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HOME OFFICE

A Review of Conceivable Health Hazards to Firemen in the Light of Current Knowledge

SCIENTIFIC RESEARCH & DEVELOPMENT BRANCH

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REPORT NO 6/77

FIRE RESEARCH

A REVIEW OF CONCEIVABLE HEALTH HAZARDS TO FIREMEN IN THE LIGHT OF CURRENT KNOWLEDGE

Summary

This report reviews the possible health hazards to firemen due to recently occurring threats such as toxic products from burning plastics and to factors present since the formation of the fire service reconsidered in the light of current knowledge. The latter include the effect of sub-toxic levels of carbon monoxide, carbon dioxide and heavy metals together with the possible deleterious consequence of asbestos, heavy exercise, heat stress, psychological stress and oxygen deficiency.

Recommendations for future research and possible amendments to the initial and subsequent medical examinations are suggested.

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1. INTRODUCTION

Research into the occupational health of firemen is at present a wide and unexplored field. Although the problem has been brought to the notice of a much wider range of concerned workers since the advent of the new types of plastic being used both industrially and in the home, many of the possible hazards due to fire fighting have been present since the inception of the fire service. The research required into the health hazards of firemen involves a large area of physiology, encompassing such specialities as respiratory -, cardiovascular -, olimatic -, and neuro-physiology and extends into disciplines as diverse as toxicology and epidemiology.

The acute effects of carbon monoxide, still the major gaseous threat from fires, are known, but a better understanding of the possible neurological and cardiovascular effects of sub-toxic levels of this fire product require further quantification. Another feasible cause of cardiovascular/kidney disease is the likely presence of heavy metals such as antimony and cadmium in (which are components of flame retardants and metallic plating etc respectively) smoke in low concentrations, until recently, considered non-toxic.

In the case of firemen handling asbestos products such as gloves and blankets, there is as yet no sound evidence of any danger to health. However, the potential risk from asbestos particles in a fire originating from the wide range of products using asbestos as a filler needs careful study together with the toxic products from the many types of plastic materials now on the market, in order to give the fireman protection and advice on any risks he may be taking.

Heat stress is a possible hazard by itself, but when it is compounded by the other factors to which a fireman is subjected, it is essential that some investigation is carried out in this area to ascertain the long term effects. The risk of eye deterioration due to heat or flame, similar to glass-blowers cataract, is not beyond the realm of possibility.

The psychological stress of being suddenly alerted for duty and always on 'active' service with the responsibility for human lives and property, in addition to working under arduous conditions, is an unknown factor worthy of further study, especially in view of the current theory that repetitive stress is a significant factor in the causation of heart disease.

Regular heavy exercise executed in a controlled manner, is now known to protect against heart disease. However the irregular manner in which it is carried out by firemen, without any warming-up period and in combination with the other stresses so far mentioned, could have serious adverse effects. More information is needed in order to allay fears and provide reliable guidance.

Some knowledge of the level of oxygen and carbon dioxide at fires is also required in order to protect against the known physiological effects of a deficiency in the former and excess of the latter.

One area in which it could be intuitively expected that the fireman has cause for particular concern is that involving the condition of his lungs. Research* is at present being carried out on approximately 1000 London firemen to discover whether or not there is any decrement in lung function due to their working environment and also to detect signs of heart and lung disease by means of the standard MRC respiratory questionnaire.

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* Pulmonary function study of London firemen by Dr D Douglas (London School of Hygiene and Tropical Medicine).

Although this report is concerned mainly with environmental hazards there is much that could be done in the category of what may be loosely described as 'preventive medicine'. For instance it is now considered that participation in the occasional ball game has no lasting effect, other than to improve the performance in that particular sport. Even the beneficial effect of strenuous exercise is lost after about three days if the training regimen is not maintained. Therefore the development of some form of systematic daily strenuous exercise schedule designed to tone up the cardiovascular system and prepare the heart for stress is of prime importance to enable the fireman to be suitably prepared to carry out his duties with the minimum risk of disease or injury.

Finally the possibility of using some form of testing as an aid in assessing the psychological factors influencing behaviour in dangerous team situations may deserve further consideration.

2. CARBON MONOXIDE

2.1 ACUTE EFFECTS OF CARBON MONOXIDE

The major gaseous threat from fires is still carbon monoxide;[79] as a general rule it can be assumed that any smoky fire involving organic material will have carbon monoxide present also.[21] Although plastics such as polyvinyl chloride (PVC), polyurethane and polystyrene also emit some hydrogen chloride, toluene di-isocyanate (TDI) and styrene respectively, these products normally reach their toxic levels after the toxic level of carbon monoxide has been attained.[80] Therefore, as far as the cause of death is concerned, carbon monoxide can still be considered the culprit at present. Inspiration of 10,000 ppm carbon monoxide can culminate in a loss of consciousness without the classical symptoms of headache, nausea and vomiting.[76] An exposure of approximately 2 hours to carbon monoxide concentrations between 200–1,200 ppm will result in a progression through the following stages:

35% blood carboxyhaemoglobin (COHb) – impairment of manual dexterity;

40% COHb – mental confusion;

67% COHb – death.

If recovery from a coma due to carbon monoxide poisoning takes place, but the tissue hypoxia has been too severe there may be central nervous system involvement such as impairment of memory, vision, hearing and speech.[76] Carbon monoxide can be lethal when as little as 1000 ppm is inspired[93] for 2 hours.

2.2 NEUROLOGICAL EFFECTS OF NON-LETHAL DOSES OF CARBON MONOXIDE

The neurological effects of non-lethal doses of carbon monoxide are well documented. Beard and Wertheim[11] demonstrated a deterioration in the ability to discriminate short intervals of time after 90 minutes exposure to 50 ppm of carbon monoxide and for proportionally shorter times after exposure to higher concentrations up to 250 ppm. Similar results were obtained by Rose and Rose.[64] However no such interference with the mechanism of time perception was found by Stewart et al[77] using carbon monoxide levels of up to 500 ppm for exposure of up to 5 hours duration. Vigilance was also impaired according to Horvath et al[42] after breathing 111 ppm of carbon monoxide sufficient to raise the blood COHb level to 6.6%. The heart rate and minute volume were not affected by this concentration of gas. These authors also showed the necessity for measuring the blood COHb levels during exposure in order to compare the carbon monoxide exposure with other variables.

Breathing concentrations of carbon monoxide as low as 10 ppm (2% COHb) is claimed to have an effect on the central nervous system according to Ross.[63] That the effect of carbon monoxide, in low concentrations, is to act on the

higher central nervous system centres in the brain that control some of the cognitive and psychomotor abilities was also maintained by Shulte.[70] He reported impairment of cognitive abilities at COHb levels of only 5%. It is worth pointing out that this level of COHb can be attained after breathing a concentration of carbon monoxide as low as 30 ppm for 8 hours.[63]

A study carried out on human fire victims in Chicago showed significant levels of COHb in each patient who, in retrospect, showed the signs and symptoms of carbon monoxide poisoning (ie headache, weakness, confusion and reckless behaviour).[93] The study suggests a surprisingly high incidence of COHb in house fire victims and firemen and points to the use of oxygen therapy at the site of fire when required. In addition physical exertion in the face of the decreased oxygen delivery capacity of the blood exacerbates the effect of carbon monoxide and increases its uptake. This is of particular concern to fire fighters. The degree of confusion and extent of the clinical symptoms were more prominent than originally appreciated by the victims or by the treating physician. These findings are of great significance to firemen for whom loss of judgement and confusion accompanying carbon monoxide inhalation may cause inappropriate acts unwittingly.

Howlett and Shephard[43] have studied the synergistic effect of carbon monoxide and hypoxia in aircrew and found it significant when carrying out demanding tasks. This combination of stresses is very likely in a fire situation. But even without the added stress of hypoxia they found impairment in psychomotor performance following a 2-3% augmentation of blood COHb. The maximum allowable atmospheric concentration of carbon monoxide was in fact reduced from 100 ppm to 50 ppm (for 8 hours) in 1964 because of new evidence of possible adverse effects mostly on the central nervous system from exposure in the range 50-100 ppm.[38]

2.3 CARDIOVASCULAR EFFECTS OF CHRONIC CARBON MONOXIDE EXPOSURE

a. ATHEROMA FORMATION

There is a considerable body of opinion nowadays, although not unanimous, pointing an accusing finger at carbon monoxide as being an atherogenic compound when the exposure is either for long exposures of low dosage or possibly short exposures of high dosage.[39] Most of the laboratory work carried out to investigate the relationship between atheroma formation and carbon monoxide has of necessity involved small mammals, mainly rabbits. Rabbits are a good model for this type of study as they form atheroma readily and with many types of atherogenic compounds.

In 1972 Kjeldsen et al[45] demonstrated ultrastructure and intimal changes in the rabbit aorta, using an electron microscope, after moderate carbon monoxide exposure (180 ppm for 2 weeks). Apart from actual plaque formation the surface structures were also markedly swollen and irregular compared to the control animals. The results supported earlier findings[9] of the toxic effect of low concentrations of COHb on the arterial walls provoking increased endothelial permeability and formation of oedema leading to changes indistinguishable from early atherosclerosis.

