinated polyvinyl chloride (CPVC)	2	22.25 ± 0.69
Polystyrene	2	23.10 ± 4.33
Polyphosphazene flexible foam	2	24.05 ± 0.09
Styrene-butadiene rubber (SBR)	1	24.11
Polychloroprene flexible foam	6	25.88 ± 1.24
Chlorinated polyethylene	2	26.08 ± 1.80

Table 4. Effect of Fire Retardants on Toxicity of Off-Gases

Material	Time to Death min
cotton batting (California)	
untreated	10.16 ± 1.43
treated (believed to be boric acid based)	10.44 ± 0.95
cotton batting (Louisiana)	
untreated	9.66 ± 1.29
8% boric acid, vapor phase	11.20 ± 1.25
10% boric acid, padded on	14.58 ± 1.06
viscose fibers	
untreated	13.71 ± 0.37
12.3% Sandoflam 5060	14.39 ± 0.59
15.5% Sandoflam 5060	14.24 ± 0.31
17.5% Sandoflam 5060	14.58 ± 1.45
polypropylene fibers	
untreated	20.35 ± 0.18
3.1% Sandoflam 5070	17.87 ± 0.23
5.0% Sandoflam 5070	19.14 ± 1.22
vinyl fabric	
untreated	15.64 ± 0.95
FR to Boston code	17.49 ± 1.07
polyurethane flexible foam, A	
untreated	19.21 ± 1.57
FR (Sb, Cl) to California requirements	20.13 ± 1.06
polyurethane flexible foam, C	
untreated	18.28 ± 0.91
FR (P, Br) to California requirements	17.12 ± 1.72
polyurethane flexible foam, D	
untreated	10.16 ± 0.68
FR (P, Br) to California requirements	11.18 ± 0.74
polyurethane flexible foam, E/F	
untreated	9.69 ± 0.62
FR (P, Cl) to California requirements	12.68 ± 0.94
FR (P, Cl) to FAA requirements	13.18 ± 2.16
polyurethane rigid foam	
untreated	23.92 ± 2.14
10% RF-230	23.74 ± 2.11
7% Fyrol 6	25.69 ± 0.80

TOXIC GAS . . .

to be significant. Polyimide flexible foam exhibited the greatest toxicity under these particular test conditions – followed by polyurethane flexible foam, polyphosphazene flexible foam and polychloroprene flexible foam.

Plastics are compared on the basis of generic type in Table 3. The foam plastics are included in this comparison, and wood is included to provide a reference for comparison. The majority of the generic types of plastics appear to be less toxic than wood under these particular test conditions.

Fire retardants are widely used to increase the fire resistance of materials, and there has been interest in the effect of fire retardants on the toxicity of off-gases. Comparisons of various materials without and with fire retardants are presented in Table 4. In the majority of comparisons, the addition of fire retardants decreased relative toxicity under these particular test conditions.

References

1. C.J. Hilado, "Screening Materials for Relative Toxicity in Fire Situations," *Modern Plastics*, Vol. 54, No. 7, 64-66, 68 (July 1977).
2. C.J. Hilado, H.J. Cumming, and C.J. Casey, "Relative Toxicity of Materials in Fire Situations Determined in New Testing," *Western Fire Journal*, Vol. 30, No. 2, 32-33 (February 1978).
3. C.J. Hilado, H.J. Cumming, and C.J. Casey, "Relative Toxicity of Materials in Fire Situations," *Modern Plastics*, Vol. 55, No. 4, 92, 94, 96 (April 1978).
4. C.J. Hilado, "Toxicity of Pyrolysis Gases from Materials," *SAMPE Quarterly*, Vol. 9, No. 3, 14-15 (April 1978).
5. C.J. Hilado and H.J. Cumming, "Relative Toxicity of Pyrolysis Gases from Materials: Effects of Chemical Composition and Test Conditions," *Fire and Materials*, Vol. 2, No. 2, 68-79 (April 1978).
6. C.J. Hilado, H.J. Cumming, and C.J. Casey, "Toxicity of Pyrolysis Gases from Natural and Synthetic Materials," *Fire Technology*, Vol. 14, No. 2, 136-146 (May 1978).
7. C.J. Hilado, H.J. Cumming, and A.M. Machado, "Relative Toxicity of Pyrolysis Gases from Materials: Specific Toxicants and Special Studies," *Fire and Materials*, Vol. 2, No. 4, 141-153 (October 1978).
8. C.J. Hilado and D.P. Brauer, "How Test Conditions and Criteria Effects Impact on Pyrolysis-Gas Toxicity Findings," *Modern Plastics*, Vol. 56, No. 3, 62-64 (March 1979).
9. C.J. Hilado, H.J. Cumming, and J.E. Schneider, "Relative Toxicity of Pyrolysis Gases from Materials: Effects of Temperature, Air Flow, and Criteria," *Fire and Materials*, Vol. 3, No. 4, 183-187, (December 1979).
10. C.J. Hilado and P.A. Huttlinger, "Concentration-Time Relationships in Off-Gas Toxicity," *Fire Technology*, Vol 16, No. 3, 192-203 (August 1980).
11. C.J. Hilado and P.A. Huttlinger, "Rising vs. Fixed Pyrolysis Temperature in Evaluating Toxicity," *Modern Plastics*, Vol. 57, No. 9, 89-94 (September 1980).

Fire-Resistive Fabrics Tested For Toxicity

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Five samples of flame-resistant fabrics were evaluated for toxicity of pyrolysis gases, using the USF-NASA toxicity screening test method. The samples were identified as wool, flame-retarded cotton, Nomex, unstabilized polybenzimidazole. Under these particular test conditions, time to death with wool was less than half

that of the other four fabrics.

Fabrics used in applications which require a certain degree of flame resistance are selected from materials which exhibit a natural flame resistance, which have been flame-retardant treated or which are inherently more thermally stable. The potential toxicity of the gases produced upon overheating or burning is one aspect which must be considered.

The USF-NASA toxicity screening test method has been used to evaluate a wide variety of materials for toxicity of pyrolysis gases under specified test conditions. We shall present the toxicity test data on five samples of flame-resistant fabrics.

Five samples of flame resistant fabrics were supplied in the course of the four-year NASA grant which supported the development of this screening test method. These samples were wool, flame-retarded cotton, Nomex, unstabilized polybenzimidazole (PBI) and stabilized PBI.

The samples were conditioned at a room temperature of 21 degrees Celsius, plus or minus one degree Celsius, and at a relative humidity of 50 percent, plus or minus five percent.

There is need for a brief summary of the USF-NASA toxicity screening test. Four Swiss-Webster male mice of 25 to 40 grams of body weight are placed in a hemispherical chamber and the pyrolysis gases from a one gram of sample material are admitted into the chamber. The test animals are observed for responses such as staggering, convulsions, collapse and death. The times to the observation of each response in each animal is recorded.

A wide variety of test conditions can be used. The test conditions used for evaluating over 300 materials were at a rising temperature of 40 degrees Celsius per minute from 200 degrees to 800 degrees Celsius without forced air flow, in an essentially closed system which provided for accumulation of the toxic gases produced over this temperature range. These particular test conditions are intended to simulate the pre-ignition stage of a fire. It should be remembered that no single set of test conditions can simulate all the possible conditions which a material could encounter in an actual fire.

The toxicity test data obtained are presented in Table 1. Times to the various animal responses in individual tests, as indicated by a test reference, are reported as mean, plus or minus standard deviation within experiment (between animals).

Times to the various animal responses for each material are reported as mean plus or minus standard deviation between experiments.

The toxicity test data on the reference material (bisphenol A polycarbonate), evaluated at the same time, are comparable to historical data on this particular material. The data in this study can therefore be considered comparable to previously

Table 1. Toxicity of Pyrolysis Gases from Flame-Resistant Fabrics

material	test reference	weight charged g	weight pyrolyzed g	time to staggering min	time to convulsions min	time to collapse min	time to death min	CO conc. ppm	CH_2 conc. ppm
wool	EMU-114	1.00034	0.72706	4.83 ± 0.30	5.77 ± 0.94	5.91 ± 0.93	6.90 ± 0.64	60	
	EMU-118	1.00012	0.75087		5.65 ± 0.46	5.83 ± 0.42	8.26 ± 0.62	230	
	mean			4.83	5.71 ± 0.08	5.87 ± 0.06	7.58 ± 0.98		
FR cotton	EMU-115	1.00007	0.67329	7.28 ± 0.15	13.62 ± 0.26	14.05 ± 0.45	16.43 ± 2.45	10,400	1,060
	EMU-120	1.00006	0.67865		15.64 ± 2.59	16.48 ± 2.32	19.44 ± 3.60	11,240	990
	mean			7.28	14.73 ± 1.57	15.27 ± 1.72	18.94 ± 0.71		
Nomex	EMU-113	1.00000	0.45691	15.63	16.71 ± 0.33	16.94 ± 0.37	18.44 ± 0.83	6,720	920
	EMU-119	1.00016	0.44056	14.00	16.03 ± 0.62	16.28 ± 0.56	17.87 ± 0.94	5,930	700
	mean			14.82 ± 1.15	16.37 ± 0.68	16.61 ± 0.67	18.16 ± 0.40		
unstabilized PBI	EMU-111	1.00010	0.19814		16.75 ± 0.40	16.97 ± 0.41	18.24 ± 0.47	2,490	1,270
	EMU-116	1.00046	0.19980	15.53 ± 0.07	16.10 ± 0.14	16.26 ± 0.13	17.52 ± 0.14	2,540	920
	mean			15.53	16.43 ± 0.46	16.62 ± 0.50	17.88 ± 0.51		
stabilized PBI	EMU-112	0.99957	0.31287		17.37 ± 0.28	17.51 ± 0.14	19.74 ± 0.68	2,600	210
	EMU-117	1.00040	0.34180	13.10	17.79 ± 0.08	18.00 ± 0.12	19.65 ± 0.42	2,540	210
	mean			13.10	17.58 ± 0.30	17.76 ± 0.35	19.70 ± 0.06		
reference CUH-71	EMU-110	1.00014	0.84139	16.08 ± 0.80	17.23 ± 0.13	18.52 ± 1.10	21.80 ± 1.96	9,440	6,200

published data obtained under these particular test conditions.

Under these particular test conditions, time to death with wool was less than half that with the other four fabrics, about eight minutes compared to 18 to 20 minutes. Time to staggering, normally the first sign of incapacitation, was about five minutes with wool, seven minutes with Nomex, and 13 to 16 minutes with PBI.

Time to convulsions, an indication of effect on the central nervous system, was about six minutes with wool, 15 minutes with flame-retarded cotton, 16 minutes with Nomex, and 16 to 18 minutes with PBI.

Carbon monoxide appears to have been the principal toxicant in the gases from Nomex and cotton, and a significant toxicant in the gases from PBI. The toxicants in the gases from wool seemed to be gases other than carbon monoxide, and seemed to affect the animals more rapidly.

The stabilized PBI fabric appeared to exhibit slightly less toxicity than compared to the unstabilized PBI. The stabilized fabric exhibited a weight loss of 32.7 percent, compared to 19.9 percent for the unstabilized fabric.

This difference may be related to the respective times to death of 19.7 and 17.9 minutes, which means that the stabilized fabric sample was at a temperature of 800 degrees Celsius for 1.8 minutes longer before being removed.

One aspect of the potential toxic effects which cannot be readily quantified is the degree to which the gases found noxious by humans. The gases from wool were particularly noxious, making this material perhaps the most unpopular among the laboratory personnel performing the test.

Under these particular test conditions, the wool sample appeared to be the least desirable from a toxic hazard viewpoint, and the Nomex sample may be the most desirable.

REFERENCES

1. C.J. Holado, "Screening Materials for Relative Toxicity in Fire Situations," Modern Plastics, Vol. 54, No. 7, 64-66, 68 (July 1977)
2. C.J. Hilado and H.J. Cumming, "Relative Toxicity of Pyrolysis Products from Upholstery Fabrics: Effects of Fabric Variables and Test Conditions," Journal of Combustion Toxicology, Vol. 4, No. 3, 393-414 (August 1977)
3. C.J. Hilado and H.J. Cumming, "The Effect of Test Conditions on the Relative Toxicity of the Pyrolysis Products from Some Plastics," Fire Technology, Vol. 13, No. 4, 325-328, 383 (November 1977)
4. C.J. Hilado, H.J. Cumming, and C.J. Casey, "Relative Toxicity of Materials in Fire Situations," Modern Palstics, Vol. 55, No. 4, 92, 94, 96 (April 1978)
5. C.J. Hilado and H.J. Cumming, "Relative Toxicity of Pyrolysis Gases from Materials: Effects of Chemical Composition and Test Conditions," Fire and Materials, Vol. 2, No. 2, 68-79 (April 1978)
6. C.J. Hilado, H.J. Cumming, and C.J. Casey, "Toxicity of Pyrolysis Gases from Natural and Synthetic Materials," Fire Technology, Vol. 14, No. 2, 136-146 (May 1978)
7. C.J. Hilado, H.J. Cumming, and A.M. Machado, "Relative Toxicity of Pyrolysis Gases from Materials: Specific Toxicants and Special Studies," Fire and Materials, Vol. 2, No. 4, 141-153 (October 1978)
8. C.J. Hilado and D.P. Brauer, "How Test Conditions and Criteria Effects Impact on Pyrolysis-Gas Toxicity Findings," Modern Plastics, Vol. 56, No. 3, 62-64 (March 1979)
9. C.J. Hilado, "Laboratory Test Methods for Evaluating the Fire Response of Materials," Fire Journal, Vol. 73, No. 6, 69-75, 94 (November 1979) ★

Heart disease in fire fighters Part 1

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Heart disease is a general term which includes many abnormalities of the heart. Two of these heart problems are listed in the right-hand portion of Figure 1. Valve disease, which includes stenosis of the aortic valve and regurgitation in the mitral valve, is unlikely to be a major heart problem in fire fighters since it is readily detected in a routine physical examination and should therefore prevent the individual from entering the fire service. Moreover, it is unlikely that fire fighters who pass an entrance exam would develop a significant number of valve disease problems.

Arrhythmia is a heart condition in which the various chambers of the heart do not beat in normal sequence. Generally, individuals with major types of arrhythmias (frequent ventricular arrhythmias) would not be allowed to enter the fire service because of the possibility of ventricular fibrillation and sudden death. However, fire fighters may develop arrhythmias as a result of exposure to toxic gases. That factor will be discussed later in this series.

There are many other relatively common heart problems, but there is no evidence that their incidence is significant in fire fighters.

The term ischemia appears at left in Figure 1. Ischemic heart disease is a condition in which the supply of oxygen to the heart muscle is inadequate to meet the demand. If ischemia becomes severe, a myocardial infarction or heart attack occurs. Ischemic heart disease is common in the American population. It is due primarily to coronary heart disease (atherosclerotic heart disease), which is the accumulation of cholesterol and other substances within the coronary arteries. This accumulation decreases blood flow and oxygen delivery to the heart muscle. Ischemia may also be caused by other factors which reduce oxygen delivery, *i.e.*, carbon monoxide, or factors that greatly increase oxygen requirements, such as high levels of catecholamines (adrenalin). These causes of ischemia are uncommon in our general society, but may be an important aspect of the heart disease problem among fire fighters.

In 1976, the International Association of Fire Fighters published the results of a fire fighter mortality study[1] based on examination of the death certificates of 101 fire fighters who died in the line of duty during a 15-month period in 1975-76. Of the 101 deaths, 45 were caused by heart attacks. The results may seem alarming, but they might be obtained in any occupation since, in our society, more people die from heart attacks than from any other cause.

There are data, however, that show that the incidence of ...eath from heart attack and ischemic heart disease is far higher ...ong fire fighters than among the general population. (See ...s 1 and 2, which summarize statistics published by the ...ublic Heath Service.)[2,3,4]

... data show that in the older age groups (55-64), where ...st percentage of the deaths occur, the death rate ...ovascular diseases, including atherosclerotic heart ...hest in fire fighters. Personnel in this age group ...0 to 30 years' exposure to the stress of fire ...hat the death rate from atherosclerotic heart

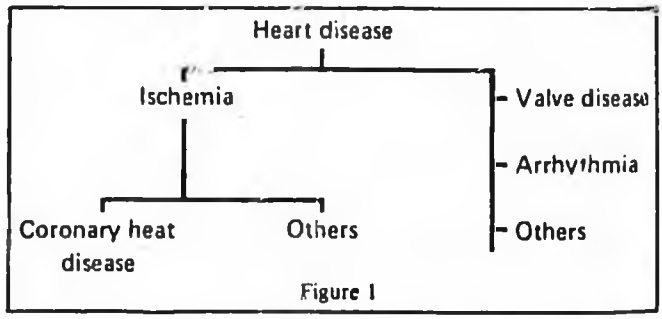


Figure 1

disease in fire fighters ages 55-59 and 60-64 is more than double the average death rate for the "All occupations" category. Furthermore, these data show that the death rate from atherosclerotic heart disease is higher in fire fighters than in any other occupation studied.

Exercise and electrocardiographic stress testing is a common clinical procedure used to test for the presence of ischemic heart disease.[5,11] The Los Angeles City and Los Angeles County Fire Departments, California, use the procedure to eliminate fire fighter applicants who have ischemic heart disease. In a study conducted at the UCLA Medical Center on a random sample of 90 active Los Angeles City fire fighters, Barnard, *et al.*,[6] found that ten percent of the group had electrocardiographic changes indicative of ischemic heart disease. These data were compared to data on a group of Los Angeles insurance executives, who had only eight percent ischemic responses.

Studies conducted on 529 Los Angeles County fire fighters revealed 10 percent ischemic responses.[7] This was a higher percentage than that in other Los Angeles County safety personnel, including sheriffs, marshals, and lifeguards.

Other groups at "high risk" for atherosclerotic heart disease have been shown to have much lower incidence of ischemic electrocardiographic responses to exercise testing. Spangler, *et al.*,[8] found only 4 percent ischemic responders in a group of 362 aircraft pilots and controllers, while McHenry[9] reported 5 percent ischemic responders in a group of Indiana state policemen.

Risk factor analysis in Los Angeles City fire fighters

Data compiled by the city of Los Angeles, California, disclosed that fire fighters receive more disability pensions for heart disease than for any other illness. When compared with policemen, the fire fighters receive more disability pensions for heart disease despite the fact that they employ half the number of men. Since fire fighters are admitted to the department only after rigorous medical screening and reaching a high level of physical conditioning, the findings suggest some specific occupational hazard of fire fighting. The present study was undertaken to determine whether the apparent high incidence of heart disease in fire fighters is related to the usual risk factors associated with coronary heart disease or whether some unique phase of the fire fighters' lifestyle might be responsible.

Table 1. Deaths per 1000 People — All Diseases of the Cardiovascular System

Occupation	Age 35–44	45–54	55–59	60–64
Firemen	.87	8.63	21.18	38.51
Policemen	1.99	8.71	18.94	28.98
Longshoremen	2.13	7.79	16.37	23.76
Lumbermen	1.40	5.23	11.89	20.66
Construction Workers	2.32	6.98	11.73	18.83
Smelter & Furnacemen	.74	3.27	7.91	9.43
All Occupations	1.49	5.42	11.26	17.20

Table 2. Deaths Per 1000 People — Atherosclerotic Heart Disease

Occupation	Age 35–44	45–54	55–59	60–64
Firemen	.67	5.97	13.95	20.40
Policemen	1.40	5.82	12.94	18.26
Longshoremen	1.04	4.10	7.73	11.88
Lumbermen	.64	2.55	7.05	10.77
Construction Workers	.99	3.19	5.81	9.62
Smelter & Furnacemen	.37	1.71	4.75	6.29
All Occupations	.79	3.25	6.75	9.90

The study consisted of investigating the electrocardiographic (ECG) response to near-maximal exercise of 90 randomly selected fire fighters between ages 40 and 59, along with documentation of their blood pressure, serum cholesterol, and smoking habits. The fire fighters were then compared to a similar age group of insurance underwriters previously studied in our laboratory.[10]

Methods

The subjects of this study were 90 men from the Los Angeles City Fire Department. They were selected using a random table and the employee numbers of all active fire fighters 40 years of age and older. The age range was 40 to 59 years, with a mean of 46 years. All of the subjects were considered to be in good health without any overt symptoms of heart disease.

The testing was conducted in two phases. During the first phase the men reported to the hospital where they were given a history questionnaire. Height and weight were recorded and a standard 12-lead electrocardiogram was obtained. Blood pressure was also measured at this time. One week later the men reported to the stress testing laboratory in a fasting state for phase two. The history questionnaire, which contained information pertaining to health history as well as physical activity habits, was examined by the doctor and discussed with the subject.

Blood pressure was again measured and a blood sample obtained for cholesterol determination. Skinfold fat measurements were taken from the 10 sites described by Allen, *et al.*,[9] using Best (USAMRN) calipers. The subjects were then given a continuous, near-maximal (> 90% of predicted max) multistage treadmill test as described previously.[10] The ECG was continuously monitored from a single transthoracic lead with the positive electrode at the V_5 position and the negative electrode at the RV_5 position. An ischemic ECG change was defined as at least 1 mm of horizontal or down slanting depression of the S-T segment observed either during exercise or in the 5-minute post-exercise recovery period. All subjects with ischemic ECG responses were returned to the laboratory on another date for retest purposes. During the additional visit to the laboratory, the treadmill test was repeated and another blood sample obtained for cholesterol, triglyceride, and lipoprotein determinations.*

The data obtained from the fire fighters were compared to data previously obtained from a group (N = 232) of Los Angeles insurance underwriters 40 to 59 years of age ($\bar{X}$ = 47 yrs.)[10]

Results

Risk factor analysis — Table 3 gives the data on coronary heart disease (CHD) risk factors. Cholesterol values ranged from 154 to 305 mg% with only 12 percent of the men having values greater than 260 mg%. None of the men had values higher than normal ranges established by the National Heart and Lung Institute. Mean systolic and diastolic blood pressure values were well within the normal range. Only 2 percent of the fire fighters had values greater than 160/90 mm Hg. Thirty-two percent of the men were smokers. Only one man had elevated cholesterol, was a smoker, and was hypertensive at the time of testing. Only five men had two of the three risk factors elevated, and 47 had none of the three risk factors elevated. Total skinfold fat measurements in excess of 150 mm were considered to be approaching obesity, and 24 percent of the men fell into this category.

Near-maximal treadmill test — Figure 2 shows the workloads at which the fire fighters reached their near-maximal heart rates. The maximal oxygen uptake, estimated from the workload oxygen cost, was 49.9 + 0.5 ml/min/kg. These data are compared to data previously obtained from the group of Los Angeles insurance executives. The estimated maximal oxygen uptake for the insurance executives was 41.9 ml/min/kg. Nine of the fire fighters had ischemic ECG responses indicating the presence of coronary heart disease. One subject in the random sample completed phase 1 of the testing having a normal resting ECG. Two days later he had a myocardial infarction at a fire and died in the hospital. One other subject (age 47) had a normal ECG during the stress test and then suffered a myocardial infarction while taking a shower in tepid water 20 minutes after exercise.

Fire fighters with ischemic ECGs — Of the nine fire fighters who had ischemic ECGs during the near-maximal stress test, only one had an abnormal resting ECG. Only one subject had a lipid abnormality (type IV hyperlipoproteinemia) and only one was hypertensive (165/100). Three of the men were smokers at the time of testing. All nine men had ischemic responses during exercise. One subject had previously experienced chest pains at a fire and was taken to a hospital where his ECG was reported to be within normal limits. This same subject experienced angina during the stress test. None of the other subjects admitted experiencing any chest pains or discomfort.

Discussion

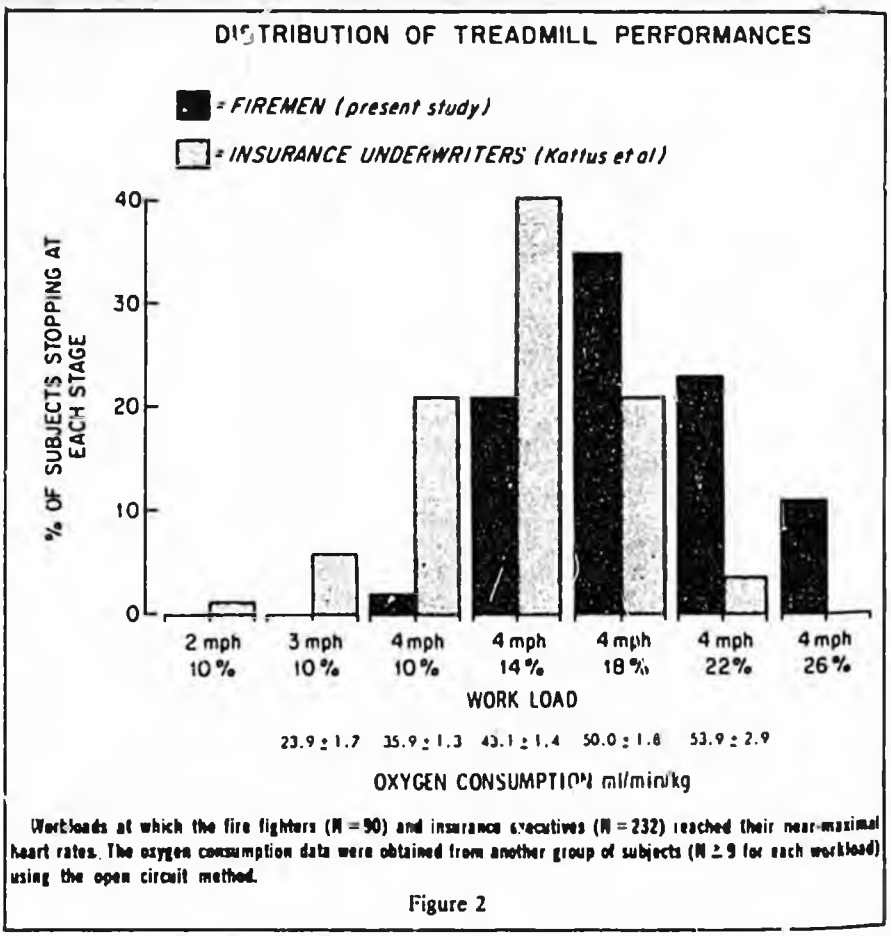
The value of the exercise electrocardiogram as a predictor of subsequent coronary heart disease has been extensively

Table 3. CHD Risk Factor Data* for Los Angeles City Fire Fighters

	Cholesterol (mg%)	Systolic B.P. (mm Hg)	Diastolic B.P. (mm Hg)	Skinfold Fat (mm)	Smoking (%)
40–50 yrs N = 68	221 ± 4	124 ± 2	77 ± 1	149 ± 4	40
51–59 yrs N = 22	226 ± 7	131 ± 3	82 ± 2	139 ± 6	5

*Values are means ± standard error

*All blood determinations were done by Laboratory Procedures Division, the Upjohn Co., Woodland Hills, CA 91364.



