AN EVALUATION OF BREATH CARBON MONOXIDE MEASUREMENT AND ITS USE IN THE ASSESSMENT OF ANTISMOKING HEALTH EDUCATION AMONG CORONARY HEART DISEASE PATIENTS

by

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Sponsored
by
Ministry of Higher Education
Egypt

A thesis submitted to the University
of Glasgow for the degree of Doctor
of Philosophy in the Faculty of Medicine November 1986

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DEDICATION

To my wife, Howyda and children,
Ahmed and Alaa for their
tolerance and endurance.

CONTENTS

ABBREVIATIONS

S	=	Scotland
WI	=	Western Infirmary, Glasgow
RH	=	Ruchill Hospital, Glasgow
X_1^2	=	Chi-square with one degree of freedom etc.
r	=	Pearsons product moment correlation coefficient
LR	=	Linear regression
ROC	=	Receiver Operating Curve
P	=	the probability of a given result occurring by chance.
df	=	degree of freedom
SD	=	Standard deviation
N.S	=	not significant.
LDH	=	Lactic acid dehydrogenase enzyme
MI	=	Myocardial Infarction
O	=	Observed number
E	=	Expected number
COHb	=	Carboxyhaemoglobin
CO	=	Carbon monoxide
SCN	=	Serum thiocyanate
IHD	=	Ischaemic Heart Disease
CK	=	Creatine Kinase
CHD	=	Coronary Heart Disease
CVD	=	Cardiovascular Disease
I	=	Intervention group
NI	=	Non-intervention group
RT	=	Row total
CT	=	Column total
GMI	=	Gas Measurement Instruments Ltd. (portable carbon monoxide monitor)
SGOT	=	Serum glutamic oxalacetic transaminase
WHO	=	World Health Organization

ACKNOWLEDGEMENTS

I would like to thank the Government of my native Egypt for sponsoring this research, and the staff of the Egyptian Educational Bureau for their sincere cooperation. I extend my grateful thanks to my supervisors Dr.R.P Knill-Jones, Professor A.J. Hedley and Professor G.T. Stewart for their patience and encouragment. This work could not have been possible without the useful guidance of the staff of the Department of Community Medicine, Ruchill Hospital; the Department of Cardiology in the Western Infirmary, Glasgow; Clydebank Health Centre; Glasgow High School and the permission of Drs. H. Dargie and MacArthur to interview their patients in the Western Infirmary, Glasgow and Clydebank Health Centre for access to patients' records.

Worthy of mention are Mr. David Hole, Statistician in the West of Scotland Cancer Surveillance Unit; Ms. Alison Raine, Computer Services; the Headmaster of Glasgow High School; the Administrator of Ruchill Hospital; Mr. MacLean, Health Visitor in the Western Infirmary, Glasgow and the Manager and staff of Gas Measurement Instruments Ltd., Renfrew, for their efficient services and facilities given by them in aid of this work in supplying the new breath carbon monoxide monitor.

My thanks also to the staff of the host Department of Community Medicine, particularly Dr. R. Knill-Jones, Senior Lecturer; Professor A.J. Hedley, Head of Department, and Mr. Harper Gilmour, Lecturer in Medical Statistics for their

continuous help, advice, invaluable suggestions and friendliness throughout the study, for reading this thesis and their criticisms and suggestions.

I am grateful to Mrs. W. Douglas for her typing the first draft of the manuscript. There were many, too numerous to mention, to whom I am greatly indebted for the completion of this work.

Finally, my special thanks are extended to the host Faculty of Medicine, University of Glasgow, Britain.

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SUMMARY

SUMMARY

A new method has been validated and used for measuring breath carbon monoxide (CO) as an indicator of success of two health education programmes for the prevention of smoking in patients admitted to the coronary care unit (CCU) in the Western Infirmary in Glasgow. Healthy adults (Ruchill Hospital employees) and Glasgow High School children were used to standardise methods for measuring carbon monoxide and to establish reference ranges for smokers and non-smokers.

In 212 patients (155 males, and 57 females) admitted to a CCU, 15% were non-smokers, 30% were ex-smokers and 55% were current smokers. Smokers (n=116) were divided (for practical purposes according to their addresses) into 2 groups which were similar in respect of age, sex, smoking habit, social class and the presence or absence of myocardial infarction. The non-intervention group was given routine health care and routine health education. The second (Health education group) was given the same care and, in addition, intensive (personalised) health education about giving up smoking from a health visitor.

Study of the total CCU patients showed no significant relationship between age and smoking habit. Males were less likely to have never smoked. There was no significant relationship between smoking habit and social class but there was the expected trend, with more smokers in the lower socioeconomic groups. There was no significant relationship between MI and the presence of stress in each group.

Evaluation of the anti-smoking intervention was carried out after one year. 41% of previous smokers in the non-intervention group had given up smoking. In the health education group 63% had given up and the overall comparison between the two groups was not significant (X^2 =3.1; 1df; P=N.S). Those who remained smokers after follow up in the health education group showed a significant reduction in the average number of cigarettes reported to be smoked per day ($p<0.001$). In comparison the difference in the non-intervention group was not significant. In each group there was no significant relationship between social class and patient's knowledge about coronary heart disease (CHD) risk factors. An intensive health education programme of limited duration carried out under the conditions of the study did not seem to provide a significantly increased stimulus for a long term change in smoking habit in patients admitted to CCU, although it did seem to reduce the average amount smoked.

The study also compared two methods used for measuring blood COHb. There was no significant relationship between blood carboxyhaemoglobin (COHb) and smoking habit or the number of cigarettes smoked per day. This finding disagrees with published data and the possible reasons are inaccuracy of the spectrophotometric method used (CO-oximeter 282) or oxygen inhalation during emergency treatment. Poor agreement was found between measurements obtained by the Gas-chromatography method and the spectrophotometric (CO-

oximeter) method. In addition, blood COHb was compared with breath CO. Blood COHb was mostly higher than breath CO, and the 95% confidence limits for the mean of differences between the two were 1.2 and 2.9. A higher coefficient of variation was present at low CO concentration and vice versa.

Breath CO is known to decrease over time. The calculated half life in this study was 182 minutes (SD = 15).

Study of breath CO changes and the smoking habit of Glasgow high school children (n=105) and Ruchill Hospital employees (n=143) showed that although 34% of the pupils were smokers, 72% of the smokers smoked only 1 to 4 cigarettes per day and they were mostly weekend smokers. In these children there was no significant relationship between smoking habit and breath CO but in contrast a highly significant relationship was found in the group of 143 Ruchill Hospital employees ($p<0.001$). When breath CO was used to detect smokers the minimum error rate (2.0 - specificity - sensitivity) was 0.37 at cut off points between 8-12 ppm CO for the employees, and 0.83 at 7 ppm for pupils. In addition to low sensitivity and low specificity for measuring breath CO in children, this CO monitor showed also a cross reactivity between breath hydrogen and breath CO. Therefore the value of using the objective measurement test (GMI CO monitor) of smoking behaviour is doubtful in children with low and sporadic consumption of tobacco.

The poor agreement between the two methods used for measuring COHb favours the use of breath CO. Measuring breath CO by the GMI hand-held CO monitor is simple and easy. The instrument is not sensitive to CO_2 or water vapour but is sensitive to hydrogen which puts some restrictions on its use, particularly in sugar malabsorption syndromes. This should be taken into account when interpreting breath CO measurements by this method.

INTRODUCTION

CHAPTER ONE : INTRODUCTION

1.1 GENERAL ASPECTS REGARDING TOBACCO CONSUMPTION AND ITS RELATED HEALTH HAZARDS

Le Riche believes that preventive medicine, like charity, should begin at home but usually doesn't. "The consumer" he said, "isn't interested in doing anything for his health that requires discipline, like eating less or exercising more. He wants to do things that are passively convenient and expects government or some other agency to find the solutions to his problem"(89). Le Riche believes also that we live in an era of self-induced disease, arising for example from the chemical manipulation of food, which has a far greater effect than people believe because we don't know what we are putting into food or what we're taking out of it.

Considering the problem of smoking, human beings are the only animals who deliberately take smoke into their bodies (63). This they have done for centuries, before the introduction of tobacco into Europe. Hippocrates prescribed smoke inhalation for asthma and certain other ailments, but the smoke came from dried cow dung or herbs. The word 'tobacco' is derived from a Y-shaped pipe called a 'tobaco', used by the American Indians, who inhaled smoke by inserting its two forks into their nostrils. The physician Francisco Hernandes was the first person to bring the tobacco plant to Europe

from Mexico in the 15th century. The French ambassador to Portugal, Jean Nicot sent seeds of the plant to the Queen Mother of France and it is from his name that the word nicotine is derived.

Over the centuries smoking habits have changed according to fashion, and also as a result of wars (63). Cigarettes began to be popular during World War I. Before that, tobacco consumption was mainly pipes, cigars, chewing tobacco and snuff (95). The annual consumption of manufactured cigarettes in the USA between 1920-1960 rose from 37 to 195 packs per adult. In 1968, the average medical practitioner found that 51% of his male patients and 33% of his female patients were regular smokers. Over the past decade the number of women smokers has increased by 50% and there has been a slight increase in smoking in males. Women are rapidly approaching men in terms of prevalence of cigarette smoking (120,158).

Smoking is, however, an international problem which has already begun to involve the developing countries(130). In the absence of resolute action, it will become an unavoidable epidemic and the resulting ill-health will replace the problems of infections and nutritional disease that are currently and rightly seen as public health priorities. The smoking problem in developing countries should be recognized from the start to be linked to activities in developed countries. In many developed countries certain forms of tobacco advertising and promotion have been restricted and levels of tar, nicotine, and carbon monoxide have been

reduced. Few such restraints apply in developing countries where cigarettes are promoted in disregard of their consequences for health(130). Smoking is less prevalent among females than among males. In children the figure rose from 6% to 21.8% in 1981 at the age of 15 in Colombo, Sri lanka. Smoking was practised by 38% of 18 year olds in Southern Brazil in 1981.

The main tobacco producers are China, USA, India, Brazil, USSR, Greece, Turkey, Japan, Bulgaria, Canada, Korea and Zimbabwe (130). The tobacco corporations in the developed world are encouraged by their governments to promote tobacco growing and sales in the third world.

In nearly all producing countries, tobacco is one of the most valuable crops grown and its contribution to total agricultural income is almost invariably significant (148). The value of world exports in 1979-1981 averaged US $ 4,000 million of which about US $ 1,600 million accrued to developing countries. The World Bank notes that tobacco consumption over the current decade is likely to increase at one percent a year in industrialised countries and nearly four percent in developing countries. America exports nearly half of the tobacco it produces. In 1981, nearly $ 600 million of mainly Virginia tobacco was sold overseas. The exports of tobacco leaf and tobacco products were worth over $ 2.7 billion. The new markets for it are now in the Third World.

For nearly twenty five years the US Federal Government helped tobacco exports to Third World countries by including the crop in the "Food for Peace" programme (148). This programme was to combat hunger but at the same time to develop new markets for US farm products, to dispose of surplus agricultural commodities and promote American foreign policy. Tobacco was excluded from the list of commodities in 1977 by a vote in the House of Representatives.

As regards the health hazards due to smoking, it plays a fundamental role in the cause of lung cancer and makes a major contribution to Ischaemic Heart Disease (63). Other diseases related to cigarette smoking are diseases of the digestive tract, chronic bronchitis, peptic ulcer and pulmonary tuberculosis (Doll and Hill 1964, 1966)(95). Rates of spontaneous abortion, low birth weight, neonatal death, and stillbirth are higher in mothers who are smokers. They also have a higher percentage of infants with congenital heart disease(120).

In India, Africa and China reports have confirmed that smoking plays an important role in the development of cardiovascular disease and to a lesser extent cerebrovascular disease(130). Although lung cancer is common among cigarette smokers in developing as in developed countries, cancer of the oesophagus is more common in developing countries than in the developed ones, and tobacco consumption plays an important role in its causation.

From 1955 to 1967, cigarette production has expanded by one half in the developing and 40% in developed countries[48]. There is no suggestion of a decrease in cigarette smoking in developing countries where tobacco consumption has yet to be influenced by health considerations. Therefore, measures are needed to stop the growth of the habit in developing countries to decrease premature deaths and disability[48,130]. In order to do that, specific recommendations on advertising, reductions in tar yield, health warnings and education are necessary and if no action is taken the cost will be counted in many lives lost[130]. Therefore in developing countries just as much as in developed countries the prevention of cigarette smoking is an essential part of any programme in preventive medicine[158].

Coronary Heart Disease (CHD) forms one of the major public health epidemics of the 20th century [46]. It was first described as a clinical entity in 1912, and it is recognized with increasing frequency as being a major health problem. By 1940 it was the leading cause of death in the United States and its frequency continued to rise through the 1950s, reaching a peak in the middle of 1960. During the last 20 years the mortality from CHD has decreased markedly in the United States and in several other countries such as Canada, Australia, but the rates have remained constant in most West European countries and have increased dramatically in Eastern Europe and some countries of the Third World.

Therapeutic medicine at present can do very little to reduce mortality due to smoking [125]. Nearly one third of all initial attacks of diagnosed myocardial infarction (MI) are fatal before the arrival of the physician and even in those who survive long enough to come under medical care, the myocardium has frequently suffered severe and irreversible damage. Therefore it is apparent that the only hope for control of this disease lies in discovering and applying effective preventive measures.

At 1979 prices, the cost in million pounds to the National Health Service (NHS) of hospital tratment in England and Wales for road accidents was £75 M, alcohol related diseases £50-69 M, accidents at home £87 M and the highest £115 M for smoking related diseases [136].

In USA it was estimated that the excess of working man-days lost in the year was 77 million, of days spent ill in bed 88 million[48]. In Britain, the Royal College of Physicians estimated that the smoking habit results in the loss of 50 million working days every year and surveys have shown that smokers of more than 20 cigarettes a day have twice as much time off work due to illness as do non-smokers [136].

In USA cigarette sales had declined in 1964 by 11 billion dollars as the effect of antismoking public service advertising on radio and television began to be felt. In 1964 52% of men and 32% of women 21 years of age and over

smoked. By 1975 these figures had declined to 39% and 29% respectively [90]. Since 1974 the number of cigarettes sold in the U.K. has been decreasing steadily. Sales of other forms of tobacco have also decreased slightly, 1978 was the first year since 1973 when the weight of tobacco sold (113.1 million Kgs.) increased from the previous year (104.5 million Kgs.) [136]. Since 1978 the weight of tobacco sold has been gradually decreasing.

There is a growing awareness of the dangers in cigarette smoking at work and of its redefinition as an environmental hazard; whether or not passive smoking is harmful to the recipient, it is increasingly perceived as harming the rights of non-smokers[130].

The effect of passive smoking on infants and children is evident from Greenberg et al's study in North Carolina [59] Nicotine and cotinine in the saliva and urine were significantly higher in the exposed groups than in the non-exposed group. There was a direct relation between Cotinine excretion by the infants and self-reported smoking behaviour of the mothers during the previous 24 hours.

Russell [131] explored the realistic goal of safer smoking and he thought that total abstinence from smoking is unrealistic and doomed to fail. He argued that the carbon monoxide yield of cigarette brands should be added to the official tar and nicotine tables. The safer cigarette, he suggested, is likely to be the one with low tar and CO yields

but a higher rather than low nicotine yield. However, the goal of acceptably safe smoking will probably require the elimination of cigarette smoking in favour of non-inhaled smoking of pipes or cigars with the combined effects of health education coupled to selective taxation. For this more realistic goal, success is not only possible but probable.

RESEARCH HYPOTHESIS

The health hazards associated with tobacco smoking are too numerous to mention. Nowadays a substantial reduction in tobacco smoking has been achieved through health education. Indirect methods of measuring smoking prevalence in a population are important for checking self-reported smoking habit. A simple and reliable breath carbon monoxide measuring device might provide an alternative method.

The success of focussed health education on a coronary heart disease high risk group also needs evaluation.

1.2 AIMS AND OBJECTIVES

The principal aims of this project were to:

1. Test the reproducibility of carboxyhaemoglobin and breath carbon monoxide measurements due to lack of published data on the subject.

2. To study changes over time and to compare blood carboxyhaemoglobin and breath carbon monoxide as a critical measuring method and to set the range for smokers and non-smokers.

3. Compare the outcome of two short-term health education interventions to combat smoking by use of carbon monoxide measurements. Group I (non-intervention) received routine advice about smoking and health education, Group II (health education) received additional personalised intensive health education advice from a health visitor including follow-up interviews in their homes.

4. Compare the characteristics of the group of patients who succeeded in stopping smoking with the failures, with respect to factors such as age, sex, social class, physical and mental status.

REVIEW OF LITERATURE

CHAPTER TWO : REVIEW OF LITERATURE

2.1 HISTORY OF CORONARY HEART DISEASES IN GENERAL AND EGYPT IN PARTICULAR.

Ancient Egyptians had described most of the blood circulation in the body and some related diseases and they were ahead of the Greeks who, 2,000 years later, never mentioned the pulse rate (57). Egyptian papyri tell us a lot more in this matter e.g. that the heart is located on the left side from which it is displaced only by disease. The heart pours into a receptacle (the aorta) from which vessels branch to all the organs.

Especially interesting is prescription 855e of the Ebers papyrus that explains weakness of the heart (57). Peripheral circulatory insufficiency expressed by such expressions as "the heart is dumb in the limbs" for example this remarkable description of cardiac ischaemia "if you examine a man for illness in his cardia, and if he suffers from pain in his arms, in his breast and in one side of his cardia it is death threatening him " (Ebers 191). Such observations may be at the origin of the belief in a nervous connection between the heart and the ring finger. Theologians explained creation as being the making of the heart that conceives and of the tongue that commands. The heart came thus to be held responsible for man's actions. In scenes of the final judgement in the Book of the Dead, it was weighted against the

feather of truth. The results were recorded by the scribe of the gods, standing behind the jackal-headed god of the dead, Anubis, who tested the tongue on the scales, in front of the monster Amam who stood ready to devour the damned.

After the golden age of papyri, nothing happened for over a thousand years (57). The main cause of these dark periods was the repeated havoc of wars and invasions. The House of Life, which more or less corresponded to our academies was rebuilt later on. After Alexander's death, the Ptolemies established a grand tradition and made their city (Alexandria) an international emporium of trade and ideas that attracted the finest scholars of the known world. Two of these showed exceptional brilliance. They were Herophilus and Erasistratos who separated arteries from veins, differentiated arterial from venous blood and nearly discovered circulation. It is difficult to tell how deeply these two scientists might have been inspired by stray copies of the Pharaonic "Beginning of the Physician's Secret" or the other papyri kept in the libraries of Alexandria and Memphis for, according to Galen, Greek physicians still studied there in the second century A D. But some notions seem curiously common to the Alexandrian and Pharaonic physicians: the relationship of the heart to the pulse, the notion of the pulse rate and its importance in diagnosis, and the concept of healthy and morbid circulating matter of different kinds.

2.2 MORTALTY FROM CORONARY HEART DISEASE

In the UK and the USA one third of all male deaths between 35 and 64 years of age are due to coronary heart disease (CHD) [48]. In the USA 25% of the deaths caused by CHD each year are also thought to be smoking-related. Cigarette smoking also accounts for 1 in 5 cancer deaths [48,63].

Prospective surveys have indicated that among young men the coronary mortality risk is five to ten times higher in heavy smokers than non-smokers. (Hammond, 1966; Doll and Hill, 1964) [125]. The risk ratio diminished with advancing age. At all ages it is substantially less than for lung cancer; but because heart disease is so much commoner it is found that cardiovascular disease rather than lung cancer accounts for most of the excess mortality of cigarette smokers.

The age adjusted mortality ratio for male cigarette smokers in the USA for all causes of death was found to be 70% higher than that for non-smokers [63]. For lung cancer, the death rate in smokers was nearly 10 times higher than for non-smokers. Many other diseases including cancer of the larynx and cancer of the oesophagus showed significantly higher mortality rates for smokers.

The impression of many physicians that serious cardiovascular diseases are now more frequently seen in younger subjects has been confirmed by statistical reports from the Registrar General's Office (18). The death rate from coronary heart disease in males aged 30 to 49 years has increased by 37% between 1950 and 1974.

According to the Royal College of Physicians of London, the calculated deaths in Scotland in 1980 from coronary artery disease, lung cancer and chronic bronchitis were over 9,000 deaths attributable to smoking compared to 740 from road accidents (134).

The recent slight decrease in mortality from lung cancer, bronchitis and CHD in both sexes in Scotland may hopefully indicate a trend which will continue (134). Certainly the steady decrease in lung cancer mortality in early middle aged males has been related to the decrease in male smoking, which is at least partly attributable to health education. The failure of female mortality to fall until 1979-80 is consistent with the failure of overall female smoking to fall significantly.

The Advisory Committee of the Surgeon-General reported a markedly lower mortality ratio for ex-smokers than for current cigarette smokers and half of the excess deaths associated with cigarette smoking were attributed to CHD (14,61).

The relation between the number of cigarettes smoked daily and death rate is clear (48,63). Thus the death rate from all smoking related diseases was increased by 40% for smokers of 1-9 cigarettes per day, 70% for 10-19 per day and 90% for 20-39 per day. Mortality also was greater for inhalers than for non-inhalers.

Middle-aged men smoking more than 20 cigarettes each day are about 20 times as likely to die from lung cancer, and about twice as likely to die from CHD as their non-smoking counterparts. Out of 100 excess deaths from all causes observed among heavy smokers, about 16 may be due to lung cancer but 50 might result from heart disease (48,58,118) Sudden death was four times more frequent in smokers than non-smokers (95).

The time of death after myocardial infarction is important: in a study in Kuwait, 85% of myocardial infarction deaths occurred in the first 48 hours (41). In a coronary care unit most deaths occured within the first 12 hours. In Scandinavia it has been reported that the case fatality rate within the first hour was 15.5 % for males and 9.3% for females and within a year was 44.2% for females and 37.4% for females (52).

In the USA the major reduction in smoking has been in men, while the prevalence of smoking among young women has increased. Yet, both had an equal decline in coronary

mortality (158). Several factors probably explain this. The greatest decrease in smoking in the USA has been in men, particularly among middle-aged and older men who are at high risk of coronary disease and account for most coronary deaths. The increase in smoking occurred among young women, who are at extremely low risk of coronary disease and account for little of the overall coronary mortality.

The conclusions of the Maryland conference in 1978 (34) on the decline in CHD mortality were (1) the decrease in coronary heart disease is real and not a result of artefacts or changes in death certificate coding;(2) both primary prevention through changes in risk factors and fundamental and clinical research leading to better medical care have probably contributed to, but not fully explained, the decline, and (3) a precise quantification of the causes requires further studies.

In the USA, one large prepaid health care plan found that the number of coronary heart disease patients discharged has decreased each year since 1971 while the case fatality rate has not changed (34). In contrast, within a community with a comprehensive medical record system, the incidence of new coronary events among those having no previous coronary disease has not changed over recent years, while the frequency of deaths among those with known coronary heart disease has decreased. These limited observations cannot be extrapolated to the whole country and the question of trends

in heart attack incidence remains unanswered. Changes in coronary heart disease care have probably had an impact on the decline but the contribution is difficult to ascertain; the same applies for changes in risk factors for atherosclerosis. Mortality from hypertensive heart disease and stroke began to decline some years before effective medical therapy for hypertension was available and during a time when coronary heart disease mortality was increasing. Although there is general agreement that the decline in CHD is real, the probable cause or causes could not be precisely identified.

Ischemic heart disease mortality information reported to WHO shows downward trends that are similar but less marked than in USA for Canada , Australia, Finland, Belgium and possibly Britain between 1968 and 1976. Upward trends are noted in two Scandinavian countries (Sweden and Denmark), France, Poland, three Balkan countries (Yugoslavia, Romania, Bulgaria) and possibly in the Federal Republic of Germany, Austria, and Italy. The possible causes were not mentioned in this paper although the increase may be due to social or environmental conditions (42). In eight other countries IHD rates appeared to show little change.

Data on international CHD mortality trends have not been reported on a comprehensive scale since data have not been analysed separately by sex. Cause-specific death rates have been used but these are subject to various possible errors and uncertainties. The reported mortality trends need more

detailed analysis (42). A more definitive report about CHD mortality trends should take into account competing causes of death with attention to any differences between the rates for men and women. The social and environmental conditions that affect CHD risk factors are likely to show different time trends not only between, but also within countries. Future analysis should take also account of such variation if the necessary data are available, since they could throw light on the reasons underlying differential trends.

The influence of cigarette smoking on prognosis after a first myocardial infarction was studied by Sparrow and Dawber (1978) (144) in the Framingham study. Follow-up of 458 subjects developing myocardial infarction in terms of recurrent myocardial infarction, mortality and cigarette smoking revealed that of 82.8% of patients who survived the initial attack 53.3% had been cigarette smokers and 45.9% non-smokers. There was a 62.0% reduction in mortality among patients who stopped smoking compared to those who continued, over a 6 year period. However no significant difference in recurrent myocardial infarction rates was observed between the two groups.

Coronary heart disease (CHD) and cerebrovascular disease form the first and third most important causes of death in Israel (43). A comparison of the international trends reveals that Israeli Jews lagged behind only American and Australians in the rate of decline of coronary deaths (53). They have a peculiar feature also which is the unusually low male :

female mortality ratio and in 1970 -71 it declined from 1.8 at ages 25 to 34 to 1.05 at more than 75 year. During the corresponding period, CHD mortality rates in Israeli Jewish women were among the highest in the world. The sex ratio of Israeli CHD mortality rates (averaged for ages 40-69 years) was the lowest reported in any country (1.92), while the highest was in Finland (4.74). This could reflect a sex-genetic-environment interaction.

The importance of examination of changes in mortality from CHD by examining the male/female ratio rather than by using conventional methods was illustrated by Anderson (1973) in Toronto (5). The fluctuation in rates caused by changes in terminology or diagnostic methods have tended to affect both sexes to an equal extent and, therefore, largely cancel out when the ratio of the two rates is calculated and this approach simplifies the interpretation of male mortality trends over the years. The ratio for the age group 45-64 for all heart disease rose from 1.1 in 1920 to 3.3 in 1968. By 1968 the rate for all-cause male deaths rose to more than double the female rate (ratio 2:1) compared to only a 10 % male excess in 1920.

2.3 SMOKING AND RISK FACTORS FOR CORONARY HEART DISEASE AND THE CHANGING PATTERN AMONG SMOKERS

2.3.1 CORONARY HEART DISEASE RISK FACTORS.

It is accepted that smoking can cause a disease if [48](1) a temporal relationship is present (2) there is a strong association between cause and effect (3) there is consistency of results from several studies (4) it is biologically plausible (5) the incidence of the disease is quantitatively related to increasing exposure to cigarette smoke; (6) the incidence decreases in those who stop smoking, and (7) the disease could be produced or exacerbated by cigarette smoking. The evidence is strengthened if the disease can be produced in animals by exposure to cigarette smoke.

Paul et al (107) in their 4 year prospective study of coronary heart disease (CHD) carried out in an industrial population found that approximately one per 100 men per year will develop CHD and there was an association with an early age of death of father, history of chest discomfort, history of chronic cough, history of shortness of breath, presence of increased skin fold thickness, elevated BP, elevated blood cholesterol, ST and T abnormalities in the electrocardiogram and use of cigarettes and coffee. No relation was encountered between body weight, mean blood sugar levels, lipoprotein lipase level or diet (other than coffee) and the development of CHD. Similarly there is no association with job type and no certain relation to physical activity off the job.

In contrast to the Paul et al study, diabetes mellitus in a Kuwaiti myocardial infarction study was present in 51.0% of the Kuwaiti patients and 31% in non-Kuwaiti patients (41). The occurrence of the risk factors hypertension, smoking, family history and previous CHD was very similar in the diabetic and non-diabetic group. The prevalence of diabetes in the population was not mentioned in this paper.

Under present nutritional and behavioural conditions in the western world it is now apparent that lipid infiltration of coronary vessels may occur by the age of 20 years (7,67).

2.3.1.1 AGE, SEX, MARITAL STATUS AND SMOKING HABIT

Kannel et al(82) in 1965 in the Framingham study mentioned that in both sexes it is quite clear that susceptibility to CHD increases with age. The male however is more susceptible than the female. Women develop MI with comparable frequency some 20 years later in life but overall more men than women develop CHD, and they are more susceptible to the lethal forms of CHD. Only angina pectoris has similar incidence in both sexes.

In the Kuwait study, most of the MI patients (61.3 %) were under the age of 60 years and 38.7% were older (41). There were nearly five times as many males (85.2%) as females (14.8%) among the MI patients. The prevalence rate of IHD correlated positively with age, marital status (divorce or widowhood).

The New Zealand study of smoking among 4,000 children from nine schools showed that boys began smoking at about 11 years and girls at 12 years (63). A comparison was made with a similar study of children in English secondary modern schools. New Zealand children appeared to have a higher proportion of smokers than English children.

Until recently boys smoked more than girls (136). Information from a number of countries now suggest strongly that smoking by girls under 16 is increasing and sometimes surpasses that by boys. Limited surveys in the UK suggest that the gap between girls and boys has largely disappeared. For example, there was little information on the smoking habits of school children in Scotland until the report "Smoking among secondary school children " was published in 1983 (133). It was disturbing that 24% of the children under age 18 said they smoked, 15% regularly (smoking one or more cigarettes a week) and 9% occasionally. The proportion of regular smokers rose from 4% of first year children to 10% in the second year, 19% in the third year and 26% in the fourth year. The proportion of boys and girls smoking regularly was similar, 15% of boys and 14% of girls. About one-fifth of the regular cigarette smokers in the sample had already acquired a habit of smoking more than ten cigarettes per day. The proportion of children who had never smoked decreased from 60% of the first year children to 40% in the second year, 31% in the third year and 26% in the fourth year.

Study of age and myocardial infarction

In a study of the registered cases of acute MI and sudden death due to CHD in 1971 in two Swedish and two Finnish cities during a one-year period,[52] the incidence increased steeply with age. In all groups it was far higher in males than in females. The case fatality rate was 44.2% for males and 37.4% for females and the difference was evident in the first hour after the onset of the attack. In acute MI patients, a history of previous cardiovascular disease was much more common than in the general population; such a history was found more often in patients who died suddenly than in acute MI patients who did not die suddenly.

In India, Sharma et al (1976) [138] studied patients admitted to a CCU in Indore. They found that CHD was more common among age group 41-60 years, among professional workers and in persons having a higher social economic status with sedentary habits. This finding which is related to smoking is opposite to the smoking pattern and social classes in Britain [25,103].

2.3.1.2 PHYSICAL ACTIVITY AND CORONARY HEART DISEASE

Physicians and other health professionals are actively promoting physical exercise as preventive and therapeutic measures for CHD, yet critically controlled studies are rare

and short in defining the exact role of physical activity in coronary artery disease (50). The consistent finding of many studies included decreased heart rate at rest and increased during exercise allowing increased myocardial perfusion at any work load. An important question is whether or not these haemodynamic and functional changes increase the quality of life. Psychological improvement is often noted clinically but the effect on morbidity and mortality is unknown. In a case-control study the results showed that there was no marked difference in physical activity of the job between coronary and non-coronary groups (163).

The relationship of smoking habits and customary levels of physical activity at the time of infarction to both CHD incidence and early mortality has been studied by Frank et al in New York, 1966 (49). Age-adjusted incidence rates for smokers and non-smokers of differing physical activity levels showed that physically inactive men show higher incidence of first myocardial infarction than more active men, among both smokers and non-smokers. Smokers exhibit an incidence of first infarction which is almost twice that of non-smokers. No difference in incidence of initial infarction was found between the least active non-smokers and the relatively more active cigarette smokers. The highest incidence is in inactive smokers and the lowest by the relatively more active non-smokers, with nearly three-fold difference between these two groups. Smoking had no significant relation to the immediate prognosis for either the least active or relatively more active men. The age-adjusted death rate from first

myocardial infarction showed a higher rate 3.29 among the least active men in comparison with 0.89 for more active men. This reflects the combined influence of increased incidence and increased risk of death shortly after onset [49].

2.3.1.3 JOB STRESS AND CORONARY HEART DISEASE

The relation between job stress and risk factors for CHD had been studied in 1972 by Shirom et al among five occupational categories in Israel [140] Most of the CHD morbidity rates, the average values of CHD risk factors and average score of job stress and strains did not significantly differ among managerial and professional workers, clerical workers, craftsmen, factory workers and agricultural workers. In order to investigate the patterns of association of job stresses and strains with CHD risk factors, smallest space analysis was performed on the data matrix of each of the four major occupational categories separately. White collar workers were most disposed to obesity as a possible risk factor. Agricultural workers were most likely to experience job stress and strains associated with increased CHD risk, this has not been found in U.K. or European contries, and it may be peculiar to Israel.

2.3.1.4 WEIGHT.

Nemery et al in their study about smoking, lung function and body weight reported that smokers (76.1 Kg) were found to weigh significantly less than non-smokers (81.6 Kg) [105].

The lower weight of smokers was attributable to a group with airflow obstruction (FEV_1/VC) <66%, who weighed less (by 4.8 Kg; $p<0.05$) than smokers with normal FEV_1/VC ratio. In smokers only body mass index and FEV_1/VC ratio were closely related ($r = 0.34$; $p<0.001$). This association was apparently not due to an effect of body weight on lung function.

2.3.1.5. GENETIC COMPONENT IN CORONARY HEART DISEASE.

Deaths due to CHD among relatives of coronary patients were studied on the basis of death certificates among equal numbers of matched controls. They were one and a half fold higher among first degree relatives [149]. The average age at death to second degree relatives was lower. The age at onset of MI did not affect the risk to relatives whereas sex-related influence was significant. The risk increase was found to be the greatest to first degree male relatives of female propositi, or over 7-fold to fathers and brothers. Mothers and sisters of both male and female propositi had a 4-5 fold risk increase over the expected value among coronary mortality relatives of controls which is close to that of the general population. These findings indicate a genetic component in the aetiology of CHD.

2.3.1.6 CORONARY HEART DISEASE RISK FACTORS AMONG CHILDREN.

Paediatricians have done well in keeping children healthy as children but at the same time they have fallen far short of creating healthy adults [165]. Williams and Wynder demonstrated that risk factors for chronic diseases can be identified among young children and are highly prevalent as well. Twenty-five per cent of New York high school students were smokers, 5-15 % were overweight and 20% or more of school children aged 5-15 years may be hypercholesterolaemic. In order to fill this blind spot the American Health Foundation in New York City developed the " Know your body program " to improve health-related behaviour in a school-age population. As with other educational endeavours health-related behaviour can most effectively be influenced by lessons learned early in life.

Risk factors for coronary heart disease (CHD), can be identified among children [166]. It was reported that 46% of a sample of 8 to 12-year old boys in California had one risk factor and an additional 14% had two or more risk factors. Preliminary data from a sample of 3000 11 to 14-year old public school children in the "Know Your Body" study indicate that about 40% have one or more risk factor for CHD (overweight, elevated cholesterol, current cigarette smoker, poor physical fitness, hypertension and diabetes).

2.3.1.7 CORONARY HEART DISEASE RISK SCORE CALCULATION.

Rose and Hamilton [126] in their randomised controlled trial on the effect on middle-aged men of advice to stop smoking selected the cigarette smokers from the Whitehall study of male civil servants in London with high cardiorespiratory risk as follows :-

A risk score was calculated for each man who smoked 5 or more cigarettes per day. This score was based on a modification of the multivariate linear discriminant function coefficients. The complete cardiorespiratory risk score was calculated thus:

0.07 X (age in years)
+0.01 X (plasma cholesterol in mg/100 ml)
+0.02 X (systolic blood pressure in mm Hg)
+0.01 X (blood sugar in mg/100 ml two hours after a 50 gm glucose load)
+1.5 for history of angina
+0.5 for possible history of MI
+2.0 for history of intermittent claudication
+3.0 for major ECG findings (Minnesota codes 1.1-2)
+1.0 for minor ECG findings
+1.5 for history of phlegm for 3 months each winter
+0.5 for history of exertional dyspnoea
+2.0 for FEV1.0 <2.2
+1.0 for FEV1.0 >2.2<3.0

The cut-off point was determined (10.24) such that the score of 10% of all men and 32% of smokers exceed this value [126].

After one year, 63% of the intervention group reported that they were not smoking any cigarettes and 29% in the normal care group, indicating a highly significant effect of giving advice about smoking cessation.

2.3.1.8 RELATIONSHIP BETWEEN CORONARY HEART DISEASE RISK FACTORS.

In order to study the relationship between risk factors, Kok et al tested the hypothesis that risk factors are inter-related (86). They studied the simultaneous occurrence of smoking, inadequate nutrition, obesity and physical inactivity in a random sample (n=1951) of the Dutch adult population by comparing observed and expected rates for up to four risk factors. Kok et al found that 20 % of the studied population had no risk factors, 66% had one or two, 10% had 3, and all four risk factors were found in 1%. Almost equal proportions with one or two risk factors were found in the two sexes. Significant sex differences were observed for the presence of zero risk habits (men 14%, women 24%). The effect of differing life styles was examined by using Fisher's linear discriminat function. The order in which important cardiovascular risk factors were reported was as follows:- stress (70%), diet excessive in fat (46%), hypertension (40%), smoking and physical inactivity shared the fourth place with 35%, and hyperlipidemia (31%) was the least important. The independent demographic and socioeconomic determinants of "risky" life style were found to be sex (men), education (low), and occupation (low); marital status and familial social class were no longer discriminating between the two categories of life style, so

men with a low level of education and occupation constituted the target group in which three or four risk factors were aggregated. The results did not suggest systematic clustering. The assumption of independence of these risk factors also could not be maintained .

In the national cooperative pooling project, the 10 year rates for first major coronary event was 20 per 1,000 for men aged 30-59 at entry who had none of the three risk factors (hypertension, elevated cholesterol level, and smoking) as contrasted with a rate of 171/1,000 for those with all three factors [93]. Similar findings were found in the prospective Framingham study. The removal of all three factors was expected to reduce the risk of a major coronary event by over 80%. This is what happened in the Veteran Administration study where there was a clear reduction in the occurence of MI, congestive heart failure, and sudden death. The findings in other intervention trials are generally similar.

2.3.1.9 SMOKING AND MYOCARDIAL INFARCTION.

The importance of the relationship between smoking and myocardial infarction has been studied by Uhl and Farrell [151]. They found that smoking was the most common risk factor in two groups of MI patients. They compared the risk factors and clinical course of 165 patients under 40 years of age having MI (group I) compared to 100 patients over 40 (group II). Six risk factors were analysed : smoking 20 pack-years, hyperlipidaemia, hypertension, family history of

ischaemic disease, diabetes mellitus and obesity. Patients under age 40 had more risk factors than those over 40 although 61% of the younger patients were heavy smokers compared with 75 % of the older patients [51,64]. The higher prevalence of heavy smoking in group II was probably due to heavy smoking defined as 20 or more pack years [151]. The younger patients would have had to smoke two packs/day or more to be included in the heavy smoker category. Group I had hyperlipidaemia, obesity and family history more commonly than did group II, while hypertension was more frequent in the older patients, a prior history of angina was present in nearly half of group I and II but physical exertion just prior to MI was more common in group I than in group II. The complication rate was similar in both groups but mortality rate was lower in young MI patients.

Unfavourable changes were observed in smokers only for serum triglycerides and there was a gradient within the smoking habit [44]. There was no consistent pattern of cholesterol change with smoking habits. Erikssen and Enger concluded that smoking can promote CHD development independently of the accepted CHD risk factors.

The characteristics of 200 MI patients as regards age, sex, smoking habits and country of origin, have been analysed by Eisenberg et al (1981) [40]. They found that patients over 65 years were characterised by an overall low-risk profile. Women with MI, especially under the age of 65, were characterised by a high risk profile, although fewer smoked

cigarettes and their high density lipoprotein cholesterol levels were higher than in men. In patients of Asian-African origin, hyperlipoproteinaemia of the type II b variety was more prevalent and the plasma triglyceride levels were higher than in the other patients. The analysis has thus indicated several distinct risk patterns among the study population, in particular plasma and lipoprotein lipid levels were found to be an important predisposing factor for coronary artery disease in young patients, in women and in immigrants from Asian and African countries.

The combined experimental and epidemiological evidence in the past few years strengthens the view that cigarette-smoking is a major risk factor for both fatal and non-fatal MI (161). Cigarette smoking acts both independently and synergistically with other major risk factors such as hypertension and elevated serum cholesterol. In most studies the risk increases in direct proportion to the number of cigarettes smoked, and there is accumulation of evidence that CO plays an important part in the mechanism whereby smoking increases CHD.

The association of cigarette smoking for most of adult life with subsequent development of clinical coronary disease is significant and it was natural that there was a relation between the number of cigarettes smoked and the manifestations of coronary disease identified (107). Acute MI patients were almost six times as likely as controls to be smokers 95% confidence limits 2.8-12.3,$p<0.0001$) (3).

Cigarette smokers have two to four times the risk for CHD compared to non-smokers and the risk is dose related [4]. In the presence of high serum cholesterol and hypertension, the risk is eight times higher. The associations are confirmed by smoking cessation, a substantial reduction in risk for CHD within a few years of quitting and, after 10 years, the risk is almost equivalent to that of non-smokers [4,49].

2.3.1.10 CORONARY HEART DISEASE RISK FACTORS IN DIFFERENT POPULATIONS.

In a study done to compare the risk factors for CHD in Edinburgh and Stockholm men aged 40, Logan et al (1978)[92] found that Edinburgh men were shorter and fatter, had higher systolic and diastolic BP, smoked more cigarettes, drank more alcohol, had more ECG abnormalities and lower exercise tolerance. Edinburgh men also had a higher level of total serum triglycerides but serum cholesterol and low density-lipoprotein cholesterol were similar in both cities. Cholesterol in very low density-lipoprotein (VLDL) was higher and cholesterol in HDL was lower in Edinburgh men and these metabolic factors may provide new information on the CHD pathogenesis.

The study of the coronary risk factors in medical students in Jerusalem participating in a class excercise to determine their own risk factors , showed that systolic and

diastolic blood pressure, body-mass index, and proportion of smokers were all lower in females and gave them a significantly lower estimated coronary heart disease risk function [143]. There was no sex difference for measures of serum cholesterol. Comparison of the Israeli for CHD data with those from three studies of American medical students revealed that Israelis smoke more than their American counterparts but otherwise appear to have similar risk profiles.

In order to determine whether MI in different countries is associated with different risk factors, Dolder and Oliver studied the risk factors for MI in young men aged 40 or less in nine countries [37]. In seven centres in developed countries there was a high prevalence of risk factors, particularly of hyperlipidaemia and cigarette smoking. The prevalence of hypertension, obesity, hyperglycaemia and hyperuricaemia varied from centre to centre. Risk factors were less prevalent in Bombay and Singapore. The most common risks operating in Bombay seemed to be cigarette smoking and hyperglycaemia, while in Singapore cigarette smoking was the commonest. Serum cholesterol levels of 7.25 mmol/l or more were present in 25% of all patients and serum triglyceride levels of 2.26 mmol/l or more in 35%. Eighty per cent of patients were smokers and 15% were either treated for hypertension before MI, or had a raised BP after MI. Obesity was found in 19% of all patients and serum uric acid levels over 0.5 mmol/l in 17%. Ten per cent of all patients were either treated for diabetes mellitus before MI or showed an abnormal

glucose tolerance after MI.

In Egypt (1976) Mahmoud et al (94) studied the effect of smoking on the prevalence of CHD in 2318 males from four social groups, urban, shifted, commuting and rural. In this paper the authors do not exactly define these groups. Higher prevalence (17.4% in urban, 4.7% in the rural) was seen among smokers than non-smokers (12.7% urban and 2.8% in rural). The rate and duration of smoking correlated positively with the prevalence of CHD. They concluded that smoking appears to be among the important factors responsible for an increased ischaemic heart disease with urbanisation in Egypt.

In Puerto Rico, Garcia et al (1978) (55)found that rural men had a lower average BP, serum cholesterol, blood sugar, heart rate and relative weight than urban men. The age-adjusted CHD incidence rate for urban men was 1.5 times that of rural men and among rural areas the most rural had the lowest incidence. He suggested also that geographical mobility may have a role.

2.3.1.11 SMOKING HABITS OF THE FAMILY AND ITS EFFECTS ON CHILDREN.

It is widely accepted that although there is a direct relationship between the cigarette smoking of parents and their teenage children the observed influence is only transitory and does not endure into adulthood (169). However, identification theory would predict the opposite, that a young adult's smoking behaviour should directly relate to his parents' smoking behaviour.

Wohlford,(146) in his study, found that the results support the identification and interpretation of the father's non-smoking pattern, as father-son smoking behaviour was directly related in intact families. The variable of family intactness was a highly relevant moderator of the parent-son smoking pattern.The mother and daughter smoking patterns remained enigmatic. Family involvement was necessary to affect smoking attitudes and behaviour. Peer influence and self esteem were also related to smoking knowledge, smoking attitudes, future smoking intention and the "purchase" of cigarettes.

Despite recognition of the deleterious effects of passive smoking, quantitative information on the intake of environmental tobacco smoke is still lacking. Matsukura et al (1984) (97) examined the relationship between urinary cotinine excretion in 472 non-smokers and the smokiness of their environment. The urinary cotinine levels of non-smokers who lived with smokers were higher than those of non-smokers who did not, increasing with the combined daily cigarette consumption of smokers in the family. The presence of smokers in both the home and the workplace also increased the cotinine levels. Urban non-smokers had more cotinine in their urine than rural non-smokers. They concluded that the deleterious effects of passive smoking may occur in proportion to the exposure of non-smokers to smokers in the home, the workplace and the community.

2.3.2 CIGARETTE SMOKING IN UK

The tobacco industry is made up of some of the richest and most powerful multinational companies in the world (148). The political and economic iterests forms what is called the "Smoke Ring" (148) which has protected the tobacco industry for the past twenty years. Unfortunately governments are themselves part of the smoke ring. The economic interest lies for example in the income from cigarette advertising, tax collection (4 billion pounds) and the huge number of employees in the tobacco industry e.g. 180,000 in EEC countries. These factors may persuade governments that the wealth provided through the tobacco industry could come before health of the nation.

The proportion of adults (over 16) in Britain who smoke cigarettes has been decreasing since the early 1970s (136). Since 1976 smokers have been a minority in the population and the number of adult cigarette smokers is around 18 million.

Prevalence of cigarettes smoking	1972 %	1974 %	1976 %	1978 %	1980 %	1982 %	1984 %
Men	53	51	46	45	42	38	36
Women	41	41	38	37	36	33	32

The prevalence of smoking in Scotland is consistently

higher than in Great Britain as a whole. In Scotland the percentages of adult men and women who were current smokers were 45.6% and 42.0% respectively in 1980 [24].

The number of male smokers has been steadily falling since 1972 when the General Household Survey (GHS) showed it to be 52.7%, whereas the proportion of female smokers remained unchanged [21]. These trends narrow the gap between the smoking habits of both sexes in Scotland. The figures for England and Wales collected in the same way (GHS) show a decline similar to Scotland for men but at a lower level, the figure for 1980 was 42.1%.

Since 1948 the proportion of adult male cigarette smokers in Britain has fallen by about 20% [25]. However over the same period the proportion of female cigarette smokers has shown relatively little change and now there is a little difference between the percentages for men and women. In recent years the downward trend for men has been clear and there is some evidence of a fall for women also. During the 1960's this decline in prevalence was accompanied by the emergence of a marked social class gradient, particularly for men, with the lowest percentage of smokers in class I and the highest in class V. Only women in social class V showed a rise in the prevalence of smoking between 1961 and 1972 - though by 1976 the figure had fallen back to the level for 1961.

SOCIO-ECONOMIC STATUS AND SMOKING.

From many studies it is evident that smoking behavoiur is related to social class. The prevalence of smoking is higher in the lower income groups, moreover these smokers have been found to start smoking at an earlier age. Occupations normally associated with higher education tend to have fewer smokers (103). For example in Glasgow smoking was clearly related to social class, (III Manual, IV, V, 46 % in contrast to 37 % amongst social classes I, II, III Non-manual) (75).

A sample from central Scotland and the Highlands and Islands, studied by Aitken et al, showed that the percentages of male and female smokers were slightly higher than in the General Household Survey (GHS) study (25). They were 54% and 45%. This study also demonstrated geographical variation within Scotland, particularly in Glasgow and Edinburgh. The percentage of smokers in Glasgow was marginally higher than in Edinburgh (2).

In Glasgow, 44% were smokers and the habit was equally common amongst men (45%) and women (43%). There was considerable variation with age , the habit being most strongly established among 35-54 years olds. Cigarettes smokers were 37% in age group 16-24, 46% in age group 25-34, 55% in age group 35- 54, 47% in age groups 55-64 and 24% in age group over 65 (75).

The Glasgow 2000 project is a new antismoking initiative which was launched in 1983 by the City of Glasgow District Council, the Greater Glasgow Health Board, Strathclyde Regional Council and the Scottish Health Education Group [85]. The main aim of this project is to reduce the incidence of smoking related diseases in Glasgow and to move towards having a nonsmoking population by the year 2000. The baseline survey indicated that over 60% of smokers in Glasgow had tried to give up and failed. Glasgow 2000 provides self-help material and courses to train group leaders. Prevention of the uptake of smoking by children has been identified as a major target particularly in primary schools.

In the General Household Survey of 1982, 38% of men in Britain, 45 % of men in Scotland and 45% in Glasgow, and 33% of women in Britain, 39% of women in Scotland and 43% of Glasgow women were found to be cigarette smokers compared with 53% for men and 42% for women in 1972 in Britain [75,133]. The decline in smoking has occured in every age and in every socioeconomic group while this represented a continuously declining trend in men the percentage of women smoking remained between 42% and 44% throughout the 1970s. It was hoped that the 1982 figure of 39% will be the begining of a downward trend amongst women comparable to that amongst men. The downward trend amongst each sex has been mostly in 25-49 age group and has now shifted to the 50-59 age group. The difference in the prevalence of smoking between the two sexes overall has been almost entirely due to the large

difference in the oldest (60+) age group. The prevalence of cigarette smoking amongst women aged over 60 is 20-25% compared with that in men of 38-49%.

The average weekly number of cigarettes consumed per smoker amongst men rose from 130 in 1972 to 142 in 1975 but then decreased to 132 in 1982 (133,136). Amongst women , there was an increasing trend from 95 in 1972 to 116 in 1980 followed by a drop to 111 in 1982. Male smokers in each group in each year between 1972 and 1982 have a higher average consumption than female smokers and the difference is decreasing because of the increase in consumption by women.

In Britain, since the early 1960s about 9 million people have given-up smoking, 65% of them found it easy to do (136).

The general decline in prevalence is due to the greater effect of the increase in the rate of giving up smoking and a decrease in that of taking it up (25). Despite this general decline in prevalence, the total number of cigarettes sold in the UK rose fairly steadily from 1948 to a peak in 1973. However, changing patterns in 1960's, particularly the change from plain to filter cigarettes, resulted in a fall in the total weight of tobacco sold as cigarettes. Since 1973, total weight of tobacco sales have fallen each year, although cigarette consumption per smoker has increased substantially over the last quarter of a century, particularly for women, and more noticeably for the 16-34 age group than for older smokers.

Wald [155] in 1976 compared the changes in the type and quantity of cigarettes smoked in the UK from 1956 to 1971 with changes in the death rates due to lung cancer and CHD from 1956 to 1973. Despite little change in the number of cigarettes smoked, the change to filter cigarettes was associated with a decrease in lung cancer mortality among men aged less than 60 years. In contrast CHD mortality has continued to increase in both sexes but to a greater extent in women. These changes are consistent with the hypothesis that in tobacco smoke, tar is the principal aetiological factor in lung cancer, whereas CO or other gaseous constituents are involved in the development of CHD.

The tar, nicotine and CO yields of most cigarette brands on sale in UK are regularly determined by the government chemist [45]. They have declined progressively since the 1960s but CO more slowly than tar or nicotine. The incidence of lung cancer, bronchitis, and heart diseases are strongly related to cigarette smoking. Evidence suggests that the recent decline in mortality rates from these diseases in men is related to the decline in total consumption and in the amounts yielded by cigarettes.

2.4 PREVENTION OF CORONARY HEART DISEASE WITH SPECIAL REFERENCE TO HEALTH EDUCATION

2.4.1 PUBLIC KNOWLEDGE ABOUT CORONARY HEART DISEASE.

Until recently, public knowledge about the risks of life and health that smokers run has steadily increased through many channels of information, particularly through the media (130). But much ignorance remains, eg. about 30% of smokers deny that they risk harming their health by smoking and many who accept a possible risk consider it applies only to those who smoke more than they do.

Shekelle and Liu, (139) in their study in Chicago, clarified the widespread lack of information about major causes of heart attacks among persons younger than 60 years. Twenty eight per cent named cigarette smoking, 21% named high BP, 13% named cholesterol, 50% did not name any of these risk factors and only 1% named all three. Although 75% believed that heart attacks are preventable, few named specific behaviours would effectively reduce risk factors other than smoking: the prevalence of these responses increased with level of educational attainment. Sustained community education programmes about reducing coronary risk factors are clearly needed. A similar nationwide survey carried out in 1973 showed that 37% named emotional pressure or anxiety, 33% overweight, 29% smoking, 13% high BP and 8% fatty foods. The results were similar to those obtained in the Chicago survey

and suggests that the Chicago findings may be broadly applicable.

In Egypt in a study about the teacher's smoking behaviour and anti-smoking behaviour, the researcher found that the sources of informations related to the health hazards of smoking were, mass media in 91.4%, health experience of self, family and friends 67.3%, scientific researches 45.1%, scholastic experience 8.0%, teaching experience 5.6% and other sources 1.9% (119).

2.4.2 BEHAVIOURAL ASPECTS INVOLVED IN SMOKING.

In 1972 Hetzel defined the environment as both physical and social and he stressed the difficulty in monitoring the social environment in contrast to the physical environment (67). The social indicators include health and illness, social mobility, income and safety. The physical environment includes science, art, participation and alienation. He referred also to 4 different epidemic health poblems in the last 20 years in Australia: traffic accidents, lung cancer, CHD and suicidal behaviour. He emphasised the close connection between these epidemics and patterns of behaviour - alcoholism, smoking, diet and the use of sedative pills.

Children do, however, learn by imprinting, particularly from their parents (110). If the parents live a life that we associate today with reduced risk of CHD, it can be expected

that the children will follow the same pattern and probably will benefit more than the parents. The effects of environment on the smoking behaviour of children was evident from their beginning of smoking at an early age [48]. However the characteristics that distinguish smokers from non-smokers were conformity to a family smoking pattern, patterns of low achievement and patterns of peer group influence. Children usually use cigarette smoking to heighten their emotional state or to decrease negative feelings. Therefore the quality of school programmes influencing smoking as a determinant to health is important, particularly as teaching by example can be more powerful than teaching by precept.

Normally by the age of 20 years, personal identity is reached as a result of a process of socialisation that dates from birth [67]. By that age also a life style is adopted which includes drinking and smoking patterns and these patterns may be due to many factors including birthplace, cultural background and social class. Other characteristics and life patterns, e.g. cigarette smoking, hypertension, hyperlipidaemia, hyperglycaemia and lower level of physical activity, may also affect the pathological changes underlying CHD [7].

The few relevant studies suggest that there are several different patterns of experimenting with, initiating regular smoking of, and habituating to cigarettes [168]. Whether or not an adolescent smokes probably involves a complex matrix of his personal dispositions and social factors, including

his attitudes towards his parents (conformity to, or deviation from their expectations) and his attitudes towards his significant peer groups (conformity to, or deviation from their expectations). Cigarette smoking is significantly related to a wide variety and somewhat contradictory array of personal dispositions, including rebelliousness, extroversion and psychopathic behaviour. Although the relationship in adults between extroversion and smoking has been replicated most often, the relationships between rebelliousness and smoking is solidly supported in longitudinal data regarding the pre-smoking personal disposition.

It has been mentioned also that a number of factors may influence a young person to begin to smoke and they are :- (136)

a) The example of parents, teachers and older brothers and sisters.

b) Pressure from smoking friends.

c) Lack of interest in schools.

d) The general social acceptability of smoking.

The psychosocial forces that account for the initiation of the smoking habit differ from those that account for continuation and habituation, and that even smokers who have indulged equally long and who consume the same daily number of cigarettes may differ from one another in the social and psychological forces that influence their smoking behaviour (70). Among several reasons for taking up smoking, perhaps the most important related to the fact that smoking

among adolescents represent both a symbolic striving for adult status and a rebellion against adult authority [58].

An example of a possible reinforcer which demonstrates that the distinction between social and psychological reinforcers may not be complete is that of the reinforcement of a male adolescent smoker's self-image afforded by smoking a cigarette [102]. Psychological reinforcements in smokers are related to nicotine which stimulates, tranquillizes, sharpens concentration and may facilitate social communication.

The expectation of reward or punishment being a consequence of a particular behaviour may be sufficient to control the performance or otherwise of that behaviour [102]. The act of smoking a cigarette may represent a complex of consequences to the smoker, some of which may be pleasant and some not so pleasant. A smoker may perceive distant health benefits arising from a decison to stop smoking and an immediate increase in the ability to cope with social and personal tensions arising from a decision to continue smoking. The balance of these two positive outcomes may decide whether the smoker continues to smoke or not.

Some personal characteristics were found to be associated with an increased probability of smoking cessation [73,126]. These are: smoking less than 20 cigarettes a day, a report of non-inhaling, use of filter tips, previous attempts at stopping, marital status (other

than married), professional or executive employment grade, and neuroticism. Lower recidivism rate has also been reported in light smokers (73).

Wallace and Wallace in Newcastle (1977) (159) tested the hypothesis that the patients who had had post-coronary education at Inpatients Advice Group (IAG) would have a better psychological adjustment to their post infarct state than those who had none, and that the reinforcing effect of the coronary education group (CEG) would be followed by a better psychological adjustment in most cases compared with patients who had had IAG alone. In fact the reverse happened, due to more anxiety in the CEG group's education. In spite of this drawback the meetings were helpful, particularly in explanation of mechanism, management and prognosis in MI. The authors recommended a further study in this area and group education should be compared with bedside instructions both for efficacy and prognosis generally.

For prevention of acquisition of the smoking habit, three approaches were suggested by Wynder and Hoffmann (171). The approaches were youth anti-smoking programmes to prevent the acquisition of the smoking habit, smoking cessation programmes to help current smokers quit and a less harmful cigarette for those who cannot or will not quit smoking. However, since changes in attitudes, knowledge and beliefs about cigarette smoking do not necessarily precipitate corresponding changes in smoking behaviour, it seems to matter little whether the students are approached by teachers

or peer groups.

Simple recognition of the hazards of smoking is not the total answer because in spite of the evidence of the gravity of cigarette smoking problems, the majority of habitual smokers may continue to do so even if they are patients with acute coronary insufficiency or survivors of MI [95]. Therefore giving up cigarettes is the patient's choice to become a non-smoker. The decision, however, to smoke or not to smoke, is an intellectual as well as an emotional process; you can lead a horse to water but you can't make him drink[162].

A healthy lifestyle programme was introduced by Simons as a part of the Sydney Coronary Heart Disease Prevention programme [141]. The aims were to influence health attitudes, knowledge, behaviour and perhaps risk factors in three main areas (i) nutrition (ii) physical activity and fitness and (iii) alcohol, tobacco and drug use. The attitude and knowledge parameters in girls improved with age, which may be due to home science offered in secondary education. On the other hand, boys showed little improvement.

Prevention and treatment of illness is increasingly a matter of motivating and helping to alter lifestyle and behavioural patterns that place one at increased risk for poor health [71]. For example, cigarette smoking, obesity, excessive drug or alcohol use, carelessness resulting in accidents, are better understood as regards health consequences than those of a diet high in saturated fats and

cholesterol and sedentary or type A coronary prone, time-pressured, lifestyles. A meaningful change in lifestyle can be achieved by behavioural techniques based on social learning theory. Behavioural approaches use 3 strategies to alter the environmental factors that maintain health threatening lifestyles. The first strategy is reinforcement control which involves building skills and rewards for healthful alternatives. The second, stimulus control, involves rearranging social and physical environments and the third, self-control strategies, emphasise personal responsibility for lifestyle change.

2.4.3 SMOKING CESSATION

In order to design a smoking cessation programme, several principles should be considered; [171] (1) scientific character (the method has been developed and evaluated on an experimental basis, using a statistical method of assessment; (2) efficacy (the method has been demonstrated and compared with other therapies); (3) permanence (reliable data exists on recidivism and cure for at least one year); (4) economic-viability (the cost is low relative to the results obtained, makes maximum use of non-professional staff and time); and (5) broad range of application (can reach as many smokers as possible).

A lot of behaviour modification techniques or methods pof giving up smoking were published and they can be summarised as follows:

1) Behaviour changes.
2) Positive rewards for non-smoking behaviour.
3) Stimulus control.
4) Giving up activities associated with the onset of cigarette smoking.
5) Identification of stress associated with the onset of smoking and the use of relaxation to reduce or avoid it [73].

Three kinds of aims can possibly be distinguished in a smoking controlled prevention trial [125]. Does normal modification of a suspected cause lead to a reduction in

incidence? How large is the benefit and what are the risks and costs of the proposed regime? To what extent can smoking habits be changed and by what means? Rose[125] mentioned also the value of the use of a high risk group. First, the reduction in the size and cost of the trial. Secondly, the subjects selected will be receptive to health advice. Thirdly, concentrating efforts on those in special need.

Sivarajam et al found that fewer than half of the MI patients were smoking at the time of admission to hospital [142]. The high rate of quitting and reducing the number of cigarettes smoked per day is similar to or better than those reported in other studies that provided no specific intervention. At three months, 45% of the initial smokers had stopped with 78% of those citing the recent myocardial infarction as the reason for giving up smoking. But some investigators suggest that behavioural techniques applied at the time of MI generally achieve good compliance as long as the subject receives supervision but compliance often regresses after the intervention stops.

In the Glasgow 2000 project, 33% of smokers had succeeded in giving-up. Two thirds of the remaining smokers had tried to give-up, but failed. The main reasons for trying to stop were concerns over health (71 %) and cost (38 %) [75].

The result of the multicomponent behavioural programme which uses a number of approaches simultaneously (social support, smoke aversion, non-smoking skill training and

contingency contracting) are twice as favourable as results achieved with traditional medical and mental health approaches (71).

Most attempts to influence the smoking behaviour of the young have had little success (150). A variety of studies have compared teaching methods and/or message themes but the studies themselves are often not comparable. Probably the most common message theme aimed at youth has been that smoking is unhealthy. Programmes testing the effectivenes of this theme have reported little success. On the contrary, quite promising results have been reported from a prospective study involving adolescent Swiss males. Matched controls were used and evaluation was conducted four years after the start of the programme. Inclusion of smoking education within a comprehensive health education programme was successful in preventing non-smokers from becoming smokers, although it did not persuade smokers to stop smoking. Generally, programmes for adult smoking education have shown mixed results. Anti-smoking campaigns have had little reported effect on smoking behaviour. On the contrary, smoking withdrawal clinics and individual counselling have shown the most success, producing abstinence rates of 20 - 35% one year after treatment. These would appear to be the most consistently effective techniques reported in the literature, but all usually undertaken as self-selected, highly motivated, populations.

Experimental studies which used treatment and control groups fell into two groups: those dealing with emotional

role playing and those involving aversive conditioning. Results were equivocal (150). Roughly one-half of those studied in each group demonstrated a significant difference between the experimental and control group smoking habits. Once the programme was over the other half of each group showed no significant difference between experimental and control subjects.

Behaviour researches clarified that the process of giving up smoking begins with simple awareness of the dangers of smoking requiring decision making immediately to give up and a continued determination not to resume (48).