Wanstrup et al[86] also found focal degenerative changes, related to atherosclerosis, in the aorta of rabbits exposed to 90 ppm carbon monoxide for 3 months (approximately 11% COHb). Their findings confirmed that tissue hypoxia via carbon monoxide exposure seems to play a basic role in the development of focal vascular changes of the atherosclerotic type. It is suggested that increased endothelial permeability caused by the hypoxic state represents a basic phenomenon leading to focal oedema and subsequent regenerative and reparative vascular changes eventually leading to plaque formation.

Kjeldsen et al and Garbash et al[34,47] also found similar gross arteriosclerotic changes after exposing rabbits to short daily periods of systemic hypoxia for two weeks (without any carbon monoxide). The alterations in the rabbit aortae had features in common with human arteriosolerosis. Similar changes at the biochemical level were also demonstrated by Garbash et al.[35] Microscopic changes in the hearts of rabbits exposed to carbon monoxide levels of less than 100 ppm and 180 ppm were also demonstrated by Thomsen and Kjeldsen[82] and Kjeldsen et al[46] respectively.

Thomsen[81] extended the above work into the primate kingdom using juvenile macaca monkeys. After exposure to 250 ppm of carbon monoxide for 2 weeks the monkeys developed changes in their coronary arteries consistent with developing atherosclerotic disease. Although the above type of experiment cannot be carried out using human subjects there is some evidence that people with a blood COHb level of 5% or more, due to tobacco smoking, are more likely to be affected by certain atherosclerotic diseases, including ischaemic heat disease[83,85]. Siggaard-Andersen et al[74] also demonstrated a significant increase in capillary filtration after inhalation of carbon monoxide by normal human subjects. This phenomenon has been implicated as a precursor in the mechanism of atheroma formation by some workers.[7]

b. INDUCTION OF ANGINA

There is now much evidence to show that a low level exposure to carbon monoxide (50-100 ppm) giving COHb levels up to 4.5% can be instrumental in shortening the duration of exercise possible before the onset of pain and also in prolonging the duration of the pain in angina patients.[2,3] Aronow et al[5] also claims to have found a similar decrease in exercise performance before angina develops, after the carbon monoxide exposures obtained during freeway travel in heavy traffic. Lescoe[50] however, claims that Aronow's samples were too small and measurements too subjective. Goldsmith[37] even goes so far as to suggest that there may be no threshold for the effect of carbon monoxide exposure on patients with angina pectoris.

c. DECREASE IN EXERCISE TIME IN NORMAL SUBJECTS

Even in normal healthy subjects the effect of breathing 100 ppm carbon monoxide for 1 hour, on maximal exercise, was found to be a significant decrease in the mean time of exercise until exhaustion.[4] Differences have also been found, in cardiopulmonary responses to exercise, between young male cigarette smokers and non-smokers due to the chronically elevated COHb levels (> 4%) in smokers.[20] Levels of COHb between 5-10% have also been shown to enhance the development of arrhythmia (any variation from the normal rhythm of the heart beat) during exercise in subjects older than 40 years of age.[8]

d. LOWERING OF VENTRICULAR FIBRILLATION THRESHOLD WHEN BREATHING CARBON MONOXIDE

Debias et al[23] demonstrated in monkeys that ventricular fibrillation (rapid unco-ordinated contractions of the heart ventricles) is more easily induced in those animals exposed to 100 ppm of carbon monoxide. Bellet et al[12] found a similar reaction in dogs although at the time they suggested that it might be due to nicotine in the cigarette smoke used.

e. INCREASE IN HEART WEIGHT FOLLOWING CARBON MONOXIDE EXPOSURE

Exposure of rats to carbon monoxide levels of up to 500 ppm has also been shown to cause an increase in heart weight by at least three groups of workers.[58,59,60]

f. INCREASED CORONARY BLOOD FLOW DURING EXPOSURE TO CARBON MONOXIDE

The above group of studies (a-e) may or may not have an immediate effect on fire fighters depending upon whether or not they have any latent disease present. One physiological response is undoubtedly operating. This is the increase in coronary blood flow. The increase in coronary blood flow is the body's response to a decrease in oxygen content of the blood which is a concomitant of carbon monoxide uptake.[92] Thus the coronary reserve capacity of the individual would be reduced. Any further stress that would require an increase in coronary flow above the maximum attainable by the heart would cause ischaemia of the myocardium. This could lead to irreversible cardiac damage or transient damage that would reduce the overall functional ability of the heart to deliver blood to the rest of the body.[8,92] A normal heart can increase its output safely but a diseased heart may not be able to do so.[22] Even so, when an exposed fire fighter with a normal heart is in a situation that demands maximum exertion he will fatigue more easily. Any attempt at maximum exertion can place severe strain on the heart.[78]

Another significant fact is that carbon monoxide is more readily taken up by the body at higher temperatures.[39] Thus the environment of the fire fighter due to exercise and heat from the fire, presents ideal conditions for obtaining the maximum ill-effects from carbon monoxide.

On an examination of 36 men of the Oklahoma City fire department, some evidence for myocardial (heart) damage was obtained as indicated by an increased level of total lactic dehydrogenase (LDH), heat stable lactic dehydrogenase (LDHS) hydroxybutyric dehydrogenase (HBD) and creatine phosphokinase (CPK) in the blood.[65]

3. HEAVY METALS

3.1 DANGERS OF HEAVY METALS

Another conceivable hazard to fire fighters, although to date this has not been considered, is that due to heavy metals notably cadmium and antimony. When looking for a possible cause of the increased incidence of cardiovascular/renal disease found by Mastromatteo[52] these metals should be given some consideration.

It has been known for many years (over 100 years in the case of cadmium) that cadmium and antimony are toxic metals. In 1950 Friberg[33] called attention to the level of renal disease and emphysema in workers exposed to cadmium oxide dust over long periods in a battery plant. Recently, however, it has become apparent that they are capable of causing adverse effects at dose levels well below those normally regarded as poisonous. Such low doses, in the case of antimony, have been shown to possess unpredictable and sometimes fatal effects on both the heart and the liver.[18] In the case of very low daily doses of cadmium (5 ppm), which is a sub-toxic dose, a series of experiments carried out by Schroeder[68] showed that the pattern of induced hypertension was remarkedly similar to that prevalent among humans. Not only was the life span of the cadmium treated animals 20% shorter than the controls, but the high blood pressure was accompanied by such typical human symptoms as enlargement of the heart, excessive lipids and arteriosolerosis of the kidney.

3.2 ANTIMONY: SOURCE AND PHYSIOLOGICAL EFFECTS

Antimony is widely used as a flame retardant, in the form of antimony oxide, in paints and textiles, in the compounding of rubber and in ceramic glazes.

Thus in any fire situation it is feasible that a fire fighter could be exposed to some trace of antimony. Little is known about the long term effects of chronic antimony exposure or understanding its power to disrupt the function of cardiac muscle.[83] It is also suspected of causing lung cancer.

3.3 CADMIUM: SOURCE AND PHYSIOLOGICAL EFFECTS

Cadmium is present in galvanised materials, in plastic and copper piping, in almost every kind of wood finish and in some paints. It also lies beneath many chromium plated surfaces and is a common constituent of hard solders. In recent years cadmium has also been used as a heat stabiliser for PVC in the form of a cadmium/barium system. The increased degree of suspicion of it being a toxicological hazard can be judged by the development of new heat stabilisers now in progress.[28] As in the case of antimony, there is every reason to suggest that some cadmium could be present in the atmosphere surrounding a fire.

One unique property of cadmium is that it accumulates with enormous efficiency in the kidneys of mammals where it becomes bound as a metallo-protein and is released and excreted extremely slowly. Another property, which appears to be unique, and has been revealed experimentally in small animals, is that at minute doses (10–40 mg per 100g body weight) injection of cadmium into the blood stream produces an immediate increase of blood pressure.[61] Translated into human terms the author suggested that this might equal a daily ingestion of only a few hundred micrograms of cadmium. At higher levels of cadmium the opposite effect is observed. Other work by Schroeder et al[69] showed that the only sign in rats due to sub-toxic levels of cadmium (5 ppm) was that of hypertension.

Cadmium which was shown to accumulate in the kidneys of humans, with advancing age[69], was also found to be elevated withe respect to zinc more frequently in persons dying from hypertensive causes than in persons dying from other major causes.[67] It has also been considered as an antimetabolite for zinc, zinc being necessary for the formation of many enzymes – a factor only recently realised. Cadmium also possesses the ability to decouple the enzyme processes of the liver and kidneys so that stages in the natural de-toxifying processes cease to function.[83]

A recent study by Glauser et al[36] showed that a group of normal humans had a blood-cadmium level of 3.4 ±0.5 µg/ml compared with untreated hypertensive humans who had a much higher level of 11.1 ± 1.5 µg/ml. It was suggested that the Cd^{++} ion replaces another divalent ion in the enzyme that synthesizes pressor agents and that the enzyme is more active with cadmium than its normal divalent ion.

