THE UNIVERSITY OF OKLAHOMA

GRADUATE COLLEGE

OCCUPATIONAL HEALTH OF FIREFIGHTERS: FULMONARY FUNCTION STUDY

A DISSERTATION

SUBMITTED TO THE GRADUATE FACULTY

in partial fulfillment of the requirements for the

degree of

DOCTOR OF PUBLIC HEALTH

BY

ATIWAY E. AUSBIE

Oklahoma City, Oklahoma

1973

OCCUPATIONAL HEALTH OF FIREFIGHTERS:

PULMONARY FUNCTION STUDY

APPROVED BY

Raymond A. Mill

Charles H Lawsuna

Michael L. Adess

DISSERTATION COMMITTEE

ACKNOWLEDGEMENTS

It is of immense importance that an advisor set a fine example by judicious guidance, valued criticism and continous encouragement for the successful completion of a graduate career for his students. To Dr. R. A. Mill, I express my deep sense of gratitude and indebtedness for all of this. His guidance will have a lasting and positive effect upon me throughout my professional career.

The author expresses his appreciation to his graduate committee Dr. R. A. Mill, Chairman, Dr. C. H. Lawrence, Dr. R. L. Coleman, Dr. E. D. King, Dr. M. A. Adess, and Dr. C. A. Lynn for their guidance and help throughout the course of my graduate studies and in preparation of this document.

Recognition is extended to the Oklahoma City Firefighters' Association, local Military units and faculty, students, and employees of the University of Oklahoma Health Sciences Center, College of Health, Oklahoma City, Oklahoma. These organizations and personnel provided data which was invaluable to the completion of this study.

Individual consideration is extended to Dr. Donald Parker and Judy Duncan for assistance in Statistical analysis, Dr. Carl Nau, Dr. M. H. Ivey, Dr. P. E. Smith, and Dr. W. W. Schottstaedt for the early impetus provided toward the opportunity of fulfilling this goal and for their confidence in me as a person and as a graduate student.

Recognition and appreciation is extended to the U. S. Public Health Service whose support under the Public Health Training Program Grant No. C1214016 made this study possible.

I acknowledge the typing assistance given on various documents by Mrs. Beverlyann V. Austin and Yvonne Kobus, Miss Florene Ausbie for the excellent job she did on the typing of this manuscript for me.

I am greatly indebted to my mother, Mrs. Nancy Ausbie and mother-in-law, Mrs. Alma Miller, for their spiritual strength and continuous prayer, my brother, Hubert Ausbie for encouragement during the study.

The author wishes to express his sincere appreciation to his wife, Mrs. Doris Jean Ausbie, for her patience, encouragement and assistance, and daughters, Vanessa, Deborah, and Sharonda for their many sacrifices in paternal comradeship. Their cherished and appreciated love is a lasting reward which we together shall never forget.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF ILLUSTRATIONS

OCCUPATIONAL HEALTH OF FIREFIGHTERS:

PULMONARY FUNCTION STUDY

CHAPTER 1

INTRODUCTION

An old Arabian proverb stated "he who has health has hope, and he who has hope has everything." It indicates that all of man's endeavors, all of his aspirations, can be achieved only if his mental and physical health are protected. It is this indispensable base on which all else in life rests (1).

At the present time Firefighting represents a high-risk group with respect to occupational exposure by smoke inhalation. The health effects associated with smoke inhalation by certain population groups have been recognized for many years; however, concern as an occupational contaminant has been a new development. Firefighting has now replaced mining as the most dangerous profession in the United States. The rates of on-the job accident and illness for firefighters have risen steeply and the on-the-job death and injury rates have become the highest of any occupational group in the country. The health hazards faced were unknown a few years ago and too little recognized today.

The dangerous and unpredictable nature of the Firefighters' occupation has caused emotional reaction and severe stress. The job experience

places a heavy strain upon them, in which death, suffering and anguish have been part of their daily lives. They have been exposed repetitively to severe stresses in form of alarm reactions, threat to life and demand to preserve life and property (2).

Every year, many Firefighters suffer illness, injury and even death as a result of what has been called "smoke inhalation." It has represented one of fire's major hazards, both to the victims of fire and Firefighters. It is well documented that about 80 per cent of the victims of fires have not been touched by the flame, but have died as a result of exposure to smoke. To date, a good method to prevent smoke inhalation has not been discovered, and as a result, this hazard has remained through history. Research on the health of Firefighters and their daily work conditions has been superficial (2).

The introduction of new chemicals and gases into manufacturing and production has exposed Firefighters to hazards far beyond those to which they have been accustomed. New construction methods have made their job even more dangerous. In the course of their work, Firefighters have been exposed to a variety of toxic fire gases and other adverse health factors. Quite often they are required to enter buildings, confined spaces, and other places where they may be exposed to extremes of heat, smoke, lack of oxygen, and toxic gases created by the conbustion process. Many fire situations add to the health problems by increasing the breathing and heart rates. Firefighters, therefore, have placed severe demands on their cardiovascular, nervous, and respiratory systems (3).

Damage to the lungs themselves may result from exposure to a group of gases and vapors called pulmonary irritants which present an increasing hazard to the Firefighters. Classic examples of these gases are

chlorine, phosgene, nitrogen dioxide and ammonia. The inhalation of one of these gases causes multiple physiologic and pathologic changes, which are essentially the same regardless of the gas inhaled. Upon entering the lung, these gases react chemically with body fluids to produce strong acids or alkalic. A violent inflammatory response takes place with destruction of the lung tissue, followed by extensive formation of scarred and fibrous tissue. The pulmonary lesions associated with smoke and fire have been almost exclusively due to the effects of these chemical pulmonary irritants. This lung damage may be severe enough to produce permanent disability and even death (2, 3).

Firefighting is one of the largest occupational groups in the country and unlike other occupational groups, in which hazards are controlled, Firefighters are paid to face with routine performance of duty, these unnecessary and uncontrollable hazards. Every fire is a gamble with the unknown, a venture into an unique complex of combustible materials and fire dynamics; therefore, risk becomes a substitute for certainty and intuition for firm knowledge.

At the present time, there is no complete study of all facets of the Firefighters' health; consequently, many have been forced to leave their departments or retire early because of occupational diseases. In view of the public's dependence on this group, it is imperative that their health and safety be protected. Thus, Firefighting as an occupational group, are exposed to hazardous conditions in significant numbers and merits additional study.

CHAPTER II

LITERATURE REVIEW

Respiratory Physiology

The pathologic and physiologic alterations in the respiratory tract caused by burns and thermal injury have been described in both man (4-10) and animals (11-14). There may be immediate cessation of ciliary movement which can be followed by peribronchial and alveolar edema. Damage to the alveolus with capillary damage allows erythrocyte extravasation and perivenous advential cuffing by edema fluid. This is associated with a rapid elevation in the blood potassium and histamine (12, 15). These pathologic changes usually lead to physiologic findings of decreased systemic volume, decreased systemic pressure, increased pulmonary arterial pressure, and increased central venous pressure (15). Early blood gas measurements generally show a decreased P_{CO_2} and slight decrease in P_{O_2}, O_2 saturation and O_2 content with an elevated pH (16). Later, the P_{CO_2} is raised, while the P_{O_2}, O_2 saturation and content fall as does the pH until anoxemia leads to death (15, 17).

There has been widespread recognition that dangerous pulmonary lesions occur from thermal trauma (12, 15, 18, 19). Edema of the bronchi, bronchioles, and possibly even the alveolar wall appear to have been the main pathologic changes. Sevitt and Gallagher (20) found that in surface burns, there is an initial edema with injury followed by a secondary

edema which appear to have been mediated by a chemical or neural chemical factory. Mast cells which are normally abundant in the respiratory tract, concentrate in an injured area within an hour, and is believed to have released histamine and other edema forming substances (21).

Respiratory Tract Pathology

At first glance, one would tend to think in terms of a "fire" or respiratory tract "burn" when an individual has extensive body burns. The term "tracheobronchial burn" has been used extensively in the literature. Experimental and clinical studies, however, demonstrated that in survivors of conflagrations, damage to the respiratory tract was caused mostly by the effects of smoke or products of incomplete combustion rather than the heat (6, 22). Due to the very efficient heat exchange in the upper respiratory tract, thermal burns are usually limited to the anterior portion of the nose and mouth (13, 22). Heat sufficient to produce thermal burns of the pharynx and larynx has been found to produce acute obstructive laryngeal edema. Except in already unconscious individuals, even the larynx is usually spared from heat sufficient to cause laryngeal thermal burns. As pointed out by Taylor and Gumbert (10), no alert human being will allow flame to reach his face if he were conscious. Accordingly, Reed and Camp (23) expressed their view relating to damage of the laryngotracheobronchial system in terms of the effects of chemical irritation and destruction of tissue by smoke and chemical products of combustion rather than the effects of actual heat.

This concept has been demonstrated on animals by Moritz et al. (13) and Stone et al. (22). These investigators conducted experiments with anesthetized dogs wherein the respiratory tract was exposed to

overheated air, flame and steam. They found that it was necessary to introduce the heated air, flame or steam directly to the trachea by an insulated translaryngeal cannula to produce any effect upon the tracheobronchial tree. Any of these agents applied directly to the upper respiratory tract caused immediate laryngeal and pharyngeal edema and the animals were killed instantaneously, before any damage could be done to the tracheobronchial tree.

Stone et al. (22) used rats in their investigations, comparing the variables of heat, humidity and smoke, and found it necessary to use an insulated tube to bypass the larynx in order to study the effects of direct heat on the tracheobronchial mucosa. The mortality rate during the 10 days after exposure as the criteria of severity of damage was used and it was found that mortality was directly proportional to the amount of heat exposure. The mortality rate was increased with increased humidity, but more important was the indication that exposure to smoke increased the mortality more than humidity. A heavy concentration of smoke in the inhaled air was almost as important regarding mortality as was the actual temperature to which the animal was exposed.

Pathologic findings by Stone et al. (22) demonstrated extensive destruction of bronchial mucosa, dilation of large and small bronchi, hemorrhagic edema of the peribronchial connective tissue, generalized hyperemia and hemorragic edema of the peripheral bronchi and alterations in the alveolar capillary membrane in experimental animals. Autopsy findings in humans dying of pulmonary burns confirmed their finds of animals exposed to heat and smoke inhalation (7, 9, 10, 22, 24, 25). These findings were described as intense tracheitis, bronchitis and necrosis of

the tracheobronchial epithelium with diffuse hemorrhagic, and focal membranous reaction in the tracheobronchial mucosa. Pulmonary changes were manifested by one or more of the following: (a) pulmonary edema, (b) atelectasis, (c) intra-alveolar hemorrhage, and (d) disruption of the alveolar capillary membrane. The amount of damage was dependent upon the severity and length of exposure.

Unfortunately, in the autopsy reports of the literature, very little has been reported regarding the histopathologic changes in the larynx of the individuals succumbing to burns. In the few cases found, the laryngeal histopathologic changes were described as edema and necrosis of the surface epithelium, overlying an intact basement membrane (4, 7, 24).

Direct Injury

As shown in Figure 1, many etiologic factors are interrelated. Direct heat injury to the lung is not a major contributing factor. Moritz et al. (13) demonstrated that a direct burn of the respiratory tract below the larnyx was extremely difficult to produce because of the very effective cooling of air by the upper airway. A blast of hot air caused reflex closure of the vocal cords reducing even further the chance of direct heat injury to the lower respiratory tract. Live steam with a heat carrying capacity of 4,000 times that of hot air can produce direct heat injury of the lower respiratory tract which was unusual and did not occur in the studies of Achauer et al. (26). Only one patient of 697 burn cases at Brooke Hospital demonstrated evidence of direct heat necrosis and this extended only a few centimeters below the glottis (27). However, this was not considered as an indication that the lungs do not sustain direct

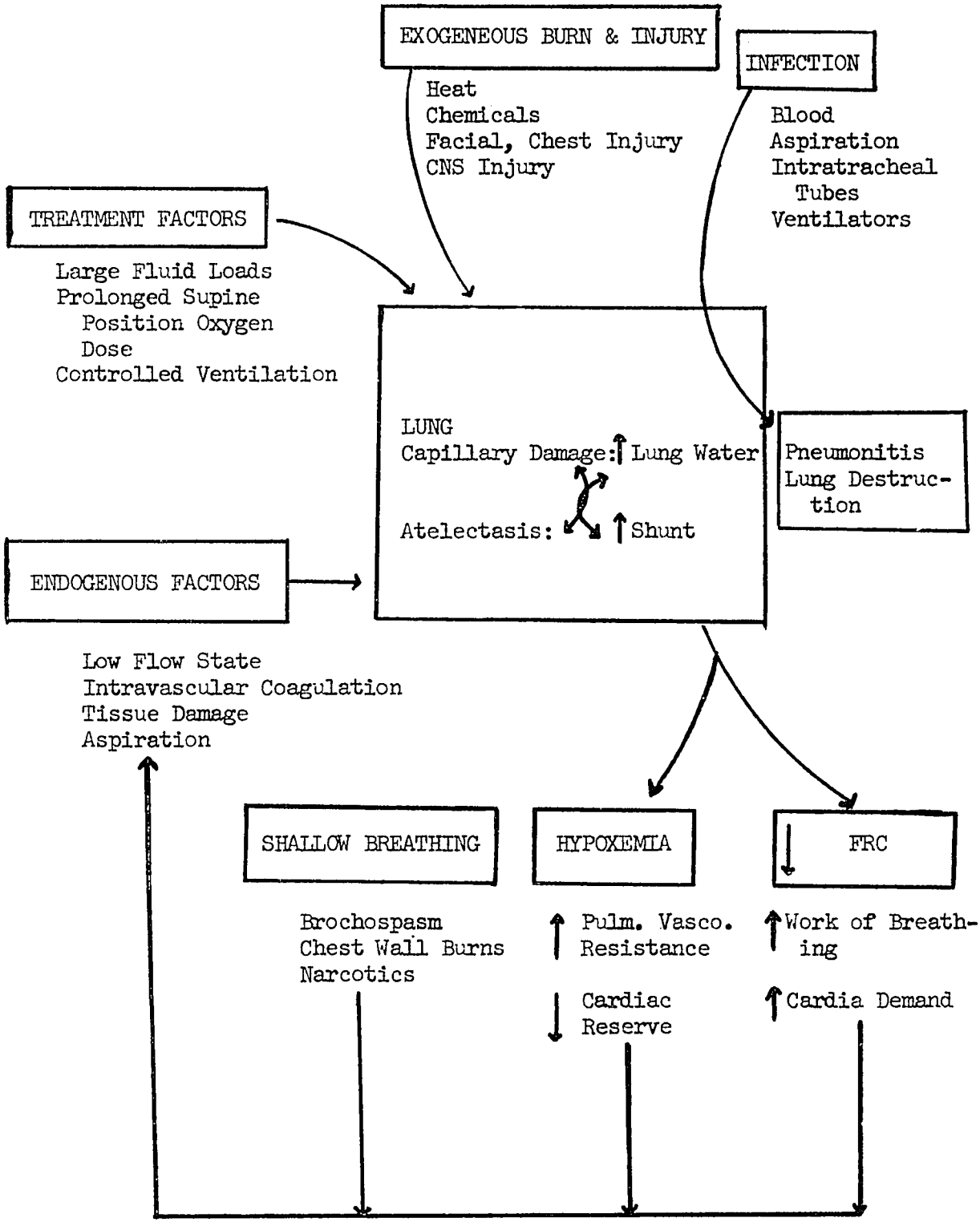

Figure 1 - Pathogenesis of pulmonary insufficiency in burned persons.

injury. The combustion products of various common substances are very toxic to airways and alveoli (26), initially, mucosal cilia are paralyzed according to Beal et al. (28) and cease to clear the airway of debris. Upper airway obstruction secondary to inflammed mucosa or plugs of soot and mucus may occur. Bronchospasm may be triggered by irritating chemicals and a loss of surfactant has been reported as a consequence of thermal injury (29).

Pulmonary Infiltration with Eosinphilia Loffler's Syndrone

Recent reviews (30, 31, 32) emphasized the numerous and varied agents which may be responsible for the clinical syndrome of pulmonary infiltration with peripheral blood eosinophilia. It became increasingly apparent that, in spite of the multiple causes, the basic pathogenesis probably represented a hypersensitivity reaction to various antigens to which the pulmonary tissues may have been exposed.

It is well known that the inhalation of noxious fumes may produce an immediate pulmonary inflammatory reaction by direct irritant effect of the gases. Tschamy (33) reported a case of transitory pulmonary infiltration and eosinophilia (Loffler's syndrome) following smoke inhalation. Among the more dangerous primary irritants present in smoke fumes are aldehydes and acid anhydrites (13). There are two other noxious gases, which are particularly important since they both occur under circumstances of combustion and may produce a severe and delayed pulmonary injury. Nitrogen dioxide (nitrous fumes) may result from combination of many organic substances containing the nitrous radical. Phosgene gas may be formed as a thermal decomposition product of carbon tetrachloride and other chemicals used in fighting fires. Both of these gases produce an acute but usually

delayed bronchitis and bronchiolitis. The picture may progress to bronchiolitis fibrosis olbiterans (34, 35). Exposure to nitrogen dioxide fumes from silage has recently been reported to produce pulmonary injury in the farm population and the symptoms are reported to be similar to that above. These cases which were autopsied, showed findings of bronchiolitis obliterans.

Respiratory tract damage as a cause of death from burns

Since in many instances, more than one cause is involved, selecting a single cause of death for each fatally burned case has been neither easy nor always possible. Pulmonary damage further compromises the oxygen exchange of an individual succumbing from shock. Wound sepsis adds the final insult to a burned victim already overburned with myocardial insufficiency; nevertheless, in the majority of instances a principal cause of death can be identified. The selection is graded as to its probable reliability. Occasionally, responsibility must be divided equally between two or three causes (5).

Many explanations have been offered, including electrolyte imbalance, shock, infection, and renal, hepatic or adrenal insufficiency. Undoubtedly, these may play a definite role in the death of some individuals. The query principally concerns the individual who has survived in the hospital long enough for correction or evaluation of these factors (10).

Recently, (5, 9, 16, 24, 36, 37, 38, 39), the respiratory system has been cited as the chief cause of death. As many as 45 per cent of all those fatally burned, are said to have died as the result of respiratory tract injury. Undoubtedly, thermal injury to the lung has been a deciding

factor in some instances but it has been questioned whether this was a principal cause of death in most burned individuals.