A community based programme to control cardiovascular diseases was started in North Karelia, Finland, in 1972 and 10 years of a community intervention programme showed among the middle-aged male population, that there was 28% reduction in smoking ($p<0.001$), 3% reduction in mean serum cholesterol concentration ($p<0.001$), 3% fall in mean systolic BP ($p<0.001$), and 1% fall in mean diastolic BP ($p<0.05$) (115).

The natural discontinuance of smoking or the cessation ratio is the ratio of ex-smokers to current smokers plus ex-smokers, that is

$$\frac{\text{Ex-smokers}}{\text{Current smokers + Ex-smokers}}$$

This relation is called the "cessation ratio" (102). In the present social climate in Britain, (1971) it is unlikely

that more than 15% who smoke regularly undergo natural discontinuance to permanent ex-smoking status before the age of 60. Discontinuance tends to occur after the age of 30 and increases with age. The average daily consumption of cigarettes also tends to drop quite sharply afer the age of 60. The natural discontinuance refers to a permanent change of smoking status whereas 'ex-smokers' status per se is not reliable. The shorter the period of cessation the higher the percentage of relapse, eg. 37% relapse among ex-smokers of less than one year compared to 19% of those with 1-2 years' standing and 5% of those maintained for more than 2 years. Six reasons were mentioned also for discontinuance of smoking:

(1) Health: particularly minor ailments being more important than dramatic illness.

(2) Expense is the second motive for adults and the prime for adolescents.

(3) Social influences in the form of pressure from other people.

(4) Example: non-smoking example set by parents, teachers, doctors.

(5) Mastery: giving up smoking as a test of willpower.

(6) Aesthetics: the belief that smoking is nasty and dirty.

Reasons 4,5 and 6 are of comparatively minor importance. These reasons for giving up smoking should be useful to anti-smoking programmes of various kinds.

Another survey was carried out by Hammond (61) in 1958 to clarify the reasons which make people give up smoking. He found that 60% of the respondents to the postal survey stopped because they suffered from physical complaints such as cough. Other 17% mentioned "doctor's orders" as a cuse of giving up smoking. Two years after enrolment in the prospective study, Hammond found a larger percentage had stopped cigarette smoking among those who had been hospitalised or had heart or circulatory disease than among those not hospitalised and those who had not had heart or circulatory disease. He observed also that the longer since giving up the habit the less likely the ex-smoker will be to resume the habit.

A dentist developed a smoking cessation method "Conditioned inhibition" which include writing at least 10 reasons why he or she wants to stop smoking, five times per day (19). Smoking can be stopped in from 1 to 6 weeks and should continue for several weeks following cessation to reinforce the new behaviour.

Hall (60) used two techniques to produce smoking abstinence. The first was health motivation treatment - this included self-management skills, film model, verbal commitment and discussion of the costs and benefits of smoking and abstinence. The second was 'aversive smoking' which includes "normally paced aversive smoking", relaxation training, role playing, high risk situations and discussion of maladaptive thought patterns. He found that there was no

significant difference between the two techniques.

Study of the different approaches used by teachers to change smoking behaviour of school pupils have been done in Egypt as a part of study of the effect of teacher's smoking behaviour and anti-smoking education in schools (119). It was found that 1.2% of the teachers used direct orders, 42.6% impressed students with the hazards of smoking, 36.4% were setting a good example by not smoking, 9.9% provided or suggested educational materials and 8.0% referred students to the appropriate resources and 1.9% used other methods.

In California, a sample of physicians used the following methods to help patients stop smoking and the percentages of cessation were: direct orders 28%; impressing patients with hazards 85%; suggestion or persuasion 47%; tranquilisers 17%; educational materials 16% and a number of other methods from 2 to 14% (Schwartz, 1969) (132). Schwartz mentioned also the standardized criteria for measuring success in smoking cessation programmes which are:-

a) percent reduction in <u>amount</u> smoked.

b) smoking category at the end of the study and follow-up, eg. occasional, one half pack and none.

However, each control study can be evaluated on 4 points;

(1) whether placebos on controls were used.

(2) whether the follow-up was based on at least three-fourths of the subjects.

(3) whether a follow-up was conducted after at

least 6 months.

(4) whether reported results were based on all subjects starting the test or just those available at follow up[132].

In a study carried out by Pozen et al in 1977 [112] to evaluate the effectiveness of a nurse rehabilitator on MI patients, they found that she was effective in increasing the return to work rate ($p<0.05$) and decreasing smoking ($p<0.05$) in a randomised trial of 102 patients with MI. She was also effective in increasing patients' knowledge of heart disease ($p<0.01$). There was also an increase in individual counselling.

Improving the effect of anti-smoking advice has been tested in Oxfordshire in which 35% of 6052 adult patients in a general practice were smokers [76]. Smokers were allocated to one of four study groups - a control group, a group that received verbal and written antismoking advice from the general practitioner, a group that received this advice and also a demonstration of exhaled CO, and a group that received this advice plus the offer of further help from a health visitor. Seventy two per cent of the smokers replied to questionnaires sent after one year. The percentages of those who stopped smoking were 11% in the control, 15% in the group that received advice alone, 17% in the exhaled CO group and 13% in the health visitor group. Validation of these findings by analysis of urinary cotinine showed that between 24% and 40% of subjects may have misreported their smoking

habits but there was no indication that the rate of misreporting was higher in the intervention groups than in the control groups. Therefore anti-smoking advice has a useful effect, but using an objective measurement test may improve the results particularly in lower socioeconomic groups.

Does quitting help? Kuebler (88) agreed that cessation leads to significant improvement in an individual's risk status regarding lung cancer, heart disease and chronic obstructive lung disease. The attack rate was one half that of individuals who continued to smoke in the Framingham heart study. Other stressed that the problem was not how to get people to give up smoking but to get people to remain non-smokers once they had quit (73).

The central and most important obstacle to smoking cessation is the huge amount of money spent in cigarette advertising: in the U.K it is about 100 million pounds in comparison to the very little spent in health education against smoking (130).

2.4.4 CORONARY HEART DISEASE PREVENTION TRIALS.

Louria et al (93)in their analysis on screening and health education concerning primary and secondary prevention, showed that only a small number of diseases were preventable by risk factor modification and, in the majority, the risk factors were alcohol and tobacco.

Any programme directed at the identification of high risk populations or those with early forms of disease and attempting to reduce the number of serious diseases experienced by these groups requires four steps [110].

1. The identification of the high risk population in a primary screening.
2. The successful referral of those found at this primary screening to a secondary screening for diagnostic evaluation.
3. The initiation of appropriate therapy.
4. The creation of a system whereby appropriate intervention is maintained and the situation evaluated periodically.

Considerable attention has focussed on intervention programmes which attempt to modify the relevant risk factors for heart disease. Cigarette smoking was associated only with increased incidence of infarction and not with a higher case fatality rate [49]. In another study carried out by Doyle et al, they confirmed the observations from the Albany and Framingham experience of cardiovascular disease which indicated that men who habitually smoked 20 or more cigarettes daily experienced a risk of MI or death from all causes three times greater than non-smokers or pipe and cigar smokers. On the contrary, there was no relationship between smoking habit and angina pectoris [39].

As a result of this analysis of risk factors a number of intervention trials have been carried out. For example the primary aim of the North Karelia project (Finland) was to reduce cardiovascular disease mortality and incidence rates among the population through a reduction of smoking rates, serum cholesterol levels and elevated B.P (114). In the North Karelia project for males a 10% reduction in smoking rate, 4% reduction in serum cholesterol levels, 4% in systolic B.P. and the respective net reduction of estimated CHD risk was 17% for men and 12 % for women.

The European Multifactorial Preventive Trial of CHD in Belgium, Italy, Poland, Spain and Britain conducted in 44 pairs of factoriesd studied over 63,000 men aged 40 to 59 (100). High risk (upper 15 to 20% men were individually advised on smoking cessation, cholesterol-lowering diet, regular exercise, weight reduction and drug control of hypertension whereas mass education on these topics was given to non-high risk individuals. Changes in cigarette consumption after 4 years varied from -3.9% in Belgium to -15.6% in Britain. The data showed a net reduction of 8.8% in cigarette consumption, 2.5% in serum cholesterol, 1.3% in systolic BP and 0.5% for body weight. The high risk group showed a net reduction of 15.2% for cigarette consumption, 5.8% in serum cholesterol, 2.6% for systolic BP and 0.9% in body weight. Net reduction of coronary risk was estimated at 12% in the random sample and 17% in the high risk subjects.

In order to study the effect of advice to change eating habits and to stop smoking, Hjermann (69) in 1983 carried out an intervention study in 1,232 healthy coronary high risk middle-aged men at high risk of CHD, (the Oslo study) (69). The results showed a significant reduction in the incidence of first events of MI and sudden death. Eighty per cent of both intervention and control groups were smokers at the beginning of the randomised trial. The men had serum cholesterol levels between 7.5 mmol/L and 9.8 mmol/L, coronary risk score (based on cholesterol, smoking and BP) in the upper quartile of the distribution and SBP less than 150 mm Hg. Cholesterol was lowered by 13% triglycerides by 20%, tobacco consumption by 45% in the intervention group compared with controls. At the end of the Oslo study, the incidence of MI and sudden death was 47% lower in the intervention groups than the controls.

The Veterans Administration Clinical Diet Study showed that subjects receiving a diet high in unsaturated fat had a 24 % lower incidence of myocardial infarction and sudden death and 41% lower incidence of strokes (158). In another study cigarette smoking was found to be related to mortality from Coronary Heart Disease independently of all other risk factors (48).

The Multiple Risk Factor Intervention Trial (MRFIT) was a 6-year clinical trial to investigate the effect of reducing

cardiovascular risk factors, elevated cholesterol, hypertension and cigarette smoking in a group of asymptomatic men at high risk (73). The programme provided maintenance for those who stopped smoking and an extended intervention for those unable to quit initially. At the end of the first four months which correspond to the end of smoking intervention phase of the study there was a forty-seven per cent rate. After 4 years the quit rates were strongly associated with the initial level of smoking with lighter smokers reporting a higher quit rate.

The example given in MRFIT indicates that attempts to change the risk factors of individuals involves great effort (127). Rose (39) suggested a third preventive strategy intermediate between primary and secondary prevention. It would start with population screening for signs of early myocardial ischaemia, mentioning that early ischaemia can be diagnosed by positive response to validated questionnaires on chest pain and by positive resting ECG (Q/QS,ST segment or T wave abnormalities or LBB block). He also clarified that a self-administered questionaire was almost as effective as an ECG in predicting risk of CHD death. One must also consider the problem of screening for ischaemia which arises when the criteria for a case are set wide enough to give warning of high numbers of future heart attacks. Only a minority of positive cases will actually face serious trouble in the following few years. Stricter criteria can reduce false alarms but bring reduced benefit to the community.

Secondary prevention of CHD would consist of treatment of angina or coronary insufficiency in order to prevent MI or sudden death [93]. Hypertension is associated with both. Increasing cholesterol concentration increases the incidence of angina and coronary insufficiency. Reduction in cholesterol in uncomplicated angina reduces the subsequent major coronary events. Smoking is less strongly associated with coronary insufficiency and is not associated with angina.

Epidemiological research has demonstrated a phenomenon known as "tracking" [141]. It means that for a given variable an individual tends to remain in the same part of a distribution curve, even if in time the entire distribution has moved in one or another direction. For example , children with relatively high blood pressure or high serum cholesterol levels will be likely to retain the status into adult life, while the actual values increase still further. On this basis, many children might be already manifesting the earliest precursors of CHD.

2.4.5 METHODS USED FOR CONTROL OF SMOKING AND PREVENTION OF SMOKING-RELATED CORONARY HEART DISEASE.

The major diseases that are likely to be susceptible to primary prevention are those in which the risk factors are known, prospective studies have provided estimates of attributable risk, the data appears reasonably convincing and

the evidence is strong enough (93).

Primary prevention of coronary heart disease is an essential and readily adaptable component of a comprehensive school health program (166). By combining screening for disease risk factors, giving children their results in a "Health Passport" and providing innovative learning activities in a health education curriculum, children can be motivated to assume responsibility for their own future health and modify their lifestyles in a direction of reduced risk for disease. The initial phase of, "Know Your Body" programme has three main components :a) health screening, b) feedback of results, and c) health education. It has been demonstrated that this approach to primary prevention of chronic disease is welcomed by students, parents, and educators. Whether or not the strategy for behavioural modification of lifestyles will be successful enough to demonstrate reduced risk factor profiles attained and sustained through adolescence will constitute the focus of effort over the next few years.

The positive results of primary risk factor intervention trials of CHD and the partial explanation of declining trends of coronary mortality in some countries, represent the theoretical basis for justifying a nationwide preventive action against CHD, including a Mediterranean-type diet, directed to lower and to maintain low levels of serum cholesterol; the drug treatment of high blood pressure and the elimination of smoking, can warrant measurable reduction

of incidence and mortality from coronary heart disease (101).

Watkin (160) in his study about the efficacy of primary prevention of CHD by multiple risk factor modification suggested that primary prevention may be more effective if efforts begin early in life before behavioural and physiological characteristics that may increase the risk become established.

In another study carried out by Rost (1982) it was stressed that smoking cessation should be used for the secondary prevention of MI due to the increased risk in post-infarction patients who continue to smoke (51,129). The main objectives of secondary prevention are: (1) intervention to change some behavioural patterns (ie. diet, smoking, physical training, control of weight and stress); (2) medical treatment of internal risk factors (ie.hypercholesterolaemia, diabetes, hyperuricaemia; (3) medical treatment of CHD complications and (4) medical intervention on possible mechanisms of infarction (133). In order to reduce the health hazards and death that result from smoking a programme should have three main objectives:(48)

A. To encourage young people not to start smoking.

B. To reduce the number of current smokers.

C. To encourage the development of less hazardous cigarettes.

This can be achieved by different means, of which the most important are (48):

1. The education of young people not to take up smoking.

2. To obtain the cooperation of health workers in influencing the public.
3. To control cigarette smoking among adults by group approaches.
4 To control cigarette smoking among the public by mass approaches.
5. Restriction of advertising and promotion of cigarettes.
6. To find ways of making smoking less hazardous.

A programme accomplishing a little under each of these categories would probably be more effective than one accomplishing a great deal under one but neglecting the other methods (48).

In the Annual Health Conference of the New York Academy of Medicine in 1974, Terris (130) recommended legislative measures to control smoking, including the prohibition of cigarette advertising, a massive campaign of Health Education and taxation. The tax funds should go to educational and other programmes to prevent smoking. Another recommendation suggested that taxes raised on tobacco should be an indication of moral disapproval rather than a method of raising revenue (63). Prohibition of smoking on trains, in TV interviews and hospitals is an important preventive measure. Taxation has failed to control the degree of smoking. Anti-smoking clinics also in their long term-results are ineffective and disappointing.

In June 1981, the Egyptian government passed an antismoking law, containing the following provisions: (109)

1) No cigarette or tobacco containing more than 20 mg tar to be imported, exported or produced.
2) Tar and nicotine levels must be shown on cigarette packs.
3) Advertising to be banned in public places.
4) No smoking policy to be adopted for public transport.
5) Penalties to be introduced for those breaking the law (109).

Many smoking cessation programmes have been done, however Cummings (35) noted that commercial cessation programmes offering multifactorial approaches often achieve higher success rates. However Pederson (111) found in his study that the commercial self-help manual was not an effective non-smoking cessation aid. The cessation rate in the treatment group was 16.7%, and in the control group 25.6%.

Unfortunately, many anti-smoking programmes have not included evaluation components so that it is difficult to determine the relative effectiveness of the various approaches (171). However, the existing evidence clearly indicates that smoking programmes that merely provide information about the consequences of smoking are not able to decrease the proportion of those who become smokers (63,171) On the other hand, approaches that take into account the underlying cause of the acquisition of the smoking habit and

teach smokers to cope with various social and psychological factors that promote smoking do seem to be capable of producing behavioural changes and thus seem to be the most promising.

The best place for preventive intervention would seem to be the primary health care system, which has almost complete access to a country's population [43,84]

2.4.6 SMOKING AND RESPIRATORY SYSTEM.

A larger percentage of cigarette smokers reported cough and shortness of breath than men who had never smoked regularly (61,107). Both of these complaints were reported by a smaller percentage of ex-cigarette smokers than of non-smokers.

The above findings have been observed also in a randomised controlled trial of smoking cessation carried out by Rose and Hamilton (126). They found that 51% of the intervention group were not smoking after a one year follow-up compared to 10 % in the non-intervention group. Men in the intervention group showed a decline in the prevalence of sputum production and dyspnoea (172). Thus it is clear that this complaint diminishes quickly when a cigarette smoker gives up the habit and also shortness of breath diminished gradually with time after giving up.

It is evident that the lung parenchyma does not revert to normal after giving up smoking but thereafter it does not progress so rapidly to a more advanced distinctive stage (61). Also, ventilatory function did not improve after giving up smoking but its rate of decline was markedly slowed (126). Rose and Hamilton concluded that smoking cessation in middle-aged men improved the symptoms and progress of chronic bronchitis but that the reversibility of the effect of cigarettes to the smoker's life expectancy may have been over-estimated in observational studies (126).

2.4.7 HEALTH EDUCATION AND SMOKING.

The scientific evidence concerning the health risks of smoking first began to achieve prominence in 1955 (130). The Royal College of Physicians published its first report in 1962 about the health effects of smoking. Publication of the first report was followed up by a 5% decrease in cigarette sales. A slow recovery followed later at the rate of one per cent per year. The findings of the report were accepted by the government and this led to the start of publishing information about the harmful effects of smoking. The report also resulted in banning of direct cigarette advertising on television in 1965 and the setting up of the Health Education Council and Scottish Health Education Unit. The second report in 1971 was followed by the setting up of the organisation -Action on Smoking and Health (ASH).

The report issued by the Royal College of Physicians in 1983, " Health or Smoking ? " drew particular attention to opportunities for further initiatives in health education (13).

The trends in the prevalence of smoking indicate that health education has already had considerable success in reducing smoking in Britain (133). The decrease has been greatest among the better educated, also more among men than women. Many expert bodies, including The Royal College of Physicians of London, WHO Expert Committee and the Secretary

of State for Scotland, have emphasised the importance and the priorities of anti-smoking health education in prevention of disease.

The pessimistic view that the modification of behaviour is not possible in large groups whose members are not stimulated by personal illness does not appear to reflect the actual situation. In New York the prevalence of the major risk factors for coronary atherosclerosis in 1974 among white males is inversely related to social status and the percentage of cigarette smokers among blue-collar workers (45%) has decreased compared to five years before [110]. These differences reflect the effect of community educational programmes on cigarette smoking and dietary habits.

The Scottish Health Education Co-ordinating Committe produced a report in 1983 entitled, "Health education in the prevention of smoking related diseases", which also underlined the importance of health education as the most helpful approach in the control and prevention of smoking and its related diseases, [125,134] especially chronic bronchitis, the leading cause in Britain of sickness absence and of premature disablement [125].

Cohen in 1979 [90] stated that the possibilities for meaningfully influencing health through education and individual decision making have been severely curtailed. He stated also that many of the major causes of disease would be unaffected by lifestyle changes, eg. workplace, stresses,

socioeconomic forces and environmental pollutants.

Fletcher and Horn in 1970 [48] in their report, recommended that an action is required by the health authorities in collaboration with the education authorities to clarify the health hazards of cigarette smoking; they specified that the tasks to be undertaken are:

1) Establishment of statutory upper limits for various constituents of cigarettes .
2) Reduction of advertisements with a view to its eventual elimination.
3) Health workers should set an example bynot smoking and discourage young people from starting to smoke.
4) Health authorities should discourage cigarette smoking in medical premises and establish anti-smoking counselling services in hospitals.
5) Health authorities should help in preparation of health education programmes for schools, teacher training institutions curricula and teaching materials dealing with the health hazards of smoking.
6) Research on effectiveness of health education designed to discourage cigarette smoking and better use of existing methods.
7) The hazards of smoking should be included as a specific part of occupational health programmes in places of employment.

Many recommendations were published in the report of the working group on "Inequalities in Health" in respect to the problem of smoking in Britain [120]. They recommended that the government should sponsor an enlarged programme of anti-smoking health education and it should be started in schools. This programme would be a joint responsibility of local education authorities and health authorities. They also recommended stronger measures to reduce cigarette smoking. These included:

a) Legislation to phase out advertising of tobacco products and banning of sponsorship of sport by tobacco companies.
b) A regular annual increase in duty on tobacco products.
c) Health warning to appear on all tobacco packets.
d) Provision of non-smoking areas in public places.
e) Anti-smoking counselling services to be made available in all health districts.

Since 1978 the anti-smoking strategy of the Scottish Health Education Group (SHEG) has moved from the previous negative message of the dangers of smoking to the positive promotion of non-smoking by emphasising its benefits [133].

Health education was the main recommendation of the workshop conference held at the University of Kent for preventing CHD [170]. The main recommendation concerning health education was to establish a health education service in all district health authorities. Advice should include both health risk advice and ways of improving health. One

member of the district health education team should be allocated to develop local community action. Schools are a powerful force for health promotion. The school health education officer should review school meals and staff smoking and teaching. Employers should provide exercise facilities for staff and implement healthy eating policies in their canteens.

The most visible parts of the anti-smoking campaign carried out by HEC and SHEG are the mass media advertising and associated activities, eg the "Look After Yourself" campaign by HEC, the "Dying Scotsman" by SHEG, the "Superman campaign" (1980-1983) by HEC and the "Go for Goals" campaign [130]. In addition to the National Health education agencies major contributions have been made by the BBC and the independent broadcasting companies. The number of smoking withdrawal clinics increased from 20 in 1962 to 49 in 1980 but their direct contribution to cessation rates is small.

Sport and art sponsorship is an attractive form of promotion of tobacco [148]. In this way the tobacco companies associate cigarettes with a healthy life and create good-will among the public. Tobacco sponsorship is designed to change the public perception of cigarettes and companies who make them. Therefore the consumer is a crucial link in what has been described before as the " Smoke Ring". The activities of ASH, the only specialist organisation, have done much to counter the promotional activities of the tobacco companies.

The greatest tribute to ASH's work has come from the tobacco industry [130].

Taking into our consideration the importance of childhood in combating smoking, Wetzler (1979) [90] mentioned that the most effective primary prevention should begins in childhood because it is easier not to adopt a habit than to alter it after it has been established. Wetzler mentioned also that health education must not be restricted to lifestyle strategies and the core should concern itself with health and all its determinants.

Between a half million to one million children annually have been influenced by the school's council and health education council projects [130]. Instructions regarding the health hazards of smoking are now provided in secondary schools as a component of general health education. The support of the Department of Education and Science in 1981 for health education has given a marked impetus to the promotion of school health education. Examples of school health education projects include the "My Body" project in Sheffield, and the "Jimmy" project in Glasgow, both done for the 10 - 12 year age group. Evaluation of the "My Body" project has shown that its use leads to major gains in knowledge, favourable changes in attitudes and considerable influence on parents.

Health education programmes at local level are systematic attempts to change smoking behaviour within a

localised population [102]. However, Bradshaw, in a review of smoking control had this to say about health education undertaken to control smoking, " For the health educator the solution to the problem of control is as simple as it is obvious; disseminate the facts about the health hazards associated with cigarette smoking and the facts should speak for themselves. They do not always do so is evidenced by the negligible long-term effects of the considerable publicity given to medical opinion in the United Kingdom during the 1960's"

In order to compare methods of health education against smoking, Louria et al [93] mentioned that the impact of fear is often clearly superior to attempts at non-anxiety producing persuasion in reducing use of tobacco, automobile safety and immunisation programmes [142]. It has been mentioned that the techniques for changing risk factors are different from treatment in conventional medical practice. In order to increase the motivation of the participants, it is necessary to introduce the fact that something is wrong with the subject [164].

Health education through mass media and counselling by physicians has been the chief tools in the medical care sector's response to lifestyle health-threats [71].

In health education, mass communication needs to be tempered with an appreciation of its strengths and weaknesses [48]. Mass communication reaches large numbers of

people and an individual will act on it if he is directed by an experienced person. This is one reason for the importance of obtaining active support of health workers to reinforce mass media campaigns on the effect of smoking on health. Reducing the effectiveness of the promotion of cigarettes on television and radio is another approach. Printed advertising also is largely neglected and some control might be placed on its content. For those who will continue to smoke, establishment of limits to the amounts of various harmful substances permitted in cigarettes particularly tar and nicotine is a priority. As regards group approaches to combat smoking,few attempts to reach smokers have been carried out and they are limited to the presentation of posters, leaflets,lectures and films designed for anti-smoking purposes.

While myocardial infarction patients are in hospital (and there by inhibited from smoking) the time may be ideal to inform them of the risk of smoking and to provide specific coping strategies to face the situations when they feel the urge to smoke or when they subsequently meet others who smoke (51). The particular effects of CO and nicotine on persons who have already had an MI can often be explained to help make them quit. The value of counselling while patients are in the hospital is demonstrated by a study in which 66% of MI patients remained abstinent at 4 months.

In another study it was reported also that a significant proportion of coronary patients can be persuaded to give up smoking while in hospital and to remain non-smokers for at least a year or more [22,64]. Thus, nearly a third of the infarct patients gave up completely. Furthermore, the proportion of those smoking more than 20 cigarettes a day dropped from at least 37 to 6%. Perhaps the more dramatic illness of cardiac infarction induced a greater determination among the infarct patients to accept advice to stop smoking .

There are two main types of communications from doctors and other medical workers to patients [91]. One kind is designed to give the patient <u>information</u> about his condition, its treatment prognosis, etc., while the aim of the other types is to <u>persuade</u> the patient to follow a prescribed course of action such as taking medicine, dieting or looking after children in given ways. The criteria for judging the success of the first type are (1) has the message been understood? (2) does the patient feel satisfied that he has been adequately informed? In the persuasive communication the main success criteria are (1) has the message been understood and (2) is the advice carried out? This problem of patients' satisfaction with communications has been investigated. The results show that the percentage of dissatisfied patients ranges from 11 to 65%. Dissatisfaction in a group of studies varies with year of survey, length of time between discharge, and follow-up.

Evaluation of individual antismoking campaigns showed that none has ever produced more than a transient effect on smoking prevalence and consumption [130]. Their value lies more in drawing attention to the smoking problem.

In 1984, Jenkins et al studied patient's evaluation of a teaching programme administered by nurses [80]. Patients admitted to Coronary Care Unit (CCU) with confirmed MI were given a detailed information session by nurses, information included in detailed programme of exercise during convalescence and attitudes towards diet, smoking, sexual intercourse. Patients were asked to assess the value of these sessions using questionnaires administered during convalescence. Between 76% and 91% of patients considered the presentation necessary and there was a significant improvement in their understanding of what an infarction was. More than 80% of patients found a booklet entitled "Recovering from your Heart Attack" particularly helpful. Most of the patients believed that more health education should be given at school and that members of the general public should be taught the techniques of cardiopulmonary resuscitation. The teaching programme appears to have reduced the admitted smoking habits of patients. On admission 56% of patients were smokers. This fell to 7% at the end of 3 weeks but had risen to 17% at 6 months.

Communication frequently fails to produce the desired effects [91]. Failure in understanding contributes to the failure of information-giving communications. Forgetting is

inversely related to the amount presented and the relationship between amount presented and amount forgotten is linear. Communications from doctors to patients frequently fail due both to failure to understand and failure to remember.

In contrast, Jamrozik (76) mentioned in his study that anti-smoking advice from a physician is useful and it can have an effect on smoking cessation rates. Demonstrating the immediate personal and harmful effects of smoking with measurements of exhaled CO may enhance this effect, particularly in lower socioeconomic groups. Anti-smoking advice from a physician is also expected to reduce the risk of post myocardial infarction mortality by 20% (28).

The British Royal College of Physicians is battling to curb smoking through education and legislation (88b). Information on the harmful effects of smoking is dissiminated by health education groups to the public through the mass media and to adolescents through classroom instruction. The two main areas where action could make a difference are taxation and advertising. The ban on television advertising of cigarettes, enacted in 1965, is still the only legislation on the issue; other forms of advertising are uncontrolled or are a matter of voluntary agreements.

In a study to assess the effect of television in reducing the habit, smokers were recruited from about 4,500 persons exposed to stop smoking posters at worksites for 2 weeks before the broadcast of a special series. Smokers who

were willing to participate were given program titles, dates, and times of the broadcasts and a copy of the booklet associated with the series, and asked to complete a questionnaire on smoking habits. Only 3.4% of smokers in the total group chose to participate. A follow-up of 62 heavy smokers for 12 months showed that the BBC television series "So You Want to Stop Smoking" helped 9 to quit, a cessation rate of 14.5% [111a]. It is noted that the cessation rate is similar to those reported for clinic and physician intervention programs.

Health care leaders should set an example and pay attention to discouraging smoking in the community. In this respect the deans of several schools of public health called for discouragement of cigarette smoking as one of the most cost effective health initiatives [60a]. They suggested that a ban could be initiated in most health institutions provided that the officials involved actively discourage smoking. In this case if such a policy is adopted at just one institution it will encourage others to do the same. It will also show the public that heath care leaders oppose smoking.

The marked decline (26%) in cigarette consumption in the UK over the past 10 years is considered one of the success of preventive medicine [17a]. On the individual level, physicians can be very effective in smoking intervention by giving: advice to quit, a leaflet, and a request for a return visit for follow up. By these efforts from GPs about 25 patients per practitioner become non-smokers each year. In

each district, the health authority should have antismoking policies for all hospital wards, and a ban on cigarette sales in hospital shops (17a,60a).

The use of tobacco substitutes (nicotine chewing gum) as an aid for cessation of smoking has been shown in the specialized smoking withdrawal clinics (88a). Evaluation of different programmes to stop smoking shows that one year cessation rates for programmes that included psychological treatment were 38% for nicotine gum vs. 14% for placebo gum. The rates for group therapy were 31% vs 14%. Nicotine gum programmes were more successful when it was prescribed by a physician along with advice to quit smoking (10% of 1,550 patients were abstinent at 1 year). Nicotine chewing gum was used to allay nicotine withdrawal symptoms rather than to provide a pulse of nicotine-induced pleasure. Side effects were present in up to 55% of the users, and included bad taste, nausea, sore throat, flatulence and heartburn.

In another study in which nicotine chewing gum was used, 55 smokers recieved individual counselling and Nicorette- a nicotine containing chewing gum (150a). After instruction, smokers adjusted the use of Nicorette personally, depending upon the craving for tobacco and abstinence symptoms. Smokers attended five control consultations over a three months period. After one week 51 (93%) had stopped smoking. After 3 months, 33 (60%) remained abstinent, and after 6 months, 27 (49%) were still not smoking. Cessation was checked by measuring breath CO content in expired air. Among 27

untreated controls, 3(11%) had stopped smoking after 6 months. Nicorette was gradually withdrawn after 3 months, and after 6 months, all patients had stopped using the gum. The results showed that Nicorette was well tolerated and only a few mild side effects were observed. Young men with a higher carbon monoxide content in expired air, and higher nicotine dependency scores showed a significantly lower frequency of breaking the smoking habit.

A study for comparing the use of nicotine chewing gum and acupunture methods for giving up smoking was carried out in general practice. Six hundred out of 35,000 smokers contacted agreed to participate. They were separated into three groups 1) acupuncture 2) nicotine chewing gum, and 3) controls (29a). The self-reported abstinence rates after 1 month were 19%, 22% and 8% respectively, but results on 270 subjects at 12 months follow-up showed a decline in success rates to approximately 6% for all groups.

The effect of a smoking cessation kit has been evaluated following a television programme on the health hazards of smoking. 4,492 subjects requested a postal smoking cessation kit (34a). A one third random sample was chosen as controls and did not recieve the kit. Among 2,117 (47%) who responded to a 1 year follow up questionnaire, the reported cessation rate for those who recieved the kit (16%) was higher than for the control group (11%, $p<0.001$). Verification with salivary cotinine measurements reduced the difference, but kit users still had a higher cessation rate than controls (9% vs 7%,

$p<0.05$). Further study of nonresponders indicated that the likely effect of the kit was to induce 2% of recipients to give up smoking for at least 1 year.

In addition to the previously mentioned smoking cessation methods, hypnotherapy has been used in 683 smokers. They recieved one session of hynotherapy (30 minute consultation followed by relaxation) during which patients may detach themselves and view their quitting with composure (131b). The hypnotherapy was to reduce the conflict and anxiety surrounding abstinence so that subjects will not smoke again. During the consultation, the damaging effects of passive smoking to children and spouse were mentioned, but to avoid further anxiety, none of the other effects of smoking were pointed out. During induction, patients were told they will be calmer about quitting and the decision to quit will be accepted and tolerated more readily. It was noted that hypnotherapy confers no side effects, and relapse was more often due to regression under duress rather that treatment failure. It was suggested that additional follow-up sessions would probably improve the success rates.

A "Group-dynamics" approach in which 10 members meet once/week for 5 weeks with an interdisciplinary group of consultants (administrators, cardiologists and respiratory disease specialists) was described in a study in France (19a). Before the first session, questionnaires about smoking habits were completed by patients in the clinic. Data were given to each consultant and discussed with patients at the

first meeting. The group method allows for a progressive relationship to develop among patients and therapists, therefore each patient takes an active part in the therapy. Success depends on the smokers who must be made aware of their habit, their attitude towards smoking, and the risk factors involved in continuing. In addition, health prctitioners transmit their convictions to the patients.

The role of physicians in health education is essential and they have to be the primary health educators. Counselling for smoking cessation should be one of their most natural tasks in view of the deleterious health consequences of smoking (62a). By contrast in practice physicians appear to lack knowledge of effective smoking cessation techniques and have inadequate training in counselling skills. The use of a combination of different techniques when effectively designed and implemented by properly trained physicians seems to be efficient in reducing the number of smokers.

In general physicians can be influential in convincing patients who smoke to quit (43a). Smoking behaviour and cessation techniques were discussed in terms of a third generation health decision model that combines health beliefs, decision analysis, and behavioural decision therapy. It was suggested that physicians trying to produce smoking cessation should emphasize factors such as health beliefs, self efficacy, social support, and reduction of stress. In absence of a clear-cut advantage for any particular smoking cessation technique, physicians were encourged to provide

advice about smoking cessation as a regular part of every patient visit.

The role of primary health care, and particulartly the general practitoner, in health education has been acknowledged only recently [48a]. General practice provides a good basis for health education because family doctors are regarded as particularly reliable sources of information on health matters. Consultation with doctors frequently arise because of fear of disease and this may help to motivate behaviour changes. Most importantly, general practice has access to virtually the entire population. Communication is in the form of a one to one method of teaching which is widely recognised as the most successful, especially when communication is two way process.

One study showed that the intervention programme administered by general practitioners was effective in helping patients to stop smoking [120b]. In this study two hundred cigarette smokers who attend a general practice were allocated to either a treatment (n=100) or a non-intervention control (n=100). After the initial visit treatment constituted of an educational consultation and four follow up visits. The smoking status was assessed biochemically at six months and three years. 35 patients in the treatment group were abstinent at three years compared with eight in the control group ($p<0.001$). The success rate of the intensive treatment programme (36% at three year follow up) compares favourably with other programmes administered by general

practitioners in which success rate ranged from 3% to 23% at one year follow up.

In spite of the best endeavours of doctors in giving verbal advice many patients fail to remember much of what they are told (48b). Study patients were able to recall only 50% of the information given. Failure of comprehension and memory may be remedied to some degree by supplementing the spoken word with written information. In general it seems that general practitioners were reluctant to distribute literature (7%), compared with the 40% of health visitors who did so.

The role of general practitioners in Australia has been demonstrated in a smoking intervention programme carried out in general practice. In Australia, approximately 7 million people between 16 and 65 years of age visit their general practitioner at least once a year. About 37% of men and 30% of women between 25 and 65 years of age were smokers (120a). Of 100 smokers in the intervention programme, 36 were abstinent at 5 weeks, 30 at 3 months and 33 at 6 months. Reported abstinence was checked by blood tests for cotinine, carboxyhaemoglobin, and thiocyanate. Explanation of the results of lung function and blood tests in relation to risks of cardiovascular and respiratory diseases constituted a strong incentive to stop smoking. In general it was estimated that if one quarter of general practitioners in Australia adopted smoking intervention in their practice, approximately 150,000 new abstainers could be expected each year. The

corresponding figures in Britain, if all GPs adopted smoking intervention programmes, would be about half a million exsmokers a year (131a).

In Britain 2138 cigarette smokers attending the surgeries of 28 general practitioners (GPs) in five group practices in London were allocated to one of four groups (131a): group 1 comprised non-intervention controls; group 2 comprised questionnaire only controls; group 3 were advised by their GP to stop smoking; and group 4 were advised to stop smoking and given a leaflet to help them, and warned that they would be followed-up. Adequate data were available for 88% of patients who were followed up at one month, and 73% at one year. Of the people who stopped smoking most did so because of the advice. This was achieved by motivating more people to try to stop smoking. The effect was strongest during the first month but still evident over the next three months and was enhanced by the leaflet and warning about follow-up. The proportions who stopped smoking during the first month and were still not smoking one year later were 0.3%, 1.6%, 3.3%, and 5.1% in the four groups respectively ($p<0.001$).

The face-to-face cessation advice from physician may be persuasive, especially in the less well educated majority. During repeated examination for certification as medically unable to work, 500 claimants were asked about smoking habits and whether they had recieved advice from their doctors to quit (39a). Of those with smoking related illness, (72%) had

been advised to quit, with complete or partial success in 72%. Of patients whose illness was not related to cigarette use only 37% had been advised to quit, with some success in 23%. Although advice to quit may be less successful in patients who do not have smoking-related illness, it is still worthwhile.

Increasing attention is being paid to health education as a health care strategy, and more emphasis is being placed on its practical application. But the question frequently asked is, "Does it work?" (48c). Evaluation of health education should ideally measure outcome, but, except in certain circumstances, it may be confined to process measurement of the extent to which relevant objectives are achieved. Fowler clarified also that the health education approach is more effective than group methods, and that the least effective strategy is one involving the relatively impersonal mass media. This is not to deny the importance of mass media in health education for setting an agenda and in providing a fertile environment for the more individual approaches.

A variety of studies have investigated the effectiveness of general practice as a setting for health education using group and individual methods and of the general practice consultation as a medium for it (48c). Lecture-discussion was one of the earliest descriptions of health education in general practice but there was no formal evaluation. The most rigorous studies of health education

have investigated its effectiveness as part of the normal consultation and have been concerned with smoking cessation. The educational effect of demonstrating to smokers the level of carbon monoxide in their breath appeared to be a valuable smoking cessation technique in patients of lower socio-economic groups, those who are otherwise least likely to respond to educational intervention (48c).

In USA, the public health strategy is to facilitate and maintain smoking cessation, including those used in community trials, physician practices, worksites and selfhelp methods (106a). Community trials have demonstrated that the media are an efective tool for creating an atmosphere for change, but that sucessful change occurs only when (1) a large variety of approaches are available in a community on many different levels,(2) interpersonal and intensive approaches are included, and (3) members of the community are involved in the development and delivery of intervention programmes. Physician delivered intervention can potentially affect a large segment of smokers. Worksite programs have been supported by management to reduce health care costs. Self-help strategies may be the most cost-effective approach, but results from self-help intervention vary widely. It appears that face to face interpersonal interaction is an important programme element for many smoking cessation programmes. An integrated approach (a variety of techniques in many different settings within the smoker's natural environment) seems to hold the best promise for reducing the number of smokers in the USA.

2.6 CARBON MONOXIDE : PHYSIOLOGICAL AND PATHOLOGICAL CHARACTERISTICS IN MAN.

2.6.1 SOURCES OF CARBON MONOXIDE.

Aronow [12] clarified that carbon monoxide is the most abundant air pollutant in our environment. It is produced in large amounts by incomplete combustion of carbonaceous materials used as fuel for transportation and heating. Tobacco smoking, industrial processing and refuse burning also can produce carbon monoxide (CO).

Carbon monoxide is present in concentrations of 1-5% in the gaseous phase of cigarette smoke and carboxyhaemoglobin (COHb) levels in smokers vary from 2-15% depending on the amount smoked, inhalation and time elapsed since the last cigarette was smoked [161].

In Los Angeles, Curphey et al found in their post mortem studies that the main source of CO is automobile traffic and CO patterns tend to be inversely related to seasonal mean temperature cycles [36]. The curve for ambient CO concentration level was more approximate in shape to the CO level of non-smokers but the smokers' level varied widely. They found also that the median value for male non-smokers was greater by a factor of two than for female non-smokers.

2.6.2 PHYSIOLOGICAL CHARACTERISTICS OF CARBON MONOXIDE.

Claude Bernard was the first to point out that CO produces hypoxia through its reversible combination with blood to form carboxyhaemoglobin (COHb) (122). Smoking is the commonest source of small amounts of carbon monoxide. The alveolar air of smokers is said to contain only a small amount in the morning before smoking and this concentration is reported to rise steadily with the smoking of each cigarette or cigar. Carbon monoxide is present also in non-smokers blood under conditions where CO is not present in the inspired air (121,122). The explanation of this is that endogenous CO formation occurs in man and is increased in haemolytic conditions.

The proportion of CO combined with myoglobin in man is estimated to be only 5% of that combined with haemoglobin of the circulating red cells when the distribution of CO in the body has revealed complete equilibrium (95,122).

In order to study the direct effects of CO on cardiac function, Takano et al (147) used a haemoglobin-free living rabbit treated with perfluorochemical blood substitutes. They administered gas mixtures of oxygen with 5%, 10% and 20% of CO or nitrogen via a respirator. They found that the CO and nitrogen effects were significantly different as regards changes in arterial pressure, pulse pressure, heart rate, product of heart rate and systolic arterial pressure, in spite of the same Oxygen tension in the inhaled gases.

Therefore they concluded that CO has a direct effect on the heart not mediated by haemoglobin.

Carbon monoxide increases cardiac output by 16% during severe chronic CO poisoning [51,122]. It is reported also that it produces cardiac lesions in dogs at COHb values which are considered safe for men [122]. In addition, nicotine increases its pumping action imposing a great burden on an already damaged and failing heart [126].

It seems clear that during the early part of exposure to CO most of this gas reaching the alveoli combines rapidly with haemoglobin in the pulmonary circulation and 10% to 20% fails to diffuse into the blood because of the very low CO pressure (PCO) in the alveoli [122]. This fraction is exhaled together with the gas in the physiological dead space. At rest the concentration of CO in the expired air is about half that in the inspired air. During exercise the increased physiological dead space, and especially the reduced time allowed for diffusion, result in the expired air containing 60% of the amount of CO inspired. As the exposure continues and the blood CO content rises the PCO increases in the blood and alveoli. This decreases the CO gradient and diminishes the rate of uptake.

In a study done by Wald et al in Oxford (1981) [157] they used a COHb cut-off level of 2%. They found that 81% of cigarette smokers, 35% of cigar and pipe smokers and 1.0% of non-smokers had raised COHb levels. They found CO concentra-

tion in alveolar breath were highly correlated with COHb levels (r = 0.97) indicating that COHb levels can be estimated reliably by measuring the concentration of CO in alveolar breath.

Jones et al studied the relationship between alveolar and blood carbon monoxide concentrations during breath holding (81). They found that during voluntary apnoea after full inspiration the alveolar CO pressure (PCO) first rose abruptly, reached a short plateau between 20 and 30 seconds, and then declined with breath holding at functional residual capacity.

Carbon monoxide exerts ill effects in several ways. It interferes with oxygen delivery and causes a leftward shift of the oxyhaemoglobin dissociation curve (95). CO inhibits the electron transport chain and slows oxidation by binding directly with cytochrome oxidase. It combines with haemoglobin and reduces the ability of red blood corpuscles to carry oxygen to vital organs such as the heart (118). For this reason the hypoxic symptoms produced by CO poisoning surpass in severity those produced by anaemia, despite the same blood oxygen content.

2.6.3 RANGE OF CARBOXYHAEMOGLOBIN VALUES IN DIFFERENT POPULATIONS

In the Scottish MONICA draft manual of operations, May

1984, the normal range of breath CO was given as 2-8 ppm for non-smokers and 9-40 ppm for smokers (162).

Getler and Mattice in 1933 (56) described the normal CO content of blood in 18 non-smokers living in New York City under conditions of minimal exposure as about 1 - 1.5% of the Hb combined with CO. The average in rural localities is less than 1% COHb. In non-smoking street cleaners in New York it was found to be 3% COHb but taxi cab drivers were found to have the highest saturation of 8 - 19%.

Hanson and Hastings in 1933 (62) found that normal individuals who do not use tobacco and who are not habitually exposed to automobile gases showed an average saturation of blood with CO of 1.5%. Analysis made on the blood of subjects after smoking from 10 to 15 cigarettes showed saturation of COHb from 3.1 to 4.3%. Environmental exposure to CO causes a rise in blood CO concentration from 0.5 to 1 % (73).

2.6.4. CARBON MONOXIDE ELIMINATION.

Virtually all of the CO absorbed by man can be recovered in the expired air if this is collected over enough time (122). However only 60% to 70% of the CO lost from the blood during the first hour is found in the expired air. This means that CO is present in some extravascular compartment.

There are differences between individuals as regards CO elimination. In one study it took 250 minutes for the COHb level to fall to half its original value in an individual breathing air at sea level (122). This fall depends on the pressure gradient between blood and air CO in alveoli. Put in another way the blood loses 15% per hour of CO which was present at the beginning of that hour. This rate of elimination is lowered with increasing age of the subjects by 1% for each year over 40.

Particular individuals may vary by + or- 20% in the relation of the percentage saturation of Hb with CO to the percentage of CO in the inspired air (122). The reasons for this are variations in the ratio of tidal air to the dead space and variations in the value of the diffusion constants of the lungs.

2.6.5 FACTORS AFFECTING CARBOXYHAEMOGLOBIN LEVEL.

The body CO stores are influenced by two principal processes, the production and excretion of endogenous CO and the respiratory exchange of exogenous CO (30). The main physiological parameters are (1) rate of CO production; (2) alveolar ventilation; (3) diffusing capacity of the lung; (4) mean oxygen tension in the pulmonary capillaries and (5) concentration of CO in inspired air. They found that measured values of blood COHb compared closely with values calculated from their equations for normal patients and normal volunteers who breathed 100% oxygen for 5 to 7 hours and were probably in a steady state. It was also shown that the blood carboxyhaemoglobin measurement is not a precise

index of CO in vivo since it is markedly influenced by other parameters.

Ho-Yen et al found that smoking fewer cigarettes resulted in an increase in plasma nicotine concentration ($p<0.01$), but carboxyhaemoglobin concentration remained the same (72). They also observed while the volunteers smoked half their usual number of cigarettes they changed their inhalation behaviour. These findings suggest that advice given to patients to smoke fewer cigarettes should be accompanied by a warning against increasing inhalation.

As regards smoking non-tobacco cigarettes, Hynes (65) found that blood COHb levels for volunteers smoking non-tobacco cigarette made from Cocoa bean achieved significantly higher levels of COHb than tobacco.

2.6.6 TOBACCO SMOKING AND COHb VARIATION.

Cigarette smoke contains more than 2,000 chemicals mostly in the particulate phase (45). Volatile and semi-volatile compounds account for more than 90% of the weight of the total main stream smoke, 70% of that being nitrogen and oxygen.

In another study it was found that different cigarette preparations do not result in significant variation in a subject's COHb level (33). The author found also a significant difference in COHb between individual smokers.

They suggest that these differences may be due to inhalation practice or factors such as pulmonary functions which are of importance in determining whether an individual achieves low, moderate, or high COHb levels while smoking.

Exposure to CO in smokers of middle and low tar cigarettes was studied by Stepney. He found that CO intake from a single cigarette (measured by the rise in expired air CO concentration) was significantly less in low tar smokers (145). Longer term exposure, measured by pre-smoking CO levels, was also less in low tar smokers but not significantly so.

Wald et al (1977) studied blood carboxyhaemoglobin (COHb) levels in 343 healthy male cigarette smokers aged 35-64 (156). They found that the mean COHb level was 30% higher in those who smoked unventilated filter cigarettes than in the 41 men who smoked plain cigarettes, after adjusting for the number of cigarettes smoked ($p<0.001$). The medical implications of these results are uncertain. High COHb levels (3 - 4% or more) can exacerbate angina pectoris and intermittent claudication, and it is therefore of concern that COHb levels are higher in smokers of filter cigarettes than in smokers of plain cigarettes.

The variations in carboxyhaemoglobin level in smokers have been studied by Castleden and Cole (27). They reported that in the individual smoker the COHb level does not increase gradually during the day but is kept within

relatively narrow limits. Moderately heavy smokers get up in the morning with substantially raised COHb level because the half life of COHb is significantly longer during sleep than during the day. Women excrete their CO faster than men. It was found also that a random COHb estimation gives a good indication of the mean COHb level of an individual.

Wald et al (154) in Oxford (1975) described a method of using a single COHb measurement with a recent smoking history to estimate the average COHb (boost) produced by each cigarette , the total daily CO uptake from smoking and the mean COHb level throughout the day. The indices were reasonably reproducible within the same person and the differences between people were statistically highly significant ($p < 0.001$). In smokers who smoked 15 to 40 cigarettes a day the mean daily COHb ranged from 0.7% to 9.3%. These differences are large enough to distinguish possible differences in the risk of developing diseases such as IHD. These suggested indices depend less on the time of the blood test and on the daily smoking pattern than on COHb alone. The ratio of COHb boost to the CO yield for a cigarette may reflect the depth of inhalation more accurately than a smoker's self-assessment.

Castleden and Cole (26) found that COHb levels in a group of 11 men who smoked cigars or a pipe but who had not previously smoked cigarettes were similar (mean 2.3%) to those found in urban non-smokers. In another 39 men who had switched from cigarette to pipe or cigars, they had COHb

level (mean 5.2%) comparable to those of cigarette smokers.

In situations where CO inhalation is a hazard, automatic alarming devices often cannot be used; calculation of degree of exposure is unreliable; and direct estimation of blood COHb is impracticable (81). Simultaneous blood COHb and alveolar partial pressure of CO (PCO) analyses in 9 normal subjects on 58 occasions showed an excellent correlation, but slightly alinear though very close relationship. Correlation between COHb saturation and CO meter reading was even closer. The principle of the CO meter reading method involves use of the lungs as aerotonometers until gas pressure of pulmonary blood and alveoli approach equilibrium. The COHb % saturation is then estimated indirectly by rapid physical analysis of the CO content of expired air with model 16 Liston-Becker infra red analyzer.

2.6.7 EFFECTS OF CARBON MONOXIDE ON CARDIOVASCULAR SYSTEM

Aronow (10) reported in an editorial that many studies have documented a significant association between cigarette smoking and the incidence of myocardial infarction and death from coronary heart diseases independent of other risk factors especially in young and middle-aged men.

In a letter, Brown et al said that " It is almost impossible to prove that relatively high CO level in cigarettes will not lead to additional deaths from CHD since it is probable that various smoke constituents' interactions

affect death from this cause (23). Similarly, a role for CO is not excluded by the observation that smokers of cigarettes with reduced tar and nicotine levels and smokers of filter cigarettes show a reduced risk of coronary heart disease. They suggest also that CO yield of individual brands of cigarettes should be published with tar and nicotine levels.

The results of the Framingham study indicate that smoking's greatest contribution to CHD is at the younger age level(95). Other risk factors contribute more at the older ages. Atheroma formation is inevitable when blood fat is persistently elevated above some critical level (95). Nicotine also is an agent in the production of ischaemia and other acute effects. Nicotine liberates nor-epinephrine, causing vasoconstriction. Nicotine, however, has not been shown to be responsible for atherosclerosis. The most acceptable hypothesis is that CO of cigarette smoking plays a primary role in the genesis of atherosclerosis. Until recently, CO had not been considered to have pathogenic significance. In patients with angina pectoris, a dose-related increase in resting heart rate, aortic blood pressure and arterial CO levels and decrease in the stroke volume response to excercise were noted after cigarette smoking.

Coronary heart disease studies have shown that smoking a cigarette has an effect similar to an injection of nicotine in that it produces an immediate increase in the blood pressure, the output of the heart, and it increases the risk of coronary thrombosis (118).

Aronow (11) investigated the significant increase in sudden death from CHD in cigarette smokers and suggested it was due to CO interference with oxygen delivery to myocardium. As cigarette smoke exposes the pulmonary capillary blood to at least 400 p.p.m. of CO it is much higher than those resulting from atmospheric air pollution. The increase in sudden death from CHD is due to nicotine, which increases the myocardial oxygen demand but not the myocardial oxygen supply at times of myocardial ischaemia. Nicotine also increases thrombotic tendency by increasing platelet adhesiveness. During myocardial ischaemia, nicotine can lower the threshold to ventricular fibrillation.

The thrombogenic effects of smoking may be of multiple origin. Carboxyhaemoglobin in blood will increase erythropoiesis which in turn increases blood viscosity and haematocrit levels (95). Catecholamine release at adrenergic end-plates and circulating catecholamine are both increased leading to increase in peripheral resistance.

In another study done by Kaufman et al (83) to evaluate whether nicotine and CO of cigarette smoke is related to the risk of non fatal first myocardial infarction in young men, 502 cases were compared with 835 hospital controls between the ages of 30 and 54 years. The increased risk of MI with the number of cigarettes smoked was estimated. The relative risk for current smokers was 2.8 and did not vary according to the amount of nicotine or CO in cigarettes. They

suggested that smokers who smoke the newer cigarettes with reduced amounts of nicotine and CO do not have a lower risk of MI than those who smoke cigarettes containing larger amounts of these substances.

The effects of hypoxia or increased carbon monoxide have been studied through several experiments which indicate that either increased CO or decreased blood oxygen accelerates the atherosclerotic process in experimental animals and that increasing the oxygen retards vascular lipid accumulation (14,16,51,118). It is proposed that local hypoxia of the arterial wall results in vessel injuries, oedema and congestion of the tissues as well as vessel wall degeneration and myocardium.

Carbon monoxide probably causes enzymatic disturbances in fluid transport across the arterial wall (14). Diffuse fibrous thickening of the arterial and arteriolar walls located in multiple organ systems occurs more frequently in smokers.

A similar effect can happen after prolonged hypoxia exposure for 8-10 weeks (14,51,118). Severe effects are seen as areas of necrosis in the myocardium, occurred after 2 weeks exposure to 16-18% CO. This indicates that CO is toxic, particularly in young smokers with MI and peripheral arterial insufficiency.

Astrup (17) observed that CO promotes development of atheroma in cholesterol-fed rabbits; the cholesterol content in the aortic tissue of the experimental group was about 2.5 times higher than the control group.

The effects of moderate CO exposure in man were studied by Astrup (15). He found that intermittent exposure to CO rather than nicotine due to tobacco smoking may be regarded as a real cause of the higher risk for smokers who develop arterial diseases compared with non-smokers. This demonstrated toxicity of low concentrations of COHb and should be acknowledged when discussing air pollution and threshold limit value for CO.

Cigarette smoke with more than 2% CO can expose the pulmonary capillary blood to at least 400 ppm and angina can then be triggered by a demand for more Oxygen than the myocardial circulation can supply (58).

Another study was carried out by Cohen et al (32) to clarify the relationship between atmospheric CO air pollution and fatalities from IHD. They have studied case fatality rates for patients admitted with MI to 35 Los Angeles hospitals. The results indicate that there is an increased MI case fatality rate in "high" pollution areas and this difference is only evident during periods of relatively increased CO pollution.

The effect of cigarette filters on cardiovascular diseases has been studied by Hammond et al (171). The results clarified that the risk of heart attack is reduced among filter cigarette smokers and Koch has reported also a reduction in peripheral vascular disease. He suggested that cardiovascular events are affected by components in the particulate matter, probably nicotine. Carbon monoxide which is a major component of the gaseous phase of smoke does not have a major role since CO was not markedly reduced in filter cigarettes until products with perforated filter tips were introduced.

The association between acute coronary events (ACE) and cigarette or cigar smoking has been studied by Matroos et al in Netherlands (96). Cigarette smoking was related more to rapidly fatal than to non-fatal acute coronary events, especially in the younger age categories. The association could be shown for cigar smoking with no marked difference between inhalers and non-inhalers. In a series of sudden deaths, the non-current smokers were present only in the older age categories (50 years and older), contradicting most previous reports which stated that cigar smoking should not be considered harmless.

The risk for MI and CHD varies according to the form of tobacco used (79). Jayant et al, found that the risk ratio for bidi smokers (3.8) in India was higher than for cigarette smokers (3.3) and there was no added risk for tobacco chewers.

They explained the lower risk ratio found among tobacco chewers by postulating that it is the CO produced by smoking tobacco that increases the risk of CHD. Substitution of non-tobacco cigarette may be associated with more immediate health hazards ie (CO poisoning) than tobacco smoking (65).

Prue (113) in his study of 27 smokers in a 2 year gradual transfer to low tar and low nicotine brands (brand fading treatment) programme found that smokers showed a decreased rates of consumption and the majority had lower CO level after they changed to low-yield cigarettes at the end of treatment. Therefore low yield cigarettes would appear to be an alternative for risk reduction in individuals who cannot stop smoking.

Aronow (10) has mentioned that COHb level in tobacco smokers correlated better with the presence of CHD than the smoking history. He mentioned also that smokers of non-nicotine cigarettes at a rate of one every 30 minutes and at their normal pace of activities, developed a mean increase in venous COHb level from 1.58 to 7.79%. This increased COHb was associated with a decrease in excercise time until the onset of angina.

Carbon monoxide and angina.

Anderson et al in North Carolina (1973) studied the effect of low level CO exposure on onset and duration of angina pectoris (6). Ten adult men with stable angina

pectoris breathed air with 50 ppm CO or 100 ppm CO for four hours on 5 successive days. After each exposure a standard treadmill exercise ECG was recorded. Mean COHb level after air breathing was 1.3% rising to 2.9% after 50 ppm CO and to 4.5% after 100 ppm CO. The mean duration of exercise before onset of pain was significantly shortened after both 50 and 100 ppm CO in comparison with air. Duration of pain was significantly prolonged after 100 ppm compared with air but not after 50 ppm. ECG showed worsening of ST segment changes with earlier onset and longer duration of ST segment depression.

Aronow in 1980 also evaluated the effect of smoking five non-nicotine cigarettes and of breathing CO on exercise-induced angina (13). Smoking increased venous COHb from 1.7 to 5.3% and decreased exercise duration (until angina is noted) by 45%, increased ischaemic ST segment depression at angina from 1.3 to 1.5mm and decreased systolic BP times heart rate at angina. He compared these results after CO breathing and after smoking. Finally he suggested that tobacco components other than nicotine or CO are responsible for a small decrease in exercise performance until angina.

Angina also was found to be 70% higher among cigarette smokers in a health insurance plan in New York City. Pipe or cigar smokers, taken as a whole, have little or no excess mortality compared with non-smokers (48).

Aronow (1974) (11) showed that smoking high nicotine cigarettes causes a significant increase in systolic and diastolic arterial pressure, a significant increase in heart rate and no significant reduction in aortic systolic ejection period - changes that increase myocardial oxygen demand. Nicotine causes these changes by increasing catecholamine discharge and stimulating sympathetic ganglion cells. In addition, nicotine affects a chemoreceptor in carotid and aortic bodies reflexly causing increases in arterial pressure and heart rate. The CO levels resulting from cigarette smoking (400 ppm) are much higher than those resulting from atmospheric air pollution and eight times greater than the permitted level in industry.

2.7 CARBON MONOXIDE: METHODS OF MEASURING BLOOD COHb.

2.7.1 METHODS OF MEASUREMENT

Methods for determining carboxyhaemoglobin are well documented and innumerable satisfactory methods are available to scientists. Blackmore (1970) tested and described the various methods used. He divided these methods into three groups (20).

1. SPECTROPHOTOMETRIC METHOD.

This method involves comparison of maximum and minimum absorbence spectra for mixtures of either COHb and Oxyhaemoglobin, COHb and reduced haemoglobin or a combination are numerous. Blackmore also mentioned that Machly has critically reviewed the methodology and clarified the problems of dilution error, sulphaemoglobin formation and spectrophotometer inaccuracy (20). He suggests calculation based on readings of the minimum absorbence between alpha and beta haemoglobin bands (about 568 nm), the absorbence at 600 nm for the blood diluted in borate buffer, and the absorbence difference at the peak near 557 nm and the trough near 575 nm for the buffered solution treated with sodium dithionite.

A wide discrepancy is present in the ratios of carboxyhaemoglobin standards diluted 1 in 100 with 0.4% ammonia solution with the extinction ratios 576:560 nm and 541:560 nm plotted against percentage saturation of carboxy-

haemoglobin. This discrepancy indicates the presence of interfering pigments (20).

It was found also that large differences in carbon monoxide saturation resulted in small changes in the movement of alpha band (20). In addition to the normal spectrophotometric errors account must be taken of the errors involved by dilution of the sample.

An experienced operator can achieve an accuracy of + 10% but for a random determination mode at infrequent intervals up to 25%, differences can occur between workers in the same laboratory, all of whom use the same spectros cope (20).

Most of the reported measurements of COHb have used venous blood and analysed spectrophotometrically on a CO Oximeter 182. Many workers have reported the unstability of CO Oximeter (Instrumentation Laboratory Inc.). The technique is generally simple and applicable to analysis of lage numbers of samples (33a).

2. DIFFERENTIAL PROTEIN PRECIPITATION

This is a method of assay in which oxyhaemoglobin is precipitated by heating a buffered solution of blood at 55 ^{o}C for 5 minutes at pH 5.05. Under these conditions the carboxyhaemoglobin remains in solution. The filtered solution can be measured spectrophotometrically and compared with a 100% carboxylated blood.

Analysis with differential protein precipitation is complicated and needs:

a) Standard test tubes.

b) Accurate temperature of water bath, pH and vigorous mixing.

c) Open test tube racks so that tubes reach the same temperature at identical times.

d) Strict control of cooling after reaction.

e) Strict control of time between removal from bath, filtration and COHb measurement.

Therefore it is evident that this method needs considerable effort and time.

3. LIBERATION OF CO AND SUBSEQUENT MEASUREMENT.

This large group of methods relies on the treatment of whole blood with reagents to liberate the CO gas which is then measured by a chemical or physio-chemical method (20). The major limitation of this method lies in the problems involved in the 100% carboxylation of the control haemolysate with which the test is compared. If blood is aged, contaminated or contains haemoglobin pigments other than oxyhaemoglobin, then treatment with a strong reducing agent before carboxylation is essential. The liberated CO is measured by gas-chromatography and plotted against the time of gassing with pure carbon monoxide. This method is sensitive also to changes in potassium ferricyanide concentration, pH, detergents and time of mixing.

Blackmore concluded that spectrophotometric and differential protein precipitations techniques have been found to be unsatisfactory ; in contrast the use of gas chromatography for determining the CO content of the specimen together with determining haemoglobin and iron to calculate the total COHb content will give reliable results (20).

4. OTHER METHODS USED FOR MEASURING CARBON MONOXIDE.

A) Infra red analysis of extracted CO (121).

Measuring Carbon Monoxide in man required a method of estimating blood COHb of greater sensitivity, specificity and technical simplicity. Coburn et al (31) developed a technique involving infra-red analysis of extracted CO. In the course of this method it became obvious that significant quantities of CO can be produced in the blood during treatment of the sample. The method uses CO dissociated from Hb by oxidizing haemoglobin to Met-haemoglobin with ferricyanide and measured as a gas in infra-red analyses. The method had a SD of ± 0.006 ml/100 ml in the range from 0.1 to 1.0 ml/100 ml.

Rees et al in 1980 (23a) in London have compared estimation of alveolar CO measured either as end expired CO (after a breath hold of 20 seconds) or mixed-expired CO tension in mixed expired gas and allowance was made for estimated dead space with 2 spectrophotometric estimations of venous blood COHb. Alveolar CO was estimated by the mixed-expired method using an infra red CO analyser (Analytical

Development Company). They found that estimation of mixed-expired CO was more convenient than the end-expired method, and this estimation of CO exposure discriminated between non-smokers and smokers as well as measurements of COHb.

B) Mine Safety Appliance.

