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SOME STATISTICAL ASPECTS OF MORTALITY FROM DEGENERATIVE HEART DISEASE

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I. INTRODUCTION

MEDICAL and social progress over the past 50 years has resulted in a large increase in the expectation of life, and this, together with a declining birth-rate, has caused an increase in the proportion of old persons in the population. In 1901 less than 5% of the population of England and Wales was aged 65 and over; by 1949 the estimated percentage had increased to 11% and must inevitably increase still further in the future. A result of this is that more and more attention is being given to diseases of old age and in fact a new specialized branch of medicine, geriatrics, seems to have arisen. Heart diseases form by far the largest group of causes of death in old age and in 1949 were the certified cause of death in 37% of the deaths in England and Wales at ages 65 and over (Table 1). Even in the age-group 55–59 heart disease was responsible for 24% of all deaths in 1949.

Table 1. Deaths aged 65 and over in England and Wales in 1949

International list no.	Cause of death	Percentage of total deaths
90– 95	Heart diseases	37
83	Intracranial lesions of vascular origin	15
45– 57	Cancer and other tumours	14
104–114	Disease of the respiratory system	11
	Other causes	23
	Total	100

It is the purpose of this note to study the recent trend of mortality from degenerative heart disease in England and Wales as recorded in the published figures of the Registrar General and to give some consideration to the important question of classifying heart-disease deaths. On this matter the Registrar General (1951, p. 201) writes:

> Since more than half of all deaths now occur at ages 65 and over, and more than one third of these are attributed to heart disease, the problem of diagnosis, description and classification of these conditions is one which merits more attention than is at present being devoted to it.

Any study of deaths by cause meets with a number of difficulties which become accentuated when we wish to consider trends over a number of years. First, over a period of time there must have been changes in the precision of diagnosis, in nomenclature, and in fashions of certification. Secondly, changes have been made from time to time in the International List of Causes of Death, and the various revisions, which have been adopted by different

countries at different times, cause discontinuities in the published figures. Thirdly, the system of allocation to cause, when several are stated on the death certificate, may have varied.

Little can be done about the first difficulty beyond always bearing it in mind and relating any death-rate to the state of knowledge at the time. As regards the second and third, in England and Wales the Registrar General adopted the Fifth Revision (1938) of the International List of Causes of Death from 1940. At the same time he abandoned the previous system of classification by a set of priority rules, when more than one cause of death was stated, in favour of classification according to that cause which the certifying doctor stated to be the underlying cause (Registrar General, 1949, p. 77). The rules and classification of the causes of death are given in the *Manual of the International List of Causes of Death, as adopted for use in England and Wales, Scotland and Northern Ireland. Based on the Fifth Decennial Revision by the International Commission, Paris* 1938 (H.M.S.O. 1940). The deaths in 1939 have been classified by the methods used until that year and also by those used from 1940 and the results have been published (*Registrar General's Statistical Review* 1939). Similar dual classifications have been obtained for the years 1936–38 and the figures for the four years 1936–39 used to obtain conversion ratios (by sex and age-group) for converting the number of deaths by cause for years prior to 1939 (classified according to the 4th Revision of the International List and the set of priority rules) into the numbers corresponding to the methods used from 1940 (5th Revision and physician's preference). The conversion ratios (by sex only) are given for the various causes of death in Appendix BI of the 1940 *Statistical Review*, vol. I. Unless otherwise stated all the death-rates quoted in this note are those estimated or classified according to the methods introduced in 1940; many rates are taken from the text volumes of the *Statistical Review* (Registrar General, 1949, 1951), but some have had to be estimated by the methods indicated in Appendix I to this note. The quoted classification numbers of causes of death are according to the 5th Revision (1938) of the International List of Causes of Death.

The analysis has not been carried beyond 1949 because the Registrar General's classification of the 1950 deaths is based on the 6th (1948) Revision of the International List, which differs considerably from previous International Lists both in scope and grouping of causes.

II. PRESENTATION OF THE DATA

Heart disease

Heart disease is covered by numbers 90–95 of the International List, and Table 2 gives an analysis of the 1949 deaths in these categories in order to give some idea of the relative magnitude of the assignments to the various forms of heart disease.

It will be seen from this table that nearly 90% of all deaths classified as due to heart disease are in groups 93 and 94. The degenerative heart diseases, which alone are dealt with in this paper, consist of groups 93*c*, 93*d*, and 94; in 1949 they included 87·3% of all heart-disease deaths. Strictly speaking, part of group 92 (i.e. degenerative diseases of the valves of the heart) should probably be treated as degenerative heart diseases, but this has not been done because the degenerative valvular diseases are grouped with other diseases which are not described as degenerative. Heart diseases of known rheumatic

Table 2. Deaths from heart disease in England and Wales (all ages) in 1949

International list no.	Cause of death	Total number of deaths	Percentage of total deaths
90	Pericarditis	322	0·2
91	*Acute endocarditis*	*453*	*0·3*
92	Chronic affections of the valves and endocardium	13,796	8·9
93	Diseases of the myocardium:		
a	Acute myocarditis	206	0·1
b	Chronic myocarditis specified as rheumatic	466	0·3
c	Myocardial degeneration	85,134	55·2
d	Myocarditis not distinguished as acute or chronic	6,577	4·3
	Total diseases of the myocardium	92,383	59·9
94	Diseases of the coronary arteries and angina pectoris	42,922	27·8
95	Other diseases of the heart	4,383	2·9
90–95	Total heart diseases	154,259	100·0
	Subdivision of myocardial degeneration (93*c*)		
93*c* (1)	Cardiovascular degeneration	33,774	21·9
(2)	Myocardial degeneration described *as fatty*	679	0·4
(3)	Other myocardial degeneration not specified as rheumatic	50,681	32·9
		85,134	55·2

origin comprise group 93*b* together with certain subdivisions (not shown in Table 2) of groups 90, 92 and 95, and totalled 10,089 deaths or 6·5% of the 1949 total.

The death-rates from the various forms of degenerative heart disease in each year from 1931 to 1949 will now be presented in the form of graphs and the principal features briefly described.

Diseases of the coronary arteries and angina pectoris (94)

This group of diseases will be referred to as coronary disease and includes coronary thrombosis which has had so much publicity in recent years. The coronary arteries are the two arteries within the heart which carry the blood supply to the heart muscles, and death from coronary disease is due to the partial or complete cutting off of this blood supply either by the formation of a blood clot (thrombosis), obstruction by some foreign body (embolism), degeneration, hardening or thickening of the arteries (arteriosclerosis and atheroma), rupture or some similar cause.

Fig. 1 shows the death-rates from coronary disease for quinary age-groups from age 50 upwards. The most striking feature is the increase which has taken place over the 19 years under review. The 1949 male rates are some $4\frac{1}{2}$–5 times the 1931 rates for each age; for females the corresponding figure is 5–6 times,

but the female rates are considerably lower than those for males. It will be noticed that the curves for the different age-groups are all of very similar shape, and that in the pre-war period there was a steady increase at all ages. By 1939 the death-rate had increased to approximately $2\frac{1}{3}$ times the 1931 rate at all ages and for both sexes. Thus, if the diagrams were plotted using a logarithmic scale for the death-rates, the curves for each age-group would be approximately parallel.

From 1940 to 1941 there was a fall in the death-rates at all ages, and then the upward trend was resumed at a greater rate than before the war. By about 1947 the rates had reached the point expected if the pre-war trend had continued unaltered; thereafter the rates were above the extended pre-war line. Thus the war years after 1940 showed a discontinuity in the previous regular increase in the death-rates. This curious feature, which is also shown by some other forms of heart disease, will be discussed later.

Myocardial degeneration (93*c*)

The myocardium is the muscular mass of the heart which is also subject to various forms of degeneration and sclerosis (thickening and hardening). Diseases classed as 'myocardial degeneration, infarction, sclerosis and other chronic myocarditis' (group 93*c*) can be divided into two sections. The one consists of group 93*c* (1) (cardiovascular degeneration) which comprises (i) cardiovascular degeneration and hypertensive (i.e. high blood-pressure) heart disease and (ii) myocardial degeneration (or myocarditis) where mention is made of arteriosclerosis. The Registrar General (1951, p. 201) writes 'some of these conditions may differ from coronary disease only in description and the groups (i.e. 93*c* (1) and 94) are best considered together'. However, it is thought that useful information can be obtained by considering the two groups separately.

The other section of group 93*c*, comprising 93*c* (2) and 93*c* (3) (together with the small group 93*d* for convenience), will be referred to as 'other myocarditis' and contains conditions where no mention is made of either hypertension or arteriosclerosis. If either of these conditions is mentioned on the death certificate the death is classified in group 93*c* (1). Other myocarditis will therefore, in all probability, contain deaths which with more precise description would have been classified as coronary disease or cardiovascular degeneration. The question whether the precision of diagnosis has improved over the 19 years and resulted in the transfer of deaths from 'other myocarditis' to 'cardiovascular degeneration' or 'coronary disease' will be considered in Part III.

Fig. 2 shows the death-rates for cardiovascular degeneration (group 93*c* (1)). Here again there was a steady proportionate increase at all ages prior to the war but at a slower rate than for coronary disease. From 1931 to 1939 the death-rate increased by approximately 50% for all ages and both sexes, although there was a slight tendency for the percentage to increase with increasing age. Then, like coronary disease, there is the war-time trough. At the older ages the trough is more noticeable than for coronary disease. Thereafter the rates show a tendency to increase but, unlike coronary disease, at a slower rate than before the war. At the younger ages the trough is hardly noticeable, and from about 1942 the rates do not appear to be increasing.

The mortality from 'other myocarditis' (where hypertension or arteriosclerosis are not mentioned—groups 93*c* (2), (3) and 93*d*) is shown in Fig. 3.

Once again there is the pre-war proportionate increase, but in this case the rate, which is more or less the same for all age-groups except the oldest, differs for males and females. From 1931 to 1939 the death-rates for age-groups 50–74 increased by about 20% for males and 5% for females; for ages 75 and over the percentage increase was 35% and 24% for males and females respectively. There was a pronounced maximum in 1940 and thereafter the death-rates have in general fallen, but not proportionately at all ages. For both sexes the 1949 death-rate is only about 40% of the 1940 rate at ages 50–55, increasing to about 75% at ages 75 and over.

