

The origin of the modern epidemic of coronary artery disease in England

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SUMMARY. *A review of all the cases of the coroner's court for the Liberty of Ripon and Kirkby Malzeard in Yorkshire from 1855 to 1926, and those of 1981–83, showed that the number of deaths from acute coronary artery disease was very low in Victorian times, suddenly increased in the period 1906–10, and was very high in 1981–83. The population of the area was stable throughout the period at around 22 000. The number of postmortems for myocardial infarctions carried out in London hospitals was very low between 1907 and 1914, but greatly increased between 1917 and 1923. A study of autopsies in the City of London showed that the increase in the number of deaths from coronary artery disease began in 1909/10.*

Introduction

CORONARY artery disease has always been with us. Deaths with all the characteristics of coronary artery disease were described by Egyptian papyruses, mortuary inscriptions and tomb reliefs as early as 3000 BC. They have been described sporadically since then,¹ although the existence of a third circulation — the coronary circulation — was only recognized by William Harvey (1578–1657) in the seventeenth century. William Heberden delivered a famous lecture on angina pectoris in 1772, although he did not associate this disorder with disease of the coronary arteries. A few years later this association was noted by Edward Jenner and Caleb Hillier Parry of Bath; and in 1809 a Scot, Allan Burns (1781–1813), proposed the myocardial theory of angina pectoris, which he regarded as originating from some organic lesion of the nutrient vessels of the heart.²

For more than 100 years after Heberden's lecture clinicians and pathologists alike were preoccupied with 'fatty change and fibroid degeneration of the heart', totally ignorant of myocardial necrosis secondary to obstructive coronary artery disease. Robert Virchow (1821–1902) thought fibrosis of the heart 'inflammatory' in origin, and Richard Quain (1816–1889) was among the exponents of 'fatty degeneration of the heart', a theory persisting among medical men until the 1930s. However, in 1880 Carl Weigert broke the spell and clearly described the condition of myocardial infarction, correlating disease of the coronary arteries with myocardial changes. He recognized the aetiological role of coronary atherosclerosis and the different effects of myocardial ischaemia, fibrosis from gradual ischaemia and necrosis from abrupt ischaemia. Postmortem diagnoses of coronary artery thrombosis began to be made, and finally in 1912 James Bryan Herrick demonstrated electrocardiographic changes and brought the condition to the world's notice.² Acute coronary deaths were rare until relatively recently: the London Hospital performed as few as one or two autopsies a year for myocardial infarction and/or coronary artery thrombosis before World War I and a total of 13 between 1907 and 1914, while Guy's Hospital performed only three between 1907 and 1914. Morris³ considered the greatest increase to have been between 1917 and 1923.

At the beginning of World War I, medical opinion was that

angina pectoris could cause sudden death, but it was still considered unusual. James Mackenzie (1853–1925) recognized very few cases, and even by 1931–32, when I was clinical assistant to W.T. Ritchie (chief cardiologist in Scotland) only three or four cases of angina pectoris were admitted to his wards at the Edinburgh Royal Infirmary in a year, and in 1933 the main cardiological clinic in Vienna (the Wenckebach Clinic) only saw one or two cases a week, in contrast to the 10 cases seen by the average general practitioner each year in the 1980s.⁴

The reality of the coronary artery disease 'epidemic' has been doubted, but two recent studies have put its existence beyond question. Slater and colleagues⁵ have investigated American data from 1931 to 1979, studying age-standardized death rates and mean ages at death by sex for acute and chronic coronary artery disease deaths and for diagnoses liable to be confused with them. They concluded that there was an epidemic of acute coronary deaths which affected men more than women and whose peak has passed. This contrasted with the much more stable pattern for chronic coronary deaths. Anderson and Halliday⁶ pointed out that the sex ratio of deaths is relatively unaffected by changes in diagnosis and in classification, and used an elegant comparison of the sex ratios for deaths from various 'heart' and 'near heart' diagnoses for England and Wales for the years 1921 to 1971 to confirm the increase in acute, but not in chronic, cardiac deaths in men. The divergence in the sex ratios started in 1920, but their method⁶ uses population changes and would not be expected to pick up the earliest evidence.

