mortality is to test the specific hypothesis in different places and at different times. Earlier records are available: births and deaths were registered nationally in Sweden from 1749 and in France from 1800. New documents may be unearthed that will throw light on, for example, the actual consumption of food during the last century, and comparisons with other countries at the same time may be of special value. Preston and Nelson studied the structure and changes in causes of death using data from 165 national populations.⁷ New techniques of analysis may make easier the separation of different factors operating at the same time.^{8,9} Whatever the technique, causes are not going to be proved; the most that can be hoped is to test the various hypotheses and eliminate those that fail. On balance, the nutritional hypothesis still seems the most powerful in explaining the decline in mortality, but the mechanism is probably to do with qualitative aspects of diet rather than simply increases per head.

Finally, does understanding the cause of the reductions in nineteenth century mortality have any relevance today? There are several current questions that have a direct relation to the last century. Why are there consistent differences between the mortality of the rich and the poor? Why is mortality two to three times higher for the unemployed than the employed? Why are there regional differences in mortality within Britain? Why is mortality from ischaemic heart disease and cervical cancer falling in some countries, static in some, and rising in others? What is the relation between economic growth and national mortality in both developed and developing countries?

What we owe to McKeown is not the answer to the puzzling fall in nineteenth century mortality but the example of his challenge to the then current mythology. Griffiths, for example, believed that the understanding of anatomy and physiology, the changes in medical education, the expansion of the hospital system, the growth of dispensaries, and the introduction of midwifery services and of specific measures against smallpox were the principal causes of the decline in mortality.¹⁰ McKeown's challenge threatened the supremacy of scientific medicine and the beliefs of the medical establishment and ranks in terms of heresy with the work of Cochrane¹¹ and Illich.¹² McKeown was important because he brought to an old problem a new way of thinking that is fundamental to our understanding of many contemporary public health problems.

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- 1 McKeown T. *The modern rise of population*. London: Edward Arnold, 1976.
- 2 Szreter S. *The importance of social intervention in Britain's mortality decline c. 1850-1914: a re-interpretation*. London: Centre for Economic Policy Research, 1986. (Discussion paper No 121.)
- 3 Langer WL. American foods and European population growth, 1750-1850. *Journal of Social History* 1975;1:51-66.
- 4 Healy MJR, Jones EL. Wheat yields in England 1815-59. *Journal of the Royal Statistical Society Series A* 1962;125:574-9.
- 5 Razzell PE. An interpretation of the modern rise of population in Europe: a critique. *Population Studies* 1974;28:5-17.
- 6 Hollingsworth TH. The demography of the British peerage. *Population Studies Supplement* 1964;18:1-108.
- 7 Preston SH, Nelson VE. Structure and change in causes of death: an international summary. *Population Studies* 1974;28:19-51.
- 8 Case RAM. Cohort analysis of morbidity rates as an historical or narrative technique. *British Journal of Preventive and Social Medicine* 1956;10:159-71.
- 9 Osmond C, Gardner MJ. Age, period and cohort models applied to cancer mortality rates. *Stat Med* 1982;1:245-59.
- 10 Griffiths GT. *Population problems in the age of Malthus*. London: Frank Cass, 1967.
- 11 Cochrane A. *Effectiveness and efficiency, random reflections on health services*. London: Nuffield Provincial Hospitals Trust, 1972.
- 12 Illich I. *Medical nemesis, the expropriation of health*. London: Calder and Boyars, 1975.

Aetiology of acute appendicitis

The incidence of acute appendicitis is declining, but it remains the commonest cause of abdominal operations in Britain—and we still do not know what causes it. An imaginative attempt to solve the problem was made when the Medical Research Council Environmental Epidemiology Unit at Southampton invited a group of surgeons, physicians, epidemiologists, nutritionists, immunologists, gut physiologists, histopathologists, and microbiologists to discuss the aetiology of this disease. Many new ideas were aired, and the lack of fibre theory was made to look decidedly shaky.

