CHILD MORTALITY IN SHEFFIELD

A clinical, epidemiological and necropsy study of the 7049 Sheffield children who died between 1947 and 1979 and a comparison between death certificates and available necropsy reports of the numbers and certified causes of these deaths together with an analysis of some Sheffield childhood mortality trends since 1885.

by

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"There cannot be a study more important to the human race than that which leads to the consideration of the causes of premature death. The health of a people is of all studies most momentous, for vainly may a nation strive to be great or prosperous as long as disease and death unnecessarily thin the ranks of the people. What happiness has the father of a family if any of his children are sickening or dying? and as it is with a family so it is with a nation, for a nation is but an aggregation of families."
(Saunders, 1860)

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ABSTRACT

All original available data on the 7049 Sheffield children aged 0-15 years who died between 1947 and 1979 have been examined and recoded into a single classification. Comparing death certificates and necropsy reports revealed discrepancies in the numbers and causes of death with serious errors in 592 (15%) of the 4007 cases where histologically-verified necropsy reports were available.

Declines in all age-specific mortality rates were observed, especially in the postneonatal period. A marked decline in fatal infectious-diseases could not be correlated with medical advances. Cot deaths showed a steady overall trend with fluctuations which might be related to social interventions. A gradual decline in prematurity mortality rate showed no temporal association with medical changes and was most marked in the maternity hospital with the least equipment. Fatal malformations showed a steady overall trend. Variations in spina bifida showed only partial correlation with changes in medical management but antenatal screening was found to be effective in the short period examined. Fatal traffic accidents showed a worrying increase in the late 1970s. Malignant diseases were the least common cause of death in this study.

Historical trends of Sheffield child deaths since 1885 also failed to show any definite effect which could be clearly related to medical advances. Sanitary reforms and new housing showed temporal associations with the beginning of the decline in infant mortality. An hypothesis is proposed which suggests that there

may be a generation delay before improvements in child health are manifest in fatal disease trends. This hypothesis is compatible with the results of this study.

High necropsy rates are necessary if accurate statistics are to be obtained. The methods of recording child death may need revision. There is an urgent need for further community-based studies of all aspects of child health.

GLOSSARY

Apart from the word epinatal, all terms used in this thesis are in accordance with the definitions given in the British Medical Dictionary, the Oxford English Dictionary or the International Classification of Diseases, Injuries and Causes of Death, Ninth Revision. The word epinatal has been created as a generic term for those conditions which are at present believed to have their origin in events occurring around the time of birth. References are abbreviated in accordance with the format of the Index Medicus. Any other terms abbreviated in the text are given in full at first appearance.

SECTION 1

INTRODUCTION

1.1 Background to the study

Death in childhood remains one of the greatest of human tragedies. More lives are still lost in the first day or first year than in any other comparable period throughout life. It has been suggested that neither increasing expenditure on medical technology nor increasing the numbers of doctors has produced any direct reduction in mortality (Cochrane, 1972; McKeown and Lowe, 1974; St. Leger et al., 1978). The data used in these studies were derived from national statistics and, as will be shown below, such statistics contain insidious inaccuracies. The present study will examine this general proposition (that reductions in mortality may not be related to medical advances) by using address-standardised, necropsy-verified data gathered by a single observer and relating to a stable community with continuously updated medical services. This study will also attempt to identify some of the non-medical factors which might have contributed to the decline in child mortality rates in the hope that lessons learned from our past may be applicable to those areas of the world which still have high child mortality rates.

The level of health attained during childhood is the single most important factor in determining health throughout adult life (Kermack et al., 1934; Greenwood, 1936). Thus health in childhood has important economic and political relevance far beyond the immediate family or community. As will be discussed below, the most consistent index of child health which is widely available is its reciprocal - child mortality. It is therefore hoped that this study may have practical as well as academic relevance.

In this work the community studied and its history are described. The limitations of mortality data are outlined together with the methods used in attempting to overcome them. All available information was collected on every Sheffield child death between 1947 and 1979. The validity of the death certificate archive as a measure of child mortality in this community and the accuracy of certified causes of death are examined against the data obtained. The pattern of child mortality by age and by cause is presented using the necropsy-verified, address-standardised data collected. Explanations for the observed pattern of deaths are examined and an alternative hypothesis is presented. This hypothesis is tested against historical Sheffield child mortality data which, although still address-standardised, does not have necropsy-verified causes of death. Other important features of this historical data are discussed and then, finally, the possible significance for other communities of the findings of these studies are outlined.

1.1.1 Reasons for a community-based study

Most studies of disease are based on hospital or clinic records. To understand prevalence it is necessary to relate these hospital records to the population from which the patients are drawn. Starfield (1981) has shown that hospital-based studies could be seriously misleading because knowledge of the distribution of health problems in a community cannot be derived from hospital experience if hospital doctors are only aware of the patients referred to them. Patients often seek treatment from many sources and some only return if satisfied. Thus

doctors may not hear about failures of their management. Similarly, a hospital doctor's experience of a fatal condition may be altered if the population attending their hospital changes. The doctor will only be aware of changes in individual patients and may attribute the change in mortality to some treatment or toxin unless he is able to standardise the patients by residence and exposure. For all its limitations, the cardinal strength of a community-based study is its inherent ability to standardise the population at risk. Hospital-based experience suggests that patients are clearly different from the healthy population (i.e. that there are two discrete groups, one diseased, the other healthy). However, studies of the distribution of physiological variables in defined communities fail to identify these distinct groups but find a continuous distribution of a variable in the population. There are very few conditions which present as a discontinuous subgroup. People with a value for the measured variable at the high end of a normal distribution are usually those considered "diseased" but many of these may be symptomless. Cochrane (1972) discussed the problems of hospital or clinic based experience in deriving definitions of normality and disease using the example of blood pressure and observed that very few of medicine's diagnostic signs have been examined in this way. Similar problems exist for hospital-based mortality studies.

Statistical normality does not necessarily equate with health. One role of clinical epidemiology is to interpret statistical findings in the light of clinical knowledge.

Epidemiology, like pathology or clinical medicine, is one way of studying disease which offers techniques for the investigation of the basic questions concerning disease: What has gone wrong? How can it be put right? How can it be prevented? To the pathologist, disease is something which happens to cells and organs, to the clinician something which happens to his patient and to the epidemiologist something which happens to populations (Armitage, 1970). Without knowledge of the prevalence of disease and of the environment in which diseases cluster, the special skills of laboratory medicine may be misdirected. There is a need for a combined approach between hospital- and population-based studies to identify relevant priorities. Hippocrates suggested that the physician who is an honour to his profession would constantly consider the effects of the environment on his patients and would relate his patients' diseases to the population they come from: "Whoever wishes to investigate medicine properly should proceed thus: in the first place consider the seasons ... then the winds ... also the qualities of the waters... When one comes into a city ... consider its situation ... the winds and the rising of the sun ... the waters ... the ground ... and the mode in which the inhabitants live." (Hippocrates). In this way, relevant priorities can be established, prevention may be possible, and occasionally, aetiologies may be discovered.

1.1.2 Why death?

The health of its people is the greatest asset of any nation. Yet there is no satisfactory index of health which can be measured consistently in populations. In the absence of any

direct index of health, indirect measurements have to be used such as indices of fertility, morbidity or mortality. Fertility is of limited value as a measure of health because it is influenced by many factors unrelated to health. Morbidity data are biased by a large number of variables which influence how each person perceives their state of health before consulting the medical profession and by variations in diagnosis after consultation. Furthermore, as any patient can have any number of non-fatal diseases any number of times, routinely collected morbidity data can give little indication of the state of health in a community. Even if a perfect system could be devised to identify each episode of disease in every member of a community, complicated statistics would be necessary to produce an index of health for comparison with another, equally ideal, community. It is preferable to identify an event which has or has not occurred in the members of a community and, to achieve optimally consistent data, it is necessary to identify events which can occur only once in each individual's life. There are two such events: birth and death.

Birth statistics are influenced by the same factors as fertility statistics and are therefore of limited value as an index of health. Thus the only consistently recorded event in each individual's life from which an index of the health of a community can be inferred is the mortality record. Problems with the standardisation of the causes of death remain and will be discussed in section 1.4 but such problems are irrelevant until a consistently recorded event has been identified. Because of the totality of death, mortality

studies have universal pertinence: "Any man's death diminishes me because I am involved in mankind; and therefore never send to know for whom the bell tolls; it tolls for thee" (Donne, 1624).