According to Phillips et al.(7), difficulties resulting from the inhalation of noxious products of incomplete combustion were attributed to the respiratory tract damage. The outstanding autopsy findings were denuding of the respiratory mucosa, pulmonary congestion, edema of the tracheobronchial tree, plugging of the smaller bronchi with epithelial debris and edema fluid, and atelectasis distal to the lungs. Bacterial invasion of the damaged tissues began at once.

The victim of respiratory tract damage often has had a deceptive interval of easier breathing between his initial difficulties and subsequent respiratory distress. Obstruction of the larynx has occurred in burned individuals, but in the majority of cases obstruction took place only in the smaller bronchi. In some instances, bronchi and alveoli have been filled with debris and edema fluid and the vital capacity and other ventilatory capacities are seriously reduced by respiratory tract damage (7).

Recent reports identified respiratory damage as the primary cause of death in fatal burns and indicated that pathologic changes found in such lungs were characteristics. Some have attempted to show that these changes were by no means specific and were found to a similar degree in many other diseases which were terminal. It seems probably that fire per se cannot account for many of the abnormalities found in the lung and that death was the result of various unknown metabolic disturbances; therefore, these diseases should also be included in any analysis of terminal pulmonary lesions. Except for suffocation, early shock and electrolyte imbalance, the exact cause of death from thermal burns remains a mystery (10).

Pulmonary complications of burns

The association between thermal burns and respiratory insufficiency has long been realized. Long (40) in 1840, described hepatization and engorgement of the lung with pleural effusion in a number of fatal burn injuries. In more recent times various authors have described the increasing danger of pulmonary complications in the mortality statistics of burned individuals. Phillips and Cope (5) stated that since 1939, pulmonary complications accounted for 42 per cent of all burn fatalities. Socher and Mallory (9) reported 41 autopsy cases in which 28 persons died from pulmonary lesions. Harrison (41) reported the bronchopneumonia was the leading cause of death in their unit. Silverstein and Dressler (42) reported that between 1956 and 1964, six patients died of pulmonary complications and twelve from burn would sepsis at Boston City Hospital. From 1965 to 1968, there were 20 deaths from pulmonary complications and only 4 deaths from wound sepsis. Foley et al. (43) reported that the major cause of death at Brooke Army Hospital from 1960 to 1963 was burn wound sepsis and after the use of Mafenide Acetate in 1964, the major fatal complications shifted to the respiratory system. In later articles from the same institution however, Pruitt et al. (27) stated that no actual increase in pulmonary complications in the entire population at risk had occurred.

There were several different mechanisms involved in the problem of pulmonary burn complications. These considerations were best explained in regard to time of onset of pulmonary findings from the time of burn injury. The acute respiratory distress seen within minutes to hours from the time of thermal injury were due to smoke inhalation, carbon monoxide poisoning or problems secondary to airway obstruction (26). Following the

Cocoanut Grove Night Club fire in Boston, 1942, there were 114 casualties taken to Massachusetts General and 75 were either dead on arrival or dead within minutes (37). This group of patients with acute onset of symptom, represented an important percentage of people who died after thermal injury and were considered a small number treated in the burn centers. Airway obstruction from oral, facial edema or bronchospasm and alveolar damage from inhalation of toxic vapors or particles comprised most of the symptoms found in that group of patients.

Another group of patients were asymptomatic for 24 to 48 hours. They developed tachypnea, cyanosis, and hypoxemia 1 to 5 days after being burned. Hypocarbia and respiratory alkalosis with decreased compliance and increased airway resistance followed. Patchy bilateral fluffy infiltrates were seen on x-ray, also. The course was usually progressive and required mechanical ventilation and supplemental oxygen. Eventually, respiratory acidosis, high pulmonary vascular resistance, myocardial depression and bronchopneumonia, usually, proved fatal. A different group of patients were those that developed late complications such as pneumonia and pulmonary emboli (26).

Chemical pulmonary edema

Chemical pulmonary edema has been one of the major problems in dealing with burned individuals. There is evidence of increased pulmonary capillary permeability from direct chemical injury and pulmonary endothelium from a variety of sources. Initially, irritating chemical products of combustion was thought to be the major insult (44); however, convincing Biochemical evidence has recently been presented for the often postulated burn toxin. Peptide has been suggested as playing an important part in lung and cardiac damage (45, 46). Another factor is the low flow state

that has occurred resulting in inadequate perfusion of peripheral tissue and lactic acidosis. Prolonged poor perfusion result in cell damage and clotting in capillaries. When adequate flow is reestablished, those products are flushed back into the venous circulation. The lung acts as a sieve for this material and may sustain damage from direct effects on the pulmonary capillary endothelium from activation of vasoaction (47). Prolonged exposure to high oxygen concentration (48) and the microemboloc effects of blood transfusion has been suggested as contributing factors (49).

Lung lesions

Despite a large volume of literature on burn pathology, there has been little documentation of pulmonary lesions in fatally burned persons. It was emphasized by Socher and Mallory (9) that patients with mortal skin burn, developed characteristic upper and lower respiratory tract lesions. Each type of lung lesion occurred most frequently within a given period of time after injury. They also associated these lesions as being the major contributory cause of death in a large percentage of burned individuals. The mortality of burned patients with the characteristic lung lesions and renal tubular necrosis was found highest during the first week of survival.

The primary lesions in these burned patients were pseudomembranous tracheobronchitis, necrotizing bronchiolitis, intra-alveolar hemorrhage, and fibrin thrombus information. Of these upper and lower tract lesions, bronchiolitis assumed a role of relatively greatest importance due to its frequent occurrence, also there was possibly casual relationship to bronchopenumonia. The most constant association of

bronchiolitis in patients with bronchopneumonia, suggests an increased susceptibility to the damaged or occluded bronchioles to superimposed bacterial infection. Lending support to this possibility was the frequency of bronchopneumonia. This reached a maximum subsequent to the maximum frequency of bronchiolitis (9).

The pathogenesis of the primary pulmonary lesions revolve around the effects of fumes, flame, particulate matter, and hot air which are inhaled at the site of conflagration. Socher and Mallory (9) emphasized that no single toxic fume was responsible for the lung damage. Each individual was burned in a separate fire and each fire varied in chemical and physical content of smoke, depending on the nature of the matter being oxidized. Moritz et al.(13) and Mallory and Brickley (24) found that flames caused severe lesions in the respiratory tract, but none in the lower tract. Steam caused lesions in both loci. Moritz et al.(13) reasoned this to be due to higher latent heat of steam compared with that of dry air. In the patients studied by Socher and Mallory (9) twice the frequency of lower lung lesions occurred in those having evidence of flame inhalation.

Effects of Inhaled Heat on Air Passages and Lungs

Victims of conflagrations frequently sustain pulmonary injuries that are of equal or greater importance to survival than are the burns received on the surface of the body. In some instances, the changes are confined to the upper air passages with little or no damage to the lungs. The larynx and trachea have shown little or no evidence of injury and profound pulmonary damage sustained. In still other cases, the entire tract may be affected (13). In some conflagrations, as for example the Cleveland

Clinic disaster in 1929, none of the victims were exposed to excessive heat; however, most of the deaths were believed due to the breathing of chemical irritants contained in the smoke that was given off by the burning of Nitrocellulose x-ray films. In many conflagrations, the victims have been exposed to heat as well as to the smoke. If respiratory injuries are sustained, the question of whether they were due to thermal or chemical trauma is raised (24).

It was emphasized that no two nonexperimental fires are exactly alike, so far as their propensity for the elaboration of chemically irritating combustion products are concerned. Fires represent a series of chemical reactions each of which may contribute a different group of potentially injurious gaseous, liquids or solid irritants to the atmosphere. The nature and amount of these potentially injurious substances in smoke are determined in part by the composition of the material being burned, amount of oxygen available at different times during the conflagration, the temperatures that are attained (13).

Thermal injury of the respiratory tract has not received extensive recognition. It is uncommonly associated clinically with even extensive cutaneous burns which is rather surprising considering the exposure of the respiratory passageways to the intense heat of the atmospheric gases associated with flame or explosion burns. The clinical manifestation of respiratory tract injury has been found to follow noxious gas or smoke inhalation. Although it is unusual for burned individuals to be exposed to heat alone, thermal injury to the respiratory tract could occur and be associated with a high mortality. Fineberg et al. (50) were able to produce respiratory passageway and parenchymal pulmonary burns in dogs to determine the response of injury tissue. The cause of death in all animals

receiving respiratory tract burns was a progressive obstruction of the air passages with edema fluid.

Garzon et al.(51) showed that pulmonary resistance was increased, indicating severe narrowing and obstruction of the tracheobronchial tree from spasm of smaller bronchi, edema of walls, blocking with carbon particles, epithelium, fibrin, leucocytes and mucus.

Combustion Products of Common Substances

It has long been recognized that when fire victims perish, the external injuries are often insufficient to explain the cause of death. They are described as victims of suffocation or asphyxiation or simply as smoke inhalation or overcome by fumes (1). All individuals overcome by smoke may not have been victims of asphyxia, but may have been suffering from the effect of toxic gases, the ingestion of particulate smoke, and the effects of heat and hyperventilation (3).

Nature of smoke

Knowledge of the combustion products from common substances under different burning conditions is essential in understanding the health effects produced. In all fires, smoke, which is composed of the volitilized products of combustion of organic compounds (such as coal, wool or petroleum) and fine particles of carbon, is produced along with varying amounts of dusts, fibers, mists, vapors and gases. The amount of smoke can vary greatly from fire to fire and the amount visible represents no index of its health hazard. Smoke particles in the air stream may cool to the extent that water vapors, organic acids and aldehydes produced by fire will condense on them. Such particles, if inhaled, carry the irritants deep into the respiratory tract (1). In addition to the visible

smoke, a number of fire gases are produced by the combustion process. The composition of these gases depends on the nature of the combustion, rate of heat, oxygen content at or near the burning surface, temperature of the evolved gases, and the composition of the material being burned. Carbon dioxide and carbon monoxide are always present in varying proportions and account for the bulk of gases produced (52). A summary of the other principle toxic combustion products of common substances is presented in Table 1.

In addition to the gases produced by combustion, the smoke may contain other toxic chemicals liberated as a result of industrial fires or explosions and volatilized by the heat of the fire. In some cases, toxic chemicals may have been employed as extinguishing agents, further adding to the risks (3).

Effects of smoke exposure

Individuals exposed to the complex mixture of smoke may produce a variety of physiological disturbances which may cause them to be overcome by smoke. Hypoxia is only one of these disturbances; others may result from the ingestion and inhalation of particulate smoke and exposure to toxic, but not asphyxiating, gases and chemicals. Levin (53) suggested that this condition be called "smoke poisoning" to clearly distinguish it from "smoke inhalation". In addition to these direct effects of smoke exposure, individuals may be in difficulty due to the hyperventilation syndrome or the effects of heat (1).

Pulmonary irritants

Individuals overcome by smoke may have been exposed to two types of pulmonary irritants: the organic aldehydes, i.e., acrylic aldehyde

TABLE I

PRINCIPAL TOXIC COMBUSTION PRODUCTS OF COMMON SUBSTANCES

Substances	Pulmonary Irritants	Other Gas and Vapors
Wood, cotton Newspaper	Acetalydehyde, formaldehyde	Acetic & methane acids Formic acid
Petroleum products	Acrolein	Similar to wood
Wool, silk	Ammonia	Hydrogen sulphide Hydrogen cyanide
Nitrocellulose film Cellulose acetate film	Oxides of nitrogen None	Similar to wood Similar to wood
Polyester resins	Hydrogen chloride	
Polyurethane foam	Isocyanates	Hydrogen cyanide
Polyvinylchloride	Hydrogen chloride Phosgene Chloride	
Polyflurocarbons (e.g. Teflon)	Octafluroisobutylene	
Phenolic resins	Ammonia, formaldehyde	Hydrogen cyanide
Melamine resins	Ammonia	Hydrogen cyanide
Rubber latex foam Neoprene foam	Unknown, but exposed rats form pulmonary edema	

Carbon monoxide and carbon dioxide are produced in all cases

(acrolein) and acetaldehyde (Table 1) and the acid anhydrides. While the former have been found capable of producing severe pulmonary lesions in the laboratory (54), their toxicity under clinical conditions appeared to have been much less than that of the latter group. Aldehydes tend to condense out of the smoke before they are inhaled. The acid anhydrides normally exist in the gaseous state which allows them to enter the lungs more readily.

The pulmonary lesions due to chemical irritant seem most likely produced by chloride,phosgene and other similar compounds. These compounds may be produced from combustion, released from storage tanks, e.g., chloride, or refrigerating units, e.g., sulphur dioxide or ammonia (1). Exposure to pulmonary irritants has become more common due to the increasing use of plastics which produced these toxic gases when burned.

Phosgene is an example of a pulmonary irritants which gives rise to hydrochloric acid on contact with moisture; but, the hydrolysis rate is slow. The gas has a slight effect on the upper respiratory tract, but in the lung alveoli, it produces concentrated hydrochloric acid causing pneumonitis and pulmonary edema. This reaction may develop hours after the initial exposure. The insidious nature of phosgene renders it far more dangerous than an equivalent amount of chlorine or ammonia.

Other gases and vapors

Exposure to a number of chemicals which have not been considered pulmonary irritants, may occur as a consequence of fires. The exposure may result in the individual being overcome by smoke and/or poisoning by hydrogen cyanide, hydrogen sulphide, or hydrogen chloride. These gases may be formed as products of combustion (Table 1) from industrial fires

or may be present because of leaks in storage or processing equipment and released in large quantities. Heat can increase the vaporization of these substances and very high concentrations may be inhaled by unprotected or unwary individuals. Most of the common industrial solvents, such as benzene, toluene, carbon tetrachloride, have a narcotic action when inhaled in high concentrations (55). Varying degrees of central nervous system depression, including unconsciousness, may be produced. If fires involved a plant manufacturing known toxic substances such as insecticides, then individuals overcome by smoke may be poisoned by these agents.

Chemical poisoning may result from non-industrial fires if chemical extinguishing agents, such as carbon tetrachloride or methyl bromide, have been employed. Fortunately, these agents have been replaced by less hazardous agents, such as carbon dioxide, foam and dry powder (3).

Hydrogen sulphide. This gas may form from incomplete combustion of some substances such as wool products, sewer gas and mine gases. At concentrations above 0.02 per cent (200 ppm), the olfactory perception in man is paralyzed and increasingly higher concentrations are not distinguishable by smell. Concentrations of 0.1 per cent (1,000 ppm) are rapidly fatal and paralyzes the respiratory center in the brain (1).

Hydrogen cyanide. This is a very toxic gas which is given off in small amounts by burning silk and some plastics. It may be evolved in fighting fires in industries where cyanides are used and stored. Concentrations approximating 0.1 per cent (1,000 ppm) are quickly fatal (1).

Hydrogen chloride. This gas is extremely hygroscopic and combines with atmospheric water vapor to form hydrochloric acid. The entire respiratory tract may become irritated, therefore provoking reflex spasm to protect the lung alveoli (1).

Material Health Hazards

The common irritants found in fire gases include: acetic acid, acrolein, acetaldehyde, formic acid, formaldehyde, ammonia, furfural, tar and sulphur dioxide. These arise in various combinations in fires involving wood, paper, coal, petroleum products, textiles, animal fats and oils. Wood, silk, rubber, photographic film, celluloid and plastics, also, produce large quantities of smoke and irritant gases. Materials which burn in an excess of air, may produce fire gases different from the same materials burned in a lack of oxygen. Generally speaking, gases and smoke produced early in a fire are relatively cool, containing large amounts of toxic and irritant materials (1).

Tidey (1) and Olsen et al. (52) studied gases produced from thermal decomposition of common combustible materials and found that gases from all types of fires investigated contained toxic constituents in sufficient amounts to make breathing the gases dangerous or even fatal in a relatively short period of time. Gases from different materials differed greatly in toxicity with substances containing nitrogen or sulfur, or both, producing the most toxic gases. These investigators studied the gases produced from combustion of materials and described them as follows:

a) Of the textiles used for clothing, draperies, etc., cotton and rayon produced the least toxic gases. Silk and especially woolen materials gave off deadly gases, such as hydrogen sulfide, hydrocyanic acid, sulfur dioxide, ammonia, and carbon monoxide.

b) High toxic concentrations of carbon dioxide and carbon monoxide were produced when cellulose was decomposed by heating in the absence of air. The high percentage of acetic acid accounted for the highly

irritating and acrid effect, when persons breathe the gases from burning cellulose materials.

c) Both carbon dioxide and carbon monoxide concentrations were very much lower in gasoline fires than when wood was the burning material. The oxygen concentration was also much higher, because gasoline burned only as a gas and it produced a flame which lost heat rapidly.

d) When rubber insulation was decomposed, considerable amounts of saturated and unsaturated hydrocarbon gases, as well as free hydrogen, were produced. Hydrogen sulfide, sulfur dioxide, carbon monoxide were also found in certain amounts.

e) The decomposition of woolen and silk materials gave a much greater number and variety of gases. Not only was carbon monoxide produced, carbon dioxide, hydrogen sulfide, hydrocyanic acid, ammonia, nitrogen, and hydrogen were evolved in large quantities. Free oxygen was evolved when the material was decomposed by heat in the absence of air. Water and organic acids in considerable amounts were produced and carbonaceous residue remained after the gas has been expelled.

f) Butter and animal fats yield significant amounts of carbon monoxide, carbon dioxide and acrolein.

The most surprising result of these experiments was the presence of such highly toxic gases as hydrogen sulfide and hydrocyanic acid in high concentrations. Since ammonia is toxic, even in low concentrations, the large percentage of ammonia made these gases dangerous to breathe. The combination of these gases and the large content of carbon dioxide and monoxide made the atmosphere dangerous to breathe for even a very short time (52).

Photographic film

When nitrocellulose is burned in a deficiency of oxygen, copious amounts of nitrogen oxides are produced. However, nitrocellulose film is no longer used because of its flammability. Cellulose acetate films yield combustion products similar to wood.

Plastics

The fire gases produced with plastics depend primarily on the type of resin. In a review published by the Underwriter's Laboratories, it was noted that fire gases produced from burning plastics contain significant amounts of carbon monoxide, carbon dioxide and other irritants. Some of these have found special uses within buildings and are worthy of special comment.

a) Polyester resin reinforced with fibrous glass is used as a panel for partitions and glazing and when burned, yields dense black smoke. Fire-retardant polyester resins have been produced; however, these may yield hydrochloric acid in addition to carbon monoxide and carbon dioxide when burned.

b) Acrylic panels have been used in light diffusers. If involved in a fire, they soften and fall out but do not burn or produce smoke.

c) Polyurethane foam has been used in automobile seats, arm rest, etc. and when burning, isocyanates which known to be respiratory irritants and sensitizers, may be produced.

d) Polystyrene foam insulation was found to be no worse than wool, but the self-extinguishing varieties may produce traces of phosgene.

e) Polyvinychloride coverings may yield significant amounts of carbon monoxide, carbon dioxide and hydrogen chloride. Traces of hydrogen cyanide and phosgen are found, also.

f) Polyfluorocarbons (Teflon) when heated above 500°F may give rise to a condition known as polymer fume fever; when heated well above 700°F, pyrolysis occurs with significant amounts of breakdown products such as hydrogen fluoride and other organic fluorides emitted.

g) Polythylene produces mostly carbon dioxide, however, some also be produced.

h) Epoxy resins produce a variety of irritant combustion products when burned.

i) Phenolic resins may give off formaldehyde gas.

j) Melamine resins may give off small amounts of hydrogen cyanide.