3.4 MODE OF ACTION OF CADMIUM AND ANTIMONY

Even supposing that cadmium or antimony were present at extremely low concentrations in a fire there is the possibility that they could become adsorbed onto the carbon particles present as smoke. Fires have become much more smoky in recent years due to the increased use of plastics in buildings and furnishings.[16] It is possible that the increased density and quantity of carbon particles (smoke) may be serving as a more efficient carrier for toxic substances such as antimony and cadmium which may have always been present in fires. In fact surface adsorption is known to greatly promote toxicity in many cases.[55,66] Thus toxic substances could be carried down into the lung adsorbed onto particles of carbon, which is a very efficient material for this process. The size of the smoke particle is another important parameter which is unknown and which could have important implications, as the smaller the particle the further into the lung tree it can travel. An increased rate of breathing due to exercise will also increase the percentage deposition of particles in the lung.[24]

In the majority of cases particles that are less than 1 micro metre (μm) in diameter are of primary importance as 50–80% of these reach the alveoli of the lungs.[55] A study of particles emitted from a coal fired power plant carried out by Natusch et al[56] showed that toxic trace elements such as antimony and cadmium were in fact most concentrated on the smallest particles. It is from these small particles that trace elements are most effectively extracted into the human blood stream. Larger particles are deposited in the nasal-pharyngeal and bronchial region of the respiratory system and are removed by cilial action to the stomach where the absorption efficiency is only 5–15% for most trace elements. Hence it is important to have some knowledge of the size of the smoke particles and the identity of any adsorbed material and to be able to relate this if possible with the blood cadmium (and antimony) levels of the fire fighter.

The GLC Scientific Advisory Branch has attempted an analysis of smoke present at 'real' fires but apparently the experimental problems were greater than anticipated when first considering the project[13] and the research has been left in abeyance.

4. ASBESTOS

4.1 GENERAL REMARKS

The recent publicity concerning asbestos and its capability of causing asbestosis, lung and bronchial cancer and mesothelioma even in people outside of the asbestos industry needs to be treated with some caution (except when applied to asbestos workers or their immediate families) until more information is known. In addition to possible contact with asbestos during their employment many people could have contact with the dust unwittingly during their leisure time (eg do it yourself enthusiasts).

In the case of the firemen who handle asbestos products in the course of their duty, there is as yet no evidence that this constitutes a danger to health. However one area of potential risk which so far has not been considered is the possible danger due to asbestos particles in a fire. These could originate from products containing asbestos as a reinforcing agent. For example, asbestos is used in such diverse items as asbestos roofing, insulating material, asbestos reinforced cement and as a filler in plastics and floor tiles.

Before any sound judgement can be made a knowledge of the asbestos content and particle size in smoke etc needs to be evaluated. One carcinogenic form of asbestos (crocidolite/blue asbestos) although no longer imported is still present in many buildings.[27] Another problem in detecting possibly harmful asbestos particles is that particles too small to be seen by an optical microscope have been shown to produce asbestosis in the guinea pig.[41]

4.2 LARYNGEAL CARCINOMA

A study designed to examine the relationship between exposure to asbestos and laryngeal cancer carried out by Shettigara and Morgan[71] suggests that exposure to asbestos and smoking are potent factors in the development of this disease. (Other cancers eg ovary are suspected as well).

5. TOXIC PRODUCTS FROM BURNING PLASTICS AND RUBBER

5.1 GENERAL REMARKS

The principal toxic gas in fires involving wood is carbon monoxide plus some hydrogen cyanide when animal fibres are present. Plastic products on the

other hand can produce a wider variety of toxic fumes which may cause lasting injury or death in those casualties who survive the initial exposure.[16] As a general rule when a large volume of smoke is present and organic materials are burning, carbon monoxide is almost certainly present also.[21] The main hazards at present are from burning PVC, polyurethane, polystrene and rubbers.

5.2 POLYURETHANE FOAMS

Polyurethane foams will emit some hydrogen cyanide when ignited but in lower quantities than most other materials, natural and man-made containing carbon, hydrogen and nitrogen.[32] Although hydrogen cyanide can be produced in amounts fully comparable in toxicity to carbon monoxide[38] the lethal dose is reached significantly later than that of carbon monoxide.[80] Another possible hazard from ignited polyurethane foams is the release of free toluene di-isocyanate (TDI). Some individuals may become sensitised to this compound in such a way that very low concentrations may subsequently cause a type of allergic reaction in the lungs.[26,54]

5.3 RIGID POLYVINYL CHLORIDE (PVC)

When rigid polyvinyl chloride is ignited it will evolve carbon monoxide, hydrogen chloride, benzene and some minor products such as toluene, napthalene, ethylene, ethane, propylene, propane, n-butane, n-pentane and n-hexane which make little or no contribution to the toxicity of the decomposition products. The main toxic risks from the decomposition products of PVC are considered to be carbon monoxide and hydrogen chloride.[88] Hydrogen chloride on combination with water forms hydrochloric acid. This compound can cause destructive damage to mucous membranes. If inhaled the upper respiratory tract could be severely damaged and this may lead to asphyxiation and death.[30] On the other hand smoke itself is an irritant. Exposure can lead to eye irritation plus irritation to the mucous membrane of the respiratory tract, nostrils and throat. Nine out of ten injuries to fire fighters are claimed to be due to smoke.[21]

5.4 POLYSTRENE

In addition to carbon monoxide some styrene can be evolved from burning polystyrene. This is considered safe at concentrations below 100 ppm. Above this level it can produce irritation of the mucous membrane and other toxic symptoms.[30]

5.5 RUBBERS

When rubbers are ignited they can evolve carbon monoxide, hydrogen sulphide and sulphur dioxide together with a heavy black smoke. Inhalation of the smoke can lead to a sick headache lasting several days.[21]

5.6 PLASTICS CONTAINING ADDED PHOSPHATE FIRE RETARDANTS

Some workers eg Petajan et al[62] consider the added phosphate fire retardants also to be a possible hazard when plastics burn. More work, however, needs to be carried out in this area.

5.7 TOLUENE DI-ISOCYANATE

Toluene di-isocyanate itself which is one of the reactants in the manufacture of polyurethane, and as such, could be met by a fireman at a fire or chemical incident is now thought to have neurological effects as well as respiratory.

A recent paper by Axford et al[10] describes neurological and long term damage to the respiratory tract of firemen fighting a fire (1967) in which storage tanks containing toluene di-isocyanate were damaged. The neurological symptoms, still present 9 years later in some cases, were such that there was an immediate feeling of well being, as if drunk, followed later by lack of concentration, loss of memory, odd behaviour, personality change and depression in some victims.

In future situations such as this it is imperative that the firemen are fully protected using breathing apparatus and protective clothing and that some indication of the chemicals involved or likely to be involved are given. If necessary a simple indication device such as the Draeger indicator tubes could be used. The difficulty here is that pre-knowledge of the possible hazards would be necessary in order to select the correct indicator tube. It is not possible at present to have a small portable apparatus capable of indicating either quantitatively or qualitatively every conveivable hazard.

It might be practicable nevertheless, for sampling devices to be developed for the most serious and probable toxic chemicals known at present and for each to be the responsibility of a different member of the team at major chemical incidents. If the Draeger tube system proved suitable in the fire environment this would only involve the fireman sampling the air using a small pair of bellows, which fit into one hand and to which are connected a small glass tube. These tubes are specific for one gas and indicate its presence by a distinct colour change and its concentration by the length of the colour changed region.

6. HEAT STRESS

6.1 GENERAL REMARKS

Although stresses encountered by the fireman during the course of his duties are a complicated combination of factors, heat stress must be considered a likely problem in many situations - eg heath-fires. The response to heat stress includes such symptoms as collapse, exhaustion, dehydration and increased heart rate etc. These will be dealt with separately below.

6.2 COLLAPSE

The most likely cause of collapse would be vasodilation due to the heat, causing a drop in blood pressure and decrease in the blood flow to the brain. This mechanism is similar to a normal faint therefore some seconds warning would be given. The site at which vasodilation occurs is not fully understood. It is not at the skeletal muscle or skin level; it is believed that the intestines are involved.[44] This response to heat stress could be particularly hazardous to the fire fighter especially if it occurred whilst in a burning building unobserved by his colleagues or whilst carrying out any delicate procedure involving other people. Carrying a victim who had fainted by this mechanism, using the fireman's carry, would probably help the blood flow by shaking and 'pumping' the muscles rather than having any adverse effect.

6.3 CARDIAC EFFECTS

There is no equivalent in hyperthermia to the ventricular fibrillation caused by hypothermia. Nevertheless anyone who had a heart defect which could lead to coronary insufficiency might succumb to cardiac arrest if exposed to higher body temperatures. This would be analogous to say swimming in cold water or weight lifting etc by an older person.[44]

6.4 PROTECTIVE CLOTHING

The wearing of thick protective clothing as at present may be the best compromise between lightweight clothing and completely fireproof material such as asbestos suits, because of its insulating properties against the heat of the fire. If lighter clothing was worn in order to keep cool when carrying out duties away from the fire ground the fireman would then become vulnerable when faced with the radiant heat from the fire.[44] Nevertheless, the ideal would be a well insulated, hardwearing and lightweight garment.