Degenerative heart disease and senility

Before it is possible to consider how much the features described above are due to real changes in the prevalence of the diseases or merely to changes in nomenclature and precision of diagnosis, it is necessary to know what has been the trend of mortality from degenerative heart diseases as a whole. The number of deaths classified, rather vaguely, as due to old age and senility has been decreasing, and the use of these terms is being discouraged by the Registrar General. Although old age is at present accepted without further inquiry as a sufficient cause of death for those aged 65 and over, the Registrar General considers the use of these terms should be confined to the 'relatively small group' of old people who 'die without any evidence whatever of definite disease' (Registrar General, 1951, p. 264). Particularly at the older ages many of these deaths must have been due, in some measure at least, to heart disease, and accordingly (following the Registrar General, 1949, 1951) the causes of death 'old age' and 'senility without mention of senile dementia' (groups 162*a*, *c*) have been combined with degenerative heart diseases (groups 93*c*, *d* and 94). The death-rates are plotted in Fig. 4 and show a pre-war increase from 1931 to 1939, the rate of which decreases with age, being for males 72% at ages 55–59 and 24% at 75 and over, while for females the percentage increase falls only from 24% to 16%. Then comes the war-time trough followed by a tendency to increase, more noticeable for males than females.

Degenerative cardiovascular-renal diseases

When studying mortality by cause, the broader the classification we adopt the more likely are we to get a true picture of the trend over a period of years. Dr D. D. Reid (1951) is reported as saying

> in view of the difficulties in the interpretation of trends in mortality from specific causes, it was probably wiser to treat degenerative cardiovascular lesions, together with cerebral haemorrhage and chronic nephritis as a single epidemiological unit.

The causes of death which it was decided to group under the heading degenerative cardiovascular-renal diseases are set out in Table 3, together with the number of deaths in 1949, in order to give some idea of the magnitude of mortality from each cause. Bronchitis (which in old age is frequently associated with heart disease) is included on the advice of Dr Reid and others on the ground that it is often a pulmonary congestion due to a faulty heart. It will be remembered that Pedoe (1947) adopted this course in his study of mortality in England and the United States. It might have been preferable to have included only chronic bronchitis (group 106*b*) or bronchitis certified as having a cardiovascular-renal disease as contributory cause (groups 106(3), (4), (5)), but comparable statistics for these groups were not available over

the whole period being investigated. An examination of the bronchitis death-rates showed them to exhibit certain points of difference from the other causes classified as degenerative cardiovascular-renal diseases. For this reason and in view of the comparative magnitude of the death-rates, bronchitis is sometimes given separate consideration in this paper. At ages 65 and over the degenerative cardiovascular-renal diseases as defined above comprise 57% of the 1949 deaths.

Table 3. Deaths from degenerative cardiovascular-renal diseases in England and Wales (all ages) in 1949

International list no.	Cause of death	Total number of deaths	Percentage of total deaths
93*c* (1)	Cardiovascular degeneration	33,774	13·0
94	Coronary disease	42,922	16·5
93*c* (2), (3), *d*	Other myocarditis	57,937	22·3
83*a*	Cerebral haemorrhage	34,127	13·1
83*b*, *c*	Cerebral embolism, thrombosis and softening	23,466	9·0
83*d*, *e*	Hemiplegia, paralysis and cerebral effusions of unstated origin	1,643	0·6
97	Arteriosclerosis (excl. coronary or renal sclerosis or cerebral haemorrhage)	13,663	5·3
106	Bronchitis	30,433	11·7
131	Chronic nephritis	9,997	3·8
162*a*, *c*	Old age and senility without mention of senile dementia	12,254	4·7
	Total	260,216	100·0

The death-rates for degenerative cardiovascular-renal disease are shown in Fig. 5, and while they show certain trends these are not very pronounced. For both sexes the rates are irregular in the older age groups. Males show a slight upward tendency in the age groups 55–59 and 60–64, while the female rates have a downward trend for ages 55–74. There is some evidence of the war-time trough at most ages.

Intra-cranial lesions of vascular origin (83)

Figs. 7 and 9 show the death-rates for cerebral haemorrhage (83*a*) and for cerebral embolism, thrombosis and softening (83*b*, *c*) respectively. These curves show many features in common with the heart-disease groups.

Bronchitis (106)

Fig. 10 shows the death-rates for bronchitis. The most noticeable feature of these diagrams when compared with the other figures is the much greater irregularity of the curves. This variability makes it difficult to decide whether the war-time trough is present or not; for some age-groups it is possible that some sort of a trough is present. That 1940 showed unusually high bronchitis mortality can hardly be missed, but the high mortality in 1947, which is a feature of most of the other diagrams, is for bronchitis hardly to be distinguished from the minor irregularities. As regards trends, the female rates

show on the whole a decreasing tendency at all ages, but for males the decrease only goes as far as 1938; thereafter, except for the oldest and youngest groups shown, there is a slight upward trend.

Sex-ratio

It will have been noticed from the graphs that the mortality differs, sometimes considerably, between the sexes. Accordingly the mortality sex-ratios (i.e. the ratio of male to female death-rates) have been calculated for each calendar year and age-group. The ratios are irregular and, unlike the death-rates, do not show any features necessitating consideration of every calendar year. Therefore the ratios have been averaged in groups of five (or four) years and are given in Table 4.

Table 4. Mortality sex-ratios (male/female), England and Wales

Cause of death	Period	Age-group				
		55–	60–	65–	70–	75 and over
Coronary disease (94)	1931–35	4·30	2·91	2·55	2·31	1·97
	1936–40	3·99	2·95	2·40	2·16	1·90
	1941–45	4·03	3·12	2·37	2·09	1·83
	1946–49	4·20	3·13	2·42	2·02	1·76
Cardiovascular degeneration (93*c* (1))	1931–35	1·65	1·65	1·72	1·62	1·33
	1936–40	1·81	1·69	1·55	1·48	1·31
	1941–45	1·51	1·68	1·59	1·43	1·26
	1946–49	1·71	1·68	1·53	1·33	1·19
Other myocarditis (93*c* (2), (3), 93*d*)	1931–35	1·14	1·09	1·12	1·12	1·00
	1936–40	1·36	1·27	1·20	1·17	1·05
	1941–45	1·51	1·45	1·34	1·17	1·07
	1946–49	1·54	1·56	1·35	1·20	1·02
Degenerative heart disease and senility (93*c*, *d*; 162*a*, *c*)	1931–35	1·61	1·42	1·38	1·27	1·08
	1936–40	1·95	1·66	1·47	1·32	1·12
	1941–45	2·38	1·98	1·64	1·41	1·15
	1946–49	2·65	2·20	1·75	1·43	1·13
Bronchitis (106)	1931–35	3·15	2·10	1·55	1·29	1·04
	1936–40	3·32	2·29	1·68	1·43	1·20
	1941–45	3·83	3·24	2·25	1·63	1·33
	1946–49	4·24	4·13	2·30	2·06	1·47
Degenerative cardio-vascular-renal disease	1931–35	1·58	1·40	1·36	1·27	1·11
	1936–40	1·74	1·52	1·39	1·31	1·15
	1941–45	1·86	1·71	1·49	1·32	1·16
	1946–49	2·05	1·91	1·57	1·37	1·14

A study of this table shows a tendency, evident in each cause of death, for the ratios to decrease with advancing age. The trend of the ratios over the 19-year period is not consistent, but there does appear to be some sort of pattern. In the older age-groups the sex-ratios for both coronary disease and cardiovascular degeneration show a tendency to decrease, while the ratios for other myocarditis, degenerative heart disease, bronchitis and cardiovascular-renal disease show an upward trend in all age-groups except perhaps the oldest.

Probably the most noticeable feature of Table 4 is the extremely high ratio for coronary disease, particularly at the younger ages. Cardiovascular degeneration shows an intermediate sex-ratio and other myocarditis a rather low ratio, but the ratios for these two groups are considerably closer at the end of the 19-year period than at the beginning, due to the contrary tendencies of the cardiovascular degeneration ratios to decrease and those of other myocarditis to increase as already mentioned. The ratios for bronchitis are of comparable magnitude to those for coronary disease, but, unlike them, show a considerable increase over the 19 years at all age groups.

General

There are certain matters of fact which should be borne in mind when considering the data set out above.

(i) It is important to remember that all the death-rates for 1931–38 are approximations to those which would have been obtained if the classification adopted from 1940 onwards had been in use during the earlier years.

(ii) The deaths and populations used in calculating the death-rates are in respect of civilians only from 3 September 1939 in the case of males and 1 June 1941 for females, but in dealing with degenerative heart-disease mortality, which is very largely confined to ages over 50, any selective effect caused by the exclusion of the armed forces from the figures can be ignored.

(iii) In view of the general ageing of the population which is taking place it might be thought that the average age of the group aged 75 and over had increased appreciably from 1931 to 1949. Actually, calculations based on the 1931 census and the 1% sample of the 1951 census (Registrar General, 1952) show that the average age of this group has increased by only about 7 weeks.

III. DISCUSSION OF THE DATA

The data set out in Part II will now be discussed and an attempt made to assess the reasons and significance of the various trends and peculiarities of the death-rates. While the course of the death-rates does sometimes appear to point to certain explanations, these are usually only partial explanations and a matter of opinion. In all the discussion it will be realized that we can only consider, as Morris, Heady and Barley (1952) have pointed out, rates of diagnosis, not of incidence. Also, to quote the Registrar General (1949, p. 208):

> Diagnosis of these degenerative conditions affecting the heart is difficult in many cases, and the nomenclature used to describe them when diagnosed is continually changing, and it is necessary to keep these facts in mind when attempting to interpret the statistics.

Todd (1952) states that

> The value of many of the statements made on death certificates is highly problematical, especially the popular diagnoses given to the very old, such as myocardial degeneration, coronary arteriosclerosis, and cerebral arteriosclerosis.

He also contends that, rather than make a precise diagnosis on insufficient grounds

> those concerned with administrative medicine should ask for precision only when precision is possible. ...If it were laid down that indefinite terms like senility, cardiac failure, and 'sudden cardiac death without demonstrable basis' *should* be used when no gross

cause of death can be found, the value of the statistics relating to the definite causes of death would be enhanced.

Provided it is *not* always possible to make a precise diagnosis (which is a medical question), the logic of this point of view must be admitted. However, some evidence will be put forward below that the attempt to classify heart disease into a few broad categories has had at least some measure of success.

In addition to being used as titles for groups of diseases, the terms 'cardiovascular degeneration', 'other myocarditis', etc., will be used as collective terms to cover any of the many diseases classified under these headings, e.g. the certification of a death as due to 'other myocarditis' will mean the certification of one of the diseases classified as 'other myocarditis'.

The death-rates for degenerative cardiovascular-renal diseases (Fig. 5) show less pronounced trends than do the constituent causes, so it might be argued that there has been little real change in the mortality from the various diseases included in this group, and that the trends shown for the separate diseases are due merely to improved accuracy of diagnosis and changing fashions in certification, which have resulted, for example, in the transfer of deaths from other myocarditis to coronary disease. If this view is accepted it is useless to proceed further. However, it appears to me inconceivable that in 19 years, including a major war, there have been no real changes in the mortality from the various constituent diseases—there must surely have been improvements in medical treatment, changes due to altered social conditions and changes in diet which will have had some real effect on the various forms of cardiovascular-renal disease.

