in the presence of such a high mortality rate is evident from the variety of methods of analysis that different workers have used. Should we compare patency at a fixed follow-up time against the number of patients operated on—or against the number of patients leaving hospital with patent grafts? Should we calculate the percentage of grafts patent at the time of death, or in those alive at the time of follow-up,[3] or should we calculate accumulated patency rates from life tables from larger groups of patients with variable lengths of follow-up?[4 5]

Using the data from their own study, De Weese and Rob[2] showed that these three methods of analysis gave 10-year patency rates of 38%, 58%, and 45% respectively. Which of these figures is closest to the truth? What do we tell our patients—in the light of the knowledge that five years after operation an individual patient is more likely to be dead than have a patent graft? Patients can be reassured by being told that about 60% of grafts are patent five years after operation and 40% patent 10 years after operation—ignoring the mortality rate. But we must not let our compassion for our patients become self-deceit and forget our total inability to treat atherosclerosis, or that such figures are of little value in the presence of conditions such as diabetes and hypertension, which will worsen the prognosis of the limb and the patient.

While acknowledging our inadequacies we might also spare time to marvel at the resilience of the long saphenous vein, which can be removed from its natural site, denervated and devascularised, and, placed in a foreign site such as the arterial circulation, function as an artery for 10 years or more.[6] If we knew what factors governed such durability we might be able to make a good small-bore arterial prosthesis.

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Sickle-cell anaemia in infancy

Infants homozygous for sickle haemoglobin are said[1] to begin to show clinical evidence of this disease only when the proportion of fetal haemoglobin in the red cells has fallen below about 35%, the rest being haemoglobin S. The switchover from fetal to adult haemoglobin synthesis is normally completed by about 200 days of age,[2] but in homozygous sickle-cell anaemia the proportion of fetal haemoglobin falls more slowly,[3-5] being 34% in 4-month-old babies compared with 11% in healthy infants.[5] Some fetal haemoglobin persists throughout childhood in sickle-cell anaemia, but it tends slowly to decline. The amount varies; but populations and individuals with higher fetal haemoglobin concentrations have fewer irreversibly sickled cells in the circulation[5 6] and milder clinical disease.[7-9] So while haemolysis does occur in babies with sickle-cell disease[3 5] from an early stage—between the ages of 1 and 6 months their haemoglobin concentrations are 1-2 g/dl below normal, and reticulocytosis is evident—the high proportion of fetal compared to sickle haemoglobin generally confers protection from serious symptoms.

The description by Heygi and his colleagues[10] of homozygous sickle-cell disease presenting in the newborn period is therefore of great interest. A black girl born at term had haemolysis at birth, the cord haemoglobin concentration being 12·7 g dl; reticulocyte count 13·3%; total bilirubin 53 μmol l (3·1 mg dl), direct 19 μmol l (1·1 mg dl). Many sickled cells were seen on the blood smear. Studies of globin chain synthesis showed that homozygous sickle-cell disease was present, and haemoglobin electrophoresis showed 20% sickle and 80% fetal haemoglobin—as would be expected in a homozygote of this age. Possibly ABO isoimmunisation might have contributed to the haemolysis, but Heygi *et al* thought that several features of the baby's illness and sudden death when aged 5 days were direct complications of her sickle-cell disease: the raised direct bilirubin concentration; abdominal distension; the presence of blood in gastric aspirates; and haematuria, proteinuria, and raised concentrations of urea and creatinine. Necropsy showed congestion of all organs and abundant sickled cells, especially in the brain and liver. Additional findings included haemosiderosis of the liver, spleen, kidney, and pancreas, and severe healing acute necrotising enterocolitis of the terminal ileum and ascending colon. Heygi *et al*[10] discussed seven reports of newborn infants with sickle-cell disease and why symptoms rarely occur so early. Their own patient's blood viscosity had been increased, perinatal hypoxia possibly having caused sickling of red cells and the resultant hyperviscosity perhaps contributing to her death.

Porter and Thurman[11] reported 64 cases of sickle-cell anaemia diagnosed during the first year of life—21 within six months of birth—and described four main clinical patterns. An infection such as bronchitis or diarrhoea was the most frequent presenting symptom; next came dactylitis (the hand-foot syndrome[12]); anaemic crisis; and then non-specific symptoms such as colic and irritability. Clinical examination might show enlargement of the liver and spleen, pallor, failure to thrive, abdominal distension, jaundice, or heart murmur. Ten babies died, eight within one month of diagnosis, from infection, anaemic crisis, or both. The susceptibility of children with sickle-cell anaemia to fatal infections[13] may be due to splenic dysfunction, pneumococcal sepsis being a special risk, as it is in patients without a spleen.[14] Anaemic crises result from hyperhaemolysis, bone marrow aplasia, splenic sequestration of red cells, or folate deficiency[12-19]; often associated with infections they are responsible for most deaths in early childhood.