These gases irritate the mucous membrane of the eye and respiratory tract, and produce pain and discomfort. They are considered as secondary importance in causing quick deaths. Damage to the trachea, bronchus and lungs may be produced within 3 hours after the exposure and secondary infection with bronchopneumonia may follow. If the exposure is excessive with severe damage to the lungs, permanent loss of pulmonary function may result. These irritant gases and vapors generally, give sufficient warning of their presence. Under some conditions, however, irritant materials may condense on small particles and be inhaled deep into the lungs (1).

MacFarland and Leony (56) studied polyurethane foam and nylon fabrics coated with polyurethane and the results showed that pyrolysis products of both materials can be lethal to rats when inhaled. Lung

sections from all experimental groups were examined histologically and it was concluded that acute asphyxia from occusion to the respiratory tract was the process which produced death in rats exposed to pyrolysis products of polyurethane. With nylon products, death resulted from a slowly developing but ultimately overwhelming pulmonary edema.

Teflon

The polymer of tetrafluoroethylene known as "Teflon" is a thermoplastic resin possessing a variety of highly desirable properties. Its high melting point and outstanding resistance to organic solvents and strong acids and alkalic have promoted its expanding use in laboratory ware, chemical valve packings, gaskets and hydraulic hoses. The formed plastic is physiologically inert, insoluble, nonirritating to the skin and nontoxic when taken by mouth. Consequently, it was mentioned with increasing frequency in the literature as a valuable structural material for prosthetic devices, i.e., cardiac valves and vascular replacements, designed for indefinite contact with tissue and biologic fluids (57).

The many desirable qualities of Teflon have led to its wide use in industry, domestic and medical applications. Although stability is one of its outstanding qualities, it is recognized that at quite high temperatures, toxic decomposition products are evolved. Toxic effects on man have been only rarely reported. Apparently, none of these individuals are exposed to fumes from Teflon decomposed at such high temperatures (58).

The toxicity of heated Teflon has been ascribed to its degradation products, which varies in quantity and type according to the temperature (59). Temperatures from 200 to 300° C generate very minute quantities, measured as a weight loss of 0.0002 per cent per hour, of products. The

exposure sometimes may cause an influenza fever which is referred to as "polymer-fume fever" (60). Reported cases of polymer-fume fever has indicated prompt recovery with no after-effects.

Above the temperatures of 400° C, the quantity of pyrolysis products, rises rapidly to 10 per cent weight loss per hour at 500° C, and 50 per cent at 550° C. The nature of the fumes changes to include more toxic products such as Octafluoroisobutylene. It is extremely toxic and in experimental situations, has produced delayed pulmonary edema resembling that associated with phosgene exposure. Teppers (57) found no clearly documented reports of pulmonary edema in man due to Teflon gases.

Chemical Health Hazards

Sulfur Dioxide

Sulfur dioxide has attracted the attention of toxiciologist for several reasons. It can easily be recognized at low concentrations by its acrid odor and has been considered for a long time to be an effective gaseous disinfectant, particularly, used in the preparation of vats for the fermentation of grapes. It is a distinct contaminant of most fossil fuel (coal and petroleum); therefore, it is almost constantly present in the products of fuel combustion (61, 62). The recommended threshold limit for industrial exposure is 5 ppm by volume (63), a level readily detectable by most individuals.

While concentrations sufficient to cause mucosal irritation may frequently be reached in industrial plants, there has been no clear evidence that such concentrations produce an adverse effect on those chronically exposed. Kehoe (64) and Anderson (65) found little or no impairment of health resulting from chronic exposures which often reached 25

to 30 ppm. Anderson (65) measured vital capacity, but the conclusions were based on interviews, physical examinations, and x-rays of the chest rather than physiologic measurements.

Several investigators have studied the response of human subjects, not occupationally exposed to sulfur dioxide to different levels of the gas for short periods of time. Amdur et al. (66) reported that a more shallow, rapid respiratory pattern resulted from 10 minutes of exposure from 1 to 8 ppm sulfure dioxide; however, was not subsequently confirmed by others (67). Sim and Pattle (67) who used the interrupter's method, of Ainsworth and Eveleigh (68) measured pulmonary flow resistance and concluded that little change occurred at levels of sulfur dioxide below 30 ppm for 10 minutes or 5 ppm for 60 minutes. The investigators, however, did not publish the data on which their conclusion was based; therefore, it was felt that this finding could not be judged adequately.

Frank et al. (62) studied the effects of short term exposure to controlled concentrations of sulfur dioxide on the mechanisms of breathing in healthy subjects. The subjects were chosen because their activities did not customarily expose them to the gas, except for whatever traces may have been present in a large urban community (66). Whether frequent or prolonged exposure to sulfur dioxide might affect the degree of response seen in these experiments was unknown. To reduce the possible importance of too frequent exposures, a delay of at least 1 month between experiments was prescribed for each subject (62).

The degree to which the pulmonary flow resistance increased during administration of sulfur dioxide was related to the concentration of gas. It was not, as suggested, tantamount to an all-or-none phenomenon (62). It was noted, however, that the response could also be modified by time.

Usually, flow resistance continued rising as long as the gas was administered, a period lasting several hours (69); however, in some instances a maximal response was elicited within 10 minutes and this change was small and random thereafter (69, 70).

The fundamental cause of these changes was not known but appeared to be bronchoconstriction. There was evidence that bronchoconstriction may be mediated by both extrinsic nerve reflexes and direct action of the gas on the bronchial small muscle (62). Whether variations in the amount of secretion by the mucosal gland was an additional factor contributing to changes in flow resistance, was not certain. If, as in experimental animals, the level of irritant gas is sufficiently high, there will be ensuing tissue damage in the form of edema and hemorrhage. Extreme changes of this type were suggested as being partially responsible for the pattern of response in those studies in which there was a continuing rise in flow resistance.

Controlled animal exposure revealed that small rodents (hamsters) can withstand intermittent exposure of 650 ppm for 3 hours a day, 5 days a week, for a period of 10 months, without lethal effects (71). Only minimal histological findings were described at sacrifice in these experiments. Concentrations in excess of 3,000 ppm were necessary to kill 50 per cent of animals exposed for 1.5 hours. Rats exposed to 40 ppm daily for 3 months, in an attempt to produce bronchitis sustained the exposure without noticeable effects and without apparent evidence of historical abnormalities (72). In the same study, an exposure to a concentration of 400 ppm, 5 hours a day, 5 days a week, for a period of 6 weeks, was necessary to obtain a substantial increase in bronchial mucus secretion. Rats exposed to the same concentration, for 2 weeks, did not show gross

abnormalities in the entire respiratory system. Only after 4 weeks at 400 ppm were the initiative phenomena evident. After the exposure was discontinued, the animals sacrificed at different intervals of time showed a proportional regression of the hyper-secretion phenomenon.

The literature has been replete with work relating to adaptive reactive to sulfuric mists in the sense of a progressive tolerance in occupational exposure to high concentrations of acid sulfurous gases (65). Pattle and Cullumbine (73) reported a progressively decreasing response in repeated exposure of volunteers to sulfuric acid aerosals. Sulfur trioxide or sulfuric acid appeared to have been more of an irritant than sulfur dioxide at equal dose in man (67) and animal (69). Human volunteers exposed to sulfuric acid at concentrations in excess of 2.9 mg/m^3 for 1 hour, complained of irritation of the conjunctivae and respiratory mucosae. Several subjects showed increased airway resistance as measured by the interrupter's technique (67).

The oxidation of sulfur dioxide to sulfur trioxide is a slow process with complex equilibrium constants in which humidity, sunlight and the presence of catalysts in the atmosphere are all important. It has been proven that mixtures of sulfur dioxide and sulfuric acid are more than additive in their effects on animals and therefore may occur at levels not comparable with those commonly found in air pollution episodes (69).

Applying a suggestion originally proposed by Hemeon (74), Amdur and Corn (75) studied, experimentally, the effects sulfate inhalation had on airway resistance. These experiments indicated that airway resistance can be slightly increased in guinea pigs exposed to zinc and ammonium sulfate at level of 0.25 mg/m^3 and higher. However, the relevance of this

phenomenon in contributing to the death of 4,000 human beings was not readily available.

La Belle et al. (76) formulated a hypothesis suggesting that particle size influenced toxicity by using an aerosol composed of formaldehyde, acrolein and nitric acid to permit deep penetration in the airways. If a gas was absorbed on a certain size aerosol in reversible fashion, the locus of the gas phase deposition was modified. In other words, if the aerosol penetration was deeper than the gas phase, the inhalation of both, simultaneously in the stated conditions would deliver the gas at a deeper site in the lungs. In the case of an irritant gas, this would imply a more extensive surface of attack by the gas and perhaps an increased extent of injury. La Belle et al. (76) also presented evidence that this phenomena occurred. As stated by Goetz (77), the opposite could also occur in the sense of an inhibitory action by the aerosol on the gas toxicity. This condition occurs when the affinity of the gas molecule for the particulate surface exceeds that of tissue fluids.

The potentiation of toxic effect as determined by a deeper penetration of a gas, is not considered a general phenomenon. It specifically depends on the nature of the mechanism of injury characteristic of a given compound. If a gas is absorbed on the nasal surface, in adequate doses, it induces pain, edema and hemorrhage of the turbinates and if it is deposited in the alveolar spaces, it produces some degree of pulmonary edema (61).

Oxides of Nitrogen

Concentrations of less than 50 ppm (0.005 per cent) of oxides of nitrogen gases may be breathed freely for long periods of time (78).

Higher concentrations cause reflex inhibition of breathing with laryngospasm. Intense cyanosis develops rapidly due to the formation of methaemoglobin in the blood and arteries desaturation consequent upon changes in pulmonary gas exchange. Severe hypoxia may therefore occur, for which two mechanisms are responsible: anemic anoxia as a result of reduced oxygen capacity of the blood, and anoxia as a result of the interference with oxygen transfer in the lungs (79).

In Greenbaum's et al. (79) dog experiments, there was a tendency for the ventilatory frequency to increase. The tidal volume was decreased leading to progressive ventilatory failure manifested by rising carbon dioxide pressure. Pulmonary compliance was halved and airway resistance was markedly increased.

Pulmonary edema may occur in the acute phase (79) but with lesser degrees of exposure,it characteristically develops after a latent period of several hours. The exposure may result in death in 1 to 3 days as seemed to have been the case in the individuals involved in recent anaesthetic accidents (80). If not immediately fatal, pulmonary edema may be complicated by chronic chemical pneumonitis. There have been several reports of delayed appearance of inflammatory changes described as bronchiolitis fibrosa obliterans or chronic pulmonary fibrosis (81, 82, 83).

In Prys-Roberts' (84) investigation, the intense irritation of the lower respiratory tract following inhalation of oxides of nitrogen led to the development of a diffuse inflammatory process throughout the lungs.

Nitrous Fumes

Exposure to Nitrous fumes may result in several types of clinical reactions. One of these, acute pulmonary edema (85 - 89), develops after

a latent period, lasting in some cases up to 30 hours and is frequently fatal. It is apparently followed by complete recovering if the victim survives the acute initial episode. Nevertheless, there are records of individuals in whom the symptoms persist. In the 16 cases reported by Lindquist (89), the patient in 1 case, on resuming work months after the accident, found that he became breathless very easily. This complaint had not been noticed before his accident. Similarly, Johnstone (90) stated, in his textbook that permanent disability occurred but was rare. However, no detailed follow-up studies by clinical methods or by pulmonary function test have been reported. There was no clear description of the nature of the residual disability or the frequency of its occurrence.

A second type of reaction of these fumes is bronchiolitis obliterans (89, 91, 92). This condition could only be diagnosed with certainty by histologic methods, although the clinical course may have suggested it. Exposure and acute symptoms were followed by a latent period; apparent improvement sometimes lasted up to one month (88, 89, 91, 92, 93). Thereafter, there was a progressive development of unremitting dyspnea with severe cough, frank cyanosis and typical roentgenographic findings of irregular soft mottling throughout both lungs.

A third clinical finding following exposure of these fumes was that of pneumonia which was apparently chemical rather than bacterial (83, 88). However, no work on the incidence and nature of permanent sequelae following bronchiolitis obliterans or on chemical pneumonia following exposure to nitrous fumes was encountered. The medical literature did not reveal any clear impression as to whether permanent disability occurs after exposure to nitrous fumes.

Several patients who had recovered from an episode of acute

pulmonary edema following exposure to nitrous fumes were studied for periods up to 64 months after the accident. There was a reasonable agreement between subjective complaints and demonstrable disturbances of physiologic test. The usual pattern of physiologic disturbance was a reduction in maximal breathing capacity and an increase in expiratory resistance values. Two patients showed an increased absolute value of residual volume as well as an increase in its percentage value of total lung capacity. Such changes in the test value were commonly associated with the presence of emphysema or bronchospasm. Neither of these conditions was thought to be present in the reported cases. A possible explanation seemed to have been that the patients had some degree of bronchial and bronchiolar narrowing due to the healing by fibrosis of various degrees of bronchiolitis obliterans, following exposure to nitrous fumes (94, 95).

Nitric Acid

Serious injury by concentrated nitric acids has not been infrequent and has accounted for a significant number of the cases of disability and death from chemicals (96). Toxicologically, there are two major forms which may be regarded as identical since they are both extremely corrosive and emit dangerous fumes. Red fuming nitric acid, containing dissolved oxides of nitrogen (nitrous fumes), is toxic and decomposition of the acid on contact with oxidizable material, liberates more oxides of nitrogen. White fuming nitric acid, though containing little dissolved oxides of nitrogen, decomposes on contact with oxidizable material just as red fuming nitric acid and also liberates oxides of nitrogen (97).

The concentrated nitric acids are extremely corrosive fuming

liquids which vary in amounts of dissolved oxides of nitrogen. The[illegible]re evolved as reddish-brown fumes with small amounts of acid vapor. Nitr[illegible] oxides in contact with air are rapidly oxidized to nitrogen dioxide which readily polymerizes to nitrogen tetroxide. The inhaled fumes, at body temperature consit of approximately 30 per cent NO_2 and 70 per cent N_2O_4. Both of these oxides form nitric acid on contact with water (88). Fuming nitric acid constitute a definite hazard, causing injury by liquid contact with body surfaces and by inhaled fumes.

Severe pulmonary injury may result from inhalation of nitrous fumes. Clinical findings in acute poisoning by inhalation of the fume, include those involving the irritant gas type. There are signs of local irritation followed by a latent period of a few hours and a development of pulmonary edema and/or bronchiolitis obliterans may lead to death in 1 or 2 days (97). Variation in the clinical picture seem to have been due to the composition of the fumes. When the content of nitric oxide is high in sources of nitrous fumes, other than nitric acid, i.e. arc welding (89), there may be cerebral symptoms. When the content of nitrogen dioxide was high in nitric acid, there may be an irritant phenomena (97).

Aldehydes

Aldehydes which contaminate the atmosphere are emitted from burning natural gas, fuel oils, diesal and refuse. Salem and Cullumbine (98) investigation determined the relative inhalation toxicities of a series of aldehydes using various species of laboratory animals exposed to aerosol and vapors. All the aldehydes employed in the study were irritant aldehydes produced a bronchial construction with a resulting distention

of alveoli and rupture of the alveolar septa. The convulsions that followed were believed to be due to anoxia and death occurred quickly. The less irritant aldehydes produced a slower occurring death, and increased lung damage due to irritation on the lung tissue, rather than on the bronchi. An example was the propionaledehyde, n-butyraldehyde which produced less bronchi irritation than lung irritation.

Pattle and Cullumbine (73) found that formaldehyde was intermediate in irritation between the highly irritant unsaturated aldehydes, acrolein and crotonaldehyde and corresponding saturated aldehydes, propionic and butyric, which were considered almost nonirritant. Salem and Cullumbine (98) and Sim and Pattle (67) suggested that the unsaturated aldehydes (acrolein and crotonaldehyde) were more toxic than the saturated aldehydes.

Halogenated Hydrocarbons

Comstock and Oberst (99) studied the respiratory toxicities of 4 halogenated hydrocarbons in the presence of gasoline fires on rats and mice. Carbon tetrachloride ($C\ Cl_4$), Monochloromonobromomethane ($CH_2\ ClBr$), difluorodibromomethane ($CF_2\ Br_2$), and trifluoromonobromomethane ($CF_3\ Br$) have been widely used commercially as fire extinguishers. When any one of the 4 extinguishing fluids is applied to a gasoline fire, the resulting gaseous products include unaltered vapors, carbon monoxide and carbonyl halides. Trace amounts of halogens and halogen acids, soot and smoke may also be present. Short term exposures to small gasoline fires in well-ventilated spaces and where the concentration being produced is under ordinary condition by the contents of 1 standard fire are not as dangerous as when there is prolonged exposure in closed spaces. In one study, CF_2Br_2

appeared the most toxic to mice when fires were extinguished rapidly, However, it was noticed that the amount of carbon monoxide, carbonyl halides, and other decomposition products varied and depended on the intensity of the heat and the relative humidity.

Not all the breakdown products which are formed when heat is applied to the test compounds are known. The presence of carbon monoxide and carbonyl halides was suggested as contributing the greatest toxic effects. CCl_4 and CH_2 ClBr as extinguishers were more toxic to rats, while CF_3 Br was one of the least toxic to both rats and mice. Exophthalmor and pallor were characteristic in rats exposed to all 4 agents when the fire was extinguished slowly. Rats and mice showed congestion of the trachea and lungs after exposure to these compounds in the presence of fire. With CCl_4, there was also pulmonary edema and focal necrosis of the liver (99).

CHAPTER III

PURPOSE AND SCOPE

It has been increasingly evident that toxic or adverse effects of smoke and gas inhalation will continue to be a health problem, especially with increasing emphasis on long-term effects. The chief reason for studying the biologic effects of smoke and gas inhalation has been to understand its influence on man's health and activities.