Prior to the war the death-rates for coronary disease, cardiovascular degeneration and other myocarditis were all increasing, while deaths recorded as due to senility were decreasing rapidly. The net effect was that degenerative heart disease (with senility) was also increasing. The other diseases of the cardiovascular-renal group were on the whole decreasing, thus keeping the cardiovascular-renal mortality fairly constant. The increase in degenerative heart disease (with senility) could well have been due in large measure to what Dr Reid has called 'a good deal of switching of diagnostic labels' and also perhaps to more accurate diagnosis and a greater recognition of the importance of heart disease.

But this can hardly be the whole story, for degenerative heart diseases (with senility) show a fairly constant pre-war increase at all ages for females, though for males the increase is steeper at the young ages and gets less steep with increasing age. After the war the male rates appear to be definitely increasing while the females are only increasing slowly. From this it may be argued that there has been some real change in mortality, as it is unlikely that differences in diagnosis and certification would throw up differing features for the two sexes, unless perhaps the quality of medical treatment and diagnosis varied between the sexes (e.g. until 1948, males might have been more often treated as panel patients than females).

As stated in Part II, groups 94 (coronary disease) and 93*c* (1) (cardiovascular degeneration) contain some conditions which may differ only in description, nevertheless, the graphs for these two groups present rather different features. Those for coronary disease are much more regular in appearance and, while the war-time trough is clearly evident, it is less noticeable than in the case of cardiovascular degeneration, at least so far as the older age-groups are concerned. (The war-time trough is later suggested as having

something to do with changes in diet during the war.) Also the high mortality of 1947 (almost certainly due to the severe winter) which is a prominent feature in the graphs for cardiovascular degeneration is only evident as a slight upward distortion of the coronary-disease graphs. If these explanations of the two features are accepted, it appears that coronary disease is comparatively little affected by what may be called temporary external conditions, and also that the deaths classified as coronary disease do form a distinct group. While all deaths from coronary disease may or may not be certified as due to this cause, it does at least appear that most of those so certified will have been due to some common cause or causes.

After the war-time trough, which seems to have reached its greatest depth about 1943, coronary disease continued to increase rapidly, cardiovascular degeneration increased more slowly, while senility continued to fall rapidly and other myocarditis also fell rapidly. As mentioned in Part II, the rate of fall for other myocarditis was not proportionate at all ages (as was the case with coronary disease and cardiovascular degeneration), being greater at the younger ages than at the old ages. In my opinion this is evidence that the fall is at least partly, perhaps mainly, due to the transfer of deaths to coronary disease and cardiovascular degeneration, and the reduction in mortality is less at the older ages because of the generally acknowledged greater difficulty in certifying the cause of death in old persons. The main reason for the transfer of deaths seems likely to be an increase in the proportion of certificates which mention arteriosclerosis or hypertension (thus placing a disease previously in groups 93*c* (2), (3) or 93*d* in 93*c* (1)). Coronary disease and cardiovascular degeneration have thus increased by transfer from other myocarditis and senility.

The rapid increase in coronary disease mortality has been discussed by Ryle and Russell (1949), who concluded that the transfer of deaths from other types of degenerative heart disease was not the complete explanation. They reached this conclusion by a comparison of the actual 1945 death-rates (the latest year for which they give data) with those 'expected' by a continuation of the pre-war trend. As Fig. 1 shows the 1945 actual deaths are less than this 'expected'. Ryle and Russell therefore concluded that coronary-disease mortality was increasing at a slower rate than pre-war. However, now that figures are available for years after 1945, the nature of the war-time trough has become more apparent, and it is seen that coronary disease is now increasing faster than pre-war.

Clinical experience and Morris's (1951) investigations indicate that coronary disease is now more prevalent than it used to be. How much of the increase is due to certification and how much to a real increase remains to be determined. However, the opinion is hazarded that the observed increase is largely due to transfer of deaths from other categories of heart disease, but that there has also been some real increase.

Recently there have been encouraging accounts of the treatment of coronary thrombosis with anticoagulants. Gilchrist (1952) reports that the use of these drugs halved the death-rate in the first 6 weeks after attack—and Morris *et al.* (1952) found that 70% of the deaths from coronary heart disease in male medical practitioners aged 40–64 took place within a month of the first attack. Perhaps the present upward trend of the coronary-disease death-rates will soon be a thing of the past, although there is always the possibility that the use of anticoagulants may merely change the age distribution of the deaths by postponing ultimate death from the disease.

Social-class mortality

A well-known feature of coronary-disease mortality is its peculiar social-class distribution. Unfortunately, the latest complete information on this matter covers the years 1930–32 (Registrar General, 1938), a time when Morris *et al.* (1952) state 'the clinical recognition of "coronary thrombosis" was not yet very common in this country'. At this time the 4th International List of Causes of Death was in use, together with the old rules for classifying multiple causes of death (see Part I) and on this basis the Registrar General (1938) gives mortality figures for coronary disease (present group 94) and myocardial disease (present groups 93 *a*, *b*, *c*, *d*). Table 5 sets out these results; the columns headed 'married women' give the mortality of married women classified according to their husbands' social class.

Table 5. Social-class mortality, 1930–32

Social class	Coronary disease				Myocardial disease			
	Males		Married women		Males		Married women	
	S.M.R.	P.M.	S.M.R.	P.M.	S.M.R.	P.M.	S.M.R.	P.M.
I	237	60	157	34	77	156	54	146
II	148	34	126	21	92	172	75	162
III	95	21	93	14	94	192	99	185
IV	66	16	84	11	105	190	110	180
V	67	13	89	11	122	205	129	197

S.M.R. = standardized mortality ratio for ages 35–65.
P.M. = proportionate mortality per 1000 deaths from all causes for ages 65 and over.

Coronary disease is seen to show mortality decreasing from social class I to social class V, while myocardial disease shows an opposite gradient of increasing mortality from social classes I to V. The wives show the same features as the males but with less steep gradients. A number of suggestions have been put forward to explain the unusual mortality gradient of coronary disease; to be convincing these must apply both to men and to their wives. The Registrar General (1938) suggested social-class differences in diet, partly on the grounds that the mortality from appendicitis and some other digestive diseases showed similar social-class gradients. He also mentioned that physical exertion or nervous peculiarities may have something to do with the prevalence of coronary disease in the professional classes. Ryle & Russell (1949) and Stewart (1950) put forward the greater mental stress to which men in the highest social class are supposedly subject. Rentoul (1951), however, wonders whether we are really able to say that the manual worker does actually experience less mental stress than a professional man, while he and Stocks (1951) both say that the absence of physical effort in class I as compared with class V might equally well be the reason for the difference in the mortality rates. Platt (1951), however, in an amusing letter points out that differences in death certification may well be the cause. He argues, and his argument appears to me very cogent to the conditions of 1930–32, that the higher the social class of a man (or his wife) the more likely he is to go to a specialist or cardiologist and so eventually

have his death ascribed to the more specific coronary disease rather than the myocardial disease used for the less fortunate manual worker of class V. Logan (1952) has investigated the social-class differential for coronary disease and myocardial disease (as classified according to the 4th International List). Table 6 shows his comparison of the proportionate distribution of male deaths aged 45–74 by social class for the two categories of heart disease in 1930–32 and 1949.

Table 6. Percentage distribution by social class of male deaths aged 45–74 from coronary disease and myocardial disease

Social class	Coronary disease		Myocardial disease	
	1930–32	1949	1930–32	1949
I	9	5	3	3
II	28	19	17	15
III	39	47	41	44
IV	12	13	18	17
V	10	14	20	20
Unoccupied	2	2	1	1
Total	100	100	100	100

It will be seen that the distribution of deaths by social class in 1930–32 hardly differed from that of 1949 for myocardial disease, but for coronary disease the proportion of deaths in the higher social classes has decreased and the proportion in the lower classes increased. If, therefore, it is assumed that there have been no great changes in the proportions or age distribution of the population in each social class, it can be concluded that in 1949 the class gradient of coronary-disease mortality was still in the same direction as in 1930–32 but was less steep.

Now that the Registrar General (1952) has published the results of a 1% sample of the 1951 census records, it is possible to carry this investigation a stage further. Table 7 gives the percentages of the total male population in each social class in 1931 and 1951 for those age-groups where comparable data are available. This table shows that there has been very little change in the

Table 7. Percentage of total male population in each social class and age-group at 1931 and 1951 censuses

Social class	Age-group									
	45–54		55–64		65–69		70 and over		45 and over	
	1931	1951	1931	1951	1931	1951	1931	1951	1931	1951
I	4	3	4	4	4	3	4	4	3	4
II	18	18	19	18	19	18	21	19	19	18
III	45	48	42	44	42	44	43	44	44	46
IV	16	17	17	17	18	17	17	17	17	17
V	17	14	18	17	17	18	15	16	17	15
Total	100	100	100	100	100	100	100	100	100	100

distribution of the male population into social classes, certainly not sufficient to account for the figures of Table 6. It is therefore safe to conclude that the recorded deaths show a considerable reduction to have taken place in the social-class mortality gradient for coronary disease since 1931.

The results of this investigation mean, in my opinion, that Platt's (1951) explanation as applied to the 1930–32 situation contains a great deal of truth, particularly since the explanations of mental stress and physical activity seem unlikely to apply to the corresponding extent to the married women in each social class. However, we must await the publication of social-class mortality based on the 1951 census before this discussion can be carried further. Then it will perhaps be possible to consider the class mortality separately for the three types of heart disease for which death-rates are given in this paper, instead of only comparing coronary disease with other degenerative heart disease as was done by the Registrar General (1938); it will be interesting to see whether the social-class gradient of cardiovascular degeneration is comparable with that of coronary disease (as I would expect) or with other myocarditis.

Differences between the sexes

It might be argued that any observed variation in the mortality sex-ratio for a given cause of death must reflect a genuine change in relative mortality, since changes in diagnosis should affect either sex equally. However, the validity of this contention is open to question. It is possible, as already mentioned, that different standards of medical attention were received in the past by men and women, although the advent of the National Health Service should remove any such differentiation. Further, since the age incidence of coronary disease is different in the two sexes, improvements in diagnosis and treatment could quite easily disturb the mortality sex-ratios. Females live longer than males, and it may be that the ageing process starts later in females, so that a more appropriate comparison would be of the male death-rates with the female rates for an older age. A later onset and different rate of ageing in females could be the reason that the sex-ratios shown in Table 4 decrease with increasing age. If there is anything in this proposition the sex-ratios might well be expected to show a fictitious time trend.

