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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:

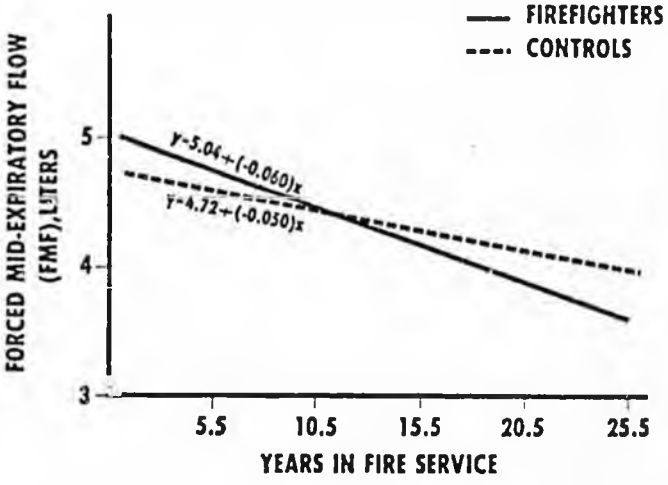
1. The vital capacity, forced vital capacity and flow rates decreased in firefighters as in controls but the firefighters mean values declined at a faster rate; even though the test population excelled the control population in height and weight.

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.

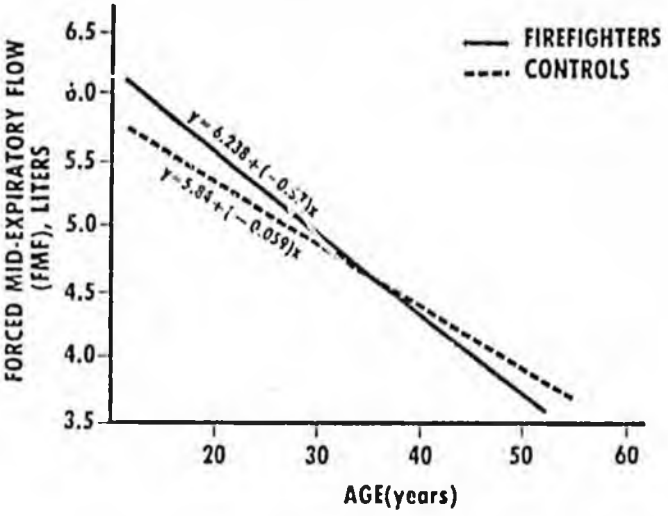
3. The observed differences between the test and control groups could best be attributed to occupational exposure as indicated by reduced volumes suggestive of airway obstructive and restrictive diseases especially when considered in light of the fact that the worst cases have been retired.