1.1.3 Why child death?

The death of a child is a tragedy for both parents and community. Any study which leads to an understanding or prevention of child death will lessen human suffering. However, the benefits from understanding the factors underlying childhood mortality could extend beyond the prevention of suffering. Health in childhood is probably the most important factor in determining the health of the adult population (Farr, in Registrar General, 1839; Kermack et al., 1934). In the past, infant mortality has been regarded as the most sensitive index of both the public health and the standard of living of a community. It is still a useful index but, at low levels of mortality, congenital malformations tend to predominate and thus the crude infant mortality rate becomes a poor indicator of public health and a poor predictor of adult health. In consequence, attention has turned from infant to other childhood mortality rates (Dyson, 1977).

In addition to being a useful index of community health, deaths in childhood are amenable to studies of causal factors because the child population is relatively non-mobile. In a stable society, the child population at risk can be accurately calculated from numbers of live births whereas adult studies depend on census figures or inter-censal estimates.

The two ages when death occurs most frequently are

childhood and old age. Studies of factors associated with death in the elderly are complicated by the multiplicity of pathologies and environmental factors which could be involved. By contrast child deaths (while not necessarily easier to understand) have relatively few confounding variables and therefore causal factors may be identified more quickly and be controlled more certainly. For example, if atherosclerosis were fatal at all ages, it would be easier to search for aetiological factors in children than in adults because a child will have had less exposure to fewer agents and because a child's environment is easier to control and study.

Finally, of all deaths, death in childhood remains the greatest tragedy. Any further understanding of these deaths, of causal factors, or of possible preventive measures should result in a lessening of suffering of the children, their parents and the community.

1.1.4 Why Sheffield child death?

To obtain accurate details of the causes of death in childhood it is necessary to limit the number of observers to the minimum, to identify all child deaths and to examine all available data on these deaths. Ideally, one observer should do all of these tasks. For one observer to do all of the data collection, correlations and analysis it is necessary to limit the size of the population studied. Two possible methods of limiting the population are: to take a random national sample, or to study all of the deaths in a selected community. A random sample of child deaths in the whole country would sample death certificates issued by a large

number of doctors and pathologists and hence introduce considerable potential for inter-observer variation. It would be very expensive in time and travel and because of the large area covered would not allow precise correlations of environmental change. The alternative, studying a single community, would limit the numbers of doctors involved and the number of pathologists could be very small. Medical staff may be available to clarify records compiled years before and the timing of changes in medical management or environmental controls could be more accurately correlated. However, if a single community is to be studied, the selected community should be stable and, if possible, representative of the nation. Sheffield fulfils these criteria for a study of child mortality in the period 1947-1979.

There is a gradient of mortality in this country whereby mortality rates increase in proportion to the distance from the south east. Sheffield lies at about the mid-point of this mortality gradient (Registrar General 1947-1973; OPCS 1974-1980; DHSS 1976a). Furthermore, in national studies of child mortality which have been conducted during the period under review, the experiences of Sheffield's children closely reflect the national average (Butler and Bonham, 1963; Royal College of Obstetricians and Gynaecologists, 1975).

The population of Sheffield has been stable at about 500,000 for the last 50 years. In contrast to other large manufacturing towns, the migration pattern to Sheffield during the Industrial Revolution was predominantly from less than 20 miles distant and very few came from outside south Yorkshire

or north Derbyshire (Pollard and Hunt, 1956). Most of the migrants were artisans or apprentices. There were very few Scots or Irish labouring immigrants. Additionally, the population of Sheffield showed little ethnic change in the last century: in the 1851 census, 14.8% of the Sheffield population were born outside Yorkshire; in 1951 the proportion was 15.6%. In recent times Sheffield has been little affected by immigration from the New Commonwealth although accurate figures must await the results of the 1981 census. Since 1973, every Sheffield child has been followed for the first two years of life as part of a study of child development. More than 97% of these children had a Sheffield address throughout the two years that they were followed (Sheffield Child Development Study, unpublished data).

Sheffield is favoured for a study of childhood disease because the Sheffield Children's Hospital is an internationally famous centre for the treatment of children. Advances in therapy were introduced quickly and Sheffield's doctors pioneered the study of many treatments. High quality, consistent diagnoses would be expected.

Since the inception of the National Health Service there has been only one paediatric pathologist in the city. The Sheffield paediatric necropsy is unusual, and may be unique, in that every organ in every child is examined routinely and blocks are taken from every organ for histological examination. Each necropsy requires some six to eight doctor-hours from first mortuary examination to completion of the histopathological analysis. Additionally, each necropsy requires sixteen to

twenty-four hours of full-time technical assistance. The histological blocks are stored after examination and the resulting store of tissue is believed to be the largest such store of paediatric tissue in the world. This store was used in the present study to clarify potentially conflicting reports. There have been only six other pathologists in the city in the period 1947-1979 who were involved with paediatric necropsies. One of these pathologists did most of the perinatal necropsies. The two forensic pathologists in the city in this period conferred with the paediatric pathologist on many of their childhood cases. The remaining pathologists examined only a few children, usually at the request of the coroner.

The Sheffield coroner maintains a close interest in childhood deaths and has encouraged local family doctors to refer cases to the paediatric pathologist if they have minor doubts as to the cause of death but do not feel that there is sufficient evidence to open an inquest. Following necropsy the pathologist would advise the coroner if any doubts remained. As a result, a much higher necropsy rate has been achieved among the home deaths. A few cases of unnatural death which may have been missed were reported to the coroner and many unsuspected, natural pathologies were revealed with improved accuracy of the death certificates.

It would therefore appear that a study of childhood mortality in Sheffield between 1947 and 1979 would be a reasonable examination of the effectiveness of the medical and environmental changes which have been introduced to prevent childhood death since the inception of the National Health Service (NHS).

Furthermore, as the causes of Sheffield child deaths have been examined by only a small number of pathologists, the certified causes of death can be correlated with necropsy reports which will have little inter-observer variation. Finally, insofar as the pattern of Sheffield child death reflects the national pattern, an examination of trends in Sheffield child mortality might explain national changes.

1.2 Background of Sheffield

Sheffield is an industrial city in the north of England with a stable population of approximately 500,000. The city is situated almost in the geographical centre of England lying just north of the intersection of lines joining the river Severn with the Humber and the Mersey with the Wash. It lies seventy miles from the east and the west coasts of Britain. The city lies in a fold of the eastern flank of the southern Pennines, the land to the west rising steeply to a moorland plateau 1500 feet above sea level. The city centre stands on the spatulate edge of a ridge formed by the rivers Sheaf and Don. The modern city has spread over the surrounding hills yet it remains almost encircled by hills with only a half-mile wide valley through which the Don flows north east to the plains of Trent and York. The city's climate is predominantly continental rather than maritime. The principal occupations are in engineering and foundry trades, with an international reputation for the manufacture of special steels, cutlery and edge tools.

1.2.1 Growth of the city

The genetic stock of Sheffield's children has, like the rest of Britain, been derived from the intermingling of many

peoples. The earliest known settlements in the region were in Neolithic times and there is evidence of settlement during the Bronze and Iron ages. Romans, Angles and Norsemen successively invaded and settled but, throughout these turbulent times, the Sheffield valleys remained relatively undisturbed as they were remote from the main trade routes.

Throughout the Middle Ages Sheffield was a secluded town surrounded by wooded hills, isolated from the natural routes of communication across the plains of Trent and York. This seclusion was one of the reasons why Mary, Queen of Scots, spent the greater part of her imprisonment in Sheffield Castle (from 28th November 1570 to 3rd September 1584).

There had been small-scale industrial development prior to the Industrial Revolution with iron working and forging from prehistory, lead mining in Roman times, coal mining since 1300 and cutlery manufacture was sufficiently well-known in Chaucer's time for him to mention Sheffield twithels (knives) in his Miller's Tale. However, by the middle of the eighteenth century Sheffield had become the centre of a region producing coal, iron and steel, and manufacturing cutlery and edge tools.

The rapid industrial development of the city was reflected in the growth of the population. Accurate population statistics are available from 1801, the first national census. Additionally, in Sheffield, there were enumerations by 'twenty-four of the most sufficient inhabitants' in 1615 and by house to house survey in 1736. These surveys, together with estimates from the Poll Tax returns of 1692 and from parish registers, allow an

estimate of the population growth (Fig. 1). The population remained relatively stable throughout the seventeenth century. Between 1692 and 1750 the population quadrupled and doubled again by 1801. During the nineteenth century it increased ninefold and continued to increase until the early 1920s. A similar increase occurred nationally and appears to be due primarily to an increase of births over deaths rather than to migration (Singer and Underwood, 1962). Sheffield parish registers show an increase of births over deaths from 1700 onwards (Pollard and Hunt, 1956). The changes would appear to be due to a decrease in the death rate rather than an increase in fertility and appear to be associated with the relative improvements in general health which followed the agricultural revolution of the seventeenth century (McKeown and Lowe, 1974).