6.5 SUDDEN CHANGES OF TEMPERATURE

The possibility exists that sudden changes of temperature and unequal distribution of temperature might be hazardous, for example, arriving at a large conflagration on a cold day and standing with the extreme heat from the fire radiating on one half of the body and cold on the other half. However this possibility has already been considered in other contexts and does not constitute a significant hazard.[44]

6.6 BEHAVIOURAL PROBLEMS ASSOCIATED WITH HEAT STRESS

Experiments carried out on human subjects exposed to various heat stresses have highlighted the fact that it is very difficult for a person to judge for how long they could continue in a given situation. For example a person might think that they could continue in a hot environment for some time and then be forced to give in after a few minutes. On the other hand they could be on the verge of giving up and then realise that they can continue for a much longer period and in fact find it not so arduous as at first thought.[44] When considering heat stress it is necessary to consider both the physiological and psychological strain.[49]

6.7 HEAT EXHAUSTION

When suddenly exposed to a high temperature there is a dilation of the peripheral vessels greatly increasing the vascular space. The heart-rate and cardiac output increase and the blood pressure may fall a little. This is because in addition to carrying oxygen to the working muscles the blood has also to carry heat from the interior of the body to the skin. The outcome is an extra burden on the heart.[6] If in addition physical work is performed, the heart may not be able to maintain the blood pressure and the condition known as heat exhaustion ensues. The symptoms are indicative of cardiac insufficiency and collapse and unconsciousness could ensue.[14] Although heat exhaustion may occur at any time after exposure to heat it usually occurs when unusual physical demands are made upon an unacclimatised subject.

6.8 DEHYDRATION

In a hot environment evaporation of sweat is the main defence against over-heating but this poses a continual threat to homeostasis because of potential bodily dehydration and associated undesirable concentration of body fluids. Unlike heat exhaustion which could occur at any time after exposure to heat, dehydration exhaustion occurs after a longer exposure to heat if the fluid loss is not replaced. In a hot environment with extensive sweating the blood volume is reduced and this reduction in the blood volume associated with the larger vascular space again produces cardiac insufficiency and exhaustion to collapse.[14]

6.9 HEAT STROKE

This is a rare condition compared with heat exhaustion. It is characterised by a rising temperature with a dry skin. The victim seemingly well, may suddenly become unconscious with a rectal temperature as high as 43°C (110°F). This is apparently caused by a complete breakdown of the heat regulating mechanisms of the body. Unless the temperature is promptly reduced, there may be permanent damage to the nervous system. Heat stroke often terminates in death. The mechanism of heat stroke is obscure but its onset usually accompanies exertion which may be quite mild.[14] This form of temperature-regulation failure is rare, possibly occurring in only one in a million persons exposed to extreme heat.[6]

6.10 ANHIDROTIC HEAT EXHAUSTION

Another type of temperature regulation failure is that known as anhidrotic heat exhaustion. The victim may have a body temperature of 38–40°C and sweat very little or not at all. He will feel very tired and may be out of breath and develop tachycardia. The main trouble is reduced sweat production. When the victim stops working and is removed to a cool place his condition rapidly improves, but it may take him a long time to regain full tolerance to heat.[6]

6.11 RELATIONSHIP BETWEEN HYPERTENSION AND PROLONGED EXPOSURE TO HEAT

A preliminary investigation into the relationship between exposure to heat and blood pressure was carried out among workers at a metallurgical plant by Koetzl et al.[48] Hypertension was found to be significantly prevalent among the labour force employed in the rolling mill and furnace operators. The difference could not be explained through difference in age, composition of the group, race, or consumption of salt supplements. A very definite relationship was found between the levels of blood pressure and years of exposure to high environmental temperature.

6.12 AGE AND SIZE DEPENDENCY

There is evidence to show that increase in age will result in a greater physiological strain in men who are exposed to heat but the actual differences in rectal and skin temperatures in men of different ages are small.[49]

Large men are at a disadvantage in heat in comparison with a man of slighter build. This has been attributed to a greater ratio of body weight to surface area in the large man. Minard et al[53] found that in unacclimatised marine recruits, exposed to unexpected heat loads, heat stroke occurred mainly in obese individuals. All of the above factors could possibly have a bearing on the efficiency and health of fire fighters and should be considered when subjecting firemen to heat stress both in training and at actual fires.

7. PSYCHOLOGICAL STRESS

As well as the obvious physiological stresses encountered by firemen in the course of their work, there are also many varieties of psychological stress.

There is the stress of suddenly being awoken by the alarm bell and the accompanying pounding of the heart testified to by most firemen. The uncertainty of the fire situation to be encountered whilst journeying to an incident must create added stress.

It is also thought that certain body types are more susceptible to cardiovascular disease than others[75] and that those body types who seek an active/dangerous occupation are most susceptible. It is likely that this category of person is more attracted to the fire service which gives this type of stimulus. Hence, the increase in cardiovascular disease claimed by some to exist in firemen when compared to the general population could be due, at least in part, to self selection by the psychological/body types.

It could also be possible that worry and apprehension of the possible outcome, following a chemical or fire incident in which the actual extent of the possible damage to health was unknown, might be more harmful psychologically than physiologically. This could be especially relevant if the fireman had not been kept fully informed of the situation and was worrying unnecessarily. With the present increase in the use of plastics and new chemicals every household and industrial fire must present the firemen with some worry of the unknown concerning the hazards to which he might have been exposed and whether it could have any long term effects either toxic or carcinogenic.

Providing the firemen with better information on the facts of the situation would be of benefit in such cases.

Finally there is the psychological stress of always being on 'active' service and responsible for human lives and property together with working under arduous conditions. This is not the case for most other occupations.

These problems warrent further investigation in view of the current theory of stress being a factor in the causation of ischaemic heart disease. For example, it may be that a different form of alarm system would be advisable at night. There is a real need for research in this area especially as it is a subject about which many firemen express anxiety.

8. THE EFFECT OF HEAVY EXERCISE

8.1 GENERAL REMARKS

Controlled regular exercise is known to protect against ischaemic heart disease. When, however, the strenuous exercise is taken together with all of the stresses so far mentioned or without a warming up period, the effect on the cardiovascular system is really unknown.

8.2 TYPES OF EXERCISE

There are two fundamental types of exercise, isometric and isotonic. Basically isometric exercise is when there is no change in length of the muscle such as occurs when pushing against an immovable object and isotonic exercise occurs when there is a change in length such as in the leg muscles when riding a cycle.

The importance of this distinction is that in the first case (isometric) the blood pressure is raised, but not necessarily so in the case of isotonic exercise. This may have relevant implications in the case of fire fighters (especially the older man) when carrying out such activities as holding a hose against a large reaction. The mechanism of raising the blood pressure during isometric exercise is not understood. Even exercise of a small group of muscles appears to cause the same rise in pressure.[44]

8.3 EFFECT OF EXERCISE ON THE HEART

The effect of exercise on the heart is to increase the cardiac output. This is achieved by some increase in the rate of the heart and by an increase in

the volume of blood ejected per beat (stroke volume).[66] It is also possible for heart abnormalities to show up on the electrocardiogram after exercise which were not indicated during the resting state. In fact according to the American Heart Association about 60% of adults, with severe cardiovascular disease, have normal 'at rest' electrocardiograms. There is a growing concensus that the resting ECG is most informative when combined with an ECG taken during an exercise routine.[19]

The added stress placed on a fire fighter's heart when carrying out a strenuous task together with the added burden of a breathing apparatus and a hot environment is a totally unknown factor. To this can also be added the effects of carbon monoxide if breathing apparatus is not worn, which as stated in section 2 can have a synergistic effect, carbon monoxide also causes a dilation of the coronary arteries in order to supply the heart with more blood to compensate for the reduced amount of oxygen being carried. Intuitively this would seem to be a severe cumulative stress for a normal heart let alone that belonging to an older man or one with latent heart disease.

The possibility also exists for an older person to suffer a cardiac arrest during heavy exercise due to his coronary arteries being narrowed by atheroma deposits or atherosclerosis. The heart of such a person would cope quite satisfactorily under normal living conditions but would be unable to obtain the increase in coronary blood flow required at high exercise levels.

For the above reasons it is important that some research is carried out in this field and also for an initial medical examination, including ECG to be carried out after the fireman has undergone some form of exercise whilst dressed in his operational gear and preferably at typical operational temperatures. The Double Master Two Step Test has been found useful in allied work.[25] This would also give a suitable ECG base line with which to compare any future records, following illness or accidents for example. There is also scope for some research on suitable physical training methods for the Fire Service.

9. LACK OF OXYGEN

9.1 INTRODUCTION

Although much emphasis has been placed in the past on the effects of 'smoke and fumes' on a fireman's health and performance. One aspect of the fire environment which is not afforded the same consideration is that of the decrease in oxygen concentration at the site of a conflagration, due to the oxygen being consumed by the fire. The exact extent of this potential hazard requires analysis of the atmosphere surrounding 'real' fires. However it is as well to consider the implications if or when such a threat presents itself.

9.2 PHYSIOLOGICAL EFFECTS OF OXYGEN LACK

In many ways the effect on the body of lack of oxygen is similar to that caused by carbon monoxide, ie Garbasch et al[34] produced changes in the rabbit aorta which had features in common with human arteriosclerosis. Similar biochemical changes, in the aorta, to chronic carbon monoxide exposure were also demonstrated by Garbasch et al.[35] Anoxia (and asphyxia) also causes a profound increase (250–300%) in coronary artery blood flow which if accompanied by maximum effort could have a deleterious effect as described for carbon monoxide in Chapter 2. Cardiac output is also increased, in man, by as much as 50% whilst breathing a gas mixture containing 8% O_2[14] for example.

Another serious problem with anoxia is that the victim can collapse without any warning due to the low partial pressure of oxygen in the blood flowing through the brain. This is unlike the simple faint which is due to a decrease in blood flow to the brain and the victim is aware of the fact a few seconds before collapsing.

