

International trends in postneonatal mortality

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Abstract

Trends in mortality in the age groups 1-5 and 6-11 months from 1966 to 1987 for Australia, Canada, England and Wales, New Zealand, and Sweden were examined. Mortality rates for ages 1-5 months differed appreciably between countries, with Sweden lower than all other countries examined. Rates have decreased in Australia, Canada, and England and Wales, but increased in New Zealand and Sweden. Mortality reported as due to the sudden death syndrome (SIDS) increased dramatically in all countries, although much of the increase was probably due to diagnostic transfer from respiratory diseases. Over 80% of SIDS deaths occurred in the age group 1-5 months and SIDS accounted for about half of all deaths in this age group. For developed countries total mortality in those aged 1-5 months was an indirect measure of SIDS mortality. A real increase in SIDS has thus occurred in Sweden and New Zealand and possibly in other countries as well. Mortality in the age group 6-11 months has approximately halved in all countries examined over the study period.

Infant mortality is often used as a performance indicator of a country's health and its health services. When used in this way, however, infant mortality is better divided into neonatal and postneonatal mortality.¹ Neonatal mortality provides an indirect measure of the quality of obstetric and neonatal services and postneonatal mortality a measure of social factors and less importantly child health services in the community. Sudden infant death syndrome (SIDS) is the major reported cause of postneonatal death in the developed world and it has increased steadily over the last two decades. Much of the increase can be attributed to diagnostic transfer from pneumonia and other respiratory causes.²⁻⁴ Recently, however, there has been concern that in England and in New Zealand there has been a real increase in SIDS.⁴⁻⁶

The aim of this study was to examine international trends in mortality in the age groups 1-5 and 6-11 months.

Methods

The mortality rates for ages 1-5, 6-11, and 1-11 months for all causes and for SIDS from 1966 onwards for England and Wales, Canada, Sweden, Australia, and New Zealand were obtained from official publications or direct

from that countries health statistics department. SIDS mortality rates by age group were not available for Sweden.

Results

The figure shows the trends in mortality rates for ages 1-5 and 6-11 months from all causes for England and Wales, Canada, Sweden, Australia, and New Zealand. In those aged 1-5 months there were appreciable international differences in mortality rates with Sweden the lowest and New Zealand the highest. Compared with Sweden in 1987 the mortality rates in New Zealand (1987), England and Wales (1987), Australia (1986), and Canada (1986) were 137%, 70%, 40%, and 10% higher respectively. Canada showed the largest decline in mortality from 1966 (60%), and reduction in mortality rates were also seen in England and Wales (28%) and Australia (22%). In New Zealand the mortality rates at 1-5 months increased by 10%. Sweden also showed a significant though small increase in mortality in this age group.

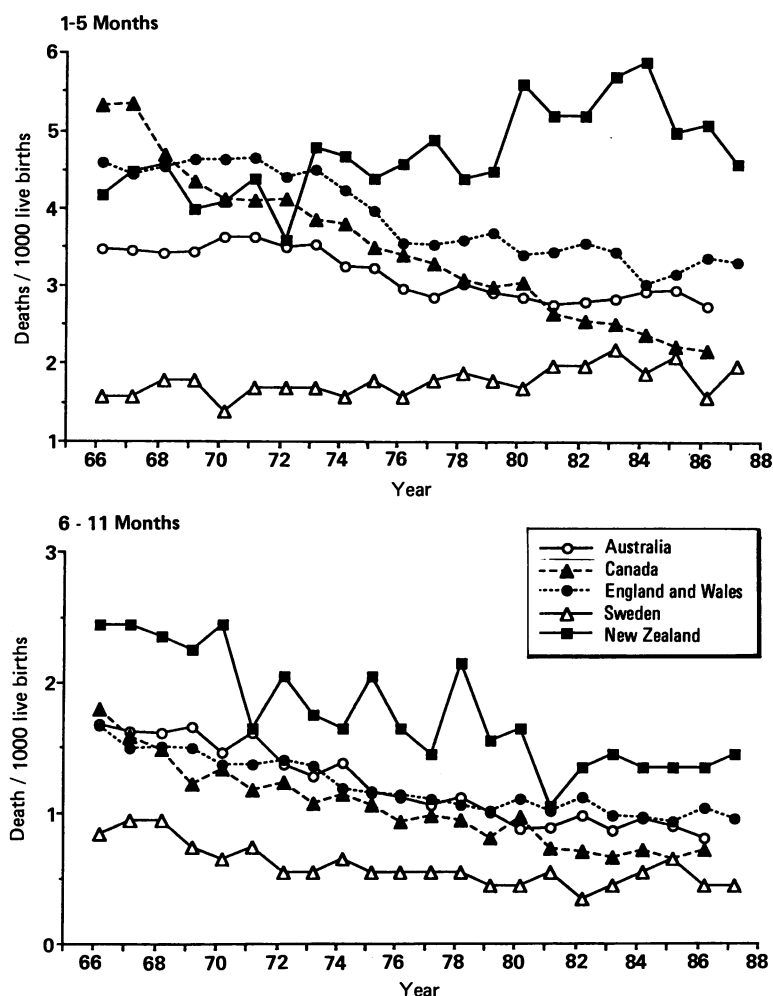
Deaths in those aged 6-11 months were much less common than in those aged 1-5 months and from 1976 accounted for only 14% to 37% of all deaths in the postneonatal age group, a figure which is remarkably constant over this period and between countries. In this age group the absolute differences in mortality rates between countries were less pronounced, although the percentage difference was greater. New Zealand again had the highest mortality rate and Sweden the lowest. Compared with Sweden in 1987 the mortality rates in New Zealand (1987), England and Wales (1987), Australia (1986), and Canada (1986) were 250%, 128%, 90%, and 68% higher respectively. All countries have shown a significant reduction in mortality rates at 6-11 months since 1966. In England and Wales there was a 44% reduction, Canada 61%, Australia 53%, Sweden 50%, and New Zealand 42%.