Thus the tendency for the sex-ratios of degenerative heart disease and cardiovascular-renal disease to increase over the 19 years could be due to the use of an unsatisfactory measure of comparison or to a real change in the sex difference or, more likely, partly to both. However, the contrasting trends at the older ages for coronary disease and cardiovascular degeneration (downward trend) and other myocarditis (upward trend) can hardly be explained in this way. Unless the standard of medical attention differs between males and females much more than it seems reasonable to suppose, then the contrasting sex-ratio gradients must be due to some real change in the sex difference and may be allied to the apparent reduction in the social-class mortality gradient for coronary disease over the 19 years which has already been discussed.

Apart from any question of trends with time and age the sex-ratio of coronary disease is very high, particularly at the younger ages, much higher than for cardiovascular degeneration or other myocarditis. Here the high value could be explained by a later start of the ageing process in females as suggested above. There are, in fact, certain physiological differences between the sexes which may have some influence on mortality. For instance, Dock (1946) has

pointed out that the thickness of the intima (or inner coat) of the coronary arteries is greater in males than in females, and he believes this to be related to the higher male mortality from coronary disease. Also if the social-class differences are to any extent occupational differences, and mental stress or physical exertion have any effect on coronary-disease mortality, then, so far as these differ in their incidence on the two sexes, sexual differences in mortality will arise. In fact, it seems certain that there is a real difference between the sexes, as regards mortality from the various forms of heart disease.

The war-time trough

The war-time trough in the trend of the death-rates is a most interesting feature of the diagrams and one which might provide a unique opportunity to throw some light on the causes of degenerative heart disease. Other investigators have usually dealt with mortality over periods of several years, so that the nature of the war-time trough has not been brought out so fully as when individual years are used.

Before proceeding further, consideration must be given to the question whether the war-time trough represents a real decrease in heart-disease mortality. A trough in the death-rates could have arisen if the registration of deaths had become defective during the war years so that all deaths were not registered. Another possibility is that the Registrar General's estimates of population (which form the denominator of the death-rates) were too high during the war. If either of these events had taken place then the death-rates from all causes excluding degenerative cardiovascular-renal disease would also show the war-time trough—in fact, these death-rates show a slight suggestion of a trough, but even if it does actually exist the shape is quite different and the minimum occurs about 1945, which is a year or two later than for heart disease (Fig. 8). These two possibilities can therefore be excluded. Changes in the accuracy and fashion of diagnosis of cause of death could produce continuous trends, but it is difficult to imagine how they could cause a decrease and then an increase in the death-rates; these changes can therefore be excluded as a possible reason for the war-time dip.

Having excluded the artificial causes, the conclusion is that the war-time trough is due to real causes, and the fact that it has different characteristics in the different heart-disease groups is further evidence for this conclusion, and also for the belief that the groups do represent different types of disease. If the trough is real it also seems reasonable to think it has been caused by some war-time change in living conditions. Many changes occurred during the war which affected the civilian population, e.g. changes in diet and food consumption, consumption of alcohol and tobacco, occupation, the state of mental anxiety, internal migration and evacuation. The war-time trough is present in the death-rates of both males and females, so any explanation must be applicable to both sexes.

Dealing with the possible causes of the reduced heart-disease mortality during the war, the Registrar General (1949) writes:

> Possibly conditions of full employment with consequent increase in available income for food in large numbers of families, coupled with a more equal distribution by rationing leading to less over-indulgence in food, were the main factors responsible. It is difficult to suggest any other reason which is adequate.

Also, as already mentioned, in 1938 the Registrar General suggested that food might have something to do with the social-class differential mortality. Thus

for some years it has been thought that food had something to do with heart disease, and there is some evidence that a certain constituent of human diet, known as cholesterol, may have some effect on heart-disease mortality. The principal dietary source of cholesterol is eggs, and it is also contained in butter, cheese, milk and some other foods. Fig. 6 shows the average consumption of cholesterol per head of population in the United Kingdom for each of the years 1940–50 as a percentage of the pre-war (1934–38) consumption. (See Appendix for details of calculation.) This diagram shows that the consumption of cholesterol fell after the start of the war, rose to a maximum (below the pre-war level) in 1945, then fell again for two years and finally rose above the pre-war level in 1950. The correlation of this curve with the mortality curves is not high, although there is some similarity; the death-rates show no evidence of the 1945 maximum in cholesterol consumption and the subsequent minimum. However, if cholesterol has anything to do with heart-disease mortality its effect is probably cumulative and comparatively long-term, which could perhaps account for the 1945 maximum having no counterpart in the death-rates and also for the fact that the mortality trough did not appear until 1941, although the high mortality due to the cold winter of 1940 (see section on effects of weather) may have masked its appearance in 1940. It must also be remembered that the food-consumption figures relate to the average consumption per head, and that in addition there have been considerable differences and changes in the food consumption of various groups of the population. For example, rationing probably had less effect on the diet of those living in rural areas than on the better-off persons in towns. On the other hand, full employment and the equal sharing of the available food resulted in an improvement in the diet of the working class in towns.

Malmros (1950) gives some diagrams showing the changes in 'arteriosclerosis (including diseases of the coronary arteries)' mortality compared with the consumption of certain foodstuffs in the U.S.A. and several Scandinavian countries. It is interesting to note that in the Scandinavian countries the consumption per head of population of eggs and fats (the principal sources of cholesterol) fell during the war and later rose; in these countries arteriosclerosis mortality also fell and rose. On the other hand, in the U.S.A. the consumption of eggs per head increased throughout the war and that of total fats only decreased slightly, while the arteriosclerosis mortality increased steadily. Strøm and Jensen (1951) also consider the relation between cholesterol consumption and mortality from diseases of the circulatory system in Norway —both fell during the war years and later rose.

The crude death-rates for heart disease in many other countries were extracted from the publications of the World Health Organization (1951, 1952). It was found that many European countries showed the war-time trough (e.g. Denmark, England and Wales, Italy, Netherlands, Scotland, Sweden and Switzerland), while the trough was not evident in other countries where it might have been expected (e.g. Belgium and France). No trough was found for Australia, Canada, Eire, New Zealand or U.S.A. However, it cannot be said that these facts, although suggestive, support the cholesterol theory without making a detailed study of the mortality and population statistics and obtaining information on the changes in diet during the war years in each separate country. (In some of the countries mentioned the methods of allocating deaths to cause leave something to be desired.)

It is not, of course, possible to prove statistically that cholesterol (or any-

thing else) is the cause of the war-time trough in rates of mortality from heart disease; it is possible only to show that the changes in the two items are correlated, and even then the correlation may merely be 'nonsense correlation'. It remains a medical and biological matter to show that the consumption of cholesterol does have an effect on heart-disease mortality. If this can be done then the statistical findings add their weight in support of the cholesterol theory.

The biological evidence has been reviewed by Firstbrook (1951) and seems to indicate some association between cholesterol and atherosclerosis (atherosclerosis is a less general term than arteriosclerosis and arteriosclerotic diseases are included in groups 93*c* (1); 94; 83*a*, *b*, *c*; 97; 131*b*). Atherosclerotic lesions have a high cholesterol content; the average concentration of cholesterol in the sera of men who have experienced myocardial infarction is higher than in those of the same age without clinical evidence of coronary disease; races living on low-cholesterol diets show low incidence of atherosclerotic disease; typical atherosclerotic lesions in animals have never been produced without the administration of cholesterol, and can be produced by cholesterol feeding in the rabbit, chicken, dog, guinea-pig and hamster, but not in the rat.

Firstbrook also mentions that severe atherosclerosis has been found to be at least 10 times as common in the obese as in the lean in each age-group over 35—obesity is often due to overeating, which presumably also involves the consumption of additional cholesterol. However, obesity also places additional strain on the heart.

Firstbrook concludes

> on the basis of present evidence it would seem that the initiating cause of experimental atherosclerosis is the administration of cholesterol.... So far as dietary cholesterol is concerned, although the *circumstantial* evidence is considerable there is no *direct* evidence for the hypothesis that dietary cholesterol is involved in human atherosclerosis.

Moreover, medical opinion seems divided on the question of whether human atherosclerosis results from ingestion of cholesterol and on the value of cholesterol-free diets for those already suffering from atherosclerosis.

It is interesting to notice that coronary disease and cardiovascular degeneration, both of which are associated with arteriosclerosis and hypertension, show the war-time trough (Figs. 1, 2). On the other hand, other myocarditis, which comprises deaths where there is no mention of arteriosclerosis or hypertension, does not show any evidence of the war-time trough (except in those aged 75 and over) (Fig. 3), although this may merely be masked by the general fall in mortality from this cause since 1940. Of the other diseases included as degenerative cardiovascular-renal diseases, cerebral haemorrhage (83*a*) shows evidence of the war-time trough (imposed on a falling tendency), cerebral embolism, thrombosis and softening (83*b*, *c*) also shows the trough (Figs. 7, 9). Arteriosclerosis (97) (a residual group excluding coronary, or renal sclerosis and cerebral haemorrhage) and arteriosclerotic kidney (131*b*) show the trough in the two oldest age-groups, other age-groups being doubtful. All these diseases contain many conditions involving arteriosclerosis and hypertension and show the war-time trough which may be due in some way to cholesterol consumption—as Firstbrook says, 'the circumstantial evidence is considerable'.

It may also be noted that the decrease in the social-class mortality gradient for coronary disease, already discussed, is consistent with the cholesterol theory. The Ministry of Food (1952) shows that by 1950 there had been a

substantial reduction in the pre-war social-class differences in food consumption, largely a levelling upwards by the poorer classes, changes which, on the cholesterol theory, tend to equalize the social-class mortality. The following figures set out the situation:

	1936–7	1950
Average food expenditure	8*s.* 11½*d.*	14*s.* 6½*d.*
Social class range	10*s.* 10*d.*	4*s.* 1*d.*
Range as percentage of average	121	28

These figures and conclusions are subject to many reservations and qualifications for which reference must be made to the original publication. It may, however, be mentioned that the social-class grouping is into four groups on an income basis, and not by occupation as is the case with the Registrar General's classification.

The above is not intended to give the impression that cholesterol is considered the principal cause of coronary and arteriosclerotic disease; it seems generally agreed that psychological causes such as mental stress and emotional shock have a definite influence and are sometimes the initiating cause, but it is not possible to investigate this from the national statistics. Heredity may also result in some persons having a predisposition to heart disease.