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

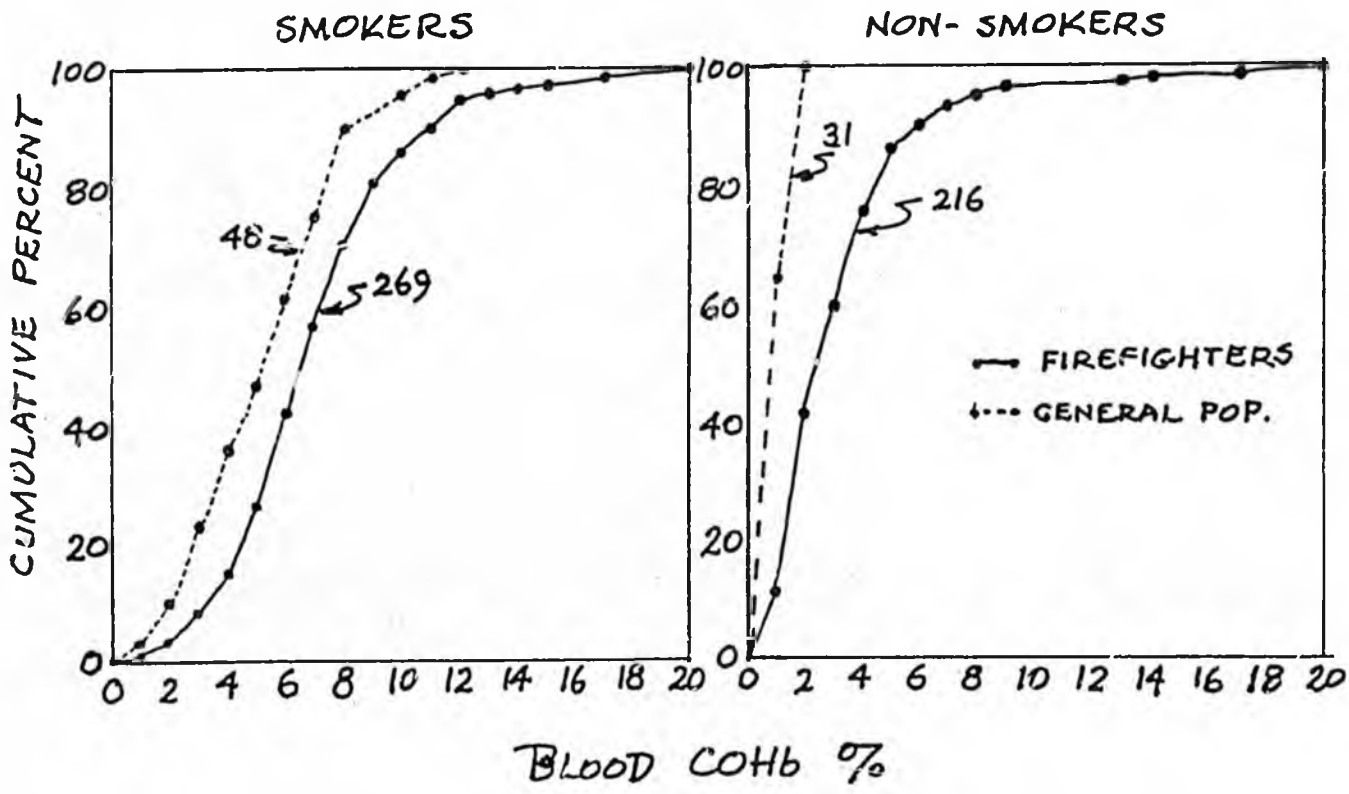


FIGURE 1—Cumulative Frequences of Blood COHb—Firefighters and Controls

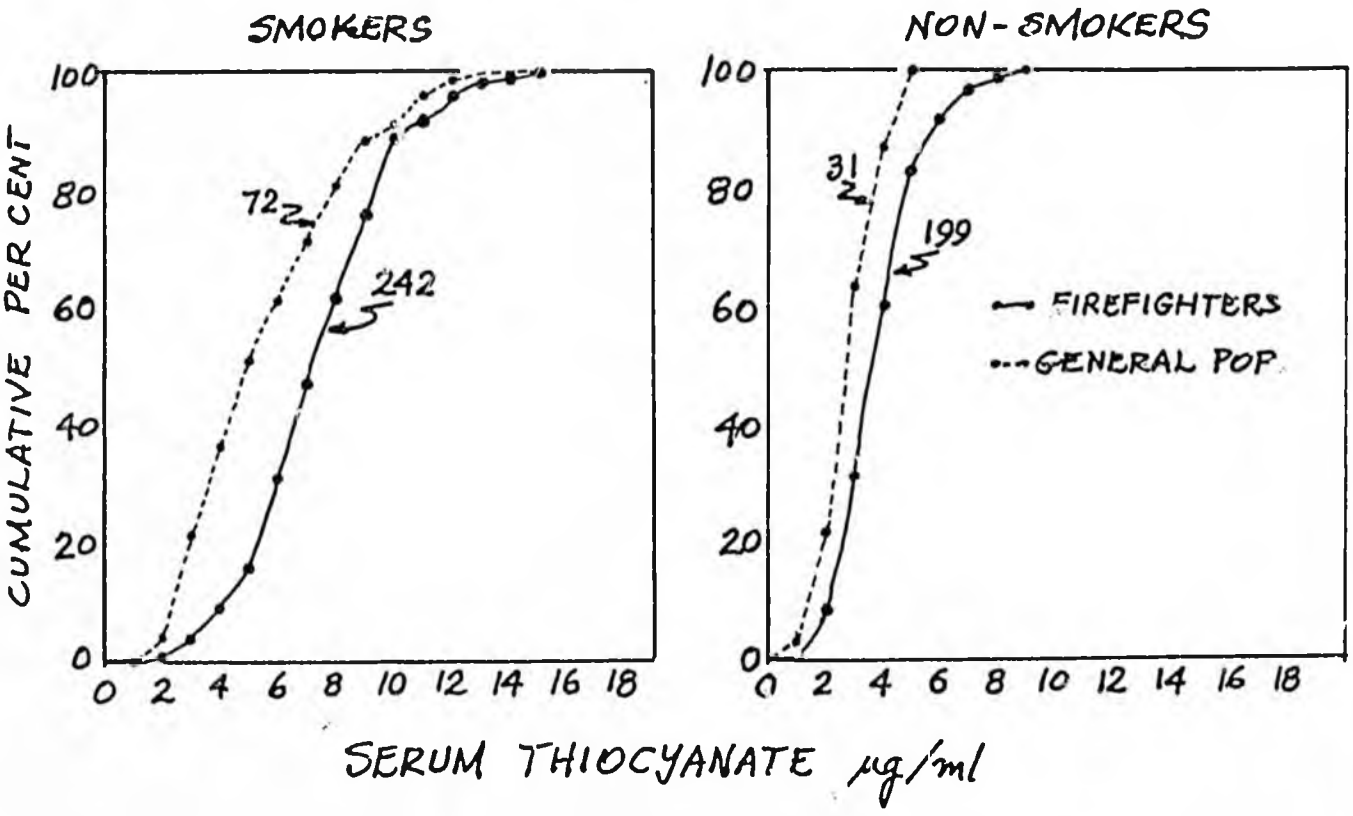