Workloads at which the fire fighters (N = 90) and insurance executives (N = 232) reached their near-maximal heart rates. The oxygen consumption data were obtained from another group of subjects (N ≥ 9 for each workload) using the open circuit method.

Figure 2

studied. Kattus, *et al.*,[18] reported that during a 2.5 year follow-up of their insurance group, 33 percent of those individuals with initial ischemic responses experienced a coronary event. In the study of insurance underwriters, Kattus, *et al.*,[18] found that 8 percent of the 232 men (ages 40 to 59) had ischemic ECG responses during the near-maximal stress test. This value is compared to the 10 percent value observed for the fire fighters. Felton[10] has reported a similar value for Los Angeles County fire fighters.

Comparison of our data obtained in Los Angeles with those reported for other areas is somewhat difficult because of different research designs, *i.e.*, different age ranges, stress tests, and criteria for ischemia. Lester, *et al.*,[21] reported finding 5 percent ischemic responders in a group of 114 Birmingham businessmen (ages 40 to 75) performing a Master two-step test. Spangler, *et al.*,[22] found only 4 percent ischemic responders in a group of 362 aircraft pilots, controllers, etc., performing a multistage treadmill test. McHenry, *et al.*,[23] reported 5 percent ischemic responses in a group of Indiana state policemen subjected to a maximal stress test. Doan, *et al.*,[24] observed 7.5 percent ischemic responses in a group of 433 Seattle volunteers also subjected to a maximal stress test. These reported incidences of ischemic responses are all lower than the incidence observed in our random sample of fire fighters; however, some investigators[21,24] have reported a higher percentage of ischemic responses.

Our data as well as the data of Felton[10] indicate that Los Angeles fire fighters have an unexpectedly high incidence of ischemic stress tests, which is difficult to explain since they are a medically-selected population and have relatively low risk factors for coronary heart disease. Only 12 percent of the fire fighters had cholesterol values greater than 260 mg% while 18 percent of the insurance executives fell into this category. Only 2 percent of the fire fighters had blood pressure values above 160/90 mm Hg while 25 percent of the insurance executives were hypertensive. Thirty-two percent of the fire fighters were smokers at the time of testing as opposed to 26 percent for the insurance executives. The treadmill performances indicated that the fire fighters were in better physical condition than the insurance executives.

Of the nine fire fighters with ischemic ECG responses, only one was hypertensive. One had elevated triglycerides and a lipoprotein pattern indicating type IV hyperlipoproteinemia. All of the cholesterol values were within the normal ranges established by the Heart and Lung Institute. One subject experienced a myocardial infarction following a normal ECG response to near-maximal exercise (HR 180). He was transferred immediately to the Coronary Care Unit where he recovered from an uncomplicated anteroseotal myocardial infarction. His blood pressure (125/80) and cholesterol (234 mg%) were within normal ranges. He was a little overweight and had skinfold measurements for the 10 sites totalling 156 mm. He had previously never experienced chest pains or other symptoms.

Although the 10 percent ischemic responses observed in the fire fighters may not be statistically different from the 8 percent observed in the group of insurance executives, the finding is still unexpected since fire fighters are a medically-selected population with low risk factors for coronary heart disease.

The observations suggest that ischemic heart disease may be job-associated. Recent electrocardiographic recordings obtained from fire fighters while on the job show that for the most part they are relatively sedentary, however, they frequently must exert themselves maximally. Extremely high heart rates recorded immediately after the alarm, during the anticipation phase while riding on the truck, and for prolonged periods during actual fire fighting, indicate an emotional stress.[21] In addition, the men are exposed to thermal stress as well as inhalation pollution. All of these factors may be related

Note: Portions of this material first appeared in the *Journal of Occupational Medicine*, November 1975, Volume 17, No. 11. The material has previously appeared in *The California Fireman*, published by the California State Firemen's Association, Inc. Used with permission.

to the pathogenesis of ischemic heart disease and may be independent of atheromatous deposits in the coronary arteries.

Summary

Near-maximal ECG stress testing and coronary artery disease risk factor analysis, including blood pressure, serum cholesterol, and smoking habits, were conducted on a randomly selected group (N = 90) of Los Angeles City fire fighters ranging in age from 40 to 59 years. The data obtained from the fire fighters were compared to data previously reported for a group of Los Angeles insurance underwriters of the same age range.

Only 12 percent of the fire fighters had cholesterol values greater than 260 mg%, while 18 percent of the insurance executives fell into this category. Only 2 percent of the fire fighters had blood pressure values greater than 160/90 mm Hg, while 25 percent of the insurance executives were hypertensive. Thirty-two percent of the fire fighters were smokers at the time of testing as compared to 26 percent for the insurance executives. Only one fire fighter had all three risk factors elevated and only five had two risk factors elevated. Forty-seven of the fire fighters had no risk factors elevated. Ten percent of the fire fighters had ischemic stress tests as compared to 8 percent for the insurance executives. Of the nine fire fighters with ischemic stress tests, one was hypertensive, one had elevated serum triglycerides, and three were smokers at the time of testing.

Since the fire fighters are a medically-selected population with low risk factors for CHD, the observed incidence of ischemic stress tests is surprising and suggests that ischemic heart disease may be job-associated.

Editor's note: Next month, in Part 2 of "Heart Disease in Fire Fighters," Dr. Barnard will discuss a follow-up study conducted on Los Angeles City fire fighters, and analyze data obtained from the medical records of 1055 New York City fire fighters.

About the author

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His research interests are in the general area of exercise physiology. "My major areas of interest at the present time," he says, "include biochemical adaptations in myocardial and smooth muscle, and the value of exercise as a preventive measure for vascular diseases." In recent years he has participated in and published papers on several research projects involving fire fighters.

Dr. Barnard's research has been supported by grants from the American Heart Association and the John P. Redmond Fund. Most of his work, however, is supported by contributions from fire fighter associations or individuals. Money is needed to continue this project, and donations may be made to: UCLA Foundation — Dr. R. J. Barnard, 405 Hilgard Avenue, Los Angeles, CA 90024.

References

1. "Fire Fighter Mortality Report." International Association of Fire Fighters, Washington, D.C., May 1976.
2. "The Comparability of Reports on Occupations from Vital Records and the 1950 Census." U.S. HEW Public Health Service, Vital Statistics, Special Reports, Vol. 53, No. 1, June 1961.
3. "Mortality by Occupation and Industry Among Men 20-64 Years of Age: United States, 1950." U.S. HEW Public Health Service, Vol. 53, No. 2, September 1962.
4. "Mortality by Occupation and Cause of Death Among Men 20–64 Years of Age: United States, 1950." U.S. HEW Public Health Service, Vol. 53, No. 3, September 1963.
5. Master, A.M.; Friedman, R.; and Dack, S.: Electrocardiogram after standard exercise as a functional test of the heart. Amer. Heart J. 24:777, 1942.
6. Rosenfeld, I.; Master, A.M.; and Rosenfeld, C.: Recording the electrocardiogram during the performance of the Master two-step test. Circulation 29:204, 1965.
7. Scherf, D., and Schaffer, A.I.: Electrocardiographic exercise test. Amer. Heart J. 43:927, 1952.
8. Lepeschkin, E.: Exercise test in the diagnosis of coronary heart disease. Circulation 22:986, 1960.
9. Mattingly, T.W.: The postexercise electrocardiogram: Its value in the diagnosis and prognosis of coronary heart disease. Amer. J. Cardiol. 9:395, 1962.
10. Robb, G.P., and Marks, H.H.: Latent coronary artery disease: Determination of its presence and severity by the exercise electrocardiogram. Amer. J. Cardiol. 13:603, 1964.
11. Brody, A.J.: Master two-step exercise test in clinically unselected patients. JAMA 171:1195, 1959.
12. Rumball, C.A., and Acheson, E.C.: Latent coronary heart disease detected by electrocardiogram before and after exercise. Brit. Med. J. 1:423, 1963.
13. Bellet, S.; Roman, L.; Nichols, G.J.; and Muller, O.F.: Detection of the coronary prone subject in a normal population by radioelectrocardiographic exercise test: Follow-up studies. Amer. J. Cardiol. 19:783, 1967.
14. Barnard, R.J.; Gardner, G.W.; Diaco, N.V.; and Kattus, A.A.: Near-maximal ECG stress testing and coronary artery disease risk factor analysis in Los Angeles City Fire Fighters. J. Occup. Med. 17:693, 1975.
15. Felton, J.S.: Conducting cardiopulmonary evaluations of County of Los Angeles Safety Personnel. Health Service Reports 88:515, 1973.
16. Spangler, R.D.; Horman, M.J.; Miller, S.W., *et al.*: A submaximal exercise electrocardiographic test as a method of detecting occult ischemic heart disease. Am. Heart J. 8:752, 1970.
17. McHenry, P.L.; Fisch, C.; Jordan, J.W.; and Corya, B.R.: Cardiac arrhythmias observed during maximal treadmill exercise testing in clinically normal men. Am. J. Cardiol. 29:331, 1972.
18. Kattus, A.A.; Jorgensen, C.R.; Worden, R.E.; and Alvaro, A.B.: S-T segment depression with near-maximal exercise in detection of preclinical coronary heart disease. Circulation 44:585, 1971.
19. Allen, T.H.; Peng, M.T.; Chen, K.P.; Huang, T.F.; Chang, C.; and Fang, H.S.: Prediction of total adiposity from skin folds and the curvilinear relationship between external and internal adiposity. Metabolism 5:328, 1956.
20. Felton, J.S.: Conducting cardiopulmonary evaluations of County of Los Angeles Safety Personnel. Health Service Reports 88:515, 1973.
21. Lester, F.M.; Sheffield, L.T.; and Reeves, T.J.: Electrocardiographic changes in clinically normal older men following near maximal and maximal exercise. Circulation 36:5, 1967.
22. Spangler, R.D.; Horman, M.J.; Miller, S.W., *et al.*: A submaximal exercise electrocardiographic test as a method of detecting occult ischemic heart disease. Am. Heart J. 80:752, 1970.
23. McHenry, P.L.; Fisch, C.; Jordan, J.W.; and Corya, B.R.: Cardiac arrhythmias observed during maximal treadmill exercise testing in clinically normal men. Am. J. Cardiol. 29:331, 1972.
24. Doan, A.E.; Peterson, D.R.; Blackmon, J.R.; and Bruce, R.A.: Myocardial ischemia after maximal exercise in healthy men. Am. Heart J. 69:11, 1965.
25. Goldbarg, A.N.; Moran, J.F.; and Resenekov, L.: Multistage electrocardiographic exercise tests. Am. J. Cardiol. 26:84, 1970.
26. Cumming, G.R.: Yield of ischemic exercise electrocardiograms in relation to exercise intensity in a normal population. Brit. Heart J. 34:919, 1972.
27. Barnard, R.J., and Duncan, H.W.: Heart rate and electrocardiographic responses of fire fighters. JOM 17:247, 1975.

Heart disease in fire fighters Part 2

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Ischemic heart disease in fire fighters with normal coronary arteries

Los Angeles City fire fighters, like fire fighters from most other cities, receive more disability pensions for heart disease than any other illness. Our initial study,[1] conducted on a random sample of Los Angeles City fire fighters, revealed that 10 percent had ischemic ECG responses to near-maximal stress testing. This section presents follow-up data obtained from the men who had ischemic ECG responses during the stress tests.

Methods

The subjects were nine men from a random sample of Los Angeles City fire fighters who had ischemic responses to near-maximal stress testing. An ischemic ECG change was defined as at least 1 mm of horizontal or down slanting depression of the ST segment observed either during exercise or in the five-minute post exercise recovery period.[2]

Following the initial testing when the nine men were identified, one additional visit to the laboratory was scheduled. During this visit, the treadmill stress test was repeated and an additional blood sample obtained for cholesterol, triglyceride, and lipoprotein determinations.*

All of the men were subsequently given the option to undergo cardiac catheterization and angiography. (Six of the men elected to do so.) The catheterizations were done at Cedars of Lebanon Hospital using the Seldinger or Judkin techniques. The study included right and left heart catheterization, selective coronary angiography, left ventricular angiography, coronary sinus catheterization, and atrial pacing.

Results

Table I gives the coronary heart disease (CHD) risk factor data obtained from the nine men. One subject (B.N.) was obese, as indicated by his skinfold fat measurements, and hypertensive. Another subject (D.I.) had elevated triglycerides and a lipoprotein pattern suggesting Type IV hyperlipoproteinemia. Two of the men were smokers at the time of testing. In general, these men with ischemic stress tests had low risk factors for CHD. One subject (S.J.) had ST abnormalities in his resting ECG.

The cardiac catheterization and angiographic studies revealed that only two of the six men had CHD. However, the four with "normal" coronaries had signs of abnormal myocardial function during the atrial pacing studies.

• • • •

Discussion

Our initial observation[1] that 10 percent of a random sample of active Los Angeles City fire fighters had ischemic responses to near-maximal exercise was surprising. Los Angeles City fire fighters are a medically selected population, are generally in good physical condition, and have low risk factors for coronary heart disease. The observations suggest that some job associated stress(es) may be responsible for the relatively high incidence of ischemic stress tests.

The follow-up studies conducted on the nine fire fighters with ischemic stress tests are even more surprising. When the risk factors for coronary heart disease (lipids, hypertension, and smoking) were evaluated, five of the nine men had no abnormal risk factors, while only one subject had two abnormal risk factors.

Six of the nine men elected to undergo angiography and cardiac catheterization. Only two of the six men had significant ($\geq 50\%$ obstruction) coronary artery disease. The 66 percent "false positive" stress tests which were confirmed on two different tests is far higher than that reported by other groups. Several investigators have reported studies in which both stress testing and angiography were conducted on the subjects. Mason, *et al.*,[3] compared the results of exercise stress testing on the ergometer with angiography in 84 patients. Only 12 percent of the individuals with ischemic stress tests had normal coronaries. In a similar study using maximal treadmill testing, Martin and McConahay[4] studied 100 patients and found that only 11 percent of the ischemic responders had normal coronaries. McHenry, *et al.*,[5] reported that treadmill stress testing in 80 patients showed only 5 percent false ischemic responses as evaluated by coronary cinearteriography. Roitmen, *et al.*,[6] using maximal treadmill stress testing in 46 patients, found a specificity of 87 percent for coronary artery disease, while Kaplan, *et al.*,[8] studied 200 patients with ischemic stress tests and found that only 9.5 percent had less than 25 percent narrowing of any major branch of the coronary tree. Froelicher, *et al.*,[7] studied 76 asymptomatic air crewmen with ischemic stress tests and found that 47 percent had insignificant coronary artery obstruction. However, some of their subjects had ECG changes due to vasoregulatory problems. No mention was made of abnormal left ventricle (LV) function in those individuals with normal coronaries.

Atrial pacing studies conducted on the four subjects with normal coronary angiograms provide further insight into the ischemic responses. Two individuals had abnormal lactate metabolism. Subject C.S. had −28 percent lactate extraction with moderate hypertrophy and hypokinesis of unknown etiology. Subject D.L. had 7 percent lactate extraction with a normal functioning ventricle. Moderate LV hypertrophy and hypokinesis was observed in another subject (J.E.), with normal lactate metabolism (24% extraction). Subject S.J. had a normal ventriculogram and normal lactate metabolism during atrial pacing. He did, however, experience angina and ST segment depression. Thus three of the four subjects with normal coronary angiograms had abnormal left ventricle function, which might explain the ischemic stress tests.

Although the abnormal LV function may explain the ischemic stress test, none of the data obtained can explain the

*Blood determinations were done by Laboratory Procedures Division of the Upjohn Co., Woodland Hills, California.

Table 1. Data from fire fighters with ischemic stress tests.

Subject	Age	Blood Pressure (mm Hg)	Skinfold Fat (mm)	Cholesterol (mg%)	Triglycerides (mg%)	Lipoprotein (Electrophoresis)	Smoking
L.S.	45	115/75	144	222	75	normal	No
C.S.	52	128/85	149	171	—	—	No
S.J.	45	110/75	91	210	49	normal	Yes
M.W.	53	110/70	105	174	168	normal	No
D.L.	53	145/90	147	278	200	possible type IV	No
J.E.	59	140/85	153	261	120	normal	Yes
B.J.	54	125/90	99	212	35	normal	No
R.R.	48	125/75	129	192	50	normal	No
B.N.	56	165/100	195	261	105	normal	No

abnormal LV function. All subjects had normal valve function and pressures which were within normal limits. The two subjects with moderate LV hypertrophy were avid handball players, which may explain the hypertrophy. However, hypertrophy resulting from athletic training is not associated with hypokinesis and abnormal LV function.[9]

We realize that angiography is not 100 percent accurate and that we have studied only a small number of subjects. However, the present data suggest that fire fighters have ischemic heart disease and/or abnormal LV function not due to coronary artery disease or any other known cause. The obvious conclusion is that stress(es) associated with the job of fire fighting is having a detrimental effect on the myocardium itself. Myocardial ischemia, necrosis, and even infarction in people with normal coronaries have previously been reported.[10-14] Eliot and Bratt[10] and Likoff, *et al.*,[11] reported myocardial ischemia and necrosis in patients with normal arteriograms but abnormal hemoglobin-oxygen dissociation. Although we did not check for this possibility in our subjects, it seems very unlikely that they were suffering from this abnormality since most of them had very good work capacities during the treadmill test.

Exposure to carbon monoxide, which reduces oxygen delivery to the heart, can cause ischemia and even myocardial infarction.[15,16] This is a hazard to which fire fighters may be exposed.[17] If exposure to carbon monoxide is frequent, it could produce myocardial necrosis and hypokinesis. High levels of catecholamines have also been shown to produce myocardial ischemia and necrosis.[18,19] Our previous studies[10] have reported rapid heart rate acceleration and electrocardiographic changes while responding to the alarm. Near maximal heart rates for prolonged periods and electrocardiographic changes have also been reported during actual fire fighting.[17,20] These observations suggest high sympathetic nervous system activity which may result in the secretion of massive amounts of catecholamines.

Summary

Near maximal stress testing conducted on a group (N-90) of randomly selected Los Angeles City fire fighters (ages 40-59) revealed that 10 percent had ischemic ECG changes. These ischemic ECG responses were confirmed during a second test conducted two to four weeks after the initial test. Coronary heart disease (CHD) risk factor analysis revealed that in general the men were not at high risk for CHD.

Six of the nine men elected to undergo cardiac catheterization and angiography. One patient had severe triple vessel disease and subsequently underwent aorto-coronary bypass surgery. Another had 50 percent obstruction in the circumflex branch of the left coronary, while the other four men had no visible signs of coronary obstruction. The men with "normal" coronaries, however, did show signs of abnormal cardiac function during atrial pacing. One man had cardiac enlargement, hypokinesis, ischemic ECG, and abnormal lactate metabolism. Another had abnormal lactate metabolism and ischemic ECG. A third man had moderate cardiac enlargement with anterior wall hypokinesis. The fourth had ischemic ECG changes with angina but otherwise normal cardiac function. All four of these men had pressures which were within normal limits.

These data show that some fire fighters have ischemic heart disease with patent coronary arteries. This disease may be due to job related factors (*i.e.*, carbon monoxide and other noxious fumes, catecholamines, etc.), which reduce myocardial oxygen supply or greatly increase myocardial oxygen demands.

Heart disease in New York City fire fighters

The data presented here were obtained from the medical records of 1055 fire officers from the New York City Fire Department. These fire fighters underwent a cardiopulmonary stress test administered by Cardiogramics Inc.

Methods

The fire fighters reported to the Cardiogramics Laboratory on an off-duty day and in a fasted condition. They completed a history questionnaire and then had a blood sample drawn for cholesterol analysis.* A standard 12-lead electrocardiogram (ECG) was obtained and resting blood pressure measured prior to the exercise ECG stress test.

A submaximal exercise test was performed on either a treadmill or bicycle ergometer, following the Bruce protocol and guidelines published by the American Heart Association.[21]

Results

The results of the submaximal stress test are outlined in Figure 1. Of the 1055 men tested, 179 (17.0%) had signs of cardiovascular disease. Twenty (1.9%) had abnormal ECGs at rest and were not even given the stress test as recommended by

*Cholesterol analysis was done by Metpath, Hackensack, N.J., using an autoanalyzer.

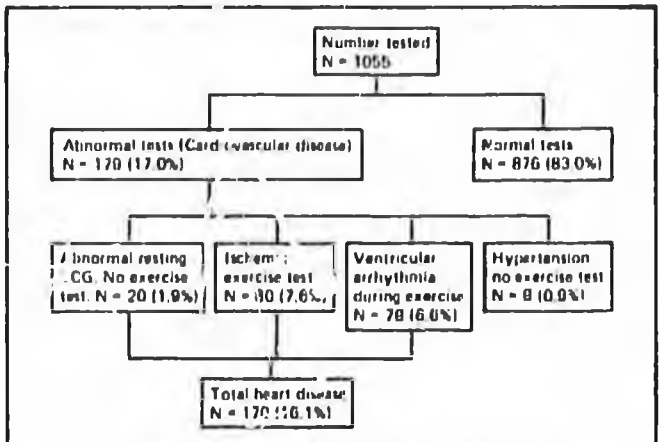

Figure 1. Electrocardiographic stress test results from New York City fire fighters age 40 and over.

Table 2. Coronary heart disease risk factors in New York City fire fighters.

Age Group	Total Men	Cholesterol >260 mg%	Blood Pressure >160/90mmHg	Smoker
40–49	542	86	52	150
50–59	479	86	69	107
≥60	34	3	4	6
Total	1055	175	125	263
%	(100)	(16.6%)	(11.8%)	(24.9%)

the American Heart Association. An additional nine (0.9%) were classified as hypertensive and also were not given the stress test. Of those who took the stress test, 150 (14.2%) had abnormal ECG responses; 80 (7.6%) had ischemic ST segment depression, and 70 (6.6%) had significant ventricular arrhythmias. A total of 170 (16.1%) had ECG signs of heart disease either at rest or during exercise.

Table 2 gives the coronary heart disease risk factor data for various age groups. Of the 1055 men, 175 (16.6%) had cholesterol values greater than 260 mg%, 125 (11.8%) had blood pressure greater than 160/90 mmHg, and 263 (24.9%) were smokers.

Two items on the history questionnaire provided some interesting information about the stress of fire fighting. A total of 442 (41.9%) fire fighters answered yes to the question: "Have you ever gone to the hospital for smoke inhalation?" Only 79 (7.5%) answered no to the question on smoke exposure and breathing problems: "Have you ever received a real shellacking or pasting?" To the question on the frequency, many answered numerous times, and two said 1000.

Discussion

New York City, like Los Angeles, admits fire fighters to the department only after they pass a preliminary medical screening and obtain a high level of physical conditioning. Therefore, one would anticipate finding an incidence of heart disease lower than that of the average population of adult men. Of the New York City fire fighters, 20 (1.9%) had abnormal resting ECGs, and 80 (7.6%) had ischemic ECG responses during the submaximal exercise test, for a total of 100 (9.5%) abnormal tests.

It must be emphasized that these results were obtained with a submaximal exercise test. Bellet and Roman[22] have reported that maximal exercise testing increases the percentage of ischemic responses by 4 percent over that observed with a submaximal test. This would indicate that the true incidence of ischemic heart disease in New York City fire fighters is close to 14 percent. This value of 14 percent is even higher than that observed in Los Angeles City and County fire fighters. This difference may be due in part to the higher incidence of exposure to the actual stress of fire fighting.

In looking at the coronary heart disease risk factors, it can be seen that the New York City fire fighters had a lower rate of abnormal risk factors than the Los Angeles insurance executives, but slightly higher than the Los Angeles fire fighters. Of the New York City fire fighters, 25 percent were smokers, compared to 26 and 32 percent for Los Angeles insurance executives and fire fighters, respectively. Twelve percent of the New York City fire fighters had blood pressure values greater than 160/90, while 25 percent of the Los Angeles insurance executives, but only 2 percent of the Los Angeles fire fighters were hypertensive. Seventeen percent of the New York City fire fighters had cholesterol values greater than 260 mg%, while only 12 percent of the Los Angeles fire fighters, but 18 percent of the Los Angeles insurance executives had values greater than 260 mg%.

These data on New York City fire fighters also support the hypothesis that stress associated with the job of fire fighting is involved in the high incidence of ischemic heart disease.

