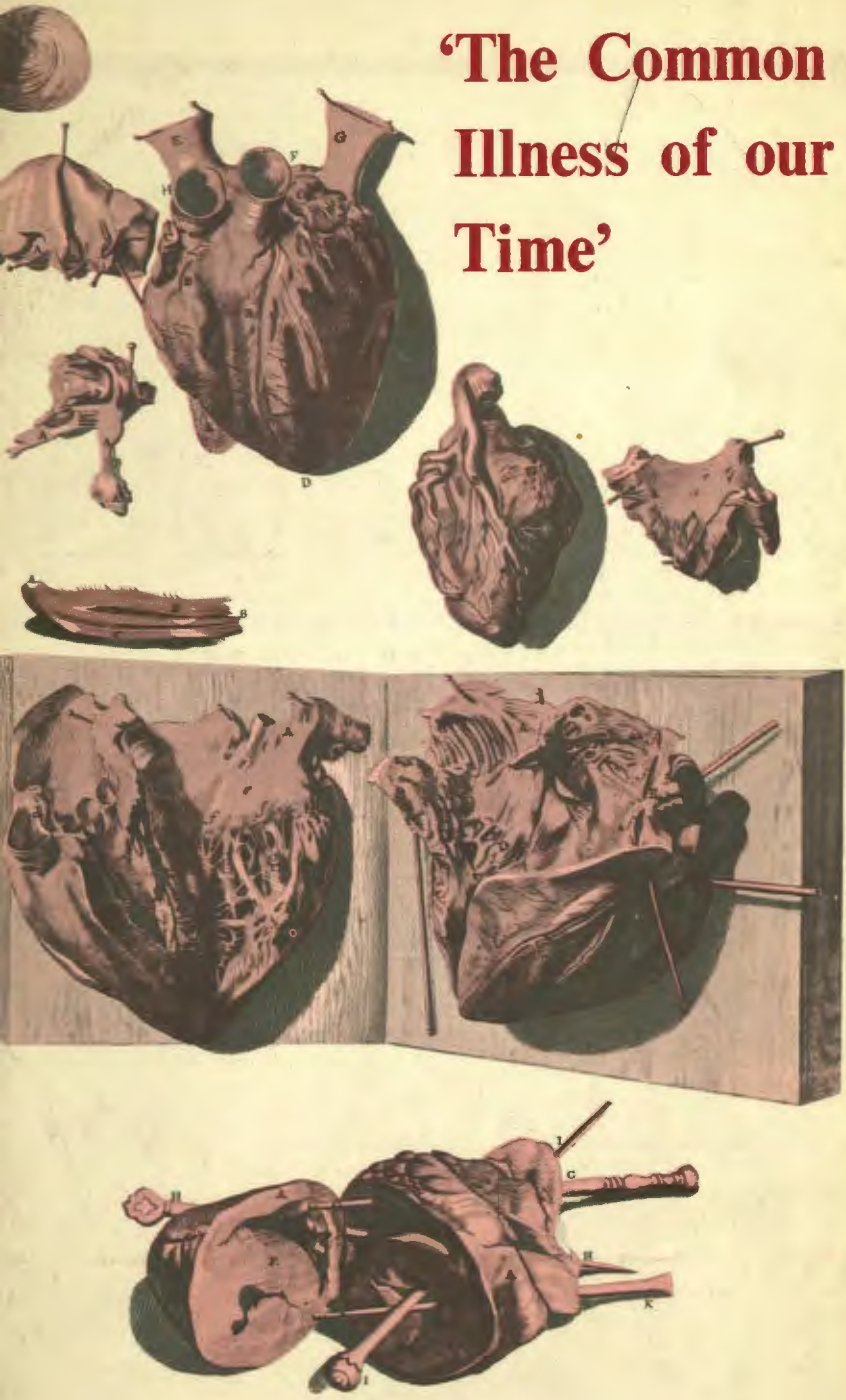


# 'The Common Illness of our Time'



Number 19 in a series of papers on current health problems published by the Office of Health Economics and sent without charge to selected readers. Additional copies are available at 2s. 6d. For previous papers see page 43.

© May 1966, Office of Health Economics

Cover illustration by courtesy of the Wellcome Historical Medical Library.

# 'The Common Illness of our Time'

---

*a study of the problem  
of ischaemic heart disease*

---

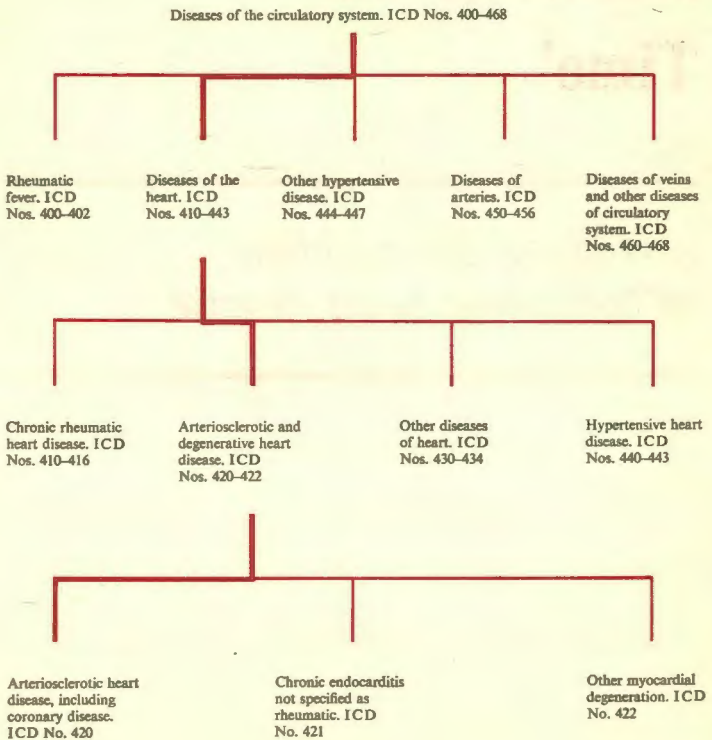


Office of Health Economics

62 Brompton Road London SW3

# Classification of diseases of the heart.

Source: Registrar General's Statistical Review of England and Wales. 1963. Part 1.



These categories and numbers are those used by the Registrar General and World Health Organisation in accordance with the International Statistical Classification of Diseases, Injuries, and Causes of Death (ICD), 1955—7th Revision. This paper is mostly concerned with ischaemic heart disease and will equate this to the ICD No. 420 'arteriosclerotic heart disease including coronary disease'. 'Diseases of the heart' is equated with ICD Nos. 410-443.

## 'The Common Illness of our Time'

In England and Wales in 1963, 187,023 deaths were certified as due to *diseases of the heart*. These included 154,815 deaths due to *arteriosclerotic and degenerative heart disease* of which 107,856 were due to *arteriosclerotic heart disease, including coronary disease*.

Figure 1 shows the standardised death rates both from all causes and from diseases of the heart during the present century. While the death rate from all causes fell substantially, the rate from heart disease since the 1930s has remained relatively constant. In the first quarter of the century deaths from diseases of the heart represented approximately one in eight of all deaths and by the 1960s this had risen to some one in three. (The overall heart disease rate conceals a slight rise for males over the last 25 years and a slight fall for females.)

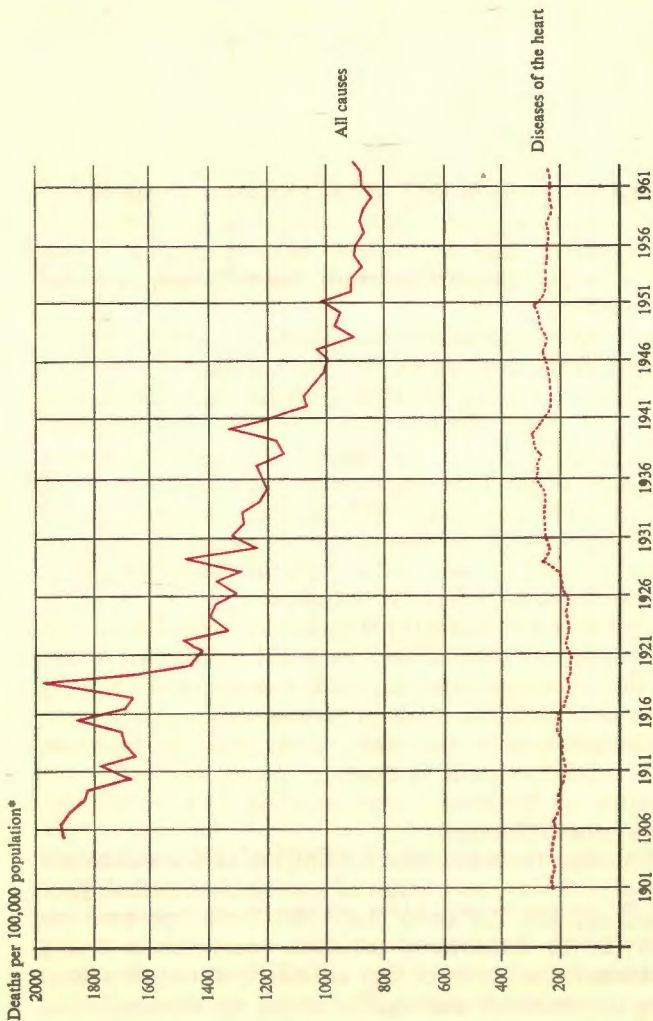
There are, however, serious limitations and pitfalls in examining the mortality and morbidity statistics for heart disease, and especially ischaemic heart disease, even within a country at one point in time. The difficulties are greatly increased when making comparisons for different times or between different countries. The difficulties arise in two ways; firstly from the technical progress which has made it easier to detect and define the exact nature of the disease, and secondly from consequent changes in nomenclature.

The first steps towards understanding the nature and causes of heart disease came as a result of post mortem pathological investigations, and for some time past there has been no ambiguity in the detection of ischaemic heart disease during such examinations. However they are clearly not applicable in assessing the incidence and significance of the disease among the living and they are not generally performed even to confirm

# 1

## Mortality from all causes and from diseases of the heart. All persons. England and Wales. 1901-1963.

Source: Derived from Registrar General's Statistical Report of England and Wales. 1948-49 Text volume. 1956 Part 1. 1963 Part 1.



• Adjusted to 1938 population.

the cause of death. Progress towards accurate clinical diagnosis therefore had to await the development of the electrocardiograph. Only since its introduction and with other improvements in technique has clinical diagnosis, in advanced countries at any rate, been possible on a sound and uniform basis. Even now, accurate classification without a post mortem or precise clinical history may still not be easy. For instance, among the elderly, chronic bronchitis with myocardial degeneration is a common cause of death. Such deaths will be recorded as due either to bronchitis or heart disease depending on the judgement of the doctor. Furthermore, if the cause is diagnosed as heart disease a decision must sometimes be made whether to record the death under *arteriosclerotic heart disease, including coronary disease* or one of the other headings. It has been shown that even the interpretation of electrocardiograph readings is to some extent subjective, and the opinions of different specialists can vary.