Effects of weather

The relationship between mortality and weather is a subject which merits more attention than appears to have been given to it. It is well known that the number of deaths from all causes has a seasonal incidence, being higher in the winter months and lower during the summer; deaths from heart disease also show this feature. Table 8 shows the percentage of the deaths in the years 1940–49 which have occurred in each quarter of the year from the three categories of heart disease and also from three of the additional causes which were combined with heart disease and designated degenerative cardiovascular-renal diseases.

For each cause and sex the quarters arranged in order of decreasing magni-

Table 8. Percentage of deaths in 1940–49 according to quarter of occurrence.—England and Wales

Cause of death	Males				Females			
	1st qtr. (Jan. to Mar.)	2nd qtr. (Apr. to June)	3rd qtr. (July to Sept.)	4th qtr. (Oct. to Dec.)	1st qtr. (Jan. to Mar.)	2nd qtr. (Apr. to June)	3rd qtr. (July to Sept.)	4th qtr. (Oct. to Dec.)
Coronary disease (94)	28·8	22·9	20·8	27·5	28·7	22·8	20·5	28·0
Cardiovascular degeneration (93*c* (1))	32·6	23·6	18·6	25·2	32·3	23·3	19·0	25·4
Other myocarditis (93*c* (2), (3), *d*)	34·5	22·8	18·0	24·7	34·0	22·6	18·4	25·0
Cerebral haemorrhage (83*a*)	30·6	23·6	20·6	25·2	29·5	23·8	21·2	25·5
Cerebral embolism, etc. (83*b*, *c*)	31·3	23·2	20·2	25·3	30·2	23·3	20·9	25·6
Bronchitis (106)	46·4	17·8	10·4	25·4	46·9	15·0	7·7	30·4

tude of percentage of deaths, are first, fourth, second and third, and the average (1906–35) temperatures over England and Wales for the four quarters increase in the same order. Also it is well known that disease is more prevalent during the winter, so it seems reasonable to assume that temperature variations or some other factors correlated with temperature have something to do with the seasonal variation of deaths.

The three categories of heart disease given in Table 8 show certain differences which are of interest. The percentages for coronary disease show much less variation over the year than do those for the other two causes. This is consistent with the remarks made earlier about the comparative insensitiveness of coronary-disease mortality to external influence. It may also be noted that Morris *et al.* (1952) in their examination of the mortality of doctors from coronary disease find no concentration of deaths in any part of the year.

Other myocarditis shows a pronounced maximum of deaths in the first quarter of the year, while the percentage for cardiovascular degeneration is intermediate between those for coronary disease and other myocarditis. These differences do give some confidence in the belief that the present system of classifying heart disease does at least have some measure of success in separating the deaths into three distinct groups and that we are not attempting the impossible and making distinctions without differences. It is, however, still possible to argue that cardiovascular degeneration includes a mixture of deaths which more properly belong to one of the other two groups.

The percentages for the two cerebral-disease groups given in Table 8 are similar and correspond most nearly with those for coronary disease (with which the cerebral diseases have much in common), although the range of variation is somewhat greater. Bronchitis deaths show a wide seasonal variation, nearly half occurring in the first three months of the year, thus differing considerably from the other diseases shown in Table 8. It might therefore be argued that deaths certified as due to bronchitis form a distinct group and should not have been included as due to a degenerative cardio-vascular-renal disease; this matter will be discussed in the next section.

The argument that cold weather tends to increase deaths from heart disease leads one to expect higher mortality in years with severe winters. This is in fact evident from the mortality diagrams already discussed, nearly all of which show 1947 to have had mortality above the level of the current trend and the following year, 1948, to be below this level, perhaps indicating that the severe winter of 1947 had resulted in the death of a number of persons rather earlier than would otherwise have occurred. Table 8 shows that the greatest proportion of deaths occurs in the first quarter of the year, and an examination of the monthly average temperature over the years 1931–49 shows that the largest deviations below the average also occur in the first quarter. Accordingly, the effects of a severe winter would be expected to be shown by an increase in the percentage of deaths in the first quarter of the year; we shall therefore consider the average temperature and the proportion of deaths in the first quarter of each year. Table 9 gives, for England and Wales, the deviation of the average temperature in the first quarter of the year from the first-quarter average of the thirty years 1906–35.

It will be seen that 1947 had by far the most severe winter and was followed the next year by a warm winter. The next most severe first quarters were, in order of decreasing severity, 1942, 1940 and 1941; that of 1940 was preceded by at least nine years without any severe cold in the first quarter.

Table 9. Deviation of temperature from average (1906–35) in first quarter of year—England and Wales

Year	1931	1932	1933	1934	1935	1936	1937	1938	1939	1940
Deviation from average (° F.)	−1·2	+0·5	+0·4	−0·4	+1·7	−0·3	+0·1	+3·4	+1·1	−3·6
Year	1941	1942	1943	1944	1945	1946	1947	1948	1949	
Deviation from average (° F.)	−3·0	−4·5	+2·2	+0·5	+1·0	+0·3	−5·8	+2·6	+1·3	

The above figures are one-third of the algebraic sum of the differences from average for each of the three months January to March.

Many of the mortality curves show a maximum in 1940, but these are usually less conspicuous than in 1947, and sometimes, although being a maximum, the point for 1940 is below that obtained by continuing the trend of previous years. The mortality of 1941 is nearly always less than that of 1940 (as in 1947 and 1948), but 1941 was nearly as cold as 1940, and, although 1942 was the coldest of the three years, there is no sign of abnormally heavy mortality in 1942. Several explanations can be suggested for this:

(i) The deviation from the average temperature is not the best measure of the effect of severe weather on heart-disease mortality—perhaps a short period of very low temperatures has more effect than a longer period of less severe cold or some other factor, e.g. humidity, has more effect than temperature.

(ii) The cold years 1940 and 1941 may have already resulted in the death of many who would otherwise have succumbed to the cold of 1942.

(iii) The increased mortality may have occurred but have been masked by the war-time trough in mortality already discussed.

Table 10 shows the percentages of deaths in the first quarter of each year from 1940 to 1949. Only the male figures are given, those for females being so similar as not to warrant the space required to exhibit them. Percentages could be calculated from 1940 only, owing to the changed methods of classification employed by the Registrar General from that year.

Table 10. Percentage of male deaths occurring in first quarter of year—England and Wales

	1940	1941	1942	1943	1944	1945	1946	1947	1948	1949
Coronary disease (94)	31·7	30·0	29·7	26·6	27·5	29·2	28·2	31·6	26·4	28·2
Cardiovascular degeneration (93*c* (1))	35·4	32·7	34·6	26·7	30·9	32·7	32·5	38·0	28·8	33·0
Other myocarditis (93*c* (2), (3), *d*)	39·6	35·4	34·2	28·5	31·2	34·6	33·9	40·3	30·2	34·4
Cerebral haemorrhage (83*a*)	34·0	30·6	32·0	27·0	27·9	29·1	30·0	35·9	29·1	29·4
Cerebral embolism, etc. (83*b*, *c*)	34·2	32·1	33·2	27·6	28·8	32·5	31·0	37·4	28·3	28·1
Bronchitis (106)	60·7	50·0	46·1	31·6	38·4	46·0	46·3	51·3	37·3	47·0

Like those in Table 8, the percentages for the heart-disease groups in Table 10 are (except in 1942) always lowest for coronary disease and highest for other myocarditis, again showing the smaller sensitivity of coronary disease to the vagaries of weather.

The percentages for the cerebral diseases are nearly always intermediate between those for coronary disease and cardiovascular degeneration. As would be expected from Table 8 and common experience that bad weather is most likely to affect the respiratory system, the percentages for bronchitis are very variable.

Except in the case of bronchitis the percentages for 1947 are always the highest (or very nearly so), while those for 1940, 1941 and 1942 are among the highest. Thus the years having the coldest first quarters also show a high percentage of deaths in the first quarter. The high figures for 1942 indicate that the cold weather did have its effect of increasing the deaths, although this increase is not reflected in the annual death-rate. That the above cannot be the whole story is evident from the comparatively high proportion of deaths in the first quarters of 1945 and 1949. Further investigation, however, shows that January 1945 was the second coldest January of the ten years 1940–49, but in 1949 January and February were above average temperature and March only slightly below average. Bronchitis shows rather similar features except that 1940 has by far the highest percentage.

A reasonable conclusion from the evidence set out above is that the mortality from heart disease is increased in very cold weather, but that the increase is less in the case of coronary disease than for other forms of heart disease.

Bronchitis

The death-rates for bronchitis (Fig. 10) and the various figures for this disease given in the tables show marked differences from those for heart disease and the other causes grouped under the title degenerative cardiovascular-renal diseases. As compared with these diseases, bronchitis shows (i) greater variability of the death-rates, (ii) a high sex-ratio which increases considerably over the 19 years, (iii) a much higher proportion of deaths occurring in the first quarter of the year (nearly 50% as compared with about 32% for heart disease). In view of these differences some justification seems necessary for including bronchitis as a degenerative cardiovascular-renal disease.

A medical text-book (Maxwell, 1948) states that chronic bronchitis is really a degeneration of the mucous membrane on which the effects of infection are often superimposed. It is further stated that the conjunction of chronic bronchitis with heart and similar diseases is so close as to 'give rise to the belief that chronic bronchitis is primarily due to an underlying vascular degeneration rather than to infection'. That bronchitis is often associated with heart disease is statistically evident from the fact that 36% of the deaths certified as due to bronchitis in 1939–45 were stated to have had either myocardial disease (group 93), arteriosclerosis (97) or chronic nephritis (131) as a contributory or secondary cause of death. It is found that, of the bronchitis deaths associated with one of the degenerative cardiovascular-renal diseases mentioned, the proportion occurring in the first quarter of the year is almost the same as the proportion shown in Table 8 for all bronchitis deaths. This may be some evidence that the deaths associated with the cardiovascular-renal diseases do not form a distinct group differing from the remainder of the bronchitis deaths. It therefore seems that, in old people, bronchitis should

not be regarded as the real cause of death but as the initiating cause which only results in death if there is also present some more fundamental degeneration of the organs, often the heart. In this connexion it is interesting to note that in the U.S.A. the certified death-rate from bronchitis is negligible (Pedoe, 1947), a fact which can only be satisfactorily explained by differences in certification practice. If it is agreed that bronchitis is the initiating and not the real cause of death, then the greater variability and high proportion of certified bronchitis deaths in the first quarter of the year do not lend support to the exclusion of bronchitis from the degenerative cardiovascular-renal group, for these facts are merely a reflexion of the incidence of the superimposed symptoms of bronchitis. By similar arguments the high sex-ratios are only evidence of the differing sexual incidence of bronchitis.