Until the end of the seventeenth century, Sheffield retained its mediaeval street plan. During the eighteenth century, the population grew faster than the town causing the disappearance of open spaces. New brick houses of two or three stories replaced stone cottages. Further expansion was provided by building on at the rear, eventually creating a warren of buildings and laying the seeds of eventual squalor and overcrowding. Most cutlery artisans worked in backyard workshops, tending to occupy areas of the city which have remained centres of the cutlery industry to the present. The steel foundries, on the other hand, developed in the valley floor and spread along the Don. This industrial area was surrounded with back-to-back terrace houses (two rows of dwellings under one roof, each house sharing three walls with

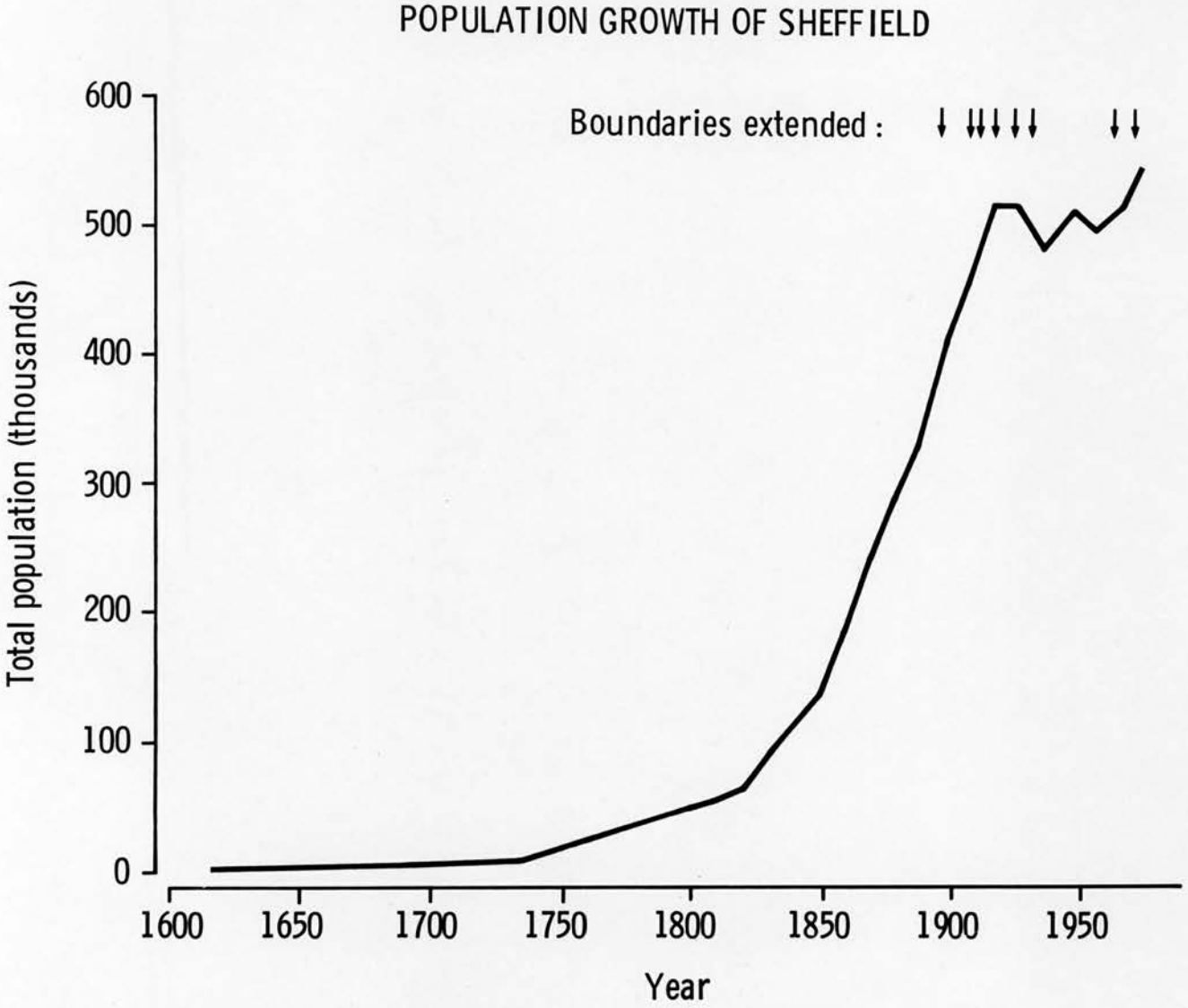

POPULATION GROWTH OF SHEFFIELD
Boundaries extended :
Total population (thousands)
600
500
400
300
200
100
0
1600
1650
1700
1750
1800
1850
1900
1950
Year

Fig. 1

neighbours). In the nineteenth century the original town around the castle was transformed with terraced houses into a densely populated zone. Despite this rapid development of cheap housing, Sheffield had no inhabited cellars - in 1841 12% of Manchester's population and 7682 of Liverpool's people permanently inhabited cellars (Holland, 1843).

During the present century municipal housing estates were built on the surrounding hills while the more affluent increasingly moved into the south west corner of the city. Sheffield has almost avoided inner city development schemes, has relatively few tower blocks of flats and there is little commuting as the majority of the commuting classes still live within the city boundary and participate in the community.

1.2.2 Mortality in Sheffield

During the nineteenth century, Sheffield's crude mortality rate was one of the highest in the country. A major reason for the high crude mortality rate was the very high childhood mortality - approximately 50% of all deaths in late nineteenth century in Sheffield were among children aged less than five years. Taylor (1873), commenting on the observation that half of Sheffield's children did not reach their fifteenth birthday, said "the manner in which children are killed off in Sheffield is something fearful to contemplate".

The principal cause of the high childhood mortality was the prevalence of infectious diseases. There were 2686 child deaths from smallpox between 1857 (the earliest record) and 1872. Vaccination of infants by four months of age was compulsory but enforcement had been lax prior to 1872.

Following mass vaccination, smallpox deaths fell from 1000 in 1872 to 5 in 1873, and apart from small outbreaks in 1884 and 1888, there were no more smallpox deaths. Prior to 1920, epidemics of diphtheria, scarlet fever, measles and whooping cough ravaged Sheffield's children almost every year. There was an epidemic of infant diarrhoeal deaths between the 28th and 41st week of each year until 1912. For reasons which are unknown, the epidemic of diarrhoeal deaths began when the soil temperature four feet below the surface was greater than 53.5^{o}F and remained until this temperature fell below 53.5^{o}F (Sheffield 1874-1973).

Taylor (1873) postulated that the causes of the high child mortality in Sheffield must be preventible because the rates in other large manufacturing towns were lower and also because the rates varied within the different townships of Sheffield. Successive Medical Officers of Health (MOH) had drawn attention to the high childhood mortality in the poorer overcrowded areas of the town which were in the heavily polluted industrial atmosphere and close to the sewage-laden river.

Sheffield was no different from other manufacturing towns in the neglect of children or the abuse of child labour. Working class mothers returned to work soon after childbirth, leaving their babies in the care of other children or untrained child minders. There was little knowledge of a child's needs and in 1878 more than 1600 children under two years were seen at the Children's Hospital with 'ailments arising from improper feeding' (Harvey, 1976). Children began work at six or seven years of age and worked a 14-hour day in the steel mills or

grinding shops. When introducing the Prevention of Cruelty to Children Act 1889, A. J. Mundella (a Sheffield MP already associated with the 1870 Education Act) said "By this Bill I am really anxious only that we give (children) the same protection that we give in the Cruelty to Animals Act" which had been passed 13 years earlier (Harvey, 1976).

1.2.3 The development of hospitals in Sheffield

By the beginning of the eighteenth century, forty English towns had a public infirmary. Yet Sheffield had none until the late 1790s, paying £2.12. 6 per year for the use of beds in York some 80 miles away. In 1789 Dr. William Young, an Edinburgh graduate, wrote a letter anonymously which drew attention to Sheffield's lack of an infirmary and suggesting that a public subscription be organised. With the encouragement of the vicar of Sheffield £16,000 was raised between 1792 and 1795 and the Sheffield Infirmary was built without delays or debts. In 1832, a Public Dispensary (later the Royal Hospital) was founded and in 1864 the Jessop Hospital for Women was opened. The town council built a fever hospital in 1884 principally for the isolation of scarlet fever patients and, in 1878, an isolation hospital was built on Lodge Moor.