Breysse et al (21) had used two field methods for measuring COHb%. The first method was the Mine Safety Appliance CO poisoning kit. The concentration of CO in the expired air was determined by a field device using the length of stain in indicator tubes as well as by gas chromatography.

C) CO poisoning kit.

The second method used by Breysse et al (21) measured CO concentration in the expired air and COHb % can be calculated from the following equation:

$$COHb\ \% = 0.5 + (CO\ ppm/5)$$

Both methods proved reasonably reliable when the results were compared with COHb % determined from blood specimens.

D) Measurement of breath CO by Ecolyzer.

Since carbon monoxide in alveolar air after breath holding is in equilibrium with the concentration of carboxyhaemoglobin in the blood. Jarvis et al (77), in their study on the development of a simple breath test of tobacco smoke, found that the amount of CO in end-expired alveolar air provides a rapid and accurate non-invasive indirect measure of carboxyhaemoglobin. They used the carbon monoxide

analyser, Ecolyzer Series 2000, which is portable and inexpensive. The method has a potentially wide application as an objective test of the amount smokers inhale and of their claims to have stopped. Impaired lung function cannot be an important factor in normal smokers. The closeness of the relationship between COHb and expired air CO suggests that there is little to be gained from analysing a sample of blood rather than expired air.

The relationship between percent saturation of Hb and CO to the percent of CO in inspired air has been studied. In order to calculate blood COHb% at rest for an individual (ventilation 6 litres per minute, pulse 70 per minute), the concentration of carbon monoxide in the inspired air in percent (%CO) times the minutes of exposure (t) multiplied by 3 (122).

$$\% \text{ COHb} = 3 \times (\%CO) \times t.$$

If COHb measured during light activity 5 is used instead of 3 and for heavy work 11 will be used in place of 3.

In the Multiple Risk Factor Intervention Trial in Alabama, breath carbon monoxide was measured by Ecolyzer to provide positive feedback for smoking changes (73). Breath CO tended to increase with the increase in number of cigarettes smoked, depth of inhalation and other smoking-related factors and to decrease after cessation.

2.7.2 METHODS FOR DETECTING SMOKING USING OTHER METABOLITES OF TOBACCO.

It is well known that tobacco smoke is a mixture of gases and millions of minute particles from which some 300 different components have been identified (63). Nicotine is one of them; it is rapidly degraded to non-toxic products and there is no evidence that chronic toxicity to this substance occurs in smokers.

Cotinine, which is a metabolic product of nicotine, has a longer half life (19 - 40 hours as compared with less than 30 to 110 minutes for nicotine) (68). Therefore cotinine measurements provide advantages over those for nicotine. The former can be measured by sensitive radioimmunoassays and its presence is an indicator of chronic exposure to tobacco, whereas that of nicotine is an indicator of recent exposure.

Nicotine, cotinine and carboxyhaemoglobin levels in long-term (> 1 year) smokers of a given cigarette should be compared with the corresponding nicotine and CO levels in the smoke of that cigarette; only such long term studies can determine what adjustment, if any, is made in smoking patterns when smokers switch to low tar, low nicotine cigarettes (171).

In order to verify the smoking histories of MI patients by measuring both urine nicotine and cotinine, Wilcox et al (1979) found that while in non-smokers detectable

concentrations were always below the confidence limits set for the method (6.04 ± 0.73 mmol/L), smokers were always above these limits (163). Forty six to 53% of previous smokers had actually stopped smoking compared with the 63% who said that they had stopped. Therefore assays of urinary nicotine and cotinine at the same time may be a useful means of verifying patients' current smoking habits.

The study of the relationship between plasma cotinine and COHb by Hill et al clarified a higher significant correlation between both (r = 0.70: r= 0.67 men:women) and with nicotine (r = 0.73: r = 0.74 men:women) than with thiocyanate (r = 0.53: r = 0.59 men:women) (68). They found also when smokers were separated into those smoking cigarettes yielding more or less than 1mg nicotine per cigarette, COHb was the better indicator of daily exposure and absorption. With increasing consumption, COHb and plasma nicotine showed a comparable progressive increase in men and women in a linear relationship.

In addition to CO gas, other components in cigarette smoke are toxic (68). These include hydrogen cyanide, which is converted to thiocyanate. Thiocyanates accumulate in body fluids and inhibit cellular respiration.

A significant correlation has been found between carboxyhaemoglobin and plasma thiocyanate concentrations with self-reported daily cigarette consumption (152). The extent to which inhalation patterns affected the intake of cigarette

smoke constituents was determined from the partial correlation between carboxyhaemoglobin and plasma thiocyanate concentrations after the number of cigarettes smoked per day had been allowed for. Thus, 23% of the variation in carboxy-haemoglobin and thiocyanate concentrations was accounted for by the way a cigarette was smoked and a further 21% by the number smoked per day. Furthermore, the relation between COHb or plasma thiocyanate and daily cigarette consumption was not linear but reached an asymptote at consumption rates above 25 cigarettes a day.The remaining variability may be due to difficulties in CO or hydrogen cyanide yields of cigarettes, excretion, and environmental or nutritional factors.

Vogt et al (153) have studied the baseline carbon monoxide and serum thiocyanate levels as predictors of outcome. In a San Francisco clinic, 162 male smokers participated in a smoking cessation programme. They underwent analysis of expired air carbon monoxide and serum thiocyanates (SCN) at entry and one year later. They found that persons who failed to stop smoking (58%) had higher base line CO and SCN and smoked more cigarettes per day than those who succeeded in stopping. They also noticed that persons who failed to quit reduced the reported number of cigarettes smoked per day by one third. At the same time there was no corresponding reduction in CO and SCN, suggesting that self-reporting of a reduction in number of cigarettes smoked may not lead to corresponding decline in exposure measured independently.

2.7.3 DETECTION OF TOBACCO METABOLITES IN PASSIVE SMOKERS.

In recent years there is an increasing concern as regards non-smokers chronically exposed to tobacco smoke in their daily lives who may suffer a significant risk to their health (78). Therefore in order to study the reliability of a non-invasive marker and validated questionnaire measures for assessing the degree of exposure in non-smokers, Jarvis et al (1984) examined the levels of COHb, saliva thiocyanate, plasma nicotine and cotinine. They studied also the relation between the levels of self reports by questionnaire in non-smokers exposed to tobacco smoke. The results clarified that cotinine level showed a significant variation with exposure. Thiocyanate and carbon monoxide levels were unrelated to exposure as was the concentration of nicotine in plasma, so cotinine can provide a sensitive guide to the extent of recent daily passive smoke exposure in stringently defined non-smokers. The concentration of nicotine in saliva was related to exposure, but only exposure on the day of testing. Therefore the non-invasive samples of urine and saliva are particularly suited to epidemiological investigation.

MATERIALS AND METHODS

CHAPTER THREE: MATERIALS AND METHODS

3.1 DESCRIPTION OF POPULATION, DEFINITION OF TERMS AND HEALTH EDUCATION METHODS

This project was based on a follow up study for the validation of a breath carbon monoxide test as an indicator of smoking cessation among patients admitted to a coronary care unit (CCU).

The populations studied were:-

1- A population of patients admitted in 1983 to the coronary care unit (n=212) at the Western Infirmary irrespective of the clinical diagnosis. It included all ages and both sexes and was used to evaluate two health education interventions. In addition random sub-samples of these patients were taken when required for detailed study of blood carboxyhaemoglobin (COHb) and breath carbon monoxide (CO).

2- Glasgow High School pupils (n=105) and Ruchill Hospital employees (n=143) were also included in the study for evaluating the repeated use of a carbon monoxide (CO) breath measuring device in normal adults and children.

The admission rate of patients to the Coronary Care Unit at the Western Infirmary was about 60 cases on average every month. Patients were admitted from the Western Infirmary catchment area which extends from the city centre (Post code G1) to the Clydebank area (Post code G81). Clydebank patients constituted about 20% of the total CCU admissions.

A pilot study was done to investigate the difficulties which would be encountered in the study. Difficulties were found in following-up patients in their homes (other than Clydebank area) because they were scattered over a large catchment area. Searching for homes was time-consuming and sometimes a house had to be visited many times to see the patient. The cost of transport was another difficulty. It showed that Clydebank patients were comparable with the other patients with regards age, sex, social class distribution and smoking habit.

Sample size was estimated before the study began as follows (47) :- The successful follow-up of 80 patients in group I (non-intervention group) and 25 in group II (health education group) together with an expectation of a 30% reduction in smokers in group I and 60% in group II, was calculated to give an 80% chance of detecting a significant difference ($p < 0.05$).

Fresh venous blood samples for blood carboxyhaemoglobin analysis were collected immediately after admission. All CCU patients (n=212) included in the study filled in a questionnaire based on the previous Midspan Study in Renfrew and Paisley (Appendix A2) (103). This was done after a patient's condition became stable. Such a self-administered questionnaire is reliable enough to allow confident estimation of the variables within groups and between different groups, eg. smokers and non-smokers.

The questionnaire included standard demographic material and the standardized and validated questions on respiratory symptoms developed by the Medical Research Council Committee on the aetiology of chronic bronchitis (98,99). In addition to the standard questions on respiratory symptoms, chest illness and detailed smoking habits, a series of questions on occupation, family history of smoking, and chest pain were incorporated. These questions were developed at the London School of Hygiene and Tropical Medicine and approved by WHO for use in field studies (123,124). All questions were coded to facilitate analysis by a computer file (Appendix C).

Each questionnaire was checked immediately and standard investigations were carried out which included:

1. Measuring weight and height of subjects in indoor clothing (without shoes).
2. Measuring FEV 1.0 and FVC using a Garthur Vitalograph with the subject sitting. The best expiration was recorded, the one second vital capacity determined with vitalography, and the results expressed as a percentage of the total vital capacity (66).
3. The social and laboratory data and results of investigations were recorded from medical records eg. ECG findings.
4. Blood pressure measurement was done at rest after admission, by a Mercury sphygmomanometer with a cuff width of 14 cm, on the right arm. Diastolic blood pressure was taken at the disappearance of the fifth Korotkoff sound.
5. Plasma levels of cholesterol, triglycerides, glucose

and cardiac enzymes (CK, SGOT and LDH) were measured in a venous blood sample.

6. The reported daily number of cigarettes, cigars and pipes smoked were summed and values were presented as mean tobacco consumption per person.
7. A chest X-ray was carried out for each patient.
8. Exhaled Carbon Monoxide was measured, the method for which is described later in a separate section (Section 3.3).

The 212 cases studied were given the basic health education material (Appendix E1)(108) and divided into two groups comprising 116 smokers and 96 ex and non-smokers. Smokers were followed up. The response rate was 94 (81%) out of 116 patients who were smokers. The entry and follow-up data were collected by the research student.

Smokers from Clydebank were also given the Middlesex Hospital Questionnaire (MHQ Appendix A3), an Illness behaviour Questionnaire (IBQ Appendix A5) and a dietary questionnaire (Appendix A4) in the CCU to assess their psychological profile and feeding habits. Assessment was done by the CCU health visitor by scoring the answers given to each question.

This group was given also another booklet about health education and heart disease "Beating heart disease" (135) (Appendix E2). It contains details about what heart disease is, why some people get it, how to help yourself to avoid it,

what is meant by angina and a heart attack, who suffers from heart disease and what are its causes. Advice was given about smoking and heart disease, diet and what the patient can do for himself; and about high blood pressure - what is it, what the patient can do. A section is included on stress and the heart, and how to stay fit and healthy after a heart attack.

In the CCU these Clydebank patients were given intensive personalised health education by the trained health visitor about smoking and its hazards, the benefits to be gained from smoking cessation, and the important role of such risk factors as smoking, diet, obesity and hypertension in coronary heart disease according to the previously assessed psychological profile of the patient. It included also at least one home visit for follow-up after discharge from the hospital. Home visits lasted on average 45 minutes.

Visiting patients at home is an important part of intensive health education because most patients live with their families and usually are affected by family feeding habits and psychological and social factors in the house. Therefore it might be useful to include the spouse in the health education process to manage the new circumstances properly.

The presence of a suitable clinic in the Clydebank Health Centre under supervision of Consultant Cardiologists from the Western Infirmary facilitated further follow-up of the patients. The health visitor who was providing health

education for patients in the Western Infirmary also worked in the follow-up clinic in Clydebank health centre. He was an ex-smoker at the time of the study. Ease of access to patients in this area made follow-up and home visits more likely to be successful than following up patients living in other parts of the Western Infirmary's catchment area.

DETAILED METHODS OF HEALTH EDUCATION.

Basic health education material: This was standard for all CCU patients in the form of a booklet "Advice for those recovering from a heart attack" published by the Scottish Health Education Unit (Appendix E1) (108) and it included the following titles:

1. What is a heart attack?
2. Physical activity during the time of illness and after recovery.
3. Advice about returning back to work, the suitable time and type of activity at work.
4. Sleep and rest.
5. Advice concerning the diet and weight reduction if the patient is overweight.
6. Advice about giving up smoking.
7. Advice for women as regards household duties and family planning methods.

Intensive health education was undertaken after every Clydebank patient (smoker) filled in the MHQ, IBQ and a dietary questionnaire (Appendix A4) in the hospital, and was continued after dicharge. This was specifically tailored to

their responses after scoring the answers and assessing the psychological profile and it included advice and health education with regard to:

1. Hazards of smoking and the best way to give up smoking.
2. Advice in tackling the psychological problems of the patient and also physical fitness training (Appendix A6).
3. Recommendations about dietary changes according to the patient's answers to the dietary questionnaire.
4. Advice about exercise according to the fitness of the patient.

All patients in this group were also given the detailed booklet 'Beating Heart Disease' (135 and Appendix E2). It was essential that the patient should trust our judgement and our concern for his wellbeing. The aim of the advice in general was to avoid raising major fears and to emphasise the positive merits of the health advice, that is mostly the gains from stopping smoking rather than the danger of continuing.

Follow-up

One year after discharge an invitation letter was sent to patients who were smokers (n=116) at the time of admission. All smokers were invited to return to the Western Infirmary except for Clydebank patients who were asked to attend the Clydebank Health Centre. Non-respondents were invited twice to participate in the study by letter and

phone-call (Appendix A7). A response rate of 81% (94/116) was achieved. Of the 22 non-respondents, 2 patients refused to participate and 20 could not be traced. Of the 94 responses, 7 patients (7.5%) were found to have died after discharge from the hospital within that year. The main purpose of the follow-up examination at one year was to compare the status of the two groups with regard to smoking habits and other risk factors and to evaluate the effectiveness of the health education procedures.

Awareness about cardiovascular risk factors was studied by having the subjects identify the correct risk factors from a list of questions and answers generally associated with coronary heart disease (Appendix A, page 297)(86). Their current smoking, dietary history, and their ability to recall the advice they had been given 1 year previously were measured. Smoking history was checked against both blood COHb and breath CO, allowing a further comparison of the two methods.

Confidentiality

Assurance was given to respondents that this informatiom was for the purpose of research only.

Data processing

The completed questionnaires were coded and the data were stored in a computer file. The Statistical Package for Social Science (SPSS) (106) and the Minitab Statistical Package (104) were used for analysis of the data.

Definition of terms (103)

Age is counted from the date of birth to the date of hospital admission and thus involvement in the study.

Occupation and social class: according to Classification of Occupation (OPCS 1980) (29).

Stress index is obtained from the sum of all the positive answers to a series of related questions (103) (Appendix B).

Smoker is any person who still smokes at the moment of the research or before admission and he or she can be:

a) cigarette smoker. b) pipe smoker. c) cigar smoker.

In this study smokers were classified as light smokers who smoke less than 15 cigarettes per day, moderate who smoke between 15-25 cigarettes per day and heavy smokers who smoke more than 25 per day.

Non-smoker is any person who has never smoked a cigarete, pipe or cigar before.

Ex-smoker includes those who have smoked for some time but were not smokers at the time of the study.

Knowledge about cardiovascular risk factors was assessed from a list of questions. The maximum possible score is 16. (Appendix B)

Cardiorespiratory symptoms For reference see Appendix B.

3.2 CROSS-REACTIVITY OF CO WITH BREATH HYDROGEN

A study was made of cross-reactivity between breath carbon monoxide and hydrogen in the 2 groups of healthy individuals- Ruchill Hospital employees and Glasgow High School pupils. Ruchill Hospital employees included in this study comprised both sexes, smokers and non-smokers. Everyone was invited to take this breathing test through a notice circulated to all hospital departments. One hundred and forty three personnel responded to take the test. About 10 personnel, mainly cleaners refused; all were smokers. The employees were asked to take the test in the Coffee Lounge on the specified dates; each one was asked to answer a few questions, particularly about their smoking habit. Each person was asked to take a breath hydrogen test at the same time as the breath carbon monoxide test.

Because nearly all hospital employees were over 18 years of age and had a different smoking pattern from younger age groups (<18 years), I arranged to do the same procedure on Glasgow High School children for the purpose of comparison.

One hundred and five pupils from Glasgow High School carried out breathing tests in confidence - that is without personal identification. It included breath carbon monoxide measured by a GMI (Gas Measurement Instruments Ltd) portable carbon monoxide monitor and breath hydrogen measured by a GMI breath hydrogen monitor (Appendix D2). Each test was done twice at the same time for each pupil. Each pupil was asked a few questions regarding his smoking habits, age, family

smoking habits and, for those who smoked, the time since their last cigarette.

3.3 TECHNIQUES OF CARBON MONOXIDE MEASUREMENT: COMPARISON BETWEEN METHODS, CHANGES IN CO OVER TIME, AND ACCURACY

Blood COHb was measured for each patient at entry and at follow-up as well. For detailed study of blood COHb sub-samples from the total patient population were used. The relationship between blood COHb and breath CO was studied in a random sample of 42 patients. Changes in COHb over time was studied in 8 persons. Study of the effect of storage was done on 21 patients. In order to calculate the half life of breath CO, six healthy subjects carried out the breath test every 15 minutes for 3 hours.

Blood COHb is measured by the CO-oximeter 282 as percentage saturation and for the purpose of comparison in some tables it is converted to ppm (1% saturation is nearly equal 5 ppm)(122). Normal levels were measured up to 1.9% saturation (about 9 ppm)(62,161,162). Higher levels of blood COHb (>2% saturation) were divided arbitrarily into moderately high (2-4%) and high levels (>4% saturation). In this study CO was measured by three different methods which were:

3.3.1 Spectrophotometric method for measuring blood COHb

A 3cc fresh venous sample was collected in a heparinised (one drop of heparin 5,000 I.U.) airtight syringe immediately after admission, for immediate analysis. Carboxyhaemoglobin was measured by CO-oximeter 282

(Instrumentation Laboratory Inc. Appendix D3) according to the instruction manual (74). Five COHb measurements were taken for each blood sample. The averages of each five readings were recorded. A subsample was used for COHb measurement by a second CO-oximeter 282 machine and the COHb measurements were similar for both machines. The measurements were in %COHb and in order to transfer %COHb to carbon monoxide content the following equation was used.

$$1.39 \times THB \times \frac{\%COHb}{100} = \text{ml CO/100ml blood}$$

(THB = the total amount of haemoglobin in the sample calculated also by CO-oximeter 282 at the same time).

Light absorption at certain wave length was the underlying theory of this CO-oximeter and of spectrophotometry; the wave lengths used are 541 nm, 560 nm and 576 nm (74). Light absorption at 548 nm is related to changes in total haemoglobin concentration. Absorption at 568 nm to 548 nm is related to carboxyhaemoglobin and 578 to 548 is related to oxyhaemoglobin. The range for readings is 0-100% for carboxyhaemoglobin, 6-20gm/100ml for blood haemoglobin and 0 to 100% for oxyhaemoglobin. Reproducibility of this machine is reported to be ±1% (74).

3.3.2 **Gas chromatography method for measuring carboxyhaemoglobin.**
The principal of this method is liberation of bound carbon monoxide from haemoglobin and its subsequent measurement.
Apparatus: Gas-chromatograph fitted with Katharometer detector, linear amplifier and gas sampling valve with 0.5ml and 5.0ml loops (20).

Reagents: Distilled water, saponin and de-gassing reagent.

Procedure:

(1) Put 1ml whole blood + 3ml of distilled water + 10mg of white saponin then mix for 10 minutes and centrifuge for 15 minutes at 3000 rpm.

(2) Remove the supernatant liquid and pipette 1ml of it into a 5ml plastic syringe fitted with a short length of plastic tube. Into the syringe draw 1ml of air, into another syringe place 3ml of degassing reagent and attach this syringe into the other end of the plastic tube. Mix the two solutions by transferring the degassing agent to the syringe containing the haemolysate.

(3) Mix on a rotary mixer for 10 minutes and the air space containing the CO is then transferred to the empty syringe.

(4) Transfer the air space to a 5ml gas loop of the gas chromatograph.

(5) The recorder reading shows the separation of oxygen, nitrogen and carbon monoxide obtained from such an injection at the optimum sensitivity of the detector and Katharometer. The volume of carbon monoxide liberated from an aliquot of haemolysate is calculated, with allowances for difference in amplifier attenuation, as follows:

$$\text{Volume of CO from test} = \frac{\text{Peak height of test}}{\text{Peak height of CO}} \times \frac{\text{Attenuator setting for test}}{\text{Attenuator setting for CO}} \times \text{Volume of pure CO}$$

From the relationship 100mgHb = 0.34mg iron = 136 ul of CO, the total volume of CO that would represent 100% carboxylation of haemolysate can be calculated.

Per cent saturation is equal to the volume of CO liberated from 1ml of haemolysate devided by the volume of CO that would combine with 1ml of haemolysate as calculated from Hb and iron concentration.

3.3.3 **Breath CO measurement by GMI portable CO-monitor.**

Breath carbon monoxide was measured by a carbon monoxide monitor developed by Gas Measurement Instruments Ltd., (GMI). It is based on an electrochemical cell which has recently become available (Appendix D1). This CO monitor is hand held and it works with a rechargeable battery for about 20 hours continuously. The monitor was calibrated by carbon monoxide gas 93 ppm. After switching on, but before using, it is allowed to settle to zero, otherwise the zeroing screw has to be readjusted. Everyone breathed into a sterile disposable mouthpiece connected to the GMI CO monitor from full inspiration to the end of expiration. Expiration was continued as long as possible and samples were taken at end - expiration in order to include a good sample of alveolar air. The test was repeated five times for every person with all measurements made at rest.

The mean, standard deviation and coefficient of variation for each group of 5 readings were obtained for both COHb and breath CO readings.

Simple breath CO measurement is of importance as measuring CO in blood or measuring urinary cotinine is time consuming, expensive and requires considerable experience in laboratory work; on the other hand measuring breath CO is cheap, quick and simple using portable monitors. Because of this they can be used in field studies concerned with measuring breath CO.

3.4 STATISTICAL METHODS USED

1. Terminology(8).

Null Hypothesis (Ho) :states that there is no difference in the frequency of a risk factor or in the prevalence of a stated condition between two or more groups.

Significance level: this is the probability of rejecting Ho when it is true. Often the 5%, 1% and 0.1% (ie. 0.05, 0.01, 0.001 respectively) levels are chosen as important.

Critical value: this is the value found in statistical tables which if exceeded by the test statistic will lead to the rejection of H_o at some significance level. The main statistical significance tests used for the tabulated data are:

2. Chi Square (8).

Chi-square (X^2) test is used for making a conclusion about the prevalence data. In its basic form the X^2 test compares the observed frequencies with those which would be expected under some theory or hypothesis. Thus X^2 = the sum

of the squared differences between the observed and expected values divided by the expected value ie.

$$X^2 = \Sigma \frac{(O-E)^2}{E}$$

If X^2 is small, the implication is that the observed and expected frequencies do not differ markedly, and there is no strong evidence against the null hypothesis (ie. cannot reject null hypothesis). The larger the value of X^2, the greater the discrepancy between observed and expected frequencies and the less likely it is that the hypothesis is true (ie. reject null hypothesis). There is a critical value of X^2 such that any value of X^2 greater than this critical value implies disagreement between observed and expected frequencies. This critical value can be found in X^2 distribution tables and the value depends on the degrees of freedom of the problem under consideration. Fisher's exact test was used for some 2 x 2 tables with small numbers and results are given under each table.

3-Use of Correlation Coefficient for comparison between two methods

Comparison of a new measurement technique with an established one is often needed to see whether they agree sufficiently for the new to replace the old (20a). Such investigations are often analysed inappropriately, notably by using correlation coefficients. The use of the correlation coefficient is misleading because (1) it measures the strength of a relation between two variables, and not the agreement between them unless the points are on the line of equality with similar scales. (2) it is not affected by

change in the scale of measurement which certainly affects agreement. (3) correlation depend on the range of the true quantity in the sample. (4) the test of significance may show that the two methods are related, but it would be very surprising if two methods designed to measure the same quantity produced values which were not related in some way. (5) data which seem to be in poor agreement can still produce quite high correlations.

However an alternative approach, based on graphical techniques and simple calculation is appropriate. Precision of the estimated limits of agreement can be obtained by using standard errors and confidence intervals to see how close the agreement is (20a).

4. Oneway Analysis of Variance (Oneway ANOVA) (8).

This test is used to compare the means of more than two populations, where it is inappropriate to analyse the data by performing multiple t-tests, ie. the more t-test that are performed, the greater the probability of establishing an apparently significant difference (in the absence of a real difference) purely by chance. Therefore, it is better to perform the analysis of variance on the data, and to examine it more closely by performing t-tests between pairs of groups only if the initial analysis of variance demonstrates a significant difference between groups (giving an indication that at least two groups differ from one another, but not indicating which groups they are). The test statistic is:

$$F = \frac{\text{(between groups sum of square) / (no. of groups -1)}}{\text{(within groups sum of square)/(no. of observations- no. of groups)}}$$

ie. $$\frac{BSSq \,/\, (k-1)}{WSSq \,/\, (n-k)}$$

when k = no. of groups and n = no. of observations.

The degrees of freedom are k-1, n-k. We reject the null hypothesis if F is larger than the critical value from F distribution tables.

5. Exponential decay model

COHb concentration in blood decreases gradually over time (provided the subject refrains from smoking) due to excretion through the lungs. The reported half life of COHb in publications is 4 hours (122).

It is useful to apply the exponential decay model to observed COHb measurements at time t hours after last cigarette to find out what the value was at time 0. The equations used are

The concentration at time t = concen. at time 0 X $2^{-\frac{1}{\text{half life}} \times t}$

therefore to calculate COHb concentration at time 0 is equal to:

$$C_0 = C_t \,/\, 2^{-\frac{1}{H}.t}$$

H = half life in hours.

6. Measurment of Reliability

Reliability of observations is different from their accuracy eg. findings of two physicians may agree (be

reliable), and yet be wrong as compared to an independent standard of accuracy (87). Agreement of physicians is termed inter-observer agreement and agreement of a physician with himself regarding repeated observations is termed intra-observer agreement. Overall agreement is the proportion of all patients about whom observers agree on the presence or absence of an abnormality. Specific agreement is the proportion of patients about whom observers agree on the presence of an abnormality. Neither overall nor specific agreement rates take into account the varying contribution of chance agreement rates.

As regards the reliability of self-reported smoking behaviour, it is generally felt that confidential smoking questionnaires can give reliable results but this reliability may be improved by the use of a "visible" objective test (141).

Accuracy of many diagnostic procedures is subject to impairment from errors in technique of examination and interpretation (55). Even experienced physicians are found to have a measurable degree of 'observer error'. Accuracy in diagnosis can be improved by independent examination either by the same observer on two different occasions or by two different observers. The observer error in different fields should provide a stimulus to greater care in examination, to increased use of consultation and, above all, to continued attempts at elucidation and correction of the factors involved.

7-Sensitivity and specificity

The most useful reliable investigation fulfils a discriminating function (128). The results are either positive or negative, normal or abnormal, diagnostic or non-diagnostic. The reliability of tests in separating diseased from non-diseased subjects may be measured in terms of their sensitivity and specificity (Vecchio, 1966). Sensitivity is a measure of whether the test will identify the disease in question. Specificity is the ability of the test to separate subjects with the disease from those without it. The possible outcomes of using the test are:-

		Disease	
		Positive	Negative
Test result	Positive	True positives	False positives
	Negative	False negatives	True negatives

To maximise the yield of true positives an operator must accept a degree of contamination by false negatives. The functions of individuals interpreting results may be calibrated on a 'receiver operating characteristic' (ROC) curve. In any given investigation the distribution of criteria for diagnosis or exclusion will seldom permit complete separation from those with a disease from those without.

8. Use of analysis of variance to estimate reliability of measurements (167).

For a person who has a magnitude of (π) for a specified characteristic the observed score after using a measuring device will be (π+n) therefore, a measurement on person (i) with measuring device (j) will be

$$X_{ij} = \pi_i + n_{ij}$$

where Xij = observed measurement

π_i = true magnitude of characteristics

n_{ij} = error of measurement

After the assumption that π_i is constant upon repeated measurements and n_{ij} is assumed to vary, the mean of (k) repeated measurements will be

$$\frac{\Sigma_i X_{ij}}{k} = \bar{P}_i = \pi_i + \bar{n}_i$$

and the variance within person (i) is due to error of measurement and the pooled within-person variance also estimate variance due to error of measurement.

The reliability of the mean of (k) measurements is the variance due to the true scores divided by the sum of the variance due to the true scores and variance due to error of measurement.

Reliability of the mean of a group of measurements =

$$r_k = 1 - \frac{\text{MS within people}}{\text{MS between people}}$$

The reliability of Xij which is a single measurement is

$$r1 = \frac{\text{MS between people} - \text{MS within people}}{\text{MS between people} + (K-1)\ \text{MS within people}}$$

MS = mean squares.

rk also can be expressed in terms of the reliability of a single measurement as follows:

$$rk = \frac{kr_1}{1+(k-1)r_1}$$

The assumptions underlying the validity of this formula are those underlying the analysis of variance and they are

1 - The error of measurement is uncorrelated with the true score.

2 - The sample of n subjects (or people) is a random sample.

3 - The sample of k measuring instruments is a random sample from a population of comparable instruments.

4 - The within-subject variance may be pooled to provide an estimate of σ^2_n .

INTERPRETATION OF THE RESULTS

The term rk means that if the experiment is repeated with another random sample of judges but with the same subjects the correlation between the mean ratings obtained from the two sets of data for the same people would be the value of rk. This assumes that the variance due to

differences between the means of judges is part of the error of measurement and does not represent a systematic source of variation. But if these represent a systematic source of variation then the source of variation due to differences between those means should not be considered part of the error of measurement. In order to adjust for the frame of reference the deviation of the means of a judge from the mean of all judges should be calculated. By the adjustment procedure the SSq between people is not affected. However, SSq within people for the adjusted data differs from the corresponding sums of squares for the unadjusted data by SSq judges. So adjustment was made to eliminate the between-judge variation from the within-subject variation.

The mean square within people (MSW) for the adjusted data is numerically equal to the mean square residual (MS res) for the unadjusted data and the estimates of reliability are

$$rk = 1 - \frac{\text{MS-residual}}{\text{MS between people}}$$

$$r1 = \frac{\text{MS between people - MS res}}{\text{MS between people + (k-1) MS res}}$$

The reliability of a single rating (r1) for the adjusted data is approximately equal to the average intercorrelation between ratings given by pairs of judges.

RESULTS

CHAPTER FOUR: RESULTS

4.1 DESCRIPTION OF THE TOTAL PATIENT POPULATION

This part of the study was done to validate the carbon monoxide (CO) breath test as an indicator of giving-up smoking as a result of the Health Education intervention. All patients came from the Western Infirmary catchment area in Glasgow which extends from the Clydebank area (Post code G81) to the City centre (Post code G1). The total number of patients was 212. All patients had been admitted to the CCU in Western Infirmary and they were given standard medical care as well as basic health education. 55% out of whom were current smokers (see Table 1). The 116 current smokers were followed-up and 94 (81%) responded, drop-outs were invited to participate in the study twice by letters and a phone call to arrange a suitable appointment. Twenty two patients could not be traced because they had moved during that year. Patients from Clydebank (Post code 81) were in addition given the intensive personalised health education.

The social class distribution was as follow:

Social class I and II 12%, social class III non-manual 11%, social class III manual 24%, social class IV , V 11%, retired 16% , unemployed 11% and housewives 15%.

AGE AND SMOKING HABIT FOR ALL CASES.

In Table 1 most (50%) of the patients are in the age group 55-65 years, and only 13% are less than 45 years. Elderly patients of more than 65 years form 9% out of the total patients. 55% were current smokers at the time of the study, ex-smokers 30% and non-smokers 15%. It is evident that the majority (85%) of patients have smoked or were current smokers at the time of the study. Smokers comprised different percentages in each age group, being lower at the extremes, 50% in age group less than 45, 61% in age group 45-55, 56% in age group 55-65 years and 37% of the elderly above 65 years. Statistically (X^2) there is no significant relationship between age and smoking habit.

SEX AND SMOKING HABIT FOR ALL CASES.

Table 2 shows the distribution of smokers by sex, 57% of males and 47% of females are current smokers. There is no significant relationship between smoking habit and sex (X^2 =5.51; 2 df; N.S). However combining "smokers" and "ex-smokers" to give one group resulted in a significant relationship and males are less likely to have never smoked. (X^2 =4.5; 1 df; $p<0.05$). Ex-smokers are equally frequent in both sexes.

AGE WHEN SMOKING BEGAN AND NUMER OF CIGARETTES SMOKED PER DAY FOR SMOKERS.

In Table 3a about half of the male patients (48%) started smoking between age of 15 to 25 years, 31% started smoking before age 15 years and 22% of the patients started at age more than 25 years. There is a significant relationship between age when smoking began and number of cigarette smoked per day at admission ($p<0.05$). Table 3b is for female smokers, collapsed to a 2X2 table due to small numbers. The relationship was also significant ($p<0.05$) as in males. This means that in each sex, the younger the age when smoking began the higher the number of cigarettes smoked per day later in life.

SMOKING HABIT AND CARBOXYHAEMOGLOBIN LEVEL FOR ALL CASES

In Table 4, there was no significant relationship between COHb and smoking habit at entry in the study. The mean COHb for smokers was 10.3 ppm and 10.2 ppm for non-smokers. This result was not expected when the study was initially planned which may be due to inaccuracy and instability of the spectrophotometric method used (20,33a). Inhaled oxygen during the emergency phase could be another factor.

Table 5 shows the relationship between the number of cigarettes smoked per day and COHb in smokers. Mild smokers (less than 15 cigarettes per day) comprised 40%, 32% smoked between 15-25 cigarettes per day and 28% smoked more than 25 cigarettes per day. Mean COHb for mild smokers was 9.6 ppm,

moderate 10.6 ppm and 10.5 ppm for heavy smokers. There was no significant relationship between the number of cigarettes smoked per day and COHb level.

THE PRESENCE OF ANGINA AND COHb LEVEL FOR ALL CASES.

From Table 6, 74% of patients with angina (n=83), had COHb over 10. The mean COHb for patients with and patients without angina is 10.2 ppm, 10.5 ppm respectivley. There is no significant relationship between angina and COHb level (Chi-square= 3.7; 4df).

Table 7 shows the distribution of COHb levels in patients with (48%) or without (52%) myocardial infarction. 70% of patients with myocardial infarction have a COHb over 10 ppm. 85% of patients without MI have a COHb level over 10 ppm. The table shows a significant inverse relationship between myocardial infarction and COHb with a tendency for patients with MI to have a within normal COHb. This relationship may not be more than a chance finding.

STRESS AND SMOKING HABIT IN ALL CASES.

Due to small frequencies in each group, the two group 'no stress' and 'mild stress' were combined to form one group, and the other consisted of 'moderate' and 'severe' stress. Table 8 shows that 23% of the smokers,13% of ex-smokers and 9% of non-smokers had no stress or mild stress as estimated from the questionnaire. It is evident also from

table 8 that 77% of smokers, 88% of ex-smokers and 91% of non-smokers suffered from moderate or severe stress. X^2 shows no significant relationship between smoking habit and stress (X^2=4.6, df=2).

MYOCARDIAL INFARCTION AND LIFE EVENTS IN THE YEAR BEFORE HOSPITAL ADMISSION.

In Table 9, more than half of the patients (56%) did not suffer from any of the listed life events (job loss, family member death, financial problems or residence change). Few patients (14%) suffered from more than one event. Death of a family member forms the most frequent life event (11%), followed by financial problems (9%). The percentage who had had more than one life event and not had a myocardial infarction was 19% compared to 8% had MI. There is no significant relationship between the presence or absence of life events and myocardial infarction (X^2=0.73; df=1).

Study of the presence of life events and smoking habit shows also no significant relationship between the two variables.

ANALYSIS OF OTHER FACTORS INCLUDED IN THE STUDY FOR ALL CASES.

It was evident from a study of respiratory symptoms among patients that 68% of those recording sputum were smokers compared to only 48% of those without sputum. Statistically there is no significant relationship between smoking habit and the presence of sputum for as much as three months in the winter each year. However recurrence of sputum lasting for 3 weeks or over for more than one period in the last three years before admission was much more common in smokers (46%) than in non-smokers (11%) ($p< 0.01$).

Further analysis of the other factors included in this study showed no significant relationship between presence or absence of acute myocardial infarction with any of the following variables: age, angina, indoors or outdoors work, tea , coffee or alcohol consumption, haemoglobin, blood sugar or blood triglycerides. The relationship between the presence of acute myocardial infarction and having a high cardiac enzymes (SGOT, CK, LDH) is highly significant ($p<0.001$) , reflecting the criteria used for the diagnosis of myocardial infarction. The relationship between the presence of acute myocardial infarction and having chest pain later on is significant ($p<0.05$). It is not surprising that patients suffering from MI are more likely to suffer from chest pain later on. The relationship between smoking habit and respiratory function tests (FVC or FEV_1) was also not significant.

4.2 COMPARISON BETWEEN BREATH CARBON MONOXIDE AND BLOOD CARBOXYHAEMOGLOBIN

COMPARISON BETWEEN BLOOD COHb AND BREATH CO.

Table 10 and Fig 1 show a comparison between breath carbon monoxide in a randon subsample of 42 patients measured by GMI CO-monitor in ppm and blood COHb measured by CO-oximeter-282. In each case the mean of 5 readings was calculated. The mean for blood COHb is 7.98 ppm and for breath CO is 5.96 ppm. For comparison the differences between the value obtained by the two methods in each patient have calculated. The overall mean of the differences ($\bar{d}$) is 2.02 ,variance 10.7 and SD is$\pm$ 3.3. Statistically (paired t-test for comparison between two means) CO is apparently present in higher concentration in blood than in breath CO ($p<0.001$) as might be expected. A 95 % confidence limit for $\bar{d}$ is 1.2 and 2.9. Figure 1 shows the differences between both methods used for each patient and it is evident that although the mean difference is small (2.02), some of the individual differences are quite large which may indicate poor agreement between the two methods.

Figures 2, 3 and table 10 show at different CO means for the 42 patients the relationship between blood COHb and its coefficient of variation (CV) and breath CO and its coefficient of variation. There is an inverted (J) shaped curve; this implies that the variability is proportionately higher in smaller CO concentrations.

4.3 CARBON MONOXIDE MEASUREMENTS IN BLOOD AND EXPIRED AIR: CHANGES OVER TIME AND ACCURACY

Table 11 shows the reliability of blood COHb measurements analysed immediately after the sample was taken and also 24 hours later for 8 blood samples. Each sample was analysed five times. Reliability for the measurements for the whole group analysed immediately is 0.99 and for each measurement in this group is 0.99. Reliability of COHb analysed 24 hours after the samples were taken is slightly lower, 0.98 for the group and 0.94 for each single measurement. Comparison of the difference between the 2 methods for the 8 patients shows that the overall mean difference is 0.05, SD $\pm$ 0.55 and variance is 0.3 .

Table 12 shows mean and coefficient of variation of COHb measurements for a further group of 21 patients. The samples were measured by CO-oximeter 282. Each sample was measured five times. The relationship between the mean COHb and coefficient of variation also again is an inverted J shaped curve which means that the variability is proportionately higher in smaller COHb concentration. By using the analysis of variance reliability test, reliability of each measurement is = 0.94. This high reliability value indicates that the average intercorrelation between ratings given by pairs of judges (the two methods) is high (167).

Table 13 shows the effect of time and storage on further 21 venous blood samples analysed immediately after the sample was taken and 24 hours after by CO-oximeter-282. The mean for the first half (immediate analysis) is 4.4% saturation. The mean for the second half is higher (5.6% saturation). The mean of differences between the two methods for each patient is = 1.2, SD ±2.0 and variance 4.0 . There is no evidence however of significant increase in COHb concentration with storage. (Paired t test = 1.6; df=20; N.S).

Table 14 compares two methods for COHb measurement ; gas-chromotography and spectrophotometry by CO-oximeter-282 in 31 venous blood samples. The mean COHb measured by spectrophotometry methods is 6.1 ppm, SD ± 6.6 while the mean COHb measured by gas-chromatography is 5.6 ppm, SD ± 2.4. The mean of differences between the two methods for each patient is $(\bar{d})$ =0.55, SD ±7.2 and variance is 52.1. A 95 % confidence limit for the mean of differences is -1.7 and 2.8 which means that there is no evidence of bias between the two methods but many of the individual differences are very large (Fig. 4). It is evident that there are large disagreement between the measurements, but without a reference standard it cannot be deduced which is "better".

RAPID CHANGES IN CO MEASUREMENTS IN BLOOD AND BREATH.

Table 15 shows the change in blood COHb level in 6 random subjects measured every 30 minutes. 3 samples were taken from each subject 30 minutes apart and analysed by CO-oximeter 282. Reliability of the whole group is (rk) = 0.95 and reliability of each single measurement (r1) = 0.87.

In a second experiment the decay curve of breath CO was done in a group of 6 different subjects after smoking a single cigarette. The results are shown in Table 16 (raw values) and Figure 5 (values after logarithmic transformation). Breath CO was measured every 15 minutes by GMI portable CO monitor. The fitted regression lines (Fig.5) for values after smoking show a gradual decrease in breath CO over time. The "outlier" in Fig. 5 was excluded in the calculation of CO half life. The mean slope for breath CO regression is - 0.0048 and the mean intercept is 3.7. Calculation of the half life for each subject showed that it was on average 182 minutes with SD $\pm$ 15.3.

4.4 BREATH CARBON MONOXIDE STUDY FOR GLASGOW HIGH SCHOOL PUPILS AND RUCHILL HOSPITAL EMLOYEES

Age and smoking habit

Out of 105 total pupils included in the study 83% were in age 15-18 years, 17% were less than 15 years. Two-thirds of the pupils examined were non-smokers and nearly one third admitted to being smokers (34%). Out of the smokers, 72% smoke only from 1 to 4 cigarettes per day, the other (28%) of smokers use between 5 and 10 cigarettes per day. In the sample males (61%) outnumbered females (39%).

Family smoking habit:

Fifty six per cent of the pupils' families had no members who were a smoker. In 16% of the families only the fathers were smokers and in 13% of the families one or other parent was a smoker. In the remaining 15% , either the brother or sister were the only smokers in the family.

SMOKING HABIT AND BREATH CO OF THE HIGH SCHOOL PUPILS

Table 17 shows that most of the pupils (85%) have a breath CO within normal range and 15% have a high level (>10 ppm). 25% of the smokers (n=36) have a high breath CO.

Breath CO measurements were divided into two groups less than 10 ppm and more than 10 ppm(161), the sensitivity of breath carbon monoxide is 0.25, and specificity is 0.9. The

predictive value of a positive test is 56% and for a negative test is 70%. Chi-square test shows a significant relationship between smoking habit and breath CO with a higher level of CO in smokers. (X^2 =4.04; 1 degree of freedom; $p<0.05$).

Table 18 shows the time between last smoking and breath carbon monoxide test for Glasgow High School pupils who smoke (n=36). It is evident that 81% out of the smokers had not smoked within 12 hours of the test. 24% (7/29) of the smokers had a high breath CO (10ppm and over) and they had not smoked for more than 12 hours. 29% (2/7) of smokers had a high breath CO and smoked within 12 hours of breath test.

CROSS-REACTIVITY WITH BREATH HYDROGEN.

Table 19 shows no significant relationship between smoking and breath hydrogen among pupils (X^2 =0.06; 1df). Breath hydrogen in the pupils was highly correlated with breath CO (r= 0.75) and it was highly significant ($p<0.001$). In hospital employees breath hydrogen was poorly correlated with breath CO (r=0.19).

SMOKING HABITS AND BREATH CO FOR RUCHILL HOSPITAL EMPLOYEES

Age characteristics: Out of the total of 143 employees seen in the study 42% were aged between 20-30 years, 19% were between 30 and 40 and 16% were between 40 and 50 years. In the sample females (67%) outnumbered males. There was little sex difference as regards smoking. 38% of males were smokers compared to 42% of females.

Smoking habit: Most of the employees (59%) were non-smokers and 41% were smokers. 4% of the employees were a pipe smokers. Only 15% of smokers smoked less than 5 cigarettes per day, 16% smoked 5-15 cigarettes, 47% of the smokers smoked between 15-25 cigarettes and 19% smoked more than 25 cigarettes per day.

Table 20 shows the relationship between breath CO and smoking habit for Ruchill Hospital Employees. 74% of smokers have more than 10 ppm CO, compared to 11% of non-smokers. The sensitivity using this cut-off point is 0.74, and specificity 0.89. The predictive value of the positive test is 83 % and for the negative test is also 83 %. There is a significant relationship between smoking habit and breath CO, with much higher levels in smokers in this population (X^2 =60.17; 1 df; $p<0.001$).

Table 21 shows time between the last cigarette smoked and breath CO for Ruchill Hospital employees. Sixty four per cent of the smokers finished their last cigarette less than one hour before the test, 72% of them having a high breath CO (more than 10ppm). Comparison between an interval of less than one hour and more than one hour shows that there is a significant relationship between breath CO and time since last cigarette the longer the time the lower the CO level (Chi-square= 16.7; 2df; $p<0.001$).

Table 22 shows that breath hydrogen was similar in smokers and non-smokers, the exact probability test being not significant at the 5% level (P=0.09). Breath hydrogen was poorly correlated with breath CO in the hospital employees ($r=0.19$, $p<0.001$). This result, which is in contrast to that found in school children, may be due to the differences in their smoking habit.

4.5 ACCURACY OF BREATH CO: SENSITIVITY AND SPECIFICITY

Table 23 shows comparison between sensitivity and specificity for breath CO at different CO levels for Glasgow High School pupils and Ruchill Hospital employees. It is evident from these tables that as expected, increasing the **cut-off level** decreases sensitivity and specificity correspondingly increases. The same finding is found in the results from the pupils.

Figure 6 shows the Receiver Operating Characteristics Curve for breath CO in both Ruchill Hospital employees and Glasgow High School pupils. In this curve the X axis represents the False positive percentage which is equal to 100- (specificity X 100). The Y axis represent the true positive percentage. The curve for the hospital employees is higher than that for the school pupils. Assuming what they said about their smoking habit was true, the minimum error rate for breath CO occurs at cut off points between 8 and 12 ppm for the hospital employees , and at 7 ppm for the High School pupils.

4.6 GENERAL DESCRIPTION OF THE FOLLOWED-UP GROUPS AND COMPARISON BETWEEN THEM

The psychological profile for the health education patients was assessed by the health visitor involved in the health education. The distribution of health and psychological problems was as follow:- 39% had normal psychlogical profile defined as within 1 S.D of the published means (141a). Out of the rest who had high scores for at least one factor, 69% of the patients had free floating anxiety, 50% had phobic anxiety, 18% had obsessive compulsive traits, 64% had somatic symptoms, 11% had depressive tendency and "hysterical trait" was noted in 10% of males and 23% of females.

4.6.1 CHANGES OF SMOKING HABITS FOR BOTH GROUPS OF PATIENTS

Table 24 shows that 33% of patients in the non-intervention group were aged 45-55 and 54% were older than 55 years. Males comprised 75% and females 25% of the non-intervention cases. Table 25 shows that in the health education group 25% of patients were younger than 45 and 54% were older than 55 years. 67% were males and 33% were females. Fisher's exact probability test was used for comparison of the frequency of observations in a fourfold table when the numbers are too small for a Chi-square test. Within each group, the age distribution of each sex was similar. Further comparison of the age distribution between the two groups was not significant (Chi-square=2.54; 2df) and for sex distribution was also not significant (Chi-square =0.55; 1df).

AGE DISTRIBUTION AND ACUTE MYOCARDIAL INFARCTION IN BOTH GROUPS

43% of the patients in the non intervention group and 67% in the health education group suffered from acute MI. In each group and between both groups there is no significant relationship between age and presence of acute MI.

AGE WHEN SMOKING BEGAN AND NUMBER OF CIGARETTES SMOKED PER DAY AT THE TIME OF STUDY

In the non-intervention group, 37% began smoking under the age of 15. 46% began smoking between 15 and 25 years. In comparison 15% of the health education group began smoking under the age of 15, and 75% began smoking aged between 15 and 25. In each group the older the age of starting smoking the lower the number of cigarettes smoked now per day ($p<0.001$). Comparison of the age when smoking began in both groups shows no significant difference ($X^2 = 4.6$; 4 df).

SMOKING HABIT OF THE PATIENT'S FAMILIES

Study of the smoking habit of the patient's families showed that in the non-intervention group, 71% of the smokers had at least one family member who was a smoker, 42% of them had more than one. In comparison with the health education group 73% of the smokers had at least one family-member who smoke, 64% of them had more than one smoker. The difference was not significant.

SMOKING HABIT AND SOCIAL CLASS FOR BOTH GROUPS

In Table 26, the social class distribution of the non-intervention group was as follows:- patients in social

classes I and II 15%, III NM 7%, III M 35% and in classes IV,V 14%. 29% were unemployed or retired (X^2=0.25; 1df; N.S). In Table 27, the health education group social classes was distributed as follows:- I, II and III NM 25% and 30% in social classes III M, IV and V , unemployed and retired patients were 45%. Fisher's exact probability test was used for collapsed table (social class I,II,IINM and the others) due to small expected frequencies (P=0.31; N.S). As expected most of the patients were in low social classes. In each group there is no significant relationship between social class and smoking habit. The distribution was similar in both groups.

SOCIAL CLASS AND RESIDENCE AREA FOR MOST OF THE PATIENTS LIFE

Comparison between the two groups as regards the area of residence shows that in each group most of the patients were living in cities for most of their life. There is no significant difference between the two groups as regards their social class distribution and their area of residence for most of their lives.

RELATIONSHIP BETWEEN PATIENTS KNOWLEDGE ABOUT CORONARY HEART DISEASE RISK FACTORS AND SOCIAL CLASS.

Awareness about CHD risk factors was assessed by having the subject identify the correct answers from a list of possible factors (see Appendix A1). The maximum possible score was 16. The mean score and SD for each social class group is shown in tables 28 and 29. Oneway ANOVA for non-

intervention group F = 2.08; df= 2,60. F was 0.56; df= 2,21 for the health education group; both were non-significant. In each group there was no significant relationship between social classes (I,II, IIINM) and the others and knowledge score.

MYOCARDIAL INFARCTION AND PATIENT'S KNOWLEDGE ABOUT CORONARY HEART DISEASE PROBLEM

In non-intervention group, 43% (n=26) had acute MI and 73% of them had a satisfactory amount of knowledge. In the health education group, 67% (n=16) had a myocardial infarction 76 % of them had good knowledge about CHD problem. In each group there is no significant relationship between patient's knowledge about CHD and the presence or absence of myocardial infarction. There is no significant relationship also between both groups as regards patient's knowledge about CHD problem and presence or absence of myocardial infarction.

LIFE EVENTS IN THE YEAR BEFORE HOSPITAL ADMISSION

Table 30 shows that in non-intervention group, 29% of patients (n= 18) suffered from at least one stressful life event in the year before hospital admission. Patients who had more than one event comprised about 15%. Chi-square =0.0; 1df between non and presence of any life events. Table 31 shows that in health education group 37% patients reported at least one stressful life event (Chi-square =0.08; 1df; N.S). There is no significant relationship between those have and those not reported life events in the past and the presence or absence of myocardial infarction.

STRESS AND MYOCARDIAL INFARCTION

From Table 32, in the non-intervention group, 68% suffered from moderate or severe stress, and 32% had mild or no stress (Chi-square =0.48; 1df ; N.S). In the health education group as evident from Table 33, 88% suffered from moderate or severe stress and 12% had mild or no stress (Fisher's exact probability test = 0.22; N.S). There is no significance difference in each group between stress and the presence or absence of MI. There is no significant relationship also between the two groups as regards the presence or absence of stress (X^2 =3.26; 1df).

COMPARISON BETWEEN LABORATORY FINDINGS IN BOTH GROUPS

Study of laboratory findings for both groups in Table 34 showed that there is no significant difference between the two groups as regards the presence of MI, presence of high triglycerides, cholesterol, Haemoglobin, blood sugar, high cardiac enzymes (CK > 160 U/L, LD enzymes >230 U/L) between both groups. In each group, high cardiac enzymes were significantly related to the presence of myocardial infarction as expected.

CARBOXHAEMOGLOBIN AND DAILY CONSUMPTION OF CIGARETTES

Tables 35 and 36 show that at entry to the study 44% of the non-intervention group, smoked less than 15 cigarettes per day and 30% smoked between 15-25 cigarettes per day. 90% have high blood COHb over 2% saturation. The mean COHb was 3.0% saturation for patients smoking less than 15 cigarettes per day, 3.5% for those smoking between 15 to 25 and 3.4% for

those smoking over 25 cigarettes per day. There is no significant relationship between COHb and the number of cigarettes smoked per day in the non-intervention group. (Oneway ANOVA done on raw values gives F=0.98; df= 2,60; P=NS). In the health education group 83% have high blood COHb (over 2% saturation). The mean COHb for the group smoking less than 15 cigarettes per day is 2.5% saturation and 2.9% for those smoking more than 25% cigarettes per day. There is no significant relationship between COHb and the number of cigarettes smoked per day in the health education group. Oneway ANOVA done on raw values gives F=0.16, df=1,22; N.S.

FOLLOW-UP COHb LEVEL AND SMOKING HABIT

In Tables 37 and 38 patients having a high COHb (more than 2%) comprised 51% of the non-intervention and 46% of the health education group. In the non-intervention group, 41% are non-smokers, most of them (89%) having a normal COHb. The mean COHb for patients who had given up smoking, those smoking less than 15 cigarettes per day, 15-25 per day and more than 25 per day were 1.2, 2.2, 2.6 and 1.8 respectively. In the health education group 63% were non-smokers and most of them (73%) had a normal COHb. The mean COHb for patients who had given up smoking, those smoking <15 and 15-25 per day were 1.5, 2.2 and 2.4% saturation respectively. Oneway ANOVA was used for comparison between the means of COHb for more than 2 groups at the same time. Oneway ANOVA is significant in the non-intervention group (F=13.8; df= 3,59; p<0.001). In the health education group this relation is not significant (F=2.86; df= 2,21). Comparison between the groups as regards

COHb distribution shows no significant difference in follow-up COHb (normal and high COHb levels) between the two groups (X^2 = 0.17;1 df; N.S). The proportion of smokers with COHb of over 2% who stopped smoking was 11.5% (3/26) in the non-intervention group and 37% (4/15) in the health education group. The difference was not significant (X^2=1.55; 1df;N.S).

COMPARISON BETWEEN BLOOD CARBOXYHAEMOGLOBIN MEASUREMENTS AT ENTRY AND FOLLOW-UP.

In Tables 39 and 40 the difference between entry COHb and follow up COHb were calculated. The mean of differences in the non-intervention group is 0.83% saturation and - 1.16% saturation in the health education group. There is no significant difference between COHb level in the non-intervention group at entry and follow-up (Paired t test= 1.85; 62 df;N.S). In the health education group there is also no significant difference between the COHb level at entry and follow-up (Paired t test =0.93; 23 df;N.S).

CARBOXYHAEMOGLOBIN AND TYPE OF HEATING IN THE HOUSE

In Tables 41 and 42, the type of heating in the house has been studied because persons may be exposed to other sources of CO other than smoking eg. open fire or gas in absence of sufficient ventilation can be a source of CO. It was found that 36% of the followed-up patients used electricity for heating and, 25% use central heating 14% use mixed types (more than one type). The mean percentage saturation of COHb for those use electricity as a source of heating is 0.05 %, 4.1 % for those use open fire or gas, 2 %

for those using central heating and 1.0% for those using mixed types. About twenty five percent use gas or open fire heating. There is no significant relationship between the type of house heating and blood COHb (Oneway ANOVA =1.02; df= 2,81; N.S).

COMPARISON BETWEEN INITIAL NUMBER OF CIGARETTES SMOKED PER DAY FOR BOTH GROUPS.

Tables 43 and 44 show the differences between the number of cigarettes smoked per day at entry minus the number of cigarettes smoked at follow up. The mean of the differences in the non-intervention group is -1.03 and SD ±7.3 and in health education group is 8.2, SD ±9.1. There is no significant decrease in the number of cigarettes smoked in the non-intervention group (Paired t test = 1.12; df=62; N.S) compared to the highly significant decrease in the health education group (Paired t test=4.4; df=23; $p<0.001$).

CHANGES IN SMOKING HABIT BETWEEN INITIAL AND FOLLOW-UP EXAMINATIONS

The follow up self-reported smoking habit in Table 45 shows that in the non-intervention group, 41% of the patients who were originally smokers (n=63) had stopped smoking after the initial examination. In the health education group 63% had stopped smoking. All patients in non-intervention and health education group were originally smokers. Patients restarting smoking having originally been recorded as non-smokers were not assessed. Comparison of the changes in smoking habit of the two groups at the follow-up examination shows no significant difference between both groups (Chi-

square=3.14; 1df; N.S).

Comparison between smoking habit of the two groups at entry and follow up by use of breath CO monitor at a cut off point of 9 ppm (Table 45a) shows that 8% (5/63) of the patients in the non-intervention group had misreported their smoking habit in comparison to 17% (4/24)in the health education group. Statistical comparison between the two groups defined this way was also not significant (X^2=1.17; 1df; N.S).

SMOKING HABIT AND MYOCARDIAL INFARCTION

In the non-intervention group 38% of MI patients were smokers and in the health education group 60% of MI patients in this group were smokers.

FOLLOW-UP BREATH CARBON MONOXIDE AND SMOKING HABIT

From Tables 46 and 47 it was evident that at follow-up, 59% of non-intervention group were smokers, 78% of smokers had a high breath CO more than 10 ppm. Out of the ex-smokers 19% had a high breath CO (X =21.5; 2df; $p<0.001$). Table 47 shows that in the health education group 38% were smokers. Out of the smokers 89% (n=8) had a high breath carbon monoxide. 27% of ex-smokers (n=4) had a high breath CO (X^2 =8.7; 1 df; $p<0.01$ for collapsed table <10 and 10 ppm and more). In each group there is a significant relationship between smoking habit and breath CO with a higher level of CO among smokers than non-smokers as expected.

COMPARISON BETWEEN FOLLOW UP BLOOD COHb AND BREATH CO.

The opprtunity was taken to compare blood COHb and breath CO in the combined groups, (Table 48) as done earlier (Table 10). The mean of the differences between blood COHb minus breath CO was - 7.7 (SD± 7.1) and the paired t test =10.1; df=86; $p<0.001$. This highly significant difference between the two variables is seen in both the non-intervention group (Paired t test= -9.6; df=62; $p<0.001$) as well as in the health education group (Paired t test = -3.8; df=23; $p<0.001$).

4.6.2 RESULTS OF FURTHER ANALYSIS OF OTHER FACTORS INCLUDED IN THE STUDY

Respiratory symptoms

In the non-intervention group, 52% had cough, 49% suffered from morning sputum , all of them were smokers. 51% suffered from chest wheezes, 74% suffered from sputum for 3 months and 50% suffered from sputum for 3 weeks more than once in the last 3 years.

In health education group, 29% had cough, 51% suffered from morning sputum. Thirtythree per cent suffered fron chest wheezes, 69% suffered from sputum for three months and 44% suffered from phlegm for three weeks more than once in the last three years. There is no significant difference between both groups as regards the previously mentioned variables. There is no significant relationship between either FEVOL1 or FVC and the number of cigarettes smoked per day in each groups.

Electrocardiogram findings and myocardial infarction.

In both groups the incidence of T-inversion, presence of arrythmia, bundle branch block and left ventricular hypertrophy were similar. The realtionship between ST elevation or depression and MI is significant in the non-intervention group ($p<0.001$). The presence of Q wave and myocardial infarction also is significant in each group ($p<0.001$) indicating that there is a relationship between Q wave, ST changes and the diagnosis of acute myocardial infarction.

Myocardial-infarction and recurrence of chest pain

In non-intervention group at follow-up patients reporting chest pain (64%) are nearly double those without chest pain (36%). In the health education group, the figures are 52% with and 48% without chest pain.

In the non-intervention group there is a significant relationship ($p<0.05$) which means that in this group patients initially suffering from acute myocardial infarction were more liable to suffer from chest pain later on. The results are not significant in the health education group.

Comparison between some other characteristics in the two groups

Table 49 shows no significant difference between the two groups as regards age, sex, weight, height,consumption of alcohol tea or coffee, systolic BP, diastolic BP and activity in work. Finally there was no significant relationship between carboxyhaemoglobin and intermittent claudication in

each group.

4.6.3 CHARACTERISTICS OF THE GROUP GIVING UP SMOKING

Study of some of the characteristics of patients who gave up smoking and those who continue to smoke showed that there was no significant difference in the age distribution of those gave up smoking between both groups (X^2 =0.95; 2df). There was also no significant relationship with sex (non-intervention group X^2 =1.13; 1df; and health education group X^2 =0.53; 1df; N.S). Of those who gave up smoking, 28% of the non-intervention group were in upper social classes (I,II,IIINM) compared to 44% in the health education group.

In each group there was no significant difference between those who gave up smoking and those who continued to smoke as regards either the presence or absence of chest wheezes, angina, and amount of knowledge about CHD (in non-intervention group X^2 =0.24; 2df and in health education group X^2 =1.4; 2df)

Comparison between inhalation behaviour of both groups at entry and at follow up showed that there was no significant difference between the two groups (X^2 1.14; 1df; at entry and X^2 =1.32 at follow up.)

DISCUSSION

5.1 CHARACTERISTICS OF THE STUDIED POPULATION AND CORONARY HEART DISEASE RISK FACTORS

It is well known that coronary heart disease (CHD) is a major public health problem in terms of the number of patients who need treatment and in terms of the number of deaths. The adverse effect of cigarette smoking on non-fatal myocardial infarction (MI) is well recognised.

For CHD as for many other diseases, diagnosis, together with secondary and tertiary prevention, is not sufficient, and primary prevention is essential. For such an approach, whether at individual or community level, health education is a necessary part of health promotion.

It is recognised that although the public knows about CHD, few individuals are ready to apply what they know because they may think that the scientific evidence is still controversial. Alternatively, they may think that there are more important things to worry about than one's own health in the distant future, or they may not care very much about longevity. Epidemiological studies have indicated that the risk of CHD increases with cigarette consumption. Two major constituents of tobacco smoke, nicotine and carbon monoxide (CO), are of prime importance in exacerbating cardiovascular events. A rapid reduction in the risk of myocardial infarction has been reported for ex-smokers (4,49). The

stress of hospital admission with acute chest pain gives an invaluable opportunity for secondary prevention, for example by reducing cigarette smoking which even at this stage can reduce the risk of future MI (142).

5.1.1 HEALTH EDUCATION AND SMOKING HABIT

This study compared the effectiveness of two methods of health education in preventing smoking. The first method was the normal care and advice given to all patients admitted to the coronary care unit (CCU) and the second was intensive health education which was given to CCU patients who lived in Clydebank. The second method was for a health visitor to give tailored health advice, particularly about smoking, to patients in hospital, with follow-up visits in their home after discharge. Breath and blood COHb were used to measure the success of anti-smoking health education in each group of patients.