The lack of fibre theory was proposed early in this century, when appendicitis was a new disease and still increasing.¹ The theory was boosted in the 1970s by evidence that the disease was still rare in many rural areas of developing countries, where stools are bulky and gut transit is fast.^{2,4} Recently workers in South Africa have had second thoughts: they noticed that urban blacks had for several years been eating little fibre, even less than the whites, yet they remained free of appendicitis.⁵ Furthermore, the British mortality from appendicitis began to decline during the 1930s and has continued to fall ever since, whereas fibre intakes have increased only recently.⁶ Falling mortality may not be caused wholly by falling incidence. But there are other unexplained facts—for example, hospital admissions for appendicitis are 70% commoner in Eire than in England.⁷

Some workers have reported that the diet of patients with appendicitis contains less fibre than that of the general population, but others have not found this.⁸⁻¹³ An odd dietary finding is that when different areas of the country are compared potato consumption is positively correlated with the disease (also sugar consumption, but less consistently), whereas a high intake of fruit and vegetables other than potato is associated with a lower incidence.¹⁴ Extension of these comparisons to include Scotland and Ireland confirms these findings and also shows that rates of appendicitis are highest in communities with high cereal consumption.¹⁵ Fitting all these facts into a neat and all embracing dietary hypothesis is hard, but environmental factors must be important in a disease whose incidence seems to change so fast. What other possibilities are there?

The infection theory is reviving in a new form. In the fully fledged disease bacteria invade and destroy the appendix, but bacteriology cannot explain why this happens. Most people believe it happens only when there is an obstruction of the appendicular lumen with stasis distally. In older people an impacted faecolith is sometimes found but not invariably; in young patients swelling of the lymphatic tissue at the base of the appendix is a more likely cause of obstruction. This tissue swells with virus infections but atrophies with age.

Barker has combined these considerations with new epidemiological findings to generate a "hygiene hypothesis" for appendicitis.^{6,15} This attributes the rise in appendicitis to improvements in sewage disposal and water supplies in Britain in the late nineteenth century. These improvements in hygiene greatly reduced the exposure of infants and children to enteric organisms, which in turn altered the children's response to later virus infections so that they now triggered appendicitis. The virus did this either by causing vigorous lymphoid hyperplasia that blocked the appendix or conceivably by "devitalising" the appendiceal mucosa so that bacteria could invade.¹⁵

Barker attributes the fall in appendicitis in the past 50 years to improved housing leading to less overcrowding and

so fewer infections in adolescence. He explains the link between appendicitis and the consumption of potato and sugar by the association of these cheap foodstuffs with poverty, which in turn goes with overcrowding and more virus infections.

This is an ingenious and plausible theory even if it has overtones of having your cake and eating it—it blames absence of infections in infancy and then the presence of infections in adolescence. No serious objections were raised to it at the Southampton conference, but the hypothesis needs more supporting data and will not explain everything. Some older people do have faecoliths blocking their inflamed appendix. Perhaps constipation is the main risk factor in older people and lymphatic swelling induced by viraemia the main one in young people. Other factors—such as a change in bacterial flora or spasm in the caecum—could also play a part. Much research still needs to be done before we can say we fully understand acute appendicitis.

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- 1 Short AR. The causation of appendicitis. *Br J Surg* 1920;8:171-88.
- 2 Burkitt DP. The aetiology of appendicitis. *Br J Surg* 1971;58:695-9.
- 3 Burkitt DP, Walker ARP, Painter NS. Effect of dietary fibre on stools and transit-times, and its role in the causation of disease. *Lancet* 1972;ii:1408-12.
- 4 Walker ARP, Walker BF, Richardson BD, Woolford A. Appendicitis, fibre intake and bowel behaviour in ethnic groups in South Africa. *Postgrad Med J* 1973;49:243-9.
- 5 Segal I, Walker ARP. Low-fat intake with falling fiber intake commensurate with rarity of noninfective bowel diseases in blacks in Soweto, Johannesburg, South Africa. *Nutr Cancer* 1986;8:185-91.
- 6 Barker DJP. Acute appendicitis and dietary fibre: an alternative hypothesis. *Br Med J* 1985;290:1125-7.
- 7 Barker DJP. Epidemiology of appendicitis. In: *The aetiology of acute appendicitis*. Southampton: Southampton General Hospital, 1986:3-8. (MRC Environmental Epidemiology Unit Scientific Report No 7.)
- 8 Nelson M, Morris J, Barker DJP, Simmonds S. A case-control study of acute appendicitis and diet in children. *J Epidemiol Community Health* 1986;40:316-8.
- 9 Brender JD, Weiss NS, Koepsell TD, Marcuse EK. Fiber intake and childhood appendicitis. *Am J Public Health* 1985;75:399-400.
- 10 Arnbjornsson E. Acute appendicitis and dietary fiber. *Arch Surg* 1983;118:868-70.
- 11 Cove-Smith JR, Langman MJS. Appendicitis and dietary fibre. *Gut* 1975;16:409 (abstract).
- 12 Nelson M, Barker DJP, Winter PD. Dietary fibre and acute appendicitis: a case-control study. *Hum Nutr Appl Nutr* 1984;38A:126-31.
- 13 Donnan S. Appendicitis in Hong Kong. In: *The aetiology of acute appendicitis*. Southampton: Southampton General Hospital, 1986:16-8. (MRC Environmental Epidemiology Unit Scientific Report No 7.)
- 14 Barker DJP, Morris J, Nelson M. Vegetable consumption and acute appendicitis in 59 areas in England and Wales. *Br Med J* 1986;292:927-30.
- 15 Morris J, Barker DJP, Nelson M. Diet, infection and acute appendicitis in Britain and Ireland. *J Epidemiol Community Health* (in press).