Note. Portions of this material first appeared in the *Journal of Occupational Medicine*, December 1976, Volume 18, No. 12. The material also previously appeared in *The California Fireman*, published by the California State Firemen's Association, Inc. Used with permission.

References

1. Barnard, R. J.; Gardner, G. W.; Diaco, N. V.; and Kattus, A. A.: Near-maximal ECG stress testing and coronary artery disease risk factor analysis in Los Angeles City fire fighters. *JOM* 17:693, 1975.

2. Kattus, A. A.; Jorgensen, C. R.; Worden, R. E.; and Alvaro, A. B.: S-T segment depression with near-maximal exercise in detection of preclinical coronary heart disease. *Circulation* 44:585, 1971.

3. Mason, R. E.; Likar, I.; Biern, R. O.; and Ross, R. S.: Multiple-lead exercise electrocardiography. Experience with 107 normal subjects and 67 patients with angina pectoris, and comparison with coronary cinearteriography in 84 patients. *Circulation* 36:517, 1967.

4. Martin, C. M., and McConahay, D. R.: Maximal treadmill exercise electrocardiography. Correlations with coronary arteriography and cardiac hemodynamics. *Circulation* 46:956, 1972.

5. McHenry, P. L.; Phillips, J. F.; and Knoebel, S. B.: Correlation of computer-quantitated treadmill exercise electrocardiogram with arteriographic location of coronary artery disease. *Amer. J. Cardiol.* 30:747, 1972.

6. Roitman, D.; Jones, W. B.; and Sheffield, L. T.: Comparison of submaximal exercise ECG test with coronary cineangiocardiogram. *Ann. Int. Med.* 72:641, 1970.

7. Froelicher, V. F.; Yanowitz, F. G.; Thompson, A. J.; and Lester, M. C.: The correlation of coronary angiography and the electrocardiographic response to maximal treadmill testing in 76 asymptomatic men. *Circulation* 48:587, 1973.

8. Kaplan, M. A.; Harris, C. N.; Arrow, W. S., *et al:* Inability of the submaximal treadmill stress test to predict the location of coronary disease. *Circulation* 42:250, 1973.

9. Raskoff, W.; Goldman, S.; and Cohn, K.: The "athletic heart." *Circulation* 52 (Suppl. II): 115, 1975.

10. Eliot, R. S., and Bratt, G.: The paradox of myocardial ischemia and necrosis in young women with normal coronary arteriograms. *Amer. J. Cardiol.* 23:633, 1969.

11. Likoff, W.; Segal, B. L.; and Kasparian, H.: Paradox of normal selective coronary arteriograms in patients considered to have unmistakable coronary disease. *New Engl. J. Med.* 276:1063, 1976.

12. Khan, A. H., and Haywood, L. J.: Myocardial infarction in nine patients with radiologically patent coronary arteries. *New Engl. J. Med.* 291:427, 1974.

13. Proudfit, W. L.; Shirey, E. K.; and Sones, F. M., Jr.: Selective cinecoronary arteriography: correlation with clinical findings in 1000 patients. *Circulation* 33:901, 1966.

14. Boudoulas, H.; Cobb, T. C.; Leighton, R. F.; and Wilt, S. M.: Myocardial lactate production in patients with angina-like chest pain and angiographically normal coronary arteries and left ventricle. *Amer. J. Cardiol.* 34:501, 1974.

15. Rajalakshmi, C.; Chuquilmia, R.; Chawla, K.; and Towne, C.: Myocardial injury as a manifestation of carbon monoxide intoxication. *Circulation* 5 (Suppl. III) 151, 1974.

16. Anderson, B. F.; Allensworth, D. C.; and DeGrant, W. D.: Myocardial toxicity from carbon monoxide poisoning. *Ann. Int. Med.* 67:1172, 1967.

17. Kurt, T. L., and Peters, J. M.: Epidemiology of cardiac risks in fire fighters with Holter electrocardiograms and carbon monoxide sampling. *Circulation* 52 (Suppl. II): 204, 1975.

18. Szakacs, J. E., and Mahlman, B.: Pathologic changes induced by L-norepinephrine. *Amer. J. Cardiol.* 5:619, 1960.

19. Szakacs, J. E., and Cannon, A.: L-norepinephrine myocarditis. *Amer. J. Clin. Pathol.* 30:425, 1958.

20. Barnard, R. J., and Duncan, H. W.: Heart rate and electrocardiographic responses of fire fighters. *JOM* 17:247, 1975.

21. Exercise testing and training of apparently healthy individuals: A handbook for physicians. American Heart Association. NY, 1972.

22. Bellet, S. and Roman, L.: Comparison of the double two-step test and the maximal exercise treadmill test. *Circulation* 36:238, 1967.

Heart disease in fire fighters Part 3

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We stated earlier in this series (see Part 1, *Fire Command*, August 1979) that the most common cause of ischemic heart disease is coronary artery disease, also known as coronary heart disease (CHD), or atherosclerotic heart disease. In this section, we will discuss the cause of coronary heart disease and see how the stress of fire fighting might be a major factor in the high incidence of this disease in fire fighters.

Coronary heart disease and the stress of fire fighting

The exact etiology of coronary heart disease is not known; however, the most widely accepted theory is that damage to the inner lining (endothelial cells) of the artery is the initial stage.[1,2] Once the artery lining has been damaged, cholesterol and other fats enter the artery wall from the blood stream, and atherosclerosis develops. Factors which have been shown to cause damage to the artery linings include high levels of cholesterol, hypertension, smoking, high levels of adrenalin, and high levels of carbon monoxide.[3-11] Of these five factors, two, carbon monoxide and adrenalin, are major concern to the fire fighter. In addition to causing damage to the artery lining, high levels of carbon monoxide and adrenalin have been shown to cause direct damage to the heart muscle.[12-16] These observations may explain the heart muscle damage which Barnard, *et al.*, reported finding in Los Angeles City fire fighters.

In a recent study conducted in conjunction with the Los Angeles City Fire Department, Barnard and Weber[17] observed carbon monoxide (CO) levels as high as 3000 parts per million (ppm). Examples of CO measurements are shown in Table 1. Higher values (4100 ppm) were reported by Kurt and Peters[10] in a study conducted in Boston. The Boston fire fighters who were exposed to these high CO levels had carboxyhemoglobin (COHb) levels in the range of 18 percent.

In the normal environment, CO levels are generally very low (1-10 ppm) with carboxyhemoglobin levels of less than two percent for nonsmokers and 2-4 percent for smokers.[11]

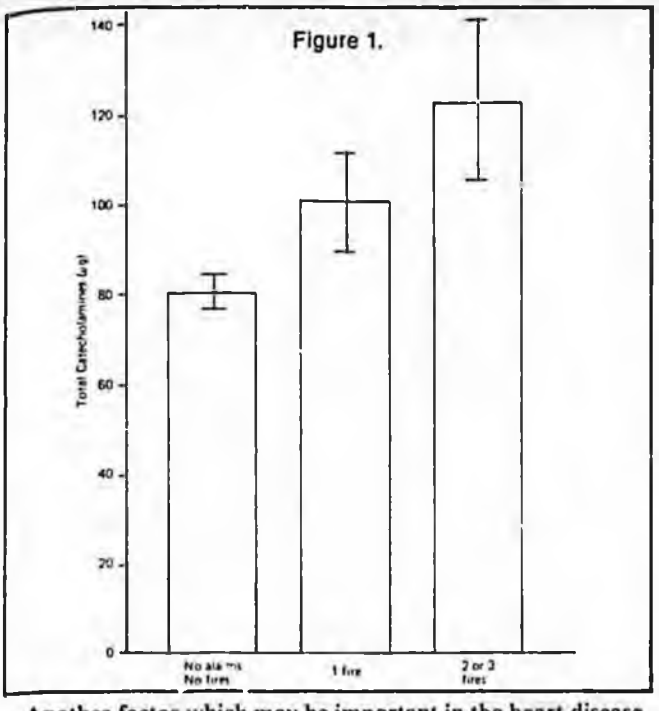
In a study conducted by Gordon and Rogers with the Denver Fire Department,[21] Colorado, it was reported that in nearly two-thirds of the instances of fire fighting a value of 10 percent COHb or higher was reached; in nearly one-fourth of the exposures a COHb value of 20 percent or greater was observed; one individual had a very high COHb level of 44 percent. Gordon and Rogers concluded, "By all criteria available, this exposure to carbon monoxide is both frequent and severe." These investigators also measured elevated levels of serum enzymes (LDH and CPK) and concluded ". . . that exercise alone does not elevate the blood enzymes in the manner, frequency, or degree seen in the exposure to fires." Other investigators[11,21] have also reported elevated COHb levels in fire fighters. De Bias, *et al.*,[16] reported that exposure to CO in levels sufficient to elevate COHb levels to 9 percent significantly increased susceptibility to ventricular fibrillation, which may account for some of the fire fighter deaths that occur on-duty.[11]

While the CO problem in fire fighting has been well documented, little is known about adrenalin levels in fire fighters. Kurt and Peters[10] reported in a pilot study that urinary adrenalin levels in four Boston fire fighters were above normal ranges. Blimkie, *et al.*,[22] reported an 81 percent increase in the heart rate of 15 fire fighters responding to an alarm and a 154 percent increase in plasma or adrenalin levels. These investigators concluded, "In this study, the immediate response of fire fighters to an alarm was one of intense physical arousal which was accompanied by a substantial, but lesser, degree of emotional arousal." In a study that is presently being conducted with Los Angeles City Fire Fighters, Barnard and Duncan[23] have found increases in 24-hour urinary catecholamines with increased fire activity (Fig. 1). To date, unfortunately, no data have been obtained with more than three fires and none during major fires lasting 45 minutes or longer.

Table 1. Carbon monoxide levels in the fire fighting environment

Incident Description	CO ppm	Comments
Foam rubber pillows burning in bathroom	150	front part of apartment, light smoke
	1600	closed bathroom, dense black black smoke
Greater alarm structure fire	200	1st floor
	600	mezzanine, light smoke
	2000 +	2nd floor, fire area
Two-story dwelling fire on first floor living room	900	immediately involved area, light smoke
	1600	2nd floor, uninvolved but heavy smoke
	2000 +	1st floor closet
Two-story dwelling fire on first floor and spread to second floor	300	1st floor involved area
	800	2nd floor uninvolved area
Dwelling area under house involved, rags soaked with flammable liquids	1000	inside house, no fire but heavy smoke
Dwelling service porch involved, carburetor cleaner fluid	1800	partially ventilated room
	3000	unventilated, uninvolved bedroom

Summary:
1. In every instance where CO levels were significant, the smoke was quite heavy and noxious.
2. In some instances where the smoke was heavy and noxious, the CO level was of minimal significance.
3. In two-story structures, the higher concentrations of CO were found on the second levels along with heavy smoke.



Another factor which may be important in the heart disease problem of fire fighters is exposure to other toxic gases such as hydrogen chloride. Polyvinyl chloride (PVC) toxicity in fire fighters is becoming a major problem because numerous household items, electrical wiring, etc., are made of PVC. Dyer and Esch[9] reported that between 1970-1975, 175 Washington, D.C., fire fighters experienced symptoms from PVC toxicity from one to four occasions; one died. Genovesi, *et al.*,[10] reported on 21 Los Angeles City fire fighters who were exposed to dense smoke containing PVC. Nineteen were found to have mild to moderately severe hypoxemia and 13 required hospitalization. All of the fire fighters were using self-contained breathing apparatus while fighting the fire.

Heart rate and ECG responses of fire fighters

A previous study from our laboratory revealed that fire fighters had a high incidence of ischemic responses to a standard near-maximal exercise test, even higher than that observed in a similar age group of insurance underwriters.[11] This observation was surprising because the fire fighters, a medically selected group, were in good physical condition with low blood pressure and cholesterol levels. These findings suggest that ischemic heart disease in fire fighters may be job associated.

Other studies from our laboratory[12,13] showed that performing sudden exercise without the benefit of warm-up can produce an ischemic condition in the heart. Since fire fighters are required to perform sudden exercise without the benefit of warm-up and also are required to work under thermal stress as well as inhalation pollution, the present study was undertaken to observe electrocardiographic and heart rate responses during the fire fighters' normal 24-hour work day.

Methods

The subjects of this study were 35 fire fighters from the Los Angeles City and County Fire Departments. The men ranged in age from 23 to 42 and were all considered in good health without any overt symptoms of heart disease. Their service ranged from one-half to 19 years.

At approximately 0700 hours, the men were connected to Avionics Model 375 Mini Recorders. The electrodes were placed to record the ECG from the manubrium to the V_5 position with the ground at the RV_6 position. The men kept a log book which included the exact time the recorder was started, the time of all alarms, and a description of their activities. The tapes were changed every four hours. Most subjects retired between 2300 and 2400 hours with a new tape in the recorder.

The tapes were transcribed to paper printouts on an Avionics Model 650 Scanner. Analysis of the ECG was made before the alarm sounded, for 30 seconds after the alarm, and then every minute until the time the men returned to the station. Movement artifact prevented accurate analysis of the ECG in many cases; however, heart rate data could be obtained from the QRS complex. To be included in the analysis, a man had to have responded to at least four alarms.

Discussion

The data obtained from these 35 fire fighters show that for the most part they are relatively inactive, but at times are called upon to perform at or near their maximal heart rate for prolonged periods. The heart rate responses observed immediately after the alarm sounded, as well as on the truck approaching a fire, often indicated a state of high anxiety. Why a man's heart rate might increase by only 15 to 20 beats/minute in response to one alarm and then 70 to 80 beats/minute in response to another alarm could not be determined from our data. The fire fighters themselves recognize these different responses but cannot explain them. Some men indicated that they did not think that they experienced anxiety when the alarm sounded, but their heart rate indicated they were anxious on some occasions. From our preliminary data,[11] we suggested that less experienced fire fighters showed a more excited response to the alarm than did more experienced men. However, the present, more complete data did not support this earlier suggestion. In addition, more experienced as well as less experienced fire fighters experienced similar periods of high heart rates during actual fire fighting. No consistent pattern of heart rate increase was observed for any one individual. Although fire fighters knew that a high percentage of box alarms turn out to be false, they still showed an excited response at times.

Summary

Data were obtained from 35 fire fighters responding to 189 alarms. Fifteen to 30 seconds after an alarm, heart rate showed a mean increase of 47 beats/minute (range: 12 to 117 beats/min.). Approximately one minute after the alarm, while on the truck, heart rate still showed a mean increase of 30 beats/minute (range: 1 to 80 beats/min.) above that recorded before the alarm. S-T segment changes were observed in the ECG shortly after the alarm sounded. Upon approaching a fire, heart rates as high as 150 beats/minute were observed before the men got off of the fire truck.

During actual fire fighting, extremely high heart rates were observed for prolonged periods of time. One fire fighter had a mean heart rate of 188 beats/minute for 15 minutes during the initial stages of a structure fire.

The heart rate responses observed immediately after the alarm as well as on the truck approaching a fire indicate that the men experience a state of high anxiety. The extremely high heart rates observed for prolonged periods during fire fighting may also indicate a state of high anxiety coupled with the heavy work performed in a hot environment.

Repeated exposure to states of high anxiety, as well as inhaling pollutants such as carbon monoxide, may be related to the high incidence of ischemic stress tests previously observed in fire fighters.

Note: The preceding section, "Heart rate and ECG responses of fire fighters," is excerpted from an article by the same title, published in the *Journal of Occupational Medicine*, Vol. 17, No. 4, April 1975. Reprints are available by request to: Department of Medicine, UCLA School of Medicine, Los Angeles, CA 90024. Attn.: Dr. Barnard.

Conclusion

The purpose of this report was to investigate the heart disease problem in fire fighters and to see if any of the information known about the stress of fire fighting could be related to heart disease in fire fighters.

Statistics published by the U.S. Department of Health,

Education, and Welfare show that the death rate for all cardiovascular diseases, and in particular atherosclerotic heart disease (coronary heart disease), is higher in fire fighters than in police officers, longshoremen, lumbermen, construction workers, smelters, and furnacemen. In fact, the death rates for the age groups 55–59 and 60–64 years were twice as high in fire fighters than the average for all of the other groups studied.

The second line of evidence which shows that fire fighters have an abnormally high incidence of heart disease comes from the electrocardiographic stress test studies. Published reports from both the Los Angeles City and County Fire Departments revealed a higher percentage of abnormal ECG responses than values reported for insurance executives, sheriffs, lifeguards, police officers, and airline pilots and controllers. The data presented in this report on the New York City Fire Department shows similar results. Since fire fighters are a medically selected group, the high percentage of abnormal ECG stress tests is indeed indicative of an abnormally high incidence of heart disease.

Although the exact etiology of coronary heart disease is not known, experimental studies have shown that damage to the endothelial lining of arteries can lead to the premature development of atherosclerosis. Several factors, including elevated cholesterol, hypertension, smoking, carbon monoxide, and high levels of adrenalin, have been shown to cause damage to the endothelial cells. Of these, carbon monoxide has been shown to be a major factor in fire fighting. Measurements made in both the fire environment and the fire fighters themselves strongly suggest that carbon monoxide may be a major factor in the abnormally high incidence of heart disease in fire fighters.

Although not extensive, the information on adrenalin suggests that an emotional response to fire fighting results in the elaboration of higher than normal amounts of adrenalin.

Heart rate and electrocardiographic data obtained from men during actual fire fighting show that their hearts are operating at near maximal to maximal limits, sometimes for prolonged periods. Any factors that would limit oxygen delivery, *i.e.*, CO, PVC, or factors that would significantly increase oxygen demands, *i.e.*, adrenalin, could cause direct damage to the heart muscle. Heart muscle damage has been reported in some Los Angeles City fire fighters.

In conclusion, the data overwhelmingly support the contention that fire fighters do indeed have an abnormally high incidence of heart disease. The information available on the stress associated with the job of fire fighting strongly suggests a causal relationship with this abnormally high incidence of heart disease.

References

1. Constantinides, P.: Endothelial injury in the pathogenesis of arteriosclerosis, In Advances in Experimental Medicine and Biology, Vol. 16A, S. Wolf, ed. Plenum Press, New York, p. 185, 1971.
2. Ross, R., and Glomset, J. A.: The pathogenesis of atherosclerosis. *New Engl. J. Med.* 295:369, 1976.
3. Shimamoto, T.; Kobayshi, M.; and Numano, F.: Infiltration of globulin, fibrinogen and lipoprotein into blood vessel wall by atherogenic stress visualized by immunofluorescence. *Proc. Japan Acad.* 48:336, 1972.
4. Thomsen, H. K., and Kjeldsen, K.: Aortic intimal injury in rabbits. An evaluation of a threshold limit. *Arch. Environ. Health* 30:604, 1975.
5. Kjeldsen, K.; Thomsen, H. K.; and Astrup, P.: Effects of carbon monoxide on myocardium. Ultrastructure changes in rabbits after moderate chronic exposure. *Circ. Res.* 34:339, 1974.
6. Thomsen, H. K.: Carbon monoxide-induced atherosclerosis in primates. An electron microscopic study on the coronary arteries of *Macaca irus* monkeys. *Atherosclerosis* 20:233, 1974.
7. Wanstrup, J.; Kjeldsen, K.; and Astrup, P.: Acceleration of spontaneous intimalsubintimal changes in rabbit aorta by a prolonged moderate carbon monoxide exposu. *Acta. Path. Microbiol. Scand.* 75:353, 1969.
8. Webster, W. S.; Clarkson, T. B.; and Lofland, H. B: Carbon monoxide exaggerated atherosclerosis in the squirrel monkey. *Exp. Mol. Path.* 13:36, 1970.
9. Astrup, P.; Kjeldsen, K.; and Wanstrup, J.: Enhancing influence of carbon monoxide on the development of atheromatosis in cholesterol-fed rabbits. *J. Atheroscler. Res.* 7:343, 1967.
10. Armitage, A. K.; Davies, R. F.; and Turner, D. M.: The effects of carbon monoxide on the development of atherosclerosis in the white carneau pigeon. *Atherosclerosis* 23: 333, 1976.
11. Birnstingl, M.; Hawkins, L.; and McEwen, T.: Experimental atherosclerosis during chronic exposure to carbon monoxide. *Europ. Surg. Res.* 2:92, 1970.
12. Bhattacharya, S. K.; Chakravarti, R. N.; and Wahi, P. L.: Noradrenaline-induced myocardial infarction in monkeys given an atherogenic diet. *Atherosclerosis* 20:241, 1974.
13. Anderson, R. F.; Allenworth, D. C.; and deGroot, W. J.: Myocardial toxicity from carbon monoxide poisoning. *Ann. Int. Med.* 67:1172, 1967.
14. Markey, Z., and Piejko, M.: Circulatory failure in acute carbon monoxide poisoning. *Forensic Sci.* 1:419, 1972.
15. Rajalakshmi, C.; Chugulmia, R.; Chaula, K.; and Towne, C.: Myocardial injury as a manifestation of carbon monoxide intoxication. *Circulation* 50 (Suppl. III):151, 1974.
16. Szakacs, J. E., and Mahlman, B.: Pathologic changes induced by L-norepinephrine. *Am. J. Cardiol.* 5:619, 1960.
17. Szakacs, J. E. and Cannon, A.: L-norepinephrine myocarditis. *Am. J. Clin. Pathol.* 30:425, 1958.
18. Haft, J. I.; Gershengorn, K.; Kranz, P. D.; and Oestreicher, R.: Protection against epinephrine-induced myocardial necrosis by drugs that inhibit platelet aggregation. *Am. J. Cardiol.* 30:838, 1972.
19. Barnard, R. J., and Weber, J.: Carbon monoxide levels in the fire fighting environment. (In Preparation, 1978.)
20. Kurt, T. L., and Peters, J. M.: Epidemiology of cardiac risks in fire fighters with Holter electrocardiograms and carbon monoxide sampling. *Circulation* 52: (Suppl II) II-204, 1975.
21. Carbon monoxide. National Academy of Sciences, Washington, D.C. 1977.
22. Gordon, G. S., and Rogers, R. L.: Project carbon monoxide. Merkle Press, Inc., Washington, D.C., 1969.
23. Radford, E. P., and Levine, M. S.: Occupational exposure to carbon monoxide in Baltimore fire fighters. *J. Occup. Med.* 18:628, 1976.
24. Sammons, J. H., and Coleman, R. L.: Firefighters occupational exposure to carbon monoxide. *J Occup. Med.* 16:543, 1974.
25. Stewart, R. D.; Stewart, R. C.; Stamm, W.; and Seelan, R. P.: Rapid estimation of carboxyhemoglobin level in fire fighters. *JAMA* 235:390, 1976.
26. DeBias, D. A.; Banerjee, C. M.; Birkhead, N. C.; Green, C. H.; Scott, S. D.; and Harrer, W. V: Effects of carbon monoxide inhalation on ventricular fibrillation: *Arch. Environ. Health* 31:42, 1976.
27. Blinkie, C. J. R.; Rechnitzer, P. A.; and Cunningham, D. A.: Heart rate and catecholamine responses of fire fighters to an alarm. *Canad. J. Appl. Sport. Sci.* 2:153, 1977.
28. Barnard, R. J., and Duncan, H. W.: Catecholamine levels in fire fighters. (In Preparation.)
29. Dyer, R. E., and Esch, V. H.: Polyvinyl chloride toxicity in fires. Hydrogen chloride toxicity in fire fighters. *JAMA* 235:393, 1976.
30. Genovesi, M.; Tashkin, D. P.; Chopra, S.; Morgan, M.; McElroy, C.; and Coulson, A.: Transient hypoxemia in firemen following inhalation of smoke. *Chest* 71:441, 1977.
31. "Fire Fighter Mortality Report." Int. Assoc. of Fire Fighters, Washington, D.C., May 1976.
32. Gardner, G. W.; Barnard, R. J.; Diaco, N. V.; and Kattus, A. A.: Near-maximal ECG stress testing and CHD risk factor analysis in Los Angeles City Firemen. *Med. Sci. Sports* 6:71, 1974.
33. Barnard, R. J.; Gardner, G. W.; Diaco, N. V. *et al.*: Cardiovascular responses to sudden strenuous exercise — heart rate, blood pressure, and ECG, *J Appl. Physiol.* 34:833. 1973.
34. Barnard, R. J.; MacAlpin, R. N.; Kattus, A. A.; and Buckberg, G. D.: Ischemic response to sudden strenuous exercise in healthy men. *Circulation* 48:936, 1973.
35. Barnard, R. J.: Electrocardiographic and heart rate responses to sudden strenuous exercise and to the fire alarm. In: Survival in the Fire Fighting Profession, p. 26, Int. Assoc. of Fire Fighters, Washington, D.C., 1974.

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The Lubbock fatalities

Information supplied by the
Fire Investigations Department
National Fire Protection Association

In Lubbock, Texas, on 25 March 1979, an early morning fire believed to be electrical in origin caused moderate damage to a restaurant undergoing renovation. The one-story building of ordinary construction was not equipped with automatic sprinklers, fire detectors, or an alarm system.

An employee of a nearby motel reported the fire at 4:30 a.m., and the Lubbock Fire Department's first unit responded at 4:32 a.m. Fire fighters found the kitchen area heavily involved and used 1 ½-inch hose lines, controlling the fire within 20 minutes. During overhaul, three fire fighters wearing self-contained breathing apparatus (SCBA) entered a rear dining room to check for fire extension. They apparently became lost because of limited visibility and eventually succumbed to carbon monoxide (CO) intoxication.