Nomenclature used to describe ischaemic heart disease has been, and often still is, varied. As detection of the disease becomes more exact so the terms used become more precise. Infarction, thrombosis and occlusion were originally pathological terms but, with improvements in the accuracy of diagnosis, are now often used in clinical medicine. Atheroma, atherosclerosis, arteriosclerosis and degenerative heart disease are all terms used in both pathology and clinical medicine.\* Although even now they are not always used correctly by clinicians the official morbidity and mortality statistics often depend on them. As statistics concerning the disease ultimately depend on descriptions used by doctors, they are not always uniform over time or throughout the world; hence the difficulties in interpreting them. Because of all these variations it is important when discussing ischaemic heart disease to understand its nature, and the terms which are used to describe it.

### *The Nature of Arterial Disease*

Progressive changes occur in the arteries as they age. For instance, the calcium content of the arteries rises gradually and this is associated with a progressive loss of elasticity. But, in addition to these changes, there is a patchy degeneration made up of arterial plaques which are either of the nature of fatty

\*Some of these terms are described in the next section, and all are defined in Appendix A.

streaks or raised lesions. This condition is found to some extent in most persons from youth onwards although the rate of development varies. It is known as atheroma or atherosclerosis. As this process goes on, the lumen, or passageway of the artery, begins to narrow and, if the artery is small, the blood flow is reduced. When the coronary arteries, which supply the heart itself with blood, narrow in this way the blood supply to the heart is reduced, the heart becomes less efficient and as parts of the heart muscle become increasingly short of oxygen they degenerate.

The narrowing of the artery, together with destruction of the lining of the artery over the surface of a plaque, frequently causes a thrombus, or clot, to form, thus suddenly occluding the artery. If this occurs in a coronary artery the heart muscle supplied by it is starved and damaged, causing a myocardial infarction. It is properly recorded under *arteriosclerotic heart disease, including coronary disease* on health documents.

However, in some cases no such sudden and dramatic event occurs, instead there is a gradual and progressive narrowing of the coronary artery so that the blood supply to the muscle is slowly cut off. This can lead eventually to angina pectoris and may lead to congestive heart failure. Angina pectoris is a symptom rather than a disease, but where it is diagnosed as a cause of death it, too, is properly recorded under *arteriosclerotic heart disease, including coronary disease*. If neither myocardial infarction nor angina is diagnosed but death is considered to be due to a reduced blood flow to the heart, the cause is usually recorded as *other myocardial degeneration*. However myocardial degeneration is often caused by myocardial infarction, and as this fact is recognised the non-specific diagnosis is becoming less common.

Thus, coronary disease involves both the formation of atheroma in the coronary arteries, and also ischaemic heart disease (particularly the syndrome of thrombosis, occlusion and infarction). Coronary atheroma is the early stage, which cannot yet be measured clinically by diagnostic techniques. As the disease advances it is recognised in life at the stage when it gives rise to symptoms or alterations in the pattern of electrical impulses from the heart muscle, as recorded by an electrocardiograph. It is then termed coronary or ischaemic heart disease.\*

\*Ischaemic heart disease is the more accurate term and will be used throughout this paper wherever applicable. Where official statistics and the results of medical enquiries are discussed the terms used will generally be those of the original source.



## The Trends

When changes in the age structure of the population have been taken into account, total mortality from *diseases of the heart* has changed little since the early 1930s (Fig. 1); but mortality ascribed to diseases of the coronary arteries has risen spectacularly. Figure 2 shows the death rate for males and females in four parts. Prior to 1927 there was a rubric *angina pectoris* but in 1927 the Registrar General decided that many deaths which had previously been assigned to general arterial disease should be assigned to angina pectoris and from 1928 a new rubric *diseases of the coronary arteries, angina pectoris* was formed. This change was probably associated with the introduction of the electrocardiograph at that time, which not only improved the accuracy of diagnosis but indicated the position and size of the infarction. In 1940 the description was changed to *arteriosclerotic heart disease, including coronary disease* and in 1957 some minor adjustments were made. It can be seen from Figure 2 that there has been a substantial rise in the rates from 1928 onwards. Although the rates are increasing is much less certain whether the disease itself is increasing at the same pace, for better diagnostic methods and an increased realisation of the existence of the disease may contribute to the increase in rates. In particular, the trends suggest that there may have been a considerable shift in diagnosis from the category *other myocardial degeneration* to *arteriosclerotic heart disease, including coronary disease*. Table A shows the standardised mortality ratios (based on the years 1950-52 as 100) from 1949 to 1963 for men and women for these two categories, together with all diseases of the circulatory system.

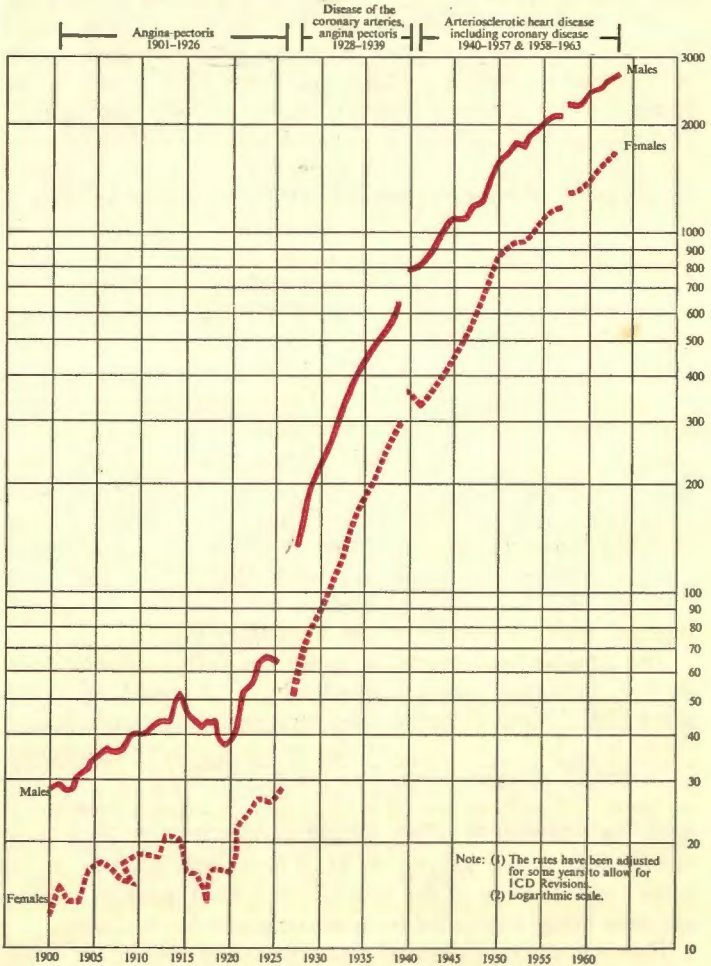
The standardised mortality ratios for *arteriosclerotic heart disease, including coronary disease* have increased by a half since 1950-52 and those for *other myocardial degeneration* have fallen by a half. This may be due to more accurate diagnosis. Myocardial degeneration was probably used to describe coronary thrombosis which is now usually being diagnosed as such. In addition it seems almost certain that at least some deaths that would previously have been attributed to causes other than diseases of the circulatory system, such as senility, are now being attributed to arteriosclerotic heart disease.

However the standardised mortality ratios conceal the extent of changes in the actual rates for individual age groups. Table B compares the death rates by sex at ages 45 to 64 from

## 2

Crude death rate per million living from angina pectoris; diseases of the coronary arteries, angina pectoris; arterio-sclerotic heart disease including coronary disease, by sex. England and Wales. 1901-1963.

Source: Registrar General's Statistical Review of England and Wales. Various years.



*arteriosclerotic heart disease, including coronary disease and other myocardial degeneration* in 1953, 1958 and 1963. In this age group, particularly for men, the cross-compensation is not complete. This evidence is confined to the last ten years but a real increase in pathologically confirmed coronary heart disease over the last fifty years has been reported.<sup>1</sup> An analysis of the necropsy records of the London Hospital showed about a sevenfold increase from 1907-14 to 1944-49 in the number of cases of coronary heart disease amongst persons aged 35 to 70.

**A** Standardised mortality ratios\* (1950-52=100) for arteriosclerotic heart disease including coronary disease, other myocardial degeneration and for all diseases of the circulatory system, by sex. England and Wales, 1949-1963.

Source: Registrar General's statistical review of England and Wales. 1958 Part 1. 1963 Part 1.

	MALES			FEMALES		
	<i>Arterio-sclerotic heart disease, including coronary disease</i>	<i>Other myocardial degeneration</i>	<i>Diseases of the circulatory system†</i>	<i>Arterio-sclerotic heart disease, including coronary disease</i>	<i>Other myocardial degeneration</i>	<i>Diseases of the circulatory system†</i>
1949	82	113	96	84	112	103
1950	94	102	98	96	104	101
1951	101	108	104	100	109	106
1952	105	90	97	103	88	93
1953	104	84	95	103	85	94
1954	112	80	97	108	79	91
1955	116	79	98	115	78	93
1956	121	75	99	119	74	92
1957	122	65	95	119	64	88
1958	129	65	98	129	66	90
1959	128	57	94	130	60	89
1960	137	53	96	138	57	87
1961	140	52	98	144	56	90
1962	149	49	100	151	53	89
1963	156	48	103	158	51	91

\*The standardised mortality ratio shows the number of deaths registered in the year of experience as a percentage of those which would have been expected in that year had the sex/age mortality of a standard period (1950-52) operated on the sex/age population of the year of experience.

†Includes *arteriosclerotic heart disease, including coronary disease and other myocardial degeneration* plus other rubrics. See *Classification of disease of the heart*, p.2.

## B

Death rate per million living at ages 45 to 64 from arteriosclerotic heart disease, including coronary disease and other myocardial degeneration, by sex. England and Wales. 1953, 1958 and 1963.

Source: Registrar General's Statistical Review of England and Wales 1953, 1958, 1963. Part I.

	MALES			FEMALES		
	1953	1958	1963	1953	1958	1963
Arteriosclerotic heart disease, including coronary disease	2712	3457	4333	731	876	1049
Chronic endocarditis and other myocardial degeneration	500	315	247	359	196	147
Total	3212	3772	4580	1090	1072	1196

These figures relate to acute coronary thrombosis and chronic coronary occlusion, and with them ischaemic heart disease. No increase, however, was found, for atheroma of the artery walls. Other authorities have also questioned whether the pathological condition does in fact now occur more commonly.