Sometimes skilled physicians examining a patient may disagree regarding the findings. Such disagreements reflect the imperfect reliability of the clinical methods used. Today, consumers, insurers as well as physicians and their students must become aware of the imperfect reliability of the methods and data of clinical medicine. Reflecting this concern, this study was done to validate two of the methods commonly used for measuring carbon monoxide. The first method measured blood carboxyhaemoglobin (COHb) and the second measured breath carbon monoxide (CO). It also included the

study of the relationship between breath CO and blood COHb. The repeatability and validity of each method was assessed. The results are discussed later.

Out of the total cases (n=212), 116 were smokers (55%). 95% confidence intervals are 52.8% to 66.8%, and this group was followed-up one year after hospital admission. The response rate 81% (94 cases) for the assessment of the two health education interventions was good. This could be explained by the use of a second letter and telephone call for non-responders. Successful follow-up of 80 patients in group I (non-intervention) and 25 in group II (health education group) and an expectation of a 30% reduction in smokers in group I and 60% in group II was calculated to give a 80% chance of detecting a significant difference ($p<0.05$) (47). However due to difficulty reasons in ensuring complete follow up of all patients, the study ended with a power of about 70% (47).

Mortality was 8% (7 cases and 95% confidence interval 3.0% to 14.7%) amongst the followed-up smokers (n=116). Survivors (n=87) were contacted successfully, 24 cases from the Clydebank area and 63 cases from the catchment area of the Western Infirmary. Smoking habit, social, medical, laboratory findings and other factors were compared in both groups. As regards reliability of self reported smoking behaviour,it was generally felt that confidential smoking questionnaires can give reliable results, but this

reliability was improved by the use of an objective test (141).

About 55% of the total patients at the time of admission were current smokers, 50% of the total were in the age group 55-65 (Table 1) and 28% were in age group 45-55 years. The higher percentages of smokers among the older age groups could be explained by the fact that older cohorts continued to smoke more than younger ones who did not start smoking or had given up smoking earlier. However out of the total number, 85% were either smokers or ex-smokers (95% confidence interval 79.7% and 89.0%). This percentage is much higher than that reported in Scotland (136). In Scotland 38% of men and 33% of women were smokers in 1982 and it is reported from Glasgow that 45% of men and 43% of women were smokers (75). There is no relationship between age and smoking habit. The higher percentage of smokers in this study of patients with chest pain and myocardial infarction agrees with published results (3,4,51,64,107,151).

As regards sex and smoking habit more males (57%) included in the study were current smokers (Table 2) in comparison to females (47%). Combining" Smokers " and "Ex-smokers" in one group gives a significant relationship and males are less likely to have never smoked ($X^2=5.4$; 1df; $p<0.05$). This finding is in agreement with published literature (136). The percentage in both sexes was higher than that in the general population (8). However Tables 3a and 3b show a significant relationship ($p<0.05$) between the

age at which smoking began and the number of cigarettes smoked per day at the time of hospital admission in each sex.

Out of the male smokers, 31% began smoking under the age of 15 years and 48% began between 15-25 years, which agrees with previous results (133). Out of the female smokers 36% began smoking under the age of 15 years and 64% began after age 15 which agrees with previous results (133). However there is a significant relationship between sex and the age of begining to smoke. This young age of starting smoking can be explained by the effect of peer group pressure. It may also be due to the presence of a smoker in the family, or due to lack of interest in school, or teachers who smoke and pressure from smoking friends (136). The psychological reinforcements of nicotine could be another factor, (102) or smoking may represent both a symbol for adult status and rebellion against adult authority (70).

5.1.2 RELATIONSHIP BETWEEN SMOKING AND CARBON MONOXIDE

Carbon monoxide can be produced both physiologically and pathologically, however cigarette smoking is a common and an important source of CO (132,157). The presence of CO in the blood of non-smokers can sometimes be explained by significant endogenous CO formation, particularly in haemolytic conditions. It is suggested also that CO is formed by the clearing of the porphyrin ring in choleglobin formation. Elimination of CO in expired air has been shown to be a physiological phenomenon. The strong affinity between CO and haemoglobin (Hb) plays a role in the excretion of this

product. Analysis of expired air CO as an indirect measure of COHb, which avoids the inconvenience of taking a blood sample, is now frequently used in population studies. The close correlation of breath CO with blood COHb was demonstrated in many studies done in Britain and the USA. It has been shown that breath CO is also associated with cigarette smoking as well as exposure to atmospheric pollution with CO. Therefore COHb levels can be estimated reliably by measuring the concentration of CO in breath.

Study of the relationship between smoking habit, number of cigarettes smoked per day and blood COHb at entry examination (Tables 4 and 5) shows no relationship (χ^2 = 2.0; 4df). This result was not expected when the study was planned, which may be due to inaccuracy and instability of the spectrophotometry method used (CO-oximeter 282) (20,33a). Inhaled oxygen during the emergency phase could be another factor. Similar results were obtained by a second CO-oximeter machine in Western Infirmary. The short half life of CO (between 2-4 hours) mentioned in the literature could be another explanation. Furthermore all CO absorbed by man can be recovered in the expired air if this is collected over a sufficient period of time. On the average it takes 250 minutes for CO to fall to half its original level (122), in this study it was 182 minutes.

5.1.3 SMOKING HABIT, ANGINA, MYOCARDIAL INFARCTION AND STRESS

There are indications in the literature that CO can cause earlier onset and longer duration of pain (6). Angina

was found also to be 70% higher among cigarette smokers in a health insurance plan in New York city (48). Smoking also increases the heart work load which increases oxygen demand (11). In this study (Table 6) there is no significant relationship between COHb and the presence or absence of mild or severe forms of angina in contrast to the previously published literature (15,16,95). This negative finding could be explained by the variation in smoking habit among smokers or inaccuracy and instability of the spectrophotometry method used for measuring blood COHb. Study of the relationship between COHb and the presence or absence of MI is evident from Table 7 which shows that COHb has an inverse relationship with the presence or absence of MI and patients with MI are more likely to have a COHb level within the normal reference range - a finding at variance with published results (23,79,154). This relationship may then be a chance finding.

It has been known for some individuals that smoking can relieve tension and stress as a result of the psychological enhancement produced by nicotine. This enhancement is followed by depression later. Study of the problem of stress (Table 8) by scoring the answers of questions about stress (see Appendix A) and smoking habit before hospital admission clarified that there was no significant relationship between patient's smoking habit and stress. The explanation may be that stress may not be the factor for maintenance of smoking behaviour. It has been mentioned also that the psychosocial forces accounting for the initiation of the smoking habit

differ from those that account for its continuation (70). However, smokers who suffer from moderate or severe stress contributed a 42% (89/211) of all patients. In each of the followed up groups there was no significant relationship between stress and myocardial infarction(Tables 32 and 33).

In this respect, life events in the year preceeding hospitalisation also show no significant relationship with MI (Table 9). Death of a family member was the commonest reported event (11%). 14% of the cases had reported more than one event. It is well known that CHD is a multifactorial disease, and while stress in the form of life events in the year preceding hospitalisation is a factor, it is unlikely to have an important effect on its own.

5.2 COMPARISON BETWEEN THE TWO METHODS USED FOR MEASURING CARBON MONOXIDE, CHANGES OVER TIME, AND ACCURACY.

A study carried out by Hill et al (68), they clarified the high correlation coefficient between plasma cotinine and blood COHb (about 0.7), and between both and nicotine (0.73), higher than with thiocyanate (0.56). When smokers were separated into those smoking cigarettes yielding more or less than 1 mg nicotine per cigarette COHb was the better indicator of daily exposure and absorption with increasing consumption, COHb and plasma nicotine showed a comparable progressive increase in men and women in a linear relationship.

Expired air carbon monoxide testing can efficiently dichotomize smokers and non-smokers (153). It was also found that only the number of cigarettes smoked per day and the longest period a smoker has quit smoking in the past were significantly associated with levels of carbon monoxide. In this respect the study of the relationship between breath CO and blood COHb concentration for a random subsample of 42 persons (Table 10) was carried out for comparison between the two methods used. The differences between the value obtained by the two methods in each patient have been calculated. The overall mean of differences ($\bar{d}$) is 2.0, variance 0.7 and the SD is $\pm$ 3.3. Comparison between the two means by paired t test shows that CO is mostly present in a higher concentration in blood than in breath ($p<0.001$) as might be expected (81,121,122). Nintetyfive per cent confidence limits for the mean of differences between the two methods are 1.2 to 2.9. In this table although the overall mean difference (d) is small (2.0), but some of the individual differences are quite large which may indicate a poor agreement between the two methods (Table 10).

In a paper published in 1986 by Bland and Altman (20a), it was shown that the use of the correlation coefficient for comparison between two methods is misleading. The causes mentioned were (1) Correlation coefficient measures the strength of a relation between two variables and not the agreement between them unless the points are on the line of equality with similar scales. (2) Correlation coefficient is not affected by the scale of measurements which certainly

affects agreement. (3) Correlation depends on the range of the true quantity in the sample. (4) Data which seems to be in poor agreement can still produce quite high correlations.

Figure 1 shows the difference between blood COHb and breath CO for each patient. The overall mean difference is small, once again some of the individual differences are quite large which indicate poor agreement between the two. The curves for the coefficient of variation for blood COHb (Fig. 2) and breath CO (Fig.3) have an inverted J shape which indicates a proportionatly higher variability in small CO concentrations and vice versa.

It was interesting to assess the effect of storage on blood COHb. Blood samples were analysed immediately and 24 hours later after they had been kept in a refrigerator (Table 11). The effect of storage on blood COHb was shown by the use of an analysis of variance reliability test (167). Reliability of immediately analysed COHb measurements was 0.99 and reliability for each single measurement in the group was 0.99. Analysis 24 hours after the samples were stored showed a similar reliability (0.98) for the group and slightly lower (0.94) for each single measurement in the group. The slightly lower reliability after 24 hours storage may be due to interference from pigments produced as a result of breakdown of haemoglobin which can affect light absorption in spectrophotometery (20). Comparison of the differences between the two methods for each patient shows that the overall mean of differences ($\bar{d}$) is 0.05, SD $\pm$ 0.55 and

variance 0.3. The overall mean of difference is very small and variation between the two measurements is also very small. This finding indicates that putting blood samples immediately into a refrigerator will not markedly affect CO in the sample. Storage for longer time may have an effect.

The relationship between the arithmetic mean and coefficient of variation of COHb for a further random subsample of 21 venous blood samples (measured by CO-oximeter 282) was also an inverted J shaped curve (Table 12). This relationship indicates a proportionatly higher variability in smaller COHb concentrations, as found already. The variation was marked between 2.5 to 7.5 ppm COHb. Reliability of each single measurement in this samples is 0.94 which, means that there is a high intercorrelation between ratings given by the two methods.

The effect of 24 hours storage on COHb was also studied in another random subsample of 21 venous samples (Table 13). Each sample was divided into two halves. The part analysed immediately has a mean of 4.4, SD ± 2.4 and for the later analysis the mean COHb = 5.6, SD ± 2.1. The mean of the differences is 1.2, SD ± 2.0 and variance is 4.0. There is no significant increase in COHb concentration after storage for 24 hours in the refrigerator (Paired t test =1.61; NS) which is similar to the findings from Table 11.

It has been mentioned in the literature that the Gas-chromatography method for measuring blood COHb is more

accurate than spectrophotometry (20). Therefore the two methods used for measuring blood COHb were compared, (Table 14 and Fig.4). The mean of CO measured by CO-oximeter 282 is 6.1, SD ± 6.6 and by gas-chromatography the mean is 5.6, SD ± 2.4. The overall mean of the differences ($\bar{d}$) between the two methods is small = 0.6, the SD ± 7.2 and variance 52.1 which indicate a high variability and poor agreement between the two methods. Without a standard reference it cannot be deduced which is "better".

In order to see if rapid changes occurred during measurement of blood COHb in fresh blood samples, analysis was done every 30 minutes. Reliability of COHb measured every 30 minutes on 3 occasions in 6 subjects was 0.95 for the whole group, and for each single measurement was 0.87 (Table 15). In a second experiment (Table 16) the decay curves for breath CO was measured every 15 minutes for about 3 hours for 6 healthy persons. From the fitted regression lines (Fig.5) it is evident that CO concentration decreases over time. The amount of CO decreased per unit time (minute), which is the mean slope, is equal to -0.0048 ppm. The mean intercept= 3.7. The calculated half life of breath CO is 182 minutes and SD ± 15.3. The outlier was not included in calculations. This finding is similar with the published literature that the half life for CO is about 4 hours (122).

It is worth noting that CO elimination as well as absorption is affected by many variables (30). Factors known to be responsible for inter-person variation are :-

a - Personal variation can account for 20%.

b - Diffusion of carbon monxide in the lungs.

c - The pressure gradient of CO in the alveoli.

d - The presence of lung disease.

e - Activity of the subject

In this study, only the last of these factors was controlled: the subjects were all at rest during the day. The other four factors could all have exerted some influence on the diffusion of CO in the sample subjects, therefore may explain why the calculated half-life is at variance with that published in the literature.

5.3 CARBON MONOXIDE IN THE BREATH OF CHILDREN AND ADULTS, AND CROSS-REACTIVITY BETWEEN BREATH CO AND HYDROGEN

The previous CO findings were in adults. In order to study breath CO changes at younger ages, Glasgow High School pupils (n=105) were included in the study. 34% of the pupils were smokers. 72% of the smokers smoked only 1 to 4 cigarettes per day on average, which means that they are not heavy smokers and may only be weekend smokers. This agrees with published figures (64,133). It was hoped that an assessment of the breath test for screening school children could be made. The reported cigarette smoking in children was compared with the smoking habits of parents. Although the data failed to reach statistical significance, the probability of a child reporting any smoking experience seemed to increased progressively with the number of parents who smoke (141,146).

Study of the relationship between breath CO and the self-reported smoking habit of the pupils showed that the sensitivity of breath CO is 0.25, specificity is 0.9, predictive value of the positive test is 0.6, and 0.7 for the negative test using a 9 ppm breath CO cut-off level (Table 17). This low sensitivity and high specificity can be explained by the fact that pupils smoke very few cigarettes per day on average.

The time interval since the last smoked cigarette (Table 18) was taken into account. 24% of smokers had a high breath CO (10 ppm and more) and they have not smoked for more than 12 hours before breath CO test. 29% of smokers had breath CO (10 ppm and more) and smoked within 12 hours before breath CO test. This finding may be due to the fact that children smoke markedly fewer cigarettes per day than adults and that some of them are weekend smokers.

In order to see if there is cross-reactivity between breath CO and hydrogen (Table 19) a breath hydrogen test was also carried out. Breath hydrogen was highly correlated with breath CO in school children ($r=0.75$, significance $p<0.001$) in contrast it is poorly correlated with breath CO in adult hospital employees ($r=0.19$, significance $p<0.05$). This hydrogen interference may make breath CO doubtful as a screening test in children compared to adults. At an arbitrary cut off point of 20 ppm hydrogen , the predictive

power of the positive test was 38% and for the negative test was 65%.

The findings among school pupils were similar to those in Ruchill Hospital employees. Table 20 shows that the breath CO test sensitivity is 0.74, specificity is 0.89, the predictive value of the positive test is 0.83 and for the negative test 0.84. There is a significant relationship between smoking habit and breath CO with higher levels in smoking employees ($p<0.001$). This finding agrees with published results (161). The test sensitivity for the hospital employees is higher than that for pupils and the predictive value of the positive test is higher also. This may be due to the difference in smoking habits between High School pupils and hospital employees who smoke more than the pupils.

The relationship between the time interval from last smoking and the measurement of breath CO for Ruchill Hospital employees (Table 21) was significant (X^2 =16.7; 2df; $p<0.001$) at a cut-off points of one, 1-4 and more than 4 hours. It was evident that the longer the time elapsed the lower the breath CO, which agrees with published results (122).

At an arbitrary cut off point of 20 ppm breath hydrogen for Ruchill Hospital employees (Table 22) the predictive power of the postive test was 30%, and for the negative test was 84%. Comparing sensitivity and specificity of breath CO for the pupils and Ruchill Hospital employees (Table 23) at

different CO levels shows that sensitivity in each group decreases with increasing in breath CO concentration. Sensitivity of breath CO for hospital employees is generally higher than that for pupils. Change in specificity is opposite to the change in sensitivity as expected, ie it increases as the cut-off point for breath CO concentrations increases, but the hospital employees show a higher specificity for the test than do the pupils. The minimum error rate was calculated for each group. It was found that the error rate was minimum at any CO level between 8-12 ppm for hospital employees and at 7 ppm for the pupils.

Apparently there is nothing published on the use of Receiver Operating Characterisitics (ROC) curves (Fig.6) in assessing tests for smoking. The false positive percentages (1- specificity) and true positive percentages (sensitivity) for each group were plotted to form (38). These curves were used to study the effect of varying breath CO levels measured by GMI CO-monitor and to evaluate the analytical method (breath CO monitor) used in the 'diagnosis' of smokers. The ROC curve for the hospital employees is higher than that for school pupils. Once again this is due to the different smoking habit among employees compared to school pupils.

5.4 PREVENTION METHODS USED:HOW EFFECTIVE ARE THESE METHODS.

The approach used in this study to produce a change in behaviour was an educational one. Thus accurate and practical information was provided to the CCU patients who had made the decision to implement changes in their smoking habits or diet, hence it was not prescriptive. The education was provided primarily as an individualized risk factor strategy. This approach has few limitations, since acquistion of correct knowledge is only one of the many factors that influence behaviour change (142).

The differences in methodology and duration of follow-up make comparison between studies difficult. It appears that intervention started while patients were still in the hospital and at a time when they had already stopped smoking because of hospital policies had a higher rate of success than when counselling was delayed until after the patients had gone home. It appears that nurses and physicians have an impact during the early, in hospital, rehabilitative phase after MI in encouraging patients to quit or cut down on smoking (142).

Assessment of the psychological profile was important to know how best to approach the patients for personalised health education intervention at follow up. In the MHQ (141a) basic study, the mean scores for normal healthy subjects was 5.1 S.D =3.1 for free floating anxiety, 2.9 S.D =2.2 for phobic anxiety, 5.8 S.D =3.1 for obsessive traits, 3.2 S.D =2.4 for somatic symptoms, 3.3 S.D =2.3 for depressive tendency and 7.5 S.D =3.1 for "hysterical" traits. Using the

mean ± 1 S.D as providing the normal range, the distribution of the psychological profile was as follows:39% of the total followed up patients had a normal psychological profile. Out of the remaining who had high scores, 69% of the patients had free floating anxiety, 50% had phobic anxiety, 18% had obsessive traits, 64% had somatic symptoms, 11% had depressive symptoms and "hysterical trait " was noted in 10% of males and 23% of females. Health education was directed to the patients according to the psychological profile and according to the answers to the dietary questionnaire.

The two groups were followed up to compare the effectiveness of health education intervention among CCU patients. The intensive health education was given by a health visitor who had become an ex-smoker a long time before the time of the project. The characteristics of the study groups were comparable. There was no significant difference in structure as regards age, sex (Tables 24 and 25) and social class distribution at entry in the study. Patients over 55 years of age form an equal percentage in each group (54%). Nearly two thirds of the patients in each group were males, as in the published literature (41,82,136).

Looking at social class and smoking habit at follow-up it is evident that there was no significant relationship between these variables (Tables 26 and 27). In the non-intervention group 50% (6/12) of social class I,II,III NM were smokers and 56% (15/27) of IIIM, IV and V were smokers. Chi-square for the collapsed table, social class I, II,IIINM

compared to the rest = 0.25; 1df; N.S. From many other studies it is evident that smoking is clearly related to social class; the lower income groups contain both more smokers and more earlier starters. Occupations normally associated with higher education tend to have few smokers (75,103). Due to small frequencies in the health education group Fisher's exact probability test was used for the collapsed table (social class I, II,IIINM compared to the rest P=0.31; N.S). In this study the strength of association between social class and smoking habit is less but the trend is as expected.

Public knowledge and attitudes about the risks of health is steadily increasing, particularly through the media, but much ignorance still remains (130). In another study although 75% believed that heart attacks were preventable, only a few (28%) named cigarette smoking as a factor. Twenty one per cent named high blood pressure, 13% named cholesterol, 50% did not name any of these risk factors and only 1% named all three. This reflects the widespread lack of information about major causes of heart attack (139).

In the present study 44% of the patients mentioned old age as a factor for coronary heart disease, 77% mentioned cigarette smoking as a factor, 17% mentioned high blood pressure, 89% named overweight and eating fats. These figures are much higher than those published seven years ago from the U.S.A(139) for cigarette smoking (77% versus 28%). However both studies give a similar percentage (17% and 21%) for high

blood pressure being a risk factor. The differences may be due to different populations but could also be the result of the health education given to all CCU patients, as the questions were asked at follow-up following admission. In this study, the intensive health education group have significantly higher percentages as regards these three factors, than in the non-intervention group; namely smoking 91% vs 62%, blood pressure 21% vs 6% and eating fats 96% vs 45%.

In this study Tables 28 and 29 show the relationship between social class and the mean score of patient's knowledge about risk factors forCHD. The maximum possible score is 16. Oneway ANOVA in the non-intervention group showed no significant relationship between social class and patient's knowledge about CHD (Oneway ANOVA =2.08; 2,60 df;). The result was also not significant in the health education group (Oneway ANOVA= 0.56; 2,21 df). This may be due to the effect of admission in hospital and the basic health education given to all patients.

Life events in the year before hospital admission and presence of MI were also studied. Death of a family member , financial problems and job loss form the main life events in each group. In each group there is no significant relationship between none or any life event, and the presence or absence of myocardial infarction (Tables 30 and 31). This finding may indicate that life events may not be a single factor leading to myocardial infarction.

With regards to the presence of stress before hospital admission. There is no significant relationship between myocardial infarction and the presence or absence of stress in each group (Tables 32 and 33). Other findings agree with the published literature that the role of physical activity in CHD is not completely clear. Physical activity can produce psychological improvement which is often noted clinically but the effect on morbidity and mortality is unknown [50,163]. It has been reported in a study in New York city that physically inactive men show a higher incidence of first MI than more active men among both smokers and non-smokers. The highest incidence was in inactive smokers and the lowest by the relatively more active non-smokers [49]. In a case-control study, the results showed no marked difference in the physical activity of the job between coronary and non-coronary groups [163]. Stress was the highest of the reported important cardiovascular risk factors, occuring in 70% [86].

5.5 COMPARISON BETWEEN THE FOLLOWED UP GROUPS IN RESPECT OF EFFECTIVENESS OF HEALTH EDUCATION AND CHANGES IN SMOKING HABIT AND CO

The characteristics of the study groups were comparable as it is also evident from the laboratory findings (Table 34). Furthermore there is no significant difference between the two groups as regards the presence or absence of myocardial infarction. There is a significant relationship between the presence or absence of myocardial infarction and raised cardiac enzymes in each group since it is one of the criteria used for diagnosis, but there is no significant diffference between the two groups with regard to the following variables:- High SGOT (>43 U/L), high triglycerides (>1.8 mmol/L), high cholesterol (>7.6 mmol/L), haemoglobin, hyperglycaemia.

In other studies it has been found that cholesterol, diastolic BP, highest number of cigarettes smoked per day in the past, longest period smoking has been stopped in the past, and alcohol consumption, are ineffective discriminators. That is to say that when intercorrelation is taken into account in the analysis, the variables exert no independent effect on myocardial infarction (153).

Comparison between carboxyhaemoglobin and the number of cigarettes smoked per day at entry examination in each group showed that there is no significant relationship between both variables in either the non-intervention group or the health

education group. This unexpected result could (as mentioned) be due to the time elapsed between the onset of pain, which may stop smoking, and admission when the samples were taken (Tables 35 and 36). Giving oxygen during the first few hours in emergency and inaccuracy of the spectrophotometric method (CO-oximeter 282) could also be other contributing factors.

The normal range of CO varies in different populations, being higher in a city with exposure to automobile exhaust gases, than in rural areas. The average COHb values in non-smoking street cleaners in New York was 3% COHb (56). Hanson and Hastings mentioned that 1.5% saturation was the normal level in non-smokers not exposed to automobile gases (62).

The normal range of breath CO in the Scottish MONICA project was given as 2-8 ppm for non-smokers and 9-40 ppm for smokers (162). Wald et al in Oxford used 2% as a cut off level in their study (157). A normal COHb of less than 2% or less than 10 ppm was used as a cut-off level in the study reported here.

Comparison between the follow-up COHb and the number of cigarettes smoked per day for both groups shows that non-smokers who had a high COHb (>2% saturation) comprised 12% in the non-intervention group and 27% in the health education group. This probably indicates that they were not reporting their true smoking history. Smokers who had normal COHb levels comprised 22% in the non-intervention group and 22% in the health education group (Tables 37 and 38). Comparison

between the means of COHb for the smoking groups at the same time was done by Oneway ANOVA. There was significant relationship between the two variables in the non-intervention group (Oneway ANOVA; F=13.8; 3,59 df; p<0.001) and in the health education group (F=2.9; 2,21df; Just N.S). The small overlap in the distribution of COHb levels from cigarette smokers and non-smokers was striking and surprising in view of the short half life of COHb in blood (170). Validation of this findings by assays of blood COHb suggested that 27% of subjects who say that they gave up smoking in the health education group may have misreported their smoking habit and this percentage was lower (12%) in the non-intervention group.

Comparison between entry and follow-up COHb levels (Tables 39 and 40) in the non-intervention group showed the frequencies of patients according to the difference between entry and follow up COHb, and the percentages. The mean of the differences ($\bar{d}$)= 0.83; SD ± 3.56 Paired t test =1.85; 62 df; N.S. 95% confidence interval =-0.07 to 1.73. In health education group the mean of differences ($\bar{d}$)= -1.16; SD ±6.06; Paired t test= -0.93; 23df; N.S. 95% confidence interval -3.7 to 1.41. These results shows that there is no significant change in blood COHb between entry and follow up which may be due to unsability and inaccuracy of the spectrophotometry and may be due to non significant change in smoking habit in both groups over this period.

The type of fuel used for house heating could be a

possible source of pollution by CO gas. In this study most of the houses (60%) were heated by a clean and safe method, free from CO wastes (Table 41). About quarter of the patients used gas for heating and never complained of gas fire troubles. Very few used open fires for house heating. Table 42 shows the means of COHb and SD for group of house heating. There is no significant relationship between type of heating and blood COHb level. Therefore most of CO is due to tobacco smoking. Questions on passive smoking were not included.

A small number of diseases are preventable by risk factor modification and in the majority the risk factors of interest were alcohol and tobacco (95). The primary aim of the North Karelia project was to reduce CHD risk through reduction in the risk factors and included smoking as an important risk factor, aiming at a reduction of 10% in the smoking rate (107). In the European Multifactorial Preventive Trial the primary aim was to reduce risk factors. Changes in cigarette consumption after 4 years varied from country to country, the lowest was in Belgium -3.9%, the highest was -15.6% in Britain and the average reduction in cigarette consumption was - 8.8% (100). The effect of advice about eating and smoking habits in the Oslo study, where 80% were smokers at the begining, was to reduce tobacco consumption by 45% in the intervention group over that in controls (69). In the Multiple Risk Factor Intervention Trial (MRFIT) the quit rate was 47.0% at the end of the first 4 months which was the end of smoking intervention phase (73).

The success of the intensive health education method was assessed by studying the percentage of those giving up smoking and those who decreased the number of cigarette smoked per day (Tables 43 and 44). The differences between the number of cigarettes smoked at entry minus the number smoked at follow up was calculated for each patient. The mean of differences in the non-intervention group was -1.03 and in the health education group was 8.2. There was no significant reduction in the number of cigarettes smoked per day in the non-intervention group in contrast to the marked reduction in cigarettes smoked per day (Paired t test =4.4; df=23; $p<0.001$) in the health education group. This marked reduction in the reported number of cigarettes smoked per day is likely to be due to the effect of intensive health education.

Initially all patients in the followed up groups were smokers. Changes in the smoking habit in the two groups were studied at follow-up. At entry examination 90% of patients smoked only cigarettes and the remaining 10% smoked cigarettes with either pipe or cigar. Examination of the self-reported change in smoking habit of the two groups showed that by the follow-up examination 41% had given up smoking and 59% remained smokers. In the health education group, 63% had given up smoking and 37% remained smokers. Comparison between the two groups showed no significant increase in quit rate in the intervention group (X^2 =3.14; 1 df; NS). Although non-smokers did not significantly increase in number in the health education group, they decreased the

number of cigarettes smoked per day more than in the non-intervention group (Tables 43 and 44).

Table 45a shows a comparison between the change in smoking habit of the two groups using the breath CO test to detect smokers. There was no significant change in the smoking habit of the two groups (X^2 = 1.2; 1df; N.S). The percentage who "misreported" their smoking habit was 8% in the non-intervention compared with 17% in the health education group. These are lower than the published figures for under reporting of smoking which range from 20% to 46% (76).

At follow-up the relationship between smoking habit and breath CO is significant ($p<0.001$) in the non-intervention and in the Health education group ($p<0.01$). In the non-intervention group non-smokers with a high breath CO (>10ppm) were 19% and in the health education group were 27%. Smokers with a normal breath CO comprise 22% in the non-intervention group and 11% in the health education group (Tables 46,47).

Study of the relationship between blood COHb and breath CO at follow-up showed that there is highly significant difference between blood COHb and breath CO in each group. This significant difference show that blood COHb is higher than breath CO which is as expected, due to the high pressure gradient required for CO elimination from blood.

Further comparison between other characteristics in the followed up groups show no significant difference as regards age distribution, sex, weight, height, alcoholic drinks, tea, coffee consumption, systolic, diastolic BP and activity in work (Table 49). These findings indicate that the two groups were comparable.

It is evident from this study that measuring blood COHb is inconvenient due to its expense, time consuming and needs a lot of laboratory work experience, in addition to the reported inaccuracy and unstability of the spectrophotometric method used. The alternative method for measuring blood COHb could be gas-chromatography which again requires a lot of experience, is time consuming and not practical for field studies. It was evident also that there is poor agreement between these two methods. This study demonstrated also that the hand held breath CO monitor is a simple method for measuring alveolar carbon monoxide levels provided precautions are taken to use the monitor properly to eliminate the possibility of false readings. It is sensitive to breath hydrogen therefore diseases producing excess hydrogen should be taken into consideration when measuring breath CO. However the use of this monitor for screening of smokers in children is doubtful. The instrument is not sensitive to CO2 or water vapour two factors which make it suitable for use in breath samples. The method is rapid and easy to perform and enables a person's breath CO level to be estimated while avoiding the discomfort and inconvenience of collecting blood. The CO results measured on breath CO

monitor can be seen by the subjects, which is likely to make it useful as a method of reinforcing health advice to stop smoking. It is also likely to be useful when judging the reliability of statements made about a smoking habits.

Although there was no statisticall significantly difference between the intensive health education and the non-intervention methods yet the percentage of those stop smoking was about 20% higher than in the non-intervention method. Comparison of these finding with the other published data shows a one year success rate of 2% from a smoking cessation kit (34a), 14.5% from the BBC television series "So You Want to Stop Smoking" (111a), 10% by the use of tobacco substitutes (Nicotine chewing gum) and individualised counselling (88a), 6% from accupunture (29a), 6% from nicotine chewing gum (39a), 5.1% from patients given a leaflet and advised to stop smoking compared to 0.3% in a control group (131a).

The calculated average cost of the intensive personalised health education was £6 for each home visit including an average transport cost and an assuming an average 45 minutes consultation. Each patient had on average 5 visits for this programme- a total cost of £ 30. From table 45 it appears that 5 more people stopped smoking in the health education group than expected from the non-intervention figures. The cost of helping each of these extra individuals to stop smoking is calculated to be about £ 144. This cost is above the cost of the standard intervention given in CCU.

CONCLUSIONS

6.1-EFFECTS OF HEALTH EDUCATION ON GIVING-UP SMOKING

Anti-smoking health education methods may vary depending on the target population, social attitudes, knowledge and behaviour.

The intensive personalised antismoking health education carried out in this study according to the psychological profile of the patient showed no significant additional benefit over the conventional anti-smoking advice used for changing health behaviour. Both the non-intervention and health education groups gave up smoking by similar amounts. Self-reports of smoking behaviour indicated that 63% in health education and 41% in non-intervention groups , ceased smoking. By use of breath CO measurement, about 17% misreported their smoking habit in health education group in comparison to 8% in non-intervention group.This intensive health education method is expensive in terms of time consumed (45 minutes in each home visit and in hospital) and it does not show an advantage over the usual methods of health care.

6.2 MEASURMENT OF BLOOD AND BREATH CARBON MONOXIDE

In this study three methods were used for measuring blood and breath carbon monoxide. There were some difficulties encountered during measuring CO and they are

listed below :-

1- Gas-chromatography method. This method is sensitive and requires extensive laboratory experience, which is expensive and time consuming. Although the method is accurate it is not suitable for field studies in populations. The precision of this method is good, it has been stated that the technique achieves a more accurate and specific measurement than the spectrophotometric method (20). In this study there was a poor agreement between this method and the spectrophotometric method.

2- The Spectrophotometric method for measuring blood COHb is easily affected by many intervening factors. For example the presence of pigments in haemolytic diseases may interfere with light absorption. Therefore blood samples have to be analysed immediately and should be kept in air tight containers so as to avoid direct oxygenation. Also the CO-oximeter 282 needs frequent calibration and cleaning of the cuvette which is liable to be clogged by blood clots. Handling blood samples may carry a risk unless strict precautions are followed. Added to these difficulties is the inconvenience of collecting blood samples from the subjects. An experienced operator can achieve an accuracy of +- 10%, but up to 25% differences can occur between workers in the same laboratory.

3- Breath CO measurement. In this method the GMI CO monitor, which has been used in industry for monitoring ambient CO, is

tested for measuring breath CO. This method uses a recently developed electrochemical cell which at present has to be changed every year. Breath CO is highly correlated with blood COHb, and therefore can be used for measuring blood COHb indirectly. This method is very easy to use, very cheap to run and it is practical for field studies particularly anti-smoking studies. Repeatability of this method is +- 1%. However some difficulties were encountered:

a) Cross-sensitivity was found between CO and breath hydrogen. Therefore use of breath CO as an objective measurement of smoking behaviour is doubtful in children with low and sporadic consumption of tobacco. It should be taken into account when measuring breath CO in persons with sugar malabsorption syndromes.

b) It takes a longer time to settle to zero after use for a long time.

c) The electrochemical cell used is valid only for one year.

In this study blood COHb, as measured by the spectrophotometric method, was not related to the reported smoking habit nor to the number of cigarettes smoked per day. This may be due to instability and inaccuracy of the method used. By contrast breath CO measured by the GMI CO monitor, did show a significant relationship to the number of cigarettes smoked in adults. However, use of the CO monitor was found to have low sensitivity (25%) when screening for smokers in the sample of children. This may be due to the sporadic nature of smoking in children.

RECOMMENDATIONS

1.RECOMMENDATIONS AS A RESULT OF THE FINDINGS IN THIS STUDY.

A-Personalised health education.

(i) Although there was no statistically significant advantage present in personalised health education over the ordinary health education given to all CCU patients, there was an indication that it did help smoking cessation in about 20% more patients than than those recieving ordinary health education. There was a significant decrease in the number of cigarettes smoked per day in those who continued to smoke but only in the group recieving personalised health education.

(ii) The cost of this intensive personalised health education can be reduced by training less expensive health workers to do it as a part of their daily work.

(iii) This method could be potentially valuable for reducing the number of smokers, thereby the burden of smoking related diseases and merits further investigation.

2- Objective CO measurements.

(i) Because it was mentioned in many studies that breath CO is highly related to blood COHb breath CO can be used for screening of adult smokers particularly in field and epidemiological studies.

(ii) The use of the breath CO monitor for measuring breath CO in children is of doubtful value due to sporadic and

infrequent smoking in children.

(iii) The cross-reactivity between breath CO and breath hydrogen should be taken into consideration particularly in sugar malabsorption syndromes. Due to cross-sensitivity between breath CO and breath hydrogen further development of the GMI CO monitor is required and a further evaluation study of the method should be planned in the future.

2. GENERAL RECOMMENDATIONS.

A TO HEALTH AUTHORITIES

The declining trend in tobacco smoking in Britain seems mostly due to the effects of health education, and it is worthwhile continuing antismoking campaigns. Restriction and prohibition of tobacco smoking in many places in the community can help to reduce the number of smokers. The use of an objective visible test for breath CO measurement combined with health advice can give a convincing demonstration of the effect of smoking to an individual which may reinforce advice about smoking cessation.

B TO PROFESSIONAL AND MEDICAL PERSONNEL

General practitioners, working in primary health care should provide the public with health advice against smoking and provide counselling facilities for smokers. General practitioners are in an ideal situation to do this as they are widely distributed in the country and they are in frequent communication with many of their patients.

C TO RESEARCHERS

Tobacco smoking is a multifactorial problem related to attitudes, behaviour and health habits in the population. Research workers should study all aspects related to tobacco smoking, and be aware that different populations may have different influences acting on smoking behaviour. Therefore anti-smoking health education methods should be adapted to meet these different profiles of influences, to achieve success.

Antismoking education will cost little relative to the cost of treatment of tobacco- related morbidity. This makes health education a necessity in developing and in developed countries alike.

In developing countries, smoking is markedly increasing every year and it almost certainly will produce an epidemic of disease in the future. Research workers in developing countries should study the social, economic and personal factors involved in smoking in order to plan for a strategy to combat the smoking problem.

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TABLES AND DIAGRAMS

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DESCRIPTION OF THE TOTAL STUDY POPULATION

TABLE (1)
AGE AND SMOKING HABIT FOR ALL CASES.

Smoking Habit / Age	Never-smoked	Ex-smoker	Smokers	Row Total
<45	6	8	14	28
	(21.4)	(28.6)	(50.0)	(100.0)
45-55	8	15	36	59
	(13.6)	(25.4)	(61.0)	(100.0)
55-65	13	34	59	106
	(12.3)	(32.0)	(55.7)	(100.0)
>65 Years	5	7	7	19
	(26.3)	(36.8)	(36.8)	(100.0)
Column Total	32	64	116	212
	(15.1)	(30.2)	(54.7)	(100.0)

( ) = Row percentage shown in brackets.
N.S = not significant.
(Chi-square = 5.52; 6df; N.S)

TABLE (2)

SEX AND SMOKING HABIT FOR ALL CASES.

Smoking Habit / Sex	Never-smoke	Ex-smoker	Smokers	Row Total
Male	18 (11.6)	48 (31.0)	89 (57.4)	155 (100.0)
Female	14 (24.6)	16 (28.1)	27 (47.4)	57 (100.0)
Column Total	32 (15.1)	64 (30.2)	116 (54.7)	212 (100.0)

Row percentage shown in brackets.
(Chi-square = 5.51; 2 df; N.S)

TABLE (3a)

AGE WHEN SMOKING BEGAN AND NUMBER OF CIGARETTES SMOKED PER DAY FOR MALE SMOKERS.

Cigarettes start.age	< 15	15-25	>25	Row Total
	CIGARETTES PER DAY			
Years.				
<15	4	10	13	27
	(15.0)	(37.0)	(48.0)	(100.0)
15-25	18	13	11	42
	(43.0)	(31.0)	(26.0)	(100.0)
>25	11	3	5	19
	(57.9)	(15.8	(26.3)	(100.0)
Column Total	33 (37.5)	26 (29.5)	29 (33.0)	88 (100.0)

Percentage shown in brackets.
(Chi-square =10.62; 4 df; p <0.05)

TABLE (3b)

AGE WHEN SMOKING BEGAN AND NUMBER OF CIGARETTES SMOKED PER DAY FOR FEMALE SMOKERS

Cigarettes start. age	<15 Cigarettes	>15 per day	Row Total
<15	2	8	10
	(20.0)	(80.0)	(100.0)
>15 Years	11	7	18
	(61.1)	(38.9)	(100.0)
Column total	13	15	28
	(46.4)	(53.6)	(100.0)

Percentage shown in brackets.
(Chi-square =4.37; 1df; p<0.05)

TABLE (4)
SMOKING HABIT AND CARBOXYHAEMOGLOBIN LEVEL FOR ALL CASES.

(COHb) Smoking habit	<10	10-20	>20 PPM	Row Total
Non-smokers	7 (21.9)	19 (59.3)	6 (18.8)	32 (100.0)
Ex-smokers	14 (21.9)	40 (62.5)	10 (15.6)	64 (100.0)
Smokers	25 (21.6)	63 (54.3)	28 (24.1)	116 (100.0)
Column Total	46 (21.7)	122 (57.5)	44 (20.8)	212 (100.0)

Row percentage shown in brackets.
(Chi-square = 2.02; 4 df; N.S)

TABLE (5)

NUMBER OF CIGARETTES SMOKED PER DAY AND CARBOXYHAEMOGLOBIN MEASUREMENTS FOR SMOKERS.

COHb / Cigarettes	<10 ppm	10-20 ppm	>20 ppm	Row Total
<15 (mild)	8 (17.4)	30 (65.2)	8 (17.4)	46 (100.0)
15-25 (moderate)	8 (21.6)	18 (48.6)	11 (29.7)	37 (100.0
>25 Cigt.(heavy)	9 (27.3)	15 (45.5)	9 (27.3)	33 (100.0)
Column Total	25 (21.6)	63 (54.3)	28 (24.1)	116 (100.0)

Row percentage shown in brackets.
96 cases were ex or never smoke.
(Chi-square = 4.05; 4 df; N.S)

TABLE (6)
PRESENCE OF ANGINA AND CARBOXYHAEMOGLOBIN LEVEL FOR ALL CASES.

(COHb) / Angina	<10 ppm	10- ppm	> 20 ppm	Row Total
No angina	24	79	26	129
	(18.6)	(61.2)	(20.2)	(100.0)
Grade I	13	26	8	47
	(27.7)	(55.3)	(17.0)	(100.0)
Grade II	9	17	10	36
	(25.0)	(47.2)	(27.8)	(100.0)
Column Total	46	122	44	212
	(21.7)	(57.5)	(20.8)	(100.0)

Row percentage shown in brackets.
(Chi-square = 3.73; 4 df; N.S)
- Grade I represents severe form of angina.
- Grade II represents mild form of angina.

TABLE (7)

CARBOXYHAEMOGLOBIN IN PATIENTS WITH OR WITHOUT MYOCARDIAL INFARCTION.

COHb / M.Infarction	<10 ppm	10-20	>20 ppm	Row Total
Yes	29 (29.6)	50 (51.0)	19 (19.4)	98 (100.0)
No	16 (15.0)	66 (61.7)	25 (23.3)	107 (100.0)
Column Total	45 (22.0)	116 (56.6)	44 (21.4)	205 (100.0)

Row percentage shown in brackets.
7 cases uncertain whether MI present or not.
(Chi-square = 6.4; 2 df; $p<0.05$)

There is a significant inverse relationship between Myocardial Infarction and COHb distribution; patients with MI are more likely to have a within normal COHb level.

TABLE (8)
STRESS AND SMOKING HABIT IN ALL CASES.

Stress / Smoking Habit	Moderate & severe	Mild or no stress	Row Total
Non-smokers	29	3	32
	(90.6)	(9.4)	(100.0)
Ex-smokers	56	8	64
	(87.5)	(12.5)	(100.0)
Smokers	89	26	115
	(77.4)	(22.6)	(100.0)
Column Total	174	37	211
	(82.5)	(17.5)	(100.0)

Row percentage shown in brackets.
1 missed case
(Chi-square = 4.64; df = 2; N.S)

TABLE (9)
MYOCARDIAL INFARCTION AND LIFE EVENTS IN THE YEAR BEFORE HOSPITAL ADMISSION.

M. Infarction / Life events	Yes	No	Row Total
Job loss	6	7	13 (6.3)
Family member death	12	11	23 (11.2)
Financial problems	11	8	19 (9.3)
Residence change	3	4	7 (3.4)
More than one	8	20	28 (13.6)
None	58	57	115 (56.1)
Column Total	98 (47.8)	107 (52.2)	205 (100.0)

Percentage shown in brackets.
7 cases uncertain whether MI present or not.
(Chi-square = 0.73 between none and presence of any of life events; 1 df; N.S).

TABLES AND DIAGRAMS

COMPARISON BETWEEN BREATH CARBON MONOXIDE AND BLOOD CARBOXYHAEMOGLOBIN .

TABLE (10)
COMPARISON BETWEEN BLOOD CARBOXYHAEMOGLOBIN AND BREATH CARBON MONOXIDE FOR 42 PERSONS.

Subject No.	Blood COHb Mean ppm	Blood COHb CV	Breath CO Mean ppm	Breath CO CV
1	28.1	1.9	21.0	2.9
2	3.9	24.4	6.1	11.5
3	4.2	13.1	8.0	5.0
4	2.5	20.0	4.0	11.3
5	5.2	10.6	8.2	14.6
6	27.2	2.9	24.0	3.0
7	2.5	26.0	3.0	43.3
8	4.0	40.0	1.2	36.7
9	18.0	3.1	11.0	6.5
10	5.5	36.4	3.4	15.9
11	14.5	3.4	8.6	12.8
12	4.0	13.8	3.6	15.0
13	6.5	7.7	6.6	8.2
14	15.0	16.7	7.2	6.3
15	4.0	37.5	1.2	36.7
16	18.0	2.8	11.0	6.4
17	5.5	36.4	3.4	15.9
18	14.5	3.4	8.6	12.8
19	4.0	12.5	3.6	15.0
20	6.5	7.7	6.6	8.2
21	15.0	16.7	7.2	6.3
22	6.0	10.8	3.8	28.7
23	3.0	16.7	5.0	14.2
24	3.0	48.3	4.4	12.5
25	4.0	20.0	2.2	20.5
26	4.5	11.1	4.8	27.1
27	4.5	11.1	3.8	10.5
28	4.5	11.1	1.6	33.8
29	4.0	19.5	1.2	30.1
30	18.0	2.4	11.0	5.7
31	5.5	10.0	3.4	16.2
32	14.5	3.1	8.6	13.5
33	4.0	25.3	3.6	19.3
34	6.5	8.0	6.6	9.0
35	15.0	7.4	7.2	8.1
36	6.0	10.2	3.8	17.8
37	3.0	12.3	5.0	21.3
38	3.0	16.3	4.4	12.9
39	4.0	11.1	2.2	14.4
40	4.5	13.9	4.8	16.1
41	4.5	12.9	3.8	12.3
42	4.5	14.7	1.6	29.1

CV = Coefficient of variation

Mean 7.98 5.96

SD ±6.59 ±4.62

Paired t test =4.0 ($p<0.001$).

$\bar{d}$ = 2.02 SD of differences ±3.3 Variance= 10.7

A 95 % confidence limit for $\bar{d}$ is 1.2 and 2.9

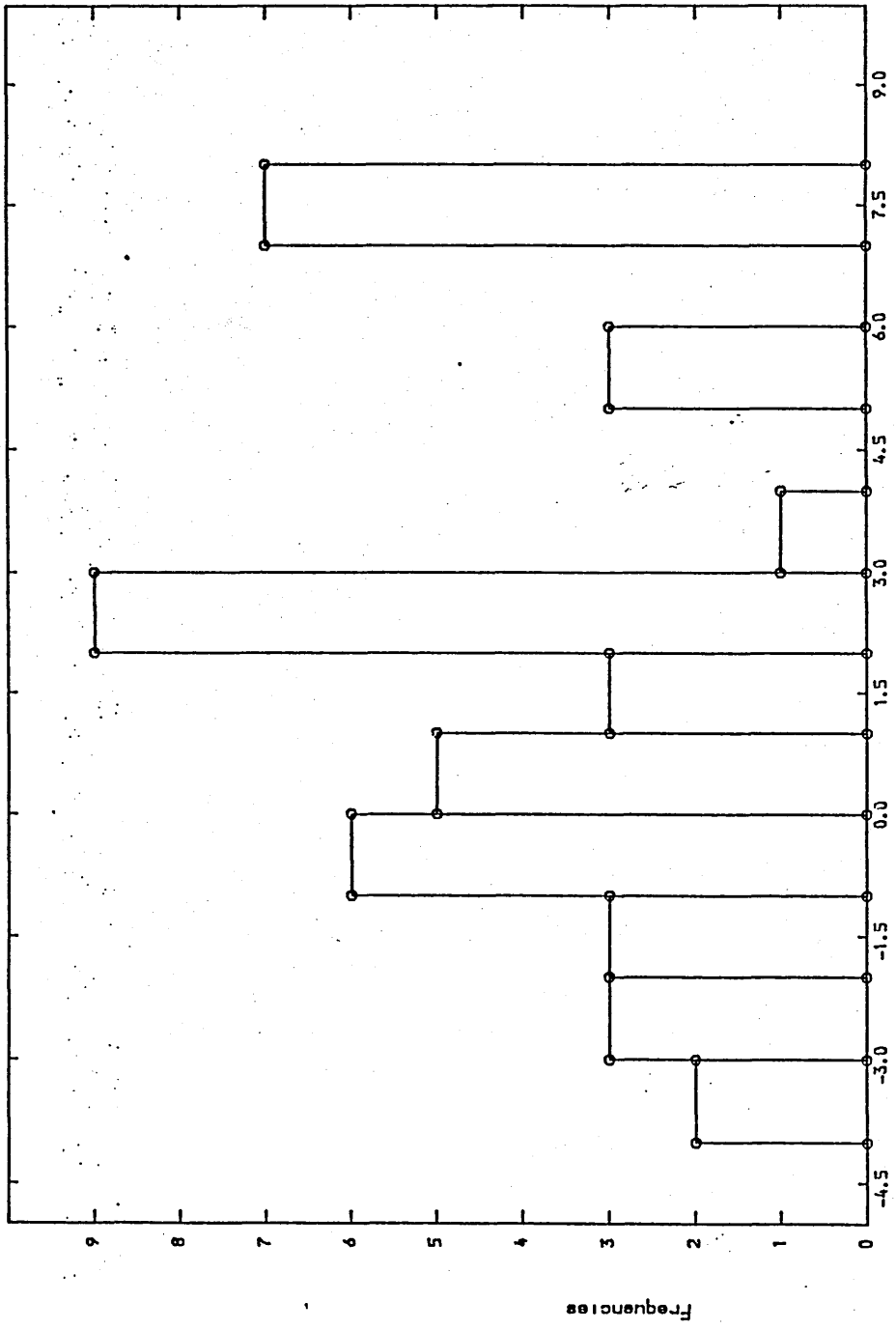

Fig. (1) Differences between spectrophotometric method (blood COHb) and breath CO for each patient

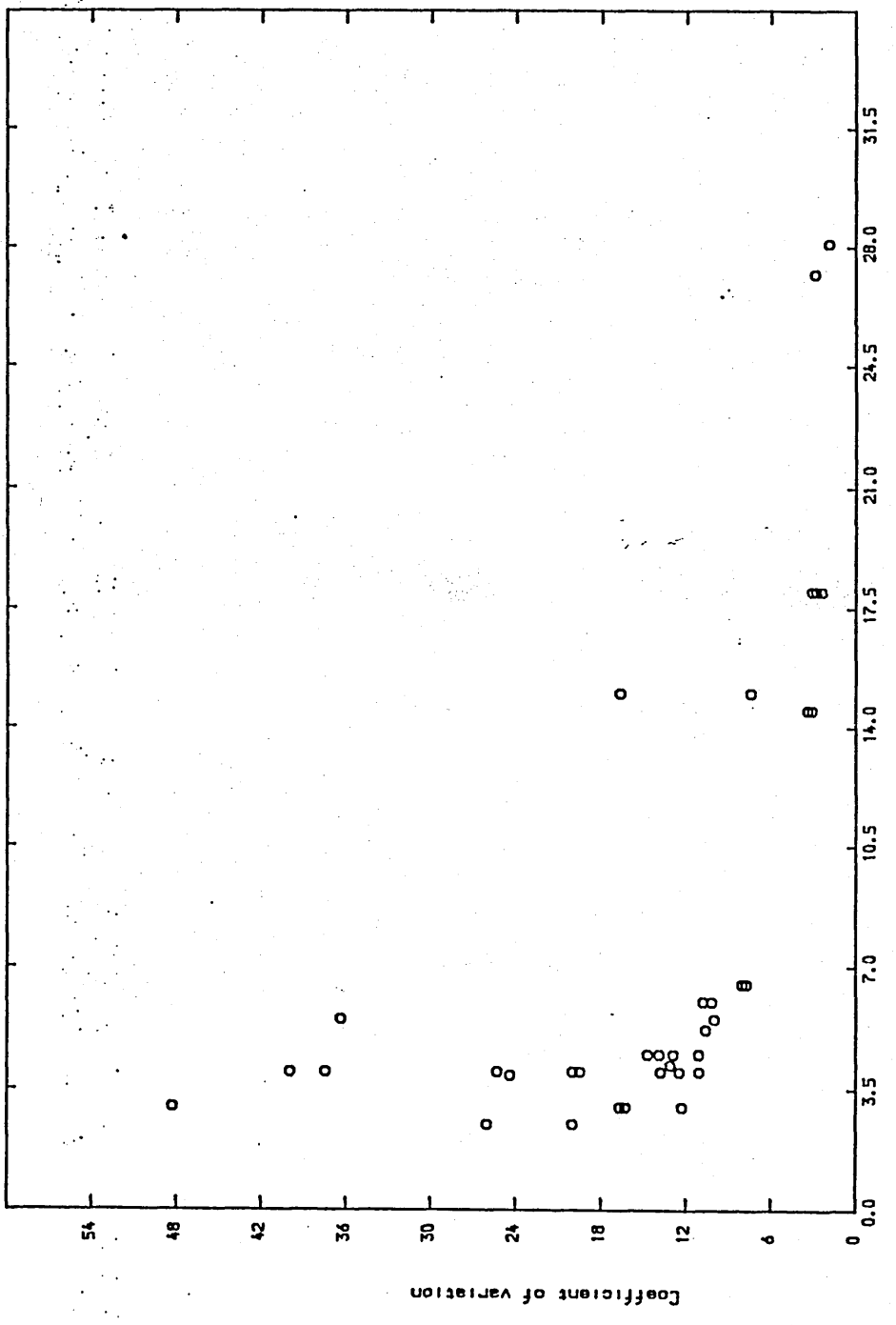

Fig. (2) Coefficient of variation of blood COHb measured by CO-oximeter-282

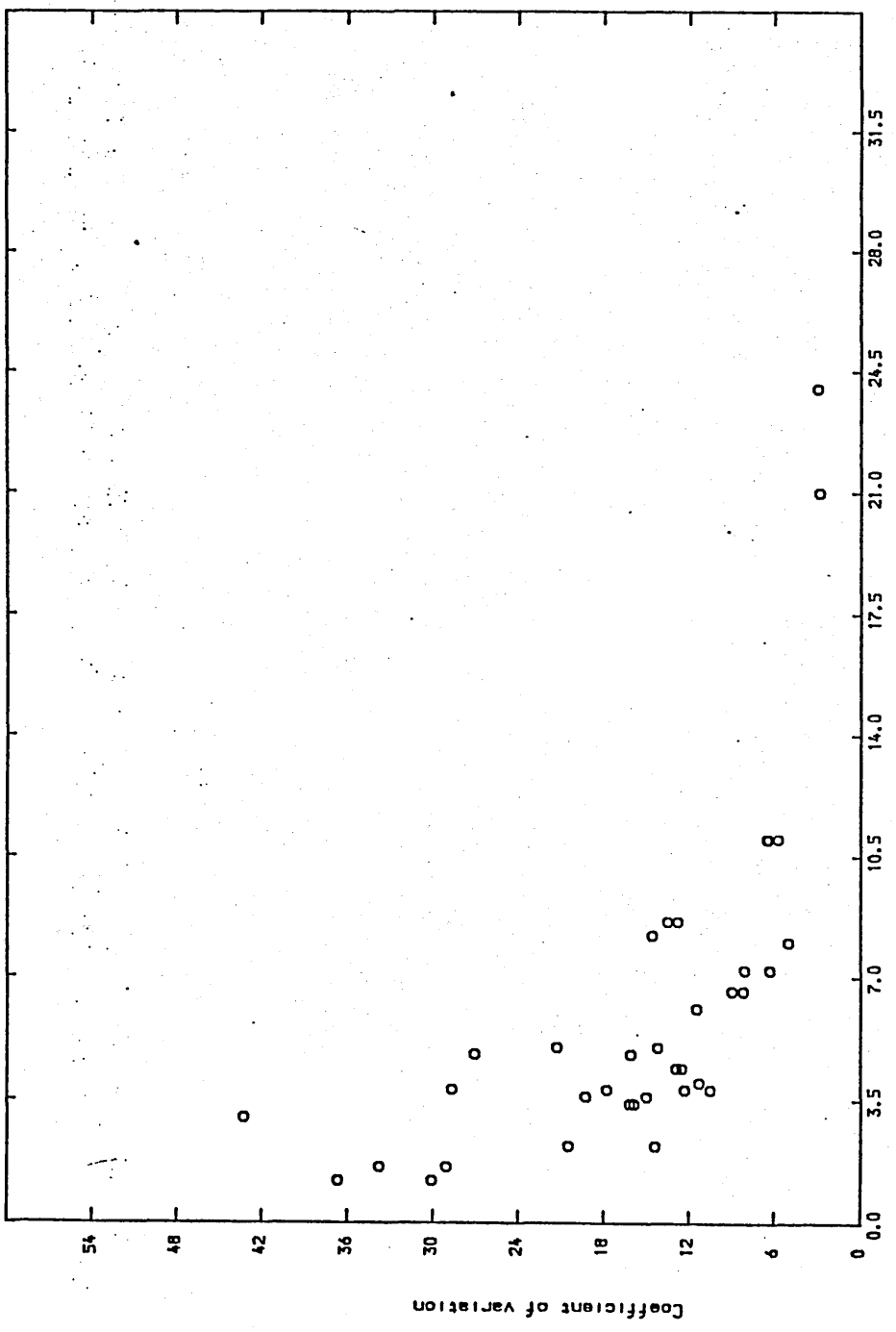

Fig. (3) Coefficient of variation of breath CO.

BLOOD AND BREATH CARBON MONOXIDE: CHANGES OVER TIME AND ACCURACY.

TABLE (11)

RELIABILITY OF BLOOD COHb ANALYSED IMMEDIATELY AND 24 HOURS AFTER THE SAMPLES WERE TAKEN

	Analysed Immediately						24 hours after the samples were taken					
Sample No.	1st Reading	2nd Reading	3rd Reading	4th Reading	5th Reading	Mean	1st Reading	2nd Reading	3rd Reading	4th Reading	5th Reading	Mean
1	4.3	4.5	5.7	4.4	4.6	4.7	4.8	4.9	5.5	4.9	5.6	5.1
2	2.8	2.8	2.8	2.8	2.8	2.8	5.9	3.4	2.2	2.6	2.3	3.3
3	2.0	2.0	2.0	2.0	2.0	2.0	1.2	0.6	1.0	1.0	0.6	0.9
4	5.0	5.1	5.0	5.0	5.1	5.0	5.2	5.2	5.6	6.1	6.1	5.6
5	2.2	2.2	2.2	2.2	2.2	2.2	2.2	2.2	2.4	3.7	2.6	2.6
6	5.4	5.4	5.5	5.4	5.4	5.4	5.6	5.3	5.5	5.4	5.5	5.5
7	2.4	2.4	2.4	2.4	2.3	2.4	2.4	2.4	2.3	2.4	2.4	2.4
8	0.3	0.4	0.9	0.8	0.8	0.6	1.0	1.1	1.0	1.0	0.8	1.0

For immediate analysis rk = 0.99 and r_1 = 0.99 (rk = reliability for the whole group)

For after 24 hours rk = 0.98 and r_1 = 0.94 (r_1 = reliability for each measurement)

Overall mean of differences ($\bar{d}$) = 0.05, SD($\bar{d}$) $\pm$ 0.55 and variance = 0.3

TABLE (12)
COEFFICIENT OF VARIATION OF BLOOD CARBOXYHAEMOGLOBIN FOR 21 PATIENTS MEASURED BY CO-OXIMETER 282.

Subject No.	Mean COHb ppm	Coefficient of variation
1	4.5	25.0
2	4.5	15.1
3	5.0	20.0
4	11.0	3.0
5	4.5	73.3
6	7.0	13.2
7	3.5	33.3
8	16.0	4.5
9	4.5	36.0
10	5.5	14.5
11	6.0	7.8
12	8.5	26.8
13	2.0	56.8
14	4.0	33.3
15	5.5	17.9
16	7.0	8.0
17	7.5	8.6
18	5.0	16.0
19	6.5	9.7
20	6.5	9.0
21	23.2	2.4

Reliability of each measurment is 0.94

TABLE (13)
EFFECT OF TIME AND STORAGE ON CARBOXYHAEMOGLOBIN IN 21 BLOOD SAMPLES MEASURED BY CO-OXIMETER 282.

Sample No.	Immediate analysis % saturation	After 24 hours
1	2.6	2.2
2	1.7	1.7
3	1.7	4.9
4	2.3	6.0
5	1.8	4.8
6	1.9	5.0
7	3.8	6.8
8	2.2	4.0
9	5.1	3.8
10	5.8	7.8
11	4.1	5.7
12	3.8	3.3
13	3.2	4.6
14	6.0	5.9
15	5.3	5.1
16	6.8	7.0
17	7.8	5.7
18	10.7	8.1
19	6.8	11.4
20	4.4	6.8
21	4.0	6.7
Mean	4.4	5.6
SD	± 2.4	± 2.1

(Paired t test =1.6; N.S)
($\bar{d}$ = 1.2; SD ± 2.0; variance = 4.0)

TABLE (14)
COMPARISON BETWEEN TWO METHODS FOR COHB MEASUREMENT ; GAS-CHROMATOGRAPHY AND A SPECTROPHOTOMETRY BY CO-OXIMETER 282 FOR 31 PATIENTS.

Sample No.	CO-oximeter 282 ppm	Gas chromatography ppm	Sample No.	CO-oximeter 282 ppm	Gas chromato-graphy ppm
1	1.0	6.8	17	2.0	5.6
2	2.5	7.2	18	10.5	5.9
3	3.0	5.5	19	4.0	7.6
4	3.0	7.3	20	2.5	6.1
5	3.5	8.0	21	16.5	2.5
6	2.5	9.8	22	23.3	3.1
7	1.5	4.3	23	15.0	3.1
8	2.5	4.5	24	3.0	6.4
9	1.0	5.2	25	6.5	6.7
10	0.0	6.9	26	11.5	7.7
11	3.5	5.0	27	16.5	5.6
12	6.0	4.4	28	13.0	6.6
13	1.5	0.0	29	0.0	7.3
14	0.5	4.3	30	11.0	11.3
15	1.5	0.0	31	20.5	4.3
16	0.5	3.6			

	CO-oximeter 282	Gas-chromatography
mean	6.1	5.6
SD	± 6.6	± 2.4

The mean of the differences between the two methods ($\bar{d}$) = 0.55, SD ($\bar{d}$) ± 7.2 and variance = 52.1.
A 95% confidence limit for the mean of differences = - 1.7 and 2.8 which means that there is no evidence of bias between the two methods but many of the individual differences are very large.