Background

The one-story restaurant at 711 34th Street in Lubbock, Texas, was of ordinary construction with concrete block walls and a varied roof system consisting of both wood and steel bar joists supporting a wood deck and built-up roof. The irregularly shaped building measured approximately 144 feet long by 70 feet wide (see figure).

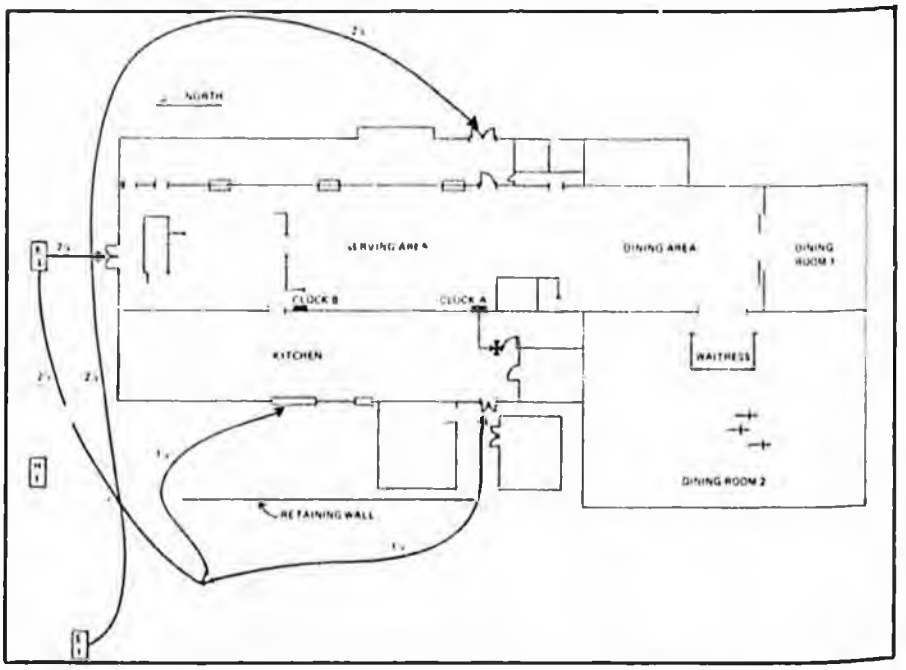
The interior had wood frame partitions, with interior finish consisting largely of ¼-inch plywood veneer paneling in the dining areas and painted concrete block in the kitchen. The kitchen ceiling was of 24-by-48-inch fiberglass reinforced plastic panels suspended in metal "T" channels. Ceilings in the serving and dining areas were of sprayed noncombustible acoustical material on wire lath. Beneath this in the serving area was a suspended ceiling of 24-by-48-inch acoustical panels in steel "T" channels.

The building was not equipped with automatic sprinklers, fire detectors, or an alarm system. Because it was undergoing renovation at the time of the fire, the interior was cluttered with equipment and building materials. The south end of the building was windowless and had no exterior door.

Lubbock covers a 90-square-mile area with a population of about 180,000. The fully-paid Lubbock Fire Department has 255 career fire fighters who operate 12 engine companies, 3 truck (ladder) companies, 4 booster companies, and 1 manpower squad.

Departmental policy required the use of self-contained breathing apparatus (SCBA). Each fire fighter received breathing apparatus training in recruit school with additional training conducted at the company level. Companies were furnished with either demand or pressure demand SCBA. There was no policy requiring operation of the pressure demand breathing apparatus in the pressure demand mode, but in training sessions fire fighters were urged to so operate the SCBA when they entered toxic atmospheres. Each shift checked the SCBA, and brief field inspections were made, but there was no general preventive maintenance check. At the time of the incident, field inspections consisted of checking air tank pressure, facepiece seal, and regulator function, by pressurizing and depressurizing the regulator. Fire department members trained by a breathing apparatus manufacturer's representative serviced malfunctioning units. A compressor and cascade system, with a recharging system configured with a mechanical filter, was used to refill air cylinders. In general, no periodic testing of air quality was conducted.

Weather conditions at the time of the incident were: temperature 42°F, barometric pressure 29.92, humidity 57 percent, wind 7 mph



from the south, and visibility 15 miles and clear.

The fire

An employee of a nearby motel saw smoke coming from the restaurant and notified the Lubbock Fire Department at 4:30 a.m. The response consisted of a chief and 14 fire fighters, assigned to Engine 1 (3 personnel), Hose 1 (1), Snorkel 1 (2), Engine 3 (4), and the Squad (4).[1] The first unit arrived at 4:32 a.m., and fire fighters found the kitchen area on the west side of the building heavily involved. In the initial attack, they advanced two 1½-inch hose lines from Engine 3 through the kitchen door and window. Engine 1 and Engine 3 also advanced two 2½-inch lines through the north and east doors, but these lines were not used. Cross ventilation was accomplished by opening doors and using smoke ejectors. The dining areas in the south end of the building had no windows or exterior doors and were not vented.

By 4:50 a.m., the fire was knocked down. Overhaul began about ten minutes later, and during it fire fighters discovered that the fire had extended to the ceiling above the serving area. They pulled portions of the ceiling and soon extinguished the fire. Both the suspended and acoustical ceilings above the serving area along the kitchen wall had to be pulled. Overhaul continued for over one hour as fire fighters completed various tasks and checked the building for additional fire extension.

It is believed that at approximately 5:15 a.m. three fire fighters entered the rear (south end) area of the building, presumably to check for fire extension. Apparently, they entered the area independently, without instructions from officers. They did not advance a hose line. The three were not seen for some time during overhaul and take-up operations. Fire fighter 1 was from Engine 1, fire fighter 2 was from the Squad, and fire fighter 3 was the driver of Hose 1 (see Table 1).

At about 6:40 a.m., the three fire fighters were found lying within five feet of one another in Dining Room 2 (see figure). They were removed from the building, and cardiopulmonary resuscitation was started immediately. At the hospitals to which they were transported, they were pronounced dead.

During the rescue effort, other smoke inhalation injuries occurred. Nine fire fighters were transported to hospitals for precautionary treatment, and two were held overnight.

Table 1

	Fire Fighter 1	Fire Fighter 2	Fire Fighter 3
Age	25	30	34
Company	Engine 1	Squad	Hose 1
Type breathing apparatus	Pressure-demand	Pressure-demand	Demand
Condition of apparatus when found			
Facepiece on	yes	yes	yes
Facepiece hose attached to regulator	no	no	yes
Air supply remaining	0	0	Approx. 800 psig
Blood gases			
CO Hb	51.5%	43.3%	46.8%
O_2 Hb	12.2%	13.8%	22.4%
NIOSH assigned number[a]	1	2	3
NIOSH leakage data[b]			
NaCl penetration	14% (d)[c] <0.002% (pd)[d]	0.0039% (d) <0.002% (pd)	7.77% (d)
Isoamyl acetate penetration	large leak	test not performed	very large leak (d)

[a] See Page 2, "Test of Self-Contained Breathing Apparatus Received From Lubbock, Texas Fire Department, June 1979," NIOSH.
[b] NIOSH, Page 9, Table 3.
[c] Demand mode.
[d] Pressure demand mode.

Damage

The fire was confined to the kitchen area, which was heavily damaged, and to the area of extension in the combustible concealed space above the serving area. Heavy smoke permeated the entire building, and the rear dining rooms received smoke damage.

Analysis

The fire is believed to have originated in an electrical panel at the south end of the kitchen. Clock A (see figure), which was plugged into a circuit fed by this electrical panel, stopped at 11:58 p.m. The fire probably smoldered for a long time before becoming free-burning. Clock B, located at the north end of the kitchen, stopped at 4:23 a.m., a time which roughly coincides with the fire's discovery.

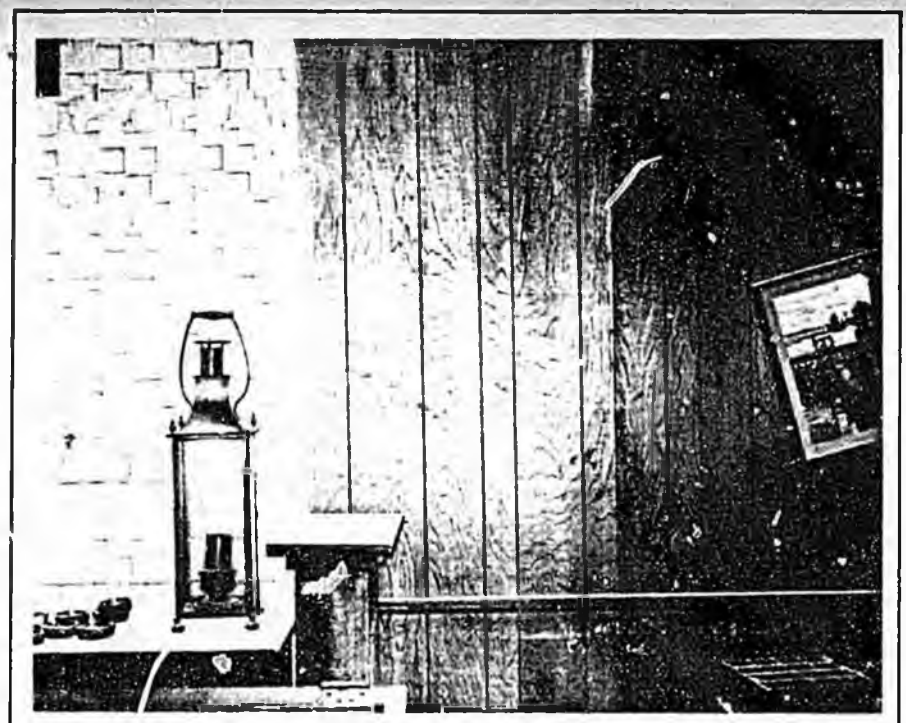
Physical evidence indicates that the fire developed in the area of the circuit breaker panel, which was mounted on wood studs and recessed in the kitchen's wood panel wall. The fire spread within the combustible concealed space and exposed the wood joists above the suspended ceiling. In general, fire exposure to the kitchen was from the concealed space above the suspended ceiling.

The three fire fighters who are believed to have entered the dining room area at 5:15 a.m. to check for fire extension were isolated from the rest of the fire fighting forces. They were not missed until about 6:30 a.m. Reports indicate that even at 6:40 a.m., when the victims were found, a heavy smoke condition existed in Dining Room 2. As noted, Dining Room 2 was windowless and was not vented.

Fire fighters 1 and 2 were involved in fire fighting and overhaul. When they were found, their air supply had been depleted. Fire fighter 3 is believed to have been wearing breathing apparatus not used during initial operations. He reportedly donned breathing apparatus from Hose 1 and joined the others some time after the fire was knocked down (4:50 a.m.).

Examination of Dining Room 2 revealed handprints and scratches from breathing apparatus on the walls and footprints in the soot on the floor (see photo). By analyzing the footprints, handprints, and other items, it was possible to trace

[1] Hose 1 runs with Engine 1 as a two-piece engine company or with the snorkel as a hose tender. Normally, Hose 1 lays lines from the fire to the water supply. The squad is a manpower unit which responds to all structural fires.



These handprints of one of the victims were on the north wall of Dining Room 2. By examining footprints, handprints, and other marks, it was possible to trace some of the three fire fighters' movements in the room. (NFPA photo.)

some of the three fire fighters' movements. They were apparently checking for fire extension when they became lost in limited visibility caused by heavy smoke. Handprints and marks on the walls, as well as handprints on tables, indicated that at least one fire fighter attempted to follow the walls to find a way out. The prints further indicated that at least one fire fighter did find his way out of Dining Room 2, but reentered it when he continued to follow the waitress station wall.

All three victims wore breathing apparatus: Fire fighters 1 and 2 wore pressure-demand breathing apparatus, while fire fighter 3 wore demand-type apparatus. Fire fighters 1 and 2 were found with their breathing apparatus hoses detached from the regulators and their air cylinders empty. Fire fighter 3, who had donned a fresh SCBA before entering the building, was found with breathing apparatus intact and about 800 psig of air remaining in the air cylinder.

Autopsy results indicate the cause of death for all three victims was "acute hypoxia due to acute smoke inhalation and carbon monoxide intoxication." Blood analyses indicated carboxyhemoglobin contents of 43.3 percent, 46.8 percent, and 51.5 percent (see Table 1). The reports do not indicate the presence of burns. In each case, the reports note that "severe pulmonary edema is suggestive of irritation by smoke in addition to carbon monoxide intoxication."

The Lubbock Fire Department submitted the breathing apparatus to the National Institute for Occupational Safety and Health (NIOSH) for testing. The NIOSH report gives a complete description of the breathing apparatus, as well as results of physical examination and performance testing.[1] The NIOSH observations were limited to the condition of the self-contained breathing apparatus as received; the exact condition at the time of the fire could not be documented.

The NIOSH report identifies several significant performance problems and states that the assemblies received differed from the applicable Bureau of Mines (BOM) and NIOSH assemblies. The report also notes that each SCBA had missing, damaged, or defective parts when compared to BOM and NIOSH approval specifications. NIOSH concludes that the SCBA do not meet the application requirements of BOM and NIOSH specifications.

[1] "Tests of Self-Contained Breathing Apparatus Received from Lubbock, Texas, Fire Department, June 1979," Division of Safety Research, National Institute for Occupational Safety and Health, Center for Disease Control, Dept. of Health, Education and Welfare.

The test results indicated significant performance problems. Of particular note were the holes in the diaphragms of SCBA worn by fire fighters 1 and 3 (labeled NIOSH No. 1 and NIOSH No. 3; see Table 1). Significant leakage occurred during tests, allowing outside air to penetrate the diaphragm and the facepiece. With SCBA in the demand mode, the NaCl penetration test method allowed leakage of 14 percent and 7.77 percent respectively in NIOSH No. 1 and NIOSH No. 3. The report contains these observations:

- NIOSH No. 1 — Diaphragm has ½-inch-by-⅛-inch hole.
- NIOSH No. 3 — Diaphragm has 3-inch-by-1/16-inch hole and is pulled away from retaining ring.

NIOSH made the following general observations about the SCBA:

1. Apparatus and regulator with damaged or displaced diaphragms, *i.e.*, NIOSH numbers 1, 3, and 4 (with diaphragm in envelope), showed excessive penetration when operated in the demand mode. (Ed. note: NIOSH No. 4 was an extra regulator not directly involved in the incident.)
2. All apparatus had airflow less than the 200 lpm required at full cylinder pressure.
3. Apparatus with dirt in facepiece, *i.e.* NIOSH numbers 1 and 2, showed excessive exhalation resistance.
4. Excessive inhalation resistance of NIOSH numbers 1 and 2 indicated possible regulator interior misadjustment, dirt, or damage.
5. Operation of remaining service time indicators was outside MSHA/NIOSH specifications on NIOSH numbers 1 and 4, indicating misadjusted, dirty, or damaged alarms.

In addition to NIOSH testing, air samples from fire fighter 3's air cylinder were tested at an independent laboratory to ensure the quality of the air in the cylinders. Initial testing was done with a threshold of 0.005 percent (5000 ppm) carbon monoxide. The results of these tests were inconclusive, *i.e.*, there was less than 0.005 percent (5000 ppm) carbon monoxide present, but this threshold test level was well above acceptable test levels for health and

safety purposes. ANSI Z88.5-1973, *Practices for Respiratory Protection for the Fire Service,* recommends a maximum level of carbon monoxide of 0.00002 percent (20 ppm).[3,4] Consequently, the air was retested with a threshold of 0.00001 percent (10 ppm). Test results were negative; *i.e.,* the air contained less than 0.00001 percent (10 ppm) carbon monoxide.

Fire fighters 1 and 2 appear to have exhausted their air supplies, and they were found with their facepieces on and their hoses detached from their regulators. This fatality scenario involving fire fighters trapped or disoriented in structures and exhausting their air supplies has been documented in other cases.[5,6,7]

Fire fighter 3 was found with his facepiece on, the hose connected to the regulator, and 800 psig air supply remaining. In this case, the 7 percent leakage reported by NIOSH indicates that fire fighter 3 was probably affected by CO from the room environment while wearing the demand-type breathing apparatus. This is substantiated by both the results of air quality tests, indicating no CO present in fire fighter 3's air supply (to a threshold of 10 ppm), and the autopsy data. Operating in the demand mode, the leakage rate in fire fighter 1's pressure-demand SCBA was 14 percent. If fire fighter 1 used the demand mode, he too could have been affected by CO from the room environment while wearing the breathing apparatus; however, the data are inconclusive.

In this incident, several factors are related to the fatalities. Among them are the lack of ventilation of the room in which the fire fighters died, the apparent limited supervision and control of fire fighters on the fireground (since the fire fighters were not missed for over one hour), and the condition of the self-contained breathing apparatus.

The lack of ventilation, either mechanical or natural draft, in the rear dining room allowed a condition of extremely poor visibility and probable high levels of toxic products of combustion to exist long into the fire incident. Dining Room 2 was a windowless area with no exterior doors, an arrangement which contributed to the nonvented condition. Additionally, the focus of fire fighting and overhaul operations was in the kitchen and serving areas, and ventilation measures were limited to those areas.

The lack of exterior doors and windows in Dining Room 2 eliminated any possible alternate escape routes for the three trapped fire fighters.

As stated, the fire fighters were not missed from the fireground for well over one hour. They apparently entered Dining Room 2 without specific instructions to do so. Unfortunately, officers on the fireground were unaware that the three fire fighters were in trouble in Dining Room 2.

The condition of the self-contained breathing apparatus, most notably the leakage through the diaphragms, in at least one case would have contributed to the effects of carbon monoxide intoxication. As previously discussed, this factor can be associated with fire fighter 3. Breathing apparatus leakage is a possible factor in the death of fire fighter 1, provided the apparatus was operated in the demand mode. Protection factors of up to 50,000 (0.00002 percent leakage) are possible with properly designed, manufactured, maintained, and used positive pressure breathing apparatus.[8] The 7 percent leakage of fire fighter 3's SCBA resulted in a protection factor of about 14 (100 percent leakage results in a protection factor of 1), and the 14 percent leakage of fire fighter 1's SCBA, assuming demand mode operation, could have resulted in a protection factor as low as 7.

Acknowledgments

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The facts and conclusions in this report are the NFPA's and do not necessarily represent the views of the NBS or the USFA. The report is based on data gathered by John A. Sharry of Code Consultants, Inc., St. Louis, Missouri, under contract to the NFPA Fire Investigations Department. The cooperation and assistance of the Lubbock Fire Department's Chief Thomas Foster and Deputy Chief Horace Anglin are acknowledged and greatly appreciated. The technical review and assistance of A. Elwood Willey, Director, NFPA Fire Information and Systems Division, and David P. Demers, NFPA Fire Analysis Specialist, are also acknowledged.

[3] "Practice for Respiratory Protection for the Fire Service," American National Standard Z88.5-1973, American National Standards Institute, Inc.

[4] "Commodity Specification for Air," American National Standard Z86.1-1973, Compressed Gas Association, Inc., New York, New York.

[5] Washburn, Arthur, and Harlow, David, "United States fire fighter deaths in the line of duty during 1978," *Fire Command,* May 1977.

[6] Demers, David P., *Fire in Syracuse — Four Fire Fighters Die,* Boston: National Fire Protection Association, 1979.

[7] Investigation Report — "Discotheque Fire, Edmonton, Alberta — August 15, 1976." Investigation conducted by NFPA under contract to the Department of Commerce, jointly funded by the U.S. Fire Administration, the National Bureau of Standards, and the National Fire Protection Association.

[8] Hyatt, Edwin C., "Respirator Protection Factors," Los Alamos Scientific Laboratory report, LA-6084-MS, September 1975.

AMERICA BURNING

The Report of
The National Commission on
Fire Prevention and Control

9

THE HAZARDS CREATED THROUGH MATERIALS

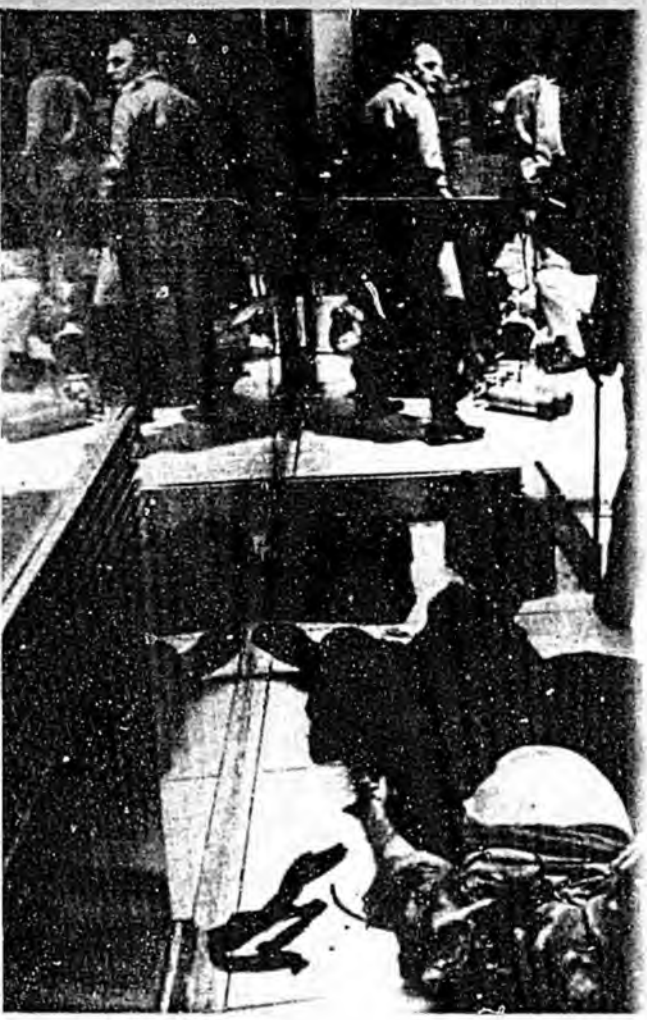
The dazzling terminal buildings at New York's John F. Kennedy Airport are virtually a museum of contemporary architecture. But one of those buildings has demonstrated that man's monuments to his technological genius can turn on him with a vengeance, at the mere touch of a flame.

The new west wing of the British Overseas Airways Corporation building at Kennedy International had not yet been opened to the public when, on August 26, 1970, it caught fire—probably at the hands of an arsonist. Swiftly, flames moved from one seat to the next along the 330-foot length of the wing. Gases from the incomplete combustion of the seats gathered in clouds along the ceiling. When flames approached the clouds, the gases ignited explosively, spreading the fire and igniting other groups of seats. The explosions knocked out the terminal's huge glass windows. As the ceiling melted, combustible liquid dripped toward the floor, further spreading the fire. In the end, all 600 seats in the wing were consumed. Damages totaled $2 million. The seats, which played the predominant role in spreading the fire, were like those in many airline terminals: layers of plastic and rubber foam covered by plastic upholstery material.

No lives were lost in the BOAC terminal fire. But 3 months later, a synthetic material was implicated in a fire that killed 145 teenagers. It happened in a door-locked dance hall in St. Laurent-du-Pont, France, that had been lavishly sprayed with a plastic foam to give the appearance of a cave. The fire raged furiously within seconds after it began, leaping "like a red panther in a small cage," in the words of one survivor.

By no means do synthetics stand alone as hazardous materials. A frame house can be a tinderbox. Restaurants decorated with natural materials, basements full of old newspapers, and warehouses storing lumber or paper products provide the fuel for major fires. Inadequately protected structural elements of steel or concrete still collapse if a fire is intense enough. Burning silk and wool release deadly quantities of carbon monoxide and cyanide gas—and these and many other natural materials ignite at lower temperatures than many synthetics do. Plastics manufacturers contend that synthetics based on carbon, hydrogen, and oxygen exclusively are generally no more toxic, when burned, than natural materials. On the other hand, other synthetics containing sulfur and the halogens are not so innocuous.

Although plastics production has doubled in the



In the modern environment of synthetic material

, gases have become increasingly important hazards.

past 7 years, it is only about one-tenth t
wood, paper, and associated products. Th
tribution of plastics to the fuel load in bui
especially older buildings where fires occu
frequently, is therefore certainly well un
percent. But their use is increasing. Wool r
giving way to synthetic fibers, wooden des
to plastics made to look like wood, glass li
diffusers to clear plastic panels. There is ha
use to which "classical" materials have be
that has not been challenged by synt
Clearly, the advantages which plastics o
consumers and manufacturers are many, an
tics will fill an increasingly large proportion
built environment.

What makes plastics relevant to our dis
of materials is not only that many of then
introduced hazards previously uncommo
that they are sold and used without ad

tion to the special fire hazards they present.
major investigation of the fire problem of
plastics by the Federal Trade Commission
highlighted a form of misleading representa-
of the combustion behavior of certain plastics.

to Die in a Fire

people, when they think of fire as a killer,
of flames. Those who have set fire safety
ards for materials have emphasized flame
nce. Yet, in a list of the five ways in which
an kill, when arranged in the listing impor-
, flames rank last.[1]

phyxiation. Fire consumes oxygen from the
unding atmosphere, thus reducing its con-
tion. If the oxygen concentration falls below

his ranking and much of the following discussion is
Irving N. Einhorn, director of the Flammability Re-
Center, University of Utah.

17 percent, thinking may be an effort and coordination difficult. Below 16 percent, attempts to escape the fire may be ineffective or irrational, wasting vital seconds. With further drops, a person loses his muscular coordination for skilled movements, and muscular effort leads rapidly to fatigue. His breathing ceases when the oxygen content falls below 6 percent. At normal temperatures, he would be dead in 6 to 8 minutes.