In 1930–32 (Registrar General, 1938) bronchitis showed a comparatively steep mortality gradient from social class I to class V. The standardized mortality ratios (ages 35–65) were 31 for class I increasing steadily to 157 for class V. It is possible that this gradient may, either in whole or in part, be only a result of more accurate certification received by those in the higher social classes, although, of course, the gradient can also be explained by a higher incidence of bronchitis among those living in more crowded or less satisfactory social conditions. Possibly the suggestion about certification may be supported by the fact that the social-class gradient of tuberculosis is in the same direction but less steep than that of bronchitis, and the incidence of tuberculosis is known to be influenced by poor social conditions.

The above arguments are the justification for treating bronchitis as a degenerative cardiovascular-renal disease, for if this were not done then a true conspectus of the mortality from this group of diseases would not be obtained. It must, however, be remembered that there is a possibility that a different type of heart disease is involved in those deaths certified as bronchitis from those recorded as heart disease. It has already been pointed out that, while mortality from other degenerative cardiovascular-renal diseases was usually particularly high in 1947, this year was not in any way notable for a high bronchitis death-rate. The extremely high bronchitis mortality of 1940 was very noticeable, much more so than for other cardiovascular-renal diseases. Similar features are shown in Table 10, where, unlike the other cardiovascular-renal diseases, the percentage of bronchitis deaths in the first quarter of the year was much higher in 1940 than in 1947, which latter year shows a percentage only slightly higher than that for 1941. It is of course possible that these differences are due to the different effect of weather conditions on bronchitis as compared with heart disease and have nothing to do with the type of heart disease associated with bronchitis deaths.

CONCLUSION

A brief summary of some of the conclusions indicated by this study will now be given.

(i) Over the period 1931–49 there have been considerable transfers of deaths between the various groups of heart (and related) diseases. Nevertheless, the three broad groups of heart disease show differing features and so point to some success in separating heart-disease deaths into distinct groups. The deaths recorded against the more precisely defined causes are likely to be correctly assigned but incomplete, while the more indefinite causes contain

those deaths where it was not possible to reach a precise diagnosis and include some of the deaths which should have been assigned to more precisely defined causes.

(ii) It is probable that there has been some real increase in the mortality from coronary disease.

(iii) The war-time heart-disease death-rates showed a trough, and it is suggested that changes in food consumption during and after the war may have had something to do with this feature.

(iv) Social-class mortality of coronary disease showed a steep downward gradient from classes I–V in 1930–32. A number of explanations have been put forward, none wholly satisfactory, although each may contain some truth. However, since the gradient appears to have become much less steep by 1949, it is felt that differential diagnosis between social classes was to a considerable extent responsible for the 1930–32 position. It is also possible that changes in food consumption may have had some influence.

(v) In severe winters the mortality from heart disease is increased, probably mainly by the hastening of certain deaths rather than causing additional deaths. This effect is of smaller magnitude for coronary disease than for other forms of heart disease.

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RECENT WORK

Since this note was written several interesting papers have been published. Morris *et al.* (*Lancet* (1953) **2**, 1053 and 1111) have investigated the incidence of first episodes of coronary disease in conductors and drivers aged 35–64 on London's double-decker buses, trams and trolleybuses in 1949–50. The work of conductors involves more physical activity than that of drivers; the conductors experienced less coronary disease than the drivers and what disease they did have was less severe. A grouping of postal workers and Civil Servants according to the degree of physical effort of work showed similar contrasts, the less active having more, and more severe, coronary disease. Several other tests supported the hypothesis that physical effort protects against coronary disease. A classification was made (Morris and Heady, *Brit. J. industr. Med.* [1953] **10**, 245) of 70 of the Registrar General's (1938) occupational groups in social classes III, IV, and V (covering about 80% of all men in these classes) into three categories according to the degree of physical activity involved. It was found that the 1930–32 mortality from coronary disease for ages 45–64 showed an increasing gradient from heavy to light physical activity, while none of myocardial disease, cerebral vascular lesions, arteriosclerosis without cerebral lesions and bronchitis (4th International List) showed any apparent association between mortality and physical activity of work. It is therefore probable that there is some degree of association between physical activity and coronary disease—Morris and Heady do not consider they have established a causal connexion.

Keys (*Amer. J. Pub. Health* [1953] 43, 1399) quotes experimental evidence that the level of cholesterol in the blood serum of human beings (unlike certain animals, as mentioned on p. 84) is little altered by changes in the dietary consumption of cholesterol but appears to be much more affected by variations in the quantity of fats eaten. Since it seems to be generally agreed that coronary disease is related to the concentration of cholesterol in the blood serum, it may be more appropriate to consider the wartime trough in the heart-disease death-rates in relation to the consumption of fats rather than of cholesterol.

If figure 6 had been drawn to show the consumption of fats instead of cholesterol, the graph would not have been fundamentally altered, so that any arguments in the note based on changes in cholesterol consumption would seem to apply equally to changes in the consumption of fats.

APPENDIX

Methods of Calculation

1. *Death-rates.* For 1939 and later years many of the death-rates required for England and Wales are given by the Registrar General (1949, 1951), otherwise they were calculated from the numbers of deaths and estimated populations published in the annual volumes of the *Statistical Review*.

For 1931–38 estimates of the death-rates were required according to the 5th Revision of the *International List of Causes of Death* and the physician's preference (referred to as 'new basis'). The numbers of deaths are only recorded according to the 4th Revision of the *International List* and the priority rules ('old basis'). Many of the estimated rates required are given by the Registrar General (1949) in age-groups for individual calendar years or groups of years. Where the rates are only given for groups of years, the annual figures have been obtained for each sex and age-group by multiplying the annual rate on the old basis by the ratio of the rates for the group of years on the new basis to the old basis. This method was used for the following causes of death: *International List* (5th Revision), numbers 83*a*, 83*b*, *c*, 83*d*, *e*, 93*c* (2), (3), 93*d* and 162*a*, *c*.

For certain causes of death no estimated rates are available. In these cases, annual rates on the new basis were obtained for each sex and age-group by multiplying the old basis annual rates by the ratio of the 1939 rates on the new basis to the old basis. The 1939 deaths are given according to both new and old bases in the *Statistical Review*, 1939, Part I, Medical. This method was used for the following causes of death: *International List* (5th Revision), numbers 97 and 131. The 1931–38 death-rates for bronchitis (group 106) were calculated by apportioning the Registrar General's estimates on the new basis of the total bronchitis deaths in each of these years to age-groups in proportion to the age distribution of the bronchitis deaths in the five years 1939–43 (new basis).

2. *Cholesterol in diet.* The quantities of the various foods per head moving into civilian consumption in the United Kingdom for each year from 1940 to 1949 and the pre-war (1934–38) averages are given by the Ministry of Food (1953).

The quantities of cholesterol in the diet were calculated on the basis of the consumption of eggs, milk, butter and cheese, by applying the cholesterol

contents of these foods as set out in Table 11, which have been extracted from the figures given by Davidson and Anderson (1947).

Table 11. Cholesterol content of foods

Food	Cholesterol in mg., per 100 g.
Eggs	600
Milk	12
Butter	250
Cheese	160

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Note on the figures.

Each of these figures has the same horizontal time scale and all (except fig. 6) show the death-rate for the stated age-groups for England and Wales per 1000 living as the vertical scale. The vertical scales of figs. 1, 2, 7, 9 and 10 are the same, but figs. 3, 4, 8 and 5 are drawn on successively smaller scales. The scales of figs. 4 and 5 have a part omitted, while that of fig. 8 does not commence at zero.

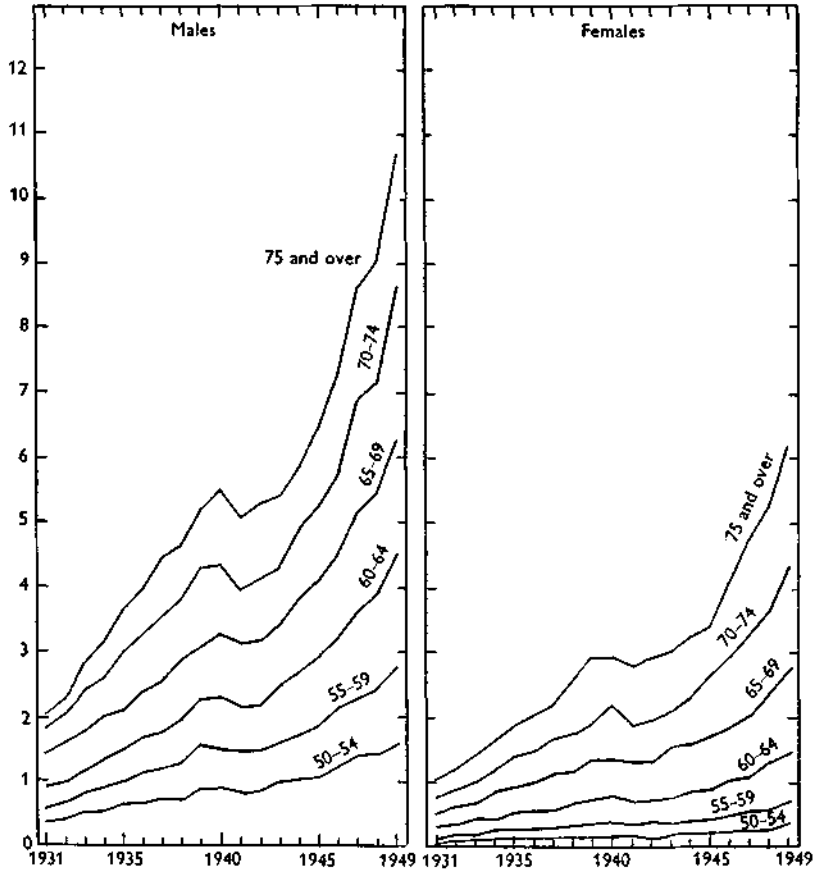

Fig. 1. Coronary Disease (94).