FIGURE 2—Cumulative Frequencies Serum SCN—Firefighters and Controls

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)

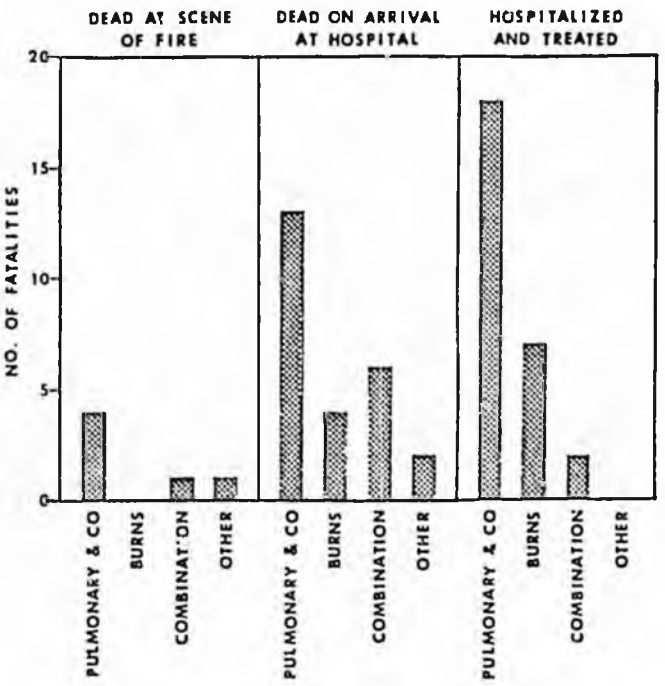
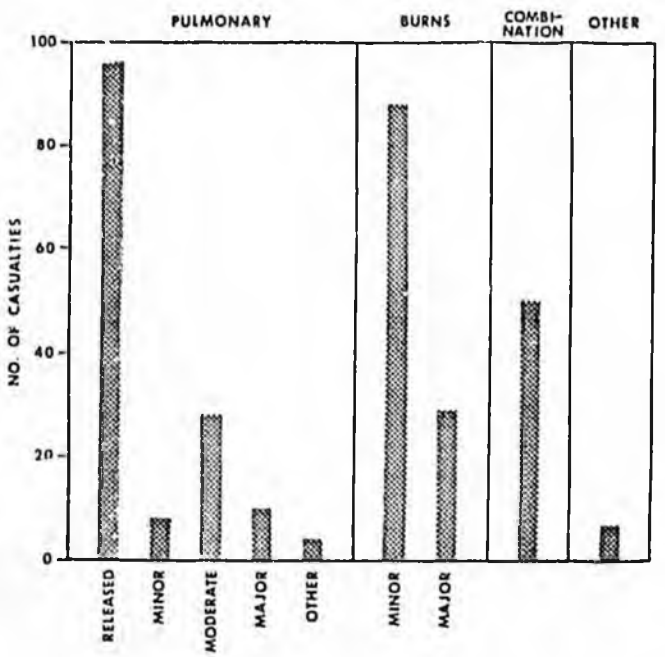