Accurate dating of the origins of the coronary artery disease epidemic could help to pinpoint the years which should be examined for causative sociological changes. This study approaches the problem by making use of a series of coroner's reports.

Method

Four years ago the author received all the records of the coroner's office for the Liberty of Ripon and Kirkby Malzeard in Yorkshire from 1855 to 1926, with the exception of the year 1895 for which there were no records. The coroner's records for this period include an inquisition file on which the decision of the court is stated, information files recording the depositions of witnesses, and the West Riding Constabulary file showing the police record; other notes are sometimes present. Nearly all the records are complete, and were kept consistently by three generations of a Ripon medical family.

The cause of death was assessed for every case, paying particular attention to deaths where a cardiac cause was a possibility. In the early years the court decision was frequently given as 'death from natural causes' or 'visitation of God' with no attempt at a somatic diagnosis. This caused some difficulty, but examination of the evidence usually indicated a diagnosis. Deciding that a death was cardiac rather than cerebral in origin caused little problem. A cerebral catastrophe was associated with antecedent headaches, signs of central nervous system involvement, red face and stertorous breathing. In most cases there was enough evidence to indicate whether sudden cardiac deaths were due to cor pulmonale, rheumatic heart disease or other cardiac pathology, or whether they were due to coronary artery disease. Cases reported to the coroner of the Liberty of Ripon and Kirkby Malzeard in 1981–83 were used for comparison.

All suggestive symptoms and reports were recorded and a sudden coronary death was noted if one or more of the following was present:

1. Evidence from a postmortem carried out by a competent pathologist. This was available for 80% of the records of the cases reported to the coroner in 1981–83; in the earlier period few cases were confirmed by postmortem and it had usually been performed by a senior general practitioner or a physician from the town of Ripon, or during World War I by officers of the Royal Army Medical Corps.
2. The opinion of the patient's own doctor that the patient had suffered from heart disease recently or that he had treated the patient in the past for a condition whose description fitted a diagnosis of myocardial infarction.
3. Evidence from witnesses that the deceased had been previously well and had had a sudden attack of angina-like symptoms, or that the deceased was known to have had a bad heart recently, or that he was found dead in bed and had had a previous history of angina pectoris or severe breathlessness on exertion.
4. The verdict of the jury; for instance that the patient 'did labour and languish with a grievous disease of the body, to wit heart disease'.

Results

The coroner's records for the early years showed few sudden cardiac deaths, with only six cases in the first 10 years (up to 1865); from 1904 there was a slight rise until in 1912 11 such deaths were noted. Numbers fluctuated around this level from 1913 to 1926 and were higher during World War I. Up to about 1894 approximately half the sudden cardiac deaths were attributable to coronary artery disease and this rose to about three-quarters between 1907 and 1926; the remainder were due mainly to cor pulmonale, valvular disease and other cardiac pathologies, and to ruptured aneurysms. The number of sudden cardiac deaths reported to the coroner each year, and the cases attributed to

coronary artery disease are shown in Figure 1 with data from the same population for the three years 1981–83 for comparison. There were 115 sudden cardiac deaths reported to the coroner in 1981–83, of which 105 were acute coronary deaths (over 90%). Figure 1 demonstrates that the increase in the number of sudden cardiac deaths over the period 1855 to 1983 occurred in those attributed to coronary disease.

There was little change in the proportion of the population over 65 years of age over the early part of this period (8% in 1861 and in 1911), though changes were apparent by 1931 (11%), and the proportion had increased to 20% by 1981 (Registrar General, decennial censuses (Yorkshire) 1861–1981). The size of the population in the area had remained fairly constant at approximately 22 000 over the period but was rather older and less rural in 1981 than in 1861.