It seems probable that there has been a real increase in death rates from ischaemic heart disease. The increase has been greater among the younger age groups but it is likely that the rate of increase has been levelling off over the last few years. It is also probable that the increase is not as large as the statistics suggest. Until recently evidence concerning the incidence of the disease has necessarily been somewhat inexact; only now are facts emerging. Nevertheless, whatever the trends, the number of people dying from heart disease presents a formidable problem.

### *Current Patterns of Heart Disease*

#### SEX AND AGE MORTALITY DIFFERENCE

In 1963 nearly seventy per cent of deaths from arteriosclerotic heart disease, including coronary disease, occurred in persons over the age of 65. Table C shows the age distributions for deaths from this cause. There are wide differences for the sexes which are due in part to their differing age structure. Forty per

# C

## Deaths from arteriosclerotic heart disease, including coronary disease, by sex and age. England and Wales, 1963.

Source: Registrar General's Statistical Review of England and Wales, 1963, Part 1.

	MALES		FEMALES		PERSONS	
	Number of deaths	%	Number of deaths	%	Number of deaths	%
Under 45	2007	3	290	1	2297	2
45-54	6981	11	1136	3	8117	8
55-64	17,515	26	5286	12	22,801	21
65-74	21,245	32	13,761	33	35,006	32
75 and over	18,092	28	21,543	51	39,635	37
All Ages	65,840	100	42,016	100	107,856	100

cent of men who died were aged under 65 compared with only sixteen per cent of women.

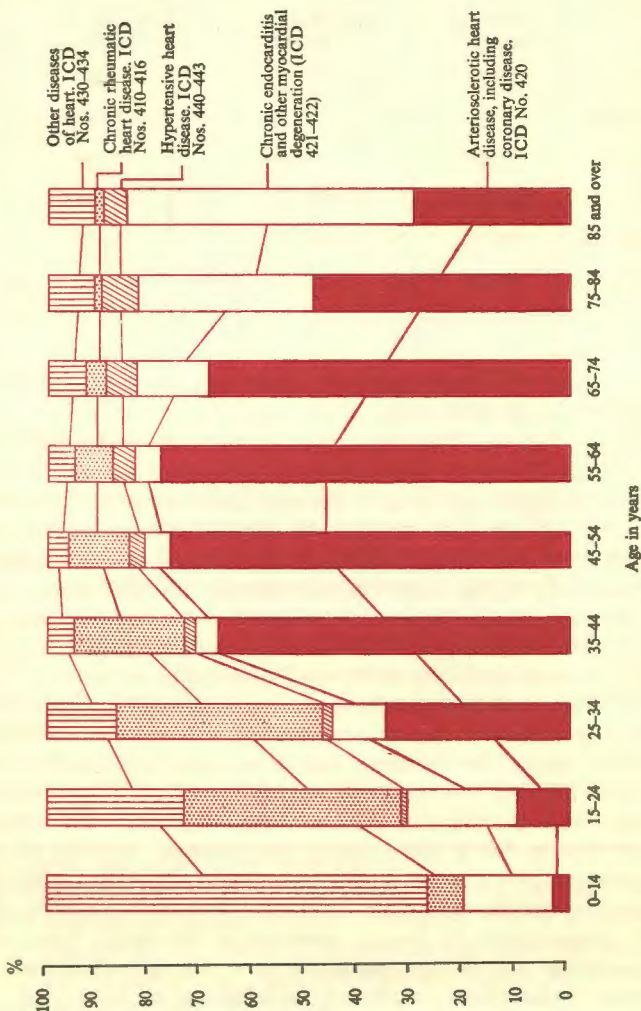
Table D shows death rates, and the sex differences are seen even more clearly. It also shows, by age group, the number of deaths assigned to *arteriosclerotic heart disease, including coronary disease* as a proportion of deaths from all causes. Death rates rise steeply with age. The overall rate for men is substantially higher than that for women and the death rates for men are roughly equivalent to those of women who are ten years older. This suggests that the same pathological processes occur in both sexes but more rapidly in men.

For men of all ages, deaths from *arteriosclerotic heart disease, including coronary disease* accounted for 23 per cent of those from all causes; for women it was 13 per cent. It reached a peak for men between the ages of 45 and 54 for whom 31 per cent of deaths were due to this disease. This, together with a more rapid rise in death rates among the younger sections of the population over the last ten years, explains the present concern regarding middle-aged men. No doubt the proportion falls with age groups over 54 largely because of the more than corresponding rise in the proportion of deaths attributed to *chronic endocarditis and other myocardial degeneration*. (Fig. 3.)

### 3

Mortality from the five groups of heart disease by age, expressed as percentage of total mortality from diseases of heart (ICD Nos. 410-443) for each age group. All persons. England and Wales. 1963.

Source: Registrar General's Statistical Review of England and Wales 1963. Part I.



# D

Death rates per million living from arteriosclerotic heart disease including coronary disease, by sex and age, and proportion of deaths from this disease to deaths from all causes. England and Wales, 1963.

Source: Registrar General's Statistical Review of England and Wales, 1963, Part I.

Age	MALES		FEMALES	
	Death Rate	% of all deaths	Death Rate	% of all deaths
0- 1	-	-	2	0
1- 4	-	-	1	0
5-14	-	-	-	-
15-24	4	0	1	0
25-34	59	5	10	1
35-44	563	23	80	4
45-54	2312	31	359	8
55-64	6646	30	1786	16
65-74	14,725	26	6397	21
75-84	25,121	19	14,533	16
85+	25,058	13	23,493	11
All Ages	2845	23	1737	13

(Note: 0 denotes less than 0.5%)

## GEOGRAPHICAL MORTALITY DIFFERENCE

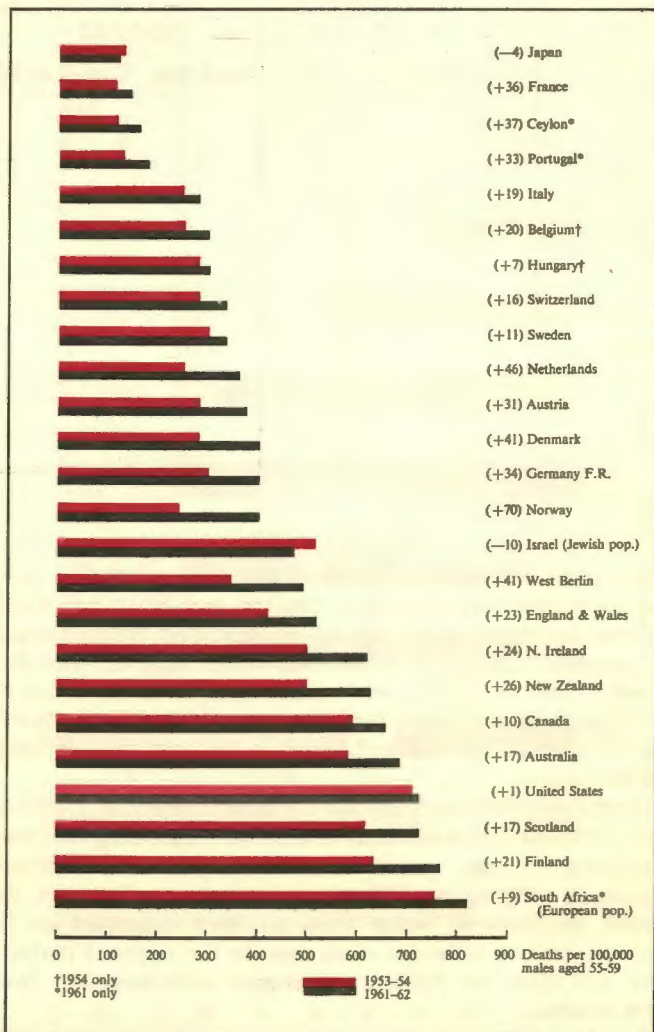
There is a clear geographical variation in mortality from arteriosclerotic heart disease in England and Wales and Table E shows the deviations from the England and Wales average for mortality from *arteriosclerotic heart disease, including coronary disease*, for men and women aged 45 to 64. It can be seen that deaths attributed to this cause are highest in the North and the West of England and lowest in the East, the Midlands and the South.

There is no simple explanation for these differences. Different habits of certification of death or more accurate diagnosis may contribute to the variation. Socio-economic conditions, population migration, differences in diet, weather and the relative hardness of water have all been suggested as influencing factors, although in no case do the regional patterns show any clear cut positive correlation with mortality from heart disease.

# 4

Deaths from arteriosclerotic and degenerative heart disease (ICD Nos. 420-422) per 100,000 males aged 55 to 59. Twenty-five selected countries. 1953-54 and 1961-62. (Figures in brackets denote percentage variation 1953-54 to 1961-62.)

Source: Epidemiological and Vital Statistics Report of WHO Vol. 15 No. 2. 1962. Annual Epidemiological and Vital Statistics Report of WHO for 1961 and 1962.





# E

Geographical variation in arteriosclerotic heart disease, including coronary disease, average death rates per thousand living and deviation from average, by sex at ages 45 to 64. England and Wales, 1959-61.

Source: Registrar General's Statistical Review of England and Wales, 1961, Part III.

	MALES	FEMALES
England and Wales Average 1959-61	3.65	0.91
REGIONS	DEVIATION FROM AVERAGE	
North Western	+0.70	+0.20
Wales	+0.56	+0.16
Northern	+0.55	+0.38
East and West Ridings	+0.41	+0.16
London and South Eastern	-0.14	-0.15
Southern	-0.35	-0.18
South Western	-0.36	-0.06
Midland	-0.40	-0.08
North Midland	-0.43	-0.11
Eastern	-0.72	-0.14

The differences found internationally are even greater. Figure 4 shows the death rates from *arteriosclerotic and degenerative heart disease* per 100,000 males aged 55 to 59 in 25 selected countries of the world. Differences in certification habits and the different patterns and standards of medical care may account for a large part of the variations. How much no one can say. To quote Allbutt, writing nearly fifty years ago, 'estimates of frequency in this region of the earth . . . depend much on the diligence, opportunities and bias of the observer. It is said to be more frequent in the United States . . . but for some time past the Americans have been closely watching their arteries'.<sup>2</sup> Figure 4 shows that the rates are highest in South Africa (European population), Finland, Scotland and the United States and lowest in France and Japan. The diagram also compares the latest available figures (1961-62) with those for 1953-54 and in 23 out of the 25 selected countries there was an increase in the rate between the two periods. Norway showed a 70 per cent increase, and the Netherlands 46 per cent. In England and Wales the rate increased 23 per cent. Only Israel and Japan showed a decrease.