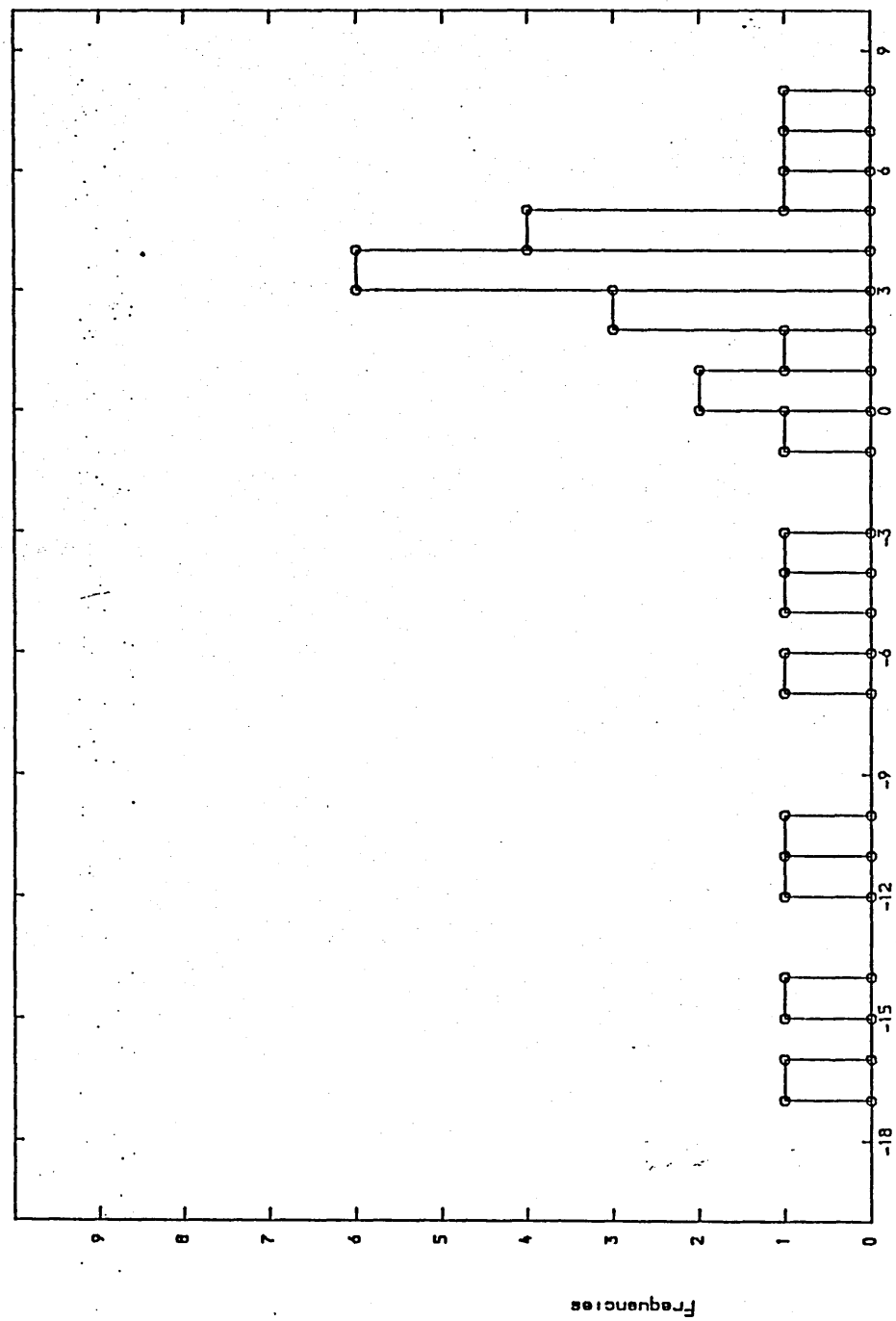

Fig. (4) The differences between spectrophotometric and gaschromatography method for measuring blood COHb

TABLE (15)
RAPID CHANGES IN BLOOOD CARBOXYHAEMOGLOBIN EVERY 30 MINUTES ANALYSED BY CO-OXIMETER 282.

Time / Subject No.	0	After 30 minutes	After 60 minutes	Variance
1	4.2	4.4	4.1	0.02
2	5.2	9.0	7.7	3.73
3	7.8	8.5	7.7	0.19
4	7.8	10.5	10.0	2.06
5	6.6	7.5	5.8	0.72
6	5.6	6.3	6.0	0.12

All subjects were smokers.
Reliability of whole group (rk) = 0.95
Reliability of each single measurement (r1) = 0.87

TABLE (16)

BREATH CARBON MONOXIDE CHANGES OVER TIME FOR SIX SUBJECTS MEASURED BY GMI CARBON MONOXIDE MONITOR

Time / Subject	Before Smoking	15 min	30 min	45 min	60 min	75 min	90 min	105 min	120 min	135 min	150 min	165 min
1	7.0	18.0	16.0	14.0	12.0	8.0	8.0	8.0	11.0	8.0	10.0	10.0
2	15.0	14.0	8.0	12.0	12.0	13.0	7.0	9.0	7.0	9.0	7.0	8.0
3	8.0	12.0	10.0	7.0	9.0	9.0	7.0	6.0	8.0	5.0	7.0	6.0
4	5.0	12.0	17.0	15.0	13.0	10.0	8.0	8.0	9.0	9.0	9.0	10.0
5	6.0	13.0	13.0	12.0	11.0	11.0	10.0	9.0	11.0	12.0	12.0	12.0
6	8.0	14.0	11.0	10.0	12.0	12.0	10.0	8.0	8.0	8.0	8.0	7.0

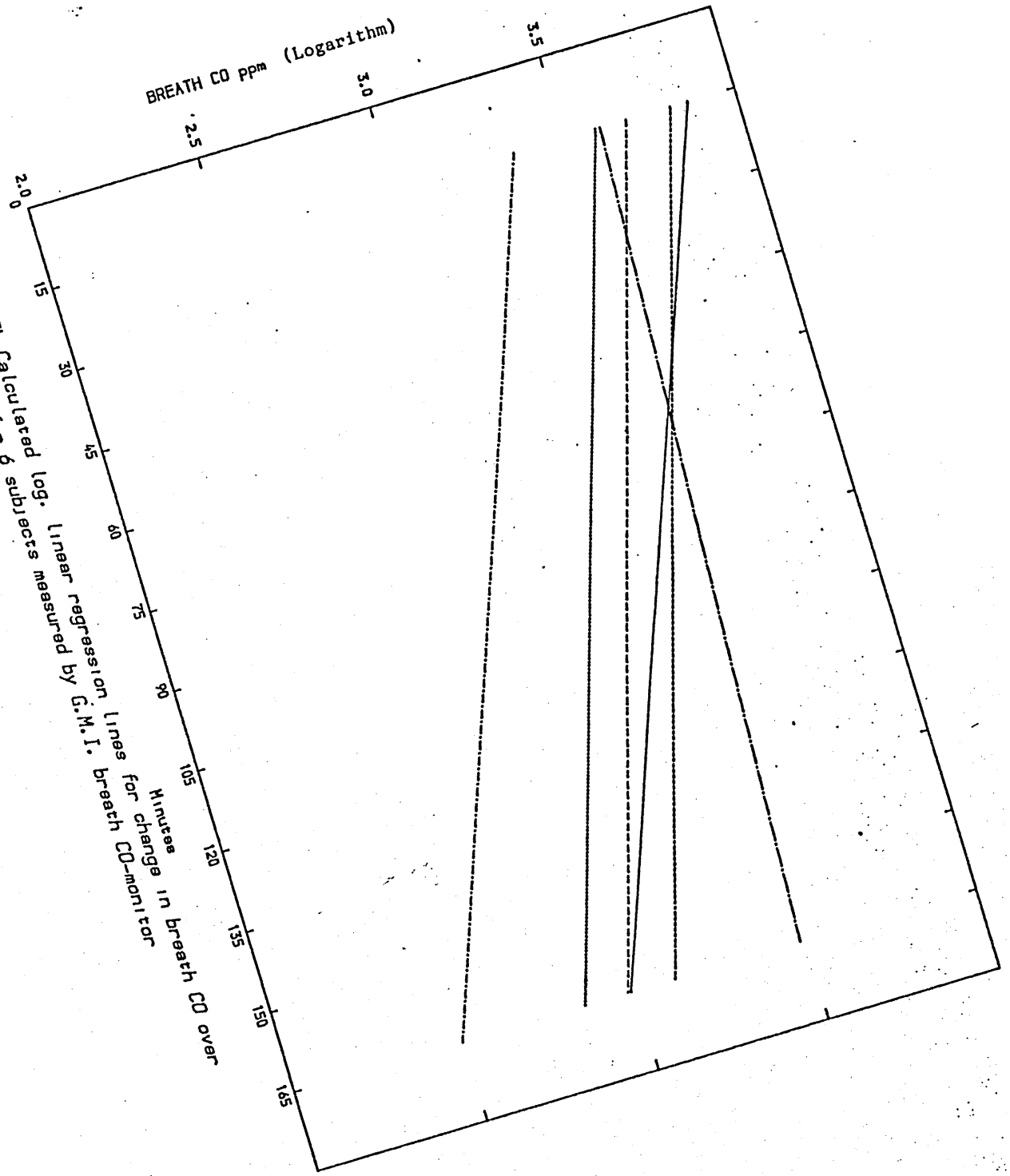

Fig (5) Calculated log. linear regression lines for change in breath CO over time for 6 subjects measured by G.M.I. breath CO-monitor

TABLES AND DIAGRAMS

CARBON MONOXIDE IN BREATH IN SCHOOL PUPILS AND HOSPITAL EMPLOYEES AND CROSS SENSTIVITY BETWEEN BREATH CO AND HYDROGEN.

ACCURACY OF CARBON MONOXIDE FOR IDETIFICATION OF SMOKERS.

TABLE (17)
RELATIONSHIP BETWEEN SMOKING HABIT AND BREATH CARBON MONOXIDE FOR GLASGOW HIGH SCHOOL PUPILS.

Smoking / Breath CO	Smokers	Non-Smokers	Row Total
10+ ppm	9	7	16 (15.2)
<10ppm	27	62	89 (84.8)
Column Total	36 (34.3)	69 (65.7)	105 (100.0)

Percentage shown in brackets.
(Chi-square = 4.04; df= 1; $p<0.05$)
Sensitivity of breath CO test 0.25
Specificity 0.9
Predictive value of a positive test 0.56
Predictive value of a negative test 0.70

TABLE (18)
TIME BETWEEN LAST SMOKED CIGARETTE AND CARBON MONOXIDE TEST FOR GLASGOW HIGH SCHOOL PUPILS.

Time / CO ppm	>12 h	<12 hours	Row Total
10+ ppm	7	2	9 (25.0)
<10 ppm	22	5	27 (75.0)
Column Total	29 (80.6)	7 (19.4)	36 (100.0)

Percentage shown in brackets.
Only 36 pupils were smokers

TABLE (19)
BREATH HYDROGEN FOR GLASGOW HIGH SCHOOL PUPILS.

Breath Hydrogen	Smokers	Non-Smokers	Row Total
>20ppm	5	8	13 (23.2)
<20ppm	15	28	43 (76.8)
Column	20 (35.7)	36 (64.3)	56 (100.0)

Breath hydrogen was done for 56 pupils.
Percentage shown in brackets.
(Chi-square = 0.06; df = 1; N.S)

TABLE (20)

SMOKING HABIT AND BREATH CARBON MONOXIDE FOR RUCHILL HOSPITAL EMPLOYEES MEASURED BY GMI CO-MONITOR.

Smoking Breath CO	Smoker	Non-Smoker	Row Total
10+ppm	43	9	52 (36.4)
<10ppm	15	76	91 (63.6)
Column Total	58 (40.6)	85 (59.4)	143 (100.0)

Percentage shown in brackets.
(Chi-square = 60.2; df =1; $p<0.001$)
Senstivity = 0.74 ; specificity = 0.89
Predictive value of positive test = 0.83 and
0.83 for negative test.

TABLE (21)

TIME BETWEEN LAST SMOKING AND BREATH CARBON MONOXIDE FOR RUCHILL HOSPITAL EMPLOYEES.

Time / CO ppm	<1 hour	1-4 h	>4 hours	Row Total
10+ ppm	30	11	1	42 (72.4)
<10 ppm	7	2	7	16 (27.6)
Column Total	37 (63.8)	13 (22.4)	8 (13.8)	58 (100.0)

Percentage shown in brackets.
(Chi-square = 16.7; 2 df; $p < 0.001$)

There is significant relationship between breath CO and time since last cigarette. The longer the interval the lower the breath CO.

TABLE (22)

BREATH HYDROGEN FOR RUCHILL HOSPITAL EMPLOYEES.

Smoking / Breath Hydrogen	Smokers	Non-Smokers	Row Total
>20ppm	3	7	10 (7)
<20ppm	55	78	133 (93)
Column Total	58 (40.6)	85 (59.4)	143 (100.0)

Percentage shown in brackets.
(Fisher's exact probability =0.09; N.S).

TABLE (23)
SENSITIVITY AND SPECIFICITY OF BREATH CARBON MONOXIDE FOR RUCHILL HOSPITAL EMPLOYEES AND GLASGOW HIGH SCHOOL PUPILS AT DIFFERENT CO LEVELS.

Breath CO level	Hospital employees		School Children	
	Sensitivity	Specificity	Sensitivity	Specificity
6 ppm	0.84	0.64		
7 ppm	0.81	0.76	0.42	0.75
8 ppm	0.81	0.82	0.33	0.80
9 ppm	0.74	0.87	0.28	0.87
10 ppm	0.74	0.89	0.25	0.90
11 ppm	0.72	0.89	0.11	0.94
12 ppm	0.72	0.91		
13 ppm	0.67	0.94		
14 ppm	0.66	0.95		
15 ppm	0.62	0.96		
16 ppm	0.59	0.96		
17 ppm	0.55	0.96		

The minimum total error for breath CO for the employee is between 8-12 ppm and that for the school pupils is at 7 ppm.
Note: School pupils have a narrow range of breath CO.

Fig. (6) Breath CO Receiver Operating Characteristics Curves (ROC) for Ruchill Hospital employees and Glasgow High School children

TABLES

ASSESSING EFFECTIVENESS OF HEALTH EDUCATION INTERVENTION

- DESCRIPTION OF THE TWO FOLLOWED UP GROUPS AND COMPARISON BETWEEN THEM.
- CHANGES IN SMOKING HABIT FOR BOTH GROUPS OF PATIENTS.
- CHANGES IN OTHER FACTORS INVOLVED IN HEALTH EDUCATION.
- CHARACTERISTICS OF THE GROUP GIVING UP SMOKING.

TABLE (24)

AGE AND SEX DISTRIBUTION OF THE NON-INTERVENTION GROUP.

Sex / Age	Male	Female	Row Total
<45	6	2	8 (12.7)
45-55	15	6	21 (33.3)
>55 years	26	8	34 (54.0)
Column Total	47 (74.6)	16 (25.4)	63 (100.0)

TABLE (25)

AGE AND SEX DISTRIBUTION OF THE HEALTH EDUCATION GROUP

Sex / Age	Male	Female	Row Total
<45	3	3	6 (25.0)
45-55	5	0	5 (20.8)
>55 years	8	5	13 (54.2)
Column Total	16 (66.7)	8 (33.3)	24 (100.0)

Percentage shown in brackets.

TABLE (26)

SOCIAL CLASS AND FOLLOW-UP SMOKING HABIT IN NON-INTERVENTION GROUP.

Smoking habit / Social class	Non- & Ex-Smoker	Smoker	Row Total
Social Class I, II &IIINM	6	6	12 (21.8)
Social Class III M,IV& V	12	15	27 (49.0)
Retired	5	4	9 (16.4)
Unemployed	1	6	7 (12.7)
Column Total	24 (43.6)	31 (56.4)	55 (100.0)

Eight cases missed
(Chi-square for collapsed table social classes I,II, IIINM and the others = 0.25; 1 df; N.S).

TABLE (27)
SOCIAL CLASS AND FOLLOW-UP SMOKING HABIT IN HEALTH EDUCATION GROUP.

Smoking / Social Class	Non- & Ex-Smokers	Smokers	Row Total
Social Class I,II,III NM	4	1	5 (25.0)
Social Class III M, IV & V	5	1	6 (30.0)
Retired	2	3	5 (25.0)
Unemployed	1	3	4 (20.0)
Column Total	12 (60.0)	8 (40.0)	20 (100.0)

4 cases social class could not be ascertained.

TABLE (28)
SOCIAL CLASS AND KNOWLEDGE ABOUT CHD IN NON-INTERVENTION GROUP.

S.class Knowledge Score	Retired & unemployed	Soc.Class I,II,IIINM	Soc.class IIIM,IV,V	Entire patients
Mean Sore	11.6	12.2	10.5	11.2
Number	22	12	29	63
Standard deviation	± 2.3	± 1.2	± 2.3	± 2.2

The maximum possible score is 16.
(Oneway ANOVA = 2.08; 2,60df; N.S)

TABLE (29)
SOCIAL CLASS AND KNOWLEDGE ABOUT CHD IN HEALTH EDUCATION GROUP.

S.Class Knowledge -Score	Retired & unemployed	Soc.class I,II,IIINM	Soc.class IIIM,IV,V	Entire patients
Mean Score	11.8	12.0	10.8	11.6
Number	13	5	6	24
Standard deviation	± 2.3	± 2.5	±1.5	± 2.1

The maximum possible score is 16.
(Oneway ANOVA =0.56; 2,21df; N.S)

TABLE (30)
LIFE EVENTS IN THE YEAR BEFORE HOSPITAL ADMISSION FOR NON-INTERVENTION GROUP.

M.infarction / Life events	Yes	No	Row Total
Job loss	1 (1.6)	2 (3.2)	3 (4.8)
Death of family member	2 (3.2)	6 (9.7)	8 (12.9)
Financial problems	4 (6.5)	2 (3.2)	6 (9.7)
Residence change	1 (1.6)	0 (0.0)	1 (1.6)
More than one	3 (4.8)	6 (9.7)	9 (14.5)
None	14 (22.6)	21 (33.9)	35 (56.5)
Column total	25 (40.3)	37 (59.7)	62 (100.0)

1 case could not be ascertained.
(Chi-square test = 0.0; 1 df; N.S between none and presence of any of the life events).

TABLE (31)
LIFE EVENTS IN THE YEAR BEFORE HOSPITAL ADMISSIN FOR HEALTH EDUCATION GROUP.

M.infarction / Life events	Yes	No	Row Total
Job loss	3 (12.5)	0 (0.0)	3 (12.5)
Death of family member	1 (4.16)	1 (4.16)	2 (8.3)
Financial problem	3 (12.5)	1 (4.16)	4 (16.7)
More than one	0 (0.0)	2 (8.3)	2 (8.3)
None	9 (37.5)	4 (16.7)	13 (54.2)
Column Total	16 (66.7)	8 (33.3)	24 (100.0)

Percentages shown in brackets.
No residential change.
(Chi-square test = 0.08; 1 df; N.S between none and presence of any life events).

TABLE (32)
STRESS AND MYOCARDIAL INFARCTION IN NON-INTERVENTION GROUP.

MI	Moderate & Severe	No & mild stress	Row Total
Yes	19	7	26 (43.3)
No	22	12	34 (56.7)
Column Total	41 (68.3)	19 (31.7)	60 (100.0)

3 cases could not be ascertained.
(Chi-square test = 0.48; 1 df; N.S)

TABLE (33)
STRESS AND MYOCARDIAL INFARCTION IN HEALTH EDUCATION GROUP.

MI	Moderate stress	No&mild stress	Row Total
Yes	15	1	16 (66.7)
No	6	2	8 (33.3)
Column Total	21 (87.5)	3 (12.5)	24 (100.0)

There was no severe stress in this group.
(Fisher's exact probability = 0.22 ; N.S)

TABLE (34)
COMPARISON BETWEEN LABORATORY FINDINGS IN BOTH GROUPS.

GROUP Investigation Type	non-intervention group	Health Education group	Difference
Myocardialinfarction	43.2 %	66.7 %	NS
Cardiac enzymes for MI patients			
SGOT (>43 U/L)	96.0 %	87.5 %	NS
CK enzyme (>160 U/L)	96.0 %	93.9 %	NS
LD (> 230 U/L)	92.0 %	68.8 %	NS
High Triglycerides (> 1.8 mmol/L)	20.6 %	16.7 %	NS
High Cholesterol (>7.6 mmol/L)	9.5 %	8.5 %	NS
Haemoglobin			
< 14 %	49.2 %	37.5 %	NS
between 14-18 %	49.2 %	53.8 %	NS
>18 %	1.6 %	4.2 %	NS
High Blood sugar	12.7 %	8.3 %	NS

N.S = not significant.

TABLE (35)
ENTRY CARBOXYHAEMOGLOBIN AND NUMBER OF CIGARETTES SMOKED PER DAY FOR NON-INTERVENTION GROUP.

COHb Cigarettes No.	< 2	2- 4	>4 % satu.	Row Total
<15	2	23	3	28 (44.4)
15-25	2	11	6	19 (30.2)
>25 Cigt/day	2	10	4	16 (25.4)
Column Total	6 (9.6)	44 (69.8)	13 (20.6)	63 (100.0)

(Oneway ANOVA done on raw values gives F=0.98; df=2,60; N.S)

TABLE (36)
ENTRY CARBOXYHAEMOGLOBIN AND NUMBER OF CIGARETTES SMOKED PER DAY FOR HEALTH EDUCATION GROUP.

COHb Cigarettes No.	< 2	2- 4 % Satu.	Row Total
<15	1	16	17 (70.8)
>15 Cigt/day	3	4	7 (29.2)
Column Total	4 (16.7)	20 (83.3)	24 (100.0)

Percentages shown in brackets.
(Oneway ANOVA done on raw values gives F=0.16; df=1,22; N.S)

TABLE (37)
FOLLOW-UP CARBOXYHAEMOGLOBIN AND NUMBER OF CIGARETTES SMOKED PER DAY FOR NON INTERVENTION GROUP.

COHb / Cigarettes No.	< 2	2- 4	>4% sat.	Row Total
Non-smokers	23	2	1	26 (41.3)
< 15	3	3	9	15 (23.8)
15-25	2	3	11	16 (25.4)
>25 Cigt/day	3	1	2	6 (9.5)
Column Total	31 (49.2)	9 (14.3)	23 (36.5)	63 (100.0)

(Oneway ANOVA ; F=13.77; 3,59 df; p<0.001 see page 180)

TABLE (38)
FOLLOW-UP CARBOXYHAEMOGLOBIN AND NUMBER OF CIGARETTES SMOKED PER DAY FOR HEALTH EDUCATION GROUP.

COHb / Cigarettes No.	< 2	2- 4	>4% sat.	Row Total
Non-smokers	11	1	3	15 (62.5)
<15	1	1	2	4 (16.7)
15-25 cigt/day	1	1	3	5 (20.8)
Column Total	13 (54.2)	3 (12.5)	8 (33.3)	24 (100.0)

Percentages shown in brackets.
The two groups 15-25 and group >25 cigt/day were combined due to small frequencies.
(Oneway ANOVA; F= 2.86; 2,21df; Just N.S see page 181)

TABLE (39)
COMPARISON BETWEEN ENTRY AND FOLLOW-UP COHb IN NON-INTERVENTION GROUP.

Entry COHb-follow upCOHb	Frequencies	Percentage %
-6 to -7.9	2	3
-4 to -5.9	3	5
-2 to -3.9	10	16
0 to -1.9	10	16
0.1 to 1.9	9	14
2 to 3.9	22	35
4 to 5.9	5	8
6 to 7.9	0	0
8 to 9.9	0	0
10 to 11.9	2	3
Total	63	100

-Mean of differences= 0.83% sat; SD ± 3.6
-Paird t test = 1.85; 62 df; N.S.
-95% confidence interval= -0.07 to 1.7

TABLE (40)
COMPARISON BETWEEN ENTRY AND FOLLOW-UP COHb IN HEALTH EDUCATION GROUP.

Entry COHb-follow upCOHb	Frequencies	Percentage %
-10 to -11.9	2	8
-8 to -9.9	2	8
-6 to -7.9	1	4
-4 to -5.9	0	0
-2 to -3.9	9	38
0 to -1.9	0	0
0.1 to 1.9	4	17
2 to 3.9	2	8
4 to 5.9	0	0
6 to 7.9	1	4
8 to 9.9	2	8
10 to 10.9	1	4
Total	24	100

-Mean differences= -1.16% sat; SD± 6.1
-Paired t test = -0.93; 23df;N.S.
-95% confidence interval = -3.7 to 1.4

TABLE (41)
TYPE OF HOUSE HEATING AND CARBOXYHAEMOGLOBIN LEVEL FOR ALL FOLLOWED UP PATIENTS

COHb / Heating	<2	2 - 4	>4 % sat.	Row Total
Electricity	1	26	4	31 (36.4)
Open fire or Gas	3	13	5	21 (24.7)
Central Heating	4	15	2	21 (24.7)
More than one	1	9	2	12 (14.1)
Column Total	9 (10.6)	63 (74.1)	13 (15.3)	85 (100.0)

2 cases missed

TABLE (42)
TYPE OF HOUSE HEATING AND CARBOXYHAEMOGLOBIN LEVEL (STATISTICAL FINDINGS)

COHb / Heating	Number	Mean COHb % satu.	SD
Electricity	31	0.05	5.59
Open fire or gas	21	4.13	11.66
Central Heating	21	1.98	4.33
More than one	12	1.03	5.13
Total patients	85	1.67	7.21

(Oneway ANOVA F= 1.02; 3,81 df; N.S).

TABLE (43)
THE DIFFERENCES BETWEEN THE NUMBER OF CIGARETTES SMOKED PER DAY AT ENTRY MINUS THE NUMBER AT FOLLOW UP.(NON-INTERVENTION GROUP)

Difference entry cigt/day - follow up cigt/ day	Frequencies	Percentage %
- 14 to-15	6	9.6
- 12 to-13	4	6.3
- 10 to-11	1	1.6
- 8 to -9	2	3.2
- 6 to -7	1	1.6
- 4 to -5	3	4.8
- 2 to -3	0	0.0
0 to -1	24	38.0
1 to 2	2	3.2
3 to 4	4	6.3
5 to 6	5	7.9
7 to 8	3	4.8
9 to 10	8	12.7

The mean of differences = 2.1 cigt/ day S.D ± 7.3
(Paired t test = 1.12; df= 62; N.S)

TABLE (44)
THE DIFFERENCES BETWEEN THE NUMBER OF CIGARETTES SMOKED PER DAY AT ENTRY MINUS THE NUMBER AT FOLLOW UP IN HEALTH EDUCATION GROUP

Differences (Entrey minus follow up) cigarettes per day	Frequencies	Percentage %
- 10 to - 11	1	4.2
- 2 to - 3	1	4.2
0 to - 1	6	25.0
1 to 2	0	0.0
3 to 4	0	0.0
5 to 6	4	16.0
7 to 8	0	0.0
9 to 10	1	4.2
11 to 12	0	0.0
13 to 14	2	8.3
15 to 16	0	0.0
17 to 18	6	25.0
19 to 20	3	12.5

The mean of differences = 8.2 Cigt per day S.D ± 9.1
(Paired t test = 4.4; df= 23; p<0.001)

TABLE (45)
COMPARISON BETWEEN SELF-REPORTED SMOKING HABIT OF THE TWO GROUPS AT ENTRY AND FOLLOW-UP EXAMINATIONS.

Follow-up smoking GROUP	Non-smokers	Smokers	Row Total
Non-intervention	26 (41.3)	37 (58.7)	63 (100.0)
Health education group	15 (62.5)	9 (37.5)	24 (100.0)
Column Total	41 (47.1)	46 (52.9)	87 (100.0)

Percentage shown in brackets.
(Chi-square = 3.14; 1 df; N.S)

TABLE (45A)

COMPARISON BETWEEN SMOKING HABIT OF THE TWO GROUPS AT ENTRY AND FOLLOW-UP EXAMINATION (BREATH CO TEST).

Follow-up smoking GROUP	Non-smokers up to 9 ppm	Smokers 10 + ppm	Row Total
Non-intervention	21 (33.3)	42 (66.7)	63 (100.0)
Health education group	11 (45.8)	13 (54.2)	24 (100.0)
Column Total	32 (36.8)	55 (63.2)	87 (100.0)

Percentage shown in brackets.
(Chi-square = 1.17; 1df; N.S)
For this table the cut-off point of 9 ppm is taken from The litrature.

TABLE (46)
FOLLOW-UP SMOKING HABIT AND BREATH CO FOR NON-INTERVENTION GROUP.

Breath CO / Follow-up smoking habit	<10	10-20	>20ppm	Row Total
Ex-smokers	21	3	2	26 (41.3)
Smokers	8	18	11	37 (58.7)
Column Total	29 (46.1)	21 (33.3)	13 (20.6)	63 (100.0)

(Chi-square = 21.5; 2df; p<0.001)
(Two-sample t test on the actual values = 5.87; 62 df; p<0.001)

TABLE (47)
FOLLOW-UP SMOKING HABIT AND BREATH CO FOR HEALTH EDUCATION GROUP.

Breath CO(ppm) / Follow-up smoking habit	<10 ppm	10 -20 ppm	>20 ppm	Row Total
Ex-smokers	11	3	1	15 (62.5)
Smokers	1	5	3	9 (37.5)
Column Total	12 (50.0)	8 (33.3)	4 (16.7)	24 (100.0)

Percentages shown in brackets.
(Chi-square= 8.7; 1df; p<0.01 for collapsed table <10 and 10 and more ppm).
(t test on the actual values =3.05; 23 df; p<0.01).

TABLE (48)
FOLLOW-UP BLOOD COHb MINUS BREATH CO DIFFERENCES FOR ALL FOLLOWED UP PATIENTS.

Follow-up COHb minus Breath CO	Frequencies	Percentage %
- 20 to - 23.9	7	8.1
- 16 to - 19.9	6	6.9
- 12 to - 15.9	7	8.1
- 8 to - 11.9	15	17.1
- 4 to - 7.9	26	29.9
- 0 to - 3.9	17	19.5
0 to 3.9	7	8.1
4 to 7.9	0	0.0
8 to 11.9	2	2.3

The mean of the differences = - 7.7 ppm S.D ± 7.1
(Paired t test = - 10.1; df= 86; $p<0.001$)

TABLE (49)
COMPARISON BETWEEN SOME OTHER FACTORS IN THE TWO GROUPS.

Characteristics	Non-intervention	Health Education group	Significance Chi-square
Number (N)	63	24	
Age			
<45	12.7%	25.0%	NS
45 - 55	33.3%	20.8%	NS
>55 Years.	54.0%	54.2%	NS
Males	74.6%	66.7%	NS
Weight			
< 50	4.8%	8.3 %	NS
50 - 60	19.0%	8.3 %	
60 - 70	33.3%	37.5%	
70 - 80	31.7%	33.5%	
> 90 Kg	11.1%	12.5%	
Height			NS
< 150	6.3%	4.2%	
150 - 160	27.0%	20.8%	
160 - 170	30.2%	50.0%	
> 170 cm	36.5%	25.0%	
Alcoholic drink			NS
Initial	61.9%	50.0%	NS
follow-up	63.5%	41.7%	NS
Tea consumption			
Initial	88.9%	95.8%	NS
follow-up	90.5%	83.3%	NS
Coffee consumption	55.4%	60.2%	NS
Activity in work	66.1%	50.0%	NS

NS= not significant.

APPENDICES

DEPARTMENT OF COMMUNITY MEDICINE, UNIVERSITY OF GLASGOW

CONFIDENTIAL

Q U E S T I O N N A I R E. C R 1.

All information which could permit identification of the individual will be treated as strictly confidential, will be used only by persons engaged in and for the purposes of this survey, and will not be disclosed for any other purpose.

Please answer all the questions in this health record, and bring it with you at the advised time and place. Put a tick in the appropriate boxes. If you cannot give an exact answer, give the best estimate you can.

DEPARTMENT OF COMMUNITY MEDICINE, UNIVERSITY OF GLASGOW

QUESTIONNAIRE. CR.1.

Surname (Mr./Mrs./Miss).....

(BLOCK LETTERS)

First Names:.....

Address:.....

Postcode

.....

Tel. No. (Home):.....

Family Doctor:.....

Address:.....

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1.01	1.02	1.03	1.04

Ref. No.

1.05

Card No.

1 a) What is your occupation? (Please describe as fully as possible)

.....

.....

b) Are you
1 ☐ a manager?
2 ☐ a foreman?
3 ☐ an employee not 1 or 2
4 ☐ self-employed without employees?
5 ☐ self-employed with employees?

1.06
1.07
1.08

c) If you are a full-time housewife, what is your husband's occupation?

.....

2 a) What is your date of birth?

Day ..... Month ..... Year .....

1.09	1.10	1.11	1.12	1.13	1.14

b) Where have you spent most of your life? (more than half number of years)
1 ☐ in the country? (isolated dwelling, hamlet or small community)
2 ☐ in a village?
3 ☐ in a town?
4 ☐ in a city?
5 ☐ elsewhere?

1.15
1.16

c) Is it industrial? 1 ☐ Yes 2 ☐ No

3 a) Sex 1 ☐ Male 2 ☐ Female

b) Are you a twin? 1 ☐ Yes 2 ☐ No

If yes, are you 1 ☐ Like 2 ☐ Unlike twin?

1.17	1.18	1.19

4 Are you 1 ☐ Married? 2 ☐ Single?
3 ☐ Widowed? 4 ☐ Separated? 5 ☐ Divorced?

1.20

5 Do you usually have a cough? 1 ☐ Yes 2 ☐ No

1.21

6 Do you usually bring up any phlegm from your chest first thing in the morning in the winter?

1 ☐ Yes 2 ☐ No (If no, go to question 9)

1.22

7 Do you bring up phlegm on most days for as much as three months in the winter each year?

1 ☐ Yes 2 ☐ No

1.23

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8 In the past three years have you had a period of increased cough and phlegm lasting for three weeks or more? 1.24

1 ☐ Yes - one period 2 ☐ Yes - 2 or more periods
3 ☐ No

9 Do you get short of breath walking with people of your own age on level ground? 1.25

1 ☐ Yes 2 ☐ No

10 Does your chest sound wheezy or whistling on most days (or nights)? 1.26

1 ☐ Yes 2 ☐ No

11 How much exercise do you get at work? 1.27

1 ☐ None 2 ☐ A small amount 3 ☐ A lot

12 How much exercise do you get on the way to and from work each day? 1.28 1.29 1.30

.....Minutes

13 How much physical exercise do you engage in outside work each day or at the weekend? 1.31 1.32

.....Hours per week

14 What is your usual physical activity at work during the past year? 1.33

1 ☐ Severe 2 ☐ Moderate 3 ☐ None 4 ☐ Unsure

15 a) Have you experienced in the last year 1.34 1.35

1 ☐ loss of job? 2 ☐ death of a family member?
3 ☐ financial problems? 4 ☐ change of residence?
5 ☐ more than one of above? 6 ☐ none of the above?

b) If so, when?

1 ☐ 1 month ago 2 ☐ 3 months ago 3 ☐ 4-6 months ago
4 ☐ 7-12 months ago

16 How many hours in twenty four do you usually sleep? 1.36 1.37

.....hours

17 How much of the following drinks do you usually take each day? 1.38 1.39 1.40 1.41 1.42

a) Milk..... Cups b) Coffee.....cups

c) Tea.....cups

18 a) Do you take sugar in tea or coffee? 1.43 1.44

1 ☐ Yes 2 ☐ No 3 ☐ Varies

b) Do you take sugar on cereals or pudding?

1 ☐ Yes 2 ☐ No

19 How much of the following are you accustomed to taking each week? 1.45 1.46 1.47 1.48 1.49 1.50

a) Spirits.....nips b) Beer.....pints

c) Wine.....glasses

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20 Do you usually eat salads? 1.51

1 ☐ Most days of week 2 ☐ Less than once a week 3 ☐ Never

21 a) Do you smoke cigarettes now? 1.52

1 ☐ Yes 2 ☐ No (If no, go to question 22)

b) If yes, are they 1.53

1 ☐ Low tar? 2 ☐ Medium tar?

c) Do you inhale? 1.54

1 ☐ Yes 2 ☐ No

d) What kind of cigarettes do you smoke? 1.55

1 ☐ Manufactured with filters

2 ☐ Manufactured without filters 3 ☐ Hand rolled

e) How many manufactured cigarettes do you usually smoke per day? 1.56 1.57

Number per day.....

f) About how many ounces of tobacco do you use per week for rolling your own cigarettes? 1.58 1.59

.....Oz.per week

g) What is the maximum number of cigarettes that you have smoked per day for as long as a year? (include hand rolled) 1.60 1.61

.....Number per day

h) How old were you when you began to smoke cigarettes regularly? 1.62 1.63

Age.....

22 a) If you do not smoke cigarettes now, did you ever smoke them regularly? 1.64

1 ☐ Yes 2 ☐ No, never (If no, go to question 23) 1.65 1.66

b) If you used to smoke regularly, what is the maximum number of cigarettes you ever smoked per day for as long as a year? 1.67

..... Number of cigarettes 1.68 1.69

c) Did you inhale? 1.70 1.71

1 ☐ Yes 2 ☐ No

d) How old were you when you began to smoke cigarettes?

Age.....

e) When did you stop smoking cigarettes? (Give year of stopping)

Year.....

23 a) Have you ever smoked a pipe regularly? Ref. No. 2.01 2.02 2.03 2.04

1 No (if No, go to question 24)
2 Used to smoke a pipe but not now (go to d))
3 Now smoke a pipe

Card No. 2.05

b) If you smoke a pipe now, about how many ounces of tobacco do you smoke? 2.06

.....Oz. per week

c) Do you inhale? 2.07 2.08

1 ☐ Yes 2 ☐ No

d) How old were you when you began to smoke a pipe? 2.09

Age..... 2.10 2.11

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e) If you do not smoke a pipe now, when did you stop? (Give the year)
Year.....

2.12 | 2.13

24 a) Does any member of your family smoke?

1 ☐ Father 2 ☐ Mother 3 ☐ Sister 4 ☐ Brother
5 ☐ More than one member of family 6 ☐ None

b) Do they smoke
1 ☐ Cigarettes? 2 ☐ Cigars? 3 ☐ Pipe? 4 ☐ More than one type?

c) What is the longest duration of smoking?
Number of years.....

2.14
2.15
2.16 | 2.17

25 a) Have you ever smoked cigars (including small cigars) regularly?

1 ☐ No (If No, go to question 26)
2 ☐ Used to smoke cigars but not now
3 ☐ Now smoke cigars

b) If you smoke cigars now, about how many do you smoke per week?
Number per week.....

2.18
2.19 | 2.20

26 a) Have you ever had any pain or discomfort in your chest?

1 ☐ Yes 2 ☐ No (If No, go to question 27)

2.21

b) Do you get this pain or discomfort when you walk uphill or hurry?

1 ☐ Yes 2 ☐ No

2.22

c) Do you get it when you walk at an ordinary pace on the level?

1 ☐ Yes 2 ☐ No

2.23

d) When you get any pain or discomfort in your chest, what do you do?

1 ☐ Stop 2 ☐ Slow down
3 ☐ Continue at the same pace

2.24

e) Does it go away when you stand still?

1 ☐ Yes 2 ☐ No

2.25

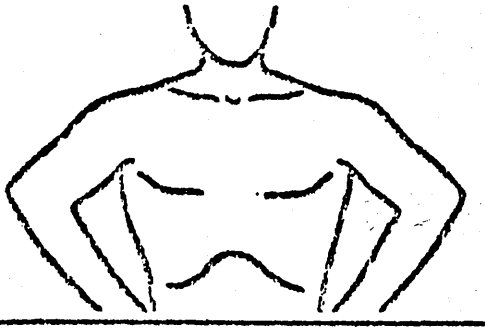
f) How soon?

1 ☐ 10 minutes or less 2 ☐ More than 10 minutes

2.26

g) Where do you get this pain or discomfort? (Mark the place(s) with an X on the diagram

2.27



17 Have you ever had a severe pain across the front of your chest lasting for half an hour or more?

1 ☐ Yes 2 ☐ No

---

28 a) Do you get a pain in either leg or foot on walking?

1 ☐ Yes 2 ☐ No (If no, go to question 29)

b) Does this pain ever begin when you are standing still or sitting?

1 ☐ Yes 2 ☐ No

c) Do you get the pain in your calf or calves?

1 ☐ Yes 2 ☐ No

d) Do you get it when you walk uphill or hurry?

1 ☐ Yes 2 ☐ No

e) Do you get it when you walk at an ordinary pace on the level?

1 ☐ Yes 2 ☐ No

f) Does the pain ever disappear while you are still walking?

1 ☐ Yes 2 ☐ No

g) What do you do if you get it when you are walking?

1 ☐ Stop 2 ☐ Slow down 3 ☐ Continue at the same pace

h) What happens to it if you stand still?

1 ☐ Usually continues for more than 10 minutes
2 ☐ Usually disappears in 10 minutes or less

---

29 Do you have diabetes?

1 ☐ Yes 2 ☐ No 3 ☐ Do not know

---

30 Do you have high blood pressure?

1 ☐ Yes 2 ☐ No 3 ☐ Do not know

---

31 Do you regularly take any medicine?

1 ☐ Yes 2 ☐ No

---

32 a) Have you ever been in hospital?

1 ☐ Yes 2 ☐ No

b) If yes, please give the following information for each admission:

Year	Nature of illness or what was wrong
1.....	.....
2.....	.....
3.....	.....

(Attach separate sheet of paper if necessary)

---

33 a) Are your parents both alive?

1 ☐ Yes 2 ☐ No

If no

b) What caused your father's death? .....

Age at death.....

c) What caused your mother's death?.....

Age at death.....

34 If you are married, does your wife/husband suffer from

a) Bronchitis? 1 ☐ Yes 2 ☐ No
b) Heart trouble? 1 ☐ Yes 2 ☐ No

2.61 2.62

35 a) What was your first regular occupation, excluding temporary work? (Please give full description)

.....

.....

2.63
2.64

b) What was your father's main occupation? (Please give full description)

.....

.....

36 Please indicate by a tick in the appropriate box in each of the following sections which description fits you best.

a) In general I am usually tense or nervous

THIS DESCRIBES ME: 1 ☐ Exactly
2 ☐ To some extent
3 ☐ Not very accurately
4 ☐ Not at all

2.65

b) There is a great amount of nervous strain connected with my daily activities

THIS DESCRIBES MY SITUATION: 1 ☐ Exactly
2 ☐ To some extent
3 ☐ Not very accurately
4 ☐ Not at all

2.66

c) At the end of the day I am completely exhausted mentally and physically

THIS DESCRIBES ME: 1 ☐ Exactly
2 ☐ To some extent
3 ☐ Not very accurately
4 ☐ Not at all

2.67

d) My daily activities are extremely trying and stressful

THIS DESCRIBES MY ACTIVITIES: 1 ☐ Exactly
2 ☐ To some extent
3 ☐ Not very accurately
4 ☐ Not at all

2.68

37 How do you feel about your present job?

1 ☐ Completely satisfied
2 ☐ Neither satisfied nor dissatisfied 3 ☐ Dissatisfied

2.69

38 a) How many brothers were there in your family (excluding yourself)?

.....

b) How many sisters were there in your family (excluding yourself)?

.....

c) Were you your mother's first child, second, third etc.?

.....

2.70 2.71
2.72 2.73
2.74 2.75

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39 How many times have you changed your job since leaving school?

..... 2.96

40 At what age did you finish full-time education?

..... years 2.97 2.98

41 How is your home heated?

1 Electricity 2 Open fire 3 Gas heaters
4 Central heating (any type) 5 Gas & electricity 6 Other 2.99

PLEASE CHECK THAT YOU HAVE ANSWERED ALL THE QUESTIONS.

THANK YOU FOR YOUR HELP.

DEPARTMENT OF COMMUNITY MEDICINE, UNIVERSITY OF GLASGOW

Confidential

Date: Ref. No.

Name: Ser.obser. & Card No.

1 - Height (without shoes) .....inches

2 - Weight (indoor clothing) .....pounds

3a Pulse rate/min ...../min

b Regularity 1 ☐ Regular 2 ☐ Irregular

4 - Blood pressure (at rest)

a Systolic BP .....mmHg.

b Diastolic BP .....mmHg.

5 - Respiratory function tests

a FEV 1.0 .....

b FVC .....

6 - X-ray

a Cardiac size 1 ☐ normal 2 ☐ enlarged

b Lung fields 1 ☐ normal 4 ☐ effusion

2 ☐ oedema 5 ☐ other

3 ☐ venous congestion

7 - ECG changes

1 ☐ Q wave 2 - ST Seg. a ☐ elevation

3 ☐ LVH b ☐ depression - measure

4 ☐ T inverted 5 ☐ A V Block

6 ☐ BBB 7 ☐ Arrythmia

8 ☐ No abnormalities

8 - Myocardial infarction

1 ☐ Yes 2 ☐ No 3 ☐ Not sure

9 - Cardiac Enzymes

1 SCOT (AST).....U/L

2 Total CK.....U/L

3 LD (Lactic dehydrogenase).....U/L

10 - Blood serum: (Fasting)

a - Cholesterol.....mmol/L

b - Triglycerides.....mmol/L

c - Blood sugar.....mmol/L

61	62	63
64	65	66
67	68	69

11_ Carboxy HB level ... .

12- Haemoglobin level.....

13- Blood Oxygen .....

14 - Urine examination

1 ☐ Blood 2 ☐ Glucose 3 ☐ Protein

4 ☐ Normal 5 ☐ More than one

70 ☐

15- B. Blocker use 1 ☐ Yes 2 ☐ No

1

71 ☐

16-Previous Myocardial infarction

1 ☐ Yes 2 ☐ No /3 ☐ Probable

72 ☐

17- Cause of Admission

1 ☐ Emergency 2 ☐ Non emergency

73 ☐

18 - Fate

1 ☐ Survived 2 ☐ Died

74 ☐

DEPARTMENT OF COMMUNITY MEDICINE, UNIVERSITY OF GLASGOW

CONFIDENTIAL

PREVENTION OF SMOKING-RELATED DISEASE: FOLLOW-UP QUESTIONNAIRE 3

FOR OFFICIAL USE

401	402	403	404

SURNAME:(Mr/Mrs/Miss)

FIRST NAMES

ADDRESS POST CODE

TELEPHONE NO.

405
4

1. What is your occupation? (Please describe as fully as possible)

406

2. How much of the following drinks do you usually take each day?

407

a. Milk.....cups b. Coffee.....cups

c. Tea.....cups

408	409
410	411

3. Do you take sugar in tea or coffee?

a. 1 ☐ Yes 2 ☐ No 3 ☐ varies

b. Do you take sugar on cereals or pudding?

1 ☐ Yes 2 ☐ No

412	413

4. How much of the following are you accustomed to taking each week?

a. Spirits.....nips b. Beer.....pints

c. Wine.....glasses

414	415
416	417
418	419

5. a. Do you smoke cigarettes now?

1 ☐ Yes 2 ☐ No. If 'No', go to next question

b. If 'Yes', are they

1 ☐ Low Tar 2 ☐ Medium tar

420
421
422
423

c. Do you inhale?

1 ☐ Yes 2 ☐ No

d. What kind of cigarettes do you smoke?

1 ☐ Manufactured with filter

2 ☐ Manufactured without filter

3 ☐ Hand rolled

424	425
426	427

e. How many cigarettes do you usually smoke per day?

Number per day...

f. How many ounces of tobacco do you use per week for rolling your own cigarettes?

.....oz. per week

g. When did you finish your last cigarette?

since.....hours.....minutes-----.

h. What is your preferred brand name of cigarette?

.....

428 429 430
431 432

6. Do you smoke a pipe now?

1 ☐ Yes 2 ☐ No 3 ☐ Used to smoke a pipe but not now

433

If 'No', go to next question

If you smoke a pipe now, about how many ounces of tobacco do you smoke?

.....oz. per week

434 435
436

Do you inhale?

1 ☐ Yes 2 ☐ No

437 438 439

When did you finish your last smoking?

since.....hours.....minutes

7. Do you smoke cigars now?

1 ☐ No 2 ☐ Used to smoke but not now

3 ☐ Now smoke cigars

440
441 442

If you smoke now, about how many do you smoke per week?

.....per week

8. Have you any pain or discomfort in your chest after discharge from the hospital?

1 ☐ Yes 2 ☐ No

443

9. Is it similar to pain you get before admission to hospital?

1 ☐ Yes 2 ☐ No

444

10. Do you have a raised blood pressure?

1 ☐ Yes 2 ☐ No 3 ☐ Don't know

445

11. Do you have diabetes?

1 ☐ Yes 2 ☐ No 3 ☐ Don't know

446

12. Have you been admitted to hospital in last 2 months?

1 ☐ Yes 2 ☐ No

447

What is the cause?.....

When? Date.....

448 449 450

Health Education Questionnaire 4 Ref Number.....

Please tick the correct answers

1- Have you given a booklet about health education and heart disease in the hospital?

1 ☐ yes 2 ☐ nb 3 ☐ not sure 4 ☐ more than one

2- Is that booklet similar to this booklet?

a- small and yellow. ☐

b- Large blue one. ☐

3-what the important points do you remember which is applicable to you as regards these booklets?

A.....

B-.....

C-.....

4- Please tick the correct answers in this section.

A- Liability to get heart diseases is increased

1 ☐ If heart disease runs in familye

2 ☐ The older the age the greater the risk.

3 ☐ women because they are more at risk than men.

4- ☐ in non smokers.

5- ☐ Those who eat to much and not getting enough excercise.

B- These things might give high blood pressure

1- ☐ Being slim.

2- ☐ smoking.

3- ☐ Eating too much salt.

4- ☐ To much stress.

5- ☐ Never Alcoholic drinks.

6- ☐ Regular physical activities.

c- I think the rules for losing weight are

1- ☐ To cut down the amout of fat in the diet.

2- ☐ To eat more meat.

3- ☐ increase physical activity and sports.

4- ☐ use less lard or oil in cooking.

5- ☐ not to cut down sugar in diet.

Thank you for your co-operation.

0_0_0_0_0_0_0_0_0_0_0_0_0_0_0_0_0_0_0_0_0_0_0_0_0_0_0_0_0

Only

HEALTH EDUCATION QUESTIONNAIRE (INVESTIGATION PART)

5- Height in cm .....

6- Weight in Kg .....

7- COHb % saturation. .....

8- Breath CO ppm .....

midspan health plan

conducted by the
Glasgow MMR Cardiorespiratory Screening Unit and the
Department of Epidemiology and Preventive
Medicine of the University of Glasgow

MMR Reference Number

Questionnaire

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1.01	1.02	1.03	1.04	1.05	1.06	1.07

All information which could permit identification of the individual will be treated as strictly confidential, will be used only by persons engaged in and for the purposes of this survey, and will not be disclosed for any other purpose.

Please answer all the questions in this health record, and bring it with you when you come to the unit. Write a tick in the appropriate boxes. If you cannot give an exact answer, give the best estimate you can.

Surname (Mr., Mrs., Miss) .....
(BLOCK LETTERS)

Christian names .....

Address .....

.....

Works no: .....

Dept no: .....

Check no: .....

1.08

1.09

Family doctor Dr. .....

Address .....

.....

1 a What is your occupation? (Please describe as fully as possible)

.....

.....

1.10	1.11	1.12

b Are you
1 ☐ a manager?
2 ☐ a foreman?
3 ☐ an employee?
4 ☐ self-employed without employees?
5 ☐ self-employed with employees?

1.13

2 a What is your date of birth?

Day of month	Month	Year
.....	.....	.....

1.14	1.15	1.16	1.17	1.18	1.19

b Were you born
1 ☐ in the country? (isolated dwelling, hamlet or small community)
2 ☐ in a village?
3 ☐ in a town?
4 ☐ in a city?
5 ☐ elsewhere? (e.g. at sea)

1.20

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c Where have you lived for the most of your life?

1 ☐ in the country? (isolated dwelling, hamlet or small community)
2 ☐ in a village?
3 ☐ in a town?
4 ☐ in a city?
5 ☐ elsewhere? (e.g. at sea)

1.21

3 Are you

1 ☐ Married?
2 ☐ Widowed?
3 ☐ Single?
4 ☐ Other?

1.22

4 Do you usually bring up any phlegm from your chest first thing in the morning in the winter?

1 ☐ Yes
2 ☐ No (If "No" go to question 7)

1.23

5 Do you bring up phlegm like this on most days for as much as three months in the winter each year?

1 ☐ Yes
2 ☐ No

1.24

6 In the past three years, have you had a period of increased cough and phlegm lasting for 3 weeks or more?

1 ☐ Yes—1 period
2 ☐ Yes—2 or more periods
3 ☐ No

1.25

7 Do you get short of breath walking with people of your own age on level ground?

1 ☐ Yes
2 ☐ No

1.26

8 Does your chest sound wheezy or whistling on most days (or nights)?

1 ☐ Yes
2 ☐ No

1.27

9 Does the weather affect your breathing?

1 ☐ Yes
2 ☐ No

If "Yes", specify type of weather

.....

1.28

10 How much exercise do you get at work?

1 ☐ None
2 ☐ A moderate amount
3 ☐ A lot

1.29

11 How much exercise do you get on the way to and from work each day?

..... minutes

1.30 1.31 1.32

12 How much physical exercise do you get outside work each day or at the weekend?

..... hours per week

1.33 1.34

13 Do you drive a car regularly?

1 ☐ Yes
2 ☐ No

1.35

14 How many hours in twenty-four do you sleep?

..... hours

1.36 1.37

15 How much of the following drinks do you usually take each day?

a Milk ..... pints

b Coffee ..... cups

c Tea ..... cups

1.38

1.39 1.40 1.41 1.42

16 a Do you take sugar in tea or coffee?
1 ☐ Yes 2 ☐ No

b Do you take sugar on porridge or pudding?
1 ☐ Yes 2 ☐ No

17 How much of the following do you usually take each week?

a Spirits ..... nips

b Beer ..... pints

c Wine ..... bottles

M.M.R. ref. no.

18 a Do you smoke cigarettes now?
1 ☐ Yes
2 ☐ No (If "No", go to question 19)

b Do you inhale?
1 ☐ Yes 2 ☐ No

c What kind of cigarettes do you smoke
1 ☐ Manufactured, with filters?
2 ☐ Manufactured, without filters?
3 ☐ Hand-rolled?

d How many *manufactured* cigarettes do you usually smoke per day?

Number per day .....

e About how many ounces of tobacco do you use per week for rolling your own cigarettes?

Oz. per week .....

f What is the maximum number of cigarettes that you have smoked per *day* for as long as a year? (Include hand-rolled)

Number per day .....

g How old were you when you began to smoke cigarettes regularly?

Age .....

19 a If you do not smoke cigarettes now, did you ever smoke them regularly?
1 ☐ Yes 2 ☐ No, never

b If you used to smoke regularly, what is the maximum number of cigarettes you ever smoked per day for as long as a year?

Number of cigarettes .....

c Did you inhale?
1 ☐ Yes 2 ☐ No

d How old were you when you began to smoke cigarettes?

Age .....

e When did you stop smoking cigarettes? (Give year of stopping)

Year .....

20 a Have you ever smoked a pipe regularly?
1 ☐ No (If "No", go to question 21)
2 ☐ Used to smoke a pipe but not now (go to d)
3 Now smoke a pipe

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1.51	1.52	1.53	1.54			1.43	1.44
1.55	1.56	1.57	1.58			1.45	1.46
						1.47	1.48
						1.49	1.50

Survey no. 1.75 1.76 1.77

Serial obs. no. 1.78 1.79

Card no. 1.80 1

2.01	2.02	2.03	2.04	2.05	2.06	2.07

2.08

2.09

2.10

	2.11	2.12
2.13	2.14	2.15
	2.16	2.17

2.18 2.19

2.20

2.21 2.22

2.23

2.24 2.25

2.26 2.27

2.28

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b If you smoke a pipe now, about how many ounces of tobacco do you smoke?
Oz. per week .....

c Do you inhale?
1 ☐ Yes 2 ☐ No

d How old were you when you began to smoke a pipe?
Age .....

e If you do not smoke a pipe now, when did you stop? (Give the year)
Year .....

2.29 2.30 2.31
2.32
2.33 2.34
2.35 2.36

21 Have you ever smoked cigars regularly?

a
1 ☐ No (If "No" go to question 22)
2 ☐ Used to smoke cigars but not now
3 ☐ Now smoke cigars

b If you smoke cigars now, about how many cigars do you smoke per week?
Number per week .....

2.37
2.38 2.39

22 a Have you ever had any pain or discomfort in your chest?
1 ☐ Yes 2 ☐ No (go to 23)

b Do you get this pain or discomfort when you walk uphill or hurry?
1 ☐ Yes 2 ☐ No

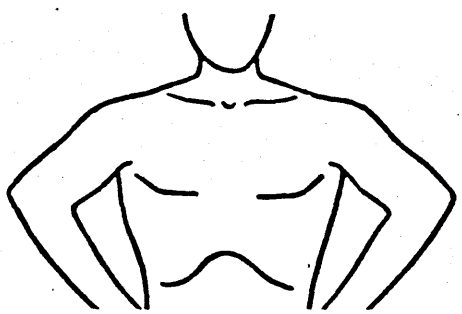
c Do you get it when you walk at an ordinary pace on the level?
1 ☐ Yes 2 ☐ No

d When you get any pain or discomfort in your chest what do you do?
1 ☐ Stop
2 ☐ Slow down
3 ☐ Continue at the same pace.

e Does it go away when you stand still?
1 ☐ Yes 2 ☐ No

f How soon?
1 ☐ 10 minutes or less
2 ☐ More than 10 minutes

g Where do you get this pain or discomfort? Mark the place(s) with X on the diagram.



2.40
2.41
2.42
2.43
2.44
2.45
9 2.46

23 Have you ever had a severe pain across the front of your chest lasting for half an hour or more?
1 ☐ Yes 2 ☐ No

2.47

24 a Do you get a pain in either leg on walking?
1 ☐ Yes 2 ☐ No (go to 25)

b Does this pain ever begin when you are standing still or sitting?
1 ☐ Yes 2 ☐ No

c Do you get the pain in your calf (or calves)?
1 ☐ Yes 2 ☐ No

2.48
2.49
2.50

d Do you get it when you walk uphill or hurry?
1 ☐ Yes 2 ☐ No

e Do you get it when you walk at an ordinary pace on the level?
1 ☐ Yes 2 ☐ No

f Does the pain ever disappear while you are still walking?
1 ☐ Yes 2 ☐ No

g What do you do if you get it when you are walking?
1 ☐ Stop
2 ☐ Slow down
3 ☐ Continue at the same pace

h What happens to it if you stand still?
1 ☐ Usually continues more than ten minutes
2 ☐ Usually disappears in ten minutes or less

25 Do you have diabetes?
1 ☐ Yes
2 ☐ No
3 ☐ Do not know

26 Over the past year have you noticed a definite increase in:

a Thirst?
1 ☐ Yes 2 ☐ No

b The quantity of urine you pass?
1 ☐ Yes 2 ☐ No

c The ease with which you get tired?
1 ☐ Yes 2 ☐ No

27 Over the same time have you noticed:

a Unexplained weight loss?
1 ☐ Yes 2 ☐ No

b Worsening of your eyesight?
1 ☐ Yes 2 ☐ No

c Boils or carbuncles?
1 ☐ Yes 2 ☐ No

d Itching of skin where you pass water?
1 ☐ Yes 2 ☐ No

28 a Have you ever been in hospital?
1 ☐ Yes 2 ☐ No

If "Yes" please give the following information for each admission:

b	Year	Nature of illness or what was wrong?
1	.....	.....
2	.....	.....
3	.....	.....
4	.....	.....

(Continue on separate sheet of paper if necessary)

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2.51	
2.52	
2.53	
2.54	
2.55	
2.56	
2.57	
2.58	
2.59	
2.60	
2.61	
2.62	
2.63	
2.64	
2.65	
2.66	
2.67	
2.68	

Survey no.	2.75	2.76	2.77
Serial obs. no.		2.78	2.79
Card no.			2.80 2

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3.01	3.02	3.03	3.04	3.05	3.06	3.07
			3.08	3.09	3.10	3.11
				3.12	3.13	3.14
					3.15	3.16

29 Are your parents both alive?
a 1 ☐ Yes 2 ☐ No
If "No"
b What caused your father's death? ✓
.....
c What caused your mother's death?
.....

30 If you are married, does your wife/husband suffer from
a bronchitis? 1 ☐ Yes 2 ☐ No
b heart trouble? 1 ☐ Yes 2 ☐ No

31 **a** What was your first regular occupation, excluding temporary work? (please give full description)
.....
.....
b What was your father's main occupation? (Please give full description)
.....
.....

3.17	3.18	3.19
3.20	3.21	3.22

32 Please indicate by a tick in the appropriate box in each of the following sections which description fits you best.
a In general, I am usually tense or nervous
THIS DESCRIBES ME:
1 ☐ Exactly
2 ☐ To some extent
3 ☐ Not very accurately
4 ☐ Not at all

3.23

b There is a great amount of nervous strain connected with my daily activities
THIS DESCRIBES MY SITUATION:
1 ☐ Exactly
2 ☐ To some extent
3 ☐ Not very accurately
4 ☐ Not at all

3.24

c At the end of the day I am completely exhausted mentally and physically
THIS DESCRIBES ME:
1 ☐ Exactly
2 ☐ To some extent
3 ☐ Not very accurately
4 ☐ Not at all

3.25

d My daily activities are extremely trying and stressful
THIS DESCRIBES MY ACTIVITIES:
1 ☐ Exactly
2 ☐ To some extent
3 ☐ Not very accurately
4 ☐ Not at all

	3.26
3.27	3.28
	3.29

33 How do you feel about your present job?
1 ☐ completely satisfied
2 ☐ well satisfied
3 ☐ neither satisfied nor dissatisfied
4 ☐ a little dissatisfied
5 ☐ very dissatisfied

34a How many brothers were there in your family? (Excluding yourself)
.....
b How many sisters were there in your family? (Excluding yourself)
.....
c Were you your mother's first child, second, third, etc?
.....

3.30	3.31
3.32	3.33
3.34	3.35

35 How many times have you changed your job since leaving school?
.....

3.36	3.37
3.38	3.39

36 When did you last change your job? (Please give year)
.....

3.40	3.41

37 At what age did you finish whole time education?
.....

3.75	3.76	3.77
	3.78	3.79

Survey no.

Serial obs. no.

Card no. 3.80 3

Please check that you have answered all the questions. Thank you for your help.

HEALTH QUESTIONNAIRE G.H.Q.

SURNAME..... AGE .....

CHRISTIAN NAMES..... .....

DATE..... HOSPITAL NUMBER.....

Instructions

The following questions are concerned with the way you feel or act. Please circle the answer that applies to you. Do not spend long on any one question.