Analysis by age

Inspection of the ages of the cases supports the conclusion that the increase in the number of sudden cardiac deaths was real and had started by 1910. Table 1 shows the number of cases of cardiac death, and those attributed to coronary artery disease, by age group for the 40 years 1855–94, the 20 years 1907–26, and for the three recent years 1981–83. In those aged less than 40 years there were sudden cardiac deaths in the earlier period but none in the recent period, mainly reflecting the improved management of congenital heart disease and the virtual elimination of rheumatic heart disease. For the 40–59 years age group there were one and a half times as many sudden coronary deaths in the three years 1981–83 as in the 40 years 1855–94. For the 60–69 and 70–79 years age groups there were respectively five times and three times as many sudden coronary deaths in the short recent period as in the early 40 years. If the differences in the numbers of years is allowed for this represents an increase of over 50 times. The increases may be marginally over-estimated if some of the earlier cases for which there was insufficient

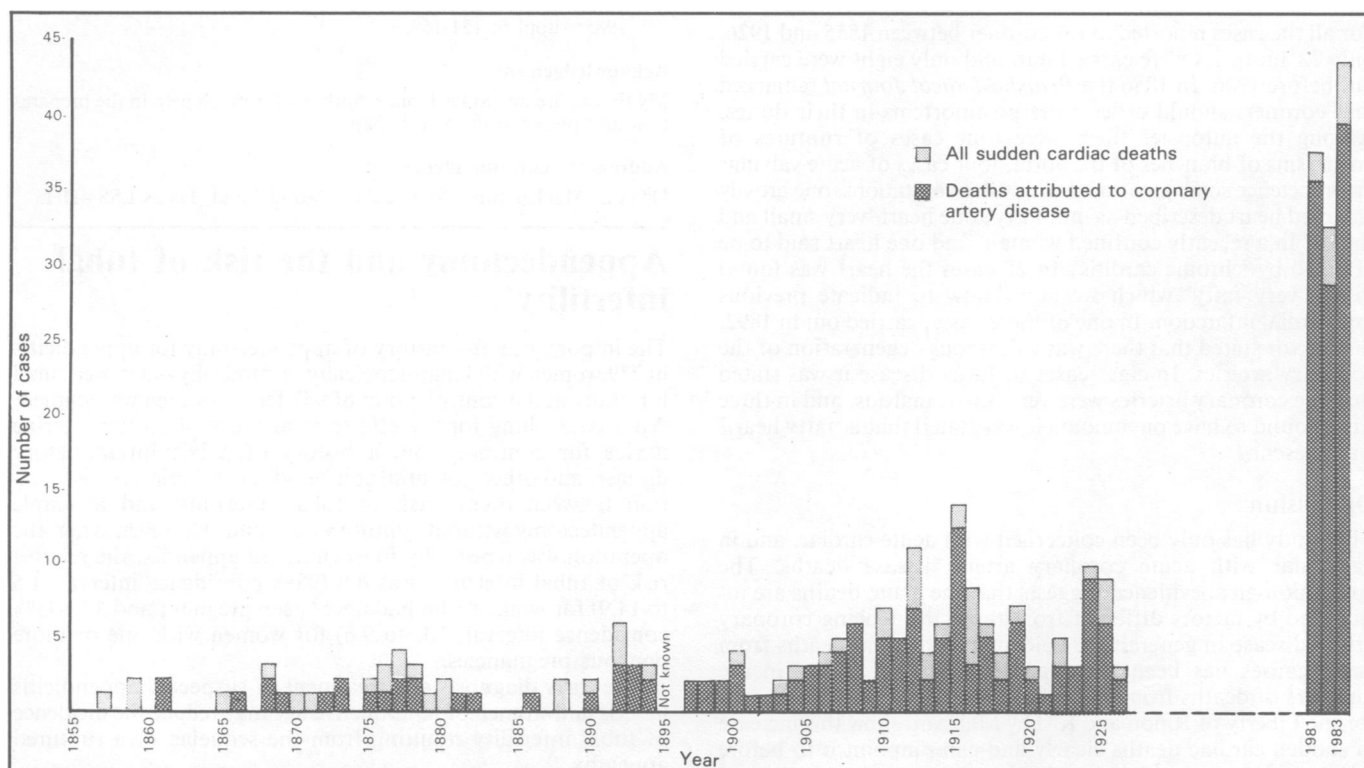


Figure 1. Number of sudden cardiac deaths reported to the coroner each year in a Yorkshire community of 22 000 people and the number of cases attributed to coronary artery disease (definitive for 1981–83, retrospective attribution for 1855–1926).

Table 1. The number of cases of sudden cardiac deaths reported to the coroner of the Liberty of Ripon and Kirkby Malzeard by age at death, and the number of cases attributed to coronary artery disease.