The better-known manifestations of sickle-cell anaemia, such as painful abdominal and bone crises, splenic and bone infarcts, jaundice, recurrent pulmonary illnesses, and haematuria, tend to occur after the first year of life.[4 20] Screening for haemoglobinopathy is technically possible in the neonatal period[21] but is practicable only in populations with, firstly, a high risk of having abnormal haemoglobins and, secondly, adequate medical and genetic counselling services to provide care, advice, and explanation to affected families. Thus in most countries diagnosis within the first year and the prevention of early deaths by prompt treatment still depend on knowledge of the clinical features of sickle-cell anaemia as it presents in young babies.

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Social class, occupation, life, and death

Mid-nineteenth century Britain abounded with people whose ingenuity opened new knowledge; it seemed that a man who could combine industry, ingenuity, and confidence was almost guaranteed world prominence. Somehow in many subjects the confidence, and with it the top-ranking, have been lost. A striking exception is health statistics, and, while the latest decennial supplement of the Registrar General[1] on occupational mortality is the 12th of a series, it is in many ways a pioneer document.

William Farr—a typical great Victorian—developed a workable system not only for collecting reliable national data on births, marriages, and deaths but also for analysing and interpreting them. Among his many interests Farr studied the inequitable distribution of mortality: the poor died young, and the rich survived. Apart from identifying diseases to which particular occupations were prone, his approach to comparing mortality patterns among sections of the population was by area or residence—in effect an attempt to classify persons by the place they lived in. It was this approach that led him to subscribe to the miasma theory of the aetiology of cholera (because those who died had resided on the low ground near the Thames) and so to make one of his few errors. Meanwhile John Snow[2] had considered place and person separately and was able to conclude over a decade before Pasteur's work bore fruit "that the quantity of morbid matter which is sufficient to produce cholera is inconceivably small . . . having the property of reproducing its own kind, must necessarily have some sort of structure, most likely that of a cell."

As this latest supplement indicates in a historical introductory section, it was T H C Stevenson,[3] a successor to Farr, who first separated the poor from the rich in mortality statistics. He aggregated occupations according to their status in the rigidly stratified society of Edwardian Britain. This fundamental statistical solution to a problem was reached long before sociology had emerged as a science. By combining information about occupation and marital status from death certificates with similar information collected in the decennial censuses it became possible to treat characteristics of place and person separately in the study of mortality.

The 12th supplement takes several more steps forward. The original system of social classification has become less valid for many reasons—in particular, more people are continuing their education beyond the minimum and more women are working throughout their married lives. The supplement gives separate analyses of mortality from principal causes in married women according to their own and their husband's occupation for the first time. These show differences; for instance, there is an excess of deaths from accidents, poisonings, and violence in social classes IV and V when women are classed by their own occupation which is not evident when they are classified by their husband's, and there are striking but more complex differences between the two groups in deaths from mental disorders. Other innovations include a lucid commentary on the social class characteristics of deaths in childhood and an attempt to disentangle those risk factors underlying fatal diseases which can be associated with occupation per se from those which are related to other aspects of social status.

The picture of the health of the nation that emerges is depressing. Living standards have risen: of the households of unskilled manual workers (the lowest of the seven social classes considered in the General Household Survey), 18% owned a car, 19% had central heating, 60% a washing machine; only 1% had no water closet. These figures were unthinkable 20 years ago. Yet, improvements in living standards notwithstanding, for every major cause of death the standardised mortality ratios (SMRs) are higher and usually far higher in social class V than in any other social class (with the exception of cancer of the breast and mental disorders in women and diseases of the blood and blood-forming organs in men). For respiratory disease SMRs in each sex are nearly twice the national average in class V, and less than half of it in class I.

In common with the earlier ones in the series, these analyses make extensive use of the SMR. Like the proportional mortality ratio (PMR), which is used for ages over 65 years, the SMR is a summary statistic with all the concomitant merits and demerits. For instance, the SMR for ischaemic heart disease in social class I was 237 in 1930-2 and 150 in 1949-53; it had fallen to 98 in 1959-63 and to 88 in 1970-2. The SMR cannot tell us that between 1951 and 1971 the crude mortality rates from ischaemic heart disease in all men almost doubled, rising from 1·8 to 3·5 per thousand. Nor do SMRs allow us to make direct comparisons of death rates between men and women in general within an occupation, or between occupations, all of which could be done if direct standardisation were used. This is all explained quite clearly in the Supplement.[1] The direct procedure, like the indirect, does have its disadvantages, but it would be interesting to see some experiments made with its use. Perhaps we can look forward to this next time; meanwhile it is good to know that reports of occupational mortality based on sampling procedures will be made available annually.

Despite all the innovations the volume is thinner and lighter than its predecessor, thanks in part to the use of new printing techniques and in part to another innovation, the preparation of many tables on microfiches which are obtainable on request. At £4·75 the price is 15 pence cheaper, yet it took four years less to produce than the previous decennial supplement, which was published in 1971. It is something to be proud of.

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