Dr. William Cleaver (another Edinburgh graduate), together with Henry Vickers a solicitor and J. D. Webster an architect, founded the Sheffield Infirmary for Children in 1876. Prior to this, children could be treated as outpatients at general hospitals but admission was not permitted under six years of age. The Infirmary for Children could initially treat only outpatients but as funds grew wards and specialist departments were set up (Harvey, 1976).

Outside of the charitable hospitals, medical care was expensive and most sick children were treated with patent medicines. Before the nationalisation of the health service, most doctors derived their income from private practice. In industrial towns like Sheffield it was rarely possible to make a living from a purely paediatric practice and most sick children were treated by adult physicians who had a special interest in children's diseases. With the establishment of a national salaried health service, a career in paediatrics became possible and children began to be treated by specialists trained in paediatric medicine.

1.2.4 Development of public health in Sheffield

Of the many changes in medicine and medical thought none have been more revolutionary than those which deal with preventive medicine (Singer and Underwood, 1962). The great public health movement began in the nineteenth century when it became apparent to some reformers that improved sanitation could greatly improve the general health of the population and therefore reduce the susceptibility to disease. The relationship between poor housing, overcrowding and illness led Kay, Arnott and Southwood-Smith to challenge the view that pauperism was due to illness, improvidence and drunkenness. They suggested that much pauperism was due to ill health as a consequence of the foul environmental conditions and other causes beyond the control of the individuals concerned. The reformers suggested that money spent on improving living conditions of the poor would ultimately benefit the whole community. The motive for these reforms was as much financial as humanitarian - in 1838, at the peak of a typhus epidemic, Chadwick pointed out to

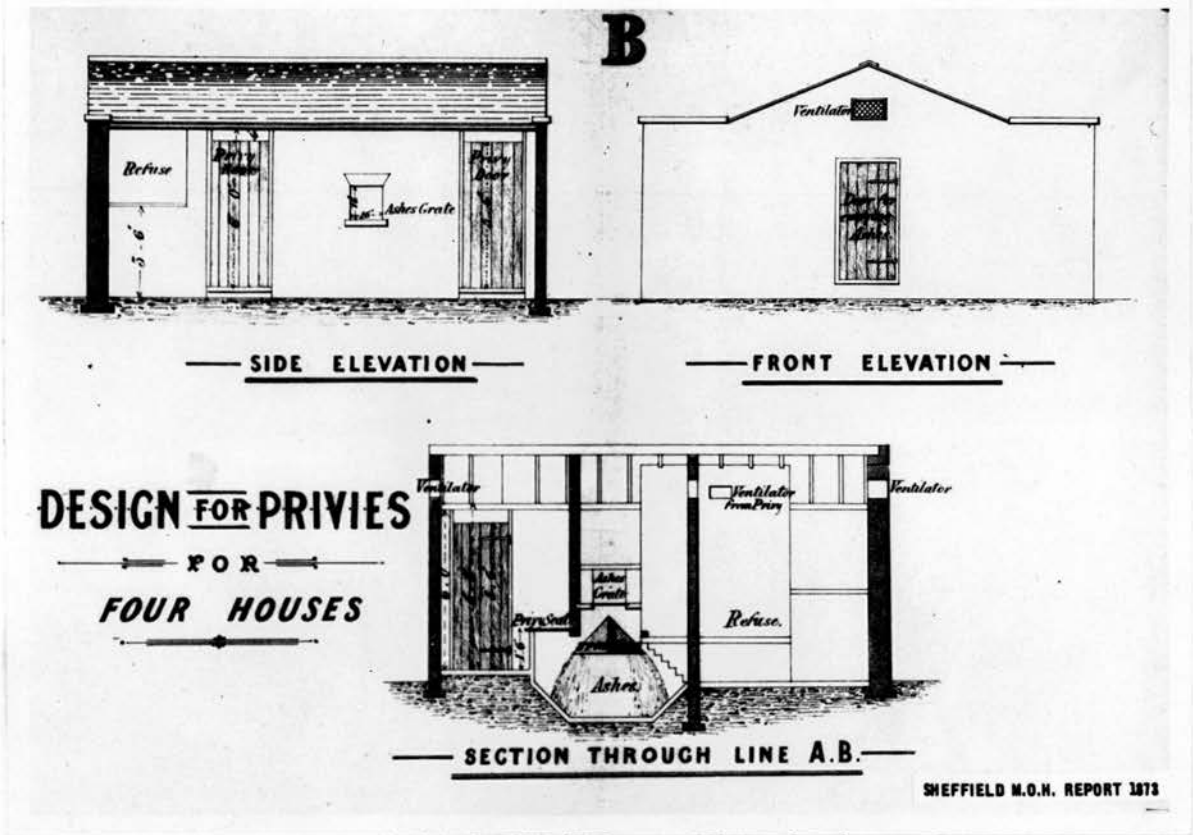
the Poor Law Commissioners that there was no limit to the amount of money which might be spent in relieving individual cases of fever unless the basic cause of the disease was removed. Their campaign culminated in the Public Health Act of 1875 (Singer and Underwood, 1962).

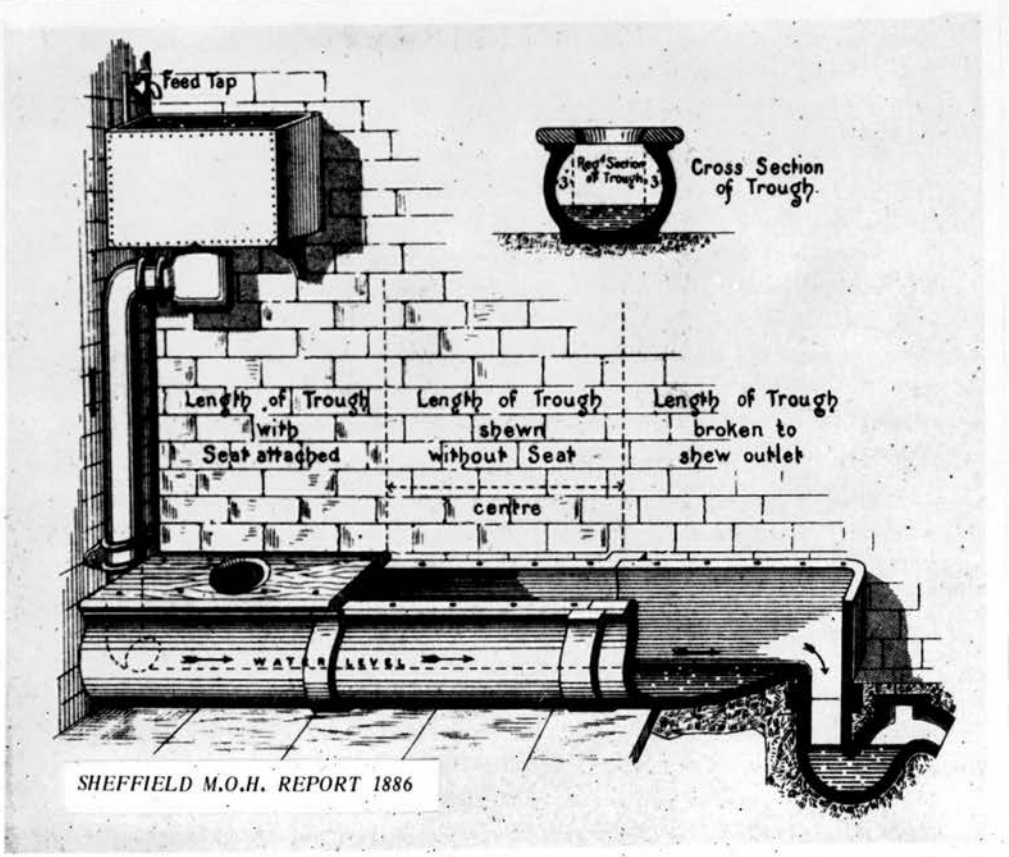
During this period there were local attempts to reform public health in Sheffield and these efforts have been summarised in three reports on the very high mortality and sickness rates in the town (Holland, 1843; Saunders, 1860; Taylor, 1873). It would appear that improvement in sanitation, water supply, slum clearance and smoke abatement may have played a major part in reducing child mortality.