It has been demonstrated that exposure to intense heat or acrid smoke can damage the respiratory tract. This has varied from mild edema of the larynx to severe pulmonary injury with pulmonary edema leading to death in man (4, 5, 6, 7, 9, 10, 16, 18, 19, 100, 101, 102).

The extent of pulmonary injury has been frequently under-assessed because of the lack of a pathophysiological understanding. This has often deprived many victims of effective therapy and has promoted acute disability and fatal pulmonary complication (103, 104). The pathologic and physiologic alterations of the pulmonary system caused by thermal injury have been described in both man (4-10) and animal (11-14).

Sudden severe alteration of pulmonary function has been a common emergency which has required the prompt institution of effective measures directed toward the restoration of function. Exposure of the respiratory apparatus to a variety of chemical substances or to superheated gas mixtures has precipitated such crises. The physiological and pathological

response to this type of injury has followed a general pattern independent, to a large extent, of the specific nature of the chemical irritant or physical state of the respired mixture (105).

For years, the disease importance of smoke and gas inhalation has been the center of repeated controversies. Yet the observations and conclusions drawn from many discussions have been more or less general and derived from inferential or purely speculative evidence. The number of experimental subjects exposed to known environmental concentrations of smoke gases and vapors or severely burned victims for short-term exposure has been fairly extensive; however, very little work concerning occupational exposure to high and/or unknown concentrations for long-term exposure has appeared. In view of the lack of comparability among existing studies, a need for further research in occupational exposure covered in this review has been evident.

Therefore, it was felt necessary to study the influence of smoke and gas inhalation on the pulmonary function of Firefighters and to determine the relationship of repeated smoke and gas inhalation effects to the changes in the pulmonary system reported in the literature. This research was designed to determine: a) the pulmonary functional capacity of the average Firefighter, b) whether there was an increased risk of pulmonary impairment associated with the length of time in the Firefighters occupation, and c) whether a career in the Fire Service has produced a higher incidence of pulmonary disease or adverse effects in active members than in the general urban population which have been daily exposed to many of the pulmonary irritant gases through breathing ambient air.

The primary purpose of this study was accomplished by utilizing 549 Oklahoma City Firefighters and 151 control volunteers. This study

was conducted over an 8-month period and the measurements from one test on each subject were used in the evaluation. Within the scope of this study was a specific objective to provide a foundation for assessing hazard effects to which Firefighters are occupationally exposed; thus, providing a long-term projection for evaluating pulmonary function involvement during the course of smoke and toxic gas inhalation, of which this study is one phase.

Pulmonary function testing has been an invaluable tool for close study on specific lung functions that have been impaired by injury or disease; therefore, it was the intention of this study to stipulate a clearer concept of pulmonary disease process, of pathologic physiology and natural course of pulmonary alteration or impairment. By ascertaining the awareness of the occupational hazards involving the Firefighter, determining his injury and disease experience, and awareness of other problems, it was felt that valuable information could be documented about exposure to smoke and toxic gas effects on lung function.

CHAPTER IV

MATERIALS AND METHODS

Selection of the Sample

This study was made possible by the cooperation from the Oklahoma City Firefighters' Association, local Military Reserve Units, Faculty, Students and Employees of the University of Oklahoma Health Sciences Center, College of Health, Ollahoma City, Oklahoma. These organizations and their personnel provided information and data to conduct this research.

The Oklahoma City Firefighters were selected as a group to study the effect of occupational hazards on their health. Of the 718 men in the Fire Service, 549 volunteered for the pulmonary function study. These men were selected from the 30 fire stations then functioning in the Oklahoma City area which covered 648 square miles and served a population of 526,805. The Fire Service was divided into three shifts, described as red "A", blue "B" and green "C" platoons and each shift was on duty for one 24-hour period, two to three times a week. Due to their assignment schedule, the firefighters were tested according to the shift on duty. If any men were absent from the fire station, they were tested on a volunteer basis at another station that same day or on an off-duty day.

The 151 control subjects were volunteers selected from local military reserve units, and the faculty, students, and employees of the University of Oklahoma Health Sciences Center, College of Health, Oklahoma

City, Oklahoma. The only criterion required for acceptance as a subject was that of never having been employed as a Firefighter.

This study was conducted over an 8-month period and the measurements from one test on each subject were used in the evaluation.

Survey Techniques

The study consisted of two parts: a questionnaire and a pulmonary function test.

The questionnaire was employed in order to record identifying information, detailed occupational, medical and smoking histories as well as any respiratory symptoms including cough, phlegm production, shortness of breath, chronic wheezing and other past respiratory conditions. The respiratory symptoms, occupational and smoking histories of the questionnaire were similar to those employed by Hepper et al. (106) and Weiss (107). The complete questionnaire, as used in the study, appears in the Appendix.

All subjects studied were males and were grouped into 5-year age groups based upon their last birthday at the time of the study. These groups were: 20 to 24, 25 to 29, 30 to 34, 35 to 39, 40 to 44, 45 to 49, 50 to 54, and 55 or above. The test subjects were also grouped according to the length of years in the fire service which ranged from less than 1 year to 28 years. The test and control groups were compared for smokers and nonsmokers. Since the number of nonwhites was too small to detect any racial difference, the subjects were not grouped by race.

The Vitalograph Spirometer utilized in this study was a single breath, wedge bellows, subject triggered instrument, used to measure and record ventilatory capacities of the lungs. The calibration was verified

by measurement against the air displacement method and valid at a standard atmospheric pressure of 760 mm Hg, a temperature of 20°C and saturated with water vapor (STPS). The vitalograph's chart speed was calibrated at 30.0 mm per second and volume rate of 29.6 mm per 1000 ml of gas at STPS. The vitalograph instrument provided a full 7.8 liter reading at body temperature, standard barometric pressure and saturated (BTPS). After one breath, the spirometer yielded a vitalogram that provided: (a) vital capacity (VC) which indicated the volumetric capacity of the subject's ventilatory system and was influenced to a great degree by height, sex and age, (b) the forced vital capacity (FVC) which added the element of forceful expiration in relation to time and revealed the entire ventilatory pattern, (c) forced expiratory volume (FEV_T) which indicated the volume in a certain period of time, (d) a forced expiratory ratio (FEV_T) which represented the volume at a given time interval, expressed as a percentage of the forced vital capacity and was determined by using the Percentage Scale provided with the instrument, and (e) forced mid-expiratory flow (FMF) which was determined with the aid of the Flow Rate Calculator using the 50th percentile of the FVC curve. This latter measurement revealed the physiological status of the smaller airways. Predicted values are available in Kory et al. (108) and Garbe and McDonnell (109).

Description of Vitalograph Spirometer System

The volume of air exhaled by the subject entered the measuring bag of the vitalograph. The bag, being of special configuration and resting on a flat surface, lay flat when empty and contained only a negligible amount of dead space. When air flowed into the bag, its top began to rise, moving through an arc. A curved stylus arm attached to the bag

carried the stylus and the stylus point described in an arc exactly identical to that of the bag top. The vertical excursion of the stylus was recorded on a chart which was driven in a horizontal direction at a constant speed.

The vertical displacement of the stylus point, when writing on the chart, was exactly related to the air volume in the bag as well as to the rate at which air flowed into it, viz., recording the volume and the rate of flow at which the subject expired into the instrument. The arrangement thus described would not be capable of recording the linear gas flow by a correspondingly linear stylus displacement. This would be due to the center of gravity of the bag top being shifted when moving through an arc and mass acceleration of the bag top for which no calibration was obtainable, as it varied from subject to subject. This potential source of error in dry spirometers was overcome in the Vitalograph by a counterbalancing system assisting the bag top at the beginning of its lift, thereby reducing inertia, and braking its movement proportionally to the rate of mass acceleration and weight reduction as it moved through the arc. This ensured a perfectly linear and therefore true recording of air volumes and rates of flow.

Spirometry Testing Procedure

The purpose of the test was explained to each subject or to a group of subjects when tested simultaneously, and the method of testing was demonstrated. The subjects were allowed to observed the tests being performed by others, therefore the test was applied by way of competition as to who produced the best results. The instruction to the subjects was a short explanation of what they were expected to do, and a brief demonstration how to do the test properly. This took no longer than a few

minutes showing: (a) how to inspire maximally, (b) how to put the mouth around the mouthpiece, and (c) how to expire forcefully and, if applicable, maximally, into the Vitalograph.

The need for avoiding leaks around the mouthpiece was emphasized. Dentures of any kind tend to cause leakage of air at the mouthpiece and subjects were asked not to wear them while performing the test. The subjects were also asked to remove tight jackets, coats and other apparel which might tend to restrict their thoracic and abdominal movements during test. For subjects suspected or subsequently found to require long periods for expiring their total vital capacity, a noseclip was applied. All questions asked by subjects were answered and every effort was made to allay apprehension and promote cooperation. All tests were performed with the subject in the standing position in order to measure maximal values, viz. without the restriction effects resulting from other body positions.

Force Expiratory Volume (FEV_T)

The subject was asked to breathe normally and quietly until an even and regular chest movement was observed. Then the subject was instructed to inspire fully until maximum inspiratory level was reached, i.e., deepest possible inflation of the lungs and maximum expansion of chest, to apply his lips firmly around the mouthpiece and to exhale as hard and as fast as possible into the instrument, viz. forced expiration.

If FEV_T only was measured, it was not necessary for the subject to exhale completely. However, in practice, it was expedient for the actual test recordings to record FVC as well. Experience showed that the first and second attempts tended to be low compared with subsequent

ones; therefore, at least two trials were allowed, followed by a least two consecutively valid attempts. If the FVC was not measured, no more than an 0.5-minute rest was allowed between trials. If the breath was to maximal expiration (viz. FVC), considerable fatique resulted and longer rest periods were necessary. Such rest periods were considered completed as soon as the subject established normal breathing, viz. an even and regular chest movement. The FEV_T test was repeated until satisfactory duplicate values of valid attempts were obtained and the maximum values recorded were used for evaluation. Forced expiratory volume may be measured over any time interval, however, in this study, the 1-second and 0.5-second measurements were employed. The 1-second and 0.5-second forced expiratory volumes are expressed as percentages of the forced expiratory volume ($FEV_{1.0}/FVC$) or $FEV_{0.5}/FVC$). The FEV/FVC ratio is useful especially in assessing the degree of obstruction. In normal subjects, $FEV_{1.0}$ should be in excess of 75 per cent, depending on age, with the normal for young, healthy subjects being 83 per cent. If in excess of 85 per cent, it will give information on the degree of restriction, or indicate a faulty attempt due to leakage or failure of the subject to inspire fully prior to the test. Air trapping as in emphysematous persons, also may be indicative, especially if occurring in multiple attempts at a reducing scale. $FEV_{0.5}$ also has been shown to be an equally valid measurement, both as a times volume measurement itself and in relation to the FVC. In normal person, $FEV_{0.5}/FVC$ should exceed 50 per cent of the total FVC. Until recently, no means were readily available for predicting normal values of FVC and FEV_T. Kory et al. (108) produced prediction formulae developed on a large population sample of male subjects and a nomogram, based on age and height, for VC, $FEV_{1.0}$ and $FEV_{0.5}$.

Forced Mid-Expiratory Flow (FMF)

The FMF or ($FEF_{25-75\%}$) was originally introduced by Leuallen and Fowler (110) and was a measurement useful in evaluating airway obstruction based on the hypotheses that the first portion of the forced expiratory spirogram showed the least delay, whereas the last portion was characteristically slower when compared to the normal. The middle 50 per cent portion of the FVC was, therefore, selected as a portion providing a more sensitive index for separating obstructive airway defects from normal states. Hyatt et al. (111) also showed that the FMF was largely independent of effort and slowed in the presence of obstruction of the small airways.

Time - volume indices, such as those given above, in the measurement of ventilatory capacity may be decreased because of poor cooperation, poor muscular effort, or because of airway obstruction. In the latter condition, the time volume indices are a useful measure of this obstruction, but should be noted these indices may be decreased for many reasons.

Average values for the FMF range from 2.0 liters/sec to 6.7 liters/sec (100 to 400 liters/minute). Prediction tables for the forced mid-expiratory flow (FMF) are found in Table 16 in the Appendix.

Forced Vital Capacity (FVC)

The test procedure for the FVC was similar to that for the FEV_T, except the subject was asked to continue exhaling after the initial rapid and forceful expiration until maximum expiratory level was reached. In normal or near-normal subjects, the FVC measurement was repeated until duplicate values within $\pm$ 5 per cent were obtained. In subjects with trapped air, the FVC may be progressively reduced; therefore, the rest periods

between attempts were considerably prolonged or a vital capacity measurement was made.

The forced vital capacity is a measure of the static volume limits of the lung, but related to maximum voluntary effort. In health, its value is equivalent to that of vital capacity. The predicted values for FVC were obtained from the nomogram described by Kory et al. (108) and Garbe and McDonnell (109).

Vital Capacity (VC)

The VC is a measure of the thoracic bellows and its volumetric value is not related to the time or effort taken for the measurement; however, this test is advised particularly in subjects in whom air trapping suspected or likely to occur. For this test, the subjects was asked to inspire maximally, following normal and quiet breathing, to apply his lips firmly around the mouthpiece and exhale into the instrument, smoothly and completely without force or haste until maximum expiratory level was reached, viz., until no further excursion of the stylus could be obtained even when exhorting the subject to exhale further. A leakproof fitted noseclip was used in some cases.

Vital capacity may be reduced by a number of factors, but in many instances, its decrease is the result of an absolute reduction due to malfunctioning lung tissue. Examples of this are mass lesions of the lungs, occlusion of a major bronchus by bronchogenic carcinoma, bronchiolar obstruction, pulmonary, obstruction, pulmonary edema, pneumonia, atelectasis, pulmonary fibrosis, pulmonary congestion and surgical excision of pulmonary tissue. Sometimes there is a decrease in vital capacity because of inability to expand the lungs or thorax, even though there is no primary

disease of the lungs. Examples of this are: (a) limitation to chest expansion caused by certain bodily positions such as a lateral position during thoracic surgery, tight strapping, scleroderma, body deformaties such as kyposcoliosis, thoracic pain (caused by fracture of a rib or by thoracic or upper abdominal incisions) or by neuromuscular disturbances such as occur in poliomyelitis, peripheral neuritis, myastenia gravis, and primary muscular disorder, (b) limitation to the descent of the diaphragm owning to ascities, abdominal tumor, pneumoperitoneum, or paralysis of phrenic nerve, and (c) limitation to expansion of the lungs because of pleural effusion, pneumothorax, diaphragmatic hernia, marked cardiac enlargement or a large pericardiac effusion (111).

Vital capacity may vary as much as 20 per cent in healthy subjects, even if of the same height, weight, age and sex. Reduced VC may occur in so many diseases that it is not characteristic of any single disorder and, in fact, it may not necessarily signify pulmonary disease; on the other hand, VC may be normal in persons suffering from pulmonary disability, as commonly found in emphysema (109, 111). The average values for vital capacity is 4.8 liters and 6.0 liters for total lung capacity. These values may change with position, age, size, sex and altitude. The nomogram used in this study was described by Kory et al. (108) and Garbe and McDonnell (109).

The data were tabulated and computed by utilizing an electronic computer. Statistical evaluation was accomplished by using the "t" test for identifying significant grouped mean values of the Firefighters and non-Firefighters. Comparisons by this method were also employed to detect differences between smokers and nonsmokers of Firefighters and non-Firefighters by age and length of time in the Fire Service. Linear

regression was also used to compare ventilatory parameters against such variables as age and length of time in the Fire Service.

CHAPTER V

OBSERVATIONS AND DISCUSSION

The observations on the pulmonary function of Firefighters and their control group are presented as a consideration of the response to occupational hazards encountered by the Firefighters. Five hundred and forty-nine Firefighters were studied from the standpoint of whether a career in the Fire Service produced a higher incidence of pulmonary impairment or adverse effect when compared to their control group. The comparisons of the Firefighters and controls are shown in Tables 2 through 15 and Figures 2, 3, and 4 in the Appendix. However, in order to interpret the type of pulmonary impairment represented by these measurements, the following preliminary discussion is presented.

Pulmonary Impairment

Pattern of diffuse obstruction of smaller airways

Obstruction of smaller airways may occur because of (a) constriction of bronchiolar smooth muscle, (b) mucosal congestion or inflammation, (c) edema of bronchiolar tissues, (d) plugging of the lumen by mucous, edemic fluids, exudate or foreign bodies, (e) cohesion of mucosal surface by forces of surface tensions, (f) infiltration, compression or fibrosis of bronchioles, or (g) collapse or kinking of bronchioles due to loss of the normal pull of alveolar elastic fibers on bronchiolar walls or to loss

of structural, supporting tissues of the bronchial walls.

The obstructive pattern may be a simple one in which airway obstruction is the only pulmonary abnormality (as in Asthma); it may be more complex, such as when structural disease of the lung causes the obstruction (as in Emphysema), or it may be present as a pattern which complicates a variety of diseases.

Collapse of airways because of weak walls or loss of traction normally provided by the elastic properties of surrounding tissues is displayed particularly by measurement of the forced mid-expiratory flow. If obstruction is reversed completely and rapidly by bronchodilation or therapy, it may have been due to smooth muscle constriction, muscosal congestion or edema, or plugging of the lumen. There is no rapid reversal in the presence of organic narrowing of the lumen (as in peribronchiolar fibrosis) or of destruction of tissues resulting in a check valve mechanism (as in obstructive emphysema). Relief of obstruction by isoproternol which is both bronchodilator and vasodilator, would suggest that smooth muscle constriction is the mechanics responsible for the obstruction. Relief following use of a powerful vasoconstrictor, administered as an aerosal, would indicate that vascular congestion is of critical importance (109, 111). The forced expiratory flow may show low rates of flow resembling an obstructive pattern when respiratory muscles are weak or paralyzed.

Obstructive airway disease displays the following patterns of altered function: (a) vital capacity may be normal if none of the airways are obstructed completely and it is decreased if the obstruction of some airways is complete or if the narrowing is so severe that airways closes completely during forced expiration; (b) the forced expiratory flow, mid

expiratory flow, FEV_T decrease far more than does the vital capacity; and (c) if an individual has bronchial asthma, these ventilatory parameters show normal or nearly normal values following bronchodilator therapy. If the person has emphysema or other disease which permits collapse of the medium size to fine airways, there is little improvement in these parameters following bronchodilator therapy. Forced mid-expiratory flow shows obstruction at its maximum extent from normality and is useful for assessing borderline cases (111 - 119).

