males killed in accidents unconnected with flying. The coronary arteries reviewed have been sectioned transversely, one section being taken from left main, the circumflex and the right main artery and a subjective assessment of the degree of stenosis made. The incidence of CAD among 114 military aircrew, 41 professional pilots and 84 private pilots was determined. "Significant CAD" is taken to include Grade 2 or a greater degree of stenosis and is regarded as a degree of atherosclerosis that could be 'significant' in the context of a possible cause for pilot incapacitation. The occurrence percentage of significant asymptomatic coronary atherosclerosis is given in Table II.

Table II
Incidence of Coronary artery atherosclerosis in aircrew

Group	Mean age	Evidence of significant CAD
Military aircrew (114)	28.8 years	16.6%
Commercial pilots (41)	39.5 years	24.3%
Private pilots (84)	36.5 years	22.6%
Controls (111)	29.7 years	18.0%

There is no good evidence for a statistically significant difference in the incidence of CAD in the four groups. Mason² found that 45 (25 per cent) of his 180 aviators with an average age of 28.3 had Grade 2 or greater coronary atherosclerosis. There are 48 (19 per cent) Grade 2 or greater CAD in the three groups of 249 "aviators" in the present series. The observed difference in proportions between Mason's figures and the present series is not statistically significant. It is concluded that the incidence of CAD in the groups studied in this series has not shown any decrease in incidence over the last 15 years.

2 The Risk Factors

A REVIEW OF NUTRITIONAL RISK FACTORS

PROFESSOR J V G A DURNIN, MA, DSc, FRCP

The hypothesis is advanced that nutritional factors are an important component in the causation of coronary heart disease, but considerable reservation must be expressed about the quality of nutritional information provided in many of the studies relating to the aetiology of CHD. Whereas an assessment of the nutritional intake of large populations is practicable and feasible, it is not anything like so easy or so accurate for individual intake. A great deal of the difficulty in interpreting the exact role of nutrition in disease patterns stems from the problems of measuring nutritional intakes exactly.

There is a vast literature of clinical, pathological and animal experimental work to support the epidemiological findings that dietary factors are fundamental to the development of severe atherosclerosis, particularly those seen in affluent

countries where there is a high intake of energy and a high intake of both total and saturated fat. The studies on humans are paralleled and supported in large part by animal studies for it has been clear over the years that atherosclerosis may be produced in a large variety of species by feeding a diet high in cholesterol. If it is accepted that cholesterol levels in the blood are related to CHD and that in turn cholesterol levels are related to diet, it emerges that the dietary factors worthy of discussion in the aetiology of coronary heart disease are the relative importance of fats, salt, sugar, water, fibre, alcohol and possibly some of the trace elements.

Dietary fats

The elementary biochemistry of dietary fats is important. Almost all the fats in the diet consist of tri-glycerides, composed of one molecule of glycerol and 3 molecules of fatty acid. The fatty acids are of many different varieties but there are basically 3 kinds — saturated fatty acids which form the predominantly hard fats characteristic of most animal fats; simple monosaturated fatty acids such as those present in olive oil which seem to have no particular effect on blood cholesterol and therefore may be regarded as reasonably neutral; and the polyunsaturated fatty acids. These are found mostly in vegetable oils and have the biological property of being able to reduce levels of blood cholesterol. Cholesterol and triglycerides are attached to plasma proteins and circulate in the form of either high density lipoproteins (HDL) or low density lipoproteins (LDL). HDL is believed to transport cholesterol from the arterial wall and hence a high concentration may be desirable to slow down the progress of atherosclerosis, whereas LDL acts in the opposite way. Thus a high concentration of LDL is associated with a high coronary risk. Factors such as obesity, cigarette smoking and lack of exercise all tend to lower the HDL concentration. In a recent comparison of CHD among middle-aged men in Edinburgh and in Stockholm, Oliver and his colleagues demonstrated that the incidence of CHD was three times greater in Edinburgh than Stockholm. Edinburgh men had a much higher level of LDL and a lower concentration of HDL. The Edinburgh population also had a higher concentration of serum triglycerides and this was suggested by Oliver as being due to a greater hepatic production of very low density lipoproteins associated with a significantly higher alcohol consumption. He also demonstrated that the Edinburgh men had a lower linoleic acid content in their triglycerides and adipose tissues, suggesting that there may have been some important dietary component affecting the levels in Edinburgh by comparison with those in Stockholm. This work is an example of a good investigation minimally marred by the absence of a well controlled dietary investigation. It is unfortunate that there was no properly controlled nutritional study.