1.2.4(a) Improvements in sanitation

The main method of sewage disposal in Sheffield until the end of the nineteenth century was by privy-midden (Fig. 2). Excrement and rubbish were deposited in a large pit usually within a few yards of the house, which was covered with ash and clinker from the fireplace. The pits were emptied manually several times a year by shovelling the contents onto the surrounding yard then carrying them to the street where the night-scavengers collected it. The privy-midden system remained effective when a midden served only one or two families and where only solid waste was used. However, the addition of urine and slops became increasingly common as the population grew. The ground surrounding the pits (where children played) thus became sodden, and the night scavengers' work became intolerable. In the second half of the nineteenth century the numbers

CORPORATION DESIGNS FOR PRIVIES AND WATER CLOSETS





using each privy rapidly increased and, as houses were built without consideration of the site of the privy, in many cases the contents of the privies had to be carried through houses to the scavenger's cart.

Despite these hazards successive Medical Officers of Health experienced great difficulty in persuading the council to adopt the water carriage system of sewage disposal (Fig. 2). Initial private introduction of water closets among the wealthy had had disastrous consequences because of defective plumbing. Ignorant of the method of ventilating sewers, plumbers connected the water closets to the main waste drains with the result that sewer gas rose by the line of least resistance to the kitchen sinks.

Following the Sheffield Corporation Act 1890 there was compulsory alteration of middens to water closets as fast as the new sewage system was developed. The Public Health Act 1894 made it the responsibility of the corporation to maintain any drain which received sewage from two or more houses. Between 1892 and 1902 there was an extensive reform of Sheffield's sewage disposal system with approximately 6000 yards of new sewer built each year. By 1911, 20739 middens (serving 42029 houses) were changed to water closets. These changes from midden to water closet began in the densest housing areas.

1.2.4(b) Water supply

In the fourteenth century, a Mr. Barker built a pool in the middle of Sheffield to supplement the water supply

from hillside springs. This pool was used for cattle and was occasionally drained to wash the town streets. Townspeople, who had no access to a spring or private well, collected the often stagnant water for all their domestic needs.

With industrial development, private water companies built dams in the upper valleys to supply the steel works and foundries. Any surplus water was sold to the town on three days each week (Holland, 1843). However, during the late summer months when the reservoirs ran low, water was often not available in the town which might have exacerbated the annual diarrhoea epidemic. Sheffield thus had a piped water supply from the early nineteenth century and little domestic water was subsequently drawn from wells which were often contaminated with sewage.

The Sheffield Council gradually gained control of the privately owned public utility companies and in 1887 completed negotiations to buy the Water Company. The Council set in motion work to provide the town with piped water under constant pressure (i.e. water would be available whenever a tap was turned). New reservoirs were built to fill the new water closets and baths, as well as fulfilling increasing industrial demand. In 1899 Sheffield became a member of the Derwent Valley Water Board (whose reservoirs supply Derby, Leicester, Nottingham and Sheffield) and a constant supply of pure water to the town was assured.

1.2.4(c) Slum clearance

The rapid growth of Sheffield in the nineteenth century led to increasing density of housing. All available land was built on, creating a warren of buildings. However, the population grew more rapidly than the housing stock with consequent overcrowding. In the poorer parts of the town, one room usually served as bed-, living- and work-room. Ventilation was inadequate and there was often no natural light. The Common Lodging House Act (1851) was intended to prevent overcrowding within dwellings but this law was evaded by letting rooms as furnished. In 1873 there were more than 800 such lodging houses where an apartment was 'a room in a filthy tenement - just large enough for a horse - partitioned so as to make two rooms for families' (Sheffield 1874).

The Housing of the Working Classes Act (1890) gave the MOH powers to condemn properties as unfit for human habitation and subsequent legislation encouraged the corporation to build their own properties. Empowered by this legislation, slum clearance began in the most overcrowded areas and new houses or flats were built on the surrounding hills - away from the smoky centre and the polluted river. In the period 1889 to 1898 there were 850 new dwellings built each year and then between 1899 and 1908 there were 2200 built each year (Figs. 3 and 44). All of these new dwellings had piped water, water closets, good ventilation and light with separate sleeping and living accommodation.

The new building at the turn of the century bears a close association with the beginning of the decline in

INHABITED HOUSES DUE FOR DEMOLITION, SHEFFIELD 1956

child mortality. However, overcrowding remained and has been revealed in surveys done for the Housing Acts of 1936, 1957, 1961 and 1969. New building and slum clearances have continued (Fig. 3). The most overcrowded and unwholesome dwellings were always in the poorest areas of the city, which have always had the highest child mortality rates.

1.2.4(d) Smoke abatement

Following the Industrial Revolution all towns had serious atmospheric pollution. The rapid growth of industry and the replacement of water-power by coal-fired steam forges led to widespread and serious atmospheric pollution in Sheffield. Attempts to control this pollution in the 1818 Sheffield Police Act and the 1854 Smoke Byelaws Committee proved unsuccessful and between 1854 and 1889 average emission of black smoke per chimney was 9-10 minutes per hour (brown, grey or other smoke was not recorded).

The effect of this atmospheric pollution on health became increasingly obvious to the citizens and eventually overrode economic objections to smoke control. In 1889 a petition from 5800 citizens led to the establishment of the Smoke Nuisances Subcommittee and, with public support, mean smoke emission had fallen to 2½ minutes per hour per chimney by 1893. However, the numbers of chimneys had increased. The Smoke Abatement Committee continued to strive for control of atmospheric pollution because of the great increase in respiratory disease compared with the surrounding rural areas.

Much of the pioneering work in understanding thermal inversions and urban atmospheric pollution was done in Sheffield University. This work became highly relevant after the great smogs in London in the early 1950s. The 1956 Clean Air Act, introduced as a consequence of these smogs, gave local authorities powers to create smokeless zones, to limit the coal consumption of new furnaces and extended previous powers by including domestic fires. There was, however, exemption for the metallurgical trades, power stations and gas works and railway locomotives were not mentioned. Smokeless zones were introduced gradually in Sheffield. The zones were carefully planned to capitalise on the prevailing winds which blow from the south west. The first smokeless zone, in 1959, was in the affluent south western suburbs. Subsequent zones were placed downwind and gradually covered the city. By this method the benefits of smoke abatement had become obvious by the time the commercial sites in the centre of the city were reached and there was little resistance to their compulsory inclusion. Finally, having included the industrial zone to the north east in 1973, Sheffield could claim to have the cleanest air of any populated space in Europe.

There were many other factors operating in the 1950s which might have contributed as much as these legal measures to the decline in atmospheric pollution. In the late 1950s British Railways changed from steam to diesel locomotives which greatly reduced the amount of soot in

the atmosphere and, as the railway steaming-up yards were in the middle of town, this change would greatly reduce pollution in the city centre. The transition from coal to oil and gas in both industry and homes may also have been encouraged by financial rather than statutory considerations.

The effect of the Clean Air Act on health was monitored by Lunn et al. (1967, 1970) who followed Sheffield children born before and after the Act. They found that pulmonary function was dramatically improved in the latter group. However, these conclusions could be affected by population movements to new housing estates, by immigration, by changes in the general health between these two groups and by the ready availability of antibiotics on prescription from family doctors after 1957 which may have reduced the amount of pulmonary damage in early childhood infections (Lunn, personal communication).

This brief historical review has indicated factors which might have influenced child mortality. No report has been traced of the effect of these factors on child mortality trends in a stable community. The present study will seek to rectify this.

1.3 History of death certification

Vital statistics were first recorded for legal and administrative purposes, to enumerate the population, to estimate the level of taxation to be borne by each parish and to obtain numbers of the population available for military service.

Parish records extend over many centuries but regular record keeping did not begin in England until after the Reformation when Henry VIII instructed every vicar to keep a 'true and exact register of all weddings, christenings and burials' (Registrar General, 1839). These parish records were gathered together and published from time to time as Bills of Mortality. From 1603 the Company of Parish Clerks issued a continuous weekly series of Bills which indicated the numbers of persons of each sex who had died from particular causes and also the numbers in each age group (although the age and cause of death were not correlated). John Graunt (1676) collected together these Bills of Mortality and derived from them inferences about mortality and fertility, including the high mortality among infants, the variation of death with seasons and the excess of male births. The parish records remained local until the establishment of the General Register Office in 1837.

The Act of 1836 which established the post of Registrar General and the General Register Office also divided the whole country into 2193 registration districts, each in the charge of a Registrar. These Registrars recorded details of births, deaths and marriages on forms described by the 1836 Act and relayed this information to the General Register Office. Registration was not compulsory until 1874. The form used for notification of death has been subsequently modified but remains essentially unchanged and is now used as the model for international death certificates (WHO 1977).

