Why did postperinatal mortality rates fall in the 1970s?

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SUMMARY Postperinatal mortality rates have shown two phases of decline since 1947 which are traditionally ascribed to social and medical improvements. These factors cannot, however, explain the arrest of decline during the 1960s. There appears to be a biological effect on child mortality rates, manifesting as a generation effect. This is due partly to continuing changes in the structure of the child population, itself a consequence of social and biological changes among the parent generation when they were children. In this study national and selected urban postperinatal deaths have been divided into two categories: “probably inevitable” and “possibly preventable”. The continuing prevalence of “possibly preventable” deaths gives cause for concern. If the number of these deaths is to be further reduced, reconsideration and redeployment of community child health staff may be necessary.

At the beginning of this century one in five children in this country died before their first birthday. Four fifths of these deaths occurred during the postperinatal period and the majority were from preventable diseases. Today the infant mortality rate is about 1% of live births and the most important causes of death now are the consequence of premature birth, death occurring in the first week of life.1

Postperinatal mortality rates (PPMR) declined exponentially during the first half of this century, correlating with the reduction in the number of deaths from infectious diseases. There was very little change in this mortality rate throughout the 1960s, and then a further fall about 1972.2 The reasons for the 1960s plateau and the subsequent decline are poorly understood.

We therefore examined the changing pattern of national and selected urban PPMR, seeking underlying mechanisms for the decline in the number of deaths from potentially preventable causes.

Materials and methods

Birth rates and details of postperinatal (1 week to 1 year) deaths were obtained from the Office of Populations, Censuses and Surveys (OPCS) for England and Wales, Birmingham, Liverpool, Manchester, and Sheffield. The cities were chosen because their population numbers were not much altered by the 1974 local government reorganisation. The deaths were divided into “possibly preventable” and “probably inevitable” categories. “Possibly preventable” deaths were those recorded as (a) unexpected home deaths (all deaths occurring at home, certified by a coroner, with any cause other than congenital anomaly or violence); (b) infectious disease deaths in hospital (all deaths occurring in hospital with any infectious cause); (c) deaths from non-accidental and accidental injuries. All other deaths (principally malformations, immaturity, and handicap) were classed as “probably inevitable”. The use of “preventable” and “inevitable” assumes acceptance of the medical realities at the time of death and previous usage of the terms in this age group.3

To verify the data, RS conducted a retrospective survey of all death certificates and necropsy reports on the Sheffield children who died between 1947 and 1979. The methodology of data collection and verification has already been described.4 5 Independently, RRG surveyed Sheffield child death certificates from 1961 to 1983. From these records details were abstracted of all postperinatal deaths: in the light of necropsy findings, and without knowledge of the year of death, they were classified into “probably inevitable” and “possibly preventable”, and mortality rates were calculated.

Results

The crude PPMRs for England and Wales are shown in fig 1 together with the crude Sheffield data. The initial fall in PPMR (between 1947 and 1952) appears to coincide with the establishment of the National Health Service and the consequent expansion of curative and preventive paediatric services. However this fall is but a continuation of the trend that began in
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1902. This initial fall, the 1960s plateau, and 1970s decline in crude PPMRs occurred in all cities examined. The decline has since ceased.

The detailed study of Sheffield data showed a 15% gross error in the accuracy of official mortality statistics. These errors were, however, reasonably evenly spread over time so that the time trends, although inaccurate, are not greatly distorted.

"Potentially preventable" postperinatal mortality rates (ppPPMR) for England and Wales and the four cities are shown in fig 2. The 1970s fall in ppPPMR occurred almost contemporaneously in all the cities. Fig 2 also shows each city's crude birth rate. Changes in birth rate and ppPPMRs show close temporal associations.

To determine the reasons for the fall in PPMR, the Sheffield data were examined further and three major fatal conditions identified: infections, malformations, and sudden infant death syndrome (SIDS, defined as sudden and histologically inexplicable deaths). Time trends of these three conditions (as three year moving means) are shown in fig 3. Infections fell from 18 per 1000 in 1947 to 1.3 in 1957 and, after a plateau in the 1960s, have now fallen to 0.2. The SIDS rate varied around 2 per 1000 throughout the whole period. Malformations account for approximately 2.5 infants per thousand, and this may represent the realistically achievable lower limit for PPMR unless there are dramatic advances in genetic or surgical technology.