FIGURE 5—Casualties (Preliminary Data)



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.

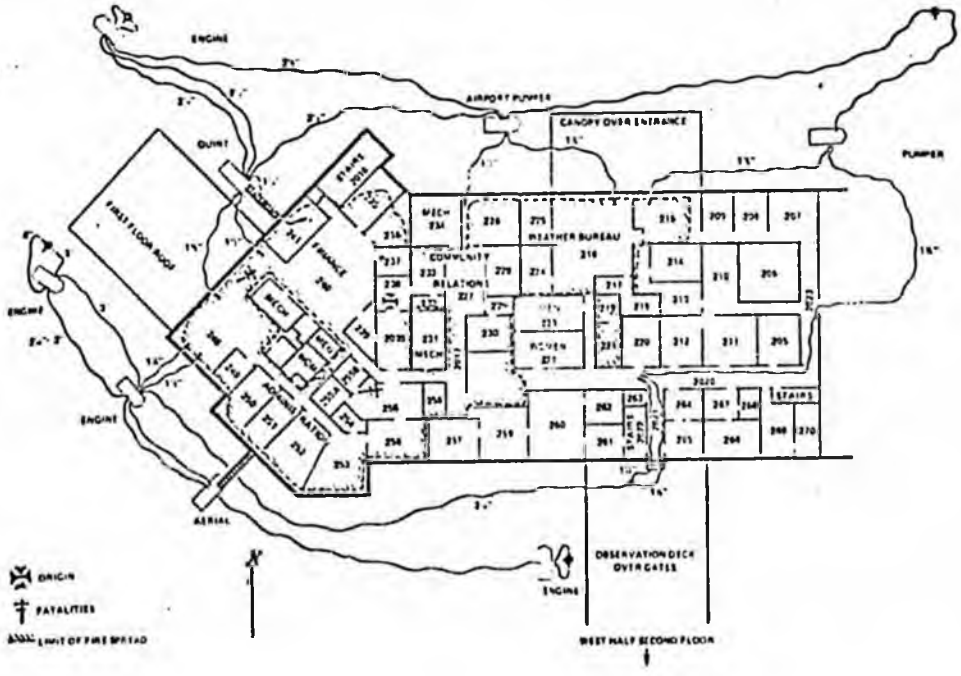
Before proceeding with this report, it should be noted that it is not my intent nor is it the intent of any of the other individuals associated with the IAFF Mortality Study to embarrass any specific fire fighter or fire department. We see no utility in pointing a finger on any one person