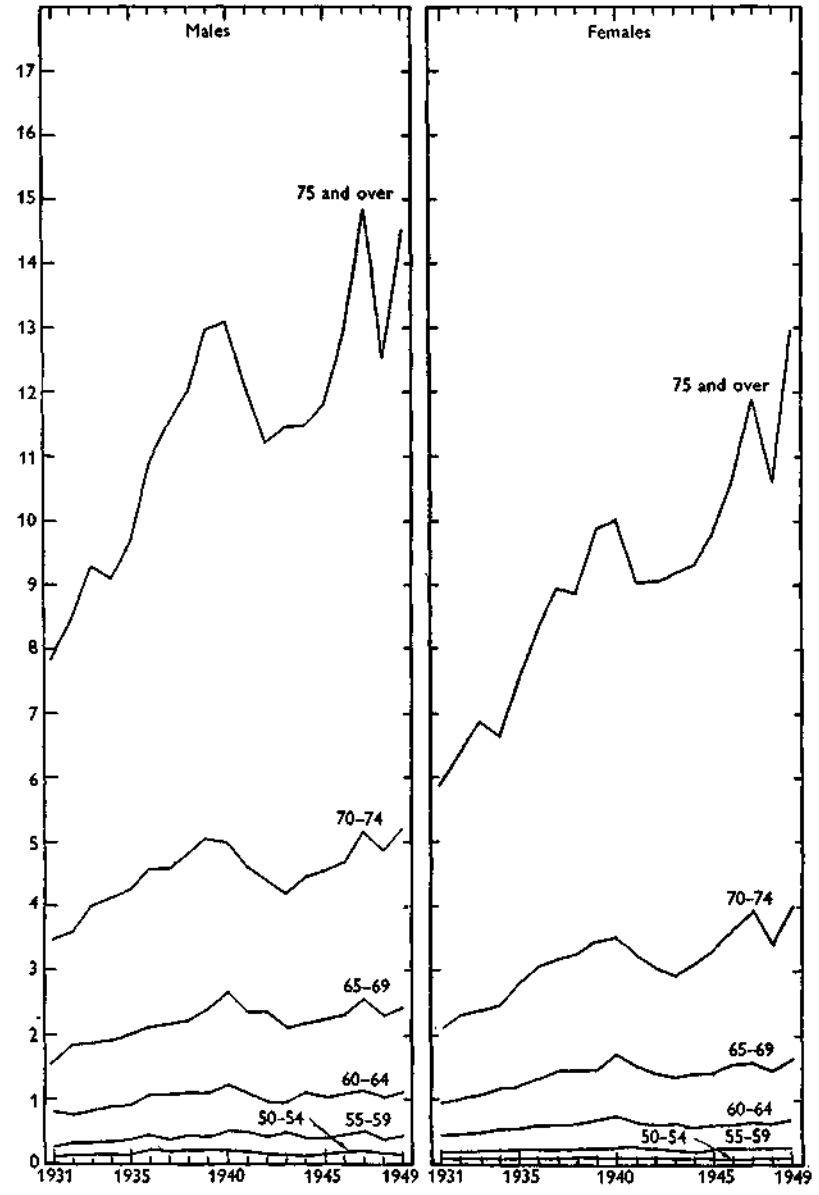

Fig. 2. Cardiovascular Degeneration (93 *c*(1)).

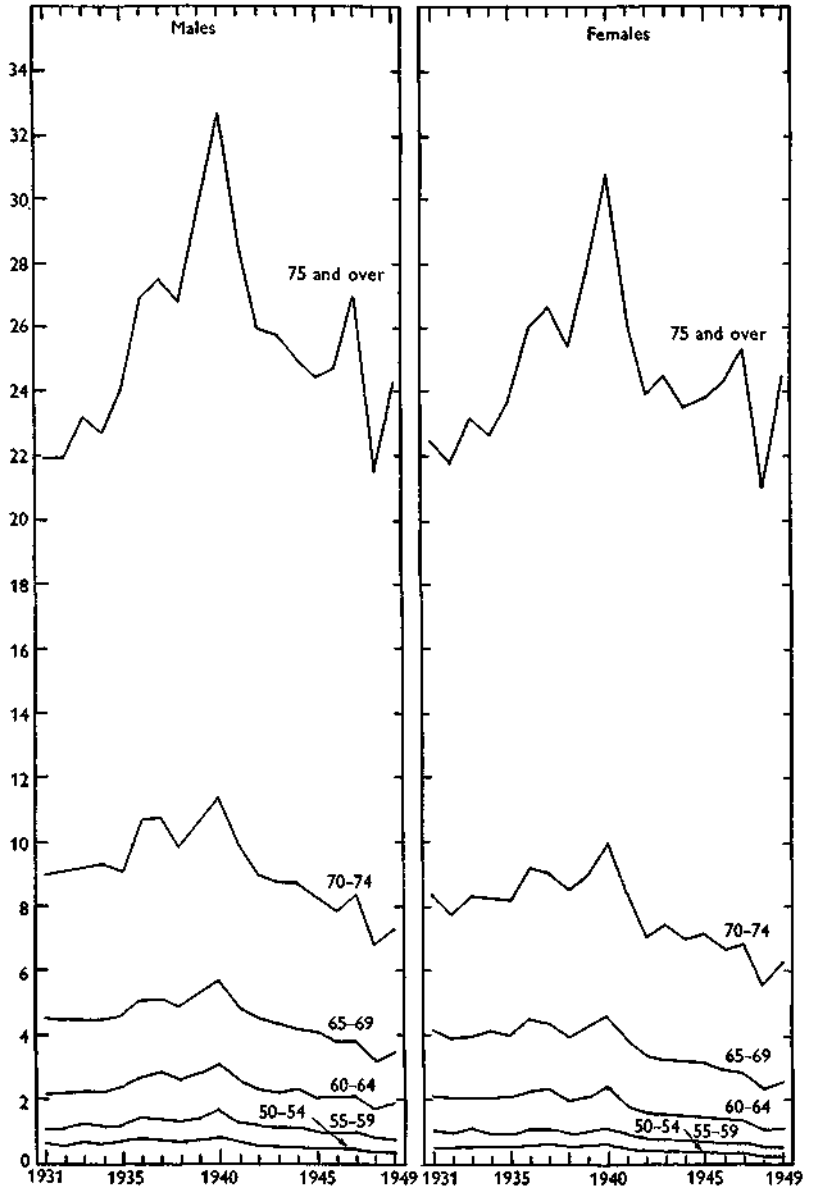

Fig. 3. Other Myocarditis (93 *c*(2), (3); 93 *d*).

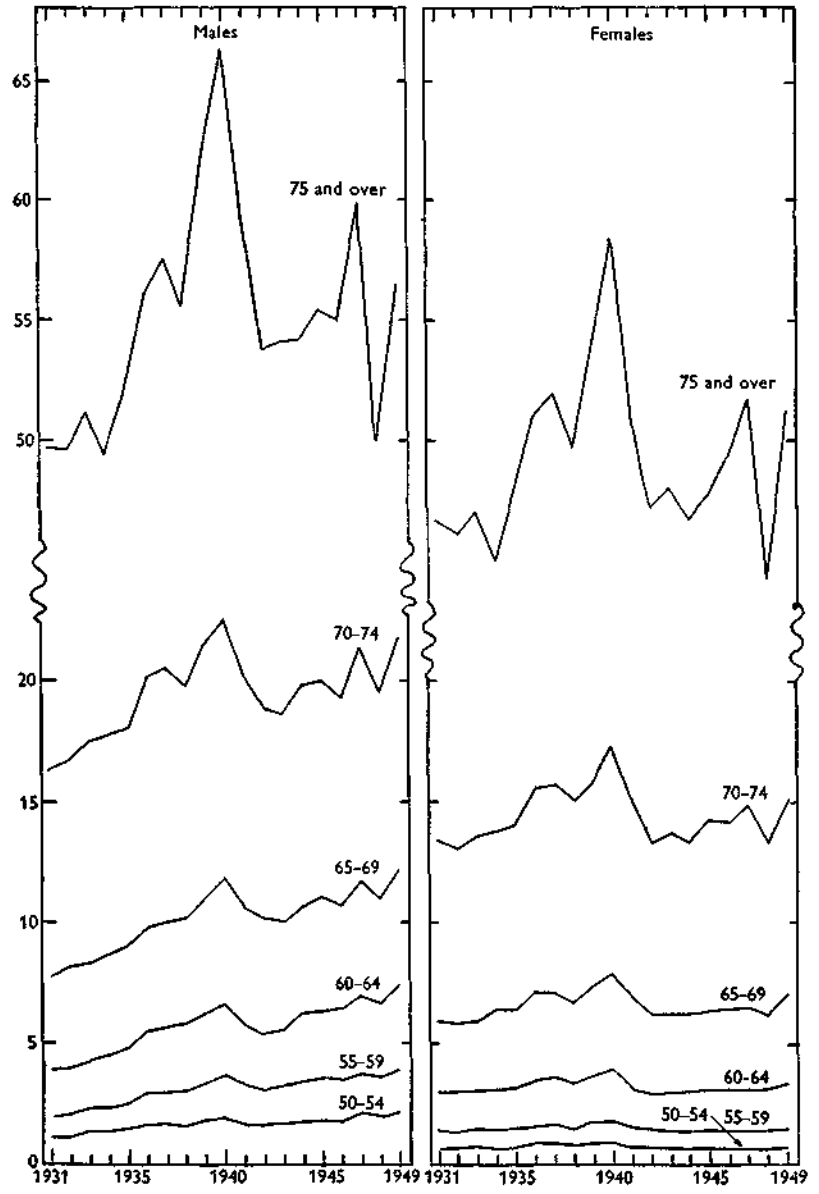

Fig. 4. Degenerative heart disease and senility (93*c*, *d*; 94; 162*a*, *c*).

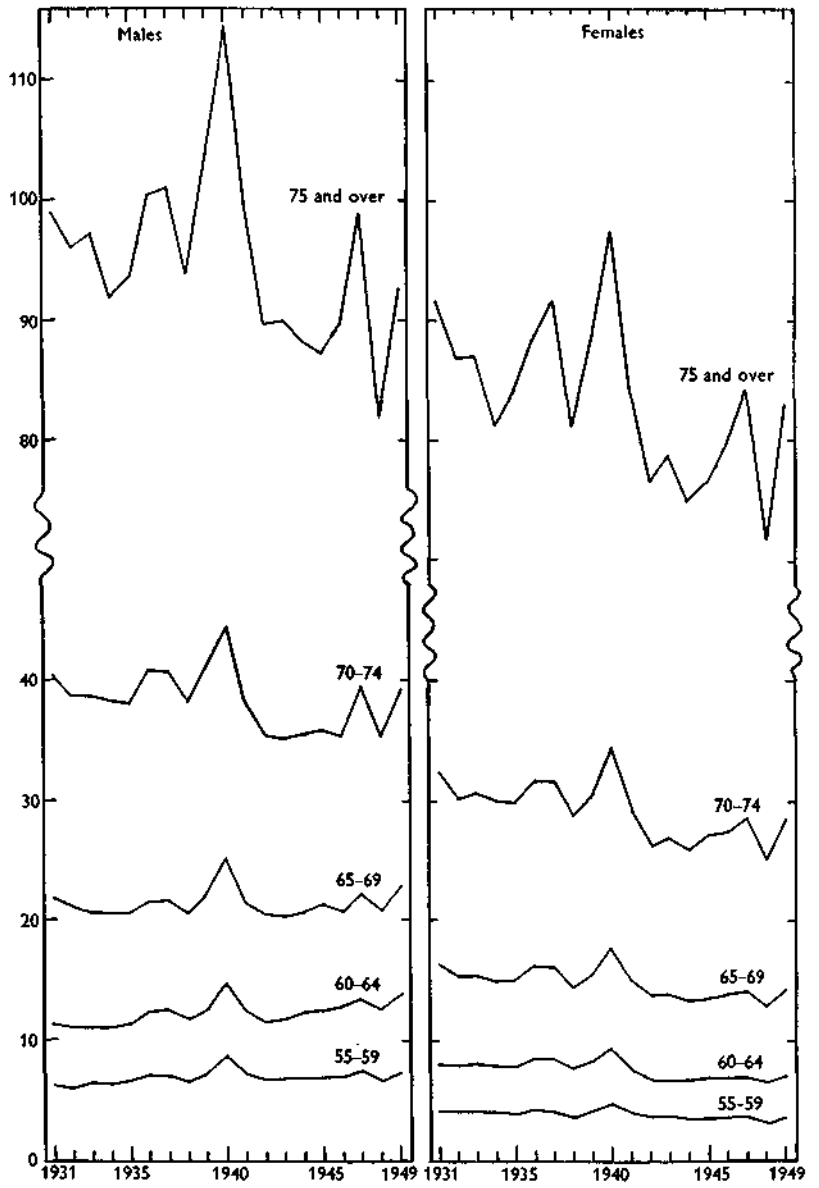

Fig. 5. Degenerative cardiovascular-renal diseases (83; 93*c*, *d*; 94; 97; 106; 131; 162*a*, *c*).