## *Morbidity Patterns*

As the development of atheroma in the coronary arteries is a continuous process starting in most individuals from childhood or early adolescence and steadily progressing, both in incidence and degree, with advancing age, there is no sharp distinction in pathological terms between healthy and diseased arteries. However there is a large variation between individuals in the amount of atheromatous change. When the pathological process has reached a certain stage, clinical symptoms manifest themselves causing illness or death. The majority of men over 65 have some atheroma of the walls of the coronary arteries but only a minority have occlusive coronary disease.

Thus morbidity statistics, as recorded by information obtained from hospitals, consultations with general practitioners, and absence from work, can only give a measure of the prevalence, in pathological terms, of the advanced stages of coronary disease.

### **HOSPITAL INFORMATION**

Figure 5 shows the estimated number of discharges or deaths from *arteriosclerotic heart disease, including coronary disease*, in hospitals in England and Wales for the years 1953 to 1964; as would be expected from mortality patterns, there is a sharp increase. 73,330 persons were discharged from (or died in) hospital in 1961, the latest official figure available. Figure 5 also shows the discharge rates by sex and age for the years 1957 to 1961; the increases occur in all groups.

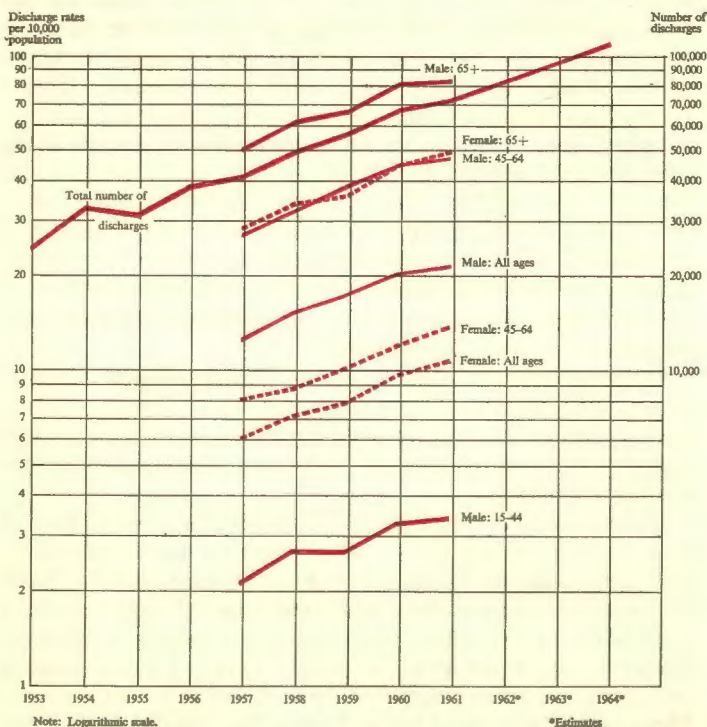
Morbidity statistics as well as mortality statistics are subject to changing diagnostic patterns; but unlike mortality figures they, as measured by hospital records, are also dependent on treatment and hospital admission policies. The figures do not, then, necessarily imply differences or changes in prevalence of the disease.

The latest available Hospital In-Patient Enquiry shows a remarkable difference of discharge rates between regions.<sup>3</sup> For example the discharge rate for men aged 65 or over was 125·3 per 10,000 population for the Liverpool Region and only 51·5 per 10,000 in the Sheffield Region. However, in contrast to regional differences in mortality, there was no general preponderance of increased discharges in the north of the country. This suggests that these rates are associated with local hospital factors, such as bed availability, rather than with the number of cases showing overt signs of the disease in an area.

# 5

Discharges and deaths from arteriosclerotic heart disease, including coronary disease. Hospitals in England and Wales. Total number (1953-1964) and rates per 10,000 of population by sex and age (1957-1961).

Source: Ministry of Health and General Registrar Office Report on Hospital In-patient Enquiry. Various years.



The majority of the persons discharged (81 per cent in 1959) had been cared for in general medical beds and less than four per cent in the cardiology department of a hospital. On average, each person remained in hospital for 33 days in 1961.

## CONSULTATIONS WITH GENERAL PRACTITIONERS

It was estimated that in 1955-56 there were some 320,000 patients who consulted their doctor and were diagnosed as having coronary artery disease (roughly equivalent to *arteriosclerotic heart disease, including coronary disease*) in England

and Wales.<sup>4</sup> One in fifty men aged 45 to 64 and one in twenty-five men aged 65 and over consulted for this disease. For *arteriosclerotic and degenerative heart disease* the estimate was about 500,000. An OHE estimate\* for 1963 gives approximately 440,000 patients described as having coronary artery disease and 570,000 as *arteriosclerotic and degenerative heart disease*.

#### SICKNESS ABSENCE STATISTICS

Sickness absence statistics only cover claims made by the working population insured against sickness with the Ministry of Pensions and National Insurance. In 1963-64 in Great Britain some 14,430,000 days were lost through sickness arising from *arteriosclerotic and degenerative heart disease* (it is not possible to obtain information pertaining specifically to the coronary arteries) and 33,010,000 days through all diseases of the circulatory system. Figure 6 shows the standardised rates (per 1000 insured population standardised to equivalent 1951 population) of spells commencing and days lost from *arteriosclerotic and degenerative heart disease* for the years 1953-54 to 1963-64. Again there is a rise in the male rate, although the female rate is relatively constant.

From the sickness absence figures it is possible to estimate that in 1963-64 some 279,000 men and 70,000 women claimed sickness benefit for *diseases of the circulatory system* and 97,000 men and 9000 women claimed for *arteriosclerotic and degenerative heart disease*.

Summarising, in England and Wales during the course of a year probably nearly half a million persons are diagnosed by their doctor as having ischaemic heart disease; nearly 75,000 of the working population have some time off; approximately 100,000 of the population as a whole are admitted to hospital; and over 100,000 die from this disease. (These are not exclusive categories and many people will be included in more than one.) These are persons recorded as having the disease. In addition many have advanced disease of the coronary arteries, unknown to their doctors. They may suddenly develop overt clinical signs or die. It has been estimated that for each man aged 45 to 65 seen by his doctor for ischaemic heart disease there are approximately three who are not seen but who could be detected as suffering from this condition on survey.<sup>5</sup>

\*This estimate is an average of figures from two sources. First the Intercontinental Medical Statistics Ltd figures of diagnosis in 1962 projected up to 1963 and adjusted to patients rather than diagnosis. Second the 1955-56 proportion of patients consulting to deaths in the year projected to 1963 assuming this proportion unchanged.

## 6

Arteriosclerotic and degenerative heart disease. Working days lost and spells commencing per 1000 insured population (of equivalent age structure of 1951 population), by sex. Great Britain 1953-54 to 1963-64.

Source: Ministry of Pensions and National Insurance. Reports for the years 1962, 1963, 1964.



## *Factors Involved In Coronary Heart Disease*

At a recent WHO conference on the prevention and control of cardiovascular diseases a group of at least eight factors were deemed to be significant in coronary artery disease: hyperlipaemia (high blood lipid levels\*), hypertension, cigarette smoking, physical inactivity, increase in weight, nervous stress, diabetes mellitus and genetic factors.<sup>6</sup> Other factors which appear to influence the pattern of the disease include diet, sex difference, blood clotting, hardness of water supplies and anthropometric differences.

In recent years there has been much speculation on the role played by each of these factors, and their relative importance. Any discussion of the subject is complicated by the difficulty in separating individual factors which are related to each other or may have a causal relationship to coronary artery disease. For example, the obese are more prone to diabetes; those under stress are often those who take little exercise; racial and genetic differences are often associated with differences in diet; obesity, exercise and diet are themselves all inter-related. Thus when examining the correlation between heart disease and any single factor, it is almost inevitable that other factors will interfere with the conclusions.†

### **OBESITY, BLOOD CHOLESTEROL AND DIET**

One group of related factors whose apparently causal relationship to ischaemic heart disease has a practical significance consists of obesity, blood cholesterol\* and diet.

There is certainly evidence to suggest that over-weight persons carry an increased risk of death not only from ischaemic heart disease, but also from many other causes. A study carried out amongst 39 to 55 year old men in Albany, USA, showed that those whose weight was more than 40 per cent above the Metropolitan Life Insurance Company's 'standard weight' had three times as great a risk of ischaemic heart

\*See Appendix A for definition.

†The Croonian lectures of 1849 delivered by Dr John Conolly provide an ironic illustration of the pitfalls in discussing the causes of a disease before they are properly understood. Concerning the causes of general paralysis of the insane—before, of course, the causal relationship of syphilis was recognised—he said, 'I have lately, whilst preparing these lectures, referred to the particulars of the 146 cases of general paralysis in men, of which all the subjects have died in the Hanwell Asylum within the last ten years. The causes were ascertained with tolerable certainty in ninety-six cases only out of this number. In sixty of these cases the malady was ascribed to what we term a moral cause; to losses, anxiety, grief, domestic unhappiness, disappointments, poverty, reverses, etc. Intemperance, singly, was assigned as a cause in twenty cases only; but in fifteen other cases of the sixty just mentioned, as a cause in combination with losses, grief, etc. The other causes mentioned, each in one or two cases only, are fever, injury of the head, hot climate, exposure to wet and cold, hereditary disposition, abuse of mercury, sensual excesses, foul air.'

disease as the population as a whole.<sup>7</sup> Lesser degrees of overweight involved relatively little increase in risk. Another prospective epidemiological survey of arteriosclerotic heart disease and hypertension which was set up at Framingham in 1950 produced similar results<sup>8</sup> (Fig. 7). However when the effect of hypertension and serum cholesterol levels were taken into account, overweight in the absence of these related causes seemed to be relatively unimportant. Other studies have shown that 'skin-fold thickness', which measures the extent of subcutaneous fat, gives a more significant correlation with ischaemic heart disease than does body weight, so that it may be 'fatness' rather than weight which is the important factor. The Albany study also suggested that it was an increase in weight rather than overweight in itself which was causally related to ischaemic heart disease.

Probably, of all the possible causative agents in ischaemic heart disease, serum cholesterol and fat in the diet have attracted most attention in recent years. From observations indicating that dietary fat, serum cholesterol levels and coronary disease are positively correlated, it has been suggested that a high fat diet causes high cholesterol levels which in turn increase the risk of developing ischaemic heart disease.