Attack by superheated air or gases. With temperatures above 300° F., loss of consciousness or death can occur within several minutes. In addition, hot smoke with a high moisture content is a special danger since it destroys tissues deep in the lungs by burning.

Smoke. Inhalation of smoke—or, more correctly, of the products of incomplete combustion—kills people who suffer no skin burns at all. In addition to carrying toxic products, such as carbon monoxide and hydrogen cyanide, thick smoke may be laden with organic irritants, such as acetic acid and formaldehyde. In the early stages of a fire, the irritants, which attack the mucous membranes of the respiratory tract, are often the more important danger. Smoke often blocks the visibility of exits.

Toxic products. Many toxic components of smoke are responsible for the damage done—including oxides of nitrogen, aldehydes, hydrogen cyanide, sulfur dioxide, and ammonia, to name only a few. There is ample evidence that the hazard of two or more toxic gases is greater than the sum of the hazards of each. Moreover, low oxygen and high temperatures increase the toxic effects. In addition to toxic gases that attack the lungs, there are irritants that attack the eyes with blinding effect, preventing escape. Some fire gases dull the senses of the victim or his awareness of injury.

Flames. Since the aforementioned factors can debilitate, confuse, blind, or kill without warning, the person who goes to sleep confident that advancing flames will provide sufficient warning for escape may be taking a fatal gamble.

Until such time as all five of these hazards have been well-studied and controlled by materials standards, too little will have been done to control the built environment and thus reduce the gamble Americans take in their daily lives.

Ironically, efforts to make materials fire-retardant—that is, with less tendency to ignite or spread flames—may have increased the life hazard, since the incomplete combustion of many materials treated to increase fire retardancy results in heavy smoke and toxic gases. The technology of fire-retardance is often unsatisfactory in other respects: The additives are generally costly, can reduce the strength and weather resistance of the material to which they are applied, and often lose their effectiveness through washing or prolonged exposure to the elements.

Where There's Smoke, There's Damage

That concern about flames alone is insufficient is pointed up by the ample evidence that smoke and toxic gases are powerful forces of destruction. Smoke from restaurant fires renders uncontainered food unusable; fabrics permeated by smoke can be altered beyond use even after cleaning. And a little smoke can go a long way: A department store recently lost $100,000 of its merchandise and 3 days' business for cleanup—all because of smoke that seeped through walls from an adjoining building on fire.

Again, efforts to make materials flame-resistant have not always been beneficial. The sooty smoke given off by many of these materials leaves a thick, black coating on whatever it touches. Moreover, the chemical compounds added to reduce combustibility often contain halogens (bromine, chlorine, and fluorine) which are corrosive and toxic.

Why Be Half Safe?

According to the Society of Plastics Industry, Inc., manufacturers of plastics spend $40 million annually on research to improve the fire safety of their products. That organization has also been helpful to manufacturers, in 1964, a fire safety standard was instrumental in setting flammability standards for cellular plastics. Fire resistance or fire classification standards for all sorts of construction materials are set by such organizations as the American Society for Testing and Materials and the National Fire Protection Association. Building codes incorporate many of these standards. Underwriters' Laboratories, Factory Mutual Research Corp., and other organizations test materials to see that they comply with such standards.

Yet, for all these efforts, the American public remains inadequately protected from combustion hazards in their midst.

Smoke and toxic gases have been underrated hazards. Recognition of these hazards has come belatedly, with the result that there is still little understanding, and hence little quantifiable knowledge, of the destructive effects of smoke and toxic gases.

As a result, *there are no nationally recognized test methods for measuring smoke production (both rate and amount)*. The American Society for Testing and Materials does have a tunnel test which measures the density of smoke produced. Development of more sophisticated tests—for example, ones which would measure toxic and corrosive products of combustion—is hampered by the complexity of the smoke problem. A single material can give off many different products of combustion under varying conditions of temperature, humidity, pressure, and other factors; burning cellulose, for example, can produce 96 different compounds.

Most tests do not simulate complexities of real fires. Nationally recognized test methods for evaluating the ignition and flame-spread hazards of conventional materials in conventional applications may not be appropriate for evaluating these materials when used in new ways or for evaluating new materials.

For example, the ASTM's tunnel test for building materials, devised long before the advent of plastics, would register a low rate of flame spread for a particular plastic, whereas, in a real fire environment, that same material will burn with an explosive intensity. As a result, architects, design engineers, building contractors, and ultimately the consuming public may grossly misinterpret or inappropriately extrapolate those test results as indicative of fire safety.

Existing large- and small-scale tests suffer from an inability to predict exact consequences of a real fire, particularly those involving foamed plastics. Improvement of test methods is dependent, to a large degree, on a better understanding of the basic processes of ignition and combustion and the mechanisms of fire retardancy and smoke generation and correlating these with actual fire experiences. **The Commission recommends that research in the basic processes of ignition and combustion be strongly increased to provide a foundation for developing improved test methods.**

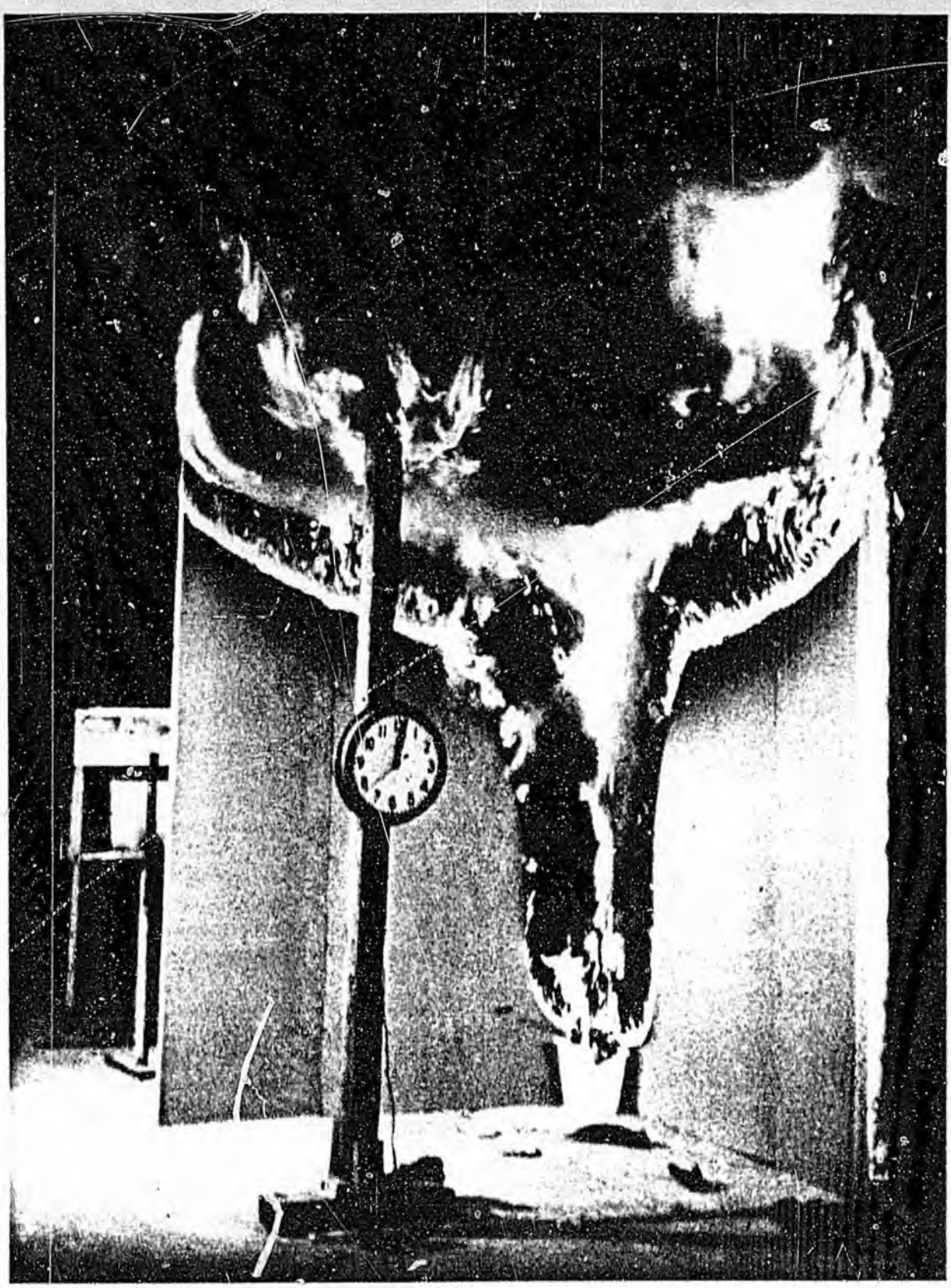
The economic interests of manufacturers, installers, vendors, and others often run counter to stringent fire safety requirements. For example, in many West Coast communities, because of industry pressures and public preferences, building codes do not outlaw untreated wood shingle roofs, despite their potential for spreading fire.

Some important hazards are not covered by building codes. The fire safety requirements of building codes apply mostly to construction materials and interior materials used on walls and ceilings. Comparatively little attention has been paid to floors and floor coverings, since in the past their contribution to fire spread was minimal. The advent of synthetic rugs and tiles has made greater attention to floors imperative.

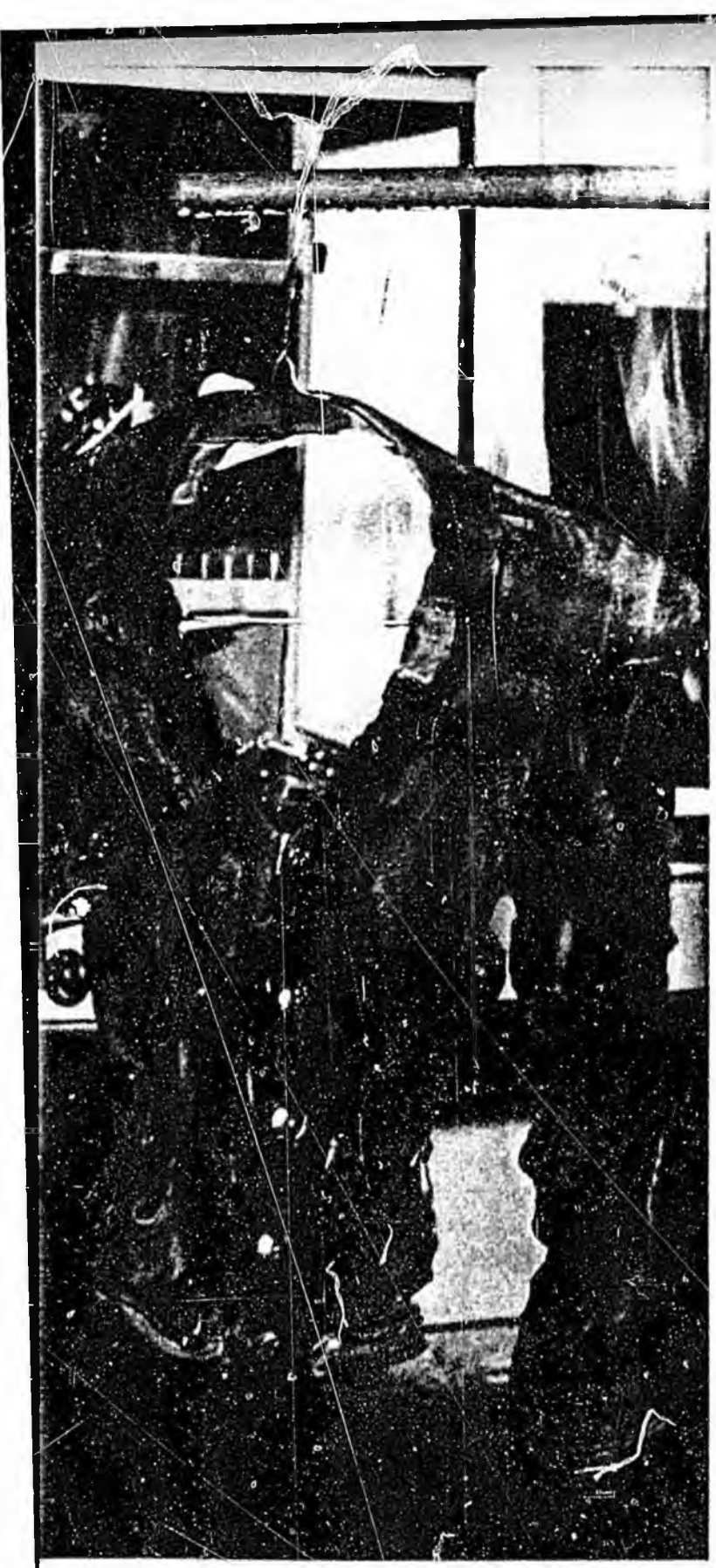
Building codes do not cover interior furnishings. While most political jurisdictions that have building codes also have fire prevention codes, designed to ensure fire safety after a building is constructed and occupied, the fire prevention codes, too, have little to say about interior furnishings. Moreover, seldom do fire prevention codes apply to private dwellings. Interior furnishings are not regulated partly because they are felt to be the province of the owner or tenant and partly because until recently there was no motivation to develop tests on which to base code provisions. They would, indeed, be difficult to regulate, since they are subject to continuing change.

While furnishings are likely to remain outside of code provisions, the fact that they contribute significantly to combustion hazards means that building codes only partly satisfy the demands of fire safety. The present practice can be compared to installing a burglar alarm at the front door and leaving the back door wide open. Only to a limited extent is this mitigated by Federal flammability standards for fabrics.

Consumers use materials with inadequate knowledge of their combustion hazards. Except for flammable liquids and the materials that are used in appliances and wiring, few of the materials that go into the home carry labels vouchsafing their fire resistance or warning of their hazards. The unlabeled hazards are found in draperies, rugs, storage cabinets, upholstered chairs, and other furniture. At present, the housewife working at the kitchen range has no way of knowing that her shiny new kitchen cabinets over-



Although considered "safe" by standard tests, this foamed plastic wallboard burns furiously in a "corner" test.



The plastic drawer fronts lack the fire resistance of the wood they simulate, and some synthetic garments burn furiously.

head are an invitation to a disastrous fire if their surface is a hot-dip polystyrene coating. A sudden flare-up from burning grease in a skillet might readily ignite the finish on the cabinets, and in no time at all fire could spread explosively throughout the kitchen.

Clearly, homeowners and building tenants need to know the relative hazards of furnishings as well as other materials so that they can minimize the risks. Fire inspectors, whether enforcing a fire prevention code or educating homeowners and tenants, need to know the hazards to carry out their tasks effectively.

New Efforts by Government and Industry

Federal initiative is needed to help close the gaps left by the voluntary action of industry and the loopholes in material standards and building codes.

In 1972 Congress created the Consumer Product Safety Commission, authorizing it to "conduct research, studies, and investigations on the safety of consumer products and on improving the safety of such products." The Commission can set standards of composition and design which

consumer products must meet; it can require labeling of hazards or instructions for safe use; it can ban products that present "an unreasonable risk of injury."

The materials that go into the built environment come under the purview of the Consumer Product Safety Commission. **This Commission recommends that the new Consumer Product Safety Commission give a high priority to the combustion hazards of materials in their end use.** Specific needs are refined understanding of the destructive effects of smoke and toxic gases, development of standards to minimize those effects, development of labeling requirements for materials, and outright ban of materials in uses that present unreasonable risks.

The development of a labeling system identifying combustion hazards is especially important. The purpose of such a system is not to regulate the lives of Americans, as an overly rigorous set of standards would do, but to enable consumers to evaluate the combustion hazards of the materials and products they bring into their homes. Further, in public buildings, nursing homes, and other occupancies subject to regulation, the labeling system would enable inspectors to verify adherence to fire load requirements. Though considerable research and testing would be needed, the eventual goal of the labeling program should be to identify fuel contribution, smoke production, and the production of toxic and corrosive gases, as well as such characteristics as ignition temperature and flame spread.

We feel we should be candid in expressing our concern that, because the Consumer Product Safety Commission is still in its formative stages, and because other hazards (many of them better publicized than combustion hazards) will be competing for attention, the problem of fire safety may become a delayed priority. The Consumer Product Safety Commission could, on the other hand, give early and deserved attention to the problem of fire safety by tapping the research capabilities of the National Bureau of Standards, universities, the national standards and testing organizations, and private industry, through contracts and cooperative arrangements.

Indeed, we do not see the Consumer Product Safety Commission supplanting the efforts in the private sector, but complementing them. For one thing, the program we have recommended is extensive and long-range. Protection of the public cannot await completion of such a program; other steps must be taken. Material producers owe to various publics—building designers, code officials, fire service personnel, and consumers—an expanded and more candid effort to explain the fire charactertistics of the materials they sell.

Further, the emergence of labeling requirements for materials will not eliminate the need for technical reports—that is, papers describing test data in detail. There will continue to be a body of technically oriented users who need detailed analyses.

Technically oriented users will, for example, have to have knowledge of fuel loads beyond that provided by the labeling system. **In this connection, the Commission recommends that the present fuel load study sponsored by the General Services Administration and conducted by the National Bureau of Standards be expanded to update the technical study of occupancy fire loads.** The information in the National Bureau of Standards' "Building Materials and Structures #149," a report on various fire loads found in different occupancies, published in 1957, is now largely out of date.

Flammable Fabrics

In 1971, the Department of Health, Education, and Welfare reported that, in recent years, more than 3,000 Americans die annually after their clothing catches on fire, and more than 150,000 are injured from this cause. One out of four whose clothing catches fire is a child under 10. Those 65 and over account for 15 percent of the clothing fires, even though they are less than 10 percent of the Nation's population. The very young and the old are also the persons least able to tolerate burns.

When clothing catches fire, the extent and depth of burns are more severe than skin burns on uncovered areas; from the standpoint of fire safety, the human species would be better off naked. A recent study by the National Burn Information Exchange showed that clothing burn victims were four times more likely to die than burn victims spared clothing fire. Their burns covered nearly twice as much body surface.

The power to set flammability standards for

fabrics now resides with the Consumer Product Safety Commission. During the 5 years that the flammable fabrics program was shared by the Department of Commerce, the Federal Trade Commission, and the Department of Health, Education, and Welfare, only a few standards were promulgated: those for young children's sleepwear (up to size 6X), rugs, small carpets, and mattresses.

These standards do nothing to protect the elderly smoker, the housewife whose sleeve passes over the kitchen burner, or the group of 8-year-olds playing with fire in a vacant lot. Notably they bypass most children between the ages of five and nine, who account for 13 percent of clothing fire accidents.

The Commission recommends that flammability standards for fabrics be given high priority by the Consumer Product Safety Commission. Specific needs are research to improve fire retardant processes, extension of flammability standards to further categories of fabric use, development of labeling requirements for other categories, and educational efforts to make consumers aware of fire hazards from clothing and other fabrics. The Commission does not favor unbridled extension of flammability standards to all categories of fabrics. Only grossly hazardous fabrics and fabrics implicated in a very large number of fire accidents should be banned from the marketplace. A preferable direction of emphasis is toward labeling requirements as to combustion hazards. This would honor the cherished principle of free choice, while at the same time informing consumers of potential risks and reminding them of the importance of fire. If reinforced by consumer education on fire safety, labeling requirements would have the effect of spurring manufacturers to improve the flame-resistance of fabrics.

Fireworks

One material hazard that has declined over the years, but not to the point of negligible concern, is fireworks. In recent years, fireworks have claimed an average of about 600 reported injuries and 10 deaths annually. Sixty years ago the annual toll from fireworks was more than 5,000 injuries and 200 deaths.

In 1938, the National Fire Protection Association published its "Model State Fireworks Law" (NFPA 494L), which, where enacted, prohibits the use of all fireworks except those in supervised public displays. Today, a majority of Americans remain insufficiently protected from fireworks accidents, since only 18 States have laws as stringent as the NFPA's model law and an additional eight have laws similar to the model but with exceptions. **The Commission recommends that all States adopt the Model State Fireworks Law of the National Fire Protection Association, thus prohibiting all fireworks except those for public displays.**[2]

The Importance of Research

Adequate regulation of materials in the built environment depends upon adequate testing, and adequate testing, in turn, depends on adequate understanding of combustion and its hazards. That is not to say, however, that progress cannot be made at all three levels simultaneously.

Improved testing methods are being pursued. Scientists and engineers at the National Bureau of Standards, for example, are utilizing a smoke chamber which measures, in addition to the density and rate of smoke produced by a sample, the concentration of specific gases emitted. Experts there and elsewhere are improving devices for measuring heat release, ignitability, flame spread, and fire endurance. Other scientists are working on model testing techniques to simulate the conditions of full-scale fires.

The technology for more sophisticated testing and the technology for basic research on fire overlap, and the two activities go hand-in-hand. It is appropriate that the National Bureau of Standards continue to provide leadership in both these areas. The Consumer Product Safety Commission should champion the strengthening of NBS efforts in these areas. At the same time, ongoing efforts of university scientists, manufacturers, and industrial testing laboratories should be encouraged and expanded.

[2] The National Society for the Prevention of Blindness, Inc., lists the following groups as supporting the limitation of all fireworks to licensed public displays only: the American Academy of Pediatrics, the American Public Health Association, the California Fire Chiefs Association, the Fire Marshals Association of North America, the International Association of Fire Chiefs, the National Fire Protection Association, the National Safety Council, the National Society for the Prevention of Blindness, Optimist International.

One basic goal of research should be to improve understanding of the dynamics of fire—not of flames alone, but of smoke, heat, toxic gases, and oxygen depletion, which together cause more deaths than flames do. **The Commission recommends that the Department of Commerce be funded to provide grants for studies of combustion dynamics and the means of its control.**

Medical research is also pertinent. In Chapter 2 we recommended that the National Institutes of Health undertake a major program of research concerning smoke inhalation injuries. One outgrowth of that research should be new knowledge concerning human tolerances of various products of combustion. From this knowledge standards can be derived setting maximum allowable outputs of various products of combustion for materials. **The Commission recommends that the National Bureau of Standards and the National Institutes of Health cooperatively devise and implement a set of research objectives designed to provide combustion standards for materials to protect human life.** It would be appropriate for NIH to bring these objectives to the attention of the community of medical scientists, to incorporate appropriate objectives in its own research programs, and to transmit to the Consumer Product Safety Commission pertinent research results.

A Question of Priorities

The hazards of materials in the built environment will never be eliminated completely, and they cannot be significantly reduced overnight. Tinderbox houses will remain in the environment until economic circumstances favor their replacement or until wear and tear dictate their removal. In settings where we are forced to live with hazardous materials, we must turn to engineering means—automatic sprinklers, for example, or early-warning detection and alarm systems—to compensate for the dangers. But for the future, we as a Nation cannot rely on these systems alone to protect us; the materials themselves must be improved for fire safety. True, a building constructed of fire-safe materials and having an automatic extinguishing system as well offers a certain redundancy of protection. But one without the other leaves open possibilities of disaster.

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DEPARTMENT OF CITY PLANNING 100 LARKIN STREET · SAN FRANCISCO, CALIFORNIA 94102

FIREFIGHTER EXPOSURE TO ENVIRONMENTAL CARCINOGENS

by

Selina Bendix, Ph.D.

to be presented at

Third International Conference on Fire Safety

San Francisco, 16-20 January 1978

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Excess Cancer Incidence in Firefighters

Up to now, what little epidemiological study has been done on occupational exposure to carcinogenic chemicals has related to occupations in which employees are exposed to a single known or suspected carcinogen amenable to routine monitoring. The firefighter is exposed to a variety of known and unknown carcinogens under conditions not amenable to monitoring. Most monitoring systems available for airborne chemical carcinogens function only under conditions where a single carcinogen is the dominant chemical species present. The complex chemical mixtures characteristic of the fire scene require something of the sophistication of gas chromatography/mass spectrometric analysis.

Although little epidemiological work has been done on firefighters, the following reports of excess cancer incidence in firefighters are available: excess buccal and pharyngeal cancer (1959),[1] excess intestinal and rectal cancer (1963),[2] and excess colon and rectal cancer (1975).[3] A 25-year study of cause of death in active firefighters[4] found a steady increase in cancer in Toronto firefighters from 15.4% in 1945-49 to 38.4% in 1965-70 (see Figure 1). I believe this increase is due to the proliferation of synthetic organic chemicals resulting in firefighter exposure to increasing numbers and quantities of carcinogens and to the latent period between exposure and appearance of chemically induced cancer.

Some Workers Compensation boards recognize that cancer in firefighters is most likely employment related and have ruled accordingly.[5, 6] In a recent (1977) report, occupational medicine specialist John Blair Webster, M.D., found excess total cancer and leukemia deaths among firefighters and concluded that leukemia in an Ohio firefighter was "the result of his exposure to carcinogens

as a firefighter and that 100% of his total and permanent disability is related to his occupation by direct cause."[7]

Benzene and Leukemia

Benzene is an accepted cause of aplastic anemia, it "...has been known for almost a century as a powerful bone-marrow poison, leading to aplastic or hypoplastic anemia."[8] Aplastic anemia may be the fairly rapid consequence of exposure to a toxic substance or it may be virtually indistinguishable from early stages of leukemia or other myeloproliferative diseases.[9]

Vigliani and Saita have described benzene-induced leukemia preceded by an aplastic phase over 10 years ago: "...the leukemia was ... often preceded by a period of apparently aplastic anemia with leukopenia."[10]

The interval between initial benzene exposure and death from leukemia ranges from 2 to 21 years.[11] It is not known how much benzene is required to produce leukemia in humans; 0.001 milliliter of benzene per week in single doses has been reported to be enough to induce leukemia in mice.[12]

Benzene Exposure of Firefighters

A 1967 California case in which a retirement board found that leukemia could not be job-related in a firefighter because he would not have been exposed to benzene on the job led me to examine the question of firefighter exposure to benzene.