Chronic bronchitis

Bronchostenosis is another name given chronic bronchitis and is characterized by decreased FEV_T, and FMF which values are little influenced by bronchodilation, vital capacity decreases significantly only when a sufficiently large number of airways are closed completely. The obstructive pattern in chronic bronchitis differs from that in asthma because bronchodilator therapy does not cause appreciable or prolonged improvement and differs from that in emphysema because in bronchitis, total lung capacity is normal and the airways resistance is increased on both inspiration and expiration.

Cigarette smoking also displays an obstructive pattern in more or less reduced FEV_T AND FMF, showing little improvement after bronchchodilation (109, 111, 120).

Obstructive emphysema

The reduced VC, FEV_T and FMF are all characteristic of an obstructive pattern. Inhalation of bronchodilator fails to produce any significant change in VC or flow rates; pointing to an irreversible type of airway obstruction, the most common cause of which is chronic pulmonary

emphysema. The obstruction may be due to (a) weak, collapsing bronchial walls on expiration, (b) occlusion of alveolar air passages due to loss of opening traction of surrounding tissues, or (c) to plugging by mucous due to excessive secretion or to decreased expiratory flow or change in ciliary action reducing the efficiency of the removal mechanism.

This test establishes (a) the degree of severe chronic pulmonary emphysema in this certain person (with insufficient function of oxygenation and removal carbon dioxide), (b) that the mechanical limitation to breathing is severe but disability is not marked, (c) that it is unlikely to gain improvement by bronchodilator therapy, (d) that positive pressure breathing therapy appears unlikely to result in improved expiratory flow and in more complete or rapid expiration (109, 120-126).

Bronchial asthma

Reduced FEV_T, VC and FMF are all part of the pattern of obstruction, restrictive lesions of the lungs and thorax are ruled out because of increased total lung capacity (109, 112, 127, 128, 129).

Pattern of impaired diffusion

Impairment of diffusion may occur because there is (a) a longer diffusion path for oxygen from alveoli to blood, or (b) a decrease in surface area for gas exchange. Vital capacity is decreased, but the FEV_T and forced expiratory flow rates may be normal even when the vital capacity is reduced considerably. This type of function is due to the pure alveolar capillary block, but is not associated with airway obstruction; the individual can still breathe in and out rapidly. A restrictive pattern is very often present also, and an obstructive one in the terminal stages (109, 112).

Pattern of pulmonary vascular congestion

This pattern is seen characteristically in mitral stenosis, left ventricular failure, left to right shunts and in hypervolemia. No single pattern is characteristic of all of these varied conditions. For this reason, the pattern presented here will be that of pulmonary congestion caused by mitral stenosis or left ventricular failure, which is fairly uniform; in certain stages it closely resembles the patterns of impaired diffusion and restriction.

The vital capacity is decreased and there is no gas trapping. Forced mid-expiratory flow and FEV_T may be normal until pulmonary edema develops (109, 112, 130, 131).

Patterns of restriction

In pure restrictive airway disease, the characteristic feature is limitation of full expansion of the lungs even though there is no neuromuscular disease. This may be due to diffuse pulmonary disease (such as interstitial fibrosis or scleroderma) pleural thickening, pleural effusion or pneumothorax, disease of the thoracic cage (Kyphoscholiosis, ankylosing spondylitis) or an unusual elevation of the diaphragm (large abdominal masses). This pattern in its pure form does not include airway obstruction; therefore, the FMF performed with quick small breaths, remains normal despite considerable reduction in vital capacity. An obstructive pattern may develop later and complicate the pure restrictive pattern; presumably this occurs because inability to breathe deeply and cough effectively which favors an accumulation of mucus, alelectasis, infection and scattered fibrotic changes.

The restrictive pattern is characterized as follows: vital

capacity decreases markedly because of limitation to full expansion; the FEV may be normal or nearly so, despite a considerable reduction in vital capacity, FEV_T per cent is often considerably above normal due to the reduced vital capacity; if the chest is rigid, the diaphragm can make full rapid excursions; if the diaphragm is crowded into the thorax, the chest cage can still move quickly; if the FEV_T is reduced, it is considerably less than the vital capacity; and the forced expiratory flow rates are usually decreased slightly, but rarely to the extent seen in the obstruction pattern (109, 112, 132, 133).

Patterns of hypoventilation

Hypoventilation exists when an insufficient volume of fresh air enters the alveoli each minute relative to the metabolic activity of the body, leading to anoxemia, carbon dioxide retention, and respiratory acidosis. It can result from many different causes; therefore, it may occur in normal subjects during deep sleep, in individuals with normal lungs and thorax, in individuals with normal lungs but a rigid thorax (Kyphoscoliosis), and in individuals with very severe obstructive or restrictive pulmonary disease (asthma, emphysema, interstitial fibrosis). Pulmonary function studies in persons with hypoventilation (but no detectable abnormality in the lungs or thorax) show the following typical patterns: vital capacity may be normal or markedly reduced if there is depression or blockage of the neural pathways from the respiratory center to the respiratory muscles; FEV_T may be normal or above normal; and the FMF is decreased when there is interference with neuromuscular condition and transmission (109, 112, 134).

Pattern of aging

Men above 50 to 60 years may have no symptoms of cardiopulmonary disease and have normal lungs on clinical and radiologic examinations. Although (in exceptional cases) subjects between the ages of 50 and 90 may have pulmonary function comparable to that of health young men, the majority of older persons display the following pattern of altered pulmonary function: (a) the VC is decreased and FEV_T and forced expiratory flow rates may also be decreased, (b) the elastic tissues of the lungs appear to change with age and (c) the muscle force decreases, which may be responsible for a decrease in flow rates. It is important in evaluating pulmonary function in older men to know that these individuals may have no pulmonary symptoms even though the FMF may be as low as 50 per cent. However, the changes produced by relatively mild pulmonary disease added to pre-existing changes caused by aging, may lead to definite symptoms because of the decrease in reserve (109, 112, 135, 136). Because of this, both Firefighters and controls were grouped into 5-year age groups and the mean values within the groups was used for comparisons.

Height and Weight Factors

The vital capacity, correlated by using the body weight, stem height, surface area, circumference and volume of the thorax, radiologic lung area or volume, age or any combination of these measurements has been utilized in predicting lung volumes (137, 138, 139). Most attempts trying to describe a linear regression have been made using body build and lung volume; therefore, good correlations was obtained only over limited ranges. To overcome this, formulas utilizing multiple linear regression have been derived.

Kelly (140), in a study of vital capacity of boys and girls, first identified the relationship between vital capacity and the cube of height. Bateman (141) later expressed the relationship of the total lung volume and its subdivisions to the third power of the body height of adults of average stature, subsequently, Moore et al. (142) Helliesen et al. (143 and Engstrom et al. (144) described this relationship in boys and girls.

Of the various measurements of body build, height generally has been found to be the single measurement with which lung volumes can be best correlated. Previous studies described lung volumes as a linear function of height dealing with persons of a limited height; over such limited ranges, the correlation was considered satisfactory. However, Hepper et al. (145) found this correlation unsatisfactory when the height range was extended. The correlation existing between lung volumes and the third power of height was suggested as a more reasonable correlation than with the first power of height, since the body is three-dimensional. This has been pointed out several times in the literature, but its use has not been widely adopted.

Since weight and height of the Firefighters were variables which influenced the measurements of the ventilatory capacities a great deal, these variables were considered as contributory factors which produced better performances for the Firefighters at the beginning of their career. Height and weight comparisons are shown in Tables 2 and 3 in the Appendix and it appears that Firefighters are physically better subjects at the younger age than the control group. This is evident in the increased lung volumes implicated in this study.

Vital Capacity (VC)

The mean value of the VC for each age group in relation to Firefighters and controls is shown in Table 4 in the Appendix. There was a steady decline in this value with increasing age in both test and controls; however, the Firefighters' values decreased at a faster rate.

The observed values for the Firefighters were higher in the younger age group of 20 through 39 and lower in the older groups when compared to the controls. Only the 20 to 24 age group was shown significant at the 0.05 level due to their better physical parameters at the early age; however, the observed mean values for the control age group 40 and above were different when compared to the Firefighters.

Forced Vital Capacity (FVC)

The mean value for the FVC for Firefighters and control in relationship to increasing age is shown in Table 5 in the Appendix. It is apparent that in all groups, the FVC exhibited a general downward trend with increasing age, but the difference between the values for the youngest age group 20 to 24 was significant at the 0.05 level due to physical measurements.

The observed values for the Firefighters were higher in all age groups, except the 30 to 34 and 45 to 49 groups, but again the decrease demonstrated a faster decline with age than the control group. While the differences between the two groups were significant only at the 0.05 level for the 20 to 24 age group, the differences of the mean values for the other groups were evident.

1-Second Forced Expiratory Volume (FEV_1)

The mean value of the FEV_1 for each age group in relation to the

Firefighter and control groups is shown in Tables 6 and 7 and Figure 2 in the Appendix. The results obtained for the FEV_1, closely parallel the trends shown in evaluation of the two previous parameters of ventilation which indicated a steady drop in the values with increasing age in both test and control groups. As noted previously, the Firefighters' mean values decreased at a faster rate. Except for the younger group, the differences between the two groups were not significant at the 0.05 level; however, the control group showed consistently higher values for age groups 25 to 29, 30 to 34 and 40 to 44. This reduced volume of timed forced expiratory in Firefighters is patterned after that found for airway obstruction and restriction due to occupation.

1-Second Forced Expiratory Ratio (FEV/FVC)

The mean value for the FEV_1 as a percentage of the forced expiratory volume for the Firefighters and controls in relationship to increasing age is shown in Table 8 in the Appendix. The decrease with age is demonstrated and a consistent difference between the rate of decrease is shown, with the Firefighters have a faster decline. The difference between the two groups was only significant at the 0.05 level for the age group 45 to 49, but a distinct difference is evident. The Firefighters were consistently lower in all groups except 35 to 39 and 50 or above and the controls were higher for the other five age groups when comparing mean values.

A decrease in timed forced capacity determination, such as forced expiratory volume in 1-second or 0.5-second reflects a lowered expiratory flow rate. This change depends on narrowing of the bronchi due to the smooth muscle spasm or mucosal edema and mucous secretion (146); therefore, occupational exposure, as a contributory factor, is suggested.

0.5-Second Forced Expiratory Volume ($FEV_{0.5}$)

As noted previously, a steady drop in the mean values with increasing age is evident (Table 9 in the Appendix) for both Firefighters and control. Mean values were consistently lower for the Firefighters except in the 20 to 24, 35 to 39 and 50 or above age groups while the controls revealed higher values for all other age groups. The difference between the two groups was not statistically significant at the 0.05 level for any age group, but the difference was distinctively different.

0.5-Second Forced Expiratory Ratio (FEV/FVC)

The mean values for the $FEV_{0.5}$ as a percentage of the forced expiratory volume for the Firefighters and controls in relationship to increasing age are shown in Table 10 (Appendix). The mean values for the Firefighters were lower in all age groups except 50 or above. Firefighters at this age are promoted to other duties not as hazardous in the Fire Service; therefore, are less occupational exposed. The mean values were not significant at the 0.05 level for any age group, but the differences were suggestive of airway obstruction or restriction disease due to occupation.

Forced Mid-Expiratory Flow (FMF)

The results obtained for the FMF patterned the trends shown in evaluation of the previous parameters of ventilation. There was a steady decline in the mean values for the FMF with increasing age in both Firefighters and Controls; but as shown in Tables 11 and 12 and Figures 3 and 4 in the Appendix, it was apparent that the Firefighters' decreased at a faster rate. Except for the age groups 20 to 24 and 50 or above, the difference was not significant at the 0.05 level, but the mean values

were distinctively different (Table 13 in the Appendix). The difference described for the younger age group 20 to 24 is suggestive of the physical condition of the Firefighters at the beginning of their career; however, that for the 50 or above age group is indicative of the promotion practice to working condition where the occupation is less hazard. This reduction, coupled with the reduction in vital capacity and timed forced expiratory volume of Firefighters, is suggestive of obstructive and resistance airway disease due to the occupation.

1-Second Forced Expiratory Volume (FEV_1) and Forced Mid-expiratory Flow for Nonsmokers and Smokers

Nonsmokers were superior to smokers in the FEV_1 and forced mid-expiratory flow parameters for both Firefighters and controls when the mean values were compared for differences. The results for Firefighters are tabulated in Table 14 and those for controls in Table 15 (Appendix).

The most consistent differences were seen in the FMF which was shown significant for both Firefighters and controls. The FEV_1 mean value difference was only significant in the Firefighters; however, the controls indicated a distinct difference between the smokers and non-smokers. The mean value difference for the Firefighters was more significant than in the controls. Significance for differences were tested at the 0.05 level.

This progressive decrease in the mean value for the FMF noted in Tables 12 and 13 was also found by Bower (147) where the FMF declined with increasing age; but on the other hand, Flick and Patton (148) found no decrease in peak expiratory flow rates in nonsmokers up to the age 70. In this study, all age groups were grouped into smokers or nonsmokers for Firefighters and controls where mean value were compared for difference.

While cigarette smoking is associated with a consistently lower mean value for the $FEV_{1.0\ sec}$ and FMF for both groups tested, the decrease in $FEV_{1.0\ sec}$ for the test group and FMF for both groups showed statistically significant difference. Although ventilation in cigarette smokers is decreased even after one cigarette (149), it has not been possible to demonstrate a cause-and-effect relationship. Matorazzo and Saslow (150) recently reviewed the pertinent literature and demonstrated many differences in the character of smoker and nonsmokers. Thomas and Cohen (151) illustrated that cigarette smokers include a higher percentage of men able to taste phenylthiourea than nonsmokers, while Snyder (152) showed that this ability to taste phenythiourea is inherited as a recessive gene. Certainly, the differences between smokers and nonsmokers is not only the fact that some inhale cigarette smoke, but probably an inborn difference in the sensitivity of their bronchi to inhaled irritants. Read and Selby (153) suggested a genetic hypothesis which would explain the fact that some nonsmokers get bronchitis and ventilatory defects while some heavy smokers remain unaffected. They postulated the existence of two alleles "V" and "v". Homozygous "VV" subjects will get bronchitis and associated ventilatory defects in the absence of any irritants; "vv" subjects remain unaffected even in the presence of irritants and "Vv" subjects will get bronchitis and ventilatory defects if exposed to any inhaled irritants.

It is apparent that the FEV_1 and FMF bear an inverse relationship to increasing age and heavy cigarette smoking. As the prevalence of pulmonary impairment increases with age and the degree of cigarette smoke, it is not surprising that the ventilatory parameters should decrease in the presence of pulmonary defects.

Many different kinds of particles give rise to increased airway resistance. Thus, it is more probably that the reaction to tobacco smoke is not dependent upon the nicotine effect along, but that it is non-specific and is elicited by reflexes from the respiratory tract, the magnitude of which are due to the sensitivity of the receptors in the bronchi (154-163).

CHAPTER VI

SUMMARY AND CONCLUSIONS

This investigation was concerned with the measurement of occupational exposure effects as evidence by alterations in the pulmonary function. Five hundred and forty-nine Firefighters were selected from the Oklahoma City Fire Service for this study, of which 440 were smokers and 109 nonsmokers (Table 17 in the Appendix). The control group was composed of 151 volunteers, consisting of 52 nonsmokers and 99 smokers. The Firefighters' age range was 20 to 63 (their length of fire service at the inception of this study was 3 months to 28 years) compared to the controls whose age range was 20 to 67 years. The smokers in both groups averaged one to two packs of cigarettes per day and all inhaled. The initial health profile did not reveal marked disease of the heart, lung, liver, kidney or nervous system, but some health issues such as bronchitis, asthma and emphysema symptoms were discovered in the initial evaluation in some of the test and control subjects.

The Vitalograph Spirometer and respiratory questionnaire were utilized in this study to better evaluate the potential hazard effects which could arise from occupational exposure.

The "t" test was employed to detect significant differences in mean values between the test and control groups. The measurements made were: (a) vital capacity (VC), (b) forced vital capacity (FVC), (c) forced

expiratory volume (FEV_T), (d) forced expiratory ratio, (e) forced mid-expiratory flow (FMF), and height and weight. The 20 to 24 age group showed significance at the 0.05 level in the VC,FVC,$FEV_{1\ sec}$, FMF and height; whereas the 45 to 49 age group showed significance at the 0.05 level for values in FEV/FVC, FMF and weight, and 35 to 39 for height and weight. The mean values of other ventilatory capacities were not significant at this level, but were distinctly different when the Firefighters and controls were compared. The following conclusions have been 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 group in height and weight with a better performance being evident in the younger age group 20 to 24.

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 FEV_1 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 represented an increased potential health hazard.

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

These findings indicated a direction and established a need for further work. It recommended, in order to eliminate some of the inherent variables, that such investigations include a longitudinal study which would make repeated measurements on the same age groups, that is, follow Firefighters in an age group through an extended part of their career.

LITERATURE CITED

1. Tidely, V. L. "Respiratory Hazards of the Fire Service", Minnesota Fire Chief, 9: 16 (1973).

2. Thomas, D. M. "The Smoke Inhalation Problem" Proceedings of a Symposium on Occupational Health and Hazards of Fire Service, Notre Dame University, South Bend, Indiana, 1971, pp. 21-27.

3. Thomas, D. M. and Conner, E. H. "Management of the Patient Overcomed by Smoke", J. Ky. Med. Assoc., 66: 1051 (1968).

4. Aub, J. C., Pitman, H., and Brues, A. M. "The Management of the Cocoanut Grove Burns at the Massachusetts General Hospital," Ann. Surg., 117: 835 (1943).

5. Phillips, A. W. and Cope, O. "Burn Therapy II. The revelation of Respiratory Tract Damage As a Principal Killer of the Burned Patient," Ann. Surg., 155: 1-12 (1962).

6. Phillips, A. W., Cope, O. "Burn Therapy III, Beware of the Facial Burn," Ann. Surg., 156: 759-766 (1962).

7. Phillips, A. W., Tanner, J. W., and Cope, O. "Burn Therapy IV. Respiratory Tract Damage and the Meaning of Restlessness," Ann. Surg., 158: 799-811 (1963).

8. Phillips, A. W. Pulmonary Complications in Burned Patients Bahama International Conference on Burns. Dorrance and Company. Philadelphia, 1964, pp. 7-24.

9. Socher, F. M. and Mallory, G. K., "Lung Lesions in Patients Dying of Burns," Arch. Path., 75: 303-308 (1963).

10. Taylors, F. W. and Gumbert, J. L. "Cause of Death from Burns: Role of Respiratory Damage," Ann. Surg., 161: 497 (1965).

11. Aviado, D. M. and Schmidt, C. F. "Respiratory Burns with Special Reference to Pulmonary Edema and Congestion," Circulation, 6: 666 (1952).