The intervening stage between normality and collapse is also hazardous in the case of anoxia. In this state the victim could think that his behaviour was quite normal and be quite unaware of the danger to himself and others through lack of self control etc and unawareness of a dangerous situation. His behaviour would be similar to that of a drunken man who is convinced that his driving is perfectly normal.

In the case of a firefighter the effect of the combination of oxygen lack, heat and carbon monoxide is unknown. This could be a cause of some of the deaths or accidents among fire fighters. It should be possible, for preliminary studies, to use the Draeger tubes, as described in section 5.7, to monitor the oxygen level and thus obtain some idea of the problem.

10. EFFECT OF CARBON DIOXIDE

10.1 GENERAL REMARKS

In the context of fire fighting carbon dioxide is usually claimed to be an inocuous gas harmful only by its displacement of oxygen and thus having a suffocating effect. This however is not the complete story.

10.2 PHYSIOLOGICAL EFFECTS

The actual physiological effects of carbon dioxide are identical whether the person accumulates his own carbon dioxide (ie by breath holding) or whether he inhales the carbon dioxide produced outside the body. The only difference is the rate of rise of the partial pressure of carbon dioxide in the blood (CO_2) which is possible. Both cardiac output and heart rate are increased in a linear relation to the pCO_2 within the range 20–80 mm Hg (3–11% CO_2) and the blood pressure is generally raised with increase of pCO_2. Elevation of pCO_2 produces unconsciousness at levels within the range of 90–120 mm Hg (13–17%). In fact carbon dioxide has in the past been used as an anaesthetic, but the amount required also caused total but reversible flattening of the electroencephalogram (EEG) and the frequent production of convulsions.[57]

However, in the case of firemen another important factor concerning the inspiration of carbon dioxide is that it causes an increase in the rate and depth of breathing. For example an atmosphere containing 2% carbon dioxide would increase the rate of breathing by about 50%.[29] Thus more of any noxious gases, vapours or particular matter present would be taken into the fireman's lungs. Such levels of carbon dioxide could be easily measured with a simple device such as the Draeger tube in order to obtain some scale of the hazard.

11. THE POSSIBLE EFFECT OF HEAT OR FLAME ON THE EYES

This possibility has no experimental backing at present. It is known that glass-blowers can develop cataracts (glass-blowers cataracts) through constantly being exposed to the heat and radiation from a hot flame or molten glass. Although firemen are not placed in this situation they are exposed to heat and flame both during training and operationally. Even if this caused a slight deterioration in eyesight it is worthy of investigation especially as the fireman ceases to be operational as soon as he needs to wear spectacles.

An analysis of the types of eye disorders suffered by fireman necessitating the wearing of spectacles compared with the normal population might throw some light on this possibility.

12. EFFECT OF FIRE FIGHTING ON LUNG FUNCTION

Finally one area in which it could be intuitively expected that the fireman has cause for concern is that involving non-specific respiratory disease of the lung. Because of this and the fact that sensitive and well developed non-invasive techniques are available lung function studies on firemen have become the starting point both in England, Scotland and America for research into the occupational hazards connected with fire fighting. The work in England and Scotland has just commenced but in America various research projects in this category have been carried out since 1970.

A long term study of Boston firemen began in 1970[72] comprising 1720 fire fighters who were examined during 1970/71 and then again during 1971/72. Their forced expiratory volume after one second ($FEV_{1.0}$) and forced vital capacity (FVC) was measured. It was concluded that experienced fire fighters had a higher chronic non-specific respiratory disease rate than new fire fighters but smoking masked the operational effect.

However, a mortality study of firemen from a Canadian city[52] covering the period 1921-1953 had previously demonstrated that firemen experienced a highly significant excess of cardiovascular deaths but a reduction in respiratory deaths. There was no significant difference in overall mortality when compared with all male deaths in the city.

It could be that due to the method of selection of firemen they are fitter than the general population and their work merely reduces their level of fitness to the average value. On the other hand the materials and toxic products from fires have changed markedly since 1921-1953 and therefore we are now dealing with a different situation. Current and proposed work in the United Kingdom* should go some way in answering this question together with clarifying the position with regard to future research.

13. CONCLUSIONS

13.1 CARBON MONOXIDE

The acute effect of carbon monoxide is still considered the major killer at fires even in the presence of burning plastics.[79] However, the neurological effects of non-lethal doses of carbon monoxide and the possibility that this may interfere with the fireman's awareness of dangerous situations[11,42] needs further investigation.

There is also evidence, from animal work, for suspecting that chronic carbon monoxide exposure may lead to cardiovascular disease via atheroma formation in the arteries.[9] This may be another avenue of research needing more effort, especially as some American studies point to cardiovascular-renal disease as being an occupational disease of fire fighters,[52] but it will be advantageous to await the outcome of the current mortality study.

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*Pulmonary function study of London firemen by Dr D Douglas (London School of Hygiene and Tropical Medicine) and the proposed mortality study of firemen.

Some workers have also shown[2,3] that angina of effort, in susceptible persons, is developed sooner when breathing fairly low concentrations of carbon monoxide - ie 50 ppm[2] and that the heart is more susceptible to ventricular fibrillation, a potentially fatal condition, under the same circumstances.[23] The latter hazard might possibly affect older firemen with latent cardiovascular disease.

13.2 HEAVY METALS

Another conceivable threat to fire fighters, if high blood pressure/ cardiovascular/renal disease are verified as being an occupational hazard, is that due to heavy metals, notably cadmium and antimony. These metals have recently come into more prominence as being hazardous in very small quantities[18] and are suspected of being a cause of the above diseases.[67,68,69] The importance to firemen is that cadmium can be used as a pigment in paints and antimony is present in many flame retardants and hence would be released during a fire and inhaled or ingested by the fire fighter.

13.3 ASBESTOS

There has been much publicity recently concerning asbestos and its capability of causing asbestosis, lung and bronchial cancer and mesothelioma after apparently slight exposures. Apart from the risk of, for example, the handling of asbestos products such as gloves and blankets. It is not so widely known that asbestos is also used as a 'filler' in some materials such as plastics as a reinforcing agent and also for cheapness of manufacture. There is at present no evidence to show that this asbestos is not released in small respirable particles along with the smoke etc at a fire.

13.4 TOXIC PRODUCTS FROM BURNING PLASTICS

Fortunately the risk from the toxic products of burning plastics has been anticipated and much research is going on in this field to isolate and identify hazardous biproducts.[88,89,90,91] One extremely toxic compound, toluene di-isocyanate (TDI) used in the manufacture of polyurethane has been known for some time, but it is now known to cause asthma in some susceptible people, in very low doses if the victim has been previously sensitized.[26,54] However, although toxicological studies are being carried out in animals to determine the products likely to be immediately toxic to the firefighter, serious consideration should be given to the possible carcinogenic effects of any emitted gases. Due to the fact that it may be many years after initial contact (up to 30 years or more in some cases) before a substance is suspected of causing cancer in humans, some attention should be given to the cheap and rapid in vitro tests being developed [17,51] in addition to the current animal tests.

13.5 HEAT STRESS

This problem has undoubtedly been with the fire brigades throughout their history and can give many symptoms such as exhaustion, dehydration, increased heart rate and thermal injury to the respiratory tract.[21] When this is combined with strenuous exercise and psychological stress it could be that the fireman is approaching the extreme limit of his physical endurance. This problem warrants further study.

13.6 PSYCHOLOGICAL STRESS

The stress of being awoken suddenly by an alarm bell and all that this entails is far from understood. Neither is the fact that people of certain body types and personalities are more prone to cardiovascular disease[75] and that men in

this category may be the type to favour fire fighting as a career.[52] It may be that this factor, if not allowed for, could bias any results obtained involving susceptibility to coronary heart disease.

13.7 EFFECT OF HEAVY EXERCISE

The heavy exercise necessary in carrying out the duties of firemen could be to their advantage in keeping healthy. On the other hand the heavy exercise by itself or in combination with other factors might be potentially hazardous for the older firemen who may have some latent heart disease. For this reason it would seem much more realistic for new recruits and firemen undergoing annual medical inspection to be subjected to some form of relatively heavy exercise and for an electrocardiogram (ECG) to be obtained during or after the exercise. This would present better conditions for showing up any heart abnormality which could easily go undetected during a normal 'static' examination.[19] There is also a need to study better methods of fitness training.

13.8 LACK OF OXYGEN

Any drastic reduction of oxygen concentration at a fire could also have repercussions on the fire fighter ranging from collapse to a feeling of euphoria.[15] In the euphoric state the firman would be completely unaware of the seriousness of the situation and may be a hazard to both himself and his colleagues. The effect of lack of oxygen would also be accentuated if the person was also suffering from carbon monoxide intoxication or was a heavy smoker. In the latter case the fireman's blood would already have some of its oxygen replaced by carbon monoxide leaving him at a disadvantage in coping with an oxygen deficient environment. This aspect requires further study involving controlled measurements at fires.

13.9 INCREASED LEVEL OF CARBON DIOXIDE

Carbon dioxide is often quoted as being a 'safe' gas and harmful only by causing asphyxiation due to it replacing air in an enclosed environment. Unfortunately in the case of a fireman this is not the only possible hazard. It is known that the inspiration of carbon dioxide can cause an increase in frequency and depth of breathing.[15] Hence this could also cause an increase in the amount of toxic gas or other irritants that a fireman would normally inspire.