Imaging pulmonary arteriovenous malformations

Acquired pulmonary arteriovenous malformations result from penetrating wounds of the chest or occur in association with liver disease, especially in children.^{1,2} The more common congenital variety results from persisting fetal anastomotic capillaries and represents pulmonary vascular hamartomas. The arterial supply is usually from the pulmonary artery, although rarely from the aorta,³ and drainage is almost always into the pulmonary venous system—but systemic venous drainage has been reported.⁴ The malformations vary in size from the microscopic to large saccular structures composed of vessels lined with endothelium in a sparse connective tissue stroma.⁵ There may be a simple fistula between an artery and a vein, or the malformation may be a complicated network of vessels. The malformations may be single or

multiple and often increase in size with age.⁶ Multiple lesions occur commonly in patients with hereditary haemorrhagic telangiectasia (Osler-Rendu-Weber disease).

Most patients with malformations do not present until the third or fourth decade of life, and many are asymptomatic. Those whose lesions allow an appreciable right to left shunt present with effort dyspnoea and cyanosis, and they may have polycythaemia and finger clubbing. Diffuse lesions consisting of many small vessels are more common in children, who present with cyanosis and heart failure.⁷⁻⁹ The proximity of most malformations to the pleura explains the occasional complication of a haemothorax^{3,10}; rupture into the bronchial tree producing haemoptysis is another uncommon but potentially fatal complication.^{4,11,12} Because the pulmonary capillary bed is bypassed paradoxical embolism can occur, causing a stroke, a brain abscess, or a minor cerebrovascular insult.^{3,4,13-17} The exact incidence of these well recognised complications is unknown, but they probably occur in about a third of patients with multiple lesions.¹⁸

On the chest radiograph a pulmonary arteriovenous malformation is typically a well defined, smooth walled, non-calcified, lobulated opacity. The lower lobe of the lung is the most likely site. Simple tomography, best performed in both anteroposterior and lateral projections, better defines its structure and allows recognition of the feeding and draining vessels. Lesions with a more complicated structure, particularly if diffuse, present less characteristic features and often appear as a network of irregular opacities; this is the pattern seen most often in children.⁷ Small lesions are invisible on the chest radiograph, and even haemodynamically important malformations may show only subtle changes or be missed because of their position—especially if they lie behind the heart or in the posterior costophrenic recesses. Because of the risk of neurological complications detecting occult malformations in patients with hereditary haemorrhagic telangiectasia is particularly important.¹⁸

Pulmonary angiography is the best imaging method. It will define structure, arterial supply, and venous drainage and disclose unrecognised lesions. Selective arteriograms are often required to provide this information, and digital subtraction techniques are a useful refinement. An alternative method is computed tomography, which will show lesions that are large enough to be haemodynamically important. The characteristic serpiginous outline will be defined, and the vascular nature confirmed by rapid sequence scanning after the peripheral intravenous injection of a bolus of contrast medium.^{19,21} Although computed tomography is a sensitive method for detecting pulmonary nodules it is unreliable for diagnosing small vascular malformations. The measurement of tissue attenuation by computed tomography poses a particular problem when the lesion is small or if the patient cannot hold his breath consistently at a fixed lung volume. Furthermore, a highly vascular tumour may be mistaken for a vascular malformation if it enhances avidly with contrast medium and the afferent and efferent vessels are not correctly identified.²²

Contrast echocardiography is a simple non-invasive method for detecting a right to left shunt.^{23,24} After the rapid intravenous injection of a normal saline or indocyanine green dye bubbles produce an ultrasonic signal as they pass through the heart, and with a right to left shunt such a signal will be seen in both right and left cardiac chambers as part of the injected bolus bypasses the pulmonary capillary bed. Unfortunately, this test does not distinguish between an