The earliest attempts at classifying diseases systematically began in the 18th century with Sauvages' Nosologia Methodica

and Linnaeus' Genera Morborum. By the beginning of the 19th century, the most widespread classification was the Synopsis Nosologiae Methodicae of William Cullen from Edinburgh. This classification was still in use, unrevised, when Farr was appointed as medical statistician to the first Registrar General. Farr showed that Cullen's nomenclature was confused, outdated and unsuitable for statistical purposes. In his annual 'letters' published in the Registrar General's reports, Farr considers the requirements of a nomenclature for statistical classification and at the first International Statistical Congress in Brussels in 1853, Farr and d'Espine of Geneva were asked to prepare a uniform nomenclature of causes of death applicable to all countries. Farr and d'Espine submitted separate lists based on different methods of classification to the Paris Congress in 1855. A compromise list was produced which was subsequently revised according to Farr's model. There was never any universal acceptance of this classification yet the general arrangement based on Farr's proposals has survived as the International Classification of Causes of Diseases and Death (ICD) now in its ninth revision (WHO 1977).

Thus British mortality records have been maintained systematically and consistently since the middle of the last century allowing a lengthy perspective of mortality patterns to be made.

1.4 Validity of death certification as a measure of child mortality

The certified cause of death is, to the layman, a matter of fact. To the doctor it is an abbreviated summary of the

pathologies which were believed to be relevant to the mortal process. It is at best a statement of what a person died with, but not necessarily what he died from.

Death certificates are still used for administrative and fiscal purposes and the control on the quality of the administrative features of death certificates is high, although an unscrupulous or lazy doctor could destroy this by falsifying the record (Havard, 1960). On the other hand, demographers, bio-statisticians and health planners using death certificates to identify important causes of mortality are entirely dependent on the quality of the medical part of these certificates. Errors here may arise from human fallibility, wilful deceit or criminal intent.

The legal aspects of death certification require an inquest to be held if there is any suspicion that death was not due to natural processes. The medicolegal enquiries centre around the questions 'Was the death natural or unnatural?' and, if unnatural, 'Was the process accidental or not?'. Coroner's Rules are designed to ensure uniformity of reply to these questions. But Havard (1960) has shown that neither the Coroner's Rules nor the requirements of death certification afford any guarantee against the surreptitious or inadvertent disposal of bodies which ought to be the subject of a medicolegal investigation.

1.4.1 The completeness of death registration

The first report of the Registrar General (1839) showed that burial records were an inaccurate indicator of the number of deaths. The average number of burials in the decade

1821-1830 was less than 250,000 per year. In the first year of death registration, there were 290,000 burials recorded but there were nearly 340,000 deaths registered. Recent studies from Carolina (Rogers et al., 1961), Alabama (McCarthy et al., 1980) and Ulster (Scott et al., 1981) have shown that deaths in the neonatal period are still underregistered. Dean and McLoughlin (1980) have similarly shown that among the elderly in a rural area of Ireland there were 8% more burials than death registrations. Puffer and Serrano (1976) found that in Santiago 56% of the neonatal deaths had neither been registered as births or deaths. As the present study is dependent on death certificate archives as the primary source of data, an estimate of the completeness of death certification will be made by comparing these records with necropsy archives.

1.4.2 Validity of the certified cause of death

In addition to problems of the completeness of registration, there have been serious misgivings about the accuracy of the stated cause of death. Graunt (1676) gave particular attention to the errors and ambiguities of the data in the Bills of Mortality because of the method of determining the cause of death: "When anyone dies, then either by tolling or ringing of a bell or by bespeaking of a grave of the sexton, the same is known to the searchers corresponding with said sexton. The searchers hereupon (who are ancient matrons, sworn to their office) repair to the place where the dead corps lies, and by view of the same, and by other enquiries, they examine by what disease or casualty the corps died. Hereupon they make their report to the parish clerk". It is reported that generous

helpings of liquor played a prominent part in these lay inquests, and inflated returns from epidemic diseases such as cholera led to public panics which provided one of the main reasons for introducing death registration (Havard, 1962).

The first report of the Registrar General also expressed concern about the accuracy of the certified cause of death: "Long before the commencement of Registration, my attention was turned towards an object admitted to be of great importance to the improvement of Medical Science, namely - to obtain a faithful statement of the cause of death.... It is obvious that such statements, in order to obtain due credit, ought to be derived, whenever it is possible, from the Medical Attendant of the deceased person" (Registrar General, 1839). However, the Sheffield MOH reported in 1885 that "6.1% of the entire number of deaths registered during the year were uncertified i.e. the cause of death was not authenticated by either a certificate of a legally qualified medical practitioner or by a Coroner's inquest". This must cast some doubt on the validity of the certified causes of death in some of the historical data.

Surveys have shown that the causes of death given by clinicians without the benefit of postmortem examination are incorrect in 50% of cases, 25% being gross errors (Waldron and Vickerstaff, 1977). Gittlesohn and Senning (1979) reviewed 25 major publications which examined the accuracy of the ascribed cause of death when compared with necropsy findings or hospital records. Agreement could be found in less than 45% of the cases studied. Other studies have assessed the accuracy and confidence of registered cause of death by

addressing questionnaires to the certifier and found serious levels of uncertainty (Alderson and Meade, 1967). These inaccuracies and uncertainties must limit the value and validity of central indexes of death certification in describing variation in fatal diseases.

There have been very few studies of diagnostic accuracy in childhood deaths. The Registrar General (1958) in a study of the accuracy of certified causes of death commented on the difficulty of ascertaining the cause of death in newborn children without a necropsy and therefore did not consider this age group further. Kane (1964) found that the rate of congenital malformations among 3700 perinatal deaths doubled if an autopsy was performed. Fedrick and Butler (1972) found that only 30% of pneumonias discovered at necropsy among neonatal deaths had been reported on their death certificates. Hook, Farina and Hoff (1977) compared the death certificates with their notes of the 301 children who had died from among the 3838 children they had seen at a cardiac clinic. Cardiac abnormalities had been diagnosed in 227 of these 301 children and this appeared somewhere on the death certificates of 187 (82.4%) but the specific abnormality was correct in only 81 (39.3%). Emery and Irvine (1958) examined the necropsy records and clinical notes on 150 consecutive deaths (aged 0 to 12.5 years) in a children's hospital. They found that in only 15% of the cases was there full agreement between pathologist and clinician, in 40% of cases the underlying certified cause of death was incorrect and in 18% they considered that a complete revision of the death certificate would be advisable.

There are therefore two important reasons for examining the present series for consistency and completeness. Firstly, there is no other reported large series of deaths throughout childhood in a defined community with so few pathologists involved where each death was reviewed by a single observer. Secondly, the validity of the present investigation depends on a complete and consistent series of cases.

1.4.3 Variations in the coding of causes of death

Even if completely accurate death certificates could be obtained, the coding of these certificates could introduce artefactual errors into central records. A number of apparently dramatic alterations in the incidence of some fatal diseases have subsequently been shown to be due to changes in the definition of these diseases following a revision of the International Classification of Diseases (Tait and Boog-Watson, 1962; Lillienfeld, 1976).

The large number of staff involved in coding death certificates introduces inter-observer variations which appear to be considerable. In one study, copies of a sample of 1000 completed death certificates were sent to six national coding centres. The coded results from each centre were compared with the codings of the WHO Centre for Classification of Diseases. Discrepancies of 15% to 34% occurred between centres and no centre agreed completely with any other (WHO 1967). In part the disagreements were due to differing interpretations of the ICD rules for classification (e.g. anuerysm not otherwise specified should be coded as syphilitic but was often coded as arteriosclerotic). However, there would also appear to be an element of human error.

Fedrick and Butler (1972) found major discrepancies between the coded causes of death of children in the British Perinatal Mortality Survey when compared with the coding of the same records by the General Register Office staff. They found again that some of the discrepancies were due to ICD grouping but concluded that even a perfect coding system could not be expected to achieve accuracy greater than 90% and possibly less than 60%.

There would appear to be a need for a study where a single observer examined the original death certificates and necropsy reports and where the causes of death were recoded into a single classification.

1.5 Previous studies of child mortality

There have been many studies on death in childhood. These can be divided into studies in which the population at risk is consistent (community-based) or variable (hospital-based). Both can be subdivided according to whether the cause of death has been verified by necropsy.

The majority of the necropsy-verified studies are based on hospital patients. The populations from which these patients are drawn are often indeterminable and most studies do not identify this population. A few studies from discrete communities served by a single hospital and mortuary approach the true community-based study, but the very few studies of child mortality over a long period of time from such centres do not report on the time trend of fatal disease rates (Östberg, 1973; Bjerre and Östberg, 1974).