Over 3 million tons of benzene per year are obtained in the United States alone.[13] Gasoline, degreasing agents, solvents, model airplane and other glues, paint strippers, etc., all contain benzene. The boiling point of benzene is 80°C or 176°F. This means that when benzene or benzene-containing products

are present at a fire site, benzene not in sealed containers evaporates at temperatures well below the boiling point of water and that the vapor pressure of benzene rises rapidly with temperature increases above ambient temperature.

Benzene is a product of combustion of polyvinyl chloride (PVC),[14, 15] polystyrene,[15] polyurethane,[16] polyphenylene oxide,[15] and polyester[17] plastics; of bismaleimide,[18] epoxy,[17] phenolic,[17] and silicone[17] resins; and of douglas fir.[18] The presence of some form of PVC plastic can be assumed at virtually every fire site in recent years because of the variety of PVC-containing products such as furniture, office equipment, electric wire insulation, car parts, water pipes, and kitchen gadgets. The ubiquitous nature of PVC and polystyrene plastics can be judged from the fact that in 1969 three billion pounds of PVC and two billion pounds of polystyrene plastics were manufactured in the United States.[19]

According to Victor Esch, M.D., Chief Surgeon of the District of Columbia Fire Department, the most dangerous period for exposure to toxic products of combustion of plastics is "the 'over-haul' or clean-up phase when firemen instinctively remove their masks for better visibility and comfort -- only to be zapped by the invisible, noxious gases that may linger for hours in confined spaces."[20]

As firefighters fight fires at gasoline stations, where benzene is a component of the gasoline; at automobile repair shops, where benzene may be present in solvents, degreasers and glues and where benzene is probably a product of combustion of auto seats and upholstery; and at homes filled with all kinds of furnishings made of synthetic materials that are more flammable than wood, many of which produce benzene when they burn, they have multiple opportunities

for exposure to benzene. This exposure may well account for much of Blair's report of excess leukemia among firefighters.

Firefighter Exposure to Other Carcinogens

Asbestos. At the end of a structural fire, when the firefighter pulls down ceilings and breaks open walls looking for smouldering fires, he is exposed to asbestos from insulation (both thermal and acoustic); acoustical tile; decorating and fire-proofing surfacing materials; patching, spackling and jointing compounds; wallboard and floor tile. Asbestos-containing materials may be found on walls, ceilings, floors, exposed structural steel, air ducts, plenums and return air spaces. Bruckman[21] has reported a 10-fold increase in mesothelioma, the specific type of cancer induced by asbestos, in the period 1935-1959, which appears to parallel cumulative asbestos consumption. Incidence of other types of cancer also rises among asbestos workers. Opportunities for firefighter exposure to asbestos rise with increasing asbestos use. Use of sprayed asbestos is now being controlled, however its presence in buildings built prior to recent institution of controls is widespread and generally unknown to building owners and occupants, much less firefighters.

Chlorinated Hydrocarbons. In fires at dry cleaning establishments firefighter exposure to the carcinogen carbon tetrachloride[22] was replaced by exposure to perchlyoroethylene, which in turn proved to be a carcinogen.[23]

Heating or burning PVC plastic, particularly in older products manufactured before the dangers of entrained, unpolymerized vinyl chloride monomer were understood, gives off varying amounts of carcinogenic vinyl chloride.[14, 24] Fires in newer buildings will encounter not only a variety of interior objects made of PVC but also PUC insulation on wires. Some form of PVC is probably

now found at the site of most fires.

Acrylonitrile. The world demand for acrylonitrile approached 5 billion lb in 1976. About 80% of this went into acrylic and modacrylic fibers and into the thermoplastic resins acrilonitrile-butadience-styrene (ABS) and styrene-acrylonitrile (SAN).[25] U. S. annual production of acrylonitrile is 1.5 billion lb. Workers exposed to acrylonitrile have an excess incidence of lung and colon cancer.[26] A major chemical tank farm fire involving acrylonitrile was reported at the 1976 Fall Meeting of the NFPA. Acrylonitrile is formed as a combustion product of durette (a polyvinyl fluoride treated with a chlorinating agent).[27]

MOCA. The human carcinogen, MOCA (4,4- methylene bis (2-chloroaniline)) is used in the manufacture of isocyanate resins in the plastics, aircraft, resin, and synthetic rubber industries, in ratio and television equipment, and in space and missile components. Cal-OSHA estimates that there are over 3,000 potential sites in California where MOCA is in use.[28] Firefighters at one of these sites would probably not know that MOCA was in use.

Benzidine. The human carcinogen benzidine is used in plastics, rubber and dyes. Cal-OSHA estimates that benzidine is in use at over 1,400 sites in California.[28] Workers routinely exposed to benzidine incur bladder tumors at a rate of 20% to over 50%.[29]

Carcinogens from Wood and Fossil Fuels

A report by the National Institute for Occupational Safety and Health indicates an increase in myeloproliferative cancers in millwrights, millmen, cabinet workers, lumber and sawmill workers.[30] In the absence of knowledge as to what substance(s) produce this effect, it is possible that firefighters are

exposed to these substances at fires in wooden structures.

Polycyclic aromatic hydrocarbons (PAH), many of which are carcinogenic, are formed on combustion of fossil fuels and other organic substances. Anthropogenic combustion appears to be the main source of PAH. These carcinogens are presumably formed at every fire.

Pesticides

Pesticides known to be carcinogenic include: chlordane,[31] heptachlor,[31] aldrin,[32] dieldrin,[32] mirex,[33] chlordimeform,[34] endrin,[35] chlorobenzilate,[36] DDT,[37] benzene hexachloride,[38] dimethoate,[39] all ethylenebisdithiocarbamates,[40] etc. The carcinogen chloroform is found in at least six pesticide products, of which five are for the control of mites, screwworms, or mange on house pets and farm animals.[41]

Pesticides are now found in grocery stores, drug stores, hardware stores and garden shops, as well as in agricultural situations and homes. It should not be assumed that only agricultural situations pose a pesticide-exposure hazard. Pesticides with high immediate toxicity are generally found only in agricultural use, but many substances with long-term effects, such as carcinogenesis, are widely distributed and presently banned substances are found in many basements.

Plastics

The proliferation of plastic products in the last two decades has resulted in a qualitative change in the nature of the compounds to which firefighters are exposed at fires. According to Esch: "Literally hundreds of compounds are given off by flame retardants, fillers, plasticizers, ultraviolet light absorbers, antioxidants, lubricants, reinforcing fibers, peroxides, coupling agents,

halogen stabilizers, biological preservatives, 'anti-static' agents, flow controls, coloring agents and other exotic chemicals. These products create an extremely complex problem in plastics fires. It is mind-boggling, to say the least, when you consider that we are just seeing the tip of the iceberg and the tremendous amount of research needed to determine the specific hazard of burning plastics."[19]

It is not known how many carcinogens may be produced by burning plastics and portable instrumentation capable of monitoring for a variety of known carcinogens at the fire site is not yet on the drawing board, nor are there enough trained toxicologists in the country to run the requisite tests.

Multiple Exposures

It is important to realize that there is little experimental evidence on the effects of simultaneous exposures to low levels of two, much less many, carcinogens. It is known that smoking "greatly increases the risk of lung cancer associated with many cancer-causing chemicals,"[28] a suggestion that multiple carcinogen exposures may have synergistic effects. Firefighters may be the unwitting guinea pigs who will tell us what the consequences are of intermittent exposure to a variety of carcinogens at levels higher than those of the general population.

Conclusions

Given that a case can be made for exposure of firefighters to a variety of carcinogens on the job, what should be done? I would make the following recommendations:

1. Firefighter exposure to inhaled carcinogens should be decreased by

use of lightweight disposable face masks when self-contained breathing apparatus is not needed.

2. Overhaul procedures should be reevaluated in the light of the potential for avoidable firefighter exposure to carcinogens during overhaul.
3. Yearly physicals and lab tests should be provided for all firefighters.
4. Records of the nature of substances at fire sites should be improved. Present records rarely include information about substances on-site, even for spill clean-up incidents. These records should be kept long enough to allow analysis of occupational cancers with 20-30 year latent periods.
5. OSHA protection of workers exposed to carcinogens[42] should be extended to firefighters.
6. Just as pneumonia in a firefighter is presumed to be occupationally related because of the effects of routine smoke inhalation, cancer in a firefighter should be assumed to be work related because of routine exposure to carcinogens.
7. Epidemiologic studies should be done to ascertain the actual level of cancer incidence in firefighters as compared to other occupations.

REFERENCES

1. Mastromatteo, E. Mortality in City Firemen, Arch. Indust. Health (1959) 20:1-7, cited in Abrams, James Lloyd, Occupational Mortality Among Professional Firefighters, Ph.D. Dissertation, Univ. of Oklahoma, 1974, p. 83.

2. Guralnick, Lillian. Mortality by Occupation and the Cause of Death Among Men 20 to 64 Years of Age: United States, 1950; Vital Statistics -- Special Reports, (1963) 53, No. 3, cited in Abrams, James Lloyd, Occupational Mortality Among Professional Firefighters, Ph.D. Dissertation, Univ. of Oklahoma, 1974, p. 85.

3. Berg, John W. and Howell, Margaret A. Occupation and Bowel Cancer, J. Toxicol. & Environ. Health, 1:82 (1975).

4. Ralph, Robert E. A Close Look At Firefighters' Deaths and Injuries; Proc. Symp. Occupational Health and Hazards of the Fire Service, 11-13 Jan 1971, p. 14.

5. Decision in case of Jean M. Snape, widow-claimant, Claim No. OD PE 5583; The Industrial Commission of Ohio (1974).

6. Decision in case of Lillian Baldwin, widow-claimant, Claim No. OD-5303-PE; The Industrial Commission of Ohio (1974).

7. Webster, John Blair. Specialist's Report in regard to Claim No. CD-PE 5969 to State of Ohio Bureau of Workmen's Compensation (1975).

8. Vigliani. E.C.; Leukemia Associated with Benzene Exposure; Ann. N. Y. Acad. Sci. 271:143 (1976).

9. Amromin, George D. Pathology of Leukemia; Harper and Row, N. Y., (1968) p. 11.

10. Vigliani, E.C. and Saita, Giulio. Benzene and Leukemia; N. Eng. J. Med. 271:873 (1964).

11. Infante, Peter F. et al. Leukemia Among Workers Exposed to Benzene; Industry-wide Studies Branch, Division of Surveillance, Hazard Evaluations and Field Studies, Natl. Inst. Occupational Safety and Health, Cincinnati, OH; manuscript for submission to Lancet, 28 April 1977, p.7.

12. Lignac, G.O.E. Die Benzolleukämie bei Menschen und Weissen Mäusen. III. Zweite Benzol-versuchsreihe -- von 54 Mäuse gehen 8 an Leukämie oder Lymphoblastoma infiltrans aleucaemicum zugrunde -- frühere Tier -- benzolversuche; Krankheitsforsch. 9:426 (1932); cited in reference 8.

13. Manufacturing Chemists Assoc. Chemical Statistics Handbook, 7th Ed.; MCA, Washington, D. C. (1971) p. 9.

14. O'Mara, Michael M. Combustion of PVC; presented at International Symposium on the Toxicity and Physiology of Combustion Products, Salt Lake City, UT, 22-26 March 1976.

15. Kimmerle, Georg. Aspects and Methodology for the Evaluation of Toxicological Parameters During Fire Exposure; presented at Flammability Characteristics of Materials, Polymer Conference Series, Univ. of Utah, Salt Lake City, UT, 11-15 June 1973, p. 35.

16. Hileman, F. D. et al. Pyrolysis of a Flexible-Urethane Foam; Flammability Research Center, Univ. of Utah, Salt Lake City, UT, 16 October 1974, Table V.

17. Madorsky, S. L., and Straus, S. Stability of Thermoset Plastics at High Temperatures, Modern Plastics (1961) 38:134-40; cited in Dufour, R. E. Survey of Available Information on the Toxicity of the Combustion and Thermal Decomposition Products of Certain Building Materials Under Fire Conditions; Underwriters Laboratories Bulletin of Research No. 53, 1963.

18. Packham, S. C. Flammability and Toxicological Aspects Pertaining to the Combustion of Douglas Fir in Flammability of Materials, Polymer Conference Series, Univ. of Utah, 7-11 July 1975.

19. Reference 13, p. 116.

20. 'Plastic fires' create new hazards for both firemen and public; Medical News, JAMA 234:1212. (1975)

21. Bruckman, Leonard. A Study of Airborne Asbestos Fibers in Connecticut; presented at the Workshop on Asbestos: Definitions and Measurement Methods, National Bureau of Standards, Gaithersburg, MD, 18-20 July 1977.

22. Christensen, Herbert E. and Luginbyhl, Thomas, Eds. Registry of Toxic Effects of Chemical Substances, 1975 Ed.; USHEW, Rockville, MD, June 1975, p. 295.

23. Solvent tied to liver cancer, San Francisco Examiner, 18 July 1977; Cleaning Solvent Tied to Cancer Risk, San Francisco Chronicle (UP), 18 July 1977.

24. Reference 20, p. 1213.

25. Acrylonitrile draws growing new markets, C&EN 4 April 1977, p. 12.

26. Bingham, Eula. Occupational Exposure to Acrylonitrile; Fed. Reg. 42:33043-4 (1977).

27. Ryan, P. W. Comparison of Flaming Combustion with Oxidative Degradation in Flammability of Materials, Polymer Conference Series, Univ. of Utah, 7-11 July 1975.

28. Cancer-Causing Chemicals: A Reference Chart for On-The-Job Health Protection, State of Calif., Dept. of Health, Occupational Cancer Control Unit, May 1977.

29. Zavon, Mitchell R. et al. Benzidine Exposure as a Cause of Bladder Tumors; Arch. Env. Health 27:1-7 (1973).

30. Study finds cancer-woodworking tie, Engineering News Record, 30 October 1974, p. 15.

31. Fredrickson, Donald S. Reports on Bioassays of Chlordane and Heptachlor for Possible Carcinogenicity: Fed. Reg. 42:48394-5 (1977).

32. Gillette, Robert. Cancer and the Environment (I): A Creaky System Grinds On; Science 186:239-45 (1974).

33. Holden, Constance. Mirex: Persistent Pesticide on Its Way Out; Science 194:301-3 (1976).

34. EPA Notified of Voluntary Recall of Insecticide Chlordimeform; EPA Environmental News, October 1976, p. 11.

35. Concentrates, Chemical & Engineering News, 26 July 1976, p. 9.

36. Allied Asks EPA to Cancel Kepone; Chemecology, August 1976, p.3.

37. Court OKs Toxics Settlement; EPA Moves on Four Pesticides; Chemecology, July 1976, p. 1.

38. EPA Proposes to Cancel "BHC" A Chemical Used in Pesticides Manufacture, Asks for Public Comment on Risks and Benefits of Pesticides Containing BHC; EPA Environment News, December 1976, p. 3.

39. Johnson, Edwin L. Rebuttable Presumption Against Registration and Continued Registration of Pesticide Products Containing Dimethoate; Fed. Reg. 42:45806-26 (1977).

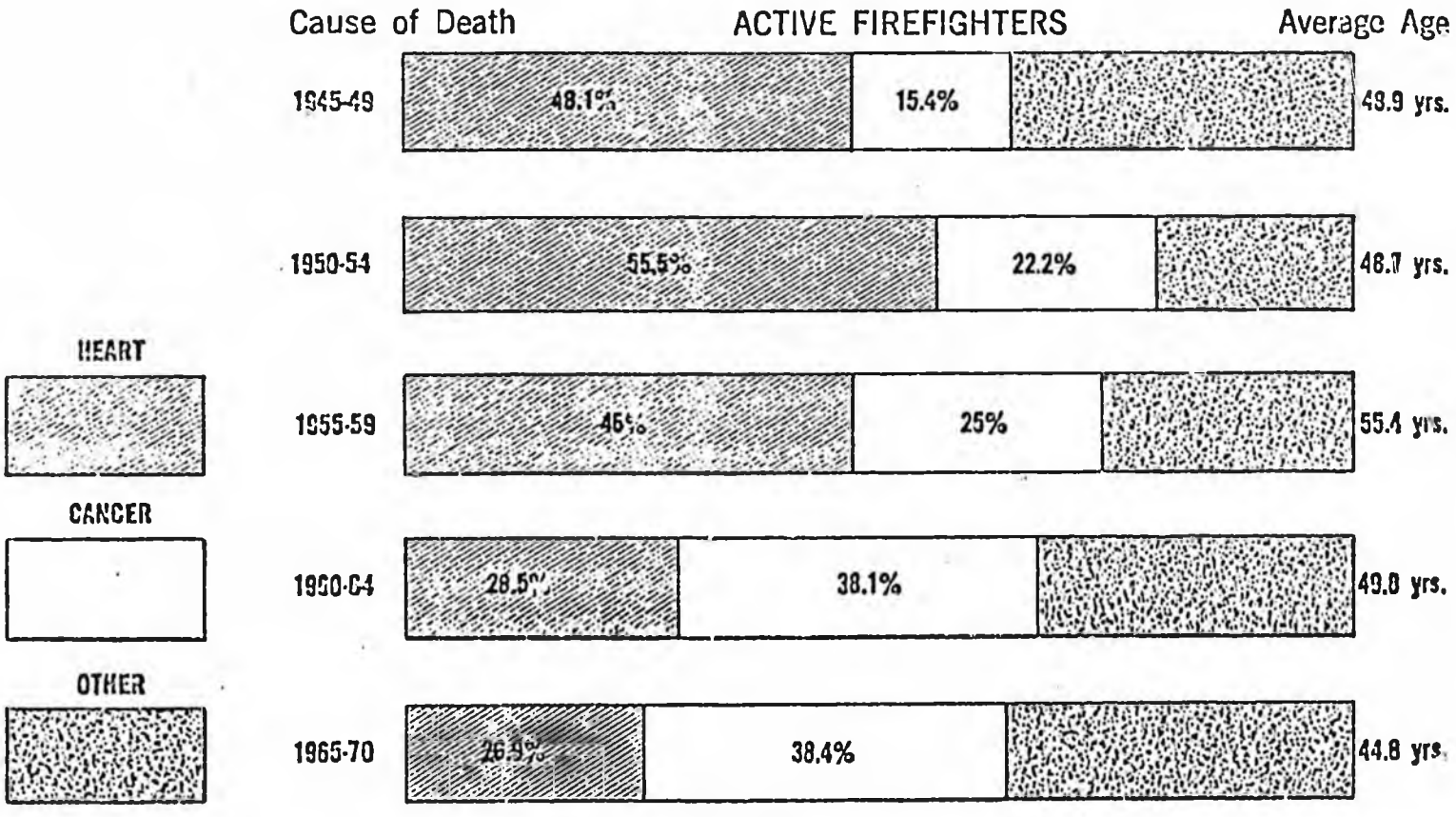
40. Johnson, Edwin L. Rebuttable Presumption Against Registratic and Continued Registration of Pesticide Products Containing Ethylene-bisdithiocarbamates; Fed. Reg. 42:40618-75 (1977).

41. EPA Acts to Limit Exposure to Chloroform, EPA Environmental News, May 1976, p. 10.

42. Dept. of Labor, OSHA. Identification, Classification and Regulation of Toxic Substances Posing a Potential Occupational Carcinogenic Risk, Fed. Reg. 42:54148-54247 (1977).

Figure 1

From Reference 42. Cause of Death in Active Firefighters.



INHALATION INJURY: DIAGNOSIS AND PATHOPHYSIOLOGY

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Inhalation injury is damage to the mucosa of the larynx, trachea or bronchus from exposure to products of incomplete combustion. In addition, these particles resulting from incomplete combustion may cause bronchiolar and alveolar (small airway) damage as well. Since the nasopharynx has tremendous cooling capabilities and since laryngospasm will supervene if heat does reach the level of the larynx, seldom if ever is heat damage seen below the level of the larynx. However, heat may cause upper airway obstruction, followed by hypoxemia, cardiac arrhythmia and death. The chemical damage to the mucosa of the trachea, bronchi or bronchioles may result in copious secretions, bronchospasm, hypoxemia, pulmonary edema and pneumonia. The onset of the clinical inhalation injury syndrome may be delayed from hours to two or three days following exposure to smoke, during which there is a relatively asymptomatic period which may be followed by severe pulmonary complications. The essence of diagnosis of inhalation injury is a careful history, a thorough physical examination, laboratory blood gas determinations, chest roentgenogram, xenon^{133} lung scan and bronchoscopy.

Since the patient may be asymptomatic, the important points in the history are burns occurring in a closed space or if in the open from a rapid combustion of petroleum products, or pre-existent depression of the level of consciousness so that the patient was exposed to smoke for a longer period of time. Burns about the nose and mouth should heighten the suspicion that an inhalation injury may have occurred, but absence of such burn injury does not eliminate the possibility of inhalation injury. The production of carbonaceous sputum during the asymptomatic period is evidence that an inhalation injury has occurred. The clinical inhalation injury picture is one of dyspnea, hoarseness or upper airway wheezing and a cough productive of fluid and carbon. The chest roentgenogram during the asymptomatic period is normal unless there is pre-existent pulmonary disease. Blood gas determinations in the asymptomatic period are also normal. However, with serial x-rays following the normal chest film on admission, one sees atelectasis and air trapping followed by patchy pulmonary infiltrates during progression of inhalation injury. In an attempt to accurately diagnose the presence of an inhalation injury during the asymptomatic period, two additional diagnostic techniques are utilized, that is, the xenon^{133} lung scan and bronchoscopy.

The xenon^{133} lung scan is performed by injecting xenon^{133} intravenously, and since the xenon gas is cleared by the lungs, serial scintiphotograms are obtained to document the pattern and rate of xenon clearance as the patient breathes. A normal scan will show complete and equal clearance of xenon^{133} within 90 seconds. A delay of clearance beyond 90 seconds or segmental retention of xenon^{133} is considered diagnostic of inhalation injury only if the patient had a normal chest x-ray prior to the performance of the test. A positive xenon lung scan in the asymptomatic period allows one to begin treatment to modify the severity of the inhalation injury syndrome before it occurs. Xenon^{133} lung scans are most helpful to identify small airway and alveolar damage.

Bronchoscopy will identify laryngeal, tracheal and bronchial inhalation injury. Positive findings at the time of bronchoscopy include observation of carbonaceous material on mucosal surfaces, excessive secretion of endotracheal and endobronchial mucosa, swelling erythema, hemorrhage and ulceration of the mucosa.

If upper airway obstruction occurs. endotracheal intubation may be necessary to insure an open airway. However, racemic epinephrine and humidification may be sufficient for mild upper airway swelling. In addition, the treatment of inhalation injury includes bronchodilators for the treatment of bronchospasm, repeated bronchoscopy for the removal of debris, humidification of inspired air, antibiotics for pneumonia, monitoring with serial chest x-ray and arterial blood gas determinations, and if indicated use of a mechanical ventilator.

The accurate diagnosis of inhalation injury is extremely important, since the patient may have a significant asymptomatic period before a rather rapid onset of clinical symptoms and signs, and thus a positive diagnosis during the asymptomatic period mandates admission to the hospital and allows treatment to begin prior to the onset of clinical signs, so that the syndrome may be modified.

BIBLIOGRAPHY

Agee, RN, Long JM III, Hunt JL, Petroff PA, Lull RJ, Mason AD Jr, Pruitt BA Jr: Use of ^{133}Xenon in early diagnosis of inhalation injury. J Trauma 16:218-224, 1976.

DiVincenti FC, Pruitt BA Jr, Reckler JM: Inhalation Injuries. J Trauma 11:109-117, 1971.

Hunt JL, Agee RN, Pruitt BA Jr: Fiberoptic bronchoscopy in acute inhalation injury. J Trauma 15:641-649, 1975.

Moylan JA Jr, Wilmore DW, Mouton DE, Pruitt BA Jr: Early diagnosis of inhalation injury using ^{133}Xenon lung scan. Ann Surg 176:477-484, 1972.

AIR CONTAMINANTS ENCOUNTERED BY FIREFIGHTERS

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Our research group at the Harvard School of Public Health has recently completed the latest in a series of studies investigating the environmental conditions encountered by firefighters in the course of performing their duties. Because this project was based, to a large degree, on the experience and results from several earlier studies, a quick review is in order.

From 1970 to 1977, occupational health studies have provided physiological and epidemiological proof that excess respiratory disease is associated with firefighting. [1,2] A project completed in 1977 involved the development and implementation of a portable system for evaluating the thermal environment in structural fires. The main finding of this study was that the predominant source of heat encountered by the firefighter was radiative as opposed to conductive, and protective clothing should be designed to reflect this fact. [3]

Our efforts to characterize the chemical constituents of "smoke" began in the early 70's with the development of a portable carbon monoxide-oxygen sampling device which provided real-time data on the exposure profiles of these two gases. [4,5] The results from 72 residential fires showed that carbon monoxide presents a much greater hazard to the firefighter than does oxygen deficiency. For 29% of the time during these fires, the carbon monoxide concentrations exceeded 500 ppm, yet in only 6 fires did the oxygen concentration fall below 18%. The maximum CO concentration measured was 27,000 ppm, a level at which death would result immediately without respiratory protection. These results are interesting in that model fires consistently show depressed oxygen concentrations.