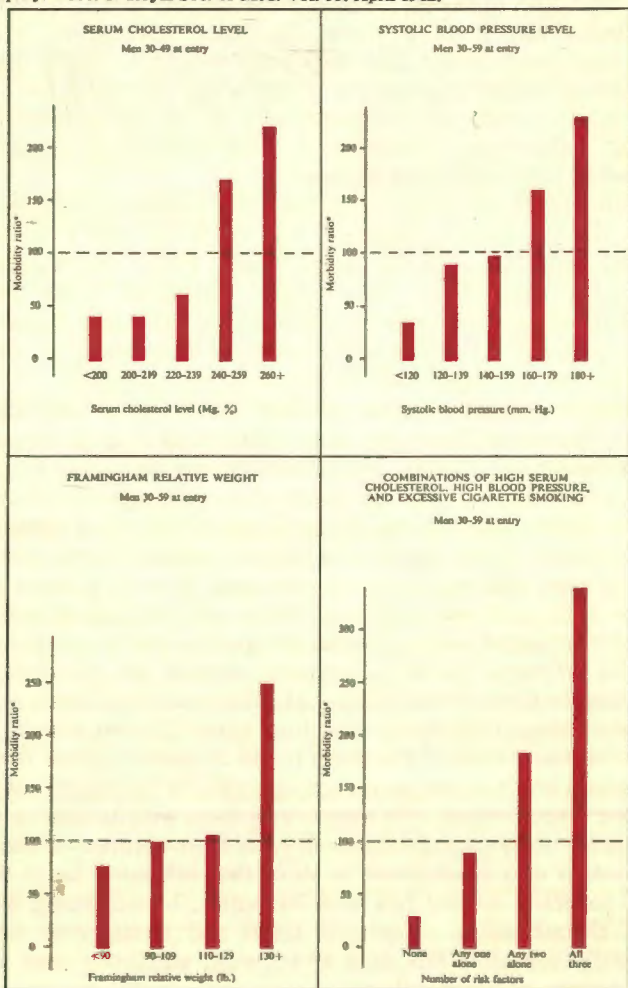
There is definite evidence to show that persons with high serum cholesterol levels are more liable to develop ischaemic heart disease than others. The Albany prospective study found that ischaemic heart disease occurred more frequently as the serum cholesterol level rose. Figure 7, taken from the Framingham enquiry, shows that the risk of developing ischaemic heart disease after eight years among men aged 30 to 49 at entry to the enquiry increased fivefold for those with the highest initial serum cholesterol levels. Another prospective study carried out by the Western Electric Company showed an association between the development of clinical ischaemic heart disease and elevated blood cholesterol after four years.<sup>9</sup> It also showed a stepwise association of the mean blood cholesterol levels from the lowest levels in men as yet not identified as having coronary disease through those with angina and those with an infarct, to the highest level amongst those who died from coronary disease.

There is also much work to show that saturated fat in the diet, generally animal fats such as butter, beef-dripping and lard, elevate serum cholesterol levels and unsaturated fats, generally vegetable fats such as corn-oil, sunflower seed oil and peanut oil depress them.

# 7

Risk of developing coronary heart disease in eight years according to: (a) *initial serum cholesterol level*; (b) *initial systolic blood pressure level*; (c) *Framingham relative weight*; (d) *Combinations of high serum cholesterol, high blood pressure and excessive cigarette smoking*. Results of Framingham Enquiry.

Source: Dawber *et al* 'The epidemiology of coronary heart disease. The Framingham Enquiry.' Proc. of Royal Soc. of Med. Vol. 55. April 1962.



\*Morbidity ratio is the ratio of observed cases to expected cases x 100



In addition, extensive international comparisons indicate that for whole populations there is a positive relationship between the intake of dietary fat with serum cholesterol levels and with the incidence of ischaemic heart disease. In populations where the intake of fat is really low, low levels of coronary disease have been found, for example, the South African Bantu, the Chimbu of New Guinea and the Japanese.\*

This relationship is, however, based on indirect evidence. Equally good relationships exist between dietary protein, income, motor cars, and purchasing power and the incidence of ischaemic heart disease. It has been suggested for example that national levels of the consumption of fat and sugar may be closely related and that fat may be an indirect factor whereas sugar may be causal.

Direct evidence as obtained from a study on 99 bank clerks aged 40 to 55, whose diets were carefully recorded and whose cholesterol was measured, yielded no evidence of any sizeable association between what these men ate and their cholesterol level.<sup>10</sup> The study showed a wide range in food intake between the men and also a wide range of cholesterol levels but these were not related to each other. They all had a relatively high consumption of saturated fat and nearly all a high cholesterol level. This suggests either that dietary fat has no direct causal relationship with ischaemic heart disease or that it is only below a certain level of fat intake that there is a direct relationship with mean cholesterol levels and ischaemic heart disease; above this level of fat intake no simple effect on cholesterol level is exerted and it is suggested that this level obtains in many western communities including the United States and Britain. Thus it is possible that a lifelong diet in a community might shift the average cholesterol values upwards, leaving other factors to produce wide individual variation in cholesterol values and to affect the risk of developing ischaemic heart disease in individuals.

In summary, therefore, it appears that the amount of fat (and probably carbohydrates and protein) in the diet of a community affects the average serum cholesterol level, and also the incidence of ischaemic heart disease. In advanced countries, however, although some correlation between individual serum cholesterol levels and the incidence of ischaemic heart disease

\*Against this general picture, however, it has been suggested that Mongolian cattle breeders, Somali tribesmen and Eskimos all live on diets rich in animal fats, without suffering from a high incidence of ischaemic heart disease.

still persists, there is little evidence of a causal relationship in individuals between diet and serum cholesterol levels.

Thus even reducing the serum cholesterol, for example by diet, in those who have naturally high levels may not necessarily reduce their risk of coronary artery disease. Research to establish whether this is so has only recently been started and necessarily involves very long-term prospective studies.

Opinion in Britain seems to be moving towards a position where normal consumption of fat or sugar are considered unlikely to have a direct causal relationship with ischaemic heart disease amongst the populations of advanced countries.\* It is nevertheless true that any over-eating can lead to obesity with general higher rates of sickness and perhaps of heart disease.

### **HYPERTENSION AND DIABETES**

As with obesity, raised blood pressure is associated with a rise in mortality generally. The American Society of Actuaries found that even a blood pressure of 150/90mm. (little above average) doubled the death rate from coronary artery disease among men under 50.<sup>11</sup> From the Framingham survey, it was found that both for males and females there was a striking increase in risk of ischaemic heart disease with increasing levels of blood pressure. For men aged 30 to 59 on entry into the survey, the risk of developing ischaemic heart disease after eight years rose from a morbidity ratio of 36 for those with an initial systolic blood pressure of less than 120mm. to 227 for those with a pressure of over 180mm. (Fig. 7.)†

Similarly, in the case of diabetes, a study in Bedford found that the age adjusted prevalence of arterial disease was lowest in a control group of persons with normal blood sugar, intermediate in persons with 'borderline' hyperglycaemia and highest among the diabetics.<sup>12</sup> The study concluded that symptomless impairment of glucose tolerance may be an important accompaniment of arteriosclerotic disease but awaits further evidence as to its causal relationship. Nor is it yet clear whether the control of hyperglycaemia necessarily reduces the risk of vascular disease.

\*In the United States, however, the American Heart Association have taken a different view, they still incriminate diets rich in animal fats.

†This study also showed that men with a combination of three 'high risk' factors, high serum cholesterol, high blood pressure and excessive cigarette smoking, had a risk of developing coronary heart disease ten times greater than men without any of these 'high risk' factors present (Fig. 7).

Controversy still rages over the factors which cause both hypertension and diabetes. Until this is resolved, it will not be known whether it is these diseases, or their underlying causes, which determine the probability of developing heart disease.

### **SMOKING**

In several investigations, in both Britain and the United States, an association between smoking habits and deaths from ischaemic heart disease has been shown. Figure 8 shows that the mortality ratios due to coronary artery disease for cigarette smokers, with non-smokers taken as unity, rose to 2.41 for heavy smokers. The figures are based on an American survey of 187,783 men over the years 1952–55. For this survey, as with those carried out in Britain, the association was with cigarettes rather than pipes or cigars.

It is unquestionable that there is a relationship between cigarette smoking and mortality from ischaemic heart disease. It has, however, been suggested that there might be some predisposing factors (perhaps stress) which make the same people liable both to become cigarette smokers and to be prone to heart disease. However, whether this is so or not, a prospective study among doctors has shown that those who give up cigarette smoking have a lower risk of premature death from ischaemic heart disease than those who do not.<sup>13</sup>

### **OCCUPATION, PHYSICAL ACTIVITY AND STRESS**

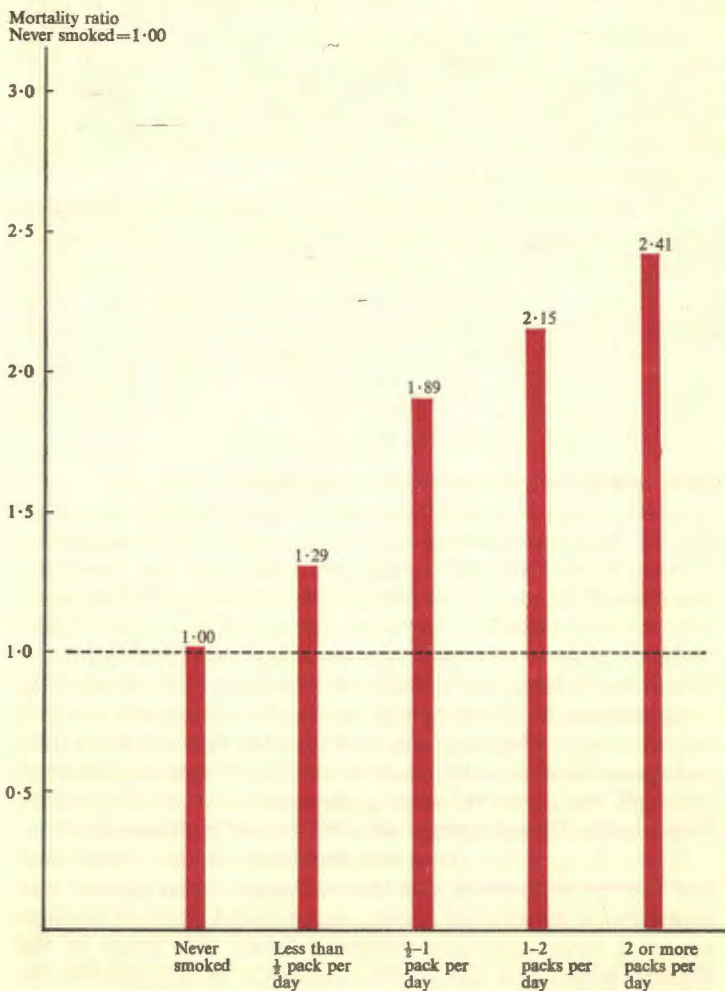
Another group of possible factors which is closely related to obesity, blood cholesterol and diet, consists of occupation, physical activity and stress. It is well established that mortality and morbidity rates for ischaemic heart disease differ between occupation groups. The morbidity survey in England and Wales in 1955–56 gave low patient consulting rates for coronary disease and angina pectoris among men aged 15 to 64 working in agriculture, horticulture and forestry.<sup>14</sup> The rate was low not only for farm labourers, etc. but also for farmers and farm-managers. Similar results were found in an American survey although the high rate among directors and administrators found in the British survey were not found in this study.<sup>15</sup>

It was thought that these different rates reflected social class and income differences and that ischaemic heart disease was primarily an upper class disease. But a recent study of employees of a large American company found that those in the highest grade had the lowest incidence of myocardial infarction.<sup>16</sup> In this country a social class gradient for mortality

## 8

**Death rates from coronary artery disease by current amount of cigarette smoking. White American men aged 50 to 69. 1952-55.**

Source: Hammond and Horn 'Smoking and death rates—report on 44 months of follow up of 187,783 men'. II. Death rates by cause. J. Amer. med. Ass. 166. (1958).



from diseases of the coronary arteries was found among men in the 1949-53 Registrar General's figures but the gradient was much diminished compared with the 1930-32 figures. It is now thought that this gradient may virtually disappear within the next few years. This prediction echoes the hypothesis of a dietary 'threshold'. Once society has reached a certain degree of affluence, individual variation in living standards no longer seem to have a direct causal relationship with ischaemic heart disease.