....	1.	Do you often feel upset for no obvious reason?	Yes = 2 No = 0
....	2.	Do you have an unreasonable fear of being in enclosed spaces such as shops, lifts, etc.?	Often = 2 Sometimes = 1 Never = 0
....	3.	Do people ever say you are too conscientious?	No = 0 Yes = 2
....	4.	Are you troubled by dizziness or shortness of breath?	Never = 0 Often = 2 Sometimes = 1
....	5.	Can you think as quickly as you used to?	Yes = 0 No = 2
....	6.	Are your opinions easily influenced?	Yes = 2 No = 0
....	7.	Have you felt as though you might faint?	Frequently = 2 Occasionally = 1 Never = 0
....	8.	Do you find yourself worrying about getting some incurable illness?	Never = 0 Sometimes = 1 Often = 2
....	9.	Do you think that 'cleanliness is next to godliness'?	No = 0 Yes = 2
....	10.	Do you often feel sick or have indigestion?	Yes = 2 No = 0
....	11.	Do you feel that life is too much effort?	At times = 1 Often = 2 Never = 0
....	12.	Have you, at any time in your life, enjoyed acting?	Yes = 2 No 0
....	13.	Do you feel uneasy and restless?	Frequently = 2 Sometimes = 1 Never = 0
....	14.	Do you feel more relaxed indoors?	Definitely = 2 Sometimes = 1 Not particularly = 0
....	15.	Do you find that silly or unreasonable thoughts keep recurring in your mind?	Frequently = 2 Sometimes = 1 Never = 0
....	16.	Do you sometimes feel tingling or pricking sensation in your body, arms or legs?	Rarely = 1 Frequently = 2 Never = 0

....	17.	Do you regret much of your past behaviour?	Yes = 2 No = 0
....	18.	Are you normally an excessively emotional person?	Yes = 2 No = 0
....	19.	Do you sometimes feel really panicky?	Yes = 2 No = 0
....	20.	Do you feel uneasy travelling on buses or the underground even if they are not crowded?	Very = 2 A little = 1 Not at all = 0
....	21.	Are you happiest when you are working?	Yes = 2 No = 0
....	22.	Has your appetite got less recently?	No = 0 Yes = 2
....	23.	Do you wake unusually early in the morning?	Yes = 2 No = 0
....	24.	Do you enjoy being the centre of attraction?	No = 0 Yes = 2
....	25.	Would you say you were a worrying person?	Very = 2 Fairly = 1 Not at all = 1
....	26.	Do you dislike going out alone?	Yes = 2 No = 0
....	27.	Are you a perfectionist?	No = 0 Yes = 2
....	28.	Do you feel unduly tired and exhausted?	Often = 2 Sometimes = 1 Never = 0
....	29.	Do you experience long periods of sadness?	Never = 0 Often = 2 Sometimes = 1
....	30.	Do you find that you take advantage of circumstances for your own ends?	Never = 0 Sometimes = 1 Often = 2
....	31.	Do you feel often 'strung-up' inside?	Yes = 2 No = 0
....	32.	Do you worry unduly when relatives are late coming home?	No = 0 Yes = 2
....	33.	Do you have to check things you do to an unnecessary extent?	Yes = 2 No = 0
....	34.	Can you get off to sleep alright at the moment?	No = 2 Yes = 0
....	35.	Do you have to make a special effort to face up to a crisis or difficulty?	Very much so = 2 Sometimes = 1 Not more than anyone else = 0
....	36.	Do you often spend a lot of money on clothes?	Yes = 2 No = 0
....	37.	Have you ever had the feeling you are 'going to pieces'?	Yes = 2 No = 0
....	38.	Are you scared of heights?	Very = 2 Fairly = 1 Not at all = 0

....	39.	Does it irritate you if normal routine is disturbed?	Greatly = 2 A little = 1 Not at all = 0
....	40.	Do you often suffer from excessive sweating or fluttering of the heart?	No = 0 Yes = 2
....	41.	Do you find yourself needing to cry?	Frequently = 2 Sometimes = 1 Never = 0
....	42.	Do you enjoy dramatic situations?	Yes = 2 No = 0
....	43.	Do you have bad dreams which upset you when you wake up?	Never = 0 Sometimes = 1 Frequently = 2
....	44.	Do you feel panicky in crowds?	Always = 2 Sometimes = 1 Never = 0
....	45.	Do you find yourself worrying unreasonably about things that do not really matter?	Never = 0 Frequently = 2 Sometimes = 1
....	46.	Has your sexual interest altered?	Less = 2 The same or greater = 0
....	47.	Have you lost your ability to feel sympathy for other people?	No = 0 Yes = 2
....	48.	Do you sometimes find yourself posing or pretending?	Yes = 2 No = 0

PLEASE CHECK THAT EACH QUESTION HAS BEEN ANSWERED

FOR HOSPITAL USE ONLY

A..... O..... D.....

P..... S..... H.....

1 .....

2 .....

3 .....

Work History since last review:

Patient has returned to work: 1=No, 2=Yes, sporadic, 3=Yes, continuous

Average hours per week, at present:

Same work as before: 1=Same employer, 2=Other employer

Modified work: 1=Same employer, 2=Other employer

If other work, specify:

I.L.O. Code

No. of weeks from infarct till return to work

Advised to return by Consultant: 1=No, 2=Yes

Did patient return to work as advised: 1=No, 2=Yes

If not, state reason:

Smoking: Rehabilitation measures; changes in habit, since last review;
1=None, 2=General reinforcement, 3=Specific (hypnosis etc)

If 3, specify therapeutic measures:

If 3, number of sessions required: at clinic
at home

Present status: 1=Stopped, 2=Continues same, 3=Restarted 4=Reduced
5=Increased 6=Non-smoker 7=Ex-smoker

If smoking: 1= 1-5cigs 2= 6-10cigs 3= 11-20cigs 4= 20+cigs
5= Pipe, light 6= Pipe, heavy 7= Cigar, light
8= Cigar, heavy 9= Variable (specify)

Exercise Status:

Rest Status:

Drugs prescribed at present: No change/Changed to:

Considered fit to return to work: 1=See again at next review
2=Yes, now. 3=In specified time

Referred to: Occupational Therapy; Social Work Dept; Psychologist/Psychiatrist;
Dietician; E.T.T.; Coronary Angiography.

Other referrals:

DIETARY QUESTIONNAIRE

Please answer all questions.

1. How many eggs do you eat each week? _____

2. Do you use butter or margarine? _____
   Do you spread it thickly? moderately thick? thinly?
   If you use margarine which type odo you use? _____

3. Do you eat cheese? YES/NO Which kind? _____
   About how much do you eat a week? _____

4. How many cups of tea do you drink each day? _____
   How many teaspoons of sugar in a cup? _____

5. How many cups of coffee each day? _____
   How many teaspoons of sugar in each cup? _____

6. How much milk do you drink each day? _____
   (Include all milk consumed in cereal, porridge, pudding, etc.)

7. Do you add sugar to cereal, porridge, puddings, etc? _____

8. What do you eat in a typical day? Give a brief history of one day's eating - time of meal and what is eaten (also add in 'elevenses').

   _____

   _____

   _____

   _____

   _____

9. How much squashes, lemondades, etc. do you drink per week? _____

10. How much alcohol do you drink per week? _____ and what kind of alcohol do you usually drink? _____

11. How much bread and rolls do you eat each day? _____

12. About how many cakes, biscuits and pastries do you eat each day? _____

13. Do you eat fried foods? _____ Which type of fat is used in cooking that food? _____

14. How often do you eat fresh fruits? Once a day/a week/a month/Never?

15. Do you often eat jam? How many teaspoons per day? _____
    Do you eat marmalade? How many teaspoons per day? _____
    Do you take honey, syrup or treacle? _____

16. Have you ever been given advice about going on a diet? _____
    What advice were you given? _____
    Who gave you that advice? _____
    How long did you stay on that diet? _____

I.B.Q. W.I.G.

Here are some questions about you and your illness. Circle either YES or NO to indicate your answer to each question.

1. Do you worry a lot about your health?	YES	NO
2. Do you think there is something seriously wrong with you body?	YES	NO
3. Does your illness interfere with your life a great deal?	YES	NO
4. Are you easy to get on with when you are ill?	YES	NO
5. Does your family have a history of illness?	YES	NO
6. Do you think you are more liable to illness than other people?	YES	NO
7. If the doctor told you that he could find nothing wrong with you would you believe him?	YES	NO
8. Is it easy for you to forget about yourself and think about all sorts of other things?	YES	NO
9. If you feel ill and someone tells you that you are looking better, do you become annoyed?	YES	NO
10. Do you find that you are often aware of various things happening in your body?	YES	NO
11. Do you ever think of your illness as a punishment for something you have done wrong in the past?	YES	NO
12. Do you have trouble with your nerves?	YES	NO
13. If you feel ill or worried, can you be easily cheered up by the doctor?	YES	NO
14. Do you think that other people realise what its like to be sick?	YES	NO
15. Does it upset you to talk to the doctor about your illness?	YES	NO
16. Are you bothered by many pains and aches?	YES	NO
17. Does your illness affect the way you get on with your family or friends a great deal?	YES	NO
18. Do you find that you get anxious easily?	YES	NO
19. Do you know anybody who has had the same illness as you?	YES	NO
20. Are you more sensitive to pain than other people?	YES	NO
21. Are you afraid of illness?	YES	NO
22. Can you express your personal feelings easily to other people?	YES	NO
23. Do people feel sorry for you when you are ill?	YES	NO

24. Do you think that you worry about your health more than most people?	YES	NO
25. Do you find that your illness affects your sexual relations?	YES	NO
26. Do you experience a lot of pain with your illness?	YES	NO
27. Except for your illness, do you have any problems in your life?	YES	NO
28. Do you care whether or not people realise you are sick?	YES	NO
29. Do you find that you get jealous of other people's good health?	YES	NO
30. Do you ever have silly thoughts about your health which you can't get out of your mind, no matter how hard you try?	YES	NO
31. Do you have any financial problems?	YES	NO
32. Are you upset by the way people take your illness?	YES	NO
33. Is it hard for you to believe the doctor when he tells you there is nothing for you to worry about?	YES	NO
34. Do you often worry about the possibility that you have got a serious illness?	YES	NO
35. Are you sleeping well?	YES	NO
36. When you are angry, do you tend to bottle up your feelings?	YES	NO
37. Do you often think that you might suddenly fall ill?	YES	NO
38. If a disease is brought to your attention (through the radio, television, newspapers or someone you know) do you worry about getting it yourself?	YES	NO
39. Do you get the feeling that people are not taking your illness seriously enough?	YES	NO
40. Are you upset by the appearance of your face or body?	YES	NO
41. Do you find that you are bothered by many different symptoms?	YES	NO
42. Do you frequently try to explain to others how you are feeling?	YES	NO
43. Do you have any family problems?	YES	NO
44. Do you think there is something the matter with your mind?	YES	NO
45. Are you eating well?	YES	NO
46. Is your bad health the biggest difficulty of your life?	YES	NO
47. Do you find that you get sad easily?	YES	NO
48. Do you worry or fuss over small details that seem unimportant to others?	YES	NO

49.	Are you always a co-operative patient?	YES	NO
50.	Do you often have the symptoms of a very serious disease?	YES	NO
51.	Do you find that you get angry easily?	YES	NO
52.	Do you have any work problems?	YES	NO
53.	Do you prefer to keep your feelings to yourself?	YES	NO
54.	Do you often find that you get depressed?	YES	NO
55.	Would all your worries be over if you were physically healthy?	YES	NO
56.	Are you more irritable towards other people?	YES	NO
57.	Do your think that your symptoms may be caused by worry?	YES	NO
58.	Is it easy for you to let people know when you are cross with them?	YES	NO
59.	Is it hard for you to relax?	YES	NO
60.	Do you have personal worries which are not caused by physical illness?	YES	NO
61.	Do you often find that you lose patience with other people?	YES	NO
62.	Is it hard for you to show people your personal feelings?	YES	NO

HOME AND EXERCISE ROUTINE

In order to get you back to full physical fitness we would advise graded exercises.

During the first week at home –

(1) Stay in the house but activities should increase daily including use of stairs.

(2) Exercises for a few minutes, other than walking, three times a day, e.g.

(a) deep breathing

(b) knee bending exercises with a straight back, hands resting on a table.

(d) stretching the arms upwards and back down to the sides together with trunk twisting.

After the first week, start taking two or three walks a day, very short at first (to the first lamp post only) but increasing gradually (maybe by a lamp post at a time at first) until by three to four weeks after leaving hospital you are walking 2/3 miles total every day. Those with outside stairs and no lift should take one walk per day only at first. Do not walk immediately after meals or on a very cold or windy day.

If exercise causes repeated chest pain or undue breathlessness or fatigue contact your doctor, but if pain, breathlessness or fatigue is slight stop for a few minutes and then continue more slowly. Take extra rest that night but do the same amount of exercise the next day. Keep at the same level of exercise per day until you can do it without stress and then steadily increase again. If you have chest pain lasting 15 minutes or longer contact your doctor immediately. Light household duties can be substituted for one of the daily walks after the first week.

With this regime you can, in time, become fitter than you were before your infarction.

DEPARTMENT OF
COMMUNITY MEDICINE

RUCHILL HOSPITAL
GLASGOW G20 9NB
TEL: 041-946 7120

May, 1984

Dear Patient,

I expect you may remember that we met while you were in the Western Infirmary. You were kind enough to answer some questions about your health, and to provide a blood specimen.

I am now in the process of following up all the patients, and I would like to see you again to ask a few more questions and do another simple blood test.

A special clinic has been set up for the purpose in the Clydebank Health Centre between 2.30 and 4.45 p.m.

Please indicate below on which Wednesday you can attent the appointment. Funds are available to defray the cost of your travel up to about £3.

I hope that you are now well and look forward to seeing you again.

Yours sincerely,

Dr H El-Gendawy

DR. H. EL-GENDAWY

After ticking the suitable date, please detach this portion and return in the enclosed envelope to me at the above address.

Name .....

Ref. No. .....

Wednesday / /84 at ..... p.m.

OR Wednesday / /84 at ..... p.m.

or Wednesday / /84 at ..... p.m.

or Wednesday / /84 at ..... p.m.

DEPARTMENT OF
COMMUNITY MEDICINE

RUCHILL HOSPITAL
GLASGOW G20 9NB
TEL: 041-946 7120

July 1984.

Dear Patient,

I expect you may remember that I sent a letter to you a short time ago in order to explain that I am now in the process of following-up all the patients I met in the Western Infirmary and I would like to see you again for about 10 minutes to ask a few questions, and to do another simple blood test.

Probably you haven't enough time but I am sure you are interested to participate in this study concerning health education as regards ischaemic heart diseases and smoking. Particularly you may know that Scotland has a higher frequency of ischaemic heart diseases.

A special clinic has been set up for the purpose in Clydebank Health Centre on Wednesday July between 2.30 and 5.00 p.m. the last day. You can come at any time between these hours and please indicate below at which time you can attend. You can come by taxi and the travel expenses will be paid up to £4.

I hope you are now well and look forward to seeing you again.

Yours sincerely,

DR. H. EL-GENDAWY

.....

Please detach this portion and return in the enclosed envelope to me at the above address.

Name.....

Ref. No.....

Wednesday 7/84 at .....p.m.

DEPARTMENT OF
COMMUNITY MEDICINE

RUCHILL HOSPITAL
GLASGOW G20 9NB
TEL: 041-946 7120

July 1984.

Dear Patient,

I expect you may remember that I sent a letter to you a short time ago in order to explain that I am now in the process of following-up all the patients I met in the Western Infirmary and I would like to see you again for about 10 minutes to ask a few questions, and to do another simple blood test.

Probably youhaven't enough time but I am sure you are interested to participate in this study concerning health education as regards ischaemic heart diseases and smoking. Particularly you may know that Scotland has a higher frequency of ischaemic heart diseases.

A special clinic has been set up for the purpose in the Western Infirmary on Friday July between 2.30 and 5.00 p.m. the last day. You can come at any time between these hours and please indicate below at which time you can attend. You can come by taxi and the travel expenses will be paid up to £4.

I hope you are now well and look forward to seeing you again.

Yours sincerely,

DR. H. EL-GENDAWY

N.B. Enter by Level 3 and the clinic is on Level 4 in the Western Infirmary. Ask the receptionist on Level 4 for directions.

.....

Please detach this portion and return in the enclosed envelope to me at the above address.

Name.....

Ref. No.....

Friday 7/84 at .....p.m.

APPENDIX B

CODING FOR STRESS

Q36 - Please indicate by a tick in the appropriate box in each of the following sections which description fits you best.

a) In general I am usually tense or nervous.

THIS DESCRIBES ME: 1- Exactly.
2- To some extent.
3- Not very accurately.
4- Not at all.

b) There is a great amount of nervous strain connected with my daily activities.

THIS DESCRIBES MY SITUATION : 1- Exactly.
2- To some extent.
3- Not very accurately.
4- Not at all.

c) At the end of the day I am completely exhausted mentally and physically.

THIS DESCRIBES ME : 1- Exactly.
2- To some extent.
3- Not very accurately.
4- Not at all.

d) My daily activities are extremely trying and stressful.

THIS DESCRIBES MY ACTIVITIES : 1- Exactly.
2- To some extent.
3- Not very accurately.
4- Not at all.

DEFINITION OF THE SCALE[103].

The scale runs from "exactly" 1 to 8 where 1 represents a response of 1 to all four questions (the highest stress) and 8 represents "not at all" responses to all four questions.

Coding procedure.

Score

1. 1111 Exactly on all four.
2. 111X or 1122 (not including 1114) "exactly" on any three," to some extent" or "not very accurately" on the fourth or "exactly" on any two and "to some extent on the other two".
3. 2222 or 1222 "To some extent" on all four or " to some extent" on any three and "exactly "on the fourth.
4. All others not specified in 1,2,3,5,6,7 or 8.
5. 44XX or 4XXX (including 1114) "not at all" or any one or any two, and any other responses on the other three or two (except 4433).
6. 4433 "Not at all" or any two "not very accurate on the other two.
7. 444X "Not at all" or any three,any other response on the fourth.
8. 4444 "Not at all" on all four.

Scale 1,2 form a severe degree of stress.

Scale 3,4 forms moderate degree of stress.

Scale 5,6,7 represents mild forms of stress.

Scale 8 means no stress.

Winter Phlegm

A positive history is a 'Yes' response to "Do you usually bring up any phlegm from your chest first thing in the morning in winter?"

Persistent winter phlegm

A respondent is considered to have persistent winter phlegm if he answered 'Yes' to the question "Do you usually bring up any phlegm from your chest first thing in the morning in winter? and 'Yes' to the question "In the past three years have you had a period of increased cough and phlegm lasting for three weeks or more ?

Dyspnoea

A positive history is a 'Yes' response to "Do you get short of breath while walking with people of your own age on level ground?

Chronic bronchitis

Defined as the presence of persistent phlegm production with an exacerbation lasting three weeks or more in the past three years, and shortness of breath while walking with people of the same age on level (ie. an examinee is considered to have a history of chronic bronchitis when he has persistent winter phlegm and dyspnoea at the same time)(103).

Angina(103)

(Question numbers refer to Questionnaire - see Appendix A1)

A positive history of angina is based on the pattern of positive answers to questions 2.26 parts a,b,c,d,e,f,and g:

a. Have you ever had any pain or discomfort in your chest?

b. Do you get this pain or discomfort when you walk uphill or hurry?

c. Do you get it when you walk at an ordinary pace on the level?

The angina assessment also depends on the answers to the following questions:

d. When you get any pain or discomfort in your chest, what do you do? Stop, slow down, or continue at the same pace?

e. Does it go away when you stand still?

f. How soon? 10 minutes or less, or more than 10 minutes? Positive answers: "Ten minutes or less"

g. Respondents are asked to mark the place(s) with X on the diagram.

Then angina is classified into four grades:

Grade 0 : No to part a. Yes to (a)&(b) and to (c) and

Grade 1: Stop or slow down to (d) and yes to (e) and 10 min. or less to (f) and X on sternum or left ant. chest +left arm.

Grade 2: As grade 1 but yes to (c)

Grade 3 : Yes to (a) + any other answer.

Grade 1,2 are combined and represent severe form of angina.

Grade 3 represent mild grade of angina.

Grade 0, means no angina, thus those with positive history of angina in fact have either grade I or grade II angina.

"Possible infarction" A respondent was classified as giving a history of possible infarction if he answered 'Yes' to the

question "Have you ever had a severe pain across the front of your chest lasting for half an hour or more?"

Hypertension

Hypertensives are those who have either systolic BP more than 150 mm Hg or diastolic blood pressure more than 100 mm Hg or both.

Criteria for Acute Myocardial Infarction[41]

1. Typical chest pain of more than 30 minutes duration.
2. Serial changes in the elctrocardiogram indicating ST segment elevation associated with terminal inversion of T- waves and the loss of initial QRS potential.
3. Rise in serum SGOT above 60 units and LDH > 500 units.

Myocardial infarction was diagnosed if at least two of the following three criteria were present; definite clinical picture, ECG changes (development of pathological Q waves or S-T, T changes) and a significant rise of Cardiac enzymes.

The Body Mass Index:(BMI) is derived by dividing weight by height squared [142]. It is used because it takes weight into greatest account and height to a lesser degree. On the basis of weight-height limits the upper limit of normal BMI for men was 25 Kg/m^2 and for women 24 Kg/m^2. Persons whose BMI exceeded these limits are considered "overweight".

Effort Pain

(Question numbers refer to Questionnaire, see Appendix A1)

If 'Yes' to question 26g and the site of pain is in the (a) sternum (upper or middle) or (b) sternum (lower) or (c) left anterior chest or (d) left arm or (e) other.

If 'No' to question 26c: grade 1

If 'Yes' to question 26c: grade 2

Possible infarction

If 'Yes' to question 27: yes

If 'No' to question 27: No

Intermittent claudication

If 10 minutes or less (question 28/h2) to (Q28/h1): Yes

If 'No' to question 28/e : grade 1

If 'Yes' to question 28/e : grade 2

If more than 10 minutes (Q28/h1) :No

```
RUN NAME        I.H.D.
PAGESIZE        NOEJECT
VARIABLE LIST   VAR101 TO VAR152,VAR201 TO VAR255,VAR301 TO VAR337
                ,VAR401 TO VAR453
INPUT MEDIUM    DISK
INPUT FORMAT    FIXED(F4.0,4F1.0,3F2.0,13F1.0,F3.0,F2.0,3F1.0,F2.0,F1.0,2F2.0,
                2F1.0,3F2.0,5F1.0,4F2.0,F1.0,F2.0,F1.0,5F2.0/
                F4.0,2F1.0,F2.0,F1.0,2F2.0,2F1.0,F2.0,F1.0,F2.0,20F1.0,
                3F3.0,F1.0,F3.0,F2.0,F3.0,F2.0,9F1.0,3F2.0,F1.0,F2.0,F1.0/
                F4.0,F1.0,3F3.0,F1.0,2F3.0,2F3.2,13F1.0,3F4.0,F2.1,5F3.1,5F1.0/
                F4.0,3F1.0,2F2.0,2F1.0,3F2.0,4F1.0,2F2.0,F3.0,F2.0,F1.0,F2.0,
                F1.0,F3.0,F1.0,F2.0,5F1.0,F2.0,18F1.0,F3.0,3F3.1)
PRINT FORMATS   VAR307 TO VAR310,VAR327 TO VAR332,VAR451 TO VAR453(1)
N OF CASES      UNKNOWN
RECODE          VAR233 TO VAR235,VAR237,VAR239(11,25=11)(14,16=14)(15,22=15)
                (17,19,20=17)
VAR LABELS      VAR101,CASE NO/
                VAR102,CARD NO/
                VAR103,S.CLASS/
                VAR104,EMPLOY/
                VAR105,HUSB OCCUP/
                VAR106,BIR DAY/
                VAR107,BIR MON/
                VAR108,BIR YEAR/
                VAR109,RESID/
                VAR110,INDUST/
                VAR111,SEX/
                VAR112,TWIN/
                VAR113,LIKE TWIN/
                VAR114,MARITAL S/
                VAR115,COUGH/
                VAR116,MORN PHLE/
                VAR117,3M PHLE/
                VAR118,3W PHLE/
                VAR119,WAK BREATH/
                VAR120,WHEEZES/
                VAR121,WORK EX/
                VAR122,WAY EX/
                VAR123,OUT EX/
                VAR124,WORK ACT/
                VAR125,PAST EXP/
                VAR126,TIME/
                VAR127,SLEEP/
                VAR128,MILK D/
                VAR129,COF DRIN/
                VAR130,TEA D/
                VAR131,SUG INT/
                VAR132,CER SUG/
                VAR133,SPIRITS/
                VAR134,BEAR D/
                VAR135,WINE/
                VAR136,SALAD/
                VAR137,CIGT SMO/
                VAR138,TAR/
                VAR139,INHA/
                VAR140,CIGT KIND/
                VAR141,CIGT NO/
                VAR142,TOBA OZ/
                VAR143,CIGT M NO/
                VAR144,SMO AGE/
                VAR145,XCIGTSM/
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VAR146,M CIG NO/
VAR147,INHA/
VAR148,SMO AGE/
VAR149,SMO STOP/
VAR150,AD DAY/
VAR151,AD MON/
VAR152,AD YEAR/
VAR201,CASE NO/
VAR202,CARD NO/
VAR203,PIPE SMO/
VAR204,TOBA OZ/
VAR205,INHA/
VAR206,PIPE SM AGE/
VAR207,STOP YEAR/
VAR208,FAM SMO/
VAR209,SMO TYPE/
VAR210,SMO DURA/
VAR211,CIGAR/
VAR212,CIGAR NO/
VAR213,CH PAIN/
VAR214,HURRY PAIN/
VAR215,PACE PAI/
VAR216,PAIN EFF/
VAR217,PAIN FATE/
VAR218,PAIN DURA/
VAR219,PAIN  SITE
VAR220,SCHEST PAIN/
VAR221,LIMB PAIN/
VAR222,REST PAI/
VAR223,CALF PAI/
VAR224,HURRY PAI/
VAR225,PACE PAI/
VAR226,WALK PA FATE/
VAR227,RESPON/
VAR228,PAIN FATE/
VAR229,DIABETES/
VAR230,BP/
VAR231,MEDI USE/
VAR232,PRE HOSPIT/
VAR233,FIR AD/
VAR234,SE AD/
VAR235,TH AD/
VAR236,PARENTS/
VAR237,FDEA CAUSE/
VAR238,F DEA AGE/
VAR239,M DEA CAUSE/
VAR240,M DEA AGE/
VAR241,SPOU BRONCH/
VAR242,SPOU HEART /
VAR243,FI SO CL/
VAR244,FATH S CL/
VAR245,TENSION/
VAR246,STRAIN/
VAR247,EXHAUS/
VAR248,DAILY ACT/
VAR249,JOB FEELING/
VAR250, BROTH NO/
VAR251,SIST NO/
VAR252,ORDER/
VAR253,JOB CHAN/
VAR254,EDUC FIN/
VAR255,HEATING/
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VAR301,CASE NO/
VAR302,CARD NO/
VAR303,HIGHT/
VAR304,WEIGHT/
VAR305,PULSE/
VAR306,REGU/
VAR307,S.BP/
VAR308,D.BP/
VAR309,FEV1/
VAR310,FVC/
VAR311,HRT SIZE/
VAR312,LUNGS/
VAR313,Q WAVE/
VAR314,ST ELEV/
VAR315,ST DEP/
VAR316,ST DEPTH/
VAR317,LVH/
VAR318,T INVER/
VAR319,AV BLOCK/
VAR320,BBB/
VAR321,ARRYTHMIA/
VAR322,NO ABN/
VAR323,M INF/
VAR324,SGOT/
VAR325,TOT CK/
VAR326,LDH/
VAR327,CHOLEST/
VAR328,TRIG/
VAR329,SUG/
VAR330,COHB/
VAR331,HB/
VAR332,OXYHB/
VAR333,URINE/
VAR334,B. BLOCK/
VAR335,OLD MI/
VAR336,ADM CASE/
VAR337,FATE/
VAR401,CASE NO/
VAR402,CARD NO/
VAR403,S.CLASS/
VAR404,MILK/
VAR405,COFFEE/
VAR406,TEA/
VAR407,DRINK SUGAR/
VAR408,C.P.SUGAR/
VAR409,SPIRITS/
VAR410,BEAR/
VAR411,WINE/
VAR412,CIGT SMOK/
VAR413,TAR/
VAR414,INHA/
VAR415,CIGT KIND/
VAR416,CIGT NO/
VAR417,TOB OZ/
VAR418,LAST CIGT/
VAR419,BRAND NAME/
VAR420,PIPE SMO/
VAR421,TOB OZ PIPE/
VAR422,INHA/
VAR423,LAST PIPE/
VAR424,CIGAR SMO/
VAR425,CIGAR NO/
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                VAR426,CHEST P/
                VAR427,SIMI P/
                VAR428,HIGH BP/
                VAR429,DIABETES/
                VAR430,HOSP ADM/
                VAR431,ADM CAUSE/
                VAR432,H ED BOOK/
                VAR433,BOOK SIMI/
                VAR434  HD LIABILITY/
                VAR435
                VAR436
                VAR437
                VAR438
                VAR439  HIGH BP/
                VAR440
                VAR441
                VAR442
                VAR443
                VAR444
                VAR445  ,WGHT LOSS/
                VAR446
                VAR447
                VAR448
                VAR449
                VAR450,HEIGHT/
                VAR451,WEIGHT/
                VAR452,COHB/
                VAR453,BREATH CO/
VALUE LABELS    VAR103,(0)UNEMPLO (1)SO CLASS 1(2)SO CLASS 2(3)SO CLASS 3NM
                (4)SO CLASS 3M(5) SO CL4(6)SO CL 5(7)RETIR(8)NOT APPL/
                VAR104(1)MANAG(2)FOREMAN(3)EMPLOY(4)S EMPLOY(5)S EMPO W EMPL/
                VAR105(0)UNEMPLO(1)SO CLASS 1(2)S CL 2(3)S CL 3NM(4)S CL3M
                (5)SO CL4(6)SO CL 5(7)RETIR /
                VAR109(1)COUNTRY(2)VILLAGE(3)TOWN(4)CITY(5)E WHERE/
                VAR110(1)YES(2)NO/
                VAR111(1) MALE (2) FEMALE/
                VAR112(1)YES(2)NO/
                VAR113(1)LIKE(2)UNLIKE(8)NOT APP(9)MISSED/
                VAR114(1)MARRIED(2)SING(3)WIDO(4)SEPA(5)DIVOR/
                VAR115 TO VAR117(1)YES(2)NO/
                VAR118(1)ONE PERIOD(2)2 PERIODS(3)NO/
                VAR119 TO VAR120(1)YES(2)NO/
                VAR121(1)NO(2)SMALL(3)LOT/
                VAR124(1)SEVER(2)MOD(3)NON(4)UNSURE/
                VAR125(1)JOB LOSS(2)F MEM DEATH(3)FINAN PROB(4)RESID CHANGE
                (5)M THAN ONE(6)NONE/
                VAR126(1)1 MON AGO(2)3 MON AGO(4)7-12 MON AGO/
                VAR131(1)YES(2)NO(3)VARI/
                VAR132(1)YES(2)NO/
                VAR136(1)MOST OF WK(2)ONCE WK(3)NEVER/
                VAR137(1)YES(2)NO/
                VAR138(1)LOW(2)MEDIUM /
                VAR139(1)YES(2)NO/
                VAR140(1)FILTER(2) N FILTER(3)ROLLED/
                VAR145(1)YES(2)NO/
                VAR147(1)YES(2)NO/
                VAR203(1)NO(2)PAST SMO(3)YES/
                VAR205(1)YES(2)NO/
                VAR208(1)FATH(2)MOTH(3)SIST(4)BROTH(5)MORE(6)NONE/
                VAR209(1)CIGTS(2)CIGAR(3)PIPE(4)COMBINED/
                VAR211(1)NO(2)PA CIG SM(3)NON SMO/
                VAR213 TO VAR215(1)YES (2)NO/
```