There were a few deaths from SIDS outside the postneonatal period and these were excluded. The mean proportion of postneonatal SIDS deaths occurring in the group aged 1-5 months was 0.85 for England and Wales, 0.93 for Canada, 0.84 for Australia, and 0.82 for New Zealand. The proportion of deaths in the groups aged 1-5 and 6-11 months attributed to SIDS increased steadily throughout the study period, so that in 1986 the proportion for Canada was 0.44 and 0.13 respectively, England and Wales 0.52 and 0.30 (1987), Australia 0.63 and 0.41, and New Zealand 0.73 and 0.50. For Sweden in 1986 the proportion of deaths attributed to SIDS increased to 0.43 for the whole postneonatal period.

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International trends in mortality for ages 1-5 and 6-11 months from all causes, 1966-87.

Discussion

Examination of causes of death over time is complicated by revisions of the International Classification of Diseases (ICD) and changes in coding practices and diagnostic fashion. During the study period there were two revisions of ICD. From 1968 to 1978 causes of death were classified according to ICD, 8th revision and SIDS was coded 795. ICD, 9th revision was in use from 1979 and SIDS was coded 798.0. Before 1968 ICD, 7th revision was in use, but it did not have specific provision for identifying infants who would now be classified as SIDS.

Changes in diagnostic fashion are very important. SIDS rates have increased dramatically during this period and now account for around 50% of deaths in the age group 1-5 months and about 25% in the group 6-11 months. During this period the number of deaths attributed to pneumonia and accidental mechanical suffocation have declined dramatically.³⁻⁴ Mortality rates from respiratory diseases declined during this period in all age groups, but the change among infants was much more pronounced. These changes probably reflect both a heightened awareness of SIDS by medical practitioners certifying infant deaths and a change in coding practice. It seems likely that much of the increase in SIDS is spurious and can be attri-

buted to diagnostic transfer. Many deaths labelled as 'pneumonia' in the past would now be labelled as 'SIDS'.

The use of total mortality rate avoids these problems. In the countries examined the mortality rate in those aged 6-11 months has approximately halved in the study period. Most of this reduction will be due to a decrease in respiratory deaths.³⁻⁴ The factors causing this decline are probably multiple.

Trends in the mortality at 1-5 months vary considerably between countries, with England and Wales, Australia, and Canada showing a decline and New Zealand and Sweden an increase. In view of the changes in total mortality in the group aged 6-11 months it seems likely that there has been a true reduction in respiratory deaths and this has probably occurred as well in those aged 1-5 months. The increase in total mortality at 1-5 months is highly suggestive of a real increase in SIDS mortality in New Zealand and Sweden. Other studies support this contention. In Sheffield, England, an increase in unexpected infant death rate has been reported.⁵ The increase was mainly confined to deaths in infants with minor disease where the father was absent or unemployed. In Norway the SIDS proportion of all infants deaths reached a maximum of 56% between 105 and 125 days of life.³ Total mortality in this age group almost doubled in 12 years suggesting a real increase in SIDS.