12. Aviado, D. M. "Therapy of Experimental Pulmonary Edema in the Dog," Cir. Res., 7: 108 (1959).

13. Moritz, A. R., Hendriques, F. C. and McLean, R. "The Effects of Inhaled Heat on the Air Passages and Lungs," Amer. Journ. Path., 21: 311 (1945).

14. Moritz, A. R., Hendriques, F. C., Dutra, F. R., and Weisiger, J. R. "Studies of Thermal Injury IV," Arch. Path., 43: 466 (1947).

15. Aviado, D. M. "The Lung Circulation," Pergamon Press, Ltd., New York, New York, 2: 783-827 (1962).

16. Baxter, C. R., DeGrosse, J. L. Green, N. J., "Pulmonary Function Following Thermal Trauma, Hypoxia," Annual Research Program Report U. S. Army Surgical Research Unit, Brooks Army Medical Center, Fort Sam Houston, Texas, 1958.

17. Levy, D. M. "Ammonia Burns of the Face and Respiratory Tract," Jour. A.M.A., 190: 873 (1964).

18. Connell, J. F., Jr. "Successful Therapy in Patients with Pulmonary Burns, Research in Burns," In C. P. Artz, Research in Burns. F. A. Davis Company, Philadelphia, Pa., 1962, pp. 19-25.

19. Shannon, D. W. "Resuscitation of the Burned Child," Proc. Roy Soc. Med., 50: 885 (1957).

20. Sevitt, S. and Gallagher, N. "Venous Thrombosis and Pulmonary Embolism: A Clinical-Pathologic Study in Injured and Burned Patients," Brit. J. Surg., 48: 475 (1961).

21. Schench, H. P. "Mast Cells in the Upper Respiratory Tract," Ann. Otol., 74: 864 (1965).

22. Stone, H. H., Rhame, D. W., Corbitt, J. D., Given, K. S. and Martin, J. D., Jr., Respiratory Burns: A Correlation of Clinical and Laboratory Results, Ann. Surg., 165: 157-168 (1967).

23. Reed, G. F. and Camp, H. L. "Upper Airway Problems in Severly Burned Patients," Ann. Otol., 78: 741-751 (1969).

24. Mallory, T. B. and Brickley, W. J. "Management of the Cocoanut Grove Burns at the Massachusetts General Hospital," Ann. Surg., 117: 865-884 (1943).

25. Epstein, B. S., Hardy, D. L., Harrison, H. N., Teplitz, C., Villarreal, Y. and Mason, A. D., Jr., "Hypoxemia in the Burned Patient: A Clinical-Pathologic Study," Ann. Surg., 158: 924 (1963).

26. Achauer, B. M., Allyn, P. A., Furnas, D. W., and Bartlett, R. H., "Pulmonary Complication of Burns: The Major Threat to the Burn Patients," Ann. Surg., 177: 311 (1973).

27. Pruitt, B. A., DiVincenti, F. D., Mason, A., Toley, F. D. and Flemma, R. J. "The Occurrence and Significance of Pneumonia and other Pulmonary Complications in Burned Patients," J. Trauma, 10: 519 (1970).

28. Beal, D. D., Lambeth, J. T. and Conner, G. H., "Follow-up Studies on Patients Treated with Steroids Following Pulmonary Thermal and Acid Smoke Injury," Laryngoscope, 78: 396-403 (1967).

29. Matsuura, Y. "Pulmonary Compliance and Surfactant Activity in Thermal Burn," Surg. Forum, 17: 86 (1966).

30. Crofton, J. W., Livingstone, J. L., Oswald, N. C. and Roberts, A.T. M. "Pulmonary Eosinophila," Thorox, 7: 1 (1952).

31. Dias-Rimera, R. S., Ramos-Morales, F. and Cirtron-Rivera, A. A. "Infiltration Eosinophila," Arch. Int. Med., 94: 102 (1954).

32. Reeder, W. H. and Gooduik, B. L. "Pulmonary Infiltration with Eosinophilia," Ann. Int. Med. 36: 1217 (1952).

33. Tschamy, W. "Pulmonary Infiltration with Eosinophilia (Loffer's Syndrome) Due to Smoke Inhalation," Ann. Int., 49: 665 (1958).

34. Hunter, D. "The Diseases of Occupation", Little, Brown and Co., Boston, pp. 521, 529, (1955).

35. Henderson, Y. and Haggard, H. E. Noxious Gases and the Principles of Respiratory Influencing Their Action, 2nd Edition, Reinhold Publ. Corp., New York, 1943, pp. 63 - 80.

36. Bull, J. P. and Fisher, A. P. "A Study of Mortality in a Burn Unit: A Revised Estimate," Ann. Surg., 139: 269 (1954).

37. Cope, O. and Rhinelanch, F. W. "The Problem of Burn Shock Complicated By Pulmonary Damage," Ann. Surg., 117: 915 (1943).

38. Jackson, T. M. and Lee, L. W., Jr. "Major Thermal Burns, A Mortality Appraisal and Review," Arch. Surg., 87: 937-948 (1963).

39. Schatzki, R. "Roentgenologic Report of the Pulmonary Lesion," Ann. Surg., 117: 841 (1943).

40. Long, J. "On the Post-Mortem Appearance Found After Burn," London Medical Gazzett, 25: 743 (1940).

41. Harrison, H. N. "Respiratory Tract Injury, Pathophysiology and Response to Therapy Among Burned Patients," Ann. N.Y. Acad. Sci., 150: 627 (1969).

42. Silverstein, P. and Dressler, D. "Effect of Current Therapy on Burn Mortality," Ann. Surg., 171: 124 (1970).

43. Foley, F. D., Moncrief, J. A. and Mason, A. D., Jr. "Pathology of the Lung in Fatally Burned Patients," Ann. Surg., 167: 251 (1968).

44. Moore, F. D., The Body Weight Burns Budget Basic Fluid Therapy for the Early Burn," Surg. Clin. North Amer., 50 - 6: 1249, (1970).

45. Allgower, M., Burri, C., Cueni, L., Engley, F., Fleish, H., Gruber, U. F., Harder, F. and Russel, R. G. G., "Studies of Burn Toxins," Ann. N. Y. Acad. Sci., 150: 807 (1968).

46. Schoenenberger, G. A. and Allgower, M. "Isolation, Activity and Physiological Characterization of a Burn Toxin from Mouse Skin," British Jour. Surg., 55: 704 (1969).

47. Blaisdell, F. W., Lim, R. C., Jr. and Stallone, R. J., "The Mechanism of Pulmonary Damage Following Traumatic Shock," Surg. Gynecol Obstet., 130: 15 (1970).

48. Morgan, A. P. "The Pulmonary Toxity of Oxygen," Anesthesiology, 29: 570 (1968).

49. Mosley, R. and Doty, D. F. "Changes in the Filtration Characteristics of Stored Blood," Ann. Surg., 171: 329 (1970).

50. Fineberg, C., Miller, B. J., Albritten, F. F. "Thermal Burns of the Respiratory Tract," Surg. Gyn. and Obstet., 98: 318-323 (1954).

51. Garzon, A. A., Seltzer, B., Song, I. C., Bromberg, B. E. and Karlson, K. E. "Respiratory Mechanics in Patients with Inhalation Burns," Jour. of Trauma, 10: 57 (1970).

52. Olson, J. C., Ferguson, C. E. and Scheflan, L., "Gases from Thermal Decomposition of Common Combustion Materials," Industrial and Eng. Chem., 25: 599 (1933).

53. Levin, M. "Smoke Poisoning Treated by Antacids," J. Med. Soc. New Jersey," 62: 215-216 (1965).

54. Browning, E. "Toxicity and Metabolism of Industrial Solvents," Amsterdam, Elsevier Publ. Co. pp. 80-121 (1965).

55. Wise, M. K. "Denver Draws Blood to Check Carbon Monoxide," Fire Engineering," 119: 28-31 (1966).

56. MacFarland, H. N. and Leony, K. J., "Hazards from the Thermodecomposition of Plastics-Polyurethane and Polyurethane Coated Nylon," Arch. Environ. Health, 4: 591-593 (1962).

57. Teppers, L. B. "Hazards to Health: Teflon," N.E.J. Med., 267: 349-350 (1962).

58. Robbins, J. J. and Ware, R. L. "Pulmonary Edema from Teflon Fumes," N.E.J. Med., 271: 360-361 (1964).

59. Fleming, A. J., D'Alonzo, C. A. and Zapp, J. A., "Modern Occupational Medicine," Second Edition, Philadelphia, Lea, 1960, pp. 200-260.

60. Harris, D. K. "Polymer - Fume Fever," Lancet, 2: 1008 (1951).

61. Battigelli, M. C. "Sulfur Dioxide and Acute Effects of Air Pollution," J. Occup. Med., 10: 500-511 (1968).

62. Frank, N. R., Arthur, J. W., Whittenberger, J. L. "Effects of Acute Controlled Exposure to SO_2 on Respiratory Mechanism in Male Adults," J. Appl. Physical, 17: 252-258 (1962).

63. Committee on Threshold Limits A.M.A. Arch Indust., 20: 266 (1959).

64. Kehoe, R. A. "On the Effects of Prolonged Exposure to Sulfur Dioxide," Brit. Jou. Int. Med., 7: 82 (1950).

65. Anderson, A. "Possible Long-Term Effects of Exposure to Sulfur Dioxide," Brit. Jour. Int. Med., 7: 82 (1950).

66. Amdue, M. O., Melvin, W. M., Jr. and Drinker, P. "Effects of Inhalation of Sulphur Dioxide by Men," Lancet, 2: 758 (1953).

67. Sim, Van M. and Pattle, R. E., "Effect of Possible Smog Irritants on Human Subjects," J. Amer. Med. Assoc., 165: 1908 (1957).

68. Ainsworth, M. and Eveleigh, J. W., Portion Technical Paper No. 331, Chemical Defense Experimental Establishment, Portion, Wilts, England, 1953.

69. Amdur, M. O. "The Physiological Response of Guinea Pigs to Atmospheric Pollutants," Inst. J. Air Poll., 1: 170 (1959).

70. Amdur, M. O. "Inhaled Particles and Vapors," Edited by C. M. Davids, Oxford: Pergamon Press, pp. 1-50 (1960).

71. Goldring, I. P. "Pulmonary Effects of Sulfur Dioxide Exposure in Syria Hamster," Arch. Environ. Health, 15: 167 (1967).

72. Reid, L. "Experimental Study of Hypersection of Mucus in the Bronchial Tree," Brit. J. Exper. Path., 44: 437 (1964).

73. Pattle, R. E. and Cullumbine, H. "Toxicity of Some Atmospheric Pollutants," Brit. Med. J., 2: 913 (1956).

74. Hemeon, W. C. L., "The Estimate of Health Hazards from Air Pollution," Arch. Indust. Health, 11: 397-402 (1955).

75. Amdur, M. O. and Corn, M. "Irritant Potency of Zinc Ammonium Sulfate of Different Particle Size," Amer. Indust. Hyg. Assoc. Jour., 24: 236 (1963).

76. LaBelle, C. L., Long, J. I. and Christofano, E. E. "Synergistic Effects of Aerosols," Arch. Indust. Health, 11: 297 (1957).

77. Goetz, A. "On the Nature of the Synergistic Action of Aerosols," Int. J. Air and Water Poll., 4: 158 (1961).

78. Hudson, L. G. "Explosives-Industry Poisons," Med. Rec N.Y., 91: 89 1971.

79. Greenbaum, R., Bay, J., Hargreaves, M.D., Kain, M. L., Kelman, G. R., Nunn, J. F., Pryr-Roberts, C. and Sielbold, K., "Effects of Higher Oxides of Nitrogen on the Anesthetized Dogs," Brit. Jour. Anaesth., 39: 393 (1967).

80. Clutton-Brock, J. "Two Cases of Poisoning by Contamination of Nitrous Oxides with Higher Oxides of Nitrogen during Anaesthesia," Brit. J. Anesth., 39: 388 (1967).

81. Zwerdling, M., Goldsworth, J. R., and Massey, F. "Maximal Expiratory Flow Tests in a Population Sample with an Application of Component Analysis," Amer. Ren. Resp. Dis., 87: 69 (1963).

82. Schultz-Braums, O. "The Fatal Poisonings by Gaseous Nitrogen Oxides (Nitrous Fumes) from Working with Nitric Acid," Arch. Path., 277: 174 (1930).

83. Lowry, T. and Schumann, M. L., "Silo-Fillers' Disease" A syndrome Caused by Nitrogen Dioxide," J. American. Med. Assoc., 162: 153 (1956).

84. Pryr-Roberts, C. "Principle of Treatment of Poisoning by Higher Oxides of Nitrogen," Brit. J. Anes., 39: 432-439 (1967).

85. Nichols, B. H. "The Clinical Effects of Inhalation of Nitrogen Dioxide," Amer. J. Roentgenal., 23: 516 (1930).

86. Schiotz, E. H. "Welding Regarded from the Medical Point of View," Acta. Med. Scandinav., 121: 537 (1945).

87. Pfenninger, E. "Effects of Exposure to Nitrous Fumes with Special Reference to Blood Findings," Schrveiz, Med. Wchnschi., 73: 1398 (1943).

88. Woods, F. C. "Poisoning from Nitric Oxide Fumes," Arch. Int. Med., 10: 478 (1912).

89. Lindquist, T. "Nitrous Gas Poisoning Among Welders Using Acetylene Flame," Acta. Med. Scand., 188: 210 (1944).

90. Johnstone, R. J. "Occupational Diseases and Industrial Medicine," W. B. Saunders Co., Philadelphia and London, 1948, pp. 218-221.

91. LaDue, J. L. "Bronchiolitis Fibrosis Obliterans" Arch. In. Med., 68: 663 (1941).

92. Blumgart, H. L. and MacMaker, H. E. Bronchiolitis Fibrosa, "Obliterans: A Clinical and Pathological Study," M. Clin. North Amer., 13: 197 (1929).

93. McAdams, A. J. "Bronchiolitis Obliterans," Amer. J. Med., 19: 314 (1955).

94. Camiel, N. R. and Berkham, H. L. "Inhalation Pneumonia from Nitric Tumor," Radiology, 42: 175 (1944).

95. Becklake, M. R. "Long Term Effects of Exposure to Nitrous Fumes," Amer. Ren. Tube, 76: 398 (1957).

96. Von Oettinger, W. F. "The Toxicity and Potential Dangers of Nitrous Fumes," Public Health Bulletin 272, Federal Security Agency, U. S. Public Health Service, 1941.

97. McAdams, A. J. Jr. and Krop, S. "Injury and Death from Red Fuming Nitric Acid," JAMA, 158: 1022 (1955).

98. Salem, H. and Cullumbine, H. "Inhalation Toxicities of Some Aldehudes", Toxicology and Appli. Pharm., 2: 183-187 (1960).

99. Comstock, C. C. and Oberst, F. W. "Comparative Inhalation Toxicities of Four Halogenated Hydrocarbons to Rats and Mice in the Presence of Gasoline Fires," Arch. Industr. Hygiene and Occup. Med., 7: 157, (1953).

100. Ball, J. P. and Fisher, A. J. "A Study of the Mortality in a Burn Unit: a Revised Estimate," Ann. Surg., 139: 269 (1954).

101. Kaltreider, N. L. and McCann, W. S. "Respiratory Response During Exercise in Pulmonary Fibrosis and Emphysema," J. Clin. Invest. 16: 23 (1937).

102. Nelson, T., Pillsbury, R. P. and Bowers, W. F. "The Use of Tracheotomy in the Burned Patient," Surg. Gynec. and Obst., p. 105 (1957).

103. Beal, D. D. and Conner, G. H. "Respiratory Tract Injury: a Guide to Management Following Thermal and Smoke Injury," Laryngoscope, 80: 25-35 (1970).

104. Moylan, J. A., Wilmore, D. W., Mouton, D. E. and Pruitt, B. A. "Early Diagnosis of Inhalation Injury Using 133 Xenon Lung Scan," Ann. Surg., 176: 477 (1972).

105. Conner, E. H., Dubois, A. B., Comroe, J. H. "Acute Chemical Injury to the Respiratory Tract and Lungs," Anesthesiology, 23: 538-547 (1962).

106. Hepper, N., Hyatt, R. E. and Fowler, W. S. "Detection of Chronic Obstructive Lung Disease: An evaluation of the Medical History and Physical Examination," Arch. Environ. Health, 19: 806 (1969).

107. Weiss, W. "Validation of a Screening Procedure for Chronic Non-specific Respiratory Disease," Arch. Environ. Health, 16: 844-853 (1968).

108. Kory, R. C., Callahan, R., Boren, H. and Syner, J. C. "The Veterans Administration - Army Cooperative Study of Pulmonary Function," Amer. J. Med., 30: 243-258 (1961).

109. Garbe, D. R. and McDonnell, H. "Lung Function Testing: An Introduction to Routine Clinical Tests Illustrated with the Use of the Vitalograph, 2nd Edition. Vitalograph Limited, Maids Moreton House, Buckingham, England, 1-68 (1964).

110. Leuallen, E. C. and Fowler, W. S. "Maximal Mid-Expiratory Flow," Amer. Rev. Tuberc., 72: 783 (1955).

111. Hyatt, R. E., Schiller, D. P., and Fry, D. L. "Relationship Between Maximum Expiration Flow and Degree of Lung Inflation," J. Appl. Physiol., 13: 331 (1958).

112. Comroe, J. H., Jr., Forster, R. E., Dubois, A. B., Briscoe, W. A. and Carlsen, E. The Lung: Clinical Physiology and Pulmonary Function Tests, Yearbook, 2nd Edition, Chicago, Illinois, 1962, pp. 7-10.

113. Lewinski, H. C., Capel, L. H. and Smart, L. "Changes in Forced Expiratory Volumes Throughout the Day," Brit. Med. J., 1: 462 (1960).

114. Lord, G. P., Gaziogln, K., Kaltreider, N. "The Maximum Expiratory Flow Volume in the Evaluation of Patients with Lung Disease," Amer. J. Med., 46: 72-79 (1969).

115. Suprenant, E. L. and Vance, J. W. "Evaluation of Methods for the Early Detection of Chronic Obstructive Ventilatory Diseases," Dis. Chest, 52: 760-766 (1967).

116. Thurbeck, W. M., Henderson, J. A., Frasier, R. G. and Bates, D. V. "Chronic Obstructive Lung Disease," Medicine, 49: 81 (1970).

117. Franklin, W., Lowell, F. C. "Unrecognized Airway Obstruction Associated with Smoking: A Probable Forerunner of Obstructive Pulmonary Emphysema," Ann. Int. Med., 54: 379 (1961).