Diagnostic criteria may not be the same for all occupational groups and varying standards of health may be required before entering certain jobs (e.g. coalmining). Also, the lower social classes may be less likely to identify and hence report a coronary incident. However the differences outlined above gave rise to further investigation as to why they might occur.

It has been shown that drivers of London's double decker buses were more likely to die suddenly from coronary thrombosis than the conductors, and that Government clerks suffer more often from rapidly fatal cardiac infarction than do postmen.<sup>17</sup> From this and other studies, it was suggested that a relationship existed between physical activity and the incidence of ischaemic heart disease. This hypothesis has been tested by studying the results of 5000 necropsy examinations performed in men aged 45 to 70 during the years 1954-56.<sup>18</sup> The conclusion was that 'physical activity of work is a protection against coronary heart disease. Men in physically active jobs have less coronary heart disease during middle age, what disease they have is less severe, and they develop it later than men in physically inactive jobs'. However the evidence on this problem is conflicting, and ischaemic heart disease does occur even among 'heavy' workers to an extent which constitutes a major health problem.

It had also been shown that the London Transport bus drivers required larger uniforms than did conductors on entry into employment.<sup>19</sup> This gave rise to the hypothesis that the men were of different constitutional types, e.g. in respect of weight or metabolism, although after allowance for this, a difference was still observed. Also, occupation in these studies was measured by the last occupation of the patient and they may have changed jobs because of their physical condition. Not all authorities agree, but on balance it is probable that physical inactivity, possibly in direct association with other factors, contributes to the development of ischaemic heart disease.

Another factor which tends to be commonly associated with occupation is stress. It is, of course, difficult to measure, and studies are few and often inconclusive, however in an investigation of 100 men between the ages of 25 to 40 with ischaemic heart disease, and 100 healthy controls, 91 of the former gave a history of severe occupational strain and only 20 of the latter did so.<sup>20</sup> The type of stress was severe and one example was of an executive who built up a successful market research business by working 70 hours a week or more. For a month prior to his coronary occlusion he often worked 24 hours a day to meet a deadline. The type of stress encountered here was internal and self-imposed as distinct from external stresses such as 'flood, famine, pestilence and war'.

Another investigation studied 40 middle aged male accountants over a period of five months.<sup>21</sup> There were two types of accountants: tax accountants whose busiest period was in April and corporation accountants for whom it was January. The subjects' diet and physical activity changed little over the period of the study, but the mean cholesterol level for the tax accountants reached its peak in April and for the corporation accountants in January.

Boris Pasternak wrote concerning the illness of Dr Zhivago: 'it's an illness I've got, sclerosis of the heart . . . It's the common illness of our time. I think its causes are chiefly moral. The great majority of us are required to live a life of constant systematic duplicity'.<sup>22</sup>

Although the medical description and the moral implications of this passage would be questioned by the profession, stress may well have a causal relationship to the development of ischaemic heart disease.

#### **OTHER FACTORS**

A correlation has been shown between parents and children with cardiac infarction. It is difficult however to separate hereditary factors from environmental ones. Studies to show the effects on ethnic groups exposed to varying environments have found differences in prevalence rates. The opposite type of study, where different races are exposed to the same environment are obviously more difficult to control. Hereditary or genetic factors certainly play an important part in diseases such as familial hypercholesterolaemia and diabetes mellitus which are closely linked with ischaemic heart disease. Sex differences have also been carefully studied and the higher

incidence of heart disease amongst males has led to a suggested hormonal effect which has pharmacological implications.

These factors are all still being extensively investigated in the hope that they will throw light on practical measures in the prevention of ischaemic heart disease. They are, however, outside the direct control of the individual and for that reason no detailed discussion of them has been included.

### *Prevention and Treatment of Heart Disease*

The Framingham study showed that over half of all patients who die with acute myocardial infarction do so within one hour. This high proportion of sudden deaths emphasises the importance of preventing or delaying the development of ischaemic heart disease. Prevention can, however, be considered in two parts. First, it is clear that the long-term objective must be to delay and possibly prevent the development of atherosclerosis of the coronary arteries, the process which starts during childhood or early adolescence. However such a fundamental and long term approach must at present be a subject for research rather than a basis for practical general medicine.

Second, it is possible to concentrate attention on those in whom the process of coronary atherosclerosis is already advanced but in whom clinical features have not developed and severe myocardial damage has not occurred. These are most likely to be affected by one or more of the possible causal factors already discussed. Therefore one of the most important preventive measures to reduce the incidence of ischaemic heart disease at present would be further health education.

Much publicity has been given to the disease and concern about its incidence has been aroused among laymen. From the evidence already discussed, it would seem that general practitioners could give a positive lead to middle-aged men who are concerned with avoiding the disease and reducing their personal risk. Such men would be well advised to refrain from excessive smoking, prevent obesity through sensible diet and increase the amount of exercise they take. It is more difficult to advise how to reduce exposure to nervous stress. Specific dietetic restrictions, other than those aimed at maintaining optimum weight, do not appear to be warranted in the present state of knowledge.

Once an acute episode of myocardial infarction has occurred it is essential to prevent immediate death and to reduce the

risk of recurrent episodes. As regards the former, it has been suggested that specialist cardiac units could reduce mortality by as much as 25 per cent,<sup>23</sup> and the first few of these units are now being established in Britain. They have facilities, for example, for continuously monitoring the patient with an electrocardiograph for the critical five days after admission, and for taking immediate action when necessary.

If death from an acute episode is averted measures must then be taken to reduce the risk of recurrence in those who have suffered a first episode. At one time it was widely thought that continuous anti-coagulant therapy would provide the answer. Now, however, it is used mainly as a short term treatment immediately following acute infarction.

Another approach is based on the hormonal factors that may underly ischaemic heart disease. The sex difference in the incidence of ischaemic heart disease are probably derived partly from physiological changes in ovarian function, particularly endogenous oestrogen secretion. The clinical features of ischaemic heart disease occur more commonly and at an earlier age in men than in women. Detailed examination has shown a regular cyclical depression of cholesterol at ovulation; a post-menopausal rise in plasma cholesterol; a rise in cholesterol after the premature removal of both ovaries; and, in pregnancy, a rise in plasma cholesterol during the last three months of about fifty per cent.<sup>24</sup> Oestrogens produce marked and continued lowering of plasma lipids. The difficulty, which has not yet been overcome, is to find an analogue which does not have a feminising effect. Androgens also reduce serum lipids when given intramuscularly. However, this reduction does not seem to be associated with a corresponding reduction in the chances of developing a second acute episode.

The pharmaceutical industry has developed a branched chain fatty acid compound (CPIB), which was originally combined with androsterone. It, too, significantly depressed serum cholesterol and other blood fats, and it now seems likely that the effect is mainly due to the CPIB alone. This not only depresses cholesterol levels but also tends to decrease thrombotic tendencies. There is hope that it will make a significant contribution to the prevention of the recurrence of thrombotic episodes and extensive clinical trials are at present being conducted. It is necessarily a very long term undertaking to evaluate the effectiveness of any treatment in preventing recurrence of acute ischaemic heart disease.



Finally, diet to reduce cholesterol levels has been suggested. However, long term studies testing the effect of diet in preventing the recurrence of coronary thrombosis have just been completed. They showed that neither diets low in animal fats, nor those in which animal fat has been replaced by corn oil reduce the risk of death from a second attack.<sup>25, 26</sup>

### *Economic Costs of Heart Diseases*

Economic costs of a disease can be divided into direct costs, which are actual expenditure, and indirect costs which are less tangible and cannot always be quantified.

#### **DIRECT COSTS**

Direct economic costs to the National Health Service can be estimated. These are in three parts; hospital expenditure, general practitioners remuneration and non-hospital prescription pharmaceutical services. (Table F). The hospital figures do not include out-patient costs.

The estimated cost to the Health Service (excluding out-patients) in 1963-64 in England and Wales for *diseases of the heart, rheumatic fever and hypertensive disease* was £43.1 million, almost £1 a year per head of population. For *arteriosclerotic heart disease, including coronary disease* the cost was £14.4 million, less than two per cent of the total expenditure by the National Health Service.

Another direct cost is research expenditure. For diseases of the heart in this country it is still comparatively small—probably in the region of five per cent of total medical research expenditure. The British Heart Foundation is currently spending some £150,000 a year. By contrast the American Heart Association's figure of £3.7 million is approximately 25 times greater. A similar ratio between the two countries probably applies to total government and private research expenditure on heart disease. The total for cardiovascular disease research in the United States was £45 million in 1963; more than twice the total expenditure on all medical research that year in Britain. None of these figures include the research expenditure by the pharmaceutical industry into heart disease—perhaps a tenth of their £150 million world-wide research budget.

A further direct cost which can be measured is the money paid out in sickness benefits. *Arteriosclerotic and degenerative heart disease* (it is not possible to isolate coronary disease)

# F

## Expenditure by NHS on diseases of the heart. England and Wales 1963-64.

Source and method: See Appendix B.

<i>Disease or condition (ICD No.)*</i>	<i>Cost</i>				<i>Proportion of total expenditure on services</i>			
	Hospitals	General Practitioners	Non-hospital Pharmaceutical	Total	Hospitals	General Practitioners	Non-hospital Pharmaceutical	Total
	£m.	£m.	£m.	£m.	%	%	%	%
Arteriosclerotic heart disease, including coronary disease (420)	12.0	1.2	1.2	14.4	3	2	1	2
Hypertensive disease (440-447)	2.8	2.0	5.1	9.9	1	3	5	2
Other (440-447 remainder)	15.3	1.8	1.7	18.8	3	2	2	3
Total: Disease of the heart, rheumatic fever and hypertensive disease (440-447)	30.1	5.0	8.0	43.1	7	7	8	7

\*Different groupings obtain in morbidity statistics compared with mortality statistics. It has therefore been possible to isolate only *arteriosclerotic heart disease, including coronary disease and hypertensive disease*. The total figure includes *diseases of the heart* (ICD Nos. 410-443), *rheumatic fever* (ICD Nos. 400-402) and *other hypertensive disease* (ICD Nos. 444-447).

cost some £10 million in benefits in 1963-64, amounting to five per cent of the total.