Discussion

Deaths of children with severe malformations causes comparatively little medical, social or political anxiety. The continuing prevalence of potentially preventable deaths is, however, a cause for public concern. Medical advances, social improvements and biological changes in the population may all have contributed to the fall in ppPPMR. The time trends observed here show no temporal association with the major medical advances that occurred in this period although the increased availability of medical care to all children following both the falling numbers of births and the increase in hospital and community based staff must have made some contribution.

During this period there have been major social and environmental improvements. These changes have
been insidious and might not be expected to show a clear association with a mortality time trend. However, neither medical nor social changes can explain the arrest of the downward trend during the 1960s.

We cannot be certain with hindsight of the reasons for the 1960s plateau of PPMR but there are three likely factors. Firstly, there was overconfidence in the efficacy of antibiotics which became generally available on prescription in the early 1960s. Secondly, the increased numbers of babies led to a shortage of available hospital cots in the winter months. Thirdly, the increasing child population contained disproportionate numbers of lower class babies.

During the 1960s plateau of ppPPMR, the major cause of death was variously described as bronchitis, tracheobronchitis, or bronchiolitis. Epidemics occurred each winter when children’s medical wards filled quickly and many were refused admission or admitted late. Some died. These deaths were not associated with bacterial superinfection (possibly because of widely prescribed antibiotics) but were due to mucus obstruction of the respiratory tract or acute hypoxic failure. The falling birth rate not only reduced the numbers at risk and the cross infection rate but also meant that those requiring admission could be treated early. Epidemics still occur but few infants now die.

Baird showed that steady biological improvements in Aberdeen children (as measured by standardised height and weight) correlated with improvements in the health of their offspring.6 The close synchrony between variations in English urban mortality and birth rates observed in this study could indicate associations in mortality risk between birth populations separated by space and time. Both are linked by changes in the structure of the child population. After the first world war there was a temporary increase in the birth rate. These “extra” babies became parents in the late 1930s, merging with the baby boom of the second world war, who in turn became parents in the 1960s. These 1960s babies will be of childbearing age in the 1980s and, liberal contraceptive policies notwithstanding, an increase in birth rate might be expected. The lower social classes are disproportionately represented in these populations, live predominantly in inner cities, and have poorer housing, more overcrowding, less family regulation, higher infection rates, and higher mortality rates. Temporal associations need not necessarily imply a causal link. However, this could explain the puzzling features of the PPMR trend and, if correct, suggests a “generation effect” which may be influencing mortality rates.

Some of the reduction in Sheffield ppPPMR in the early 1970s has been attributed to the directed use of health visitors to families at statistically increased risk of cot death.7 It has sometimes been assumed that this decline was from a consistently high rate. The earlier SIDS rate discovered by this study must change that perspective and alter the emphasis of health visiting work. There are, however, gross national discrepancies in the levels of care offered to children, and it appears that some are responding to demands rather than needs.8 There is a need for an objective method of identifying deprived, high risk children and communities. Statistical scoring systems provide this objectivity, and those that take account of daily clinical practice have increased the delivery of care to all children.9 We are not convinced by arguments for continued undirected child surveillance, especially as financial restrictions are threatening other clinical activities.10

Some have suggested that potentially serious illness is still unrecognised or not treated with appropriate urgency.3 11 It has yet to be proved that these infants were as clinically ill as they are thought to have been retrospectively. Symptoms common in children who subsequently die are seen daily in children who rapidly recover without specific treatment. There remain the problem cases where death is associated with a failure of uptake or delivery of services. The Knowelden
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The report contains a number of illustrative examples where death might have been prevented by the application of current medical knowledge if only the child and services had made contact. It has almost become traditional to blame the general practitioners for these “failures” of the service. It seems inappropriate that such a wide ranging generalist should also be expected to be the community’s primary paediatric specialist.

An alternative and cost effective method to get the services and children closer and to reduce ppPPMR further would be to modify and integrate paediatric expertise available in the community under clinical consultant leadership. Currently, health visitors and community child health doctors have no requirement for any training in paediatrics. This deficiency could be rectified quickly and should be done urgently.

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References