13.10 SIZE OF SMOKE PARTICLES

Recent reports state that the temperature of fires and the volume of smoke generated in recent years has increased due to the extensive use of plastic materials in the construction and furnishings of buildings.[16] Leaving aside the possible toxic effects of the plastics it is conceivable that the particle size of the smoke emitted from fires involving plastic materials is smaller than that from 'conventional' fires. This would mean that the particles could travel further into the smaller airways of the lung[24] and either cause more damage themselves or by adsorption of toxic gases or vapours from the fire. The release of the adsorbed substances in the lung could then produce local 'hot spots' of damage.

13.11 EFFECT OF HEAT OR FLAME ON THE EYES

It is well established that glassblowers can develop a disease of the eye known as glassblower's cataract, due to the exposure of unprotected eyes to a hot flame or molten glass for long periods. There is as yet no evidence to disprove the hypothesis that firemen also may be experiencing some form of

eyesight deterioration, albeit to a lesser extent, due to the flames and heat to which they are regularly exposed. Although there is at present no scientific basis for this assumption many firemen do claim that their eyesight deteriorated more rapidly than they would hope for their age. Another factor encouraging work in this field is that firemen are compulsory retired from active fire fighting on requiring to wear spectacles possibly cutting short a career in its prime.

14. RECOMMENDATIONS FOR FUTURE RESEARCH

It can be seen from this paper that the field of research into the occupational health of firemen is relatively unexplored and could cover a wide range of topics. Many of these investigations are comparatively easy to carry out and should be considered as soon as possible to provide early guidance. They are shown below in order of importance/ease of achievement. The need for information outlined in sections 14.1 to 14.8 is of crucial importance to the understanding of firemen's health hazards, and should be sought as soon as possible. Research suggested in sections 14.9 to 14.13, however, depends very much on the outcome of current research projects and may or may not be considered necessary.

14.1 MONITORING OF THE ECG DURING A 24 HOUR PERIOD

The ECG (which includes heart-rate) is quite easy to monitor nowadays using miniature equipment such as that developed recently at the MRC Bioengineering Laboratory, Mill Hill. Using such equipment a fireman could be monitored for a 24 hour period, for example, both on and off watch. The monitoring equipment is unobstrusive and will not interfere in any way with the fireman's duty. Such an investigation carried out on a small group of firemen would give important information about a completely unknown field concerning the stress, physiological and psychological encountered by a fireman during the course of the day. This would be an ideal way for detecting any undue stresses imposed on the fireman during the course of his duties. It would also indicate the time scale of such stresses. Dr D Douglas of the London School of Hygiene and Tropical Medicine has expressed an interest in this idea and would be willing to assist in a pilot study using a selection of the firemen taking part in the lung function study of London firemen.

14.2 ECG MONITORING FOLLOWING EXERCISE DURING ROUTINE MEDICAL EXAMINATIONS

The ECG should also be measured during the initial and subsequent medical examinations after undergoing some systematic exercise such as the Master's Two-Step Test. This should help to identify those firemen suffering from any latent heart disease and avoid any possible fatal accidents while on duty due to stress or heavy exercise. Such measurements could easily be added to the normal medical examinations. Portable ECG apparatus is now easily available for use by general practitioners or other non-specialists (in cardiology). It will be necessary, however, for all abnormal ECG's, and probably the normal ones to be read by a consultant cardiologist.

14.3 THE EFFECT OF HEAT AND EXERCISE ON THE FIRE FIGHTER

Physiological tests on firemen at varying ambient temperatures and exercise levels whilst wearing their full operational clothing and equipment is another important field for future research. Experiments to establish energy expenditure (via oxygen consumption), heat stress, cardiac output, ECG etc could be relatively easily planned and carried out at the Fire Service Technical College or in a laboratory such as those available at universities, medical

schools and MOD establishments. It is very important that some basic knowledge is obtained in this area in order to understand the stresses imposed on the firemen. This information is available for most other arduous and hazardous occupations. It is necessary to have this data to enable serious consideration to be given to the occupational health of fire fighters.

14.4 EFFECT OF LACK OF OXYGEN AND INCREASED CARBON DIOXIDE LEVEL

The physiology connected with lack of oxygen and excess of carbon dioxide is well understood. What is needed, in the context of fire fighting, is a knowledge of the level of these two gases at the 'average' fire. The monitoring of such gases together with others such as carbon monoxide and toluene di-isocyanate is comparatively easy. For a preliminary study an analytical method using indicator tubes such as the Draeger tube could be used, these tubes are specific for a given gas or vapour and change colour when the correct gas is drawn through them using a simple bellows device. This work could be carried out by a fireman if necessary. Other portable physical methods of oxygen and carbon dioxide analyser are available but these would need to be carefully calibrated each time they are used. They would also require trained operators to handle them.

14.5 SMOKING, OBESITY AND CARDIOVASCULAR DISEASE

Another useful research project would be to compare the smoking habits of firemen with the general male population and also to compare their blood pressure and body weights. Cigarette smoking is known to be an important risk factor in coronary heart disease and the main cause of lung cancer. There is some suggestion[52] that firemen on average are overweight and this alone could lead to hypertension and coronary heart disease. Some of this information should come from the survey of London firemen being carried out by Dr D Douglas on behalf of the Home Office.

14.6 PARTICLE SIZE AND COMPOSITION OF SMOKE

Although this is considered a harder task than it would appear on the surface.[13] Some information is urgently needed concerning the particle size distribution and composition of smoke in order to interpret some of the other studies outlined in this report. The Chemical Defence Establishment, Porton Down, the Fire Research Station, Borehamwood, or the Laboratory of the Government Chemist might be the best co-workers in this field.

14.7 MEASUREMENT OF CARBOXYHAEMOGLOBIN LEVELS IN OPERATIONAL FIRE FIGHTERS

To help avoid any unnecessary stress on the fireman's cardiovascular system some knowledge of the concentration of carbon monoxide to which they are exposed during fire fighting is of great importance. The realistic way for this to be carried out is by measuring the carboxyhaemoglobin value of the blood directly or indirectly by means of a breath sample. In this way an integrated value of the fireman's exposure is obtained. The techniques for carrying out such an investigation are now widely used. Apparatus and expertise are available at, for example, Surrey University (Department of Human Biology and Health) and is used for carrying out occupational health studies on other groups of workers.

14.8 NEED FOR CONSTANT SURVEILLANCE OF NEW MATERIALS

It is important that a close watch is kept on all new materials entering the home and industry with regard to their possible long and short term effects, when ignited, on the health of fire fighters and preferably to obtain early disclosure by the manufacturers of any new chemical components or additives. This would involve the Scientific Advisory Branch, Home Office in forming a close liaison with the Fire Research Station, Borehamwood.

14.9 LUNG FUNCTION STUDIES

Any further research in this field awaits the results of current experiments in the United Kingdom (Drs Moran and Kerr - Glasgow, Dr Douglas - London) but some method of screening firemen after being overcome, with regard to their respiratory system and blood COHb level may prove to be necessary. This would enable the fireman overcome by 'smoke or fumes' to have the necessary period of recuperation before returning to active fire fighting. More thought will be needed in this context when the above results are available.

The Milwaukee Fire Brigade have recently instituted such a screening test which the firemen themselves operate. To date it has been instrumental in saving the cost of many hospital admissions previously considered necessary and in protecting firemen who were more seriously affected than at first apparent.

14.10 HAZARDS FROM THE TOXIC PRODUCTS FROM BURNING PLASTICS

Research in this field is being carried out by the Fire Research Station, Borehamwood and the Chemical Defence Establishment, Porton Down. Further research in this area awaits the outcome of the present investigations which should be closely monitored by all concerned with the occupational health of firemen.

14.11 CHRONIC EFFECTS OF CARBON MONOXIDE

If the mortality study indicates an excess of ischaemic heart disease then more work may be necessary in order to substantiate the claims that chronic carbon monoxide intoxication leads to atheroma formation and thence to cardiovascular disease. This type of work would need the expertise of the universities and research workers experienced in this field. It may be possible to seek the co-operation of workers interested in this problem from another point of view such as cigarette smoking, air pollution, etc.

14.12 THE EFFECT OF HEAVY METALS ON FIRE FIGHTERS (ANTIMONY AND CADMIUM)

At this stage the exact effect of antimony and cadmium on firemen would be impossible to assess. A measure of these two metals in the blood of a group of firemen compared with the general population, in order to add or eliminate them from the list of occupational hazards should be fairly easy to accomplish. As in 14.11 this will be necessary only if the mortality study points to an above average level of heart disease.

An atomic emission spectrometer set up for this purpose is available, for example, at Surrey University (Department of Biochemistry) and undoubtedly at many other hospitals or university laboratories. The Surrey University workers are at present interested in developing analytical techniques for measuring the incidence of heavy metals in general in the blood.

14.13 CARCINOGENIC TESTS OF FIRE PRODUCTS OF CURRENT AND FUTURE MATERIALS

Finally the time elapsing between exposure to a chemical and the ultimate development of cancer can be up to 30 years or more. Animal tests for potential carcinogens are available but expensive, time consuming and non-specific for humans (2-3 years/approximately £50,000 per chemical). In vitro tests are now being developed which in the near future should be available for carrying out such tests more quickly, less expensively and it is hoped more specifically for humans.[1,17,51] In the case of the Ames test[1] it should take 3 days to carry out and cost approximately £100 per chemical. The Bridges test[17] is still under development and further information on the scope of the procedure should be available in about one year. Nevertheless, for some time all positive results will require animal studies as well. They are not yet the complete answer.