A study, completed in 1976, measured the levels of carbon monoxide, carbon dioxide, oxygen, hydrogen chloride, hydrogen cyanide and nitrogen dioxide encountered during firefighting using a prototype sampling system built into the firefighter's turnout coat. [6] This study, which included 45 fires with each of two companies, demonstrated that CO presented a potential acute hazard, hydrogen chloride was detected in only 5 of 90 samples, oxygen depression was not significant, and CO_2 and NO_2 concentrations did not approach significant toxic levels.

Using the experience gained from these studies, we developed an expanded air sampling program which would provide data on a wider spectrum of contaminants at a larger number of fires. The goal of this study was the characterization of air contaminants present during structural firefighting. The data obtained can be used for several purposes: (1) to provide further insight into the origin of observed disease in firefighter populations, (2) to improve the medical treatment of "smoke" inhalation victims, (3) to suggest modifications in firefighting procedures to reduce exposures, and (4) to provide real data to be used in the design and implementation of breathing apparatus.

As mentioned, the preliminary study was conducted with the air sampling system distributed throughout the storage pockets of the turnout coat. Because of its bulk, weight, difficulty in servicing, and lack of acceptance

by the firefighters, the expanded study required a redesigning of the sampling unit. After consultation with the firefighters, a new design for the sampler evolved. The unit which resulted was packaged in a vacuum-formed ABS plastic case, measuring 2½" x 8" x 10", weighing 4 to 5 pounds, designed to be worn over the neck.

Wherever possible, solid sorbents were used to collect the air contaminants. Some gases, unable to be trapped on these sorbents, were collected in a Tedlar plastic bag. The sampler was designed to pull air through a succession of filters and sorbent materials. The ascarite collected hydrogen cyanide, the triethanolamine treated sieve trapped nitrogen dioxide and hydrogen chloride, and the untreated activated molecular sieve collected aldehydes, specifically acrolein. Along the other circuit, the glass filter collected particulate matter, the charcoal tube absorbed aromatic hydrocarbons (benzene), and the Tedlar bag was used for the storage of carbon monoxide, oxygen and carbon dioxide.

Six units were constructed and placed in different fire houses throughout the city of Boston. Those firefighters who volunteered to participate in the project were instructed in the use of the samplers, the purpose of the study, and the results, as they became available. The reception of these units by the firefighters was positive.

On a limited number of fires, air samples were taken during active firefighting and at the overhaul operations. These data were used to evaluate the importance of the air contaminants encountered during overhaul, an issue of great concern to the fire service.

To evaluate the exposure of firefighters to air contaminants encountered during firefighting, three exposure guidelines were used. The American Conference of Governmental Industrial Hygienists publishes a list of Threshold Limit Values - Short-term Exposure Limits (STEL).[7] These limits, expressed as airborne concentrations and based on exposures to single substances, not mixtures, were developed for unprotected industrial workers, and the appropriateness of their application to the fire environment is questionable, as they represent no-effect levels for 15 minute periods.

The second industrial exposure guide considered is the concentration at which the substance poses an Immediate Danger to Life or Health (IDLH).[8] This index, which has been used in the NIOSH-OSHA Standards Completion Project in defining the application of respiratory protective devices, is defined as the concentration from which a worker might escape within 30 minutes without irreversible health effects or suffer any effects which may impede escape.

The third and probably most appropriate, standard is the Short Term Lethal Concentration (STLC), as used by Dr. J. B. Terrill [9] Each of the contaminants considered in this study was evaluated by comparing the concentrations observed with each of these indices.

Nitrogen dioxide (NO_2) may be present in the fire environment as a result of the oxidation of the nitrogenous material or the fixation of atmospheric nitrogen. Although high concentrations of NO_2 have been noted in controlled burns,[10] in our preliminary study of actual fires it was not present in concentrations greater than 1ppm. Acute exposure to NO_2 results in bronchial

irritation with subsequent dizziness, headache and weakness. Several hours later the individual may develop pulmonary edema with subsequent asphyxia and death.

A total of 212 samples were analyzed for NO_2 in this study. Four exceeded the ACGIH-STEL of 5 ppm; however, none exceeded 50 ppm, the concentration at which NO_2 is considered immediately dangerous to life or health (IDLH). The highest concentration noted over a five minute exposure period was 8.3 ppm. While it is possible that NO_2 may act in concert with other irritants to cause serious pulmonary irritation, it alone does not represent a critical exposure at the concentrations observed.

As mentioned, NO_2 may be generated from the degradation of nitrogen-containing materials or the fixation of atmospheric nitrogen. In the fires studied, the concentrations of nitrogen dioxide were low, indicating the absence of conditions conducive to the degradation of nitrogenous materials. The low concentrations also indicated that in residential fires, flame energies are not adequate to fix atmospheric nitrogen. Our conclusion is that the NO_2 concentrations observed in this series of structural fires do not constitute a respiratory hazard to the firefighter.

Hydrogen chloride gas is formed in a fire from the pyrolysis of a range of chlorinated polymers such as polyvinyl chloride, chlorinated acrylics, and flame-retardant materials.[9] In concentrations greater than 100 ppm, HCl is corrosive to the eyes, skin and mucous membranes. This gas is an upper-respiratory irritant due to its solubility in water. When associated with high concentrations of respirable particulate, it may represent a lower airway hazard.[11] Due to its irritating effect, acute exposures to high concentrations are not willingly tolerated; however, if a person is incapable of terminating exposure or cannot escape, pulmonary edema and death may occur if concentrations exceed 500 ppm for several hours.

Of 216 samples analyzed, HCl was detected in 83, with a maximum concentration of 280 ppm encountered during one seven-minute sample. Seventy-three samples exceeded the ACGIH-STEL (5 ppm), and 5 exceeded the NIOSH-OSHA IDLH (100 ppm). None exceeded the short term lethal concentration (STLC) of 500 ppm.

Firefighters commonly complain of severe eye and respiratory irritation during and after fires and personnel without breathing apparatus may be forced from the fire scene. Responsibility for this acute irritation of the respiratory tract has, in some cases, been attributed by the fire service to HCl. In fact, this action may be caused by exposure to HCl, other respiratory irritants, or a combination of irritants. In our study, HCl was present in over one third of the fires sampled, and in a small number of fires, the concentrations were sufficient to impede the escape of a person not wearing respiratory protection. However, a concentration lethal to an unprotected person exposed over a short time was not observed during the study.

Hydrogen cyanide can be formed in the fire environment from natural products such as wool and silk, and a variety of nitrogen-containing polymers, including nitriles, polyamides, nylons, and polyurethane. Because of the widespread use of polyurethanes in furniture upholstery and building materials,

the significance of HCN in fire toxicity has received much attention.[12] In our preliminary study, this contaminant was frequently found albeit at low concentrations. In the 6 fires from this earlier study in which HCN exceeded 1 ppm, 3 involved polyurethane foam mattresses, and one was a vehicle fire. Hydrogen cyanide, toxic by inhalation and skin absorption, interferes with metabolic oxidation and causes anoxia at the cellular level. Initial symptoms of acute exposure to levels above 20 ppm include weakness, headache, and confusion, then, ultimately, respiratory failure and death.

Hydrogen cyanide was detected in 27 of 253 samples. The maximum concentration noted for a minimum of 5 minutes in this series was 3.6 ppm. Neither the STEL (15 ppm), and IDLH (50 ppm), nor the STLC (350 ppm), were approached.

In a previous study, hydrogen cyanide was detected in low concentrations in approximately one half of the fires. The study included a predominance of room fires involving upholstered furniture. In the present study, HCN was detected in only about 10% of the fires; however, the fires involved a wider spectrum of building types and materials. On the basis of both data sets we conclude that the observed hydrogen cyanide concentrations do not present a health hazard to firefighters.

The formation of acrolein at the fire scene can result from pyrolysis or combustion of cellulosic materials such as wood, cotton, and papers, plastic materials such as styrene and polyolefins, and oils and fats containing glycerol.[13] Inhalation of acrolein causes irritation of the nose and throat, nausea, shortness of breath, vesicant action with extensive lung damage, pulmonary edema, and ultimately death.

Of the 118 samples which were analyzed, acrolein was detected above the STEL (0.3 ppm) in 66 and above the IDLH (5ppm) in 5. In one fire, the concentration was at the upper end of the STLC range (30-100 ppm).

The distribution of concentration data indicates that one half of the analyzed samples showed concentrations which were greater than the STEL of 0.3 ppm and that 10% of the samples were in excess of 2.7 ppm, a concentration close to that immediately dangerous to life or health.

To our knowledge, this is the first time that acrolein has been identified in life-threatening concentrations at real fires. In over one half of the samples analyzed for acrolein, the concentration exceeded levels capable of causing significant eye and respiratory injury (0.3 ppm). Firefighters operating without respiratory protective devices in the most severe of these fires might well have suffered disabling injury. The frequency of detection of acrolein at fires suggests that a range of materials contributes to its formation. This study supports the belief by Zikria[14] and Morikawa[13] that aldehydes play an important role in respiratory injury to fire victims. As these authors suggest, medical management of fire victims should routinely consider possible exposure to this contaminant.

Strucutural fires are a source of particulates which include condensation products from volatilized materials, sub-micron carbonaceous material, large particles formed by agglomeration, and char released during fires. High concentrations of particulates cause irritation of the upper respiratory tract and massive concentrations produce respiratory spasm. Particulates also absorb many of the gases and vapors present in the fire environment and present this material to the non-ciliated portion of the lung.

Data are not available on the biological effects due to the inhalation of non-specific particulates; however, concentrations in the range of 20-50 mg/m^3 will cause light obscuration, and 1000 mg/m^3 causes immediate pulmonary distress.

The effects of exposure to particulates depend on their chemical composition, size distribution, and concentration. The sampling method used in this study did not permit an analysis of the size distribution and the scope of work did not include identification of the composition, a major analytical task. The concentration data obtained in the study are consistent with our earlier study and show concentrations which would cause simple irritation of the respiratory tract. The particulates may, of course, have a much more important role in disease production by providing adsorption sites for toxic gases and vapors.

Carbon monoxide occurs as the result of incomplete combustion of virtually all organic materials. The physiological effects of carbon monoxide are well known. As with most other toxicants, the two important parameters to consider are concentration and duration of exposure. Headache, fatigue, dizziness and sleepiness are classic symptoms of low level (150-500 ppm) exposures. Unconsciousness, collapse and death occur as the concentration and the duration of exposure increases. Visual vigilance or sensitivity is impaired at relatively low concentrations. Carbon monoxide combines with the blood's hemoglobin, thus interrupting the supply of oxygen to the tissues. Permanent tissue damage may result from asphyxia. The action is enhanced by heat, humidity and physical exertion.

Found in virtually all of the fire environments studied, the CO levels exceeded the STEL (400 ppm) on 16 occasions and the IDLH (1500 ppm) 4 times. One fire approached the short-term lethal concentration (STLC) of 5000 ppm.

The concentrations of carbon monoxide found in this study are in agreement with earlier studies by this laboratory and other investigators. This contaminant is without doubt the most routine and serious acute hazard to the unprotected firefighter. It is an extremely toxic gas with varied effects. Those exposed are given no warning of its presence and, although repeatedly shown to be the cause of most fire-related deaths, this gas continues to be underrated by the fire service. In addition, inadequate awareness of the problem by medical personnel continues to exist at many urban hospitals which routinely handle fire victims.

Carbon monoxide exerts a number of effects at low concentration, including impairment of judgment, visual acuity, and decision making--all faculties crucial to the safety of the firefighter. Therefore, even when it is not the principal cause of injury, CO may contribute to serious trauma. Furthermore, CO presents additional stress to the circulatory system, contributing to the development of cardiac disease.[15]

Carbon dioxide results from the complete combustion of organic materials and has been noted in high concentrations in engineered burns. At high concentrations, CO_2 causes stinging of the eyes, nose, and throat. At 50,000 ppm the respiratory center is stimulated, causing hyperventilation. Death has been reported at exposure concentrations in excess of 10%. At 30,000 ppm it is weakly narcotic.

Of 89 samples analyzed for CO_2, 3 exceeded the STEL (15,000 ppm) and 1 exceeded the IDLH of 50,000 ppm. In no fire did the concentration exceed the STLC (100,000 ppm).

Carbon dioxide is present in all fires but has a low order of toxicity, with mild and reversible physiological responses beginning to appear for exposures in the range of ½% to 1% (5,000-10,000 ppm). Therefore, at the concentrations observed in our study, it alone would not present a significant hazard to the firefighter. However, exposure may cause hyperventilation resulting in a potentiation of the effects of other air contaminants which might be present. Although high concentrations of CO_2 (100,000 ppm and above) have been identified in engineered burns, such levels have not been noted in our studies--further evidence that caution should be exercised in extrapolating results from the laboratory to real life situations.

The sources of benzene and other aromatics in structural fires are not precisely known, although likely sources include available petroleum products acting as fuel in the fire and the thermal degradation of certain plastics such as styrene. At high concentrations there is irritation of the eyes, drowsiness, nausea and headache, and finally unconsciousness and death from respiratory paralysis. Chronic exposures may cause blood changes and leukemia.

Of 197 samples analyzed, 181 contained measurable amounts of benzene. Thirty-three were over the STEL (5 ppm); none were over the IDLH (2,000 ppm). The IDLH index is based solely on acute effects, while the STEL was developed with consideration for long term hematopoietic problems. The maximum concentration determined was 165 ppm, well below the IDLH. Concentrations of benzene and other aromatic hydrocarbons in excess of 1,000 ppm would be acutely hazardous to the unprotected firefighter due to narcosis; however, the levels in our study were consistently below 200 ppm, so an acute danger was not present. In the observed concentration range, a chronic health hazard does not seem likely since the exposure is infrequent and of short duration. The identification by NIOSH of benzene as a human leukemogen presents a more subtle evaluation problem, and a definitive statement on this potential hazard to the firefighter cannot be made based on our present knowledge.

The personal monitoring of firefighter exposures to toxic air contaminants during structural fires in Boston has confirmed the complexity of the exposure. Carbon monoxide and acrolein are the most hazardous specific air contaminants in this series of fires, while combinations of respiratory irritants (e.g., acrolein, hydrogen chloride, and nitrogen dioxide) acting in a synergistic manner may be important in selected fires. The combined effects of mixtures of these and other irritants, especially in the presence of high particulate concentrations, often make effective work by the firefighter impossible without respiratory protection. The concentrations of hydrogen cyanide and

carbon dioxide, and the degree of oxygen deficiency were not found to be significant health hazards in this study. This study confirms earlier work by this laboratory which indicates that firefighters should wear respirators at all structural fires.

A limited number of fires were sampled both during active firefighting operations and the subsequent overhauling. This allowed us to compare the data so as to ascertain the relative severity of the exposure once the fire has been "knocked down."

A statistical analysis of these data showed that:

1. The concentrations of HCl and particulates are higher during firefighting than during overhauling;
2. There was no significant difference between the concentrations of NO_2, HCN, or benzene during firefighting versus overhauling; and
3. Insufficient data were available to reach conclusions regarding acrolein, CO, and CO_2.

There are several cautions which must be considered before extrapolating these data and conclusions to other situations.

All of the fires monitored were located in one city, Boston, and fought by the Boston Fire Department. Other cities may have a predominance of newer structures built with different materials and containing different furnishings. In addition, other fire departments may not employ the same firefighting techniques as those used in Boston. Due to the nature of the sampling protocol, it was impossible to measure gas concentrations during the initial stages of the fires. Another constraint was the limited number of contaminants identified to be sampled.

Furthermore, the fires sampled in the study were predominantly structural in nature. Other situations, such as fires involving chemicals, train tunnels, vehicles, and brush would probably present an entirely different contaminant profile.

These concentration data represent environmental conditions encountered by working firefighters, usually wearing self-contained breathing apparatus. These are not, nor should they be considered to be, exposure levels.

References

1. Sidor, R. and J. Peters, "Firefighting and Pulmonary Function," Am. Rev. Resp. Dis., 109:249-55 (1974).

2. Sidor, R. and J. Peters, "Prevalence Rates of Chronic Non-Specific Respiratory Disease in Fire Fighters," Am. Rev. Resp. Dis., 109:255-61 (1974).

3. Gempel, R.F. and W.A. Burgess, Thermal Environment During Structural Firefighting, Final Report to NFPCA, 1977.

4. Sidor, R., N.H. Peterson, and W.A. Burgess, "A Carbon Monoxide-Oxygen Sampler for Evaluation of Fire Fighter Explosures," Amer. Ind. Hyg. Assoc. J., 34:264 (1977).

5. Burgess, W.A., R. Sidor, J.J. Lynch, P. Buchanan, and E.V. Clougherty, "Minimum Protection Factors for Respiratory Protective Devices," Amer. Ind. Hyg. Assoc. J., 38:18 (1977).

6. Gold, A., W.A. Burgess and E.V. Clougherty, "Exposure of Firefighters to Toxic Air Contaminants," Amer. Ind. Hyg. Assoc. J., 39:534 (1978).

7. Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1978, American Conference of Governmental Industrial Hygienists (1978).

8. Personal Communication, L. Partridge, Arthur D. Little, Inc.

9. Terrill, J.B., R.R. Montgomery, C.F. Reinhardt, "Toxic Gases from Fires," Science, 200:1343 (1978).

10. Ives, J.M., E.E. Hughes, and J.K. Taylor, Toxic Atmospheres Associated with Real Fire Situations, National Bureau of Standards, Report # 10807, 1972.

11. Stone, J.P., R.N. Hazlett, J.E. Johnson and H.W. Carhart, "The Transport of Hydrogen Chloride by Soot from Burning Polyvinyl Chloride," J. Fire and Flam., 4:42 (1973).

12. Tsuchiya, Y., "Significance of HCN Generation in Fire Gas Toxicity," J. Comb. Tox., 4:271 (1977).

13. Morikawa, T., "Acrolein, Formaldehyde and Volatile Fatty Acids from Smoldering Combustion," J. Comb. Tox., 3:135 (1976).

14. Zikria, B.A., G.C. Weston, M. Chodoff and J.M. Ferrer, "Smoke and Carbon Monoxide Poisoning in Fire Victims," J. Trauma, 12:641 (1972).

15. Goldsmith, J.R. and S.A. Landaw, " Carbon Monoxide and Human Health," Science, 162:1352 (1968).

substances, and sometimes in covering radiation diseases for the first time.

§ 41.72 Special statutes on heart and respiratory diseases of police and firefighters

An interesting recent phenomenon has been the burgeoning in all parts of the country of statutes granting special compensation coverage to firemen or policemen or both, for respiratory and heart [3.1] diseases connected with the exertions of the employment.[4] No two are quite identical. Most establish a

(*Text continued on page 7-454*)

[3.1] California has a similar statute for hernias. See Smith v. WCAB, 45 Cal. App. 3d 162, 119 Cal. Rptr. 120 (1975), § 41.72(e) *N.* 11.6 *infra*.

[4] The states having some kind of special statute in this category include: Alabama, California, Connecticut, Florida, Maine, Maryland, Michigan, Minnesota, Nevada, New Hampshire, New Jersey, North Dakota, Ohio, Oregon, Pennsylvania, South Carolina, Vermont and Wisconsin.

In several of these states, there may be some question whether the system is an intrinsic part of the workmen's compensation system, as distinguished from a separate pension program. Wisconsin, for example, was met with this question directly, and found the presumption to be part of workmen's compensation. See: City of Manitowoc v. Iowa National Mutual Insurance Co., 68 Wis.2d 722, 229 N.W.2d 577 (1975). Iowa National refused to indemnify its insured, City of Manitowoc, for survivor's benefits paid under a statute allowing a fireman's widow compensation for any work-related disease causing the death of her husband. Iowa National's argument was that the special statute for firemen was a pension statute rather than a compensation one and therefore outside the policy coverage. The court ordered payment by the insurer, rejecting this argument.

As to the Alabama situation, see the following cases:

City of Tuscaloosa v. Howard, 55 Ala. App. 781, 318 So.2d 729 (1975). The claimant, a 20-year veteran of the fire department, experienced chest pains while performing housekeeping duties at the fire station. He left work at the end of the shift and reported to his second job. He later sought medical attention. After two weeks of hospitalization, the physician determined that during the preceding week the claimant had suffered a heart attack. He was hospitalized for a total of a month and was not permitted to return to his duties for two and one-half additional months. The claimant presented his claim under the Workmen's Compensation Act under three alternative theories: first, that as a city employee, he had suffered a compensable injury while in the scope of his employment; second, that he was entitled to compensation under occupational disease provisions (Title 26, Chapter 7,

Article 2(C); third, that he was entitled to workmen's compensation under the Fireman's Heart and Lung Disability Act. The trial court found compensation due under each theory. The Court of Civil Appeals reversed and remanded, stating that, as a matter of law, the claimant could not recover workmen's compensation benefits under any of the theories. First, the Court held there was no accident within the definition of the Act, because there was no evidence that the strain and exertion of the claimant's work caused the injury. Second, the Court ruled, as a matter of first impression, that myocardial infarction resulting from progressive arteriosclerotic heart disease was not a hazard of employment of a fireman, was not particular to that occupation, and did not result from the nature of the employment. Finally, the Court held that benefits payable under the Fireman's Act are to come from separate pension funds, and are separate from and need not be identical in amount to workmen's compensation benefits.

Norris v. Seibels, 353 So.2d 1165 (Ala. 1977), 353 So.2d 1169 (Ala. App. 1978). Under the provisions of Title 37, section 450(4), all municipal firemen within the state were eligible for benefits for heart disease and hypertension as if injured in the course of their employment. The law including this section was applicable only to firemen. Subsequently, an amendment to the municipal pension laws excluded municipal firemen in cities with over 250,000 inhabitants (i.e. Birmingham) from the coverage of § 450(4). The Court of Civil Appeals held that the amendment did not violate the equal protection clause of the United States Constitution. On appeal, the judgment was reversed. The court held that there was no reasonable relationship between the classification and purpose of the pension law, to provide protection from disability to firemen disabled by occupational disease.

See also: Mitchell v. Public Employes' Retirement Board, 28 Or. App. 339, 559 P.2d 1325 (1977). The court held that the refusal to apply a Workmen's Compensation Act presumption—that a heart attack is related to a fireman's employment—to the Public Employes' Retirement system was proper.

See also, as to the Pennsylvania coordination problem:

Commonwealth v. Oil City, 15 Pa. Cmwlth. 544, 328 A.2d 170 (1974). The claimant had been employed as a fireman with the City Fire Department for approximately twenty years. In November, 1970, while fighting an especially tragic fire, the claimant had suffered a heart attack and incurred right lobar pneumonia. The incident resulted in temporary, total disability from that time until May, 1971, when the claimant returned to duty. The claimant's heart disease came within the provisions of the Pennsylvania Occupational Disease Act. He was also entitled to compensation payments during the period of temporary disability to be paid by the city. The city continued to pay the claimant his full rate of salary. The city was only required to pay $60 per week, which is the amount payable under the Occu-

presumption of work connection when these diseases result from performance of active service.

The commonest controversy generated by these statutes has centered around the strength of that presumption. The best way to measure this strength is by the negative test of how much it takes to rebut or overcome the presumption.

§ 41.72(a) Rebutting the presumption of work-connection under police and firefighter statutes.

The possible grounds for rebutting the presumption vary so widely that the end product varies from a virtually irrebuttable to a virtually worthless presumption.

In California (by statute), Michigan, and Tennessee, the presumption cannot be rebutted merely by evidence of preexisting heart disease.[5] Michigan and Wisconsin even hold that

(*Text continued on page 7-456*)

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pational Disease Act. The city did not pay the claimant's hospital, doctor, and drug bills. The claimant filed a claim seeking compensation under the Occupational Disease Act. The referee held that compensation under the Act should be suspended until a loss of earnings was shown by the claimant. It also held that the Commonwealth was liable for all hospital, medical, and drug bills. On appeal, the Board held that the Commonwealth was liable not only for medical expenses, but also for occupational disease compensation at the rate of $60 per week. It held that the city was subrogated to receive all said payments. The Commonwealth Court affirmed. It held that, pursuant to a 1965 amendment to the Occupational Disease Act, the Commonwealth was primarily liable for compensation to firemen who suffer from heart and lung diseases. When a city has made payments to a fireman under the Heart & Lung Act for a disability also compensable under the Occupational Disease Act, that city is entitled to be reimbursed to the extent the fireman is entitled to compensation under the Occupational Disease Act.

[5] "Such heart trouble or pneumonia so developing or manifesting itself in such cases shall in no case be attributed to any disease existing prior to such development or manifestation." Cal. Labor Code § 3212.5. This passage was added by amendment in 1959.

Turner v. Workmen's Compensation Appeals Bd., 258 Cal. App. 2d 442, 65 Cal. Rptr. 825 (1968). California provides that for policemen with five years' service, heart trouble developing or manifesting itself while the officer is in the service of the police department is presumed to arise out of and in the course of the employment, and that such heart trouble can not be

attributed to preexisting disease. Claimant, a police officer, suffered a heart attack while on duty, and the only medical evidence presented indicated that the attack was due to preexisting heart disease, and not in any way connected with his work. *Held:* Presumption of compensability could not be rebutted by showing of preexisting heart disease. Denial of compensation reversed.

Bussa v. Workmen's Compensation Appeals Bd., 259 Cal. App. 2d 261, 66 Cal. Rptr. 204 (1968). Decedent, a fireman, suffered a fatal heart attack working at a second job. The attack was found to have been caused by preexisting atherosclerosis, and benefits were denied. The denial was reversed, on the ground that since claimant was a fireman, a heart attack occurring during his period of service was presumed to be compensable, and this presumption could not be rebutted by attributing the attack to any preexisting disease.