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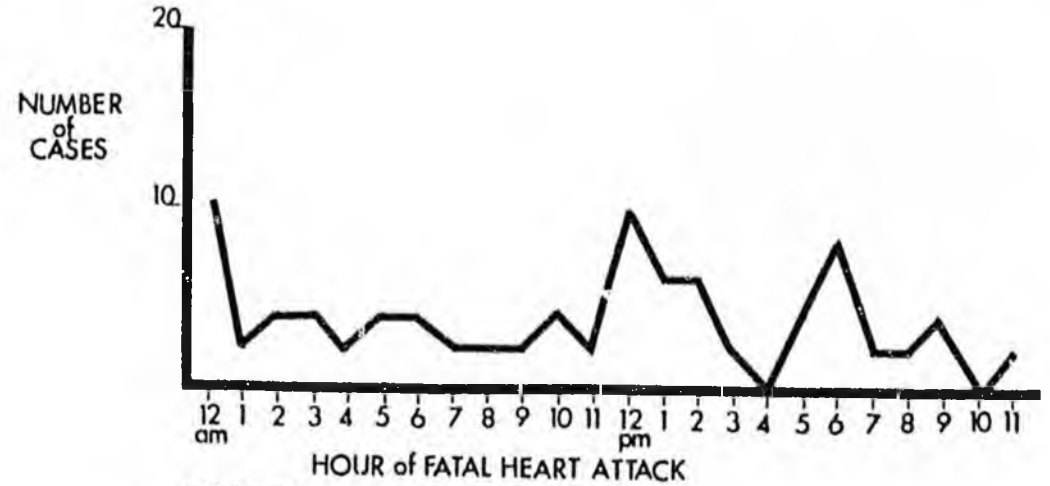
7 .Kobt. Dyer, M. D. & Victor Esch, M. D., "Polyvinyl Chloride Toxicity in Fires" JAMA, Jan. 26, 1976, page 390.

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.

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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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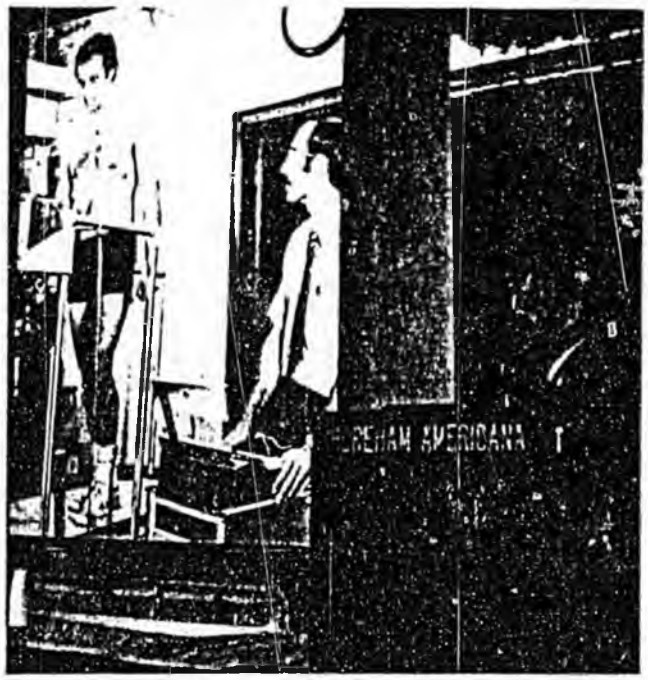
At the past three Redmond Symposia much attention was focused on the heart disease problem of fire fighters. The published proceedings from these meetings all pointed out the need to gain more information about the actual extent of the heart disease problem and how it might possibly be reduced. Since 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.

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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.

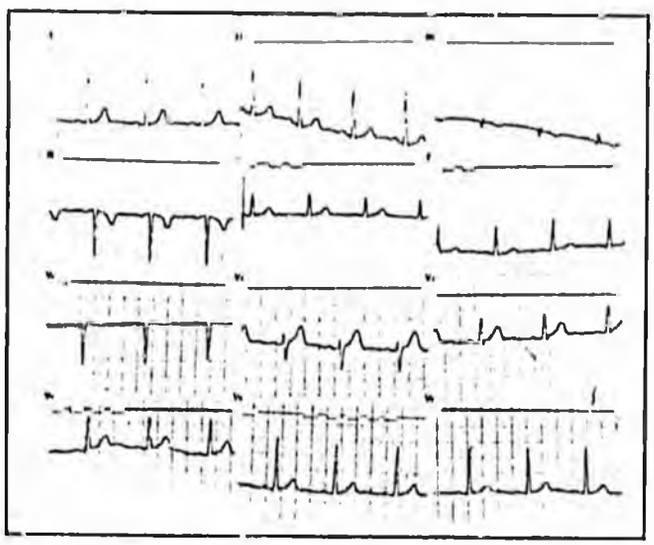
The on-the-job work portion of our study involved monitoring three areas: 1) Holter (portable continuous) electrocardiographic tape recordings were made over a work shift and played back for analysis; 2) ambient carbon monoxide was measured with a portable monitoring device worn on the belt and expired 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%)