118. Hogg, J. C., Macklem, P.T., Thurlbeck, W.M. "Site and Nature of Airway Obstruction in Chronic Obstructive Lung Disease," New Engl. J. Med., 278: 1355 (1968).

119. Simpson,T., Heard, B. and Laws, J. W. "Severe Irreversible Airways Obstruction Without Emphysema," Thorax, 18: 361 (1963).

120. Simonsson, B. "Effect of Cigarette Smoking on the Forced Expiratory Flow Rate," Amer. Rev. Resp. Dis., 85: 534 (1962).

121. Watanahe, S., Mitchell, M. and Rezetti, A. D., Jr. "Correlation of Structure and Function in Chronic Pulmonary Emphysema," Amer. Rev. Resp. Dis., 92: 221 (1963).

122. Stead, W. W., Fry, D. L. and Ebert, R. V. "The Elastic Properties of the Lung in Normal Men and in Patients with Chronic Pulmonary Emphysema," Jour. Lab. and Clin. Med., 40: 674 (1952).

123. Cole, M. B. and Robert, F. E. "A Clinical Pathologic Study of Emphysema: The Importance of Early Diagnosis," Jour. Amer. Geriat. Soc., 12: 415 (1964).

124. Fry, D. L., Ebert, R. V., Stead, W. W. and Brown, C. C. "The Mechanics of Pulmonary Ventilation in Normal Subjects with Emphysema," Amer. J. Med., 26: 80 (1954).

125. McLean, K. H. "The Pathogenesis of Pulmonary Emphysema," Amer. Med., 25: 62 (1958).

126. Azury, A., Anderson, A. E. and Foraker, A. G. "The Morphological Spectrum of Aging and Emphysematous Lung," Ann. Inter. Med., 57: 1 (1962).

127. Bates, D. V. "Impairment of Respiratory Functions in Bronchial Asthma," Clin. Sci., 11: 203 (1952).

128. Levine, G., Housley, E., MacLeod, P. "Gas Exchange Abnormalities in Mild Bronchitis and Asymptomatic Asthma," New Engl. J. Med. 282: 1277-1282 (1970).

129. Gaensler, K. A. "Ventilatory Tests in Bronchial Asthma: Evaluation of Vital Capacity and Maximum Breathing Capacity," J. Allergy, 21: 232 (1950).

130. Sharp, J. T., Griffith, G. T. and Green, D. G. "Ventilatory Mechanics in Pulmonary Oedema in Man," J. Clin. Invest., 37: 111 (1958).

131. Hardy, G. C. and Barach, A. L. "Positive Pressure Respiration in Treatment of Irritant Pulmonary Edema Due to Chlorine Gas Poisoning," J.A.M.A., 128: 359 (1945).

132. Kennedy, M. C. S. "Practical Measure of the Maximum Ventilatory Capacity in Health and Disease," Thorax, 8: 73 (1953).

133. Anderson, A. E., Jr. "Forsaker, A. G. "Morphological Aspects of Interstitial Pulmonary Fibrosis," Arch. Path., 70: 79 (1960).

134. Pierce, J. A. and Ebert, R. W. "The Elastic Property of the Lung in the Aged," J. Lab. Clin. Med., 51: 63 (1958).

135. Cournand, A. and Richards, D. W., Jr. "Pulmonary Insufficiency: Discussion of a Physiological Classification and Presentation of Clinical Tests," Amer. Rev. Tuberc., 44: 26 (1941).

136. Kaltreider, N. L., Fray, W. W. and Hyde, H. V. "The Effect of Age on the Total Pulmonary Capacity and its Subdivisions," Amer. Rev. Tuberc., 37: 662-668 (1938).

137. Whitfield, A. G. W., Waterhouse, J. A. H. and Arnott, W. M. "The Total Lung Volume and Its Subdivisions: A Study in Physiological Norm," Brit. J. Prev. and Social Med., 4: 113 (1950).

138. Needham, C. D., Rogan, M. C. and McDonald, I. "Normal Standards for Lung Volumes, Intrapulmonary Gas-Mixing, and Maximum Breathing Capacity," Thorax, 9: 313 (1954).

139. Yamada, N. and Tatar, K. "Vital Capacity, and Maximum Breathing Rate in Health Male Adults," Jap. J. Physiol., 4: 246 (1954).

140. Kelly, H. G. A Study of Individual Differences in Breathing Capacity in Relation to Some Physical Characteristics, University of Iowa Studies, Studies in Child Welfare, VII, (1933).

141. Bateman, J. B. "Studies of Lung Capacities and Intrapulmonary Mixing: Normal Lung Capacities," J. Appl. Physiol., 3: 133 (1950).

142. Moore, M., Schultz, F. W. and Cassels, D. E. "The Lung Volume and Its Subdivisions in Normal Boy 10 to 17 Years of Age," J. Clin. Invest., 31: 380 (1952).

143. Helliesen, P. J., Cook, C. D., Friedlander, L. and Agathon, S. "Studies of Respiratory Physiology in Children," Pediatrics, 22: 80 (1958).

144. Engstrom, J., Karberg, P. and Kraepelier, S. "Respiratory Studies in Children," Acta. Paediat., 45: 277 (1956).

145. Hepper, N. G. G., Fowler, W. S. and Helmholz, H. F., Jr. "Relationship of Height to Lung Volume in Health Men," Dis. Chest, 37: 314 (1960).

146. Shepherd, R., Thomason, J., Carey, G. C. R. and Phair, J. J. "Field Testing of Pulmonary Dynamics," J. Appl. Physiol., 13: 189 (1958).

147. Bower, G. "Respiratory Symptoms and Ventilatory Function in 172 Adults Employed in a Bank," Amer. Rev. Resp. Dis., 83: 684 (1961).

148. Flick, A. L. and Patton, R. R., "Obstructive Emphysema in Cigarette Smokers," AMA Arch. Intern. Med., 104: 518 (1959).

149. Simonsson, B. "Effect of Cigarette Smoking on the Forced Expiratory Flow Rate," Amer. Rev. Resp. Dis., 85: 534 (1962).

150. Matorazzo, J. D. and Saslow, G. "Psychological and Related Characteristics of Smokers and Nonsmokers," Psychol. Bulletin, 57: 493 (1960).

151. Thomas, C. B. and Cohen, B. H. "Comparison of Smokers and Nonsmokers," Bull. John Hospkins Hosp., 106: 205 (1960).

152. Snyder, L. H. "Inherited Taste Deficiency," Science, 74: 151 (1931).

153. Read, J. and Selby, T. "Tobacco Smoking and Ventilatory Function of the Lungs," Brit. Med. J., 2: 1104 (1961).

154. Nadel, J. A. "Mechanisms of Airway Response to Inhaled Substances," Arch. Environ. Health, 16: 171 (1968).

155. Brinkman, G. L. and Coates, E. O. "The Effect of Bronchitis, Smoking, and Occupation on Ventilation," Amer. Rev. Dis., 87: 684-693 (1963).

156. Morris, J. K., Koski, A. and Johnson, L. C. "Spirometric Standards for Healthy Nonsmoking Adults," Amer. Rev. Resp. Dis., 103: 57-67 (1971).

157. Hensler, N. M. and Giron, J. D. "Pulmonary Physiological Measurements in Smokers and Nonsmokers," J.A.M.A., 186: 885 (1963).

158. Larson, R. K. "The Chronic Effect of Cigarette Smoking on Pulmonary Ventilation," Amer. Rev. Resp. Dis., 88: 630 (1963).

159. Read, J. and Selby, T. "Tobacco Smoking and Ventilatory Function of the Lungs," Brit. Med. J., 2: 1104 (1961).

160. Cullumbine, H. "The Toxicity of Screening Smokers," J. Royal Army Med. Corp., 103: 119 (1957).

161. Crofton, E. and Crofton, J. "Influence of Smoking on Mortality from Various Diseases in Scotland, England and Wales," Brit. Med. J., 2: (1961).

162. Petty, T. L., Rayan, S. E. and Mitchell, R. S. "Cigarette Smoking and the Lungs," Arch. Environ. Health, 14: 172 (1967).

163. Higgins, I. T. T. "Tobacco Smoking Respiratory Symptoms and Ventilatory Capacity," Brit. Med. Jour., 1: 325 (1959).

APPENDIX

TABLE 2

HEIGHT COMPARISON OF FIREFIGHTERS VERSUS CONTROLS

Age (Years)		Test Subjects Height (inches)		Control Subjects Height (inches)
	$\bar{X}$	71.6829		69.3666
	S	2.2521		3.1784
20 - 24			N = 69 t = 3.59635*	
	$\bar{X}$	70.8861		70.2558
	S	2.1355		2.3713
25 - 29			N = 164 t = 1.61853	
	$\bar{X}$	70.6484		70.7777
	S	1.8339		3.0400
30 - 34			N = 181 t = 0.26322	
	$\bar{X}$	71.5100		70.1481
	S	2.0721		2.8107
35 - 39			N = 123 t = 2.7869*	
	$\bar{X}$	70.9062		71.4444
	S	2.2797		1.9436
40 - 44			N = 71 t = 0.67357	
	$\bar{X}$	70.8947		70.5625
	S	2.2991		3.0761
45 - 49			N = 52 t = 0.43757	
	$\bar{X}$	70.5882		69.5555
	S	2.2377		1.7400
50 or above			N = 24 t = 1.20144	

*Significant at the 0.05 level

TABLE 3

WEIGHT COMPARISON OF FIREFIGHTERS VERSUS CONTROLS

Age (Years)		Test Subjects Weight (lb.)		Control Subjects Weight (lb.)
	$\bar{X}$	181.0256		168.3666
	S	35.3676		30.8438
20 - 24			N = 67	
			t = 1.5567	
	$\bar{X}$	180.8548		178.4883
	S	23.2706		29.2829
25 - 29			N = 165	
			t = 0.53617	
	$\bar{X}$	181.4329		177.0555
	S	21.5623		19.7379
30 - 34			N = 180	
			t = 0.82392	
	$\bar{X}$	190.1145		172.1481
	S	22.8458		22.2307
35 - 39			N = 121	
			t = 3.63088*	
	$\bar{X}$	190.6825		179.0000
	S	27.3907		20.1370
40 - 44			N = 70	
			t = 1.22962	
	$\bar{X}$	191.6842		173.5625
	S	24.9508		25.3954
45 - 49			N = 52	
			t = 2.42453*	
	$\bar{X}$	185.5294		175.2500
	S	22.0003		18.5221
50 or above			N = 23	
			t = 1.14153	

*Significant at the 0.05 level

TABLE 4

COMPARISON OF VITAL CAPACITY (VC) OF FIREFIGHTERS VERSUS CONTROLS

Age (Years)		Test Subjects VC (liter)		Control Subjects VC (liter)
20 - 24	$\bar{X}$	6.0881		5.5600
	S	0.7787		0.7876
			N = 70	
			t = 2.82341*	
25 - 29	$\bar{X}$	5.7712		5.5790
	S	0.8198		0.7166
			N = 166	
			t = 1.36703	
30 - 34	$\bar{X}$	5.7194		5.6833
	S	0.7312		0.6981
			N = 181	
			t = 0.19949	
35 - 39	$\bar{X}$	5.5909		5.4963
	S	0.7892		0.8929
			N = 124	
			t = 0.53662	
40 - 44	$\bar{X}$	5.3687		5.3777
	S	0.6364		0.5932
			N = 71	
			t = 0.04014	
45 - 49	$\bar{X}$	4.9605		5.0750
	S	0.9066		0.6952
			N = 52	
			t = 0.45135	
50 or above	$\bar{X}$	4.6353		4.6555
	S	0.9089		0.5547
			N = 24	
			t = 0.0608	

*Significant at the 0.05 level

TABLE 5

COMPARISON OF FORCED VITAL CAPACITY (FVC) OF FIREFIGHTERS VERSUS CONTROLS

Age (Years)		Test Subjects FVC (liter)		Control Subjects FVC (liter)
20 - 24	$\bar{X}$	6.2785		5.6500
	S	0.0745		0.7412
			N = 70	
			t = 3.53618*	
25 - 29	$\bar{X}$	5.9040		5.7558
	S	0.7970		0.7970
			N = 166	
			t = 1.09081	
30 - 34	$\bar{X}$	5.8145		5.8500
	S	0.7416		0.8445
			N = 181	
			t = 0.18996	
35 - 39	$\bar{X}$	5.7316		5.5777
	S	0.78555		0.8125
			N = 123	
			t = 0.89455	
40 - 44	$\bar{X}$	5.5172		5.4111
	S	0.6219		0.5487
			N = 71	
			t = 0.48516	
45 - 49	$\bar{X}$	5.0210		5.2250
	S	0.9326		0.7398
			N = 16	
			t = 0.77646	
50 or above	$\bar{X}$	4.7411		4.6222
	S	0.9048		0.4763
			N = 24	
			t = 0.36602	

*Significant at the 0.05 level

TABLE 6

COMPARISON OF 1-SECOND FORCED EXPIRATORY VOLUME (FEV_1) OF FIREFIGHTERS VERSUS CONTROLS

Age (Years)		Test Subjects FEV_1 sec (liter)		Control Subjects FEV_1 sec (liter)
20 - 24	$\bar{X}$	5.2214		4.7966
	S	0.5667		0.5416
			N = 70	
			t = 3.19286*	
25 - 29	$\bar{X}$	4.9688		4.9790
	S	0.6789		0.6527
			N = 166	
			t = 0.08622	
30 - 34	$\bar{X}$	4.7632		4.8611
	S	0.7465		0.6518
			N = 182	
			t = 0.53417	
35 - 39	$\bar{X}$	4.6878		4.5851
	S	0.6997		0.6735
			N = 124	
			t = 0.68122	
40 - 44	$\bar{X}$	4.3875		4.4000
	S	0.5774		0.5000
			N = 71	
			t = 0.06167	
45 - 49	$\bar{X}$	3.8176		3.5333
	S	0.7535		0.4663
			N = 52	
			t = 1.20813	
50 or above	$\bar{X}$	3.8176		3.5333
	S	0.7535		0.4663
			N = 24	
			t = 1.02696	

*Significant at the 0.05 level

TABLE 7

REGRESSION ANALYSIS FOR YEARS IN FIRE SERVICE VERSUS 1-SECOND FORCED EXPIRATORY VOLUME ($FEV_{1.0\ sec.}$)

	Δ FMF in liter/yr. (Slope)	FMF in liter/yr. (Intercept)	Correlation Coefficient (r)
Firefighters	- 0.044	5.16	- 0.75
Controls	- 0.038	4.79	- 0.89

$\bar{X}$ = 13.0 (Years in Fire Service)

$\bar{X}$ = 4.59 liters (Firefighters)

$\bar{X}$ = 4.47 liters (Controls)

$\bar{X}$ = Mean Value

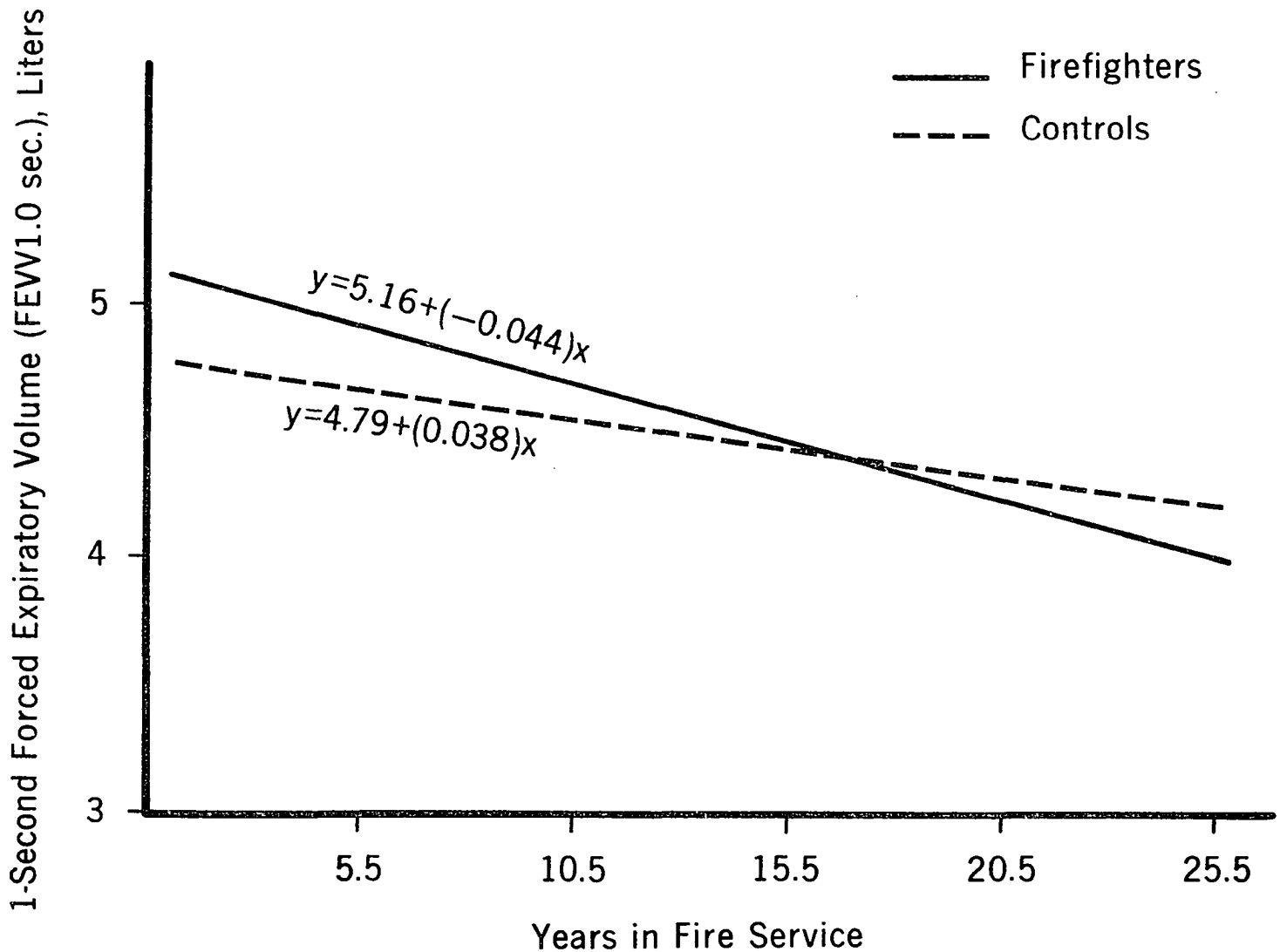

Figure 2. 1-second forced expiratory volume versus years in fire service

TABLE 8

COMPARISON OF 1-SECOND FORCED EXPIRATORY RATIO (FEV/FVC) OF FIREFIGHTERS VERSUS CONTROLS

Age (Years)		Test Subjects FEV_1 sec (Percentage)		Control Subjects FEV_1 sec (percentage)
20 - 24	$\bar{X}$	83.5268		84.6800
	S	5.7749		5.7104
			N = 69	
			t = 0.83503	
25 - 29	$\bar{X}$	83.5285		85.2954
	S	6.0617		5.3367
			N = 168	
			t = 1.71463	
30 - 34	$\bar{X}$	82.2993		83.3888
	S	5.8168		4.9751
			N = 181	
			t = 0.76424	
35 - 39	$\bar{X}$	81.4414		81.3481
	S	6.5713		5.5273
			N = 124	
			t = 0.06747	
40 - 44	$\bar{X}$	79.5921		80.8888
	S	6.2005		4.4072
			N = 71	
			t = 0.60452	
45 - 49	$\bar{X}$	78.3157		82.6875
	S	7.3953		6.1972
			N = 52	
			t = 2.07467*	
50 or above	$\bar{X}$	78.2764		77.0555
	S	5.7178		7.3195
			N = 24	
			t = 0.47032	