The majority of community-based studies have not used necropsy data to verify the causes of death. National mortality records are community-based and do, in part, use necropsy reports. There are problems with the coding and indexing of this national data (see section 1.4.3) and the large areas from which these national statistics are gathered may result in significant local changes in fatal disease being overlooked. The annual MOH reports reflected community experience although their mortality sections were based on the death certificate record. There have been a few long-term studies of child mortality in a defined community which utilised these MOH records but these did not use necropsy data to verify the causes of death (Mair and Tait, 1953; Knox and MacKintosh, 1958; Tait and Boog-Watson, 1962).

Published studies of mortality in a defined community of children which used necropsy data to verify the cause of death appear to be limited to studies of selected diseases (e.g. Neel, 1958; McKeown and Record, 1960; Campbell et al., 1961; McDonald, 1961; Froggatt et al., 1971; Chalmers et al., 1978; Reed et al., 1978), selected ages (e.g. Royal College of Obstetricians and Gynaecologists, 1948, 1975; Spence et al., 1954; Butler and Alberman, 1969; Elwood et al., 1974), or selected years (e.g. Dunn and MacGregor, 1964). No long-term comprehensive study of death throughout childhood has been traced in which the community of children at risk is known, in which detailed necropsy records have been consulted to verify the causes of death and where the changing pattern of fatal diseases with time has been analysed. There would appear to be a need for such a study.

1.5.1 Previous studies of neonatal mortality

Numerous studies of death in childhood, whether based on hospital, community or national populations have shown that deaths in the neonatal period are more frequent than at later ages and that more deaths occur in the first month than in any subsequent month of life (e.g. Dunn and MacGregor, 1964; Östberg, 1973; DHSS, 1976a).

The neonatal mortality rate in England and Wales and other large English towns will be compared with that of Sheffield between 1947 and 1979 to identify any secular trend. Because of changes following the local government reorganisation in 1974, towns will be chosen where the population altered by less than 1% following these reforms.

1.5.1(a) Effectiveness of intensive neonatal care in reducing mortality

In the late 1970s there was considerable public debate concerning the relatively poor decline of early childhood mortality rates in Britain when compared with other countries. Macfarlane et al. (1980) suggested that this debate probably started as a response to the DHSS policy of cutting maternity services (DHSS, 1976b). The debate was stimulated by the Court Report (DHSS, 1976a) which highlighted the unfavourable comparison between Britain and other developed countries and also showed the differences between regions of this country. The issue was brought to public attention by an advertising campaign organised by a national charity which used slogans such as 'If you're born British, you could be a born loser' to suggest that Britain's unfavourable position was due to a lack of provision of obstetric and intensive neonatal

facilities. A House of Commons Select Committee enquiry into perinatal and neonatal mortality was convened 'because of mounting public concern that babies were unnecessarily dying or suffering permanent damage', because 'mortality rates in England and Wales were falling more slowly than in other developed countries' and because 'there was such inequality of rates in different socio-economic groups and areas'. The report of this committee, the Short Report (House of Commons, 1980), concluded that lack of intensive neonatal facilities was partly responsible for the high mortality rates and recommended redeployment of (and additional) resources and manpower.

There have been numerous claims for the potential benefit of improved neonatal care facilities which attribute the improvement to advances in medical technology and skilled intervention (e.g. Kitchen and Campbell, 1971; Stewart, 1977; Anonymous, 1979; Stewart et al., 1981). All of the studies claiming this benefit are based on hospital populations and do not consider the possibility of a secular trend. If the neonatal mortality rate began to fall spontaneously before the introduction of intensive neonatal facilities, then claims of a change in survival attributable to these facilities must show a greater rate of decline than would otherwise have been expected. To measure this secular trend it is necessary to have a consistent population at risk. Only one such study has been traced - that by Hughes-Davies (1979) which showed that, in one hospital without intensive neonatal care, the

decline in mortality was comparable to the decline in another, superbly-equipped hospital.

Another justification for the expense of the intensive neonatal facilities has been that there will be a greater reduction in the number of handicapped survivors, with consequent eventual savings. Rawlings et al. (1971) showed that the prognosis for babies weighing less than 1500 g at birth appeared to have improved following the introduction of intensive neonatal care although they could only speculate on what might have happened without such care. However, Jones et al. (1979) showed no significant improvement over 15 years in the proportion of handicap among infants weighing less than 1500 g despite increasing complexity of care. This study stimulated a series of critical letters (Lancet, 1979) from other neonatologists giving hospital figures to support their arguments. These arguments were in essence: very lightweight babies will be the last to benefit and improvements are occurring in the 1500-2000 g group already (this argument is not valid without excluding a secular trend); that there has been no increase in handicap which might be expected as intensive care reduces mortality (this could invalidate the justification for expensive facilities); technical advances prior to the mid-1970s were unlikely to improve handicap and further follow-up will be necessary (this was not the case when expensive units were being called for in the 1960s); some babies will be handicapped before delivery (which partly invalidates the justification for expensive facilities);

and that the staff who established the unit which Jones et al. reported from left in the early 1970s and 'there must have been some setback' (this is a difficult argument as it either suggests that only a very few doctors are capable of running such units or that the remaining staff, including the authors, are not fully competent). Davies (1980) in a careful and balanced review concluded that 'frequently repeated statements that modern methods of perinatal intensive care are leading to a striking reduction in handicap must at present be considered uncritical and almost certainly untrue'.

Studies of intensive neonatal facilities in hospitals serving relatively isolated communities which did not transfer sick neonates show that minimal intervention produced similar mortality figures to intensive activity in a major teaching hospital (Hughes-Davies, 1979) and that postnatal survival and intellectual potential were not prejudiced when only experienced nursing care was available (Steiner et al., 1980). These studies were followed by critical letters from neonatologists who advanced similar arguments to those following the study of Jones et al. (1979). A limitation of the studies by Hughes-Davies (1979) and Steiner et al. (1980) is that they compare the outcome of their hospitals with that of a London teaching hospital. Although Steiner et al. (1980) showed little difference between the type of patient in their hospital and the London hospital, the possibility of some geographical factor influencing outcome cannot be excluded. There is therefore

a need for a study which examines the outcome of maternity hospitals which serve a defined stable community. This can be done in the present study as there are three maternity hospitals in Sheffield with very different facilities for neonatal care. There was no transfer of sick neonates between these hospitals prior to late 1979. Because of the possible varying fatal incidence of malformations, maternal diseases and birth trauma which a SCBU could not be expected to influence, only children dying with the diseases of prematurity will be examined.

1.5.1(b) First day mortality

Pharoah and Alberman (1981) showed that first day mortality in England and Wales remained stable from 1953 to 1962 and subsequently declined steadily in all weight groups. They attribute the decline to the presumed introduction of special care nurseries recommended by the Central Health Services Council (1961). However, they did not show which fatal diseases declined to explain the observed trend. If the decline occurred in deaths from prematurity then the explanation is tenable, but if in other diseases such as malformations, then special care nurseries could not be expected to have contributed to the decline.

Pharoah and Alberman (1981) also showed that the decline in deaths in the age group 1-27 days showed no improvement following 1963 or subsequently which might argue against the efficacy of intensive care nurseries. An alternative explanation for the decline in first day deaths is that there could be an increasing practice of registering

these deaths as stillbirths (Sunderland, 1981a). The Sheffield stillbirth and first day mortality rates will be examined together to explore this possibility. The causes of first day Sheffield deaths between 1947 and 1979 will also be analysed.

1.5.1(c) Rhesus disease

Jaundice of newborn infants has been recognised since the seventeenth century when it was regarded as being similar to adult jaundice, due to occlusion of the bile ducts by glutinous humours. Ballantyne (1892) discussed the suggestion that Hippocrates was speaking of hydrops when he described the birth of a fleshy foetus (foetus carnosus) and traced the earliest report of generalised dropsy of the foetus (hydrops foetalis) to the records of Plater (1614). The classical report of neonatal jaundice is attributed to the midwife of Marie de Medici in 1609 (Clarke 1975).

By 1930 it was apparent that the conditions known variously as erythroblastosis foetalis, congenital oedema, hydrops foetalis, congenital anaemia and icterus gravis neonatorum had many features in common. However, no rational diagnosis was possible until the description of human blood groups (Landsteiner, 1900) was advanced with the discovery of the Rhesus (Rh) factor (Landsteiner and Wiener, 1940). Following these discoveries the treatment of affected babies by transfusion and later exchange transfusion began. The first preventive steps were also taken in that no more Rh negative females were transfused

with Rh positive blood and intramuscular injections of blood products for intractable skin disease ceased (Levine et al., 1941; Diamond, 1947).