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GLOSSARY OF TERMS

Alveoli:	The minute air sacs in the lung at which exchange of gases between the blood stream and the respiratory system takes place.
Angina pectoris:	Ischaemic pain associated with an insufficient oxygen supply to the myocardium of the heart.
Antimetabolite:	Interferes with the normal products of metabolism in the cell.
Aorta:	The main artery arising from the left ventriole of the heart.
Asbestosis:	Pneumoconiosis due to the inhalation of dust containing asbestos fibre, characterised by a fine and diffuse fibrosis of the lungs and pleural plaques.
Atherogenic:	Applied to agents inducing atheroma.
Atheroma:	A 'fatty' substance gradually deposited in the walls of the arteries throughout life. More common in older persons.
Arteriosclerosis:	Degeneration of the walls of the arteries.
Bronchial:	Pertaining to the bronchi.
Bronchi:	The tubes leading from the trachea into the lungs - each lung has one large bronchus which subdivide into smaller bronchi.
Capillary:	The small blood vessels between the arteries and the veins: the main exchange between blood and tissue occurs through the capillary walls.
Carboxyhaemoglobin:	The reversible combination of carbon monoxide and Haemoglobin formed in the blood on inhalation of carbon monoxide - diminishes the oxygen carrying power of the blood.
Carcinogen:	Any cancer producing agent.
Cardiopulmonary:	Relating to both heart and lungs.
Cataract:	Opacity of the crystalline lens of the eye or its capsule.
Cholesterol:	A complex unsaturated secondary alcohol.
Cilia:	Hair like processes on the cells in the trachea and bronchi which beat in a co-ordinated rhythm to remove foreign bodies from the airways before they reach the lungs.

Cognitive:	A general term covering all the various modes of knowing - perceiving, remembering, imagining, conceiving, judging and reasoning.
Coronary arteries:	The arteries that supply the myocardium of the heart with blood.
Electrocardiogram:	A record of the electrical signals from the heart.
Endothelial:	Pertaining to the endothelium.
Endothelium:	The single celled layer forming the inner lining of the heart and blood vessels.
Edema (or Oedema):	Abnormal collection of serous fluid in the tissue.
Emphysema:	Abnormal distension of the pulmonary alveoli by air often with consequent destruction of their walls and narrowing or obliteration of associated blood vessels.
Fibrosis:	An increase of fibrous tissue in an organ.
Homeostasis:	Maintenance of constancy of the internal environment (intercellular fluid).
Hypertension:	Blood pressure above normal.
Hypertensive:	Characterised by above normal blood pressure.
Hyperthermia:	Rise of body temperature - not due to bacterial invasion.
Hypoxia:	Oxygen lack in tissues.
Intima:	The innermost layer of the three layers of a blood vessel.
Intimal:	Relating to the intima.
Ischaemia:	Lack of adequate amounts of oxygen in the tissue cells.
Ischaemic heart disease:	A diseased state of the coronary vessels that interferes with the blood supply to the heart.
Latent heart disease:	Showing no clinical symptoms.
Mesothelioma:	A tumour of the pleura or peritoneum.
Minute volume:	The volume of air breathed per minute.
Mucous membrane:	A membrane which secretes mucous.
Myocardium:	The muscular tissue of the heart.
Pharyngeal:	Relating to the pharynx (throat).

Psychomotor:	Relating or referring to the motor effects of mental (cerebral) processes.
Synergistic:	An agent acting jointly with another agent.
Tachycardia:	A rapid heart rate.
Thorax:	Chest, section of the body between the neck and the abdomen.
Vasodilation: (or Vasodilatation):	Dilation (or dilatation) of the blood vessels.
Ventricular fibrillation:	Rapid unco-ordinated contractions of the heart ventricles which prevents the normal pumping action of the heart - circulation stops immediately - usually fatal unless treated at once by cardiac massage and cardiac defibrillator.

REFERENCES

1. Ames, B - Letter to the editor
Science 191: 241-242. 1976.

2. Anderson, E W, Andleman, R J, Strauch, J M, Fortuin, N J and Knelson, J H - Effect of low level carbon monoxide exposure on the onset and duration of angina pectoris - A study of ten patients with ischaemic heart disease.
Ann Intern Med 79: 46-50. 1973.

3. Aronow, W S - Carbon monoxide effect on exercise induced angina pectoris.
Ann Int Med 79: 392-395. 1973.

4. Aronow, W S and Cassidy, J - Effect of carbon monoxide on maximum treadmill exercise - A study in normal persons.
Ann Int Med 83: 496-499. 1975.

5. Aronow, W S, Harris, C N Isbell, M W, Rokaw, S N and Imparola - Effect of freeway travel on agnina pectoris.
Ann Int Med 77: 669-676. 1972

6. Astrand, P O and Rodahl, K - in Textbook of Work Physiology
Pub McGraw-Hill Book Company. 1970.

7. Astrup P - Some physiological and pathological effects of moderate carbon monoxide exposure.
B M J 4: 447-452. 1972.

8. Astrup, P - Carbon monoxide, smoking and atherosclerosis.
Postgraduate Med Journal 49: 697-706. 1973.

9. Astrup, P, Kjeldsen, K and Wanstrup, J - Enhancing influence of carbon monoxide on the development of atheromatosis in cholesterol fed rabbits.
J Athersol. 7: 343-354. 1967.

10. Axford, A T, McKerrow, C B, Parry-Jones, A, and Le Quesne, PM - Accidental exposure to isocyanate fumes in a group of firemen.
Br J Ind Med 33: 65-71. 1976.

11. Beard, R R and Wertheim, G - Behavioural impairment associated with small doses of carbon monoxide.
Ann J Public Health 57 (11): 2012-2022. 1967.

12. Bellet, S, De Guzman, N T, Costic, J B Roman L, and Fleischmain, D - The effect of inhalation of cigarette smoke on the ventricular fibrillation threshold in normal dogs and dogs with acute myocardial infarction.
Ann. Heart J 83: 67-76. 1976.

13. Berman, A - Communication at the FRS Toxicity Seminar. May 11 1976.

14. Best, C H and Taylor, N B - in The Physiological Basis of Medical Practise 7th Ed Pub The Williams and Wilkins Company - Baltimore. 1961.

15. Best, C H and Taylor, N B - in The Living Body, 4th Ed. Pub. Chapman and Hall. 1964.

16. Bowes, P C - Smoke and toxicity hazards of plastics in fires.
Ann. occup Hyg 17: 143-157. 1974.

17. Bridges J W - The biphenyl test.
University of Surrey Report (unpublished). 1976.

18. Browning, E - in Toxicity of Industrial Metals
2nd Ed. Pub. Butterworth. 1969.

19. Burke, S R - in Human Biology in Health and Disease
Pub. John Wiley and Sons Inc New York - London - Sydney - Toronto. 1975.

20. Chevalier, R B, Krumholtz, R A and Ross J C - Reaction of non-smokers to carbon monoxide inhalation: cardiopulmonary responses at rest and during exercise.
JAMA 198: (10): 1061-1064. 1966.

21. Colburn, R E - in Fire: Protection and Suppression
Pub McGraw-Hill Book Company. 1975.

22. Debias D A, Banerjee, C M, Birkhead, NC Harrer, W V, and Kazal L A - Carbon monoxide inhalation effects following myocardial infarction.
Arch Env Health. 27: 161-167. 1973.

23. Debias, D A, Banerjee, C M, Birkhead, N C, Greene, C H, Scott, D and Harrer, W V - Effects of carbon monoxide inhalation on ventrioular fibrillation
Arch, Environ, Health. 31: 42-46. 1976.

24. Dennis W L - in Inhaled Particles III Vol I ed W H Walton
Pub. Unwin Brothers Ltd. 1971.

25. Doan, A E, Peterson, D R, Blackmore, J R and Bruce, R A - Myocardial Ischaemia after maximal exercise in healthy men.
Am Heart J 69: 11-21. 1965.

26. Editorial - Isocyanates in industry.
Lancet 1: 1375-1376. 1970.

27. Editorial - Asbestos in the air.
Lancet 1: 944-945. 1976.

28. Editorial - Heat stabilizers. P20
European Plastics. March 1976.

29. Editorial - Carbon Monoxide and fires - Fire protection Review. December 1975.

30. Einhorn, I N - Physiological and toxicological aspects of smoke produced during the combustion of polymeric materials.
Environ. Health Perspectives. 11: 163-189. 1975.

31. Fassett, D W - Cadmium: Biological effects and occurrence in the environment.
Ann Rev Pharmacol. 15: 425-436. 1975.

32. Fisbein, J (Dunlop Ltd) - letter to the editor.
New Scientist. 61: 501. 1974.

33. Friberg, L - oited in - Cadmium: Biological effects and occurrence in the environment.
Ann Rev Pharmacol. 15: 425-436. 1975.

34. Garbasch, C, Mathiessen, M E, Helin, P and Lorenzen, I - Arteriosclerosis and hypoxia: Pt I Gross and microscopic changes in rabbit aorta induced by systemic hypoxia - histochemical studies.
J Atherosol, Res 9: 283-294. 1969.