In each case, these proportions are very small compared with the level of mortality. Nineteen per cent of all deaths were assigned to *arteriosclerotic heart disease, including coronary disease*. Thus, in terms of direct costs even such a common and serious disease as ischaemic heart disease is relatively inexpensive. This is the pattern to be expected for any disease which causes many rapid deaths and for which there is no proven long-term or curative therapy.

#### INDIRECT COSTS

While direct costs are relatively low the real costs of the disease are being borne by the individual and his family. Table G shows that almost 50,000 women were widowed in 1963 through the husband dying from *arteriosclerotic heart disease, including coronary disease*. That is one in four of all those widowed in that year. Although this proportion was lower in previous years, it seems certain that at least half a million of the widows alive today lost their husbands through heart disease.

Of those widowed in 1963, some 6000 were under the age of 50. Probably only one third of these women will remarry, and the remainder face an expectation of at least 25 years of widowhood. The younger men who died not only left widows but also dependent children to be cared for. Such premature deaths result both in loss of income and in all the social problems facing a fatherless family.

Heart disease early in life, even if it does not result in death, also causes much suffering and hardship both in terms of unhappiness and uncertainty among the family as well as in economic terms. Thus it is on the patients and their families that the real burden of the disease falls.

It is important, however, also to look at the disease in terms of its total indirect cost to the State. If the disease can be prevented or controlled, the State gains from those who live on to earn and to contribute in taxes, and from not having to pay widows benefits; but, against this, it loses by having to pay out continued social security benefits (mainly pensions) to those whose lives have been saved.

Table H shows the order of magnitude of these possible gains and losses. It shows the effect, in terms of total man-years, if those who died from *arteriosclerotic heart disease, including coronary disease* in 1963 had lived on with normal expectation of life.

# G

Number of widows bereaved in 1963, through death of husband from arteriosclerotic heart disease, including coronary disease. England and Wales.

Source: Registrar General's Statistical Review of England and Wales, 1963. Parts I and II.

Age Group	MEN		WIDOWS BEREAVED		
	Number of deaths from the disease in 1963	Proportion of each age group married	Number	Average Age	Average number of years of married life which could have been expected
Years	Number	%	Number (nearest 100)	Years	Years
15-24	14	15	0	17	50
25-34	179	77	100	27	41
35-44	1814	88	1600	37	31
45-54	6981	88	6200	47	22
55-64	17,515	86	15,100	57	15
65-74	21,245	79	16,600	67	9
75+	18,092	54	9700	79	4
Total	65,840		49,300		

(Note: Average age of widow is assumed to be equal to late-husband's age less three years.)

The totals express the approximate man-years lost in a single year. (They do this by projection rather than by including survivors from previous years.) They do not take into account trends in death rates at different ages nor the change in expectation of life if the disease had been eliminated.

If no one had died from arteriosclerotic heart disease some 230,000 working man-years would have been saved; that is the total years which those under 65 at death would have lived before reaching the age of retirement. Against this more than 520,000 post-retirement man-years would have been saved. This is made up of approximately 240,000 man-years of those who were not yet 65 and a further 280,000 man-years of those who were already 65 or over.

Thus the long-term effect of preventing or substantially postponing the onset of heart disease would be to increase

# H

Expected number of future man-years lost by those who died from arteriosclerotic heart disease, including coronary disease in 1963. Males in England and Wales by age.\*

Source: Derived from the Registrar General's Statistical Review of England and Wales, 1963. Part 1.

	Col. 2	Col. 3	Col. 4	Col. 5
Age at death	Total man-years lost	Man years lost before age 65	Man years lost after age 65	Col. 3—Col. 4
Rounded to nearest hundred				
0—24	700	600	100	+ 500
25—29	1400	1200	200	+ 1000
30—34	5700	4800	900	+ 3900
35—39	16,900	13,800	3100	+ 10,700
40—44	38,000	29,500	8500	+ 21,000
45—49	57,400	41,000	16,400	+ 24,600
50—54	94,000	57,900	36,100	+ 21,800
55—59	123,800	56,300	67,500	— 11,200
60—64	132,100	25,000	107,100	— 82,100
65—69	107,700		107,700	— 107,700
70—74	85,200		85,200	— 85,200
75+	88,300		88,300	— 88,300
Totals	751,200	230,100	521,100	— 291,000

disproportionately the numbers in the population who are over the age of retirement. Approximately twice as many years of retirement as working years would be 'saved'. (If the age of retirement were shifted upwards, this proportion would obviously be reduced.) In round figures, the State takes about two fifths of income as direct or indirect taxes, and comparatively little is repaid as direct personal benefits to men at work (e.g. housing or food subsidies). Broadly speaking, therefore, if State retirement benefits equalled something of the order of one fifth of final earnings, the State would break even if ischaemic heart disease could be prevented. If retirement benefits were lower, the State would show a financial gain;

\*For each age group separately life expectancy was derived for the mid-point of the group. This expected number of years was then divided between the number of years expected before the age of 65 and the number of years expected after the age of 65. Each of these was then multiplied by the number of deaths occurring in each of the age groups in 1963.

if they were higher, as they are likely to be in the future, it would show a loss. In terms of total national production, less personal consumption, the pattern is, of course, similar.\*

This prediction is not meant, in any way, to detract from the progress that is being, and must be, made towards the conquest of heart disease. However it is wrong to imagine that the prevention or postponement of mortality from ischaemic heart disease will bring in its wake great economic benefits. Indeed the discovery of an effective treatment will not only involve expenditure on the treatment itself, but might even impose additional burdens on the nation in purely economic terms.

Thus, like the direct costs of heart disease, the indirect net costs, except to the individual family, are also relatively low. This, once again, is because of the particular pattern of morbidity and mortality which it causes. There would only be a substantial economic as well as a human gain from the control of heart disease if the age pattern of its victims could be altered so that premature mortality alone was prevented.

### *The Future*

Substantial progress has been made in the treatment of many forms of heart disease, particularly rheumatic and syphilitic heart disease, congenital malformation of the heart and hypertension from certain causes. This has been achieved by the use of medicines, surgery or dietary control. Though much research has been carried out on the aetiology, prevention and in the treatment of ischaemic heart disease it is still an enigma.

One of the difficulties is that although morbid pathological changes may eventually be associated with overt clinical symptoms there is no certain way of detecting these changes in the earliest stages. Moreover the pathological changes may be irreversible. The eventual answer must come from prevention of arterial change in its early stages or delaying its progress.

\*One other way in which the indirect costs can be measured is in terms of the loss of output to the economy, in monetary terms, due to premature death, illness and disability. The US Department of Health, Education and Welfare did so for the US on the assumption that the death and disability rates for heart disease (including ICD Nos. 400-468, diseases of the circulatory system, and ICD No. 754, congenital malformations of the circulatory system) were zero while rates for all other causes remained unchanged.<sup>37</sup> They thus added the loss of output of those who died or were ill in a single year (1962) to the loss of output of those who had died in previous years from heart disease and would otherwise have survived into 1962. They arrived at the spectacular figure of \$30,720 million (nearly £11,000 million) for the total cost of heart disease, nine tenths of which was accounted for by loss of output. This sum was approximately equal to the total US direct expenditure on all forms of medical care.

It is felt, however, that this concept of indirect costs is not meaningful. The figure gives an order of magnitude for the economic loss from heart disease, which could be compared to that of other diseases, but on its own it has little relevance.

Meantime, it is accepted that some groups carry a higher risk of developing the disease or developing it earlier than others. The difficulty is to apply this knowledge, limited as it is, to the individual patient.

Hyperlipaemia, hypertension, cigarette smoking, physical inactivity, increase in weight, nervous stress and the reaction of the individual to this stress, diabetes mellitus and genetic factors are all thought to be possible causes. In America much emphasis has been placed on the fat content of diet; in Britain this hypothesis is questioned.

Three avenues of approach to the prevention of premature mortality are possible. The first is to prevent the development of atheroma in the first place. This would need the identification of a treatment and a large scale controlled trial on 'healthy' individuals. The second is to delay, halt or reverse the atheromatous process before the lining of the artery is destroyed. With both these methods much work is in progress but no major breakthrough has been achieved. Meanwhile it has been suggested that the 'high-risk' subjects should keep their weight down, reduce cigarette smoking, take regular but moderate exercise, avoid stress and take regular holidays and weekends off. The third approach is the reduction of mortality in patients who have had a coronary thrombosis. One method to achieve this is by introducing to all parts of the country special coronary intensive care units where continuous monitoring of patients may be carried out, and modern methods of resuscitation are available.

Other important preventive measures suggested by the WHO include improvements in pre-symptomatic detection of those with a number of high risk factors, and their surveillance; refresher courses for general practitioners; the training of more specialists in cardiology; the establishment of treatment and rehabilitation centres; and emergency services for patients at the time of attack.

At present, the search for ways of prevention and cure for ischaemic heart disease may be unreal in such terms but nevertheless its containment, control or reduction in toll is a major challenge to medical science. If the challenge is met the resultant cost factors must be critically examined. Direct costs of heart disease which are now low will be raised and indirect costs, in terms of an individual's risk, which are now high will be lowered. There will thus be a shift of the cost of the disease from individuals to the community. This will be felt in two ways,

first, pressure on the medical sector and second, pressure on the welfare sector.

If treatment of even a proportion of the many hundreds of thousands of patients with ischaemic heart disease in this country is to be efficient, both economic and medical manpower resources will be severely tested. Similar pressures would be exerted if presymptomatic detection were possible, and if an early diagnosis system were put into operation.

The welfare service would need to be extended also, both in monetary outlay in the form of social security payments and in terms of manpower and facilities to meet the probable increase in the number of persons past the present retirement age in the community.

The conquest of diseases such as tuberculosis or mental illness, which without effective treatment result in long and costly illness, and frequently occur in young adults, can bring very substantial savings not only in human but also in economic terms. It is important to realise in advance, however, that the conquest of heart disease will not by itself bring comparable economic benefits. Much of the cost of progress, with its very real benefits to the individuals and their families, will have to be borne by the community. One way in which this cost could be met would be by postponing the normal age of retirement.