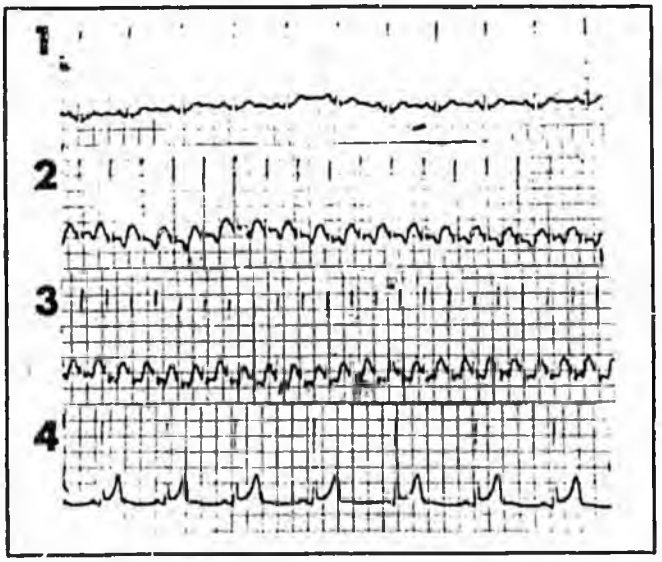
Irregular heart beats characterized as premature ventricular beats were seen in 8 of the tracings or 29%. Of significance, however, are the electrocardiographic changes described as "ischemic" in 6 of the tracings or 21% which cannot be considered normal. In their laboratory, Barnard and his colleagues 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



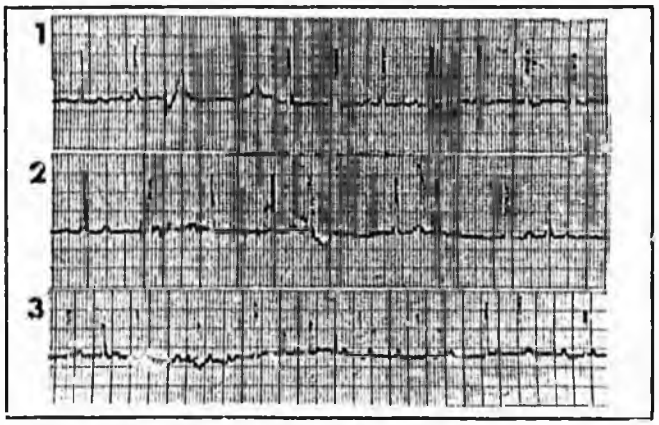
strips taken from his tape recorded electro-

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]

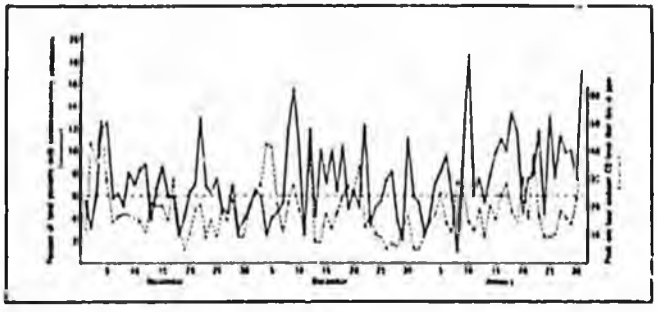
To assess work stress, urinary measurements of adrenalin-like compounds (catecholamines) were performed. Disappointingly only 4 of 31 (13%) were elevated. That these elevations occurred was important, of course, but factors which may have resulted in fewer elevations than expected were: 1) since this 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