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                VAR216(1)STOP(2)SLOW(3)CONTINUE/
                VAR217(1)YES(2)NO/
                VAR218(1)<10MIN(2)>10 MIN/
                VAR219(1)YES(2)NOT MARKED/
                VAR220 TO VAR226 (1) YES(2)NO/
                VAR227(1) STOP(2) SLOW(3) CONTIN/
                VAR228(1)CONTIN>10 MIN(2) DISAPPEAR/
                VAR229 TO VAR230 (1)YES(2)NO(3) DONT KNOW/
                VAR231 TO VAR232 (1) YES(2) NO/
                VAR233 TO VAR235,VAR237,VAR239,VAR431(1)LUNG CANCER(2)OTHER CANCER
                (3) IHD(4)CEREB V D(5)VAS D (6)OBST LUNG D(7)OTH RESP D(8)OLD AGE
                (9)INFEC (10)NERVOUS D(11)URIN T(12)GYN-OBST(13)DIGEST S(14)ACCID
                (15)OTH HRT D(16)WAR INJ(17)ENDOCRINE D(18)PSYCH(19)AUTOIMM
                (20)DIABETES(21)ANGINA(22)HRT FAIL(23)EYE(24)HERNIA(25)GENITAL S
                (26)ENT(27) BONE D /
                VAR236(1)YES(2)NO/
                VAR237(-98)NOT APPL(-99)MISSED/
                VAR238(98)NOT APPL(99)MISSED/
                VAR239(-98)NOT APPL(-99)BLANK/
                VAR240(-98)NOT APPL(-99)MISSED/
                VAR241 TO VAR242(1)YES(2)NO(8)NOT APPL(9)MISSED/
                VAR243 TO VAR244(0)UNEMPL(1)S CL1(2)S CL2(3)S CL3NM(4)S CL3M
                (5)S CL4(6)S CL 5(7)RETIR(8)NOT APPL(9)MISSED/
                VAR245 TO VAR248(1)EXAT(2)SOME EX(3)NOT ACC(4)NO(8)N APP(9)MISSD/
                VAR249(1)SATISFIED(2)NEITHER(3)DISSATS(8)NOT APPL(9)MISSED/
                VAR255(1)ELECT(2)O FIRE(3)GAS(4)CENT H(5)GAS&ELEC(6)OTHER(7)>1/
                VAR311(1)NORM(2)ELARGMENT(9)MISSED/
                VAR312(1)NORM(2)OEDEMA(3)CONGES(4)EFFUS(5)OTHER(9)MISSED/
                VAR313 TO VAR315(1)YES(2)NO(9)BLANK/
                VAR317 TO VAR322(1)YES(2)NO(9)BLANK/
                VAR323(1)YES(2)NO(3)NOT SURE/
                VAR333(1)BLOOD(2)GLUCOSE(3)PROT(4)NORM(5)MORE THAN 1(9)MISSED/
                VAR334 TO VAR335(1)YES(2)NO(3)PROBABLE(9)MISSED/
                VAR336(1)EMERG(2)NON EMERG(9)MISSED/
                VAR337(1)SURVIVE(2)DIED(0)UNKNOWN/
                VAR403(0)UNEMPLO(1)SO CLAA 1(2)SO CLASS 2(3)SO CLASS 3NM
                (4)SO CLASS 3NM(5)SO CL4(6)SO CL 5(7)RETIR(8)NOT APPL/
                VAR407(1)YES(2)NO(3)VARIES/
                VAR408(1)YES(2)NO/
                VAR412(1)YES(2)NO/
                VAR413(1)LOW (2)MEDIUM TAR/
                VAR414(1)YES(2)NO/
                VAR415(1)MANF+FILT(2)MANF W FILT(3)HAND ROLLED/
                VAR420(1)YES(2)NO(3)SM IN PAST/
                VAR422(1)YES(2)NO/
                VAR424(1)YES(2)NO/
                VAR426(1)YES(2)NO/
                VAR427(1)YES(2)NO/
                VAR428(1)YES(2)NO(3)DONT KNOW/
                VAR429(1)YES(2)NO(3)DONT KNOW/
                VAR430(1)YES(2)NO/
                VAR432(1)YES(2)NO(3)NOT SURE(4)MORE T ONE/
                VAR433(1)SMALL(2)LARGE(3)BOTH(4)NONE/
                VAR434 TO VAR449 (1)YES(2)NO/
MISSING VALUES  VAR103(9),VAR105(8)VAR106 TO VAR108(99),VAR109(9)/
                VAR109(9),VAR113 TO VAR121(9)/
                VAR122(999),VAR123(99),VAR124 TO VAR126(9),VAR127(99)/
                VAR128(9),VAR129 TO VAR130(99),VAR131 TO VAR132(9),/
                VAR133 TO VAR135(99),VAR136 TO VAR140(9),VAR141 TO VAR144(98,99)/
                VAR145(9),VAR146(99),VAR147(9),VAR148 TO VAR149(99),VAR203(9),/
                VAR204(98,99),VAR205(9),VAR206 TO VAR207(99),VAR208 TO VAR209(9),/
                VAR210(99),VAR211(9),VAR212(98,99),VAR213 TO VAR218(9),/
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                VAR220 TO VAR232(9),VAR233 TO VAR235(-99),VAR236(9),/
                VAR237(-99),VAR238(99),VAR239(-99),VAR240(99),/
                VAR241 TO VAR249(9),VAR250(99),VAR251 TO VAR252(99),/
                VAR253(9),VAR254(99),VAR255(99),VAR303 TO VAR305(999),/
                VAR306(9),VAR307 TO VAR310(999),VAR311 TO VAR327(9),/
                VAR324 TO VAR326(-999),VAR327(-9),VAR328 TO VAR332(-99),/
                VAR333 TO VAR337(9),/
                VAR404(9),VAR405 TO VAR406(99),VAR407 TO VAR408(9)/
                VAR409 TO VAR411(98,99),VAR416 TO VAR417(98,99),/
                VAR418(-98,-99),VAR419(98,99),VAR420(8,9),VAR421(98,99),/
                VAR422(9),VAR423(-98,99),VAR424(8,9),VAR425(98,99),/
                VAR426 TO VAR440(8,9),VAR450 TO VAR453(-98,-99),/
COMPUTE         AGE=(VAR152-VAR108)+(VAR151-VAR107)/12
COMPUTE         AGEGP=AGE
RECODE          AGEGP(LO THRU 24.9=1)(25 THRU 34.9=2)(35 THRU 44.9=3)
                (45 THRU 54.9=4)(55 THRU 64.9=5)(65 THRU HI=6)
COMPUTE         AGEGP2=AGEGP
RECODE          AGEGP2(1,2,3=1)(4=2)(5=3)(6=4)
VALUE LABLES    AGEGP(1)<25(2)25-35(3)35-45(4)45-55(5)55-65(6)>65/
VALUE LABLES    AGEGP2(1)<45(2)45-55(3)55-65(4)>65/
COMPUTE         WAYEX=VAR122
RECODE          WAYEX(0=0)(1 THRU 10=1)(10 THRU 20=2)(20 THRU 40=3)
                (40 THRU HI=4)
VALUE LABLES    WAYEX(0)NONE(1)<10(2)10-20(3)20-40(4)>40/
COMPUTE         OUTEX=VAR123
RECODE          OUTEX(1 THRU 5=1)(5 THRU 10=2)(10 THRU 15=3)(15 THRU HI=4)
VALUE LABLES    OUTEX(0)NO(1)<5(2)5-10(3)10-15(4)>15HOURS/
COMPUTE         MILKD=VAR128
RECODE          MILKD(1 THRU 2=1)(2 THRU HI=2)
VALUE LABLES    MILKD(0)NONE(1)<2(2)>2 MILK/
COMPUTE         MILK=VAR404
RECODE          MILK(1 THRU 2=1)(2 THRU 4=2)(4 THRU 6=3)(6 THRU HI=4)
VALUE LABLES    MILK(0)NONE(1)<2(2)2-4(3)4-6(4)>6/
RECODE          VAR129(1 THRU 3=1)(3 THRU 6=2)(6 THRU HI=3)
VALUE LABLES    VAR129(0)NO(1)<3(2)3-6(3)>6COFFCUPS/
RECODE          VAR405(1 THRU 3=1)(3 THRU 6=2)(6 THRU HI=3)
VALUE LABLES    VAR405(0)NONE(1)<3(2)3-6(3)>6 CUPS/
RECODE          VAR130(1 THRU 3=1)(3 THRU 6=2)(6 THRU HI=3)
VALUE LABLES    VAR130(0)NO(1)<3(2)3-6(3)>6/
RECODE          VAR406(1 THRU 3=1)(3 THRU 6=2)(6 THRU HI=3)
VALUE LABLES    VAR406(0)NONE(1)<3(2)3-6(3)>6/
COMPUTE         ALCOHOL=0.0
COMPUTE         ALCOHOL=VAR133+(2*VAR134)+(6*VAR135)
RECODE          ALCOHOL(0=1)(1 THRU 5=2)(6 THRU 10=3)(11.0 THRU 20=4)
                (21.0 THRU HI=5)
VALUE LABLES    ALCOHOL(1)NONE(2)1-5(3)6-10(4)11-20(5)>21/
COMPUTE         ALCOHOL2=0.0
COMPUTE         ALCOHOL2=VAR409+(2*VAR410)+(6*VAR411)
RECODE          ALCOHOL2(0.0=0)(1 THRU 5=1)(6 THRU 10=2)(11 THRU 20=3)
                (21 THRU HI=4)
VALUE LABLES    ALCOHOL2(0)NONE(1)1-5(2)6-10(3)11-20(4)>21/
COMMENT          ANGINA PECTORIS
COMPUTE         ANGINA=1
IF              (VAR213 EQ 2)ANGINA=0
IF              (VAR213 EQ 1 AND VAR214 EQ 1 OR VAR215 EQ 1 AND ((VAR216 EQ 1)
                 OR (VAR216 EQ 2)) AND VAR217 EQ 1 AND
                VAR218 EQ 1 AND VAR219 EQ 1)ANGINA=1
IF              (VAR213 EQ 1 AND VAR214 EQ 1 AND VAR215 EQ 1 AND ((VAR216 EQ 1)
                 OR (VAR216 EQ 2)) AND VAR217 EQ 1 AND
                VAR218 EQ 1 AND VAR219 EQ 1)ANGINA=2
VALUE LABLES    ANGINA(0)NO ANGINA(1)ANGINA1(2)ANGINA2/
COMMENT         SMOKING SECTION
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COMPUTE         SMOK=0
IF              ((VAR137 EQ 2) OR (VAR137 EQ 0) AND VAR145 EQ 2 AND VAR203 EQ 1
                 AND VAR211 EQ 1)SMOK=1
IF              (VAR137 EQ 2 AND VAR145 EQ 1)SMOK=2
IF              (VAR203 EQ 2)SMOK=3
IF              (VAR211 EQ 2)SMOK=4
IF              (VAR137 EQ 1)SMOK=5
IF              (VAR203 EQ 3)SMOK=6
IF              (VAR211 EQ 3)SMOK=7
IF              (VAR137 EQ 1 AND VAR203 EQ 3)SMOK=8
IF              (VAR137 EQ 1 AND VAR211 EQ 3)SMOK=9
IF              (VAR137 EQ 1 AND VAR203 EQ 3 AND VAR210 EQ 3)SMOK=10
IF              (VAR203 EQ 3 AND VAR211 EQ 3)SMOK=11
VALUE LABLES    SMOK(1)NON SMOK(2)EX CIGT SMOEX (3)PIP SMO(4)EX CIG SMO
                (5)CUR CIGT SMO(6)PIP SMOK(7)CIGAR SMO(8)CIGT&PIP SMO
                (9)CIGT&CIGAR SMOK(10)3 TYPE SMOK(11)PIPE+CIGAR SMOK/
COMPUTE         SMOK2=SMOK
RECODE          SMOK2(0,1=1)(2,3,4,=2)(5=3)(6,7,8,9,10,11=4)
VALUE LABLES    SMOK2(1)NON SMOKER(2)EX SMOKER(3)CIGT SMOK(4)CIGT+OTH SM/
COMPUTE         SMO=0
IF              ((VAR412 EQ 2) AND (VAR420 EQ 2) AND (VAR424 EQ 1))SMO=1
IF              (VAR412 EQ 1)SMO=2
IF              (VAR420 EQ 1)SMO=3
IF              (VAR424 EQ 3)SMO=4
IF              ((VAR420 EQ 3) OR (VAR424 EQ 2))SMO=5
VALUE LABLES    SMO(1)NON SMO(2)CIGT SMOK(3)PIP SMOK(4)CIGAR SMO(5)NON SMO/
COMPUTE         SMO2=SMO
RECODE          SMO2(0,1,5=1)(2,3,4=2)
VALUE LABLES    SMO2(1)NON SMOKERS(2)SMOKERS/
COMMENT         CIGARETTE NUMBER
RECODE          VAR141(98,99=0)/VAR142(98,99=0)/VAR204(98,99=0)/VAR212(98,99=0)
COMPUTE         NCIGS1=VAR141+(VAR142*30/7)+(VAR204*30/7)+(VAR212/7)
RECODE          NCIGS1(1 THRU 5.5=1)(5.5 THRU 14.99=2)(15 THRU 24.99=3)
                (25 THRU 34.99=4)(35 THRU 97=5)
VALUE LABLES    NCIGS1(0)NON SMOK(1)<5.5(2)5.5-15(3)15-25(4)25-35(5)>35/
RECODE          VAR416(98,99=0)/VAR417(98,99=0)/VAR421(98,99=0)/VAR425(98,99=0)
COMPUTE         NCIG2=VAR416+(VAR417*30/7)+(VAR421*30/7)+(VAR425/7)
RECODE          NCIG2(0=0)(1 THRU 5.5=1)(5.5 THRU 14.9=2)(15 THRU 24.9=3)
                (25 THRU 34.9=4)(35.0 THRU 97=5)
VALUE LABLES    NCIG2(0)NON SMO(1)<5.5(2)5.5-15(3)15-25(4)25-35(5)>35/
COMMENT         LAST SMOKING
RECODE          VAR418(1 THRU 60=1)(60 THRU 120=2)(120 THRU 180=3)(180 THRU 240=4
                )(240 THRU 300=5)(300 THRU 360=6)(360 THRU 720=7)(720 THRU HI=8)
VALUE LABLES    VAR418(0)NONE(1)1H(2)2H(3)3H(4)4H(5)5H(6)6H(7)6-12H(8)>12HOURS/
RECODE          VAR423(1 THRU 60=1)(60 THRU 120=2)(120 THRU 180=3)(180 THRU 240=4
                )(240 THRU 300=5)(300 THRU 360=6)(360 THRU 720=7)(720 THRU HI=8)
VALUE LABLES    VAR423(0)NONE(1)1H(2)2H(3)3H(4)4H(5)5H(6)6H(7)6-12H(8)>12HOURS/
COMPUTE         SMAGE=VAR144
RECODE          SMAGE(LO THRU 15=1)(15 THRU 25=2)(25 THRU 35=3)(35
                 THRU 45=4)(45 THRU 55=5)(55 THRU 65=6)(65 THRU HI=7)
VALUE LABLES    SMAGE(1)<15(2)15-25(3)25-35(4)35-45(5)45-55(6)55-65(7)>65/
COMPUTE         PSAGE=VAR148
RECODE          PSAGE(LO THRU 15=1)(15 THRU 25=2)(25 THRU 35=3)(35
                 THRU 45=4)(45 THRU 55=5)(55 THRU 65=6)(65 THRU HI=7)
VALUE LABLES    PSAGE(1)<15(2)15-25(3)25-35(4)35-45(5)45-55(6)55-65(7)>65YEARS/
COMPUTE         SAGEP=VAR206
RECODE          SAGEP(LO THRU 15=1)(15 THRU 25=2)(25 THRU 35=3)(35
                 THRU 45=4)(45 THRU 55=5)(55 THRU 65=6)(65 THRU HI=7)
VALUE LABLES    SAGEP(1)<15(2)15-(3)25-(4)35-(5)45-(6)55-(7)>65YEARS/
COMMENT         INT CLAUDICATION
COMPUTE         CLAUDI=2
IF              (VAR221 EQ 2)CLAUDI=0
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IF              (VAR221 EQ 1 AND VAR222 EQ 2 AND VAR223 EQ 1 AND VAR224 EQ 1 AND
                 VAR225 EQ 2 AND VAR226 EQ 2 AND ((VAR227 EQ 1) OR (VAR227 EQ 2)) AND
                 AND VAR228 EQ 2)CLAUDI=1
VALUE LABLES    CLAUDI(0)NO (1)MILD (2) SEVER/
COMPUTE         FDTHAGE=VAR238
RECODE          FDTHAGE(LO THRU 45=1)(45 THRU 55=2)(55 THRU 65=3)(65 THRU HI=4)
VALUE LABLES    FDTHAGE(1)<45(2)45-55(3)55-65(4)>65YEARS/
COMPUTE         MDTHAGE=VAR240
RECODE          MDTHAGE(LO THRU 45=1)(45 THRU 55=2)(55 THRU 65=3)(65 THRU HI=4)
VALUE LABLES    MDTHAGE(1)<45(2)45-55(3)55-65(4)>65YEARS/
COMPUTE         FDTHCAUSE=VAR237
RECODE          FDTHCAUSE(1,2=1)(4,10=2)(3,15,21=3)
VALUE LABLES    FDTHCAUSE(1)CANCERS(2)NERVOUS S(3)HEART D/
COMPUTE         MDTHCAUSE=VAR239
RECODE          MDTHCAUSE(1,2=1)(4,10=2)(3,15,21=3)
VALUE LABLES    MDTHCAUSE(1)CANCERS(2)NERVOUS S(3)HEART D/
COMMENT         PHYSICO-MENTAL STATUS
COMPUTE         PHYSMENT=4
IF              (VAR245 EQ 9 AND VAR246 EQ 9 AND VAR247 EQ 9 AND VAR248 EQ 9)
                PHYSMENT=0
IF              (VAR245 EQ 1 AND VAR246 EQ 1 AND VAR247 EQ 1 AND VAR248 EQ 1)
                PHYSMENT=1
IF              (VAR245 EQ 1 AND VAR246 EQ 1 AND ((VAR247 EQ 1) OR (VAR247 EQ 2))
                 AND ((VAR248 EQ 2) OR (VAR248 EQ 3)))PHYSMENT=2
IF              (((VAR245 EQ 2) OR (VAR245 EQ 1)) AND VAR246 EQ 2 AND VAR247 EQ
                 2 AND VAR248 EQ 2)PHYSMENT=3
IF              (VAR245 EQ 4 AND ((VAR246 EQ 1) OR (VAR246 EQ 2) OR (VAR246 EQ
                3) OR (VAR246 EQ 4)) AND ((VAR247 EQ 1) OR (VAR247 EQ 2) OR
                 (VAR247 EQ 3) OR (VAR247 EQ 4)) AND ((VAR248 EQ 1) OR
                (VAR248 EQ 2) OR (VAR248 EQ 3) OR (VAR248 EQ 4)))PHYSMENT=5
IF              (VAR245 EQ 4 AND VAR246 EQ 4 AND VAR247 EQ 3 AND VAR248 EQ 3)
                PHYSMENT=6
IF              (VAR245 EQ 4 AND VAR246 EQ 4 AND VAR247 EQ 4 AND ((VAR248 EQ 1)
                 OR (VAR248 EQ 2) OR (VAR248 EQ 3) OR (VAR248 EQ 4)))PHYSMENT=7
IF              (VAR245 EQ 4 AND VAR246 EQ 4 AND VAR247 EQ 4 AND VAR248 EQ 4)
                PHYSMENT=8
VALUE LABLES    PHYSMENT(0) NO RESPONSE(1)HIGHEST STRESS(2) STAGE2(3)STAGE3(4)
                STAGE4(5)STAGE5(6)STAGE6(7)LOWEST STRESS(8)NO STRESS/
COMPUTE         PHYSMENT2=PHYSMENT
RECODE          PHYSMENT2(1,2=1)(3,4=2)(5,6,7=3)(8=4)
VALUE LABLES    PHYSMENT2(0)NO RESPONSE(1)SEVER STRAIN(2)MODERATE(3)MILD
                STRAIN(4)NO STRAIN/
COMMENT         HIGHT&WEIGHT
COMPUTE         HIGHT=(VAR303)/.394
COMPUTE         WEIGHT=(VAR304)/2.2
COMPUTE         QI=0.0
IF              (HIGHT GT 0.0 AND WEIGHT GT 0.0)
                QI=WEIGHT*100.0/(HIGHT*HIGHT)
COMPUTE         QI=100.0*QI
RECODE          VAR450(LO THRU 150=1)(150 THRU 160=2)(160 THRU 170=3)
                (170 THRU 180=4)(180 THRU HI=5)
VALUE LABLES    VAR450(1)<150(2)150-160(3)160-170(4)>170CM/
RECODE          VAR451(LO THRU 50.0=1)(50.0 THRU 60.0=2)(60.0 THRU 70.0=3)
                (70.0 THRU 80.0=4)(80.0 THRU 90.0=5)(90.0 THRU HI=6)
VALUE LABLES    VAR451(1)<50KG(2)50-60(3)60-70(4)70-80(5)>90KGM/
COMPUTE         PULSE=VAR305
RECODE          PULSE(LO THRU 60=1)(60 THRU 80=2)(80 THRU 100=3)(100 THRU 120=4)
                (120 THRU HI=5)
VALUE LABLES    PULSE(1)<60(2)60-80(3)80-100(4)100-120(5)>120/
COMMENT         SYSTOLIC BLOOD PRESSURE
COMPUTE         SBPGP=VAR307
RECODE          SBPGP(LO THRU 119=1)(120 THRU 129=2)(130 THRU 139=3)(140 THRU 149
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                =4)(150 THRU 159=5)(160 THRU HI=6)
VALUE LABLES    SBPGP(1)<119(2)120-129(3)130-139(4)140-149(5)150-159(6)>160/
COMMENT         DIASTOLIC BP(VAR308)
COMPUTE         DBPGP=VAR308
RECODE          DBPGP(LO THRU 79=1)(80 THRU 89=2)(90 THRU 109=3)(110 THRU 129=4)
                (130 THRU HI=5)
VALUE LABLES    DBPGP(1)<79(2)80-89(3)90-109(4)110-129(5)>130/
COMMENT         RESPIRATORY FUNCTIONS
COMPUTE         FEVOL1=VAR309
RECODE          FEVOL1(LO THRU 1.99=1)(1.00 THRU 2.00=2)
                (2.01 THRU 3.00=3)(3.01 THRU HI=4)
VALUE LABLES    FEVOL1(1)<1(2)1-2(3)2-3(4)>3 LIT/
COMPUTE         FVCAP=VAR310
RECODE          FVCAP(LO THRU 2.0=1)(2.0 THRU 3.0=2)(3.0 THRU HI=3)
VALUE LABLES    FVCAP(1)<2(2)2-3(3)>3 LIT/
COMMENT          CARDIAC ENZYMES
COMPUTE         SGOTE=VAR324
RECODE          SGOTE(LO THRU 0042=1)(0043 THRU 0100=2)(0100 THRU HI=3)
VALUE LABLES    SGOTE(1)12-42 NORMAL(2)43-100(3)>100 UL/
COMPUTE          TOTALCK=VAR325
RECODE          TOTALCK(LO THRU 160=1)(160 THRU 1999=2)(2000 THRU HI=3)
VALUE LABLES     TOTALCK(1)<160 NORMAL(2)160-1999(3)>2000/
COMPUTE         LDENZYME=VAR326
RECODE          LDENZYME(LO THRU 229=1)(230 THRU 525=2)(526 THRU 999=3)
                (1000 THRU HI=4)
VALUE LABLES    LDENZYME(1)<229(2)230-525NORMAL(3)526-999(4)>1000 UL/
COMPUTE         CHOLEST=VAR327
RECODE          CHOLEST(LO THRU 3=1)(3.1 THRU 7.5=2)(7.6 THRU HI=3)
VALUE LABLES    CHOLEST(1)<3(2)3.1-7.5 NORMAL(3)>7.6/
COMPUTE         TRIGLY=VAR328
RECODE          TRIGLY(LO THRU 0.5=1)(0.6 THRU 1.8=2)(1.9 THRU 2.9=3)
                (3 THRU 3.9=4)(4 THRU HI=5)
VALUE LABLES    TRIGLY(1)<0.5(2)0.6-1.8 NORMAL(3)1.9-2.9(4)3-3.9(5)>4 MMOLL/
COMPUTE         BLSUGAR=VAR329
RECODE          BLSUGAR(LO THRU 2.7=0)(2.7 THRU 9.9=1)(10 THRU HI=2)
VALUE LABLES    BLSUGAR(0)<2.7(1)2.7-9.9 NORMAL (2)>10-HIGH MMOL L/
COMMENT         CARBOXYHAEMOGLOBIN
COMPUTE         COHB=VAR330
RECODE          COHB(LO THRU 1.9=1)(2 THRU 3.9=2)(4 THRU 5.9=3)(6 THRU HI=4)
VALUE LABLES    COHB(1)<1.9(2)2-3.9(3)4-5.9(4)>6%SAT/
COMPUTE         COHB2=VAR452
RECODE          COHB2(LO THRU 1.9=1)(2.0 THRU 3.9=2)(4.0 THRU 5.9=3)
                (6.0 THRU HI=4)
VALUE LABLES    COHB2(1)<1.9(2)2-3.9(3)4-5.9(4)>6%SAT/
RECODE          VAR453(LO THRU 9.5=1)(10 THRU 19.9=2)(20 THRU HI=3)
VALUE LABLES    VAR453(1)<10(2)10-20(3)>20PPM/
COMPUTE         HB=VAR331
RECODE          HB(LO THRU 11.9=1)(12 THRU 13.9=2)(14 THRU 15.9=3)
                (16 THRU 17.9=4)(18 THRU HI=5)
VALUE LABLES    HB(1)<11.9(2)12-13.9(3)14-15.9(4)16-17.9(5)>18/
COMPUTE         OXYHB=VAR332
RECODE          OXYHB(LO THRU 49=1)(50 THRU 70=2)(71 THRU 90=3)(90 THRU HI=4)
VALUE LABLES    OXYHB(1)<50(2)50-69(3)70-89(4)90%SAT/
COMPUTE         HD=0
IF              (VAR434 EQ 1)HD=HD+1
IF              (VAR435 EQ 1)HD=HD+1
IF              (VAR436 EQ 2)HD=HD+1
IF              (VAR437 EQ 2)HD=HD+1
IF              (VAR438 EQ 1)HD=HD+1
IF              (VAR439 EQ 2)HD=HD+1
IF              (VAR440 EQ 1)HD=HD+1
IF              (VAR441 EQ 1)HD=HD+1
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IF              (VAR442 EQ 1)HD=HD+1
IF              (VAR443 EQ 2)HD=HD+1
IF              (VAR444 EQ 1)HD=HD+1
IF              (VAR445 EQ 1)HD=HD+1
IF              (VAR446 EQ 1)HD=HD+1
IF              (VAR447 EQ 1)HD=HD+1
IF              (VAR448 EQ 1)HD=HD+1
IF              (VAR449 EQ 2)HD=HD+1
RECODE          HD(1 THRU 3=1)(9 THRU 13=2)(14 THRU HI=3)
VALUE LABLES    HD(1)POOR KNW(2)GOOD KNW(3)EX KNW/
CROSSTABS       TABLES=NCIG2 BY COHB/
                TRIGLY BY VAR323/
                BLSUGAR BY VAR323/
                HB BY VAR323/
                VAR453 BY COHB2/
                VAR453 BY SMO2/
                NCIG2 BY COHB2/
                SMOK2 BY SBPGP/
                SMOK2 BY DBPGP/
                SMOK2 BY COHB/
                SMOK2 BY OXYHB/
                NCIGS1 BY COHB/
                VAR323 BY COHB/
                ANGINA BY COHB/
                SMOK2 BY PHYSMENT/
                VAR323 BY PHYSMENT/
                AGEGP2 BY VAR323/
                VAR255 BY COHB/
                OXYHB BY COHB/
                CLAUDI BY COHB/
                VAR323 BY VAR237/
                VAR323 BY VAR239/
                VAR252 BY VAR232/
                ANGINA BY VAR323/
                VAR252 BY ANGINA /
                VAR323 BY VAR313/
                VAR323 BY VAR314/
                VAR323 BY VAR315/
                VAR323 BY VAR317/
                VAR323 BY VAR318/
                VAR323 BY VAR319/
                VAR323 BY VAR320/
                VAR323 BY VAR321/
                VAR323 BY VAR322/
                VAR323 BY SGOTE/
                VAR323 BY TOTALCK/
                VAR323 BY LDENZYME/
                VAR323 BY BLSUGAR/
                VAR323 BY VAR335/
                VAR144 BY NCIGS1/
                VAR103 BY SMOK2/
                VAR111 BY SMOK2/
                VAR103 BY VAR104/
                AGEGP BY SMOK2/
                SMOK2 BY VAR116/
                SMOK2 BY VAR117/
                SMOK2 BY VAR118/
                SMOK2 BY VAR120/
                AGEGP2 BY VAR111/
                VAR121 BY VAR320/
                WAYEX BY VAR323/
                OUTEX BY VAR323/
```

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                VAR124 BY VAR323/
                VAR125 BY VAR323/
                VAR126 BY VAR323/
                MILKD BY VAR323/
                VAR129 BY VAR323/
                VAR130 BY VAR323/
                ALCOHOL BY VAR323/
                ALCOHOL BY ANGINA/
                SMOK2 BY ANGINA/
                VAR327 BY SMOK2/
                SMOK2 BY VAR208/
                SMOK2 BY VAR209/
                VAR208 BY VAR327/
                SMOK2 BY FEVOL1/
                SMOK2 BY FVCAP/
                NCIGS1 BY FEVOL1/
                NCIGS1 BY FVCAP/
                SMOK2 BY CHOLEST/
                MILKD BY MILK/
                VAR129 BY VAR405/
                VAR130 BY VAR406/
                ALCOHOL BY ALCOHOL2/
                SMOK2 BY SMO2/
                NCIGS1 BY NCIG2/
                VAR323 BY VAR426/
                VAR323 BY VAR427/
                SEPGP BY VAR230 /
                VAR230 BY VAR428/
                VAR229 BY VAR329/
                VAR433 BY SMO2/
                COHB BY COHB2/
                VAR453 BY COHB2/
                SMO2 BY COHB2/
                SMO2 BY VAR453/
                HD BY VAR103/
                HD BY VAR323/
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OPTIONS         3,4,5,7,9
READ INPUT DATA
FINISH

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INSTRUCTIONS FOR PORTABLE CARBON MONOXIDE MONITOR

APPLICATION

The monitor is designed for the measurement of ambient levels of carbon monoxide in the atmosphere and acts as a personal safety indicator sounding an audible alarm when the carbon monoxide level rises above the pre-determined alarm level.

The actual alarm level can be displayed to the user by depressing the alarm level momentary action switch.

When the switch is released, the instrument reverts to displaying the actual ambient carbon monoxide concentration.

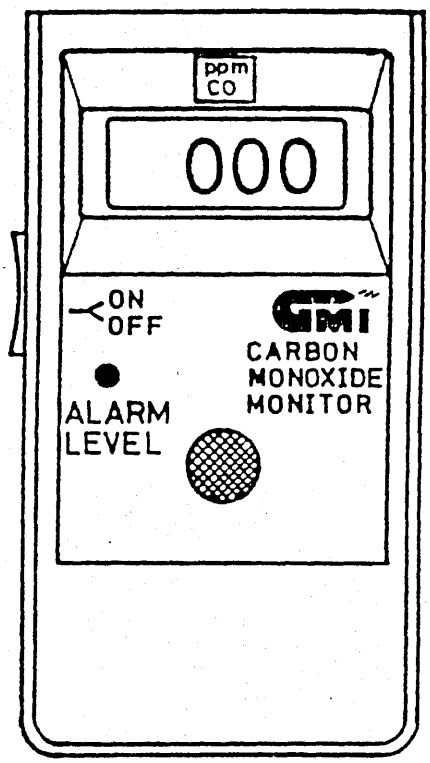


Fig. 1

Normal operation is by means of:-

(a) Allowing the ambient gas concentration to diffuse into the sensing element via the gas inlet aperture on the top of the instrument case.

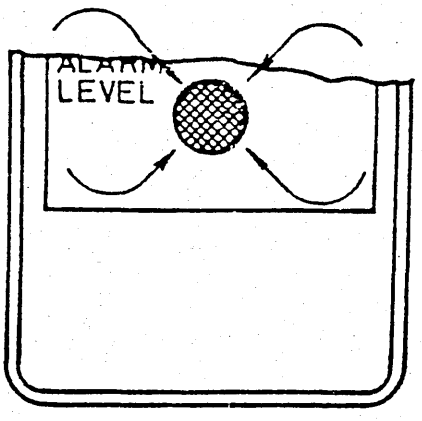


Fig. 2

(b) By connecting the calibration mask accessory to the inlet aperture, and a piece of gas sampling line and aspirator bulb to the inlet and outlet nozzles on the calibration mask and aspirating the gas sample from either a calibration bottle to allow instrument calibration, or from a remote location in order to measure the gas concentration at that location.

The above components are connected together as shown in Fig. 3.

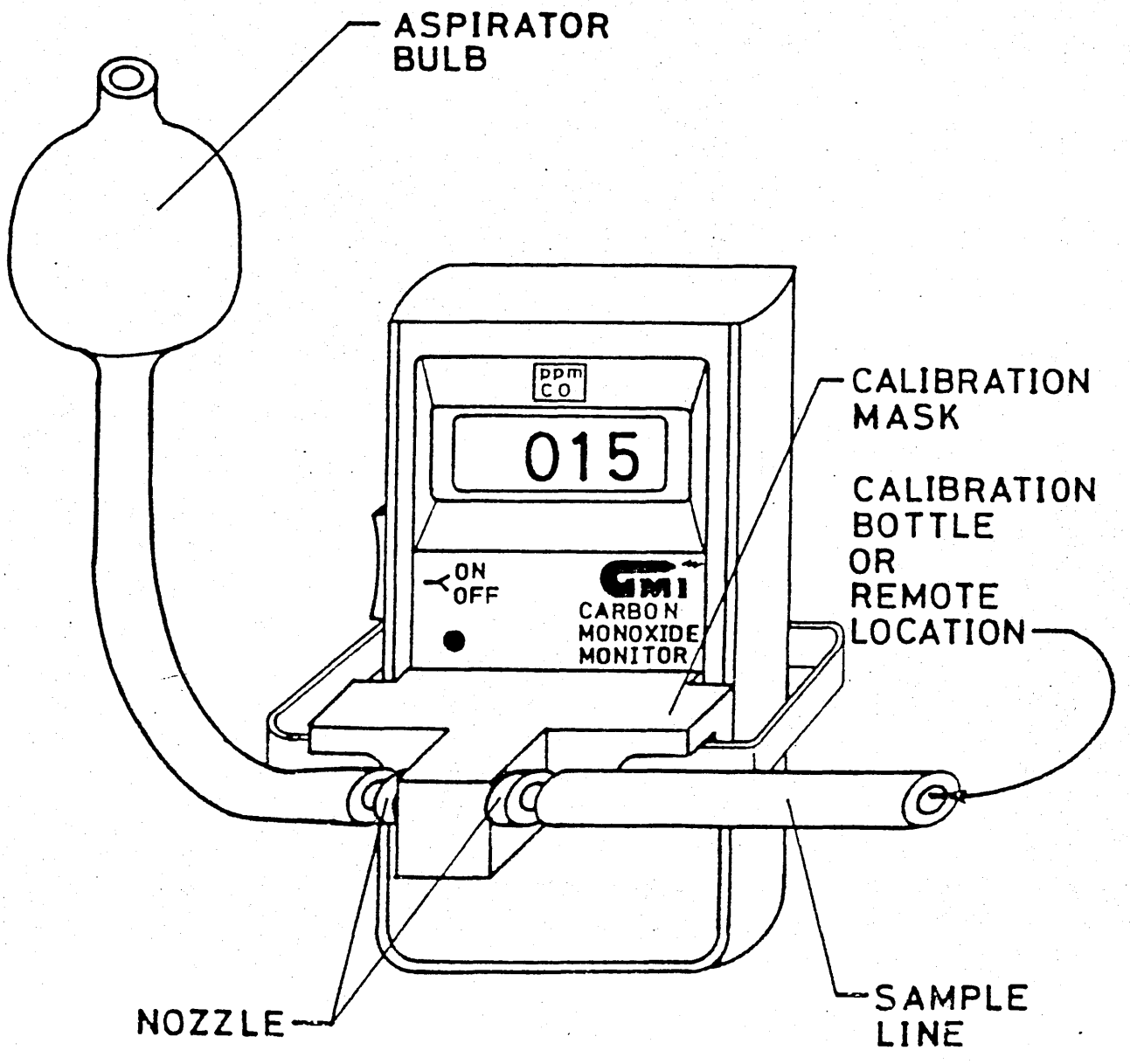

ASPIRATOR BULB
ppm CO
015
ON
OFF
GMI
CARBON MONOXIDE MONITOR
CALIBRATION MASK
CALIBRATION BOTTLE OR REMOTE LOCATION
NOZZLE
SAMPLE LINE

Fig.3

OPERATION

The operation and function of the top cover controls and indicators is as follows:

(1) Carbon monoxide concentration of gas sample in ppm CO.

(2) Battery Level Indicator - Display indicates approximately 1 hour of operating time left.

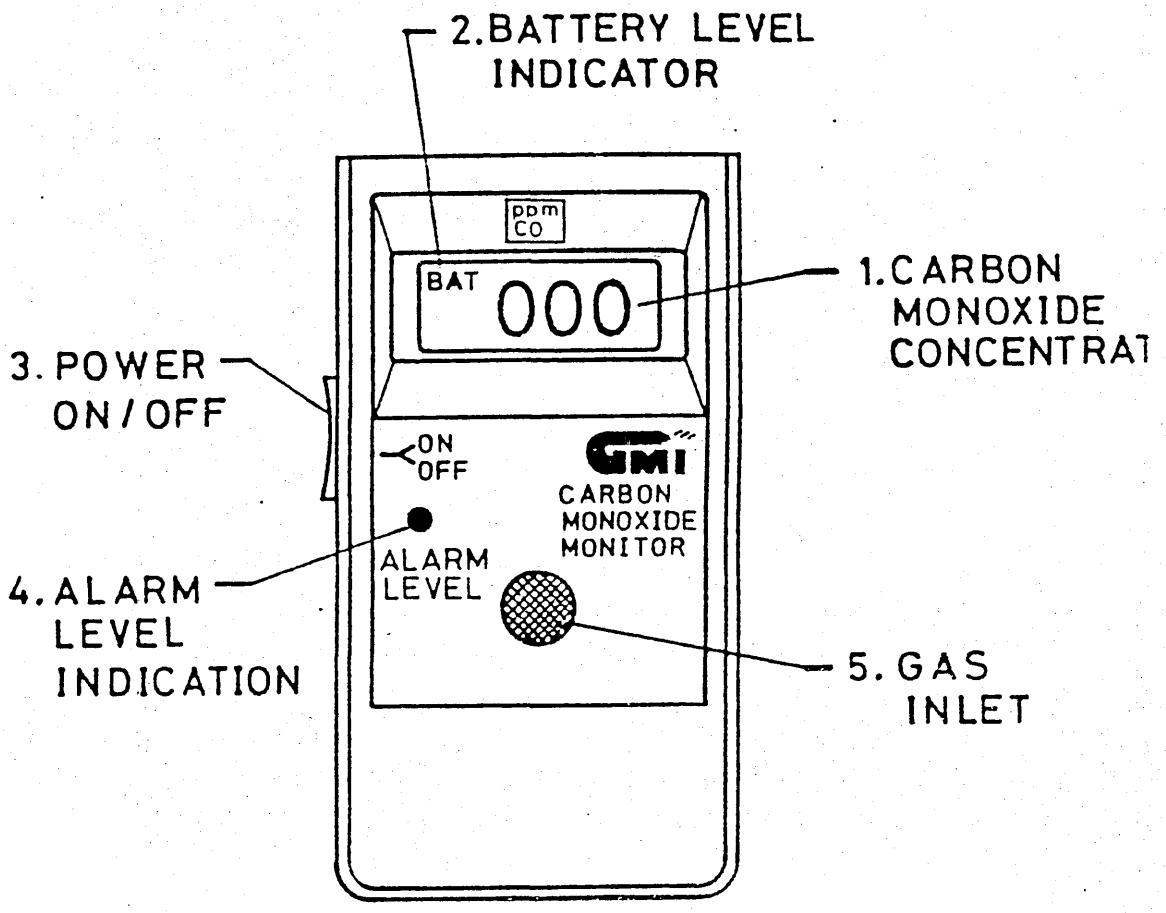


Fig.4

(3) Power On/Off - Off switches off all instrument functions while leaving sensor in the standby condition. (A 20 sec. delay can be required for stabilising the sensor after switching from the standby condition to the on condition).

On - All instrument functions on.

(4) Alarm - Depressing this switch allows the alarm level to be displayed instead of the carbon monoxide concentration.

Releasing the switch reverts the display back to the measured carbon monoxide concentration.

(5) Gas Inlet - Aperture to allow gas sample to be measured by the sensor. Do not restrict the gas diffusing through this aperture, unless instrument calibration or remote sampling is being performed while utilizing the calibration mask.

The operation and function of the end panel controls is as follows:-

(1) Charging Socket - inserting mains charger jack plug and applying mains power allows recharging of the instrument internal battery when the ON/OFF switch is in the OFF position.

(2) Charging Indicator - indicator is illuminated when the battery is being recharged.

(3) Zero Adjust - used during calibration procedure to set the zero level of the instrument.

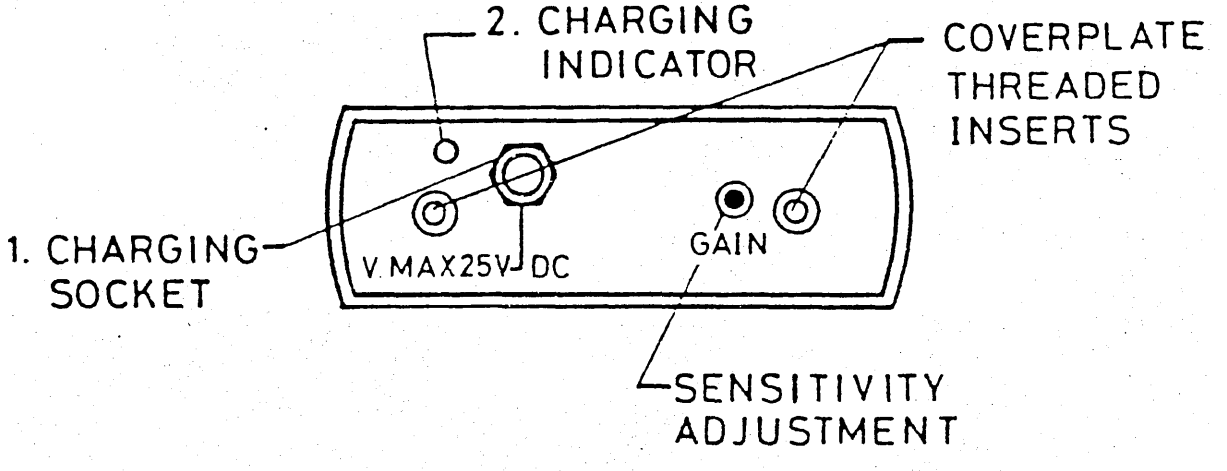


Fig. 5

SPECIFICATION

Input Voltage:-	12v to 25v dc
Input Current	20mA (Available from accompanying mains battery charger.)
Operating Temperature	- 5°C to 40°C
Rel. Humidity	90% RH non-condensing
Dimensions:-	Width 80mm Depth 35mm Length 150mm Weight 290gm

Battery Charger

Input Voltage	240v 50Hz
Operating Temperature	0°C to 40°C
Output Voltage	12v to 25v dc.
Output Current	50mA

TYPICAL PERFORMANCE CHARACTERISTICS:-

Measurement range	0 to 200 ppm CO	
Transient response (to reach 90% of final value) (diffusion mode)		
Air ⟶ Air + carbon monoxide		40 secs
Air + carbon monoxide ⟶ Air		40 secs
In the aspirated mode both responses		40 secs.
Accuracy	200 ppm	± 5% of Reading (± 2 ppm for Readings ≤ 40 pp
Linearity (in the range 0 to 200 ppm)		± 2% of Scale
Temperature coeff of zero		± 0.4 ppm/°C
Temperature coeff of span		± 0.4% of Reading/°C
Repeatability:-		± 2% of Full Scale
Accuracy of alarm set point:-		± 2 ppm
Hysterises:-		10 ppm
Audible Alarm Output		(70 dBA typical)
Variation of alarm set point:-	5 ppm to 200 ppm	(factory set to 50
Sensor operating life		1 to 2 years
Continuous operation on fully charged batteries:- (without alarm activated)		15 hours
Time to recharge batteries		12 hours
Operating time left at 'Lo BAT' indication		1 hour

CALIBRATION

CAUTION: Calibration should only be carried out in a designated non hazardous area and using known gas concentrations.

The instrument is fitted with an internal span control and external zero control. To calibrate aspirate the zero gas (Air with 0 ppm CO) and when the display has stabilized to its lowest point, adjust the zero control to give a reading of 0 ppm.

When this has been completed, aspirate calibration gas into the cell, and again when it has stabilized to its highest reading adjust the sensitivity control to give the reading as indicated on the calibration cylinder.

To preset the alarm set point, depress the alarm level pushbutton on the top of the case. This will display the present value of the alarm set point. To adjust, vary the alarm adjustment potentiometer. While the pushbutton is depressed the level can be monitored.

As soon as the button is released, the readout will revert to the actual gas concentration present.

NOTE 1 Access to the gain control and the alarm level setting control is only possible by unscrewing the special case fasteners, and removing the instrument top cover.

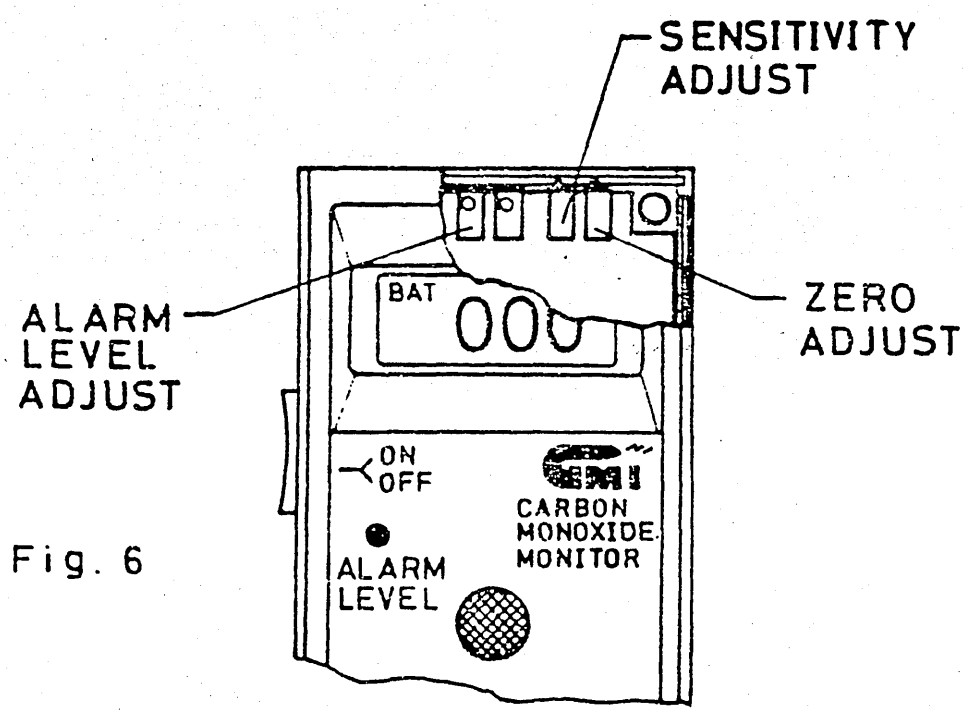


Fig. 6

BATTERY RECHARGING

The instrument internal battery is recharged by inserting the charging jack plug into the instrument jack socket, and connecting the charger mains plug to a mains outlet.

When the instrument is in the charging mode, the charging L.E.D. will be illuminated.

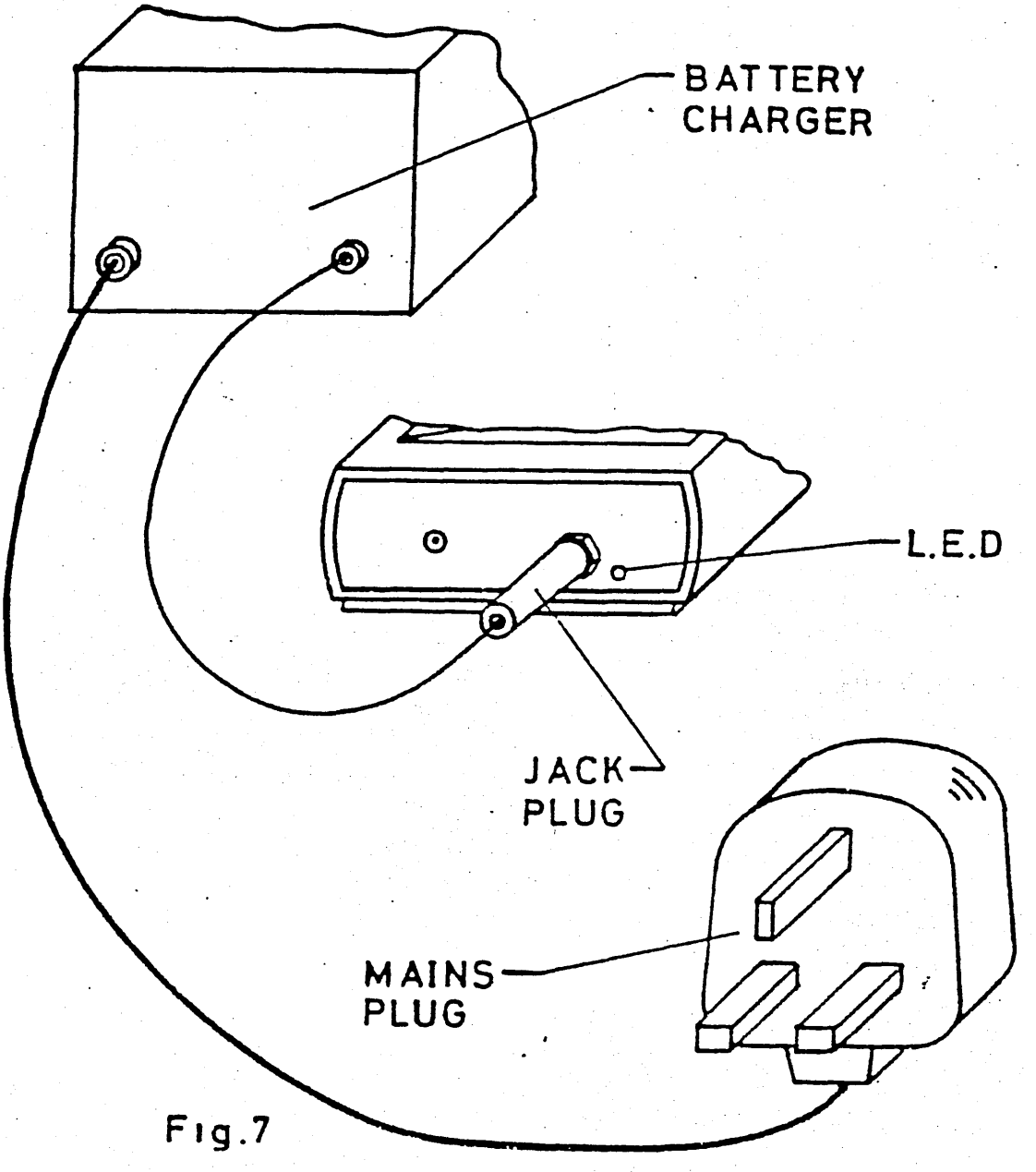


Fig.7

DESCRIPTION

The instrument is based on the measurement of parts per million (ppm) carbon monoxide using a sensitive, selective electrochemical fuel cell and is portable and capable of operating for up to 15 hours when completely re-charged.

a. The instrument is powered from a rechargeable Nickel Cadmium battery, which is accessed externally by means of a jack socket allowing for the use of a mains battery charger. A charging L.E.D. adjacent to the jack socket is illuminated when the battery is being recharged. The battery can only be recharged when the On/Off switch is in the Off position.

b. The sensor itself is housed in the main compartment of the case, and is configured in such a way as to allow satisfactory operation both in the natural diffusion mode, and the aspirated mode with only the addition of an external, push fit, calibration mask. The sensor is a three electrode sensor and as such can not be connected in circuit at all times. When not in circuit the working and reference electrodes are connected together, maintaining the bias potential at 0V. Thus when the instrument is switched on again, the concentration reading is available after a short period of time (20 s) has been allowed for the sensor to stabilize.

c. The control electronics accept a voltage output across the cell load resistor which is dependant on the carbon monoxide concentration, and amplifies this signal to a suitable level to interface with the DVM module which gives a digital readout of the signal level at its input. Compensation for variations of cell output with temperature is also included.

d. The adjustable alarm set point allows the user to preset his desired alarm level, and when the concentration of carbon monoxide exceeds the limit the audible alarm is triggered. When the concentration falls to a desired level below the alarm level the alarm ceases.

SENSOR

The Carbon Monoxide sensor is of the three electrode fuel cell typ designed to be maintenance free and stable over long periods of time There are no user replaceable parts other than the complete senso: module.

The sensors use the capillary diffusion barrier technology which ha: been proven over many years, and this results in low temperatur coefficient, a direct response to concentration (% volume), an relatively unaffected by pressure. The use of electrodes based or fuel cell technology gives a high reserve of activity which makes fo: long term stability.

Carbon monoxide diffusing to the sensing electrode (anode) react: according to the equation

$$CO + H_2O + 2e = CO_2 + 2H^+$$

At the counter electrode (cathode) the reaction is

$$\tfrac{1}{2}O_2 + 2H^+ = H_2O + 2e$$

the oxygen requirement being automatically supplied from the ambien air by controlled diffusion.

Figures 8 and 9 respectively show the electrical and mechanica details of the sensor.

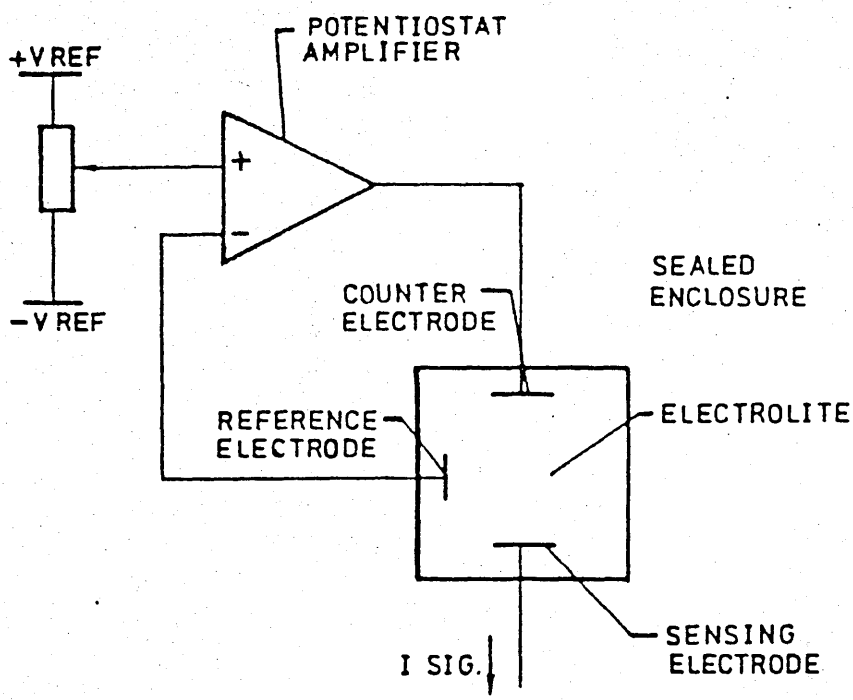

+VREF
POTENTIOSTAT AMPLIFIER
+
−
−VREF
SEALED ENCLOSURE
COUNTER ELECTRODE
REFERENCE ELECTRODE
ELECTROLITE
SENSING ELECTRODE
I SIG.

Fig.8

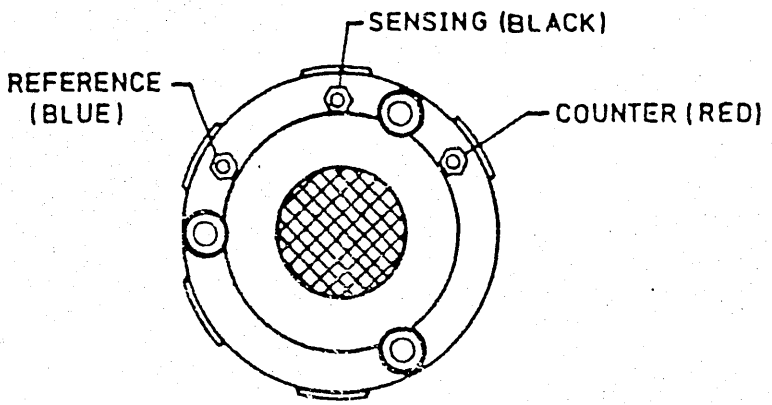

SENSING (BLACK)
REFERENCE (BLUE)
COUNTER (RED)

Fig.9

INSTRUCTIONS FOR EXHALED HYDROGEN MONITOR

APPLICATION

The monitor is designed for the measurement and recording of exhaled hydrogen in the diagnosis of malabsorption of carbohydrates which can assist in identifying such conditions as:-

1. Lactose intolerance
2. Assessment of gut transit time
3. Excessive bacterial growth in the small intestine.

The Monitor provides a range of hydrogen measurement of 0 to 250 ppm - which can be read out directly on the digital display and if required can be permanently recorded using the GMI recorder unit CR001.

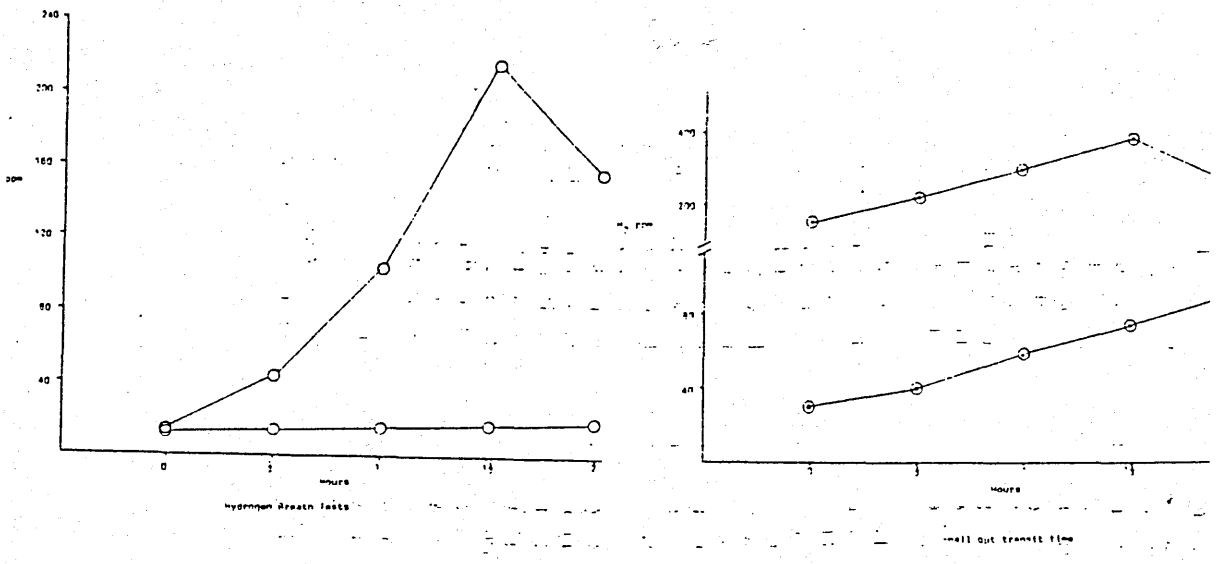


Fig. 1.

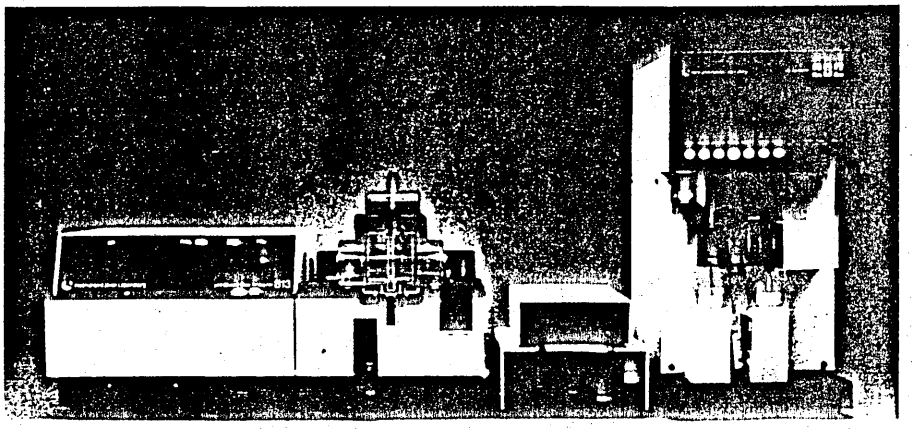
EXHYMINS

TYPICAL PERFORMANCE CHARACTERISTICS

Range :	0 - 250 ppm Hydrogen
Transient Response (to reach 90% of final value)	
Air ---- Air + Hydrogen	7 - 10 secs.
Air + Hydrogen ---- Air	12 - 18 secs.
Sensitivity	2ppm
Accuracy	+/- 2% (+/- 1 ppm for readings less than 50 ppm)
Temperature coeff of zero (1)	2ppm/°C.
Temperature coeff. of sensitivity (1)	0.5 ppm/°C
Repeatability	+/- 1 ppm
Cross sensitivity :	
Oxygen	Step change by 5% by volume = approx 2 ppm
Carbon Dioxide	Step change by 5% by volume = approx 2 ppm
Carbon Monoxide	Step change by 50 ppm = approx 7 ppm
Methane	Step change by 1% by volume = approx 1ppm
Sensor operating life	12 months
Battery back-up	20 hours

Note 1: The instrument should ideally be operated under constant ambient temperature conditions, and does not respond to rapid temperature changes. Any temperature changes should be kept to a minimum in both magnitude and rate of change.

EXHYMINS



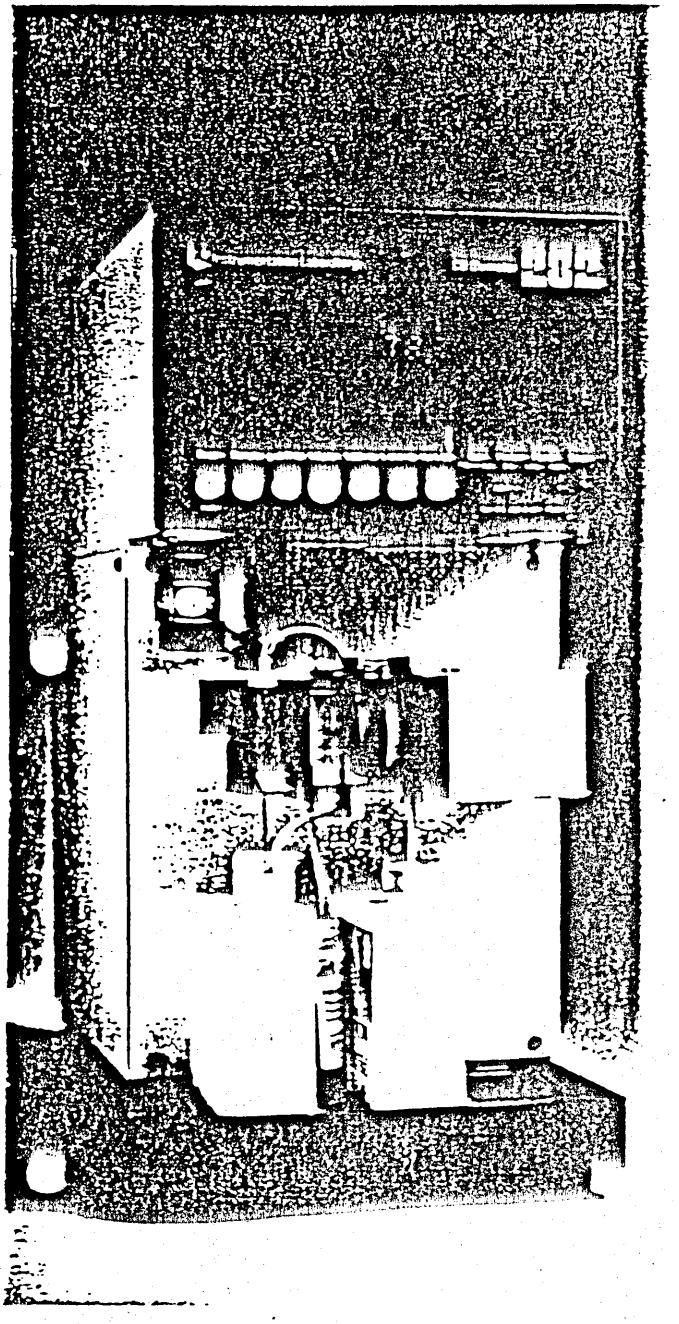
Combines with other IL instrumentation for comprehensive oxygen availability and consumption analyses.

The new IL282 CO-Oximeter can be interfaced with our IL813, 713 or 513 Blood Gas Analyzer and IL318 Printer to form a complete blood gas laboratory. This prints out a 15-parameter profile of acid/base, respiratory function and oxygen statuses. Another natural application of this system would be use in conjunction with the IL601 Cardiac Output System for oxygen consumption studies.

Phone toll-free for a demonstration of the IL282: 800-225-1481.

Instrumentation Laboratory Inc.
Biomedical Division
113 Hartwell Avenue
Lexington, MA 02173

An IL advance, to advance your patient care.



The IL282 CO-Oximeter is a dedicated spectrophotometer that analyzes whole blood specimens for 4 hemoglobin parameters, plus oxygen content. Total hemoglobin (THb), % O_2Hb, % COHb and % MetHb are determined utilizing 4 wavelengths of light and 16 extinction coefficients locked in memory. O_2 content is calculated from THb and measured % O_2Hb. A built-in microprocessor helps control the numerous automated functions.

Easy operation.

To make an analysis, you simply: present the sample, push the SAMPLE button, and watch for THb (g/dl) to come up on the LED display. Other values are then displayed at a push of their buttons. Presentation-to-display time is only 62 seconds. Data remains available for ½ hour or until the next sample is run. Zeroing, aspiration, hemolyzing, measurement, calculation and cleaning are all automatic. If the IL318 Printer or hospital computer is connected to the IL282, it receives all data automatically.
Routine sample volume is 350 µl. Microsamples of 175 µl may be presented from Natelson capillary tubes. A syringe-loading option is available.

More useful, accurate data.

This new IL instrument provides the comprehensive hemoglobin analysis that—combined with blood gas analysis—is increasingly being stressed for complete patient care, to improve assessment of oxygen saturation and transport. It is much easier and faster than manual methods.

Determines 4 hemoglobin parameters not just one or two.

The inclusion of COHb and MetHb optimizes accuracy in spectrophotometrically determining O_2Hb. And these additional "knowns" are valuable by themselves. For example, COHb is a key factor in diagnosing fire victims, smokers or workers exposed to carbon monoxide (pollution). MetHb may be significant in hemoglobinopathies and drug-related conditions.
The IL282's calculated oxygen content is highly accurate and correlates closely with Van Slyke values. It is based on the total hemoglobin and the actual percent oxyhemoglobin.

Automated stability and other features upgrade precision.

A hollow-cathode lamp in the IL282 emits unchanging wavelengths. Dual-beam optics compensate for lamp intensity variations.
The instrument cleans itself and updates its blank absorbance values after each sample, and every ½ hour when in Standby mode. It is compatible with routine blood quality control procedures. THb values correlate with those of cyanmethemoglobin reference methodology.
Audible/visual warning systems alert the operator to faults. Corrective action normally involves a simple cuvette maintenance procedure.

BEATING HEART DISEASE

The Health Education Council

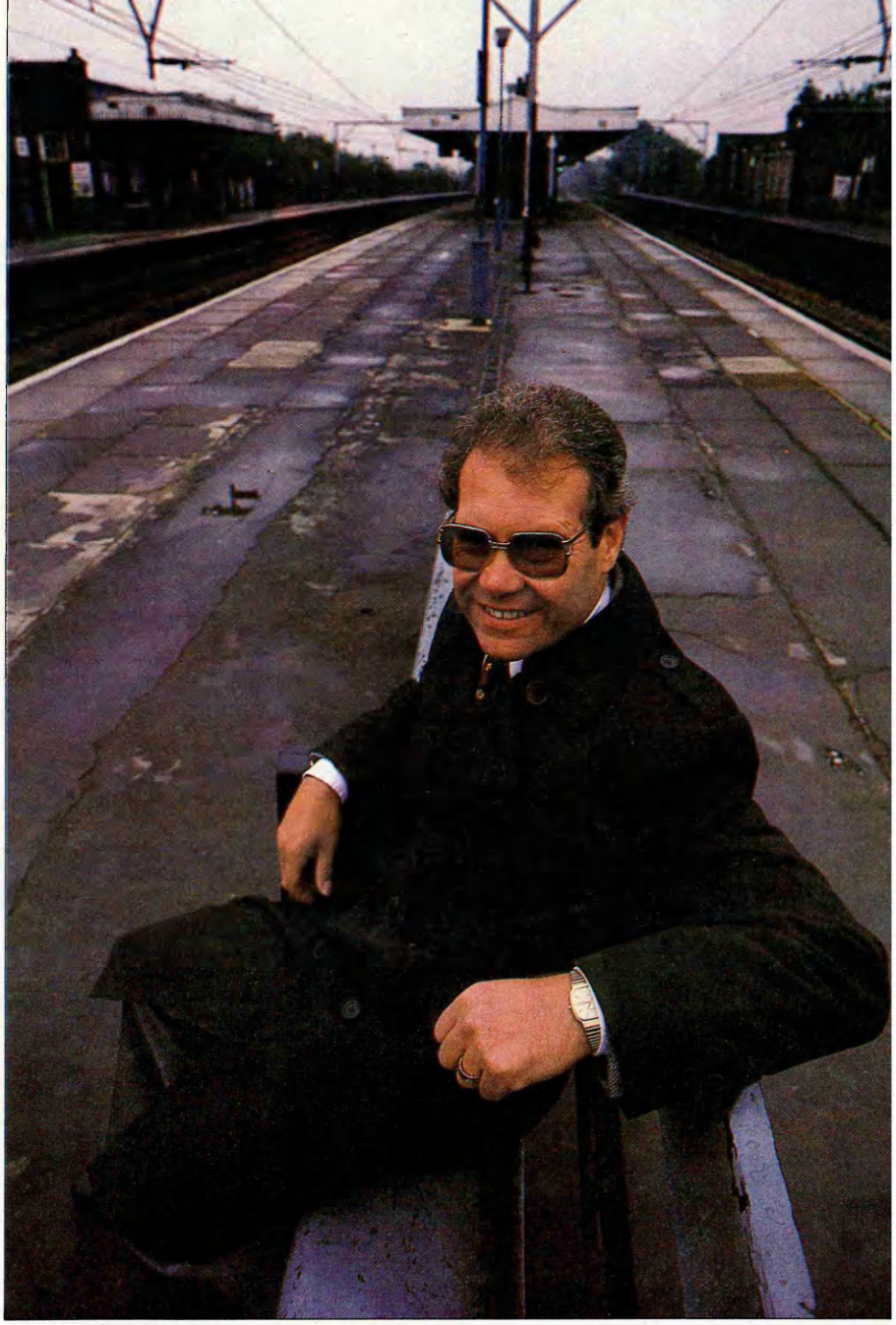
BEATING HEART DISEASE

This booklet is published jointly by the Health Education Council and the Scottish Health Education Group in association with the BBC Radio series 'Action makes the heart grow stronger'.

Designed by Information Design Workshop.

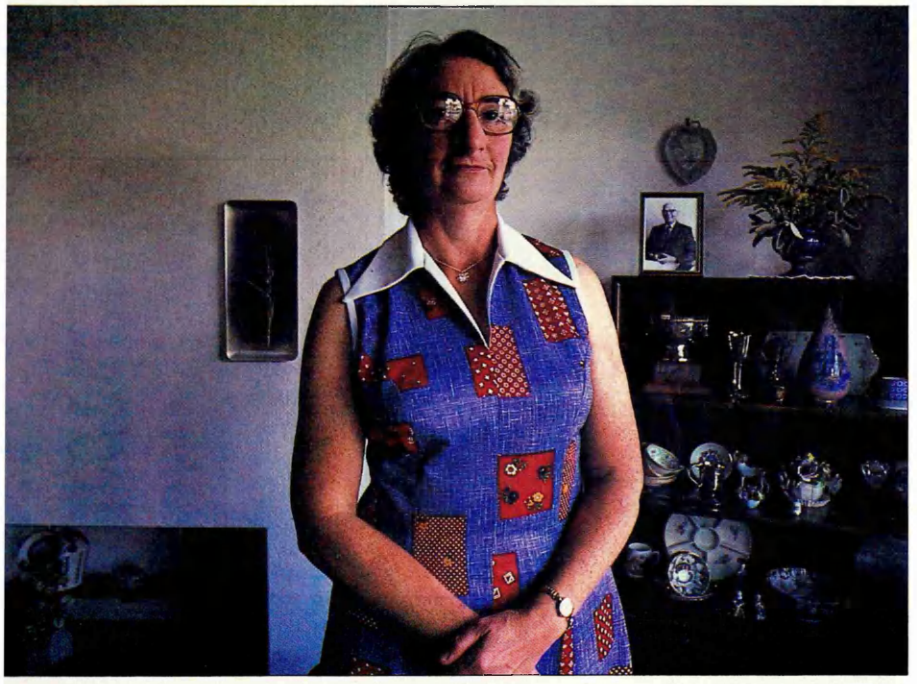
Photographs by Tony Sleep

"I used to be able to walk miles but now I only have to go down to the local shops and back and I'm out of breath. And if I'm going to do anything energetic – even if it's just the washing – I have to put a tablet under my tongue to stop the pain coming on. But usually I'm alright. I just have to be careful not to overdo it."



"I thought I'd be the last person to have any heart trouble. I've always loved sport. I was a champion cross country runner when I was younger and since then I've kept myself fit playing football fo local teams. But when I started running a busy pub I began to put on a lot of weight. I was up to 16 stone when I had my heart attack

HEART DISEASE



It could mean a heart attack out of the blue. Or years of suffering from angina. It might even mean a cardiac arrest and sudden death.

It's easy to think it couldn't happen to you. But heart disease is the biggest single killer in Britain today. It is estimated that four out of every ten men will suffer from some form of heart disease by the time they are 65 years old. And in the last twenty years there has been an increase of heart disease in younger women.

But you can do something to reduce the risk. And you'll not only be fighting heart disease. You'll soon start to feel fitter than ever before.

Take a look at this booklet and find out what you can do. It will tell you:

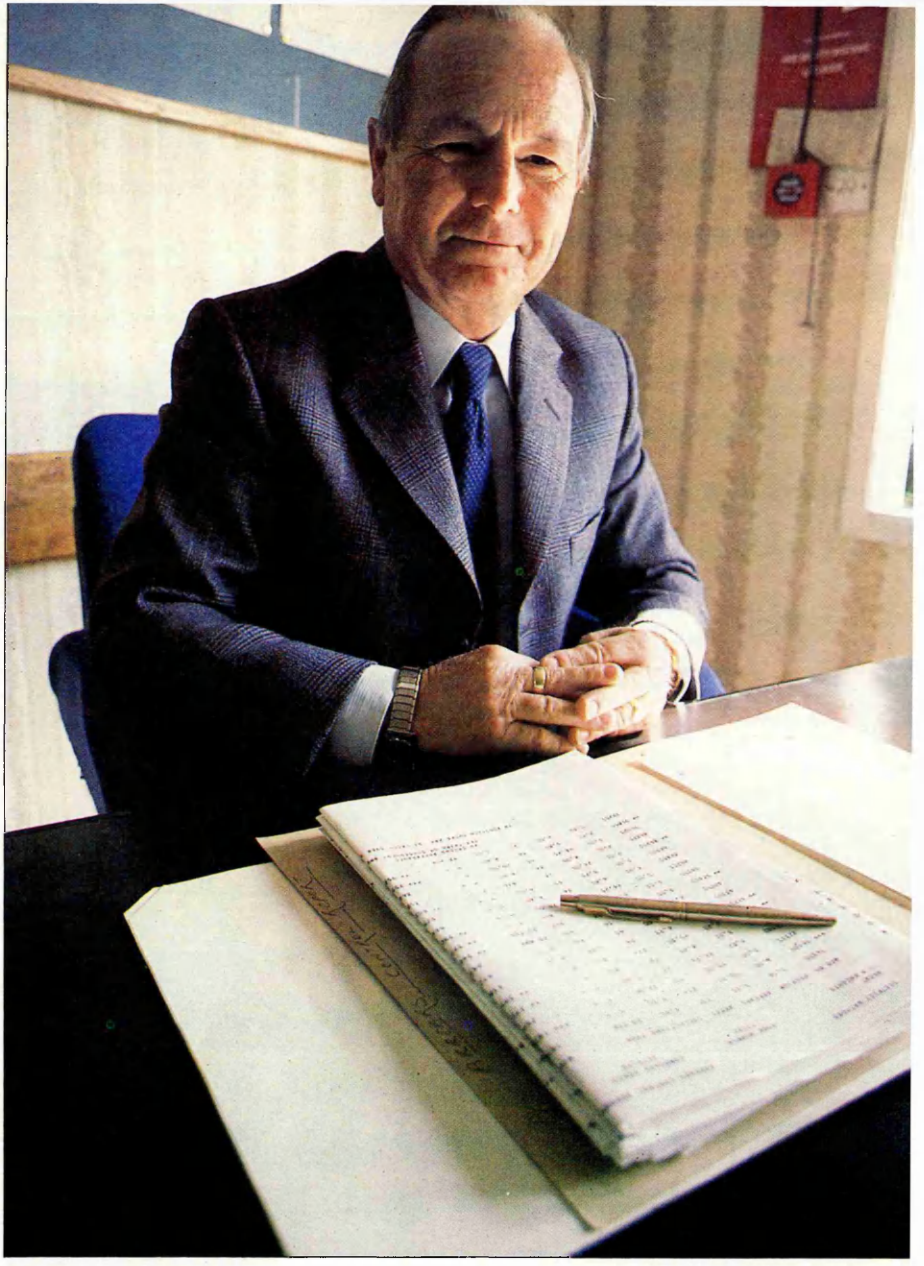
- ☐ what heart disease is
- ☐ why some people get it
- ☐ how to help yourself avoid it
- ☐ what to do for someone who's got it (including advice on emergency first aid).

Don't wait until it's too late. Do something now. Take some good advice and help yourself to a healthier heart.

"It was so sudden. I was just getting a meal ready and my husband went out to the garden to pick some vegetables. The next thing I knew he had collapsed and died. Nobody expected it – he always seemed so fit and healthy."

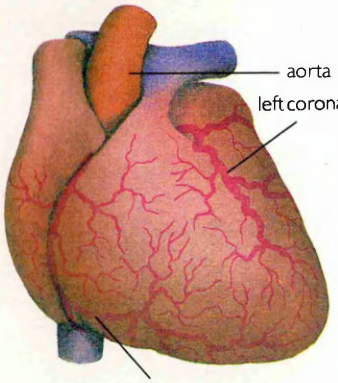
WHAT IS HEART DISEASE?

There are many different types of heart disease. This booklet is about the type that is most common and most tragic. The type that causes heart attacks. It's called **Coronary Heart Disease (CHD).**



How the heart works

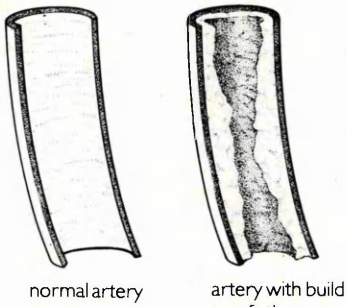
The heart is a blood-filled bag of muscle about the size of a fist. The muscle contracts about 70 times a minute to pump blood around the body. Like any other active tissue, the heart muscle needs a good supply of oxygen. It gets this from the bloodstream. Its supply is taken not from the blood which is being pumped through the heart but from separate little arteries, the **coronary** arteries. These arteries branch off from the main artery (the **aorta**) and then divide into smaller branches which fan out all over the surface of the heart.



‘Since I found out I had angina about 3 years ago, I've had to make a lot of changes in my life. For instance, we've had to move our bedroom and bathroom downstairs so I don't have to go upstairs so much. And I'm on a very strict diet. But by taking angina tablets and by following my doctor's advice I find I can cop with a fairly demanding job. You just have to be sensible about it. Nowadays if I feel tired at work I stop for a while or go home earl instead of pushing myself to the limit like I used to. In fact, these days I'm more relaxed than I've ever been! ’

What goes wrong?

Over a period of many years, starting in early adult life, the walls of these arteries can gradually become ‘furred up’ with a fatty deposit called atheroma. If the atheroma gets too thick and the arteries too narrow, the blood supply to the heart muscle can be restricted or even blocked. This is coronary heart disease. It has two main forms: angina and heart attack.



normal artery

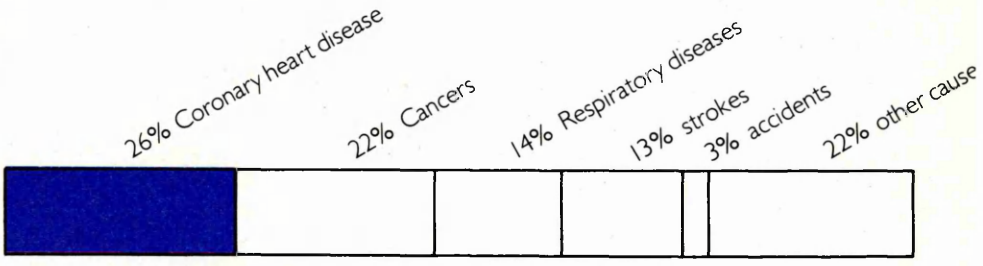
artery with build up of atheroma

What is angina?

If the narrowing of the coronary arteries is very gradual then the first signs of trouble may only be noticed when the heart is having to work harder than usual. Because the blood flow through the arteries is restricted, anything whicl makes the heart pump a little faster thar usual – even if it is only walking up step – starves the heart of blood. The classic situation is when a person is exerting hi or herself, or getting excited or angry. This brings on a heavy cramp-like pain

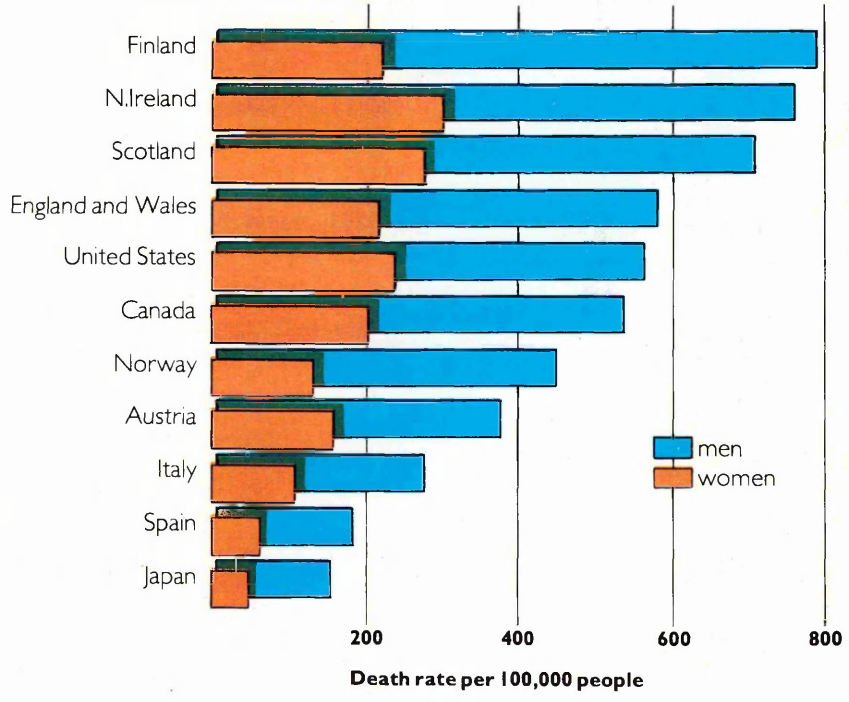
WHO GETS HEART DISEASE?

Far too many of us. As you can see from this diagram, heart disease causes more than a quarter of all deaths in this country.



Causes of death in the U.K. (as a percentage of all deaths)

Based on government figures



Death rates from coronary heart disease in different countries (35-74 year olds)

Based on figures from the World Health Organisation

You can see from this chart how people from some parts of the world are more likely to suffer from heart disease than others. Notice how England, Scotland, Wales and Northern Ireland are near the top of the league for deaths from heart disease. Now compare us with Japan.

And even within Britain there are some areas where heart disease is more common. In general, Scotland, Wales and Northern Ireland are worse off than England.

The experts are trying to find the reasons why the death rate from heart disease varies so much from place to place. There is no simple answer. As we will see later it is likely to be the result of many different factors.

ıcross the chest, like a huge weight. ɔometimes the pain spreads to the neck, houlder, arm or jaw. It usually fades ıway after a few minutes rest.

This kind of pain is known as angina. t is not the same as a heart attack ɔecause it is usually relieved by a short ɔeriod of rest or relaxation. Somebody vho suffers from angina does have a ıigher than average risk of having a heart ıttack. But angina itself can be relieved ɔr controlled by drugs and, in severe ːases, surgery.

What is a heart attack?

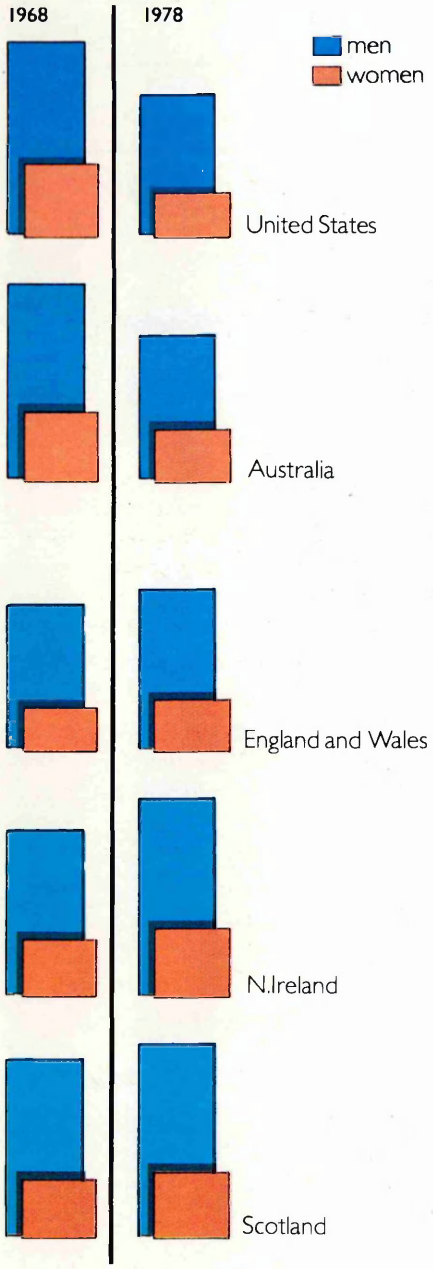
A heart attack happens when there is a ɜudden and severe blockage in one of the ːoronary arteries so that the blood supply to part of the heart muscle is cut ɔff. The blockage is usually caused by a blood clot forming in an artery already damaged by fatty atheroma. This is ːalled a **coronary thrombosis** (or sometimes a **coronary**).

The part of the heart muscle affected is severely damaged causing the pain that is the most common symptom of a heart attack. This pain is usually, but not always, a crushing vice-like ache felt in the chest. It can spread to the neck, jaw or arm. It does not usually ease off for several hours. As well as being in pain the person usually feels faint, giddy or sick.

In some cases the effects of the blockage can be so severe that the heart stops beating altogether. This is called a **cardiac arrest.** Unless the heart starts beating within a few minutes the person will die. Unfortunately about 50% of all fatal heart attack victims die within half an hour, often before medical help arrives. Some of these people could be saved by prompt emergency first aid.

WHAT CAUSES HEART DISEASE?

And things aren't getting any better. Have a look at how heart disease death rates have been changing around the world over the past few years. As you can see, we are way behind the improvement shown in other countries, for example, the United States.



Death rates from coronary heart disease in men and women aged between 40 and 69 years

Based on World Health Organisation figures

Researchers have studied entire populations to try to discover what it is in our everyday lives that increases the risk of heart attack and angina. Their results show that there is no single cause of heart disease. Instead, there seem to be several different factors which together may tip the balance against your heart.

Of course, some people are more likely to suffer from heart disease than others. For example, the tendency to die young from heart disease can run in the family. And certainly the older you are, the greater the risk of having a heart attack. The narrowing of the arteries which can lead to angina and heart attacks tends to get worse as you get older, although it may start quite young.

In general, men are more at risk from heart disease than women. A man in his late forties is five times more likely to die of heart disease than a woman of the same age. But after the menopause, a woman loses the protective effect of her hormones and her chances of suffering from heart disease are almost equal to a man's. And in the last twenty years there has been an increase of heart disease in women in their 30s and 40s.

Even though your age, your sex and your family history are all beyond your control, you can still do a lot to keep your risk of heart disease as low as possible.

Risky living

Start by looking at the way you live your life. When people think of taking risks they think of going hang-gliding, or driving too fast, or even crossing a busy road. But you might be taking a life-and-death risk every day without even realising it. By smoking, eating too much of the wrong food and not getting enough exercise and relaxation you could be gambling with the health of your heart.

Smoking

Cigarette smoking can double your risk of dying from a heart attack. And if you smoke heavily you are even more likely to die young from heart disease. For example, a man aged 50 who smokes more than 20 cigarettes a day is four times more likely to suffer from heart disease than a non-smoker of the same age. And women are just as much at risk as men. The risk for a woman who smokes is especially high if she is over 35 and is on the pill.

How does smoking affect the heart? The nicotine in tobacco smoke increases the pulse rate and raises the blood pressure. The carbon monoxide content of cigarette smoke cuts down the amount of oxygen in the blood. So your heart is having to work harder but getting less oxygen. Smoking also accelerates the 'furring up' of the coronary arteries.

What you can do

The answer is to give up smoking. As soon as you stop smoking you will start to reduce your risk of a heart attack. You could be almost back to a non-smoker's risk level within a few years.

Don't think that by cutting down or switching to a low-tar brand of cigarette you will be protecting yourself against heart disease. You might actually be inhaling even more carbon monoxide by taking longer and more frequent puffs. And smoking cigars or a pipe will only be safer than cigarettes if you don't inhale. Giving up smoking altogether isn't easy. But it's certainly worth it. To help you make your decision, think about what you gain by stopping. It's not just that you improve your chances of avoiding heart trouble. You gain in other ways too:

☐ You'll be healthier and breathe more easily – for example, when you climb stairs or run for a bus. And you'll help that smoker's cough

☐ You'll suffer fewer colds and infections

☐ You'll smell fresher. No more bad breath, stained fingers or teeth

☐ You'll save money.

If you're not sure you can do it, think of this: there are eight million people in this country who have stopped smoking. So you can do it too.

Deciding to stop smoking, and really wanting to do it, is half the battle. If you've made up your mind you're going

stop, the chances are you'll manage it. ut because it's not an easy thing to do, 's worth working out just how you're ›ing to do it.

First of all, decide on the day when ›u're going to stop and then stick to it. ake it a big day. Do something special. lan a treat at the end of the day as a ward.

From now on, try to change your ›utine so you can avoid the situations in hich you know you'll want to smoke. ›u won't have to avoid these danger tuations for ever, just for the first few eeks.

Don't think about tomorrow. Take ›ch day as it comes. If you do feel a rong urge to smoke, then do something se instead. Don't just sit there wanting cigarette and worrying about it, ecause you'll probably end up smoking. ind activities to *replace* smoking – ›ings to distract yourself, things to do ith your hands, things to help you ›lax. Do something active. Play some ›ort. Go for walks or for a run. Get thers to join you so you can share the leasure.

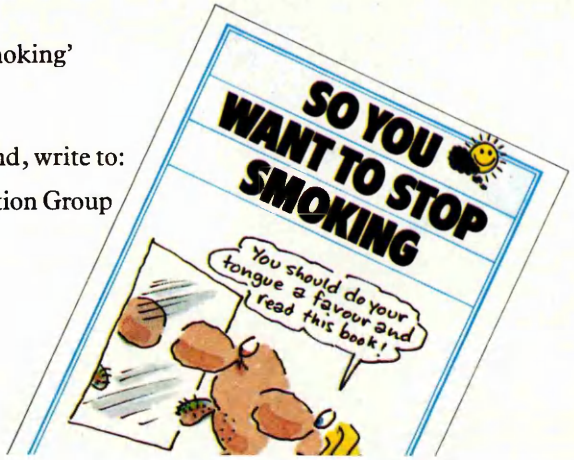
After your first enthusiasm has worn ff, *keep at it*. Remind yourself of your ›asons for giving up and what you're ›aining now you've done it. Don't be ›mpted to smoke 'just the odd cigarette.' ›ne odd one easily leads to two, or three .. You really have to work at breaking ›e habit for good if you want to give ›ur heart a chance.

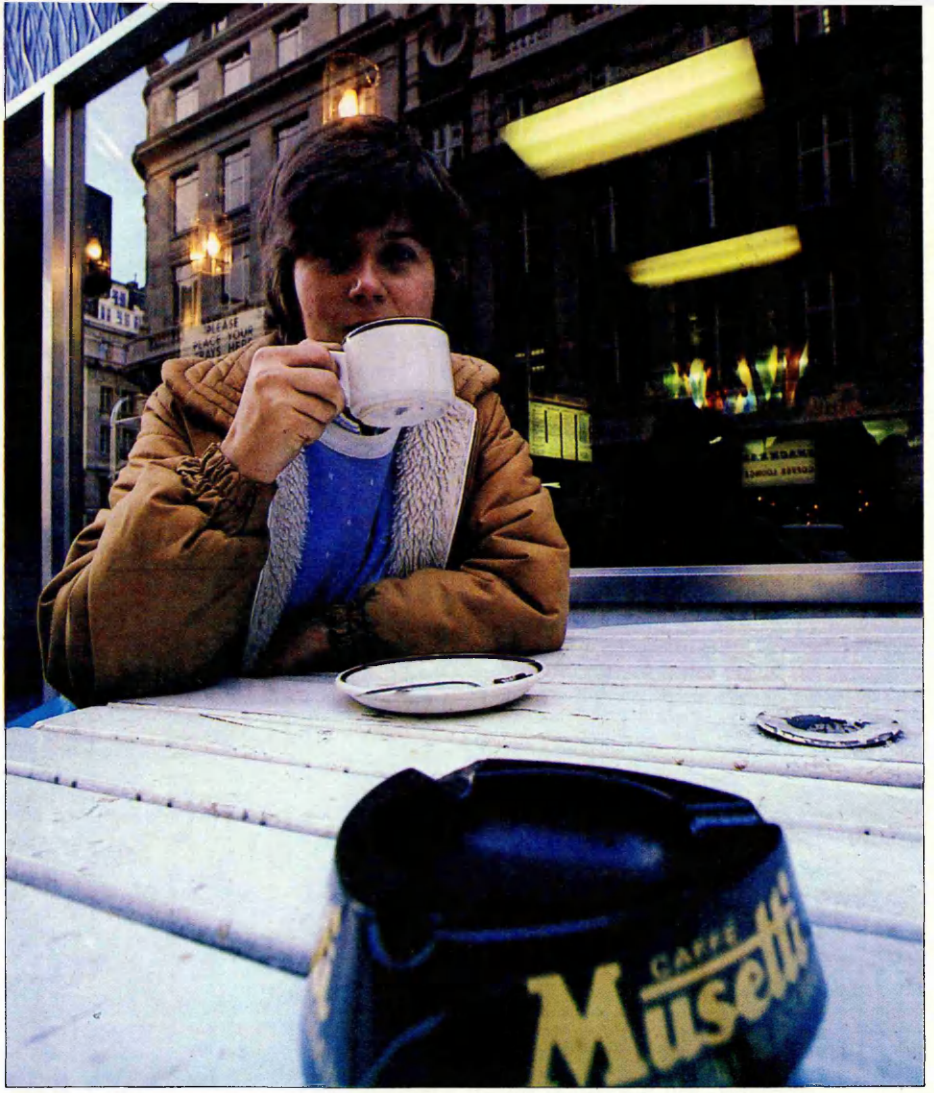
you would like a step-by-step guide to iving up smoking you can obtain a copy f *So you want to stop smoking* from your ›cal health education unit or by writing ›:

›o you want to stop smoking'
O Box 410
.ondon SE99 6YE

r, if you live in Scotland, write to:

cottish Health Education Group
Woodburn House
›anaan Lane
›dinburgh EH10 4SG.