The present compulsory age of retirement was brought in during an era of unemployment to release jobs for younger men with families to support. Today a number of factors militate against a rigid compulsory age. First, there is comparatively full employment. Second, many men in their 60's are fitter than their counterparts of the 1930's, although it is also true that more of the less fit are kept alive. Third, withdrawal from a working role can have a deleterious effect on their lives. It is often a factor in depression and anxiety in pre-geriatrics who need to do useful work as occupational therapy. Finally the work itself is often less strenuous today than it was before the war. Already many who reach the present retirement age are able and willing to work on, at least part-time or at a reduced pace. As progress in medical science makes this situation even more common, the age for complete retirement could be made more flexible, and it is possible that many would welcome this change. In several other countries the age for compulsory retirement is much later. If this were the case in Britain also, the costs which otherwise seem likely to be incurred in the conquest of diseases such as ischaemic heart



disease could be offset by the additional years of useful work, instead of by imposing a new burden on those below the present age of retirement.

## *Appendix A*

### Definition of Medical terms used.

#### *Angina pectoris*

A severe substernal crushing pain of sudden onset, which may radiate to the neck, arms or back, and is due to lack of oxygen to the heart muscle. It follows effort, which is halted by the pain, leading to automatic recovery, but recurrence on further similar effort.

#### *Arteriosclerosis*

Should be used in the general sense to include all types of arterial disease in which 'hardening of the arteries' occurs.

#### *Atheroma and Atherosclerosis*

A disease of the intima (innermost coat) of the aorta and of its main branches characterised by nodular patchy fatty degeneration resembling porridge in appearance (athêrê, porridge).

#### *Cardiac infarction*

Death of heart muscle due to decreased blood supply.

#### *Cholesterol*

One of the lipids found in bile, gallstones, brain, blood cells, plasma, eggs yolk, seeds and animal tissues, etc.

#### *Coronary occlusion*

The closure or blockage of a coronary vessel.

#### *Coronary thrombosis*

The clotting of blood during life in one of the coronary arteries.

#### *Degenerative heart disease*

A retrogressive pathological change in cells or tissues in consequence of which the functions of the heart may be inhibited or destroyed.

#### *Ischaemic heart disease*

Heart disease due to a decreased blood supply to the heart. This may be due to narrowing or occlusion of the coronary arteries.

#### *Lipids*

A general term that includes fats and fat-like compounds.

## Appendix B

### Calculations of the Costs.

Expenditure allocated to the diagnostic groups shown comprises the greater part of expenditure in England and Wales by hospital authorities and in the services administered by the Executive Councils. Local authority health and welfare expenditure, costs of central and area hospital administration and hospital out-patients have been excluded as no breakdown by diagnosis was practicable.

The costs of the Hospital Services have been estimated generally from the time spent in hospital by in-patients for the specified conditions multiplied by the cost per day of keeping a patient in hospital. The total number of bed-days was obtained for 1961 from the Report on Hospital In-patient Enquiry for the year 1961, Part II (HMSO 1964) and the cost per in-patient day from Hospital Costing Returns for the year ended 31 March, 1962, Part I (HMSO 1962). Allowance was made for varying costs between different types of hospitals and a weighted cost per day was used. Thus 1961-62 cost figures were obtained and estimates were then made to adjust these figures to 1963-64. Three factors influenced these adjustments, changes in cost per day, changes in the number of in-patients discharged and changes in the average length of stay per patient. Changes in cost per day were obtained from the 1962-63 and 1963-64 Hospital Costing Returns, Part I (HMSO 1963 and 1964) again a weighting factor was introduced to allow for patients staying in different types of hospital. Changes in the number of patients discharged were projected from the changes obtaining for the year 1957-61 derived from previous In-patient Enquiries. Changes in the average length of stay were estimated by using figures given in the Annual Report of the Ministry of Health for the years 1961, 1962, 1963 (HMSO 1962, 1963, 1964), for average duration by hospital departments. The 1959 Hospital In-patient Enquiry, Part II (HMSO 1963) figures for diagnostic group by hospital departments were used to weight the Ministry of Health figures. Percentage changes for 1962-63 over 1961-62 and 1963-64 over 1962-63 were thus estimated for these three factors for each of the diagnostic groupings and a 1963-64 estimate was derived.

Costs of the General Medical Services were allocated proportionately to the number of diagnoses made for different conditions. The total cost of the General Medical Services was obtained for the Annual Report of the Ministry of Health for the year 1964 (HMSO 1965) and this cost was then divided proportionately.

The cost of the non-hospital pharmaceutical services were derived from an analysis of the value of prescriptions given for specified diagnoses. The proportion of all diagnosis falling on the specified condition was calculated for each pharmaceutical preparation. This proportion was then multiplied by the total value of sales for that preparation and, for each condition, each of these costs were totalled. A weighting factor was applied to translate the UK data used to England and Wales and this was derived from the total cost of pharmaceutical services given in the Annual Report of the Ministry of Health for the year 1964 (HMSO 1965).

The sources for allocating the expenditure on general practitioner and the pharmaceutical services were the British Medical Index and the British Pharmaceutical Index. These publications are part of a medical research service provided by Intercontinental Medical Statistics Ltd.

Details of the IMS sample etc. are given in a previous OHE publication 'The Cost of Medical Care'. 1964.

#### *Acknowledgment*

The Office of Health Economics wishes to thank Intercontinental Medical Statistics Ltd for making available some of their statistical data on which the calculation of costs of heart diseases was based.

## References

1. MORRIS, J. N. (1951). *Lancet* i. 1 and 69.
2. ALLBUT, C. (1915). *Disease of the arteries, including angina pectoris*. Vols 1 and 2. Macmillan. London.
3. *Report on Hospital In-patient Enquiry for the year 1961* (1964). Part II. Detailed tables. Ministry of Health and General Register Office. London H M S O.
4. LOGAN, W. P. D. and CUSHION, A. A. (1958). *Morbidity Statistics from General Practice*. Volume 1 (General). *Studies of Medical and Population Subjects No. 14*. General Register Office. London H M S O.
5. MORRIS, J. N. (1964). *Uses of Epidemiology*. E. and S. Livingstone Ltd. Edinburgh and London.
6. WHO News Bulletin. June 1965. *The Practitioner*.
7. DOYLE, J. T. *et al* (1957). *Amer. J. Pub. Health*. April 1957.
8. DAWBER, T. R., KANNEL, W. B., REVOTSKIE, N. and KAGAN, A. (1962). *Proc. Roy. Soc. Med.* 55. 265.
9. PAUL, O., *et al* (1963). *Circulation* 28. 20.
10. MORRIS, J. N., *et al* (1963). *BMJ* i. 571.
11. Society of Actuaries. (1959). *Build and blood pressure study*. Chicago.
12. KEEN, H., *et al* (1965). *Lancet* ii. 505.
13. DOLL, W. R. and HILL, A. B. (1964). *BMJ* i. 1399-1467.
14. LOGAN, W. P. D. (1960). *Morbidity Statistics for General Practice*. Volume II (Occupation). *Studies of medical and population subjects No. 14*. General Register Office. London H M S O.
15. *Coronary Heart Disease in Adults* (1965). United States. 1960-1962. United States Dept of Health, Education and Welfare. Sept.
16. PELL, S. and D'ALONZO, C. A. (1963). *JAMA* 185 (II). 831-838.
17. MORRIS, J. N. *et al* (1953). *Lancet* ii. 1053.
18. MORRIS, J. N. and CRAWFORD, M. D. (1958). *BMJ* ii. 1485.
19. MORRIS, J. N., HEADY, J. A. and RAFFLES, P. A. B. (1956). *Lancet* ii. 569.
20. RUSSEK, H. I. and ZOHMAN, B. L. (1958). *Amer. J. Med. Sci.* 235. 266.
21. FRIEDMAN, M., ROSENMAN, R. H. and CARROLL, V. (1958). *Circulation* 17. 852.
22. PASTERNAK, B. (1958). *Dr Zhivago*. Collins London.
23. OLIVER, M. F. (1965). *Pers. Comm.*
24. OLIVER, M. F. (1960). *Sex Differences*. Proceedings of conference of Royal College of Physicians of London. Pathogenesis of treatment of occlusive vascular disease. Ed. Macdonald L. Pitman Medical Publishing Co. Ltd.
25. ROSE, G. A., THOMPSON, W. B. and WILLIAMS, R. T. (1965). *BMJ* i. 1531.
26. BALL, K. P. and Research Committee. (1965). *Lancet* ii. 502.
27. *Economic Costs of Cardiovascular Diseases and Cancer, 1962* (1965). US Department of Health, Education and Welfare.

# The Office of Health Economics

The Office of Health Economics is an independent organisation founded in 1962 by the Association of the British Pharmaceutical Industry with the following terms of reference:

1. To undertake research to evaluate the economic aspects of medical care.
2. To investigate, from time to time, other health and social problems.
3. To collect data on experience in other countries.
4. To publish results, data and conclusions relevant to the above.

The Office of Health Economics welcomes financial support and discussions of research problems with any persons or bodies interested in its work.

## *OHE publications still available:*

---

1. Progress against Tuberculosis	2s. 0d.
4. Pneumonia in Decline	2s. 0d.
5. Health Services in Western Europe	2s. 6d.
6. The Price of Poliomyelitis	2s. 0d.
7. The Personal Health Services	2s. 0d.
8. The Venereal Diseases	2s. 0d.
10. The Costs of Medical Care	2s. 0d.
11. The Finance of Medical Research	2s. 0d.
12. New Frontiers in Health	2s. 0d.
13. The Pattern of Diabetes	2s. 0d.
14. The Pharmacist in Society	2s. 0d.
15. The Cost of Mental Care	2s. 0d.
16. Work Lost Through Sickness	2s. 0d.
17. The Local Health Services	2s. 6d.
18. Progress in Mental Health	7s. 6d.
Factors which may Affect Expenditures on Health	Free
Survey:	
1. The Residue of Poliomyelitis	25s. 0d.

---

# The Office of Health Economics

The Office of Health Economics is a research organization established in 1963 to provide independent economic analysis of health care. It is a part of the University of York and is funded by the Department of Health and Social Security.

The Office's research is carried out in a number of areas, including the economics of health care, health care financing, and health care delivery. It also provides policy advice to the government and other organizations.

The Office's research is published in a number of journals and books. It also provides a range of services, including consultancy and training.

The Office's research is highly regarded and has led to a number of important policy changes. It is a leading organization in the field of health economics.

The Office's research is highly regarded and has led to a number of important policy changes. It is a leading organization in the field of health economics.

The Office's research is highly regarded and has led to a number of important policy changes. It is a leading organization in the field of health economics.

The Office's research is highly regarded and has led to a number of important policy changes. It is a leading organization in the field of health economics.

The Office's research is highly regarded and has led to a number of important policy changes. It is a leading organization in the field of health economics.

The Office's research is highly regarded and has led to a number of important policy changes. It is a leading organization in the field of health economics.