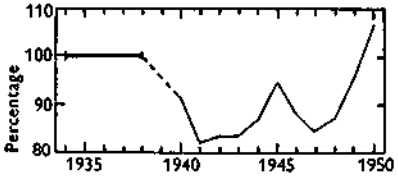

Fig. 6. Cholesterol consumption as percentage of that in 1934–38.

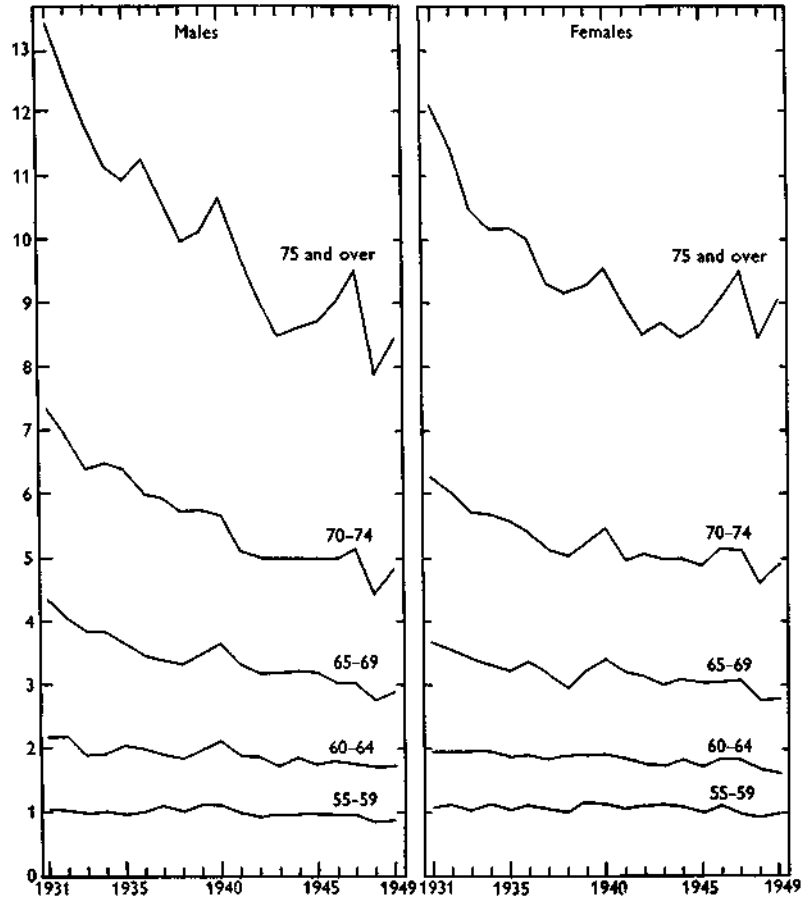

Fig. 7. Cerebral haemorrhage (83*a*).

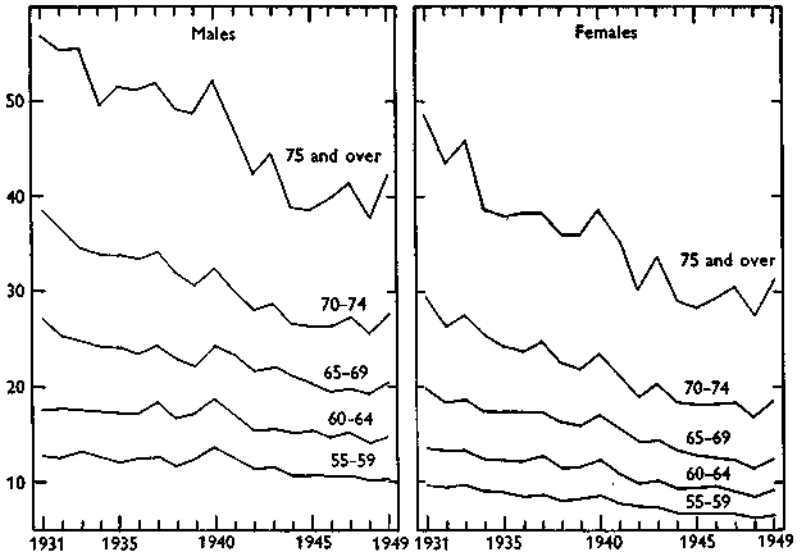

Fig. 8. All causes excluding degenerative cardiovascular-renal diseases.

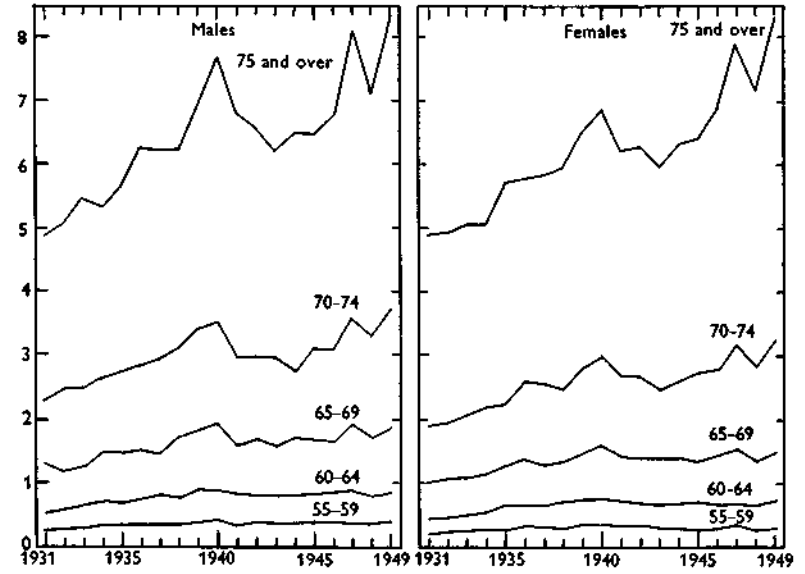

Fig. 9. Cerebral embolism, thrombosis and softening (83*b*, *c*)

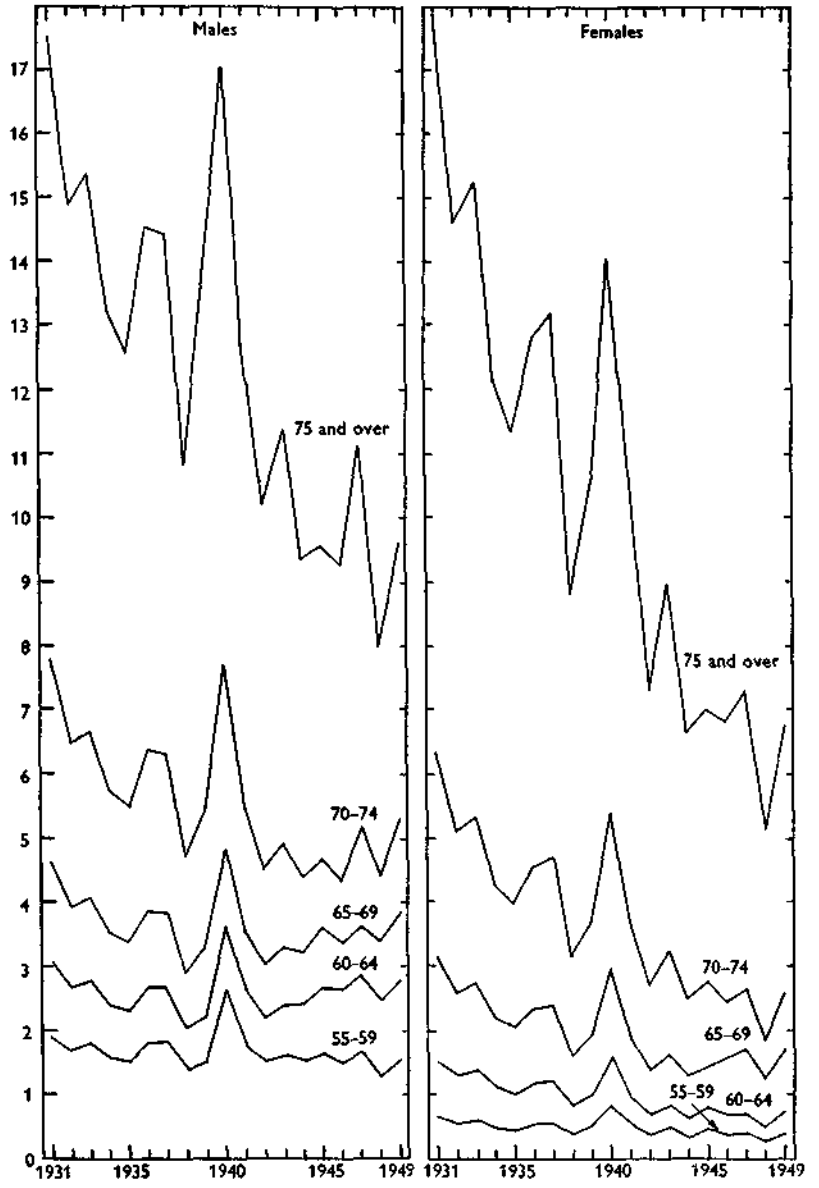

Fig. 10. Bronchitis (106).