	Age at death (years)					Not known	Total
	<40	40-59	60-69	70-79	80+		
<i>All sudden cardiac deaths</i>							
1855-94 (40 years)	2	18	7	17	3	4	51
1907-26 (20 years)	7	41	41	23	3	4	119
1981-83 (3 years)	0	16	29	37	33	0	115
<i>Sudden coronary deaths</i>							
1855-94 (40 years)	0	10	5	10	1	1	27
1907-26 (20 years)	3	27	34	21	3	2	90
1981-83 (3 years)	0	14	27	34	30	0	105

evidence were actually acute coronary deaths, but even if all the acute cardiac deaths reported to the coroner in 1855-94 had been coronary deaths the increase between the two periods in each of the older age groups was over 25 times. Although the population is rather older this makes only a minor contribution to the increase, the proportion of the population in the older age groups having increased by factors of only three to four between 1861 and 1981.

Autopsies (1855-1926)

For all the cases reported to the coroner between 1855 and 1926, only 72 autopsies were carried out, and only eight were carried out before 1906. In 1886 the *British Medical Journal* remarked that coroners should order more postmortems in their duties. Among the autopsies there were four cases of ruptures of aneurysms of branches of the aorta, four cases of acute valvular incompetence some associated with valve vegetations, one grossly enlarged heart described as 'not fatty', one heart 'very small and flabby' in a recently confined woman, and one heart said to be affected by 'chronic carditis'. In 22 cases the heart was found to be 'very fatty' which we now know to indicate previous myocardial infarction. In one of these cases, carried out in 1892, the doctor stated that there was calcareous degeneration of the coronary arteries. In eight cases of heart disease it was stated that the coronary arteries were very atheromatous, and in three cases found to have pneumonia it was stated that a 'fatty heart' was present.

Discussion

This study has only been concerned with acute cardiac, and in particular with acute coronary artery disease deaths. The epidemiological evidence suggests that the acute deaths are influenced by factors different from those influencing coronary artery disease in general: the epidemic increase in deaths from acute causes has been accompanied by little change in the numbers of deaths from chronic causes.⁶ The coroner's records for the Liberty of Ripon and Kirkby Malzeard show this increase in sudden cardiac deaths clearly and can pinpoint it to before the year 1910. As far as is possible in a retrospective assessment of this kind, the increase has been shown to have been in the deaths recognizable as being due to coronary artery disease, and

the size of the increase is such that the occasional case where the attribution of a sudden cardiac death to coronary artery disease could not be made with confidence carried very little significance. The timing agrees well with the findings of a recent review of records at the City of London mortuary which deduced, from a study of autopsy cases only, that the increase in the number of deaths from coronary artery disease had started by 1909/10.⁷

As one might expect, the increase in the number of deaths from coronary artery disease showed itself in the coroner's records a few years before it was commonly recognized on the hospital wards, where the signs, symptoms and electrocardiographic changes were only widely known from 1920 onwards. There has been a steady increase since World War I to the average of 35 coronary deaths reported to the coroner each year in 1981-83 in this semi-rural community of 22 000 people. It is highly probable that the increase in the country in general started at much the same time as in this part of Yorkshire, and that attempts to locate the causes should concentrate on changes in food, habits, and the environment introduced in the period prior to 1910.

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Appendectomy and the risk of tubal infertility

The importance of a history of appendectomy for appendicitis in 279 women with laparoscopically or surgically diagnosed tubal infertility and a control group of 957 fertile women was studied. After controlling for the effects of age, use of an intrauterine device for contraception, a history of pelvic inflammatory disease, and other potential confounding variables, no association between excess risk of tubal infertility and a simple appendectomy without rupture was found. However, when the operation was reportedly for a ruptured appendix, the relative risk of tubal infertility was 4.8 (95% confidence interval, 1.5 to 14.9) for women who had never been pregnant and 3.2 (95% confidence interval, 1.1 to 9.6) for women with one or more previous pregnancies.

The early diagnosis and treatment of suspected appendicitis in girls and women of reproductive age may reduce the incidence of tubal infertility resulting from the sequelae of a ruptured appendix.

Source: Mueller BA, Daling JR, Moore DE, *et al.* Appendectomy and the risk of tubal infertility. *N Engl J Med* 1986; **315**: 1506-1508.