"I started smoking when I was 19. A friend offered me a cigarette and I really enjoyed it. After that I was hooked and I was soon getting through about 30 a day. I did try to give up because I knew it wasn't good for me. But it was impossible – I liked smoking too much to stop. Then a couple of years ago I had to run an urgent errand at work. It meant I literally had to run about half a mile and back. By the time I got back to the office I could hardly breathe. It really brought home to me the damage smoking must be doing to me. If I felt like that at 23 what was I going to feel like at 40? I decided to give up smoking there and then. Of course, it was difficult to break the habit at first but I'm glad I kept at it this time. I have had the occasional cigarette when it's been offered to me but I don't enjoy it anymore. In fact, I feel sick if I smoke. It's a relief to have kicked the habit. And it's not just my health that's improved – my bank balance feels a lot better too! It's nice to have the 'cigarette money' to spend on other things."

Diet

Another way to beat heart disease is to watch what you eat. Your everyday choice of food can be a key factor in the health of your heart. And if you are choosing food for the family, you've got their hearts in your hands too. So beware of eating the wrong foods and putting on too much weight.

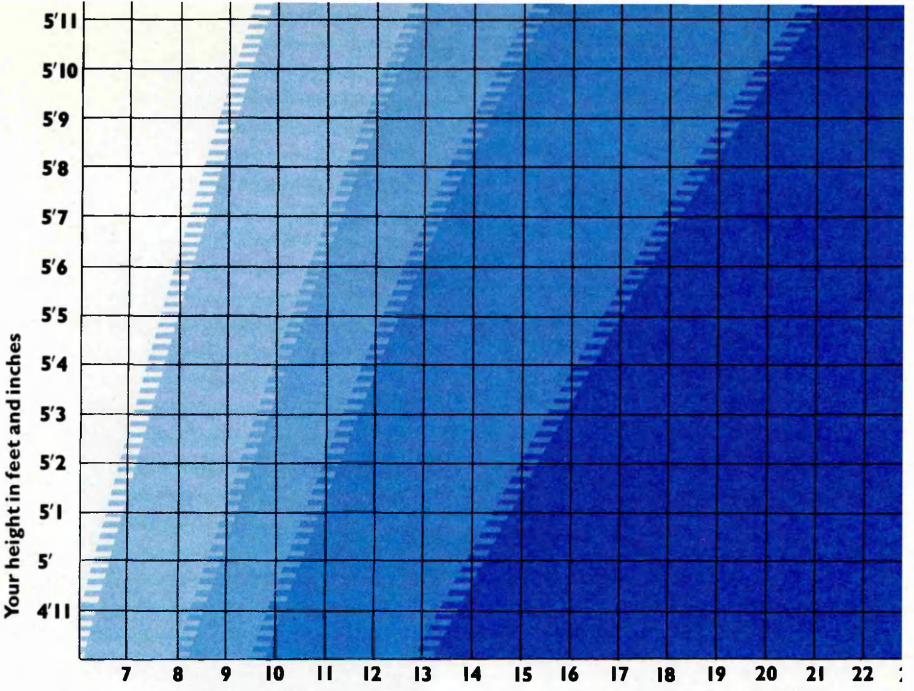
Look at the chart to see what you should weigh. Find your height (without shoes) and run your finger in a straight line across the chart. Find your weight (without clothes) and run your finger up the chart. Where the two lines cross tells you how your size rates.

The more overweight you are, the more likely you are to get high blood pressure or diabetes, both of which can lead to heart attacks and angina. So take the experts' advice and make an effort to keep your weight down.

For most people it's all too easy to put on weight. This might be because it runs in the family – whatever you eat seems to turn straight to fat. Or perhaps you aren't taking enough exercise to burn up the calories in your food.

But the most likely reason is that you are simply eating far too much of the wrong sorts of food. In other words, too many fatty and sugary foods. These are loaded with calories. Did you know that just one ounce of butter or margarine contains more calories than half a pound of potatoes? And two teaspoons of sugar contain nearly as many calories as a quarter pound of peas.

So the best way to keep your weight down is to cut down on the amount of fat and sugar in your daily diet.



From GARROW J.S. (1981) *Treat Obesity Seriously.* Edinburgh Churchill Livingstone.

Underweight. Are you eating enough?

OK. This is the desirable weight range for health.

Overweight. Not likely to have much effect on your health but don't get any fatter!

Fat. Your health could suffer if you don't lose weight.

Very fat. This is severe and treatment is urgently required.

What you can do

Eat less fat

The fat in your food is not only responsible for those extra inches on your waistline. It can also push up your blood cholesterol level. Cholesterol is one of the natural substances in the blood. It is mostly made from the fat in the food we eat. If there's a lot of fat in your diet you may have a high level of cholesterol in your blood. This can accelerate the build up of atheroma that

"Three years ago my husband was told his blood pressure was too high. Since then we've completely changed our diet. We eat more fresh fruit and vegetables, home-baked wholemeal bread and very little fat. And we've managed to cut out sugar and salt altogether. There are so many delicious things you can make without eating the wrong food. We honestly feel a lot fitter than when we were eighteen!"

eventually leads to heart disease. So the higher the level of cholesterol in your blood, the greater the risk of heart trouble.

The best thing to do is to cut down on the total amount of fat you eat by up to a quarter. You can see from the blue table how much fat there is in different foods. Use the table to help you decide where you can cut down on the fat in your diet.

- Eat less meat. Try fish or vegetable dishes. When you do eat meat, choose a leaner cut if you can – it's worth the extra cost. Or eat chicken, which has less fat than other meats and is usually cheaper. Always cut the fatty bits off meat.
- Grill, don't fry.
- Buy skimmed or semi-skimmed milk instead of full-fat milk. And try yoghurt instead of cream.
- You can see from the table that some cheeses are nearly half fat. Try a lower fat cheese.
- Spread less butter or margarine.
- Use less lard or oil in cooking.

You may have heard of **saturated** fats and **unsaturated** fats. The difference between them is their chemical make-up. They are all made up of basic substances called fatty acids. Some of these are saturated fatty acids (or **saturates**). The rest are unsaturated and include a special group called polyunsaturated fatty acids (or **polyunsaturates**). All the different fats we eat are made up of different combinations of different fatty acids. Most are high in saturates, but some are high in polyunsaturates.

Dietary experts believe that **saturates** are the main enemy so far as the heart is concerned. Too much fatty food high in saturates can push up your blood cholesterol. And that pushes up your chances of getting heart disease.

About half the fat that most people eat is rich in saturates. Experts believe that we should cut the amount of saturated fats we eat by up to a half. Take a look at the yellow table. It shows how high certain foods are in saturated fats. Use the table to work out how you can cut your intake.

Meat	Percentage fat
Fried streaky bacon	45%
Grilled streaky bacon	36%
Grilled lamb chops	29%
Pork pie	27%
Luncheon meat	27%
Liver sausage	27%
Roast lamb (shoulder)	26%
Fried pork sausages	25%
Roast leg of pork	20%
Fried beefburgers	17%
Grilled rump steak	12%
Casseroled pigs liver	8%
Stewed steak	7%
Casseroled chicken	7%
Fried lambs kidneys	6%
Tinned ham	5%
Fish	
Smoked mackerel	16%
Fried fish fingers	13%
Grilled kippers	12%
Cod fried in batter	10%
Steamed plaice	2%
Steamed haddock	1%
Cheese	
Cream cheeses	50%
Stilton	40%
Cheddar	34%
Parmesan	30%
Processed cheese	25%
Camembert	23%
Edam	23%
Cheese spread	23%
Cottage cheese	4%
Milk, butter, oils	
Oil (all kinds)	100%
Lard	99%
Butter	82%
Margarine (all kinds)	80%
Double cream	50%
Dairy ice-cream	7%
Gold-top milk	5%
Silver-top milk	4%
Yoghurt	1%
Skimmed milk	Less than 1%

The saturated fat in fatty food

These figures show the percentage of saturated fat in the total fat content of the food we eat, for example full cream milk (gold-top) is 5% fat. Two thirds (60%) of that fat is saturated.

All dairy products	
Milk Butter Cheese Cream	about 60%
Margarines	
hard (in packets)	about 40%
soft (in tubs)	
typical blends	about 30%
'high in polyunsaturates'	about 20%
Fats and oils	
Coconut oil	about 75%
Lard	about 45%
Blended cooking fat	about 40%
Corn oil Groundnut (peanut) oil Olive oil Soya oil	about 20%
Sunflower oil Safflower oil	less than 15%
Meats	
Lamb fat	about 50%
Beef fat	about 45%
Pork fat	about 40%
Chicken fat	about 35%
Fish	
Mackerel Cod Plaice	about 25%
Herring	about 20%

Remember, whenever you can, choose food **low in saturates** (sometimes labelled **high in polyunsaturates**). Look for margarines and cooking oils labelled 'high in polyunsaturates'. And think twice about choosing products labelled 'blended vegetable oils'. These contain mostly palm or coconut oil which are both rich in saturates, as you can see from the table.

Eat more fibre

Fibre is the name given to a whole range of complex plant substances. They pass right through the intestines without being absorbed into the body. By providing **roughage,** fibre not only aids digestion and helps prevent constipation. It also seems to stop too much fat and sugar getting into the bloodstream too quickly. And some scientists believe that a high-fibre diet can help keep the blood cholesterol down. What's more, fibre can give satisfying bulk to a meal without adding too many calories.

Fibre comes mainly from bread, cereals, potatoes, peas, leafy vegetables and fruit. All in all, it is the single most

important form of food likely to be lacking in your everyday diet. Look at the table below for some of the best sources of fibre.

Remember there is no fibre at all in any food which comes from animals, like meat, eggs and dairy products.

It's not difficult to put fibre in your diet. You simply need to eat more bread, including some wholemeal bread, and potatoes; choose a high-fibre breakfast cereal containing wholegrain or bran; and eat plenty of fruit and vegetables.

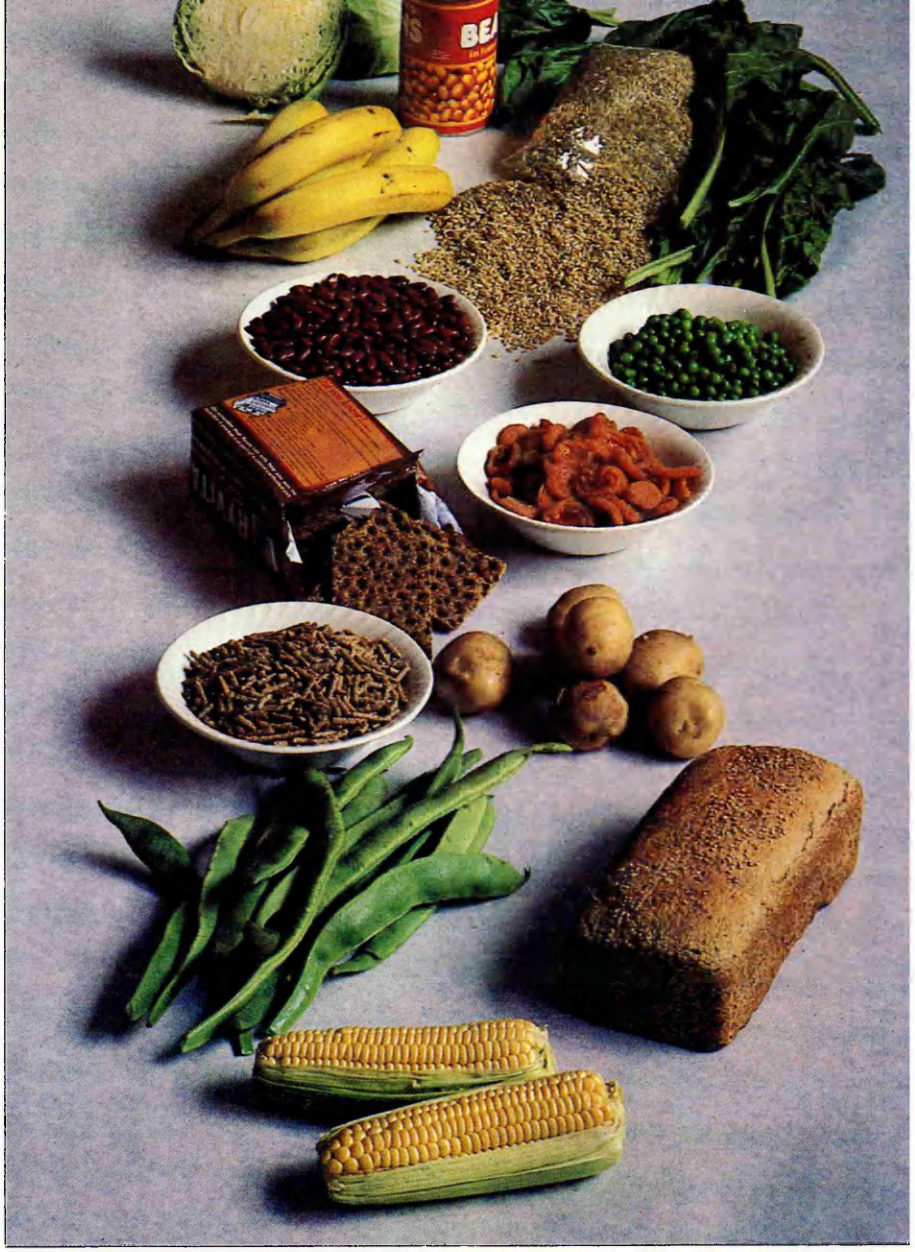
Fibre Content of Food

High Fibre Content
Peas
Bran cereal
Stewed prunes
Beans, like baked beans
Sweetcorn
Bananas
Wholemeal bread
Brown rice
Rye crispbread
Dried fruit
Leafy vegetables, like spinach
Potatoes, baked

Medium Fibre Content
Muesli
Most nuts
Most green vegetables
Apples
Oranges
Celery
Brown bread
Cornflakes

Low Fibre Content
Tomatoes
Potatoes, boiled
Grapefruit
Porridge
White rice
Lettuce
Cucumber
White bread

No Fibre Content
Sugar
Eggs
Butter
Cheese
Milk
Meat
Fish



Foods high in fibre

Eat less sugar

Not because sugar is especially harmful to the heart but because it is so tempting that it is difficult not to eat too many calories. Too many calories mean you get fat – and that can be bad for your heart.

Nobody *needs* sugar. It has no nutritional value other than its energy content. And we can get all the energy (calories) we need from the other food we eat. So try to reduce the amount of sugar you eat by at least a half.

Start by cutting down on sweets, chocolates, soft drinks, jams and other preserves and cakes or pastries. Just have them as an occasional treat. Try drinking your tea and coffee without sugar. Cut down gradually by about half a teaspoon a month. You'll soon get used to not using it. But if you really can't do without the taste, saccharin is sweeter than sugar and contains no calories at all.

It's surprising how easily you can adjust to less sugar. And you will probably start to notice all the other more subtle flavours in your food.

Eat less salt

Most of us eat far more salt than we need First of all, we buy food that already contains a lot of salt – meat, bacon, sausages, butter, cheese, tinned vegetables, bread and cereals. Then we add more salt when we cook it. And finally we sprinkle more salt onto it whe we eat it.

Some experts believe that this high intake of salt may be putting up the nation's blood pressure. You can reduce this risk by simply cutting down on the amount of salt you eat. You will be surprised how quickly you can get used to eating much less salt in and on your food.

So much heart disease could be prevented if every patient over the age of 35 had their blood pressure checked once a year during a visit to their GP.

High blood pressure

Blood pressure is the pressure which the heart and arteries apply in order to squeeze the blood around the body. When you are sitting or lying quietly your blood pressure stays at a steady resting level. In moments of exercise, excitement, anger or anxiety the level of blood pressure is raised to increase the blood flow to the brain and muscles. When the moment has passed the blood pressure drops again to its steady level. The sudden rise in blood pressure is brought about by the release of stress hormones like **adrenaline.**

What is high blood pressure?

High blood pressure (**hypertension**) means that the resting blood pressure is higher than normal. Very few young people have high blood pressure but after the age of 35 it becomes much more common, mainly because of the way we live our lives. These are some of the things that might give you high blood pressure:

☐ being overweight

☐ smoking

☐ drinking too much alcohol

☐ lack of regular exercise

☐ eating too much salt

☐ too much stress.

The trouble is that you may not even realise that your blood pressure is too high. By itself, high blood pressure doesn't feel any different. But high blood pressure makes the heart work harder and speeds up the 'furring up' of the arteries. So people whose resting blood pressure is too high are more likely to suffer from angina or a heart attack. They may also be in danger of having a **stroke,** when the blood supply to the brain is cut off by a blood clot or haemorrhage.

What you can do

There's a lot you can do to help keep your blood pressure at a normal level.

☑ Watch your weight. Keeping your weight at its correct level can help to keep your blood pressure down.

☑ Don't drink too much alcohol. Try not to drink more than two or three pints (or the equivalent), two or three times a week. Half a pint of beer is equivalent to one glass of table wine or a single whisky or other spirit.

☑ Stop smoking.

☑ Take some exercise.

☑ Eat less salt.

☑ Relax.

☑ Have your blood pressure checked. You may not know your blood pressure is high if you haven't had it checked recently by a doctor or nurse. This is especially important if you are over 35 years old. If your blood pressure is too high it can be kept at a normal level by medical treatment if necessary. There is good evidence that the control of blood pressure can prevent heart disease.

Is stress bad for the heart?

Most people would put stress at the top of their list of things that are bad for the heart. It seems obvious that worry and anxiety, or frequent crises and rows, can make your blood pressure go up and lead to a heart attack. But this is still difficult to prove, partly because stress is almost impossible to measure and define.

However, people who have a certain kind of personality – striving, ambitious, competitive, impatient, always pressed for time – seem to be more in danger of having a heart attack than more relaxed, easy-going types. We call the first heart-risk type Type A.

It seems that people who are more Type A are likely to have higher blood pressure and blood cholesterol levels than the calmer Type B. There is some evidence that this is because Type As have more stress hormones circulating in the bloodstream. This can lead, through high blood pressure and high blood cholesterol, to an increased risk of heart disease. It has also been suggested that someone who is normally calm but is frequently forced into stressful situations might eventually suffer the same effects as Type A.

How you can cope with stress

A certain amount of stress is an essential part of ordinary everyday life. It helps keep you on your toes and out of danger. Every time you cross a busy road or have an argument or watch an exciting programme on television your stress level goes up for a while. But if anxiety or

pressure of work continue for many months or years your heart may suffer. This kind of chronic stress can be difficult to recognise and sometimes impossible to avoid. Family problems, money worries, difficulties at work may not be easy to solve.

But you can help yourself by learning how to relax and trying to take things easy. When you have some free time, try to take up an activity, hobby or interest which helps keep your mind off your worries. It's difficult to worry about your problems when you are totally absorbed in doing some gardening, reading a book or watching a football match. And there are several simple ways of helping your mind and body relax during the day. Try one of them next time you feel tense about something.

Deep breathing

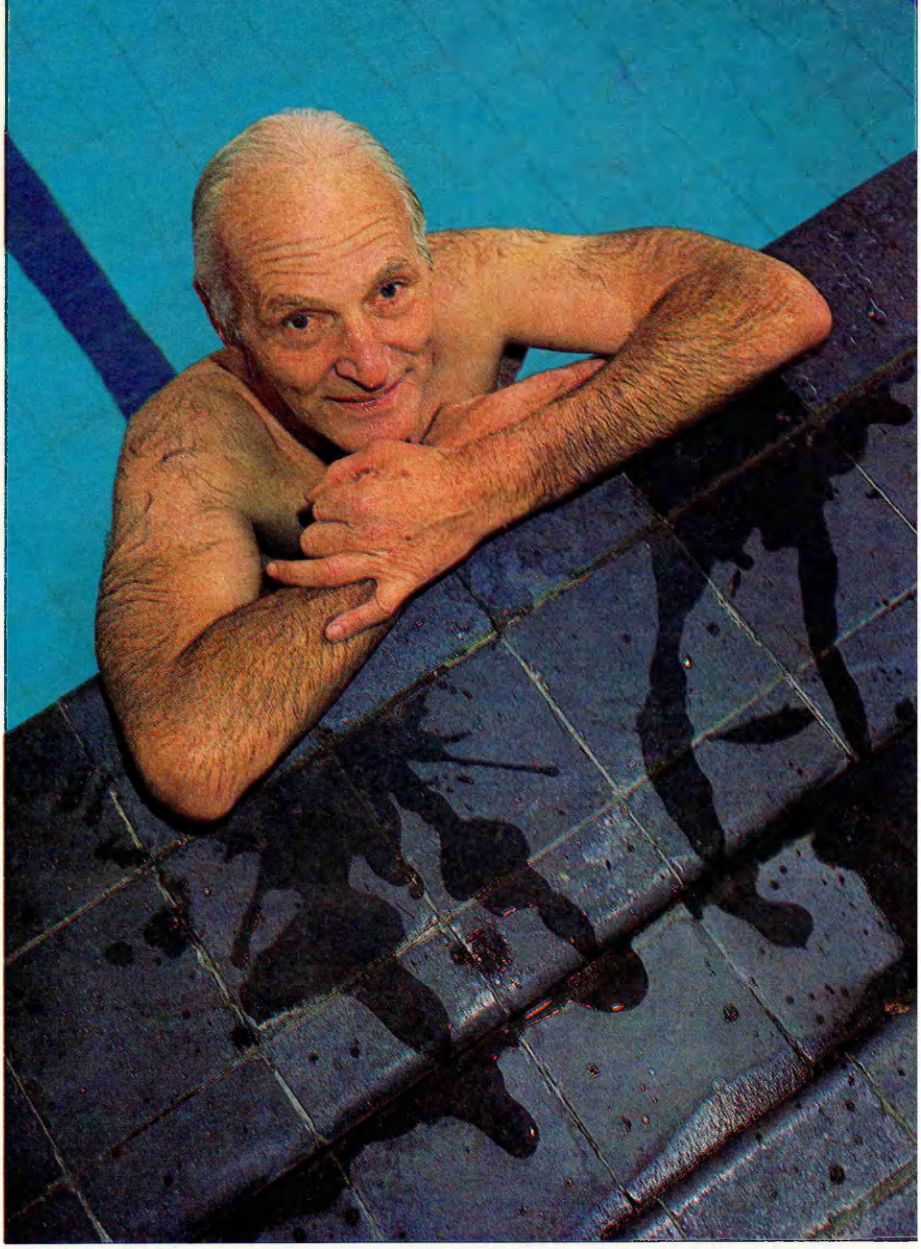
Sit or lie quietly, in as peaceful a place as you can find. Close your eyes. Picture a scene of tranquillity, like waves lapping on a sandy shore, or leaves rustling in a gentle breeze. Breathe slowly and deeply, through your nose. Make each out-breath long and soft and steady.

Clench-and-let-go exercises

Sit or lie quietly, in as peaceful a place as possible, and close your eyes. Clench both your fists for about 15 seconds. Then relax them and feel the tension draining away from your arm muscles. Repeat this twice. Then hunch your shoulders for 15 seconds, and relax, feeling the tension drain away. Repeat twice. Continue the same routine with jaw-clenching and relaxing. And finally, screw your eyes up tightly and relax them, feeling the tension disappear.

Meditation

Sit or lie quietly in as peaceful a place as possible and close your eyes. Repeat over and over to yourself a simple sound, word or phrase. Anything, so long as it's simple and easy to repeat. Just concentrate on the repetition so that it fills your mind and banishes anxious thoughts. Let yourself relax into this steady rhythm for 5 or 10 minutes.



"After my heart attack I realised I had to do something about the state of my health. I was overweight and pretty unfit. It's not easy to change your routine but I knew that unless I got some more exercise and kept myself slim I'd be digging my own grave. So I took up running and joined the swimming club at work. I really enjoy it."

Exercise

Any activity which gives you some exercise is especially good for your heart. Exercise helps relieve the tensions that have been building up inside you. And your body gets a chance to let off steam. All kinds of exercise from the gently stretching postures of yoga to the vigorous leaps and bounds of badminton will help you to combat stress.

Exercise not only helps to reduce stress. There is good evidence that regular vigorous exercise can have a protective effect on the heart. And exercise can certainly help to make you feel and look better.

Your heart will benefit most from the kind of exercise that builds up stamina. **Stamina** means staying power or the ability to keep going without gasping for breath. It depends on the efficiency of your muscles and circulation. And the most important 'muscle' of all is the heart itself.

To build up your stamina you need to choose a form of exercise that gives your body plenty of movement, and is just energetic enough to make you fairly breathless (but not gasping for breath). This is called **dynamic** exercise. The vigorous effort of moving your muscles rhythmically creates a greater demand for oxygen in the blood, and more work

Stamina Rating

Badminton	✱✱
Canoeing	✱✱✱
Climbing stairs	✱✱✱
Cricket	✱
Cycling (hard)	✱✱✱✱
Dancing (ballroom)	✱
Dancing (disco)	✱✱✱
Digging (garden)	✱✱✱
Football	✱✱✱
Golf	✱
Gymnastics	✱✱
Hill walking	✱✱✱
Housework (moderate)	✱
Jogging	✱✱✱✱
Judo	✱✱
Mowing lawn by hand	✱✱
Rowing	✱✱✱✱
Sailing	✱
Squash	✱✱✱
Swimming (hard)	✱✱✱✱
Tennis	✱✱
Walking (briskly)	✱✱
Weightlifting	✱
Yoga	✱

✱ not much effect
✱✱ beneficial effect
✱✱✱ very good effect
✱✱✱✱ excellent effect

for the heart and lungs. Regular exercise of this kind improves the balance of fatty substances in the bloodstream, lowers the resting blood pressure level and strengthens the heart muscle.

At the simplest level, brisk walking is an excellent stamina-building exercise. But you could also try running up stairs, jogging, skipping, disco-dancing, cycling or swimming.

Have a look at the chart above to help you choose the form of stamina-building exercise which suits you best. Whatever you choose make sure it's something you enjoy or you will never keep at it. Of course you can change from one activity to another; it's the level of exertion that's important.

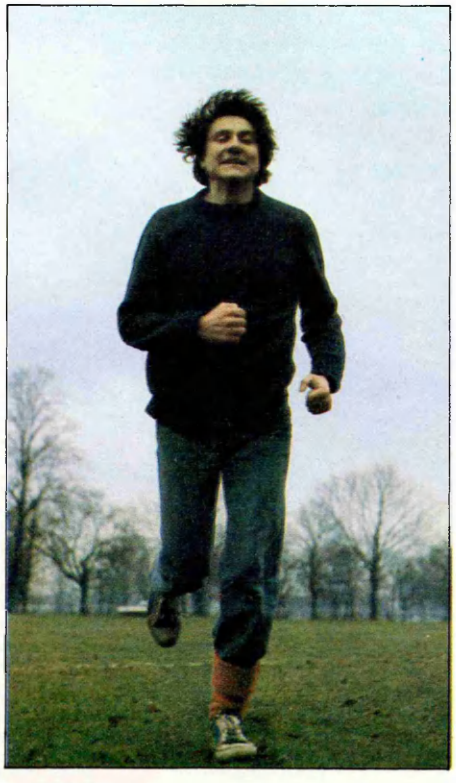
Once you have found a form of exercise which you enjoy, try to do it:

☐ **Often enough** – 2 or 3 times a week for 20 or 30 minutes at a time

☐ **Hard enough** – to make you fairly breathless but not gasping for breath

☐ **Long enough** – it must become a part of your life. For good.

Remember, whatever you choose to do, always go easy to start with and build up gradually. Don't attempt vigorous competitive games – like squash – until you've reached a good level of fitness. If you're recovering from an illness or operation or are worried that exercise may affect any other aspect of your health, consult your doctor first.



"I wouldn't say I was a fanatical jogger but I do enjoy going for a half hour run once or twice a week. I usually go early in the morning and it really sets me up for the day. I think a bit of regular exercise is especially important for someone like me who's approaching 40 and sits at a desk most of the day."

SOMEONE HAS A HEART ATTACK

A heart attack can be very painful and frightening so the first thing most people need is rest and reassurance. But for some people there is a danger that the heart might stop beating properly, or even stop altogether. If this happens the person will lose consciousness and die unless emergency first aid is given. So the critical time is the first few minutes before medical help arrives.

If you're not sure whether it is a heart attack look for these signs:

- Sudden, crushing vice-like pain in the centre of the chest which may spread to the arms, throat, jaw or back
- Ashen (waxy-white) skin and blueish lips and nailbeds
- Feeling cold, clammy, faint, giddy or sick
- Breathing and heart may stop.

If you think it might be a heart attack, send for a doctor or an ambulance (dial 999) as soon as you can. Stay with the victim and reassure him until help arrives.

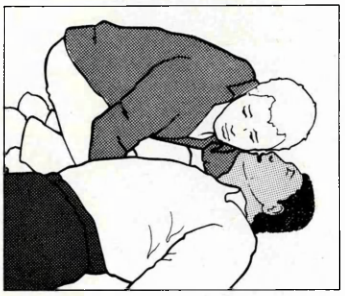
You may have to give emergency first aid before medical help arrives. Turn to the next page for a basic guide to first aid for a heart attack.

FIRST AID

Is he conscious?

no → **Quickly check to see if he is breathing.**
How to check:

Can you hear or feel the breaths? Put your ear against his nose and lips.



Can you see or feel the chest or abdomen moving?

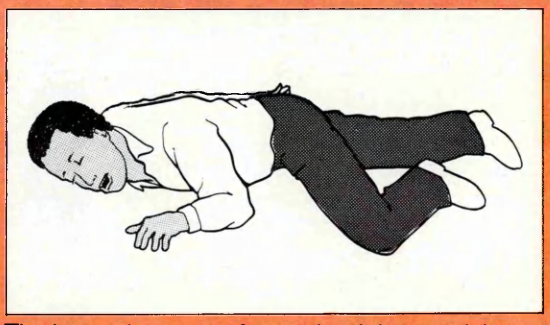
Does his face look ashen or purple? (If you can't tell because his skin is coloured, check whether the inside of the lower lip or the nailbeds are blue.)

Is he breathing?

no—

yes

1. Loosen his clothing at the neck, chest and waist.
2. Put him in the recovery position:



This keeps the tongue forward and the mouth low so that the airway is kept clear and he can breathe.

3. Stay with him, watching all the time in case the breathing or heart stop.

yes

1. Gently put him in a half sitting (semi-recumbent) position with head and shoulders raised and knees bent. This is the most economical position for the heart.

2. Loosen clothing at the neck, chest and waist.
3. If he is cold, cover him with a blanket but do **not** apply a hot water bottle – this draws the blood to the skin surface and away from the vital organs, like the heart.
4. Do **not** give him anything by mouth – not even aspirin or brandy.
5. Do **not** move him unless it is essential for safety reasons.
6. Stay with him and reassure him until the doctor arrives.

Learning about first aid

You'll be able to cope much better in an emergency if you have learnt how to do first aid properly. Heart resuscitation can only be properly learned with expert guidance.

The British Red Cross Society and the St. John Ambulance (or the St. Andrew's Ambulance Association in Scotland) run first aid courses. For information about courses in your area, contact either your local Red Cross branch (address and phone number in your telephone directory under either British Red Cross Society or Red Cross); or your local St. John Ambulance branch, or if you live in Scotland, your local branch of the St. Andrew's Ambulance Association (address and phone number in your telephone directory). The Royal Life Saving Society also runs first aid courses. See back for details.

Start mouth-to-mouth resuscitation (Kiss of Life) immediately.

Quickly clear the mouth of any dirt or vomit etc.

Bend the head backwards with one hand and push the jaw upwards with the other hand. This lifts the tongue off the back of the throat. This action alone may allow breathing to start.

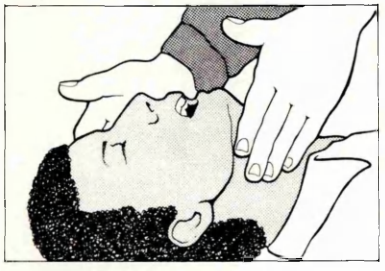
If not, quickly give him 4 full breaths:
Pinch the nostrils together then take a deep breath in, seal your lips around his mouth and blow into it. See that the chest rises as you blow in.

Take your mouth away and watch the chest fall.

After 4 full breaths, check his pulse to see if his heart is beating.

How to check:

Can you feel the carotid pulse? Put two fingers in the groove at the side of his Adam's apple and press firmly.



If you can't feel a pulse, and his face is ashen or purple, then his heart has stopped beating.

Is his heart beating?

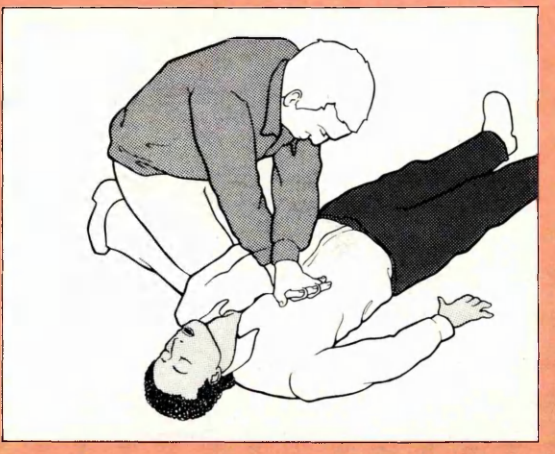
yes

Continue mouth-to-mouth resuscitation giving 16 to 18 breaths per minute (one every 3-4 seconds) until breathing starts again.

no

Start heart resuscitation.
(Never attempt heart resuscitation if the heart is beating).

1. Lie the patient on his back on the floor or other firm surface, and kneel alongside.
2. Put the heel of one hand on the lower half of the patient's breast bone, and cover it with the heel of the other hand. Press down hard on the lower half of the breast bone to a depth of about 1½ inches.



Press about 80 times a minute. To help you keep time, count out a rhythm of "one and ... two and ... three and ..." as you do it.

3. He will not start breathing again until the heart has started beating. So after pressing 15 times, stop the heart resuscitation and give 2 quick breaths by mouth-to-mouth resuscitation as described.

   Continue to give 15 presses and 2 breaths in this way.

   If there is another person with you, get them to do the heart resuscitation. Give 5 presses, 1 breath, 5 presses, 1 breath, without pausing. Press about 60 times a minute. To help you keep time, count out a rhythm of "one thousand and one ... one thousand and two ... one thousand and three ..." as you do it.
4. After one minute, check the carotid pulse to see if the heart has restarted. Then keep checking every three minutes, or until the colour of the face begins to improve.

   Continue heart resuscitation until the heart starts beating.
5. Once the heart has started beating keep on with mouth-to-mouth resuscitation until breathing starts again.
6. Once the breathing has started, put the person into the recovery position and wait for medical help to arrive.
7. Stay with the person, watching all the time in case the breathing or heart stop again.

To avoid awkward repetition of he/she him/her, the heart attack victim has been referred to throughout this chart as he.

MEDICAL TREATMENT

The main thing is to allow the patient to rest while the heart recovers. Immediate treatment after a heart attack is usually:

☐ Complete rest in bed (this is usually but not always in hospital)

☐ A painkiller (if necessary)

☐ Treatment to correct the heart beat (if necessary)

☐ Careful observation (keeping a close eye on the patient's condition).

In hospital

Most patients will stay in hospital for several days for observation. Some patients will be admitted to a special ward called the **coronary care unit.** This unit has special staff and equipment to cope with the complications of a heart attack. The patient may be connected to a monitor called an electrocardiogram (ECG) which can detect any fault in the heart beat. If anything is wrong, treatment can be started immediately.

After a few days complete rest, the patient can usually start to get moving again, and may be allowed home to be looked after by his own doctor.

AFTER A HEAR ATTACK

The heart has a remarkable capacity for healing its damaged tissue and most people who recover from a heart attack have every chance of leadin an active life afterwards.

It's natural to feel a bit shaky and insecure when you've just left hospital after a heart attack. Without the ECG and the watchful eye of trained staff it's easy to feel frightened to do anything. But it's a mistake to feel over cautious.

Getting back to normal

Most people can expect to be more or le back to normal within a few months aft a heart attack. But of course this depenc on how bad the attack was.

Your doctor can help you to work out carefully planned programme of activity This will enable you to gradually and steadily build up your physical strength and self-confidence.

At first you may only be up and abou for part of the day. Then you'll start doing a little more – going for a gentle walk everyday perhaps. Soon you will probably be able to build up to more strenuous exercise like brisk walking, swimming or cycling. Ask your doctor for advice.

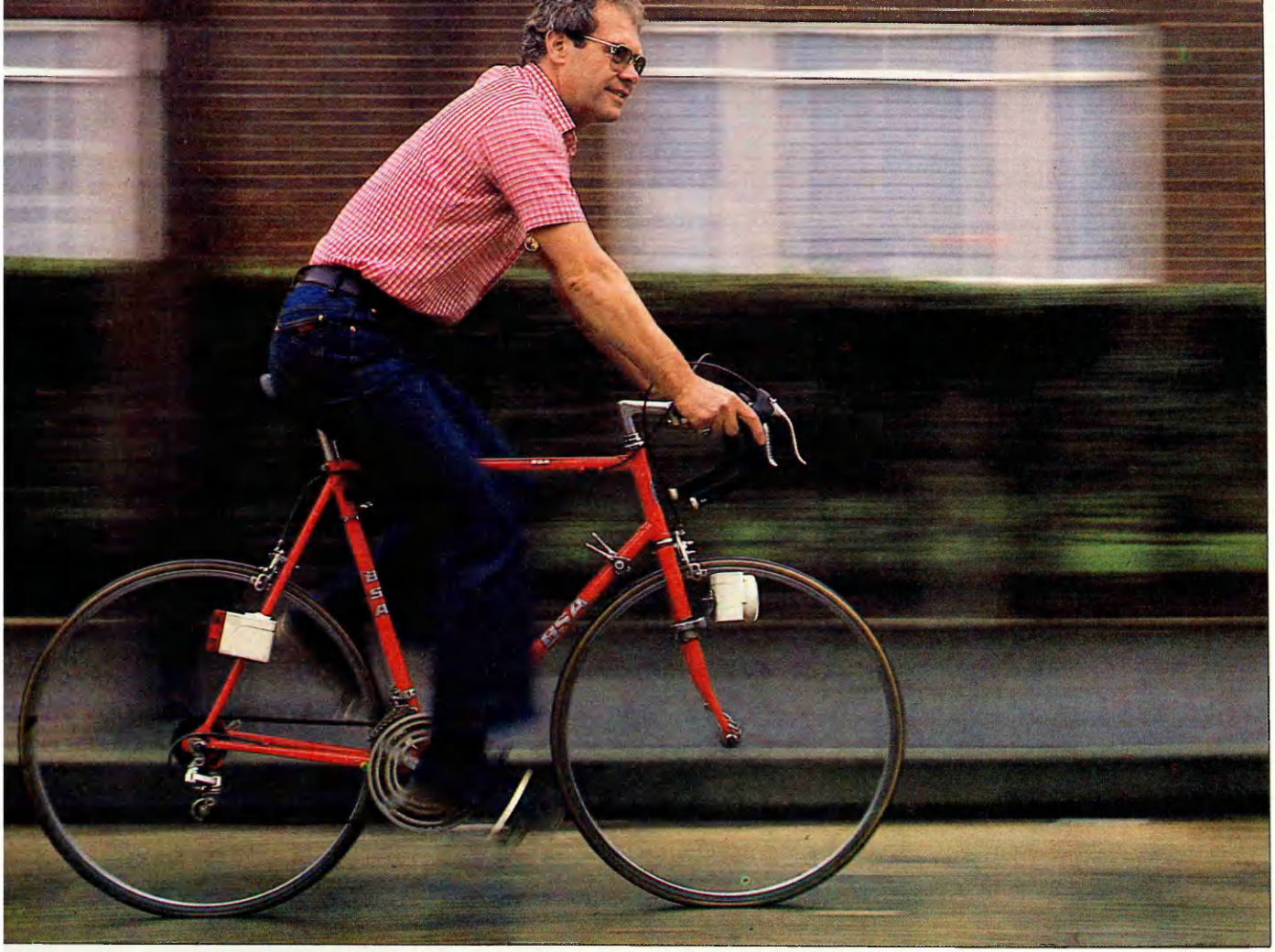
Of course, everyone's progress after a heart attack is different. It all depends c how much you feel you can do and how much your doctor thinks you can manage.

Back to work?

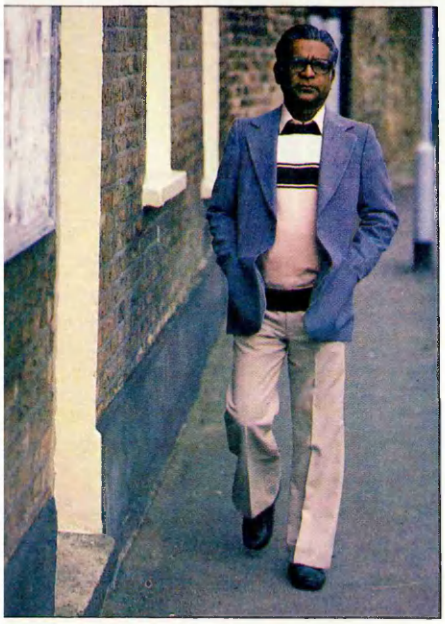
You can usually expect to return to your job within several weeks. Again, this wi depend on how bad your heart attack was. It is obviously unwise to do really hard physical work immediately after a heart attack. In some cases your doctor or occupational health services may be able to help negotiate with your employer a return to lighter work or part-time hours, before you go back to your full-time job.

Healthy eating

If you've already had a heart attack it's just as important to make an effort to ea less fatty food and keep your weight down. By eating sensibly you could help



“I used to play a lot of squash before my heart attack, but for the moment I’m happy with cycling and swimming until I feel fit enough for something more strenuous. You soon find out your limitations and do as much as is good for you.”



“This was not my first heart attack so I’m taking things slowly, trying to build up my strength gradually. I usually go for a gentle walk each day to the shops or the park so that I can get a bit of exercise and some fresh air. And every day I try to walk just a bit further than the day before.”

reduce the risk of another attack. Look at page 8 for a guide to healthy eating.

Smoking

If you haven’t already stopped smoking now is the time to give it up for good. As soon as you stop smoking you will start to reduce the risk of having another heart attack. Have a look at the section on page 7 for advice on giving up or get a copy of *So you want to stop smoking* from your local health education unit or by writing to:

‘So you want to stop smoking’
PO Box 410
London SE99 6YE

or, if you live in Scotland:

Scottish Health Education Group
Woodburn House
Canaan Lane
Edinburgh EH10 4SG.

Sex

This depends on how long it takes you to recover your physical strength. It is usually better to wait until you can manage brisk walking without discomfort or breathlessness before you start having sex again. Again, your doctor will be able to give you advice.

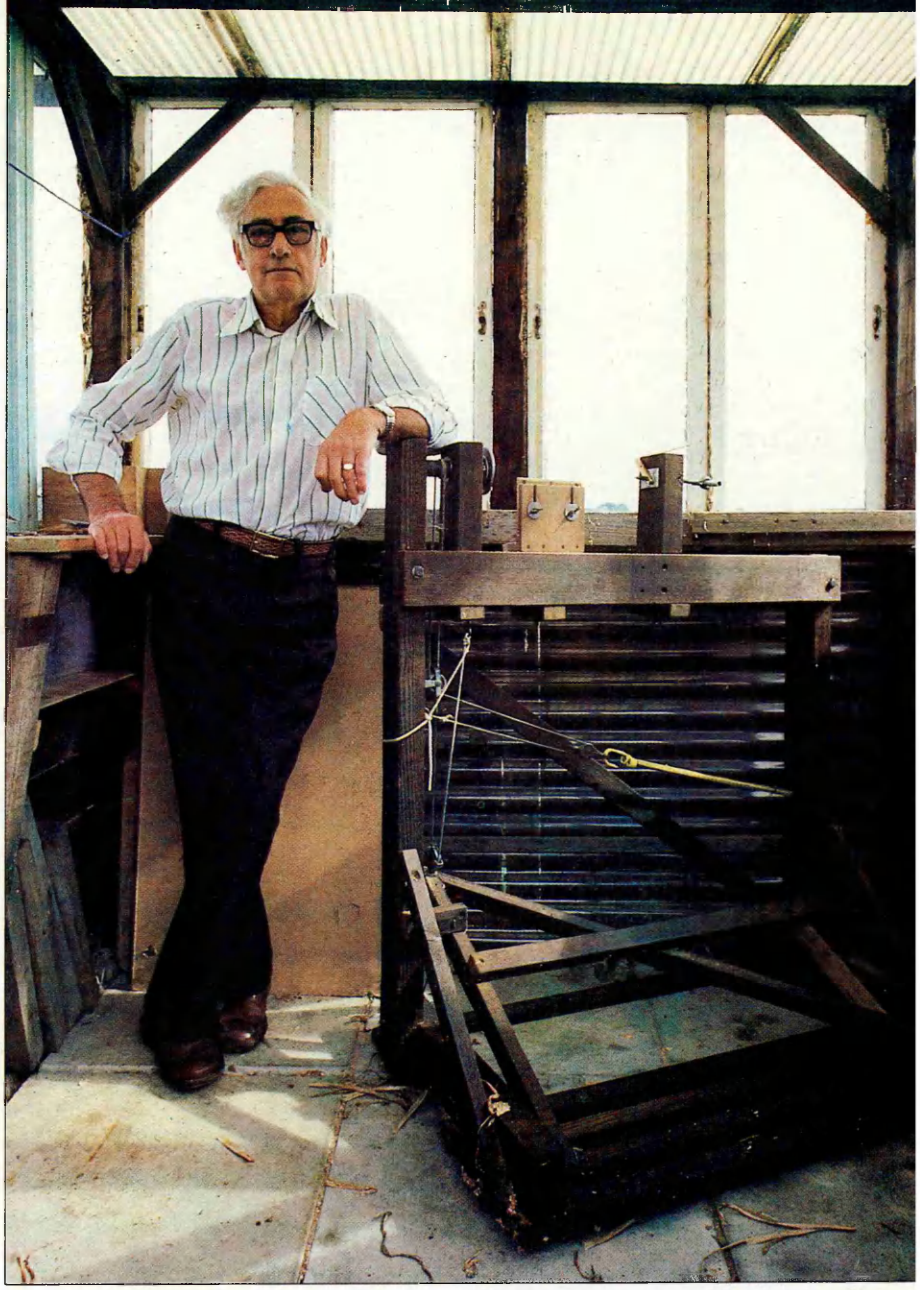
Driving

Because driving is a stressful activity it can push your blood pressure and pulse rate up to quite high levels. So you shouldn’t start driving again until your heart has recovered enough. For most people this means a few months after their attack. But it may be necessary to give up heavy driving (for example, heavy goods vehicles and public transport). In all cases check with your doctor before you start to drive again.

Staying fit and healthy after a heart attack

If nothing else, a heart attack at least gives you the chance to stop and think about the way you have been living your life. Many people who have recovered from a heart attack say their outlook and way of life have changed so much that they never felt better!

This booklet has shown how the risk of heart disease can be reduced. If you have already had a heart attack it makes sense to think about what you can do to help avoid another. It's never too late to change. Stop smoking, eat sensibly, take some exercise and learn to relax. You'll not only feel fitter than you've felt for years. You'll be giving yourself and your heart a new lease of life.



"When I had my heart attack I was only a year off retirement. I was a mechanical engineer but the company let me go back to a desk job until I retired. Now I can enjoy myself at home doing the things I never really had time for before. We go walking a lot and I've built my own workshop at the bottom of the garden so that I can get on with my woodwork."

GLOSSARY

drenaline One of the so called 'stress ormones'. Speeds up the pulse rate, uts up the blood pressure, increases lood flow to brain, heart and muscles. repares the mind and body for quick ıought and action.

ngina Constricting feeling, usually a eavy crushing pain in the chest and ometimes the neck, shoulders and ms. Comes on after exertion or notion and is relieved by rest. Caused y poor blood supply to the heart ıuscle.

orta Main artery taking oxygen-laden lood from the heart to the other arteries ıpplying the body.

rrhythmia Any variation from the ormal regular heartbeat.

rtery Vessel carrying blood away from ıe heart to supply the tissues. Has a ıick muscular wall to withstand the lood pressure.

theroma Fatty deposit containing ıolesterol which gradually builds up on ıe inner lining of arteries over a period f many years. Can cause the blockages hich lead to heart attacks and strokes.

lood pressure Pressure of blood in the ıain arteries needed to push it through ıe smaller vessels of the circulation. leasured using a sphygmomanometer. bnormally high blood pressure is called ypertension.

alorie Unit of energy. A calorie is the ıergy needed to raise the temperature f a kilogramme of water by 1°C. The verage person at rest burns about 1 alorie a minute to supply the body's asic energy needs.

apillaries Microscopic vessels that arry blood through the substance of the ssues, between the arteries and veins. lood pressure is needed to force the lood through.

arbon Monoxide Poisonous gas in igarette smoke which interferes with ıe oxygen carrying capacity of the lood.

ardiac Relating to the heart.

ardiovascular system The heart and lood vessels.

arotid arteries Pair of large arteries ıpplying the brain. Can be felt on each side of the Adam's Apple.

Cerebral Relating to the brain, eg. cerebral artery, cerebral thrombosis.

Cholesterol Fatty substance needed by the body as a building block for tissues and chemical processes. Some cholesterol is present in our food. But most is made in the body from other substances derived from food, particularly fats. Blood carries cholesterol around the body.

Coronary arteries Small arteries which encircle the heart and supply blood to the heart muscle itself. They branch off the aorta as it leaves the heart. Atheroma in the coronary arteries is the main cause of heart disease.

Coronary heart disease (CHD) Disease, such as angina or heart attack, caused by atheroma silting up the coronary arteries.

Coronary thrombosis Blood clot in a coronary artery blocking the blood supply to part of the heart muscle, and causing a heart attack (also called a coronary).

Electrocardiogram (ECG) Picture of the electrical impulses made by the beating heart.

Haemoglobin Red substance in blood which carries oxygen from the lungs to the tissue. Its action can be blocked by the poisonous gas carbon monoxide.

Heart attack Non-medical term for a sudden serious disorder of the heart. Usually refers to a coronary thrombosis.

Hypertension Abnormally high blood pressure which, if uncontrolled, can increase the risk of heart disease or a stroke.

Infarct Part of an organ which has died because its blood supply is cut off. Usually refers to the dead part of the heart muscle after a coronary thrombosis.

Ischaemia Poor blood supply usually due to narrowing or blockage of an artery.

Myocardial infarction Commonest type of heart attack in which part of the heart muscle dies because its blood supply is cut off due to a coronary thrombosis. May cause sudden death.

Nicotine Poisonous substance in tobacco. Strongly addictive. Increases pulse rate and blood pressure, and disturbs the balance of fatty substances in the bloodstream.

Obesity Condition of being grossly overweight, at least 20% heavier than the heaviest weight in the 'ideal' range for that person's height.

Palpitations Sensation of 'fluttering' in the chest, usually caused by a faster or stronger or perhaps an irregular heartbeat.

Polyunsaturated fats Fats which contain a high proportion of polyunsaturates. Thought to be less harmful to the heart and arteries than saturated fats, and may even be beneficial. Examples are fish oils and certain vegetable oils like sunflower, safflower and soya oil.

Pulse Wave of pressure in the arteries which follows each heart beat. Can be felt through the skin over some arteries. Examples are **radial** pulse (wrist) and **carotid** pulse (neck).

Saturated fats Fats which contain a high proportion of saturates. Thought to be potentially harmful to the heart and arteries if eaten in too great a quantity over a period of years. Examples are the fats in dairy products like butter, cream and cheese, in meat and meat products, in lard, and in some vegetable oils like coconut and palm oil.

Stamina Physical fitness that gives a person heart and muscle endurance or 'staying power'; the ability to keep up vigorous exercise without gasping for breath. Stamina fitness is thought to help protect the heart against heart attacks.

Sternum Breast bone.

Stress General term that covers a variety of everyday psychological pressures that might have ill-effects on the heart and arteries.

Stroke Paralysis or malfunction due to a sudden lack of blood supply to part of the brain, usually caused by a thrombosis or rupture of a cerebral artery.

Thrombosis Blockage due to a blood clot in a vessel. See coronary thrombosis.

Veins Vessels draining blood from the tissues and back to the heart, ready to be pumped round the lungs and re-charged with oxygen.

FURTHER INFORMATION

Books

Avoiding heart attacks (DHSS, 1981)
Available from HMSO bookshops

Avoiding heart trouble (Consumers Association, 1980)
Available from leading booksellers

Heart attacks: prevention and treatment (BMA, 1979)
Available by post from:
Family Doctor Publications
BMA House
Tavistock Square
London, WC1H 9JP.

First aid

You can get details of first aid courses from:

The British Red Cross Society
9 Grosvenor Crescent
London SW1X 7EJ

St John Ambulance
1 Grosvenor Crescent
London SW1X 7EF

St Andrew's Ambulance Association
St Andrew's House
Milton Street
Glasgow G4 0HR

The Royal Life Saving Society
Mountbatten House
Studley
Warwickshire B80 7NN.

First Aid Manual: (Dorling Kindersley Ltd., 1982)
The authorised manual of St. John Ambulance, St. Andrew's Ambulance Association and the British Red Cross Society. Available from leading booksellers.

The Scottish Health Education Group

Advice for those recovering from a heart attack.

The aim of this advice is to guide you concerning your activities after leaving hospital and to get you back to a normal active life. Some answers are given to questions that might have bothered you about your illness.

Prepared by Prof. M. F. Oliver, M.D., F.R.C.P.
and Dr. A. L. Muir, M.D., F.R.C.P.E.

What is a heart attack?

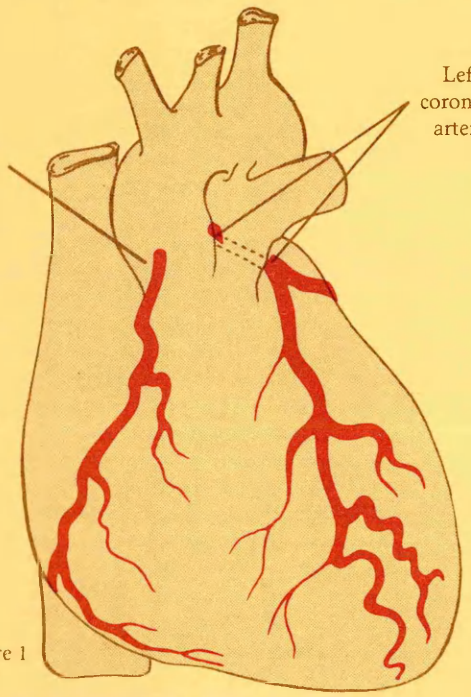


Figure 1

The heart, like any other muscle, needs to be supplied with blood to keep it working. The main arteries, called the coronary arteries, bring blood to the heart (see fig. 1) .

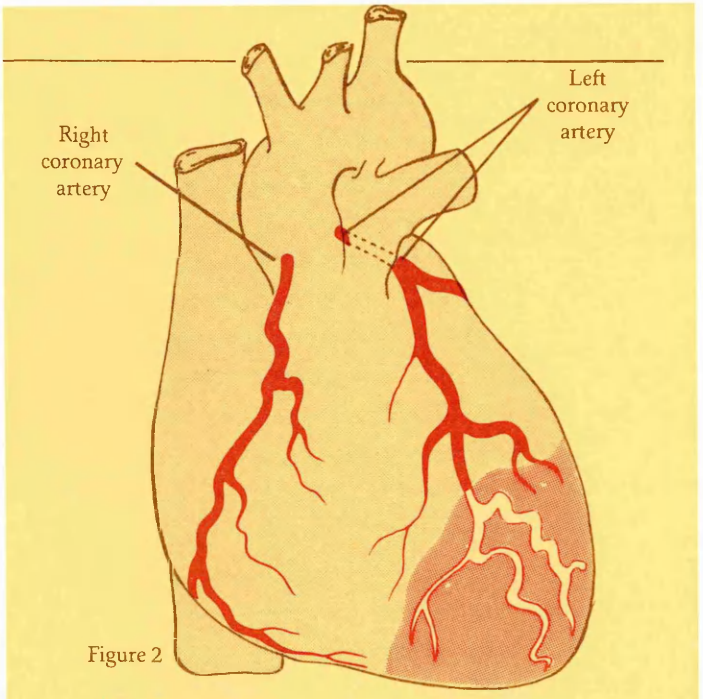


Figure 2

A heart attack occurs when there is a temporary block of one of these arteries, reducing the blood supply to part of the muscle of the heart. This causes injury to the portion of the heart muscle supplied by that small artery; the injury is similar to a bruise of the muscles in the arm or leg, occuring after a sharp blow, and like such bruising it takes two or three weeks to heal (see fig. 2). During the acute stage of injury the heart has been rested. The convalescent stage which you are now entering is based on rebuilding your heart's efficiency for a full return to fitness.

Physical Activity

Physical activity should be graduated and increased as the weeks go past.

During the first week at home you should follow much the same routine as in hospital, but don't be frightened to walk upstairs or out of your home. During the second week at home increase your activities so that by the third or fourth week after leaving hospital you should be undertaking most household and light gardening activities. Before you return to work you should be physically active again.

If you were unfit before your heart attack and found little time or interest in sport or leisure activities you should make a decision to do more and take up some activity such as hill walking, swimming, playing golf, etc. This can improve the blood supply to the heart.

You may find that you are not able to do much at first but you should concentrate on becoming more and more active, although strenuous activity should be avoided. Carrying heavy loads such as suitcases should also be avoided.

Remember you could walk to and from work, and climb stairs instead of taking the lift, but no sprinting up the stairs to see if you can beat the lift!

Return to Work

If progress is good you should be back at work within two months after leaving hospital, unless your doctor advises you otherwise. Normally you can return to your old job unless a lot of heavy manual work is involved. If your doctor says your job is not suitable, you should contact your local Job Centre or Employment Office and ask for an interview with the Disablement Resettlement Officer.

Do not be discouraged if your progress seems slow. Some people recover from a heart attack more quickly than others.

Sleep and Rest

It is common to feel tired when recovering from a heart attack and there is no reason for you to think that you are not progressing when unexpectedly you get tired. Just give in to it and have a rest—even if it is socially inconvenient. Make sure you have seven or eight hours sleep each night. Sleeping tablets are not usually necessary but your own doctor will advise you about this. A short sleep in the afternoon is good for you during the first few weeks after leaving hospital, and also later if you can manage it.

Responsibilities

While you are at home recovering, you have a marvellous opportunity to take an extended holiday and to examine the way you lived before your illness. Did you work long hours of overtime? Did you let the housework get on top of you? Did you worry unnecessarily about your job, or family, or your home life? Did you get into arguments or get upset easily? There are many ways to cut down on all this stress and strain in your life. For example, cut out the overtime, let other people help you with the work in the house or in your job, keep out of arguments, stop rushing about as if everything had to be done at breakneck speed. If you can calm down and lead a less hectic life, you will probably find yourself feeling mentally and physically healthier than you have done for years.

Diet

The chart below shows average weights for different heights. Check your weight against that shown on the chart. If you are overweight a low calorie weight reducing diet is advised (see page 13). If you are not, then no particular dietary advice is necessary.

MEN		WOMEN	
Height	Weight	Height	Weight
5' 3"	9 st 8 lb	4' 11"	8 st 3 lb
5' 4"	9 st 10 lb	5'	8 st 5 lb
5' 5"	10 st 1 lb	5' 1"	8 st 7 lb
5' 6"	10 st 4 lb	5' 2"	8 st 9 lb
5' 7"	10 st 8 lb	5' 3"	8 st 12 lb
5' 8"	10 st 11 lb	5' 4"	9 st 1 lb
5' 9"	11 st 2 lb	5' 5"	9 st 5 lb
5' 10"	11 st 6 lb	5' 6"	9 st 8 lb
5' 11"	11 st 11 lb	5' 7"	9 st 12 lb
6'	12 st 2 lb	5' 8"	10 st 2 lb
6' 1"	12 st 7 lb	5' 9"	10 st 6 lb
6' 2"	12 st 11 lb	5' 10"	10 st 10 lb
6' 3"	13 st 1 lb	5' 11"	11 st 0 lb
		6'	11 st 4 lb

However if you are under 50 years of age with high blood fat levels, specific dietary instructions may be given by your own doctor. These are outlined on page 14.

Smoking

Stop smoking cigarettes! There is clear evidence that the risk of a subsequent heart attack is substantially reduced in those who give up smoking. You should therefore stop cigarette smoking completely, and for good. Although pipe smokers are less at risk, cigarette smokers who take to a pipe may find it difficult not to inhale and inhaling pipe tobacco smoke is also harmful. If you find it difficult at first leaflets are available from the Scottish Health Education Group, Freepost, Edinburgh EH12 0PQ, giving practical hints on "How to stop Smoking".

Sexual Intercourse

Intercourse is probably best avoided during the first month after discharge from hospital. Thereafter, as you return to physical health, no restrictions are necessary.

Car Driving

You should wait two months after your heart attack before driving your car, and when you do, try to cut down the strain involved by avoiding city traffic and congestion, by allowing plenty of time for a journey and by travelling at a more leisurely pace.

You will not be allowed to drive heavy goods or public services vehicles.

Air Travel

You can still travel safely by aeroplane, although it is wise to avoid long and tiring flights for the first two months after your attack.

Angina

You may have chest pain on exercise. Not all chest pains come from the heart. Unless it is frequent and interfering with your activities, it can be ignored, although there are drugs available for its control. If it is causing distress, consult your doctor. Car driving, cold and winds can occasionally produce angina; if so, such factors should be avoided.

Special Advice for Women

Women often find themselves in a position of considerable responsibility on returning home after a heart attack. You may feel, for example, that the house has been rather neglected while you have been away, that you are responsible for the running of the household, the provision and cooking of food, and the care of your close relations. It is important during the first month or so after returning home that you do not take on your full domestic activities and that you continue to allow yourself to convalesce. If things are not just as you want in your own home, then be patient and put them right when you are completely recovered. Try to find a relative or a neighbour who will help with the shopping.

You should not take oral contraceptives. While "The Pill" is harmless in young healthy women, there is an increased risk of a heart attack in those who already have heart disease. You should consult your doctor about other methods of Family Planning.

You should not take oestrogens as hormone replacement treatment following the menopause.

Treatment

Some people require drug treatment of various kinds. Your own doctor will advise you.

Do not be discouraged because you have had a heart attack. Most people return to a full active life and some indeed become fitter than ever before.

Show this booklet to your family and friends so that they understand the advice given and can help make your recovery all the more enjoyable.

Diet for those who are overweight

SOME GENERAL RULES FOR LOSING WEIGHT

1. Eat as much as you like of:-
   Fresh green vegetables and salads, fresh fruit, and clear soup.
2. Take average portions of:-
   Meat, poultry, cheese (½lb/week) , eggs (3/week) and fish.
3. Cut down the amount of starchy foods you eat. For example reduce the size of each portion of:-
   Potato, bread, butter, milk, peas and sweetcorn, thick soups and sauces. Remember alcohol is fattening too, so try to reduce your drinking.
4. Cut out altogether sugary foods such as:-
   Cakes, sweets, biscuits, tinned fruit and sweetened fruit juices, and sugar in tea or coffee.
5. Avoid fried food and sauces or soups thickened with flour.

Diet for those with high blood fat levels

1. Eat less meat, and cut off visible fat. You can however eat poultry and fish.
2. Avoid fats such as butter, hard margarine, beef dripping and white lard both for spreading and for cooking. This means you will have to avoid most manufactured cakes, chocolates and biscuits because they also contain the wrong fats. Instead use corn oil or sunflower oil for cooking. Do not use lard or oils labelled simply 'vegetable oil'.
3. Use skimmed milk and avoid ordinary milk and cream.
4. Allow yourself no more than 3 eggs a week.
5. Reduce the amount of hard cheese eaten. Try cottage cheeses instead.
6. Eat plenty of fresh fruit and vegetables.

If you are also overweight you should cut down on starchy and sugary foods as well (see page 13).

Issued by the Scottish Health Education Group.

Printed in Scotland for HMSO by J. Swain & Son (Edinburgh) Ltd. Dd 8350545 10/80

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