Blood cholesterol concentrations can be altered significantly by changing the amount and type of fat in the diet. When the fat content of the diet was reduced to 28 per cent of the total energy intake small but significant reductions in the serum cholesterol values were demonstrated. But 28 per cent fat is an extremely low level when compared with the intake of most population groups in the UK (eg housewives, office and shop workers, factory and industrial workers, farmers,

etc) where generally the proportion of energy supplied by fat in the diet is about 40 per cent. With this mean intake assuming a normal distribution, at least 20 per cent are taking more than 45 per cent of their energy from fats. However quite small falls in serum cholesterol have been associated with a significant diminution in the rate of attacks from CHD.

Despite the findings from the major studies where an induced fall of serum cholesterol has been associated with a reduced incidence of CHD there are a number of paradoxical instances of high dietary fat intakes which seem to contradict these general findings (eg studies of Trappist and Benedictine monks in the USA, experience from tribes in Northern parts of Kenya, and evidence from the Alaskan Eskimo). These studies should not be discounted but a very limited amount of credence should be placed on the information; first because very rarely has the diet been studied in an accurate fashion and second because the data on morbidity and mortality are often done on such small numbers that it is difficult to be sure of the relative importance of the factors involved. In summary the evidence suggests, from a multitude of different sources, that while there is some doubt about the significance of the total fat content of the diet there seems to be almost no doubt about the relevance to coronary heart disease of the intake of saturated fats from the diet.

Blood Pressure

In certain populations hypertension is as good a predictor of CHD as is blood cholesterol and there is considerable epidemiological and experimental evidence to suggest that the dietary intake of sodium may be of significance in the causation of hypertension, although genetic factors cannot be discounted in deciding the individual response to sodium intake. In this connection the role of soft water may play a part, because a low calcium intake may accentuate the development of hypertension by producing indirect effects due to the sodium. The significance of soft water in this context is that the differences in the intake of calcium between hard and soft water areas can be as much as 200 mg of calcium daily, a difference sufficient to alter the amount of sodium excreted in the urine and thus predispose to sodium retention. There is some evidence that other toxic elements in the water supply such as lead and cadmium which have a known role in the causation of atherosclerosis may also be of importance in the dietary context of CHD.

Sugar

Some years ago Yudkin suggested that sugar intake was more closely related to mortality from CHD than any other single nutrient. His findings have not been repeated and it is now generally agreed that the relative importance of sugar in relation to CHD is probably very low. It is often difficult to distinguish the effect of sucrose from that of fats because there is a high correlation in most diets between the intake of the two.

Dietary Fibre

Much attention and investigation is currently being directed to the importance of dietary fibre but it is difficult at this juncture to come to any firm conclusions. An interesting suggestion that has emerged from the studies on fibre is that benefit is confined to fibre coming from cereal and that the intake of fibre from fruits, vegetables, peas and beans has little relevance in the working of the protective mechanism.

In conclusion there seems no doubt that diet is one of the main contributory factors in the causation of CHD, but the impact of the message is spoiled by the absence of properly controlled acceptable scientific studies. All the evidence suggests that it is at least worthwhile to try and reduce the total intake of fat from the diet and particularly the total intake of saturated fats. These are measures, unlike other preventive measures, which cannot possibly do anybody any harm. Conversely they will almost certainly do many people a great deal of good in reducing obesity and may even help to prevent coronary heart disease.

SMOKING IN THE ARMY

BRIG J P CROWDY, MB, MFCM

Coronary heart disease is a present day major epidemic. It causes 34 per cent of all male deaths between the ages of 35 and 64. Smoking, and particularly cigarette smoking, is an important causative factor. The excess deaths among doctor smokers are 485 per 100,000, 152 of these deaths being due to coronary heart disease. For doctors aged under 45 the risk of smoking increases the prospect of mortality by 15. Studies among civil servants have shown that the death rate from CHD was increased by 64 per cent for moderate smokers and by 75 per cent for heavy smokers (20 or more cigarettes a day).

The effect of smoking seems to be slight where other risk factors, such as serum cholesterol, are low. The danger of smoking is accentuated as other risk factors increase. The mechanism of the action of smoking in increasing the risk from CHD is still largely unknown but it is likely that carbon monoxide both increases permeability to cholesterol and also deviates up to 15 per cent of haemoglobin with consequent loss of oxygen carriage potential. There is a possible role for nicotine which is known to increase the work of the heart and perhaps to play a role in the formation of atheromatous plaques.

Smoking Habits

National smoking habits have undergone very important changes in the last 20 years. Between 1958 and 1975 the prevalence of cigarette smoking among males of social class 1 fell from 54 per cent to 29 per cent. In the same time the prevalence among social class 5 remained virtually unchanged. In general, and for the first time since the introduction of machine made cigarettes the overall prevalence of smoking in the United Kingdom has fallen below 50 per cent (47 per cent in 1975). Smokers are thus in a significant national minority.