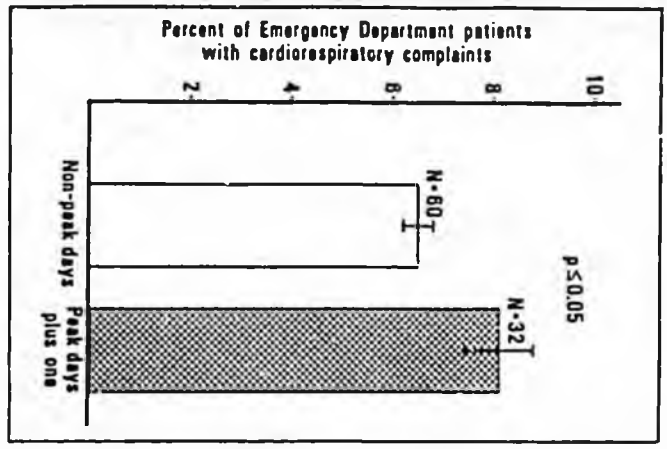
uncommon in Denver and sometimes exceed 50 ppm. Since Denver has very little heavy industry and natural gas is burned for heating rather than fuel oil or coal, sulfur dioxide and other sulfur compounds provide no major pollution.

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

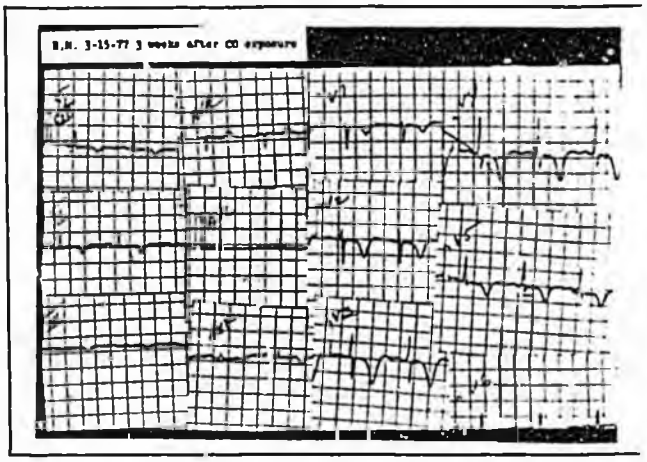
	Inversion season	Early spring
	Nov-Dec-Jan	Feb-March
Mean	18.71	12.60
SEM	1.01	0.69

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



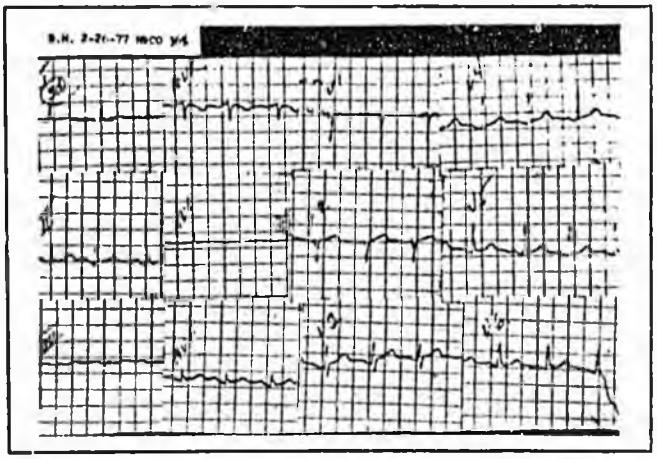
fuse T wave changes compatible with pericar ditis. An echocardiogram confirmed this show ing a small pericardial effusion (fluid formc in the sac around the heart, as an inflammato reaction). Both of these cases, plus others in th medical literature are warnings that signi cant blood levels of carbon 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.