*Significant at the 0.05 level

TABLE 9

COMPARISON OF 0.5-SECOND FORCED EXPIRATORY VOLUME ($FEV_{0.5}$) OF FIREFIGHTERS VERSUS CONTROLS

Age (Years)		Test Subjects $FEV_{0.5}$ sec (liter)		Control Subjects $FEV_{0.5}$ sec (liter)
20 - 24	$\bar{X}$	3.9476		3.7903
	S	0.4964		0.3918
			N = 71	
			t = 1.45934	
25 - 29	$\bar{X}$	3.8472		3.9439
	S	0.5345		0.6128
			N = 164	
			t = 0.96874	
30 - 34	$\bar{X}$	3.7349		3.8388
	S	0.6042		0.5574
			N = 179	
			t = 0.69736	
35 - 39	$\bar{X}$	3.6979		3.6925
	S	0.6558		0.5683
			N = 123	
			t = 0.03868	
40 - 44	$\bar{X}$	3.4500		3.6000
	S	0.5342		0.3807
			N = 71	
			t = 0.81152	
45 - 49	$\bar{X}$	3.1000		3.4375
	S	0.7938		0.5678
			N = 52	
			t = 1.53898	
50 or above	$\bar{X}$	3.1058		2.7333
	S	3.6609		0.4716
			N = 24	
			t = 1.49507	

TABLE 10

COMPARISON OF 0.5-SECOND FORCED EXPIRATORY RATIO (FEV/FVC) OF FIREFIGHTERS VERSUS CONTROLS

Age (Years)		Test Subjects FEV0.5 sec (Percentage)		Control Subjects FEV0.5 sec (Percentage)
	$\bar{X}$	64.9619		66.9866
	S	9.6255		6.7488
20 - 24			N = 70	
			t = 0.99043	
	$\bar{X}$	65.9144		68.3595
	S	7.6079		7.7714
25 - 29			N = 166	
			t = 1.79234	
	$\bar{X}$	64.5481		66.9764
	S	8.7938		8.7863
30 - 34			N = 179	
			t = 1.08383	
	$\bar{X}$	64.6867		66.1703
	S	8.7937		7.7643
35 - 39			N = 123	
			t = 0.79497	
	$\bar{X}$	62.9093		65.6666
	S	8.2688		5.6929
40 - 44			N = 71	
			t = 0.96573	
	$\bar{X}$	61.2315		65.2900
	S	9.4991		7.2733
45 - 49			N = 46	
			t = 1.2539	
	$\bar{X}$	66.7941		59.8000
	S	9.3157		8.2974
50 or above			N = 24	
			t = 1.88744	

TABLE 11

REGRESSION ANALYSIS FOR YEARS IN FIRE SERVICE VERSUS FORCED MID-EXPIRATORY FLOW (FMF)

	Δ FMF in liter/yr. (Slope)	FMF in liter/yr. (Intercept)	Correlation Coefficient (r)
Firefighters	- 0.060	5.04	- 0.74
Controls	- 0.050	4.72	- 0.87

$\bar{X}$ = 13.0 (Years in Fire Service)

$\bar{X}$ = 4.26 liters (Firefighters)

$\bar{X}$ = 4.27 liters (Controls)

$\bar{X}$ = Mean Value

TABLE 12

REGRESSION ANALYSIS FOR AGE IN FIREFIGHTERS AND CONTROLS VERSUS FORCED MID-EXPIRATORY FLOW (FMF)

	Δ FMF in liter/yr. (Slope)	FMF in liter/yr. (Intercept)	Correlation Coefficient (r)
Firefighters	- 0.57	6.238	- 0.77
Controls	- 0.059	5.842	- 0.68

$\bar{X}$ = 4.45 liters (Firefighters)

$\bar{X}$ = 4.50 liters (Controls)

$\bar{X}$ = 39 Age in years (Firefighters)

$\bar{X}$ = 38 Age in years (Controls)

$\bar{X}$ = Mean Value

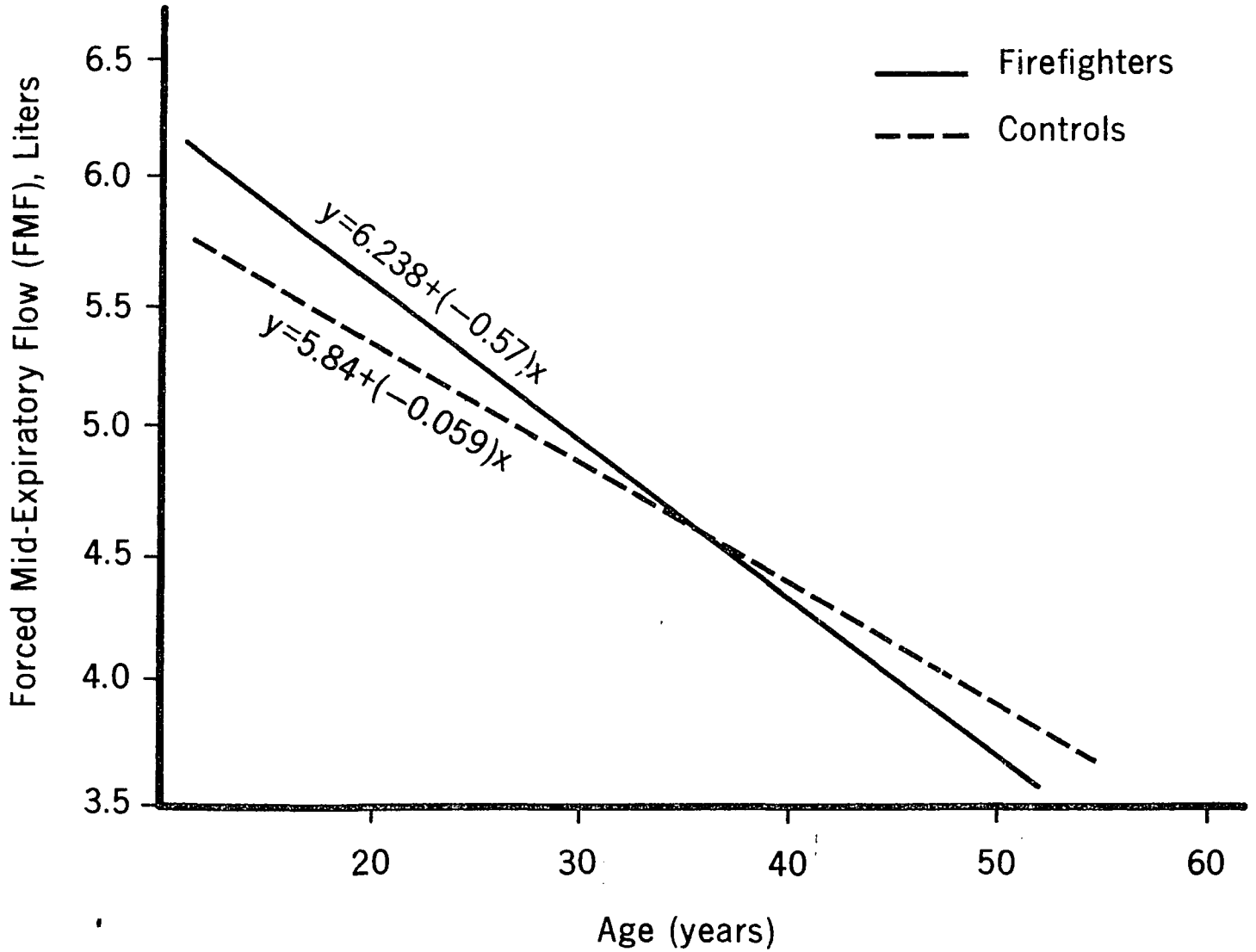

Figure 3. Forced mid-expiratory flow versus age.

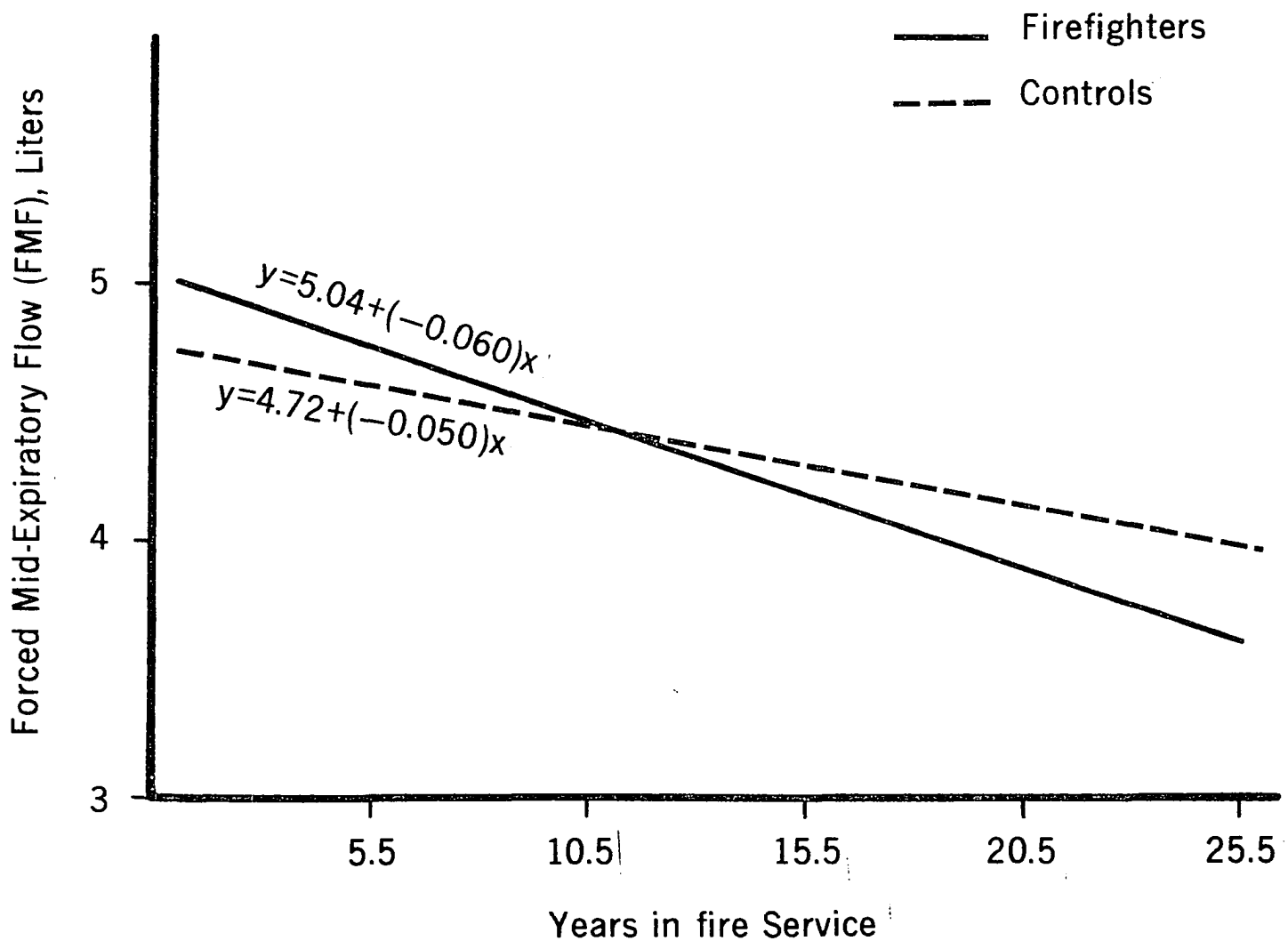

Figure 4. Forced mid-expiratory flow versus years in fire service.

TABLE 13

COMPARISON OF FORCED MID-EXPIRATORY FLOW (FMF) OF FIREFIGHTERS VERSUS CONTROLS

Age (Years)		Test Subjects FMF (l/sec.)		Control Subjects FMF (l/sec.)
	$\bar{X}$	4.9459		4.3970
	S	1.1257		0.9948
20 - 24			N = 70 t = 2.13925*	
	$\bar{X}$	4.8268		4.9474
	S	1.1288		1.1355
25 - 29			N = 166 t = 0.60317	
	$\bar{X}$	4.2253		4.6433
	S	1.1133		0.9571
30 - 34			N = 180 t = 1.53116	
	$\bar{X}$	4.3388		4.2629
	S	1.2094		1.4322
35 - 39			N = 123 t = 0.27725	
	$\bar{X}$	3.8589		3.8333
	S	1.2945		1.0427
40 - 44			N = 71 t = 0.05663	
	$\bar{X}$	3.2876		4.1625
	S	1.1478		1.3819
45 - 49			N = 52 t = 2.4062*	
	$\bar{X}$	3.1706		2.7266
	S	1.0892		1.0944
50 or above			N = 23 t = 0.97659	

*Significant at the 0.05 level

TABLE 14

COMPARISON OF 1-SECOND FORCED EXPIRATORY VOLUME (FEV_1) AND FORCED MID-EXPIRATORY FLOW (FMF) FOR FIREFIGHTER NONSMOKERS VERSUS SMOKERS

Measurement		Nonsmoker (liter)		Smoker (liter)
FEV_1	$\bar{X}$	4.9084		4.6632
	S	0.7329		0.7557
			N = 447 t = 3.01886*	
FMF	$\bar{X}$	4.9489		4.2383
	S	1.2770		1.2326
			N = 447 t = 5.2936*	

*Significant at the 0.05 level

TABLE 15

COMPARISON OF 1-SECOND FORCED EXPIRATORY VOLUME (FEV_1) AND FORCED MID-EXPIRATORY FLOW (FMF) FOR CONTROL NONSMOKERS VERSUS SMOKERS

Measurement		Nonsmokers (liter)		Smokers (liter)
FEV_1	$\bar{X}$	4.7296		4.6195
	S	0.6756		0.7414
			N = 148 t = 0.90177	
FMF	$\bar{X}$	4.8037		4.1792
	S	1.3315		1.1789
			N = 148 t = 2.98464*	

*Significant at the 0.05 level

TABLE 16

PREDICTION TABLE FOR FORCED MID-EXPIRATORY FLOW (FMF)

Subjects	Age (years)	Average Value		Normal Range	
		L./sec.	L./min.	L./sec.	L./min.
Males	20 - 29	5.4 + 1.3	324	6.7 - 4.1	402 - 246
	30 - 39	4.6 + 1.2	276	5.8 - 3.4	348 - 204
	40 - 49	4.3 + 1.3	258	5.6 - 3.0	336 - 180
	50 - 59	3.6 + 1.1	216	4.7 - 2.5	282 - 150
	60 - 69	3.0 + 1.0	180	4.0 - 2.0	240 - 120

TABLE 17

DISTRIBUTION OF FIREFIGHTER SUBJECTS IN 5-YEAR AGE GROUPS COMPARING SMOKERS VERSUS NONSMOKERS

Age (Years)	Smokers	Nonsmokers	Total
20 - 24	33	09	42
25 - 29	98	27	125
30 - 34	133	30	163
35 - 39	75	23	98
40 - 44	54	10	64
45 - 49	33	05	38
50 - 54	12	00	12
55 or above	02	03	05
Totals	440	109	549

RESPIRATORY QUESTIONNAIRE

(1) Name_____ (2) Date_____
(Last) (First) (3) Sex_____ (4) Age_____
(5) Height_____ (6) Weight_____

(7) Race Codes: White____ Black___ Am. Indian_____ Other_____

(8) Marital Status: Single___ Married___ Sep.___ Div.___ Widowed_____

(9) Fire Company Assigned_____ Prev. Company Assigned_____

(10) Date of Entry to Fire Service_____

(11) Length of Time in Fire Service_____

(12) Length of Time in Oklahoma City Fire Service_____

Check one answer after each question.

(13) Do you have a cough at the present time? . . . . . yes__ no__

(14) If yes,
(a) has it been for days?___ weeks?____ months?_____ years?_____
(b) do you bring up any mucus or phlegm from your chest? . yes__ no__

(15) Within the past six months
(a) have you had any pain or heaviness in your chest?. . . yes__ no__
(b) have you had any wheezing or whistling in your chest?. yes__ no__
(c) have you coughed up any blood or blood-streaked matter? yes__ no__

(16) Do you get short of breath
(a) when climbing a flight of stairs? . . . . . yes__ no__
(b) when walking rapidly for half a block? . . . . . yes__ no__
(c) when getting dressed? . . . . . yes__ no__

(17) Have you ever had
(a) influenza? . . .yes___ no___
(b) pneumonia? . . .yes___ no___
(c) pleurisy? . . .yes___ no___
(d) asthma? . . . .yes___ no___
(e) tuberculosis?. . . yes__ no___
(f) heart disease? . . yes__ no___
(g) chest surgery? . . yes__ no___
(h) emphysema? . . . . yes__ no___
(i) chronic bronchitis? yes__ no___

(18) How many years have you smoked?_____ years never_____

(19) What have you usually smoked? (Omit if "never" is checked above)
(a) cigarettes only_____
(b) cigarettes along with pipe or cigars_____
(c) pipe or cigars but no cigarettes_____

(20) If you usually smoked cigarettes (a or b above),
(a) how many packs a day? . . . .under 1_____ 1 to 2_____ over 2_____
(b) have you usually inhaled? . . . . . .yes_____ no_____
(c) have you smoked any cigarettes within the past hour? . .yes__ no___
(d) have you smoked any cigarettes in the past twelve months?. . . . . .yes__ no___

(21) Other pertinent information:_____

_____

_____

_____