Data on deaths at 105-125 days are not readily available. The Norwegian data, however, shows that the SIDS fraction of all deaths is greater than 40% for each three week period from 6 to 30 weeks.³ This suggests that the total mortality at 1-5 months may be as good as that of those aged 105-125 days and as data on those aged 1-5 months is more readily available may be a more convenient indirect measure of SIDS, at least in developed countries.

Many of the factors associated with SIDS, such as parity, maternal age, maternal smoking, low birth weight, and adverse social factors, are common to all these countries. Although comparative studies have been limited, it seems unlikely that the demography differs sufficiently between countries to explain the pronounced differences in total mortality rates.⁷⁻⁸ The high postneonatal mortality rate in New Zealand is unlikely to be due to poor health services, as neonatal mortality rates in New Zealand are lower than that seen in Australia and the United Kingdom and only slightly higher than in Sweden.⁹ Studies are needed to address the high postneonatal mortality rate in New Zealand and the true increase in SIDS mortality seen in many developed countries.

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Forty years ago

Coeliac disease

Forty years ago the relationship between coeliac disease and gluten had not yet been established. In a lecture given at the University of Birmingham in May 1948 Alan Brown, Professor of Paediatrics in Toronto, discussed the then current theories of the aetiology of coeliac disease.¹ He had found a higher incidence of coeliac disease (and of diabetes mellitus) in the first degree relatives of patients with the disease than in the population as a whole. Two thirds of the children with coeliac disease had never been breast fed; in those who had been breast fed the onset of symptoms was on average six months later. Brown also referred to evidence that fat was absorbed as either fatty acids, which reached the liver via the portal vein, or small particles passed through the thoracic duct to the blood. The cause of the impairment of fat absorption in coeliac disease remained unknown but the roles of infection, gastrointestinal allergy and vitamin B complex deficiency were all considered worthy of further investigation.

Sheldon postulated that intolerance of starch might have a primary aetiological role in the disease with failure of fat absorption as a secondary effect.² He therefore planned an investigation to compare the degree of fat absorption in children with coeliac disease on two consecutive diets, one containing starch, the other starch free. In the absence of information from jejunal biopsies the diagnosis of coeliac disease was established in children with a typical history of steatorrhoea by a negative tuberculin test, the presence of normal amounts of trypsin in the duodenal juice, the absence of parasites in the stools, and a flat oral glucose tolerance curve.

Fifteen children aged from 1 to 6 years were studied. In nine the diet containing starch was given for varying periods, with a minimum of six days, followed by periods of at least seven days on a starch free diet. In the other six children the order of the diets was reversed. Both diets contained normal amounts of fat. The starch free diet was made palatable by including, in place of wheat and other cereals, biscuits made with soya bean flour that contains

no starch, the carbohydrate being in the form of dextrans. At the end of each period on a test diet 12 day fat balances were carried out in all but four of the children, in whom the balances were restricted to eight days.

There was an average rise of 15% in fat absorption after the withdrawal of starch from the diet and this was highly significant. In patients with steatorrhoea isolated faecal fat estimations were found to be unreliable indicators of fat absorption. Eight day fat balances were more reliable than four day balances and no less accurate than 12 day balances.

In a further study of children with coeliac disease the finely emulsified fat particles (chylomicrons) in the serum were counted at half hourly intervals for three to four hours after a meal containing fat to produce a chylomicron curve as a measure of particulate fat absorption.³ In children with coeliac disease the chylomicron curves tended to be flat but returned towards normal as the children improved on a starch free diet. This finding led Sheldon to suggest that defective particulate fat absorption might explain the steatorrhoea in coeliac disease.

In an attempt to determine how dietary starch interfered with fat absorption Sheldon also studied the effect of raising the content of dextrin (described as the first product of starch digestion) to between 20 and 50% of the total carbohydrate in the starch free diets.³ In three children with coeliac disease the high dextrin diets caused as much impairment of fat absorption as reintroducing starch. Sheldon did not, however, draw any conclusions from this finding.

In four children with steatorrhoea due to cystic fibrosis starch free diets did not improve fat absorption.

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