City and County of San Francisco v. Workmen's Comp. App. Bd., 49 Cal. App.3d 659, 122 Cal. Rptr. 599 (1975), aff'd, 583 P.2d 151 (1978). The claimant's decedent was employed as a San Francisco police officer from 1943 to 1968. When he suffered a fatal heart failure in 1972, his widow and minor daughter claimed benefits from the city. The city challenged a state statutory presumption that a policeman's heart trouble arose out of and in the course of his employment. Medical testimony was introduced to the effect that the decedent's condition was progressive and not conclusively related to his employment. The court held that the statutory presumption was not a "denial of due process" nor "an unconstitutional invasion of the municipality's domain" by the state. The court also gave effect to an amendment providing that the statutory presumption "cannot be rebutted by a pre-existing heart disease." Benefits were awarded. **Treatise** cited in majority opinion and dissent. See further reference to the constitutional issue, which focused entirely on the passage quoted at the beginning of this note, at § 41.72 (e) *infra*.

Michigan: Schave v. Department of State Police, 58 Mich. App. 178, 227 N.W.2d 278 (1975). The claimant was a policeman who alleged his heart condition was a result of his employment. The court ruled that the statutory words "in the absence of evidence to the contrary" did not allow the presumption in the plaintiff's favor to be rebutted by evidence of preexisting heart disease or medical opinion that the occupation had no effect. In order to avoid the presumption, the defendant must introduce evidence of nonwork related causation. **Treatise** quoted.

Tennessee: City of Oak Ridge v. Campbell, 511 S.W.2d 686 (Tenn. 1974). The employee, a city policeman, suffered a myocardial infarction while performing his duties. He sought a 100% disability classification, which was challenged by employee's employer and employer's insurance carrier on the ground that the injury producing the disability was not an " . . . injury by

the presumption cannot be rebutted by medical opinion that the occupation had no effect on the weakened heart.[6] How then can the employer rebut the presumption? Michigan, in the case[6.1] holding that neither of these two[6.2] showings are sufficient, provided the answer: by affirmative proof of non-occupational causation. It is interesting to note that Michigan achieved this result under a statute that creates the presump-

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accident arising out of and in the course of employment . . . " The Court held that the employee was entitled to the 100% disability classification because the employee satisfied the statutory criteria which provided a presumption that the disability occurred " . . . due to accidental injury suffered in the course of employment." The statutory criteria which employee satisfied which gave the employee the presumption were: (1) the disabled employee was employed by a regular law enforcement department manned by full-time employees; (2) the employee suffered a disability resulting from hypertension or heart disease; and (3) prior to such claimed disability, the individual had had a physical examination which did not reveal heart disease or hypertension. The Court also held that the presumption was not rebutted by the fact that a doctor's testimony established that employee was suffering from arteriosclerotic heart disease in the day in question, and that the medical doctor would not have recommended that the employee pursue his regular nighttime duties, at which time the infarction occurred.

[6] *Michigan:* Schave v. Dept. of State Police, N. 5 immediately *supra* under *Michigan.* **Treatise** quoted.

Wisconsin: Sperbeck v. Department of Indus., Labor & Human Relations, 46 Wis. 2d 282, 174 N.W.2d 546 (1970). Wisconsin provides a rebuttable presumption that a fireman who sustains a heart attack under certain circumstances has had a compensable heart attack. The employer sought to rebut the presumption through the testimony of a doctor who was of the opinion that the occupation of a fireman had no effect on the arteriosclerotic heart disease that resulted in decedent's heart attack. The court held that this was not the type of testimony that could rebut the presumption, since in enacting the legislation that created the presumption, the legislature had rejected the school of medical opinion espoused by this doctor.

[6.1] Schave v. Dept. of State Police, N.5 *supra* this subsection under *Michigan.* **Treatise** quoted.

[6.2] When only one of the two showings is ruled out by statute, as the first is in California, the Supreme Court of California has held that this limited trimming of the scope of rebuttal does not create a constitutionally questionable "irrebuttable presumption." City and County of San Francisco v. WCAB, N.5 *supra* this subsection under *California.* See further discussion of the constitutional point *infra* at § 41.72(e).

tion "in the absence of evidence to the contrary." This "contrary" evidence, then, can not be merely negative; it must be positive proof of a cause independent of the employment.

The intermediate position is represented by the rule in Louisiana[6.3] and New Hampshire.[6.4] In those states the presump-

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[6.3] Vincent v. City of New Orleans, 326 So.2d 401 (La. App. 1975), writ denied, no error, 329 So.2d 760 (1976). Act 337 of 1968 provides that: "Any disease or infirmity of the heart or lungs which develops during a period of employment in the classified fire service of Louisiana shall be classified as a disease or infirmity connected with the employment . . . Such disease or infirmity shall be presumed, prima facie, to have developed during the employment whenever same is manifested at any time after the first five years of employment." In a suit to recover permanent and total disability, a 14-year veteran fireman alleged that his duties caused or accelerated the cardiovascular disease and angina pectoris which resulted in his retirement. The employer offered no proof to rebut the testimony of the claimant's expert witness, who stated that there was a direct correlation between the physical and emotional stress of the claimant's job and his disease. The Court of Appeal affirmed an award of benefits, stating that the statute shifts the burden of proof to the employer, who must prove the lack of causation between the disease and the employment. The Court concluded that the City had failed to prove the negative. Act 30 of 1975, passed after this decision, specifically provides that manifestation of heart or lung disease after five years employment is presumed not only to have developed during the employment, but also is presumed to have been caused by or to have resulted from the nature of the employment.

See also: Buse v. City of New Orleans, 349 So.2d 397 (La. App. 1977). The plaintiff was employed as a fireman. In 1965 he sustained a knee injury, which required him to miss over one year of work. He brought suit for compensation in 1968, alleging that the injury was work-related but had not manifested itself until 1967. In 1973 he amended his complaint, alleging total permanent disability from obstructive lung disease resulting from the inhalation of smoke at a marsh fire. The plaintiff had a pre-existing bronchitis condition. The plaintiff's treating physician testified that he thought the marsh fire had at least aggravated the plaintiff's condition. The trial court awarded permanent total disability benefits. On appeal, the defendant contended that the amendment to the old complaint should have not been allowed, because it stated a cause of action arising after the date the complaint was filed, and that the evidence was not sufficient for a finding that the plaintiff's lung condition was related to his work. The judgment was affirmed. The court held that the defendant was not prejudiced by the amendment, even though its contention was technically correct. The plain-

tion shifts the burden of proof as to employment causation, and it is possible for the employer to rebut the prima facie case based on the presumption by proving a negative: that the employment did not contribute to the injury.

At the other extreme is the rule in Oregon, which is accounted for to some degree by the particular choice of words in the statute, which speaks of a "disputable" presumption. Oregon has taken the position that if the employer presents any evidence challenging the work-connection of the injury, the presumption has been "disputed";[6.5] apparently this puts

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tiff was not entitled to the statutory presumption that his lung condition developed during his firefighting performance, because it developed prior to rather than after his first five years of firefighting work. Nevertheless, the court held that the plaintiff had proved his case by a preponderance of the evidence.

[6.4] New Hampshire Insurance Co. v. Duvall, 337 A.2d 533 (N.H. 1975). A New Hampshire statute established a prima facie presumption that heart or lung disease in a firefighter is occupationally related. Pursuant to the statute, a labor commission regulation required insurance carriers to pay compensation within ten days after a fireman proved that he had such a disease, unless the carrier offered rebutting medical evidence (defined as evidence that the "disease is not work related") within that time. The court construed the statute to permit, rather than to require, the commissioner to order such payments within ten days. It held that the regulation, insofar as it provided insufficient time for an insurance carrier to obtain rebuttal evidence and allowed no extensions of time, unconstitutionally deprived carriers of their property without due process of law.

[6.5] Norris v. State Accident Insurance Fund, 27 Or.App. 623, 557 P.2d 61 (1976). The claimant, a fireman, suffered a myocardial infarction at home after experiencing chest pains at work that day. In Oregon, claimant-firemen are aided by a disputable presumption that a heart attack is work-related. In this case, there was opposing medical testimony that the attack was not work-related. The court held that this testimony disputed the presumption, and that the claimant had failed to meet his burden of proving work-relation. Therefore benefits were denied.

Pflughaupt v. State Accident Insurance Fund, 26 Or.App. 77, 552 P.2d 284 (1976). The claimant, a fire chief, suffered a myocardial infarction at home. He filed a claim for benefits based on a statutory "disputable presumption" of work-connection for certain occupational diseases occurring in firemen with a certain tenure. The court held that the testimony of two cardiologists that there was no causal relation between the attack and his

matters right back where they would have been without the presumption, and the claimant must proceed to prove his case in the usual way. The only usefulness the presumption would have for the claimant, then, would be to support an award if no opposing evidence whatever was offered by the employer. Since it would be a rare case in which the employer could not find some testimony to "dispute" the presumption in this sense, the characterization of this kind of presumption at the outset of this subsection as "virtually worthless" does not seem exaggerated.

Expressions can also be found in Minnesota cases suggesting that a roughly similar rule has been accepted there. Thus, in *Jerabek v. Teleprompter Corp.*,[7] the court stressed that the presumption was only a "rule of law dictating decision on unopposed facts."

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employment was sufficient to dispute the presumption. Affirmative proof of what did cause the attack was not required to dispute. Thus, with the presumption gone, the claimant had no case and benefits were denied.

[7] 255 N.W.2d 377 (Minn. 1977). The claimant, a 25-year veteran of the City of Rochester fire department and part time employee of Teleprompter, experienced severe chest pains while working for Teleprompter. He had experienced less severe pain the previous day while at the firehouse. At the hearing, two doctors testified. They agreed that the employee had suffered one myocardial infarction, but disagreed as to any relation between the infarction and the claimant's job as a firefighter. The compensation judge found that the claimant's personal injury arose out of and in the course of both employments equally, and held each employer liable for a percentage of benefits equal to the percentage which each had contributed to the total pre injury earnings. On appeal, the Worker's Compensation Court of Appeals found that the claimant had performed "arduous duties" in participating in firefighting activities which led to two myocardial infarctions. Consequently, it assessed all disability payments against the city. The Supreme Court reversed, citing a lack of evidence that there were two infarctions. The court noted that there was substantial evidence to rebut the statutory presumption, embodied in Minn. St. 176.011, subd. 15, that a fireman's heart failure was occupational. "We have frequently characterized this statutory presumption as a *rule* of evidence. It is not evidence. It is, rather, 'a rule of law dictating decision on unopposed facts. . . .'" 255 N.W. at 380.

§ 41.72(b) What persons are covered

The typical statute covers police or firefighters or both. Inevitably there has been pressure on legislatures to extend the statutes to categories of employees similar or related to these. Thus, Minnesota started with members of organized fire departments in 1955;[8] in 1957 added members of organized police departments;[9] then in 1959 the highway patrol;[10] and in 1963, members of the game warden service and State Crime Bureau.[11]

The courts, however, have displayed no disposition to enlarge the protected circle by stretching the statutory language beyond its normal meaning. Thus, when California extended its police coverage to embrace campus policemen in the University of California system, the court declined to interpret it as covering California State University policemen.[11.1] Similarly, it refused to stretch coverage of full-time salaried deputy sheriffs to include a deputy coroner who was also a deputy sheriff.[11.2]

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[8] Minn. Laws 1955, Ch. 206.

[9] Minn. Laws 1957, Ch. 854. See also Minn. Laws 1955, Ch. 34, with special compensability of tuberculosis of policemen due to contact with the disease. Gray v. City of St. Paul, 250 Minn. 220, 84 N.W.2d 606 (1957), awarded compensation to policeman who had intermittently ridden in a police car with a fellow officer who had tuberculosis.

[10] Minn. Laws 1959, Ch. 20.

[11] Minn. Laws 1963, Ch. 497.

[11.1] Saal v. Workmen's Comp. Appeals Bd., 50 Cal. App.3d 291, 123 Cal. Rptr. 506 (1975). The claimant was a campus police officer employed by the California State University. After suffering a heart attack, he claimed benefits based upon statutory provisions that the heart trouble of certain classes of policemen was presumed to have arisen out of and in the course of their employment. The special provisions had been extended to cover campus policemen in the University of California system, but not California State University. The court held that the presumption did not apply to the claimant. Noting the "wide discretion" of the legislature to make classifications, the court found that the legislature "has proceeded step-by-step in conferring additional benefits upon certain peace officers with respect to certain employment hazards."

A question may arise, when statutes speak of "firefighters" or of "active firefighters," whether clerical or sedentary workers connected with the fire department are meant to be included. Pennsylvania has answered this question in the negative, in the case of a dispatcher, on the ground that his work did not expose him to the kind of special hazard contemplated by the statute.[11.3] California faced the same problem as to a maintenance employee, who, however, was required to attend fires, and held that coverage at least could not be summarily ruled out.[11.4]

§ 41.72(c) Effect of leave or retirement

Once the firefighter or police officer is within the coverage of the special presumption statute, he does not, according to the reported cases that have addressed the issue, lose the bene-

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[11.2] State Compensation Ins. Fund v. Workmen's Compensation Appeals Bd., 251 Cal. App. 2d 772, 59 Cal. Rptr. 760 (1967). Claimant worked as deputy coroner out of the coroner's office, and was also sworn in as a deputy sheriff, but was issued a sheriff's identification card marked "non-salaried." He then developed heart disease, which in the case of a "deputy sheriff employed upon a regular full-time salary" is compensable. The court denied coverage, stating that even if it was found that claimant was a salaried deputy, he could not be considered full-time in view of his primary duty as deputy coroner, and therefore was not one of those the legislature intended to benefit by this provision.

[11.3] Andes v. City of Lancaster, 23 Pa. Cmwlth. 56, 350 A.2d 457 (1976). The claimant, a dispatcher for the last four years and eight months of his employment with the fire department, sought occupational disease benefits after suffering a heart attack. The Section 108(o) of the Occupational Disease Act provided benefits for "diseases of the heart and lungs resulting in . . . disability or death, after four years of more of service in fire fighting." The court held that the claimant was ineligible for benefits because his employment did not involve exposure to the hazards of occupational disease.

[11.4] Buescher v. Workmen's Compensation Appeals Bd., 265 Cal. App. 2d 520, 71 Cal. Rptr. 405 (1968). California applies a presumption of compensability to heart trouble which develops or manifests itself during a period of service as an active fire fighter within the Division of Forestry. Decedent was employed by the Division to maintain equipment, and was required to be present at all fires in his district. Claimant held entitled to determination whether decedent was an "active fire fighter."

fit of that presumption when he is on vacation,[11.5] on disability leave, or in retirement.

California has held that, even under a statute requiring that a hernia, to be within the presumption, must manifest itself while the fireman is "in the service" of the department, the presumption applies to a fireman who sustained a hernia while on disability leave, who had intended to retire at the expiration of the leave, and who had actually moved away from the city.[11.6]

California has also applied its heart statute to an already-retired policeman who suffered a heart attack two months after the statute creating the presumption was passed.[11.7]

[11.5] Schwartz v. City of Duluth, 264 Minn. 514, 119 N.W.2d 822 (1963). Fireman's fatal coronary thrombosis while on vacation held compensable as causally related to coronary sclerosis, an occupational disease developed during the course of his employment as a fireman.

[11.6] Smith v. Workmen's Comp. App. Bd., 45 Cal. App. 3d 162, 119 Cal. Rptr. 120 (1975). A city fireman sustained a back injury during the course of his employment. On the basis of his back disability, he received full-pay disability status for one year. He spent a full year in this status and retired at the year's end after 28 years of service. He physically moved from the city at the beginning of the one-year disability period. During the eleventh month of the disability period, he sustained a hernia. He applied for workmen's compensation benefits for expenses related to this condition. His application was the granted by the referee. On reconsideration, the board denied his application on the grounds that the injury was not work-related. At the referee's hearing, the city offered no medical evidence that the hernia did *not* arise in the course of his employment. The court of appeals reversed the Board's decision, finding that the fireman was "in the service" of the city fire department within a statute raising a presumption that a hernia, which manifests itself while fireman is "in the service" arose out of and in course of employment. The fact that the fireman had physically moved from the city, that he intended to retire permanently at the end of the disability period, and that the hernia manifested itself when the disability period had almost expired were not sufficient to rebut the statutory presumption.

[11.7] Kaiser/Permanente v. Workers' Comp. Appeals Bd., 61 Cal. App.2d 408, 132 Cal. Rptr. 96 (1976). A state police officer retired in 1972 after 23 years of service. In 1973, the legislature created a statutory presumption that a policeman's heart trouble arose out of and in the course of his em-

§ 41.72(d) What injuries are covered

California has encountered some difficulty in construing the broad term "heart trouble" in its statute. In its most expansive holding, *Muznik v. WCAB*,[11.8] it concluded that the term should include a fireman's hypertension. Two years later, however, in *Coyne v. WCAB*,[11.9] it stopped short of extending the term to cerebral vascular stroke. The court had to distinguish both *Muznik* and the earlier case of *Stephens v. WCAB*,[11.10] which had reversed and remanded a refusal to apply the presumption to acute and chronic arteriosclerotic disease. The key to the distinction was that in *Coyne* the disability, although traceable to the arteriosclerotic disease, manifested itself as the result of the independent process of that disease in a part of the body removed from the heart.

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ployment. Two months later, the retired policeman suffered a heart attack. Finding sufficient evidence to establish that the heart trouble developed while he was a policeman, the court held that the officer was entitled to the benefit of the presumption. The court stated that its holding "does not result in retroactive application of the statute" under the circumstances.

[11.8] 51 Cal. App. 3d 622, 124 Cal. Rptr. 407 (1975). The claimant was a fireman for 33 years. California law provided that a fireman's "heart trouble" was compensable because it was presumed to arise out of and in the course of his employment. The court held that "the phrase heart trouble assumes a rather expansive meaning," and included the claimant's hypertension within its scope. In reversing the appeals board's denial of coverage based upon "an unduly restrictive definition," the court found that an expansive definition of the phrase was needed to effect the legislative intent.

[11.9] 69 Cal. App.3d 770, 138 Cal. Rptr. 373 (1977). The petitioner, a city-employed fireman, sustained a cerebral vascular stroke while at home, which resulted in total permanent disability. The medical evidence indicated that he had previously suffered from arteriosclerotic occlusive disease attributable to such non-industrial factors as inheritance and the aging process, and that the stroke would have occurred when it did irrespective of any prior activities. The court held that the statutory presumption of "heart trouble" arising out of and in the course of employment as a fireman was inapplicable, since the petitioner's disability manifested itself from the independent process of his arteriosclerotic disease in a part of the body removed from his heart. The Board's denial of compensation benefits was affirmed.

[11.10] 20 Cal. App. 3d 461, 97 Cal. Rptr. 718 (1971).

California had also held, in the earlier case of *Baker v. WCAB*,[11.11] that cardiac neurosis caused by the stresses of a fireman's work was not a true heart attack and did not involve heart disease. The impact of the decision was softened, however, by the conclusion that a case for compensable injury had been made independently of the presumption.[11.12]

[11.11] 18 Cal. App. 3d 852, 96 Cal. Rptr. 279 (1971). The claimant had been a full-time fireman for 26 years. In 1953, he had one particularly severe exposure to acrid fumes, and suffered severe chest pains, and expectoration of blood and mucous. The symptoms recurred with progressive intensity and frequency thereafter. By 1968, he complained that his condition had become so disabling that he could no longer work. The referee found that the claimant had developed heart disease in the course of his employment and awarded benefits accordingly. The Board on reconsideration found that claimant did not have heart trouble and reversed the award. The court of appeals agreed with the Board that there was ample evidence for a finding that the claimant had not suffered a true heart attack. However, the court found that claimant was in fact disabled from what appeared to be "cardiac neurosis" caused by the stresses and anxieties of his employment experience, and that an award on this basis was in order. Since the claimant was a fireman, there was injected into the case the argument that, by Labor Code § 3212, a fireman's heart attack is presumed to arise out of his employment. This presumption obviously was out of place here, since no heart disease was involved. The presumption relates to causation of heart disease, once it is established that heart disease is the injury involved. The presumption does not serve to aid in proving that the disability itself was in fact produced by heart trouble. Treatise cited.

[11.12] The claimant can, of course, if he prefers, prove his case without the aid of the statute.

Beckham v. City of New Orleans, 327 So.2d 460 (La. App. 1976), writ denied, no error, 332 So.2d 278 (1976). A fireman, who was suffering from coronary artery disease and angina pectoris, became dizzy and experienced chest pains while fighting a fire. A medical expert testified that the claimant's activities prior to fire-fighting played a major part in causing the heart condition. The court held that the disability was compensable under the workmen's compensation statute, even though occasioned by usual and customary activities connected with the employment. Since the claimant proved his case under the workmen's compensation statute, the court held that he need not rely on the Firefight's Heart and Lung Bill.

See also New Hampshire Ins. Co. v. Duvall, 337 A.2d 533 (N.H. 1975) § 41.72 N.4 *supra*, holding that a regulation issued under the New Hampshire statute was unconstitutional insofar as it required carriers to begin

§ 41.72(e) Constitutionality of special police-firefighters statutes

The first state to consider the constitutionality of a special police-firefighter type of statute, North Carolina, found it unconstitutional as class legislation.[12]

Since then, three states—California,[12.1] Connecticut,[12.2] and Maryland[12.3]—have held such provisions constitutional, and Alabama has also upheld its more separate Firemen's Heart and Lung Disability Act, although its coverage was constricted by an amendment excluding, in effect, firemen in Birmingham.[12.4]

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payment in these cases within ten days unless within that time they offered evidence that the disease was not work related.

[12] Duncan v. City of Charlotte, 234 N.C. 86, 66 S.E.2d 22 (1951). Commission had awarded benefits for death of a fireman which occurred on his vacation.

[12.1] City and County of San Francisco, v. W.C.A.B., 583 P.2d 151 (Cal. 1978), § 41.72(a) *supra*. Treatise cited in majority opinion and dissent. The opinion contains an excellent analysis of the background and justification for this type of statute, and of the various constitutional issues — including the constitutionality of alleged "irrebuttable presumptions."

[12.2] Grover v. Town of Manchester, 357 A.2d 922 (Conn. 1975), *app. dism'd* 44 L.W. 3182 (1975). The court held that a special statute on heart and hypertension cases of police was not unconstitutional.

[12.3] C. Colgan v. Board of County Comm'rs for Prince George's County, 21 Md. App. 331, 320 A.2d 82 (1974), aff'd, 274 Md. 193, 334 A.2d 89 (1975). The appellant, a fire fighter, claimed workmen's compensation benefits under a statute which provided that any impairment of health of a fire fighter caused by lung and heart disease could be presumed to have been suffered as a result of employment. The Workmen's Compensation Commission dismissed the claim. The circuit court affirmed, finding that the statute was in violation of the Constitution of Maryland. The Court of Special Appeals reversed, holding that the statute did not violate the state constitution. It held that the statute did not violate due process or equal protection clauses of the federal constitution.

[12.4] Norris v. Seibels, 353 So.2d 1165 (Ala. App. 1977), 353 So.2d 1169 (Ala. App. 1978), § 41.72 N.4 *supra*.

Alaska State Legislature

Senate

Official Business

Pouch V
State Capitol
Juneau, Alaska 99811

TO: Senator Richard Eliason
FROM: Senator Joe Josephson
DATE: February 27, 1984

RE: SB 262, Presumptions/Alaska Worker's Compensation

SB 262 relating to Presumptions in Alaska Worker's Compensation law relating to firefighters, police and emergency medical personnel, has been assigned to the Senate Labor and Commerce Committee.

This bill is very important and I would like to see it moved as soon as possible. I will be happy to meet with you at any time to discuss concerns you may have.

SB262
456-8354
Barry Haight
3/22/84

Glenn C. Smith
P.O. Box 874421
Wasilla, Alaska 99687

Senator Eliason
Pouch V
Juneau, Alaska 99811

Re: Sponsor Substitute for Senate Bill 262 in the Legislature of the State of Alaska Thirteenth Legislature - Second Session

Dear Senator Eliason:

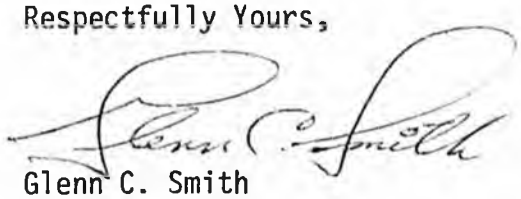
I would like to express my personal opinion on Senate Bill 262 proposed by Senator Josephson and Rodey.

As you know, this relates to "an act relating to presumption under the Alaska Workers' Compensation Act", Section AS 23.30.120. I would assume that there is a statistical base to support a bill that there is a rebuttable presumption that firefighters and policemen have a greater frequency of heart or lung disease, to include hypertension. I would at this time, submit a request for that statistical base to support Bill 262.

Judging from a laymen's observation of the physical condition of long term firefighters and policemen, I have some doubt that heart or lung disease, to include hypertension, is job related but rather self-inflicted due to neglect by the individuals, when in fact, they are called upon to be not only mentally alert but physically capable of performing their duties. Beyond that, as stated, I know of no statistical base to support the fact that heart or lung disease, to include hypertension, is more prevalent in firefighters or policemen than in general labor, trucking industry, warehousemen, or for that fact, senators.

To impose this type of presumption in addition to including, "time immediately following the termination of employment", means a period of time, not to exceed 60 months would, I believe, cause undue litigation expense in addition to liability expense to municipalities throughout the State not to mention volunteer departments in small communities. This expense would ultimately be levied back against each and every individual within the State of Alaska. I look forward to hearing from you regarding this proposed bill.

Respectfully Yours,



Glenn C. Smith

GCS/fm