35. Garbasch, C, Mathiessen, M E, Helin, P and Lorenzen, I - Arteriosclerosis and Hypoxia - Pt II. Biochemical changes in mucopolysaccharides and collagen of rabbit aorta induced by systemic hypoxia.
J Atherosol. Res 9: 283-294. 1969.

36. Glauser, S C, Bells, C T and Glauser, E M - Blood cadmium levels in normotensive and untreated hypertensive humans.
Lancet 1: 717-718. 1976.

37. Goldsmith, J R - Carbon monoxide and coronary heart disease: Compelling evidence in angina pectoris.
Ann Int Med 77(5): 808-810. 1972.

38. Goldsmith, J R and Landaw, S A - Carbon monoxide and human health.
Science 162: 1352-1359. 1968.

39. Hawkins, L - Lecturer in Human Physiology - University of Surrey 1976. (personal communication).

40. HM Factory Inspectorate Note 29 (rev).

41. Hendry, N W - The geology, occurrences, and major uses of asbestos.
Ann NY Acad Science. 132: 12-22. 1965.

42. Horvath, S M, Dahms, T E and O'Hanlon, J F - Carbon monoxide and human vigilance.
Arch Environ Hlth. 23(5): 343-347. 1971.

43. Howlett, L H and Shephard, R J - Carbon monoxide as a hazard in aviation.
J Occ Med 15(11): 874-877. 1973.

44. Kerslake, D Mc - Consultant in climatic physiology, Institute of Aviation Medicine, Farnborough, 1976.
(Personal Communication).

45. Kjeldsen, K, Astrup, P and Wanstrup, J - Ultrastructural intimal changes in the rabbit aorta after a moderate carbon monoxide exposure.
Atherosclerosis. 16: 67-82. 1972.

46. Kjeldsen, K, Thomsen, H K and Astrup, P - Effects of carbon monoxide on myocardium: Ultrastructural changes in rabbits after moderate, chronic exposure.
Circ Res 34: 339-348. 1974.

47. Kjeldsen, K, Wanstrup, J and Astrup, P - Enhancing influence of arterial hypoxia on the development of atheromatosis in cholesterol fed rabbits.
J Atheroscl Res 8: 835-845. 1968.

48. Koetzel, K, de Andrade, A E, Falleiros, J and Pactieco, J - Relation between hypertension and prolonged exposure to heat.
J Occ Med 15(11): 878-880. 1973.

49. Leithead, C S and Lind, A R - in Heat Stress and Heat Disorders pub. Caffel and Company. London. 1964.

50. Lescoe, R J - Carbon monoxide and angina.
Ann Int Med 81(4): 562-563. 1974.

51. Lewin, R - Bacteria to be used to detect cancer - causing chemicals (Ames test for mutagenesis).
New Scientist 69: 197. 1976.

52. Mastromatteo, E - II. A study of mortality in firemen of a city fire department.
Arch. Ind Hlth 20: 55/227 - 61/223. 1959.

53. Minard et al - cited in Heat Stress and Heat Disorders pub. Caffel and Company. London. 1964.

54. Moran, F - Health and Safety in firefighters.
Interfire '75.

55. Natusch, D F S and Wallace, J R - Urban aerosol toxicity: the influence of particle size.
Science 186: 695-699. 1974.

56. Natusch, D F S, Wallace, J R and Evans, C A - Toxic trace elements: Preferential concentration in respiratory particles.
Soience 183: 202-204. 1974.

57. Nunn, J F - in Applied Respiratory Physiology pub. Butterworths. 1972.

58. Penney,D - Effect of carbon monoxide on cardiac weight as compared with altitude effects.
J Appl Physiol. 37: 80-84. 1974.

59. Penney, D, Dunham, E and Benjamin, M - Chronic carbon monoxide exposure. The time course of haemoglobin, heartweight and lactate dehydrogenase isozyme changes.
Toxicol. applied. Pharmacol. 28: 493-497. 1974.

60. Penney, D, Sakai, J and Cook, K - Heart growth: Interacting effects of carbon monoxide and age.
Growth 38: 321-328. 1974.

61. Perry, HM and Yunice, A - A Acute pressor effects of intra-arterial cadmium and mercuric ions in anaesthetized rats.
Proc. Soc for exp Biol and Med 120: 805. 1965.

62. Petajan, J H, Vorhees, K J, Packham, S C Baldwin, R C, Einhorn, I N, Grunnet, M L, Dinger, B G and Birkey, M M - Extreme toxicity from combustion products of fire retarded polyurethane foams.
Soience 187(4178): 742-744. 1975.

63. Ross, R D - Carbon monoxide - in Air Pollution and Industry pub. Van Nostrand Reinhold Co. 1972.

64. Rose, E F and Rose, M - Carbon monoxide: A challenge to the physician. Clinical medicine 78(8): 12-18. 1971.

65. Sammons, J H and Coleman, P L - Firefighters occupational exposure to carbon monoxide.
J Occ Med 16: 543-546. 1974.

66. Samson Wright - in Applied Physiology.
11th Edition revised by C A Keele and E Neil. Pub. Oxford University Press. 1965.

67. Schroeder, H A - Renal cadmium and essential hypertension.
JAMA 187: 358. 1964.

68. Schroeder, H A - Cadmium hypertension in rats.
Ann J Physiol 207(1): 62-66. 1964.

69. Schroeder, H A and Vinten, W H - Hypertension induced in rats by small doses of cadmium.
Ann J physiol 202: 515-518. 1962.

70. Schulte, J H - Effect of mild carbon monoxide intoxication.
Arch Environm Health 7(4): 524-530. 1963.

71. Shettigara, P T and Morgan, R W - Asbestos, smoking and laryngeal carcinoma. Arch Environ Health 30: 517-519. 1975.

72. Sidor, R and Peters, J M - Firefighting and pulmonary function.
Am Rev Resp Dis 109: 249-254. 1974.

73. Sidor, R and Peters, J M - Prevalence rates of chronic non-specific respiratory disease in firefighters.
Am Rev Resp Dis 109: 255-261.

74. Siggaard-Anderson, J, Kjeldsen, K, Petersen, F B and Astrup, P - A possible connection between carbon monoxide exposure, capillary filtration rate and atherosclerosis - a preliminary report.
Acta Med Scand 182: 397-399. 1967.

75. Spain, D M, Brades, V A and Huss, G - Observations on atherosclerosis of the coronary arteries in males under the age of 46: A necropsy study with special reference to somatotypes.
Ann Int Med 38: 254. 1953.

76. Steward, R D - The effect of carbon monoxide on humans - a critical review.
Ann Rev Pharmacol 15: 409-423. 1975.

77. Stewart, R D, Newton, P E, Hooke, M J and Peterson, J E - Effect of carbon monoxide on time perception.
Arch Environ Health 27: 155-160. 1973.

78. Stewart, R D and Stewart, R C - Breath analyser used to test carbon monoxide levels in firemens blood.
Fire Engineering 128(8): 92-94. 1975.

79. Sylvia, D - Carbon monoxide is still the worst of lethal gases: Fire casualties seminar learns.
Fire Engineering 128(9): 31-34. 1975.

80. Taylor A (Chairman - Safety Sub-Committee - British rigid urethane foam manufacturers association) - Letter.
New Scientist 68: 658-659. 1975.

81. Thomsen, H K - Carbon monoxide induced atherosclerosis in primates. An electron-microscope study on the coronary arteries of macaca irus monkeys.
Atherosclerosis 20: 233-240. 1974.

82. Thomsen, H K and Kjeldsen, K - Threshold limit for carbon monoxide induced myocardial damage.
Arch Environ Health 29: 73-75. 1974.

83. Tucker, A - in The Toxic Metals
pub. Earth Island Ltd. 1972.

84. Wald N - Smoking filter tipped cigarettes may be more harmful than smoking untipped ones from the point of view of coronary heart disease.
Lancet 1: 136-138. 1976.

85. Wald, N, Howard, S, Smith P G and Kjeldsen, K - Association between atherosclerotic disease and carboxyhaemoglobin levels in tobacco smokers.
BMJ 1: 761-765. 1973.

86. Wanstrup, J, Kjeldsen, K and Astrup, P - Acceleration of spontaneous intimal subintimal changes in rabbit aorta by prolonged moderate carbon monoxide exposure.
Acta path microbiol scandinav 75: 353-362. 1969.

87. Willis, R A - in Pathology of Tumours.
4th edition pub. Butterworth, London 1967.

88. Woolley, W D - Decomposition products of PVC for studies in fires.
Br Polymer J 3: 186-193. 1971.

89. Woolley, W D - Nitrogen containing products from thermal decomposition of flexible polyurethane foams.
Br Polymer J 4: 27-43. 1972.

90. Woolley, W D - Studies of the dehydro-chlorination of PVC in Nitrogen and air.
Plastics and Polymers 40 (148): 203-208. 1972.

91. Woolley, W D - Toxic products from plastic materials in fires.
Plastics and Polymers 41(156): 280-286.

92. Young, S H and Stone, H L - Effect of a reduction in arterial oxygen content (CO) on coronary flow.
Aviation, Space and Environ Med 47(2): 142-146. 1976.

93. Zarem, H A Rattenborg, C C and Harmel, M - Carbon monoxide toxicity in human fire victims.
Arch Surg 107: 851-853. 1973.

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