Shortly before the description of the Rhesus factor, Darrow (1938) postulated that these congenital anaemias could be due to the baby's red cells being destroyed by some immune reaction of the mother, possibly following an accident within the placenta but she concluded that "this mechanism bears no relation to a difference in blood groups in mother and child".

Levine et al. (1941) showed that 93% of cases of erythroblastosis foetalis were due to iso-immnisation of Rh negative mothers by an Rh positive foetus and that the Rh antigen was inherited from the father. They were unable to explain why the recorded incidence of Rh disease (1 in 400) was much lower than expected from simple chance of Rh positive/Rh negative mating (1 in 20). One important reason for this remarkably low incidence was that ABO incompatibility between mother and foetus would lead to destruction of the foetal red cells before Rh sentisation could occur. Levine (1943) drew attention to the fact that erythroblastosis was lower than expected when there was ABO incompatibility but merely concluded that factors other than Rh may be responsible for abortions and stillbirths. It was Race and Sanger (1950) who first proposed that ABO incompatibility might be protective against Rh immunisation. Nevanlinna and Vainio (1956) then showed that the immunising foetus (a healthy Rh+ child born immediately before the

first affected child) was much more commonly ABO compatible than incompatible. They also showed that after immunisation had occurred, ABO incompatibility was no longer protective. Jewkes et al. (1969) attribute to Lady Clarke the original idea of using anti-Rh antibody to prevent the occurrence of isoimmunisation. Clarke (1975) has comprehensively reviewed the subsequent development of the anti-Rh gammaglobulin (anti-D).

Meanwhile an MRC controlled trial showed that exchange transfusion was superior to simple transfusion but that unselective early delivery (to reduce the baby's exposure to maternal antibodies) led to increased mortality from prematurity (Mollison and Walker, 1952). Bevis (1952) suggested that severely affected pregnancies might be predicted by measuring amniotic pigments and then selectively induced. After experimenting unsuccessfully with ferrous ions, he showed that urobilinogen concentration was a good predictor of severe disease (Bevis, 1952). The final significant steps in active management were when Liley (1963) reported the first successful intrauterine transfusion and Rodeck et al. (1981) reported the first intrauterine exchange transfusion.

These developments were quickly introduced to Sheffield and Sheffield participated in the first clinical trial of the anti-D antibody which commenced in May 1964 (Combined Study 1966, 1971). The trial was discontinued in 1967 because it was thought no longer ethical to use controls. Treatment was continued in all women found to

have foetal cells in their circulation and since January 1968 the National Health Service together with the Blood Transfusion Services have offered anti-D gammaglobulin to all Rh negative women shown to be at risk of developing Rh antibodies.

Published work has principally concentrated on the effect of preventive measures on the numbers of affected babies delivered. Little attention has been paid to the contribution of these measures in reducing infant mortality. This study will examine the changing incidence of mortality from rhesus disease with time and attempt to determine whether active treatment or preventive measures played the greater part in reducing mortality.

1.5.1(d) Comparison of Sheffield early neonatal deaths with the British Births surveys

A number of national studies have been conducted which have obtained information about early childhood mortality. These studies have taken a cohort of children born in one week in early spring and some of the studies have also examined the causes of all neonatal deaths among children born in one month (Royal College of Obstetricians and Gynaecologists, 1948, 1975; Butler and Bonham, 1963). Extrapolations have been made from these studies to produce annual mortality figures. An implicit assumption in this extrapolation is that the date or season of birth has no effect on a child's subsequent experience of illness or death. This assumption does not appear to have been tested. It can be tested in two ways: by examining the incidence

of a disease in a number of cohorts spread throughout the year or by examining the distribution of the causes of death against the season of birth. The former approach has been used elsewhere in a study of febrile convulsions (Sunderland et al., 1981). The second approach will be used in the present study.

In the 1958 and 1970 studies the Sheffield region was nearest to the national average (Butler and Bonham, 1963; Royal College of Obstetricians and Gynaecologists, 1975). The assumption concerning the null effect of season of birth on subsequent death will be tested by comparing the distribution of fatal early neonatal pathologies among the Sheffield children born in March 1958 and the week 5-11 April 1970 with the distribution of neonatal pathologies for the whole year (1958 and 1970 respectively). If the distribution of the causes of death among the sample is the same as the distribution for the whole year then the assumption concerning the null effect of season of birth on experience of disease will have been shown to be tenable for this small area of the country.

The early neonatal mortality rate for each monthly birth cohort will then be examined. If there is no significant variation then the assumption concerning the null effect of season of birth on mortality will have been shown to be reasonable.

1.5.2 Studies of malformations

Malformations have been recorded since prehistoric times but it was only after postnatal causes of child mortality

declined that prenatal causes emerged as important factors. There is some loss of life throughout pregnancy and structural anomalies of the embryo are an important cause of foetal loss in the first trimester (Warkany, 1971). Nishimura et al. (1966) examined the conceptuses removed at social abortion and found that polydactyly and myeloschisis were more than ten times commoner than in infants born at term. Carr (1967) found that chromosomal anomalies were fifty times commoner in spontaneous abortions than in live born infants. Butler and Bonham (1963) estimated that about 20% of all stillborn infants are deformed. It would therefore appear that a form of natural selection operates throughout pregnancy, removing the unfit foetuses. However, many structural anomalies which are incompatible with independent existence are quite compatible with intrauterine life and therefore there may be a large number of deaths from untreatable malformations in the neonatal period.

There have been many studies on the incidence of malformations but comparison of the reported figures is difficult because some include stillbirths, some include all foetal deaths, some exclude minor malformations and few define the populations studied. For example, Ehrat (1948) found that the incidence of malformations in the world literature varied from 0.3 to 30.3 per thousand total births. She also showed that the recorded proportion of malformed babies born in her own hospital rose from 2.6 to 14.8 per thousand live births between 1921 and 1944. These variations in the world figures can be explained by variations in the level of ascertainment in the various studies which would also be influenced by the particular interests of

the investigator. The striking increase in Ehrat's hospital figures appears to be due to an increasing quality of diagnosis and the inclusion of minor malformations (e.g. club foot) in the later years of the study. In another hospital study Nelson and Forfar (1969) found abnormalities in 470 of 8684 Edinburgh births, but 172 of these babies were stillborn and of the remainder only 1.3% were judged to have major malformations. The majority of studies of malformation incidence are hospital-based and cannot be standardised for the population at risk. Variations in reported incidence with time could therefore be due to variations in the population sample.

Leck and Record (1963) examined five community-based studies and found the incidence of malformations at birth to be between 12 and 18.5 per thousand births. They also found that the levels of ascertainment vary with the length of follow up. The incidence of malformations nearly doubles when children are followed for up to five years (Leck and Record, 1963). However, many of these community studies used notifications from public health records. Leck and Record (1963) showed that between 36% and 68% of malformations identified from hospital records are not notified - even gross abnormalities such as anencephalus. Finally, Kane (1964) dismissed studies of malformation incidence which did not use necropsy data as being "unworthy of collection, let alone publication". Detailed records of the proportion of children with malformations born in a population can probably only be achieved where the population is small enough for one observer to collect all data and where a high necropsy rate can be achieved. While malformations in survivors will give a more accurate picture, internal malformations

will probably be underdiagnosed. Therefore a study of malformations which cause death may be the best method at present of estimating the burden of serious handicap in a community.

Many studies present their results as proportions of child mortality, indicating the increasing importance of malformations as a cause of death. However, as deaths from other causes decline, it is an inevitable algebraic statement that malformation deaths will rise as a proportion of all deaths. It is more important to discover what has happened to malformation deaths as a proportion of births. It is also more useful to analyse malformation deaths by year of birth rather than year of death if aetiological factors are to be traced. However, the use of mortality data to analyse aetiology can be misleading. Warkany (1971) gives an analogy of the search for teratogens being similar to seeking the cause of a train derailment: a railway timetable may be useful because it will indicate the last moment that the damage could have been caused but an act of sabotage could have been committed immediately, or hours before the accident, or the derailment could have been due to neglect of the railbed for years or even decades preceding the disaster. Similarly a malformation may be caused by a teratogen acting in utero or due to previous environmental or genetic factors.

There would appear to be a need for a study of malformation deaths along children who were live born and have been followed throughout childhood, in whom detailed necropsies have been done and where only malformations sufficient to cause death (decided by a single observer) have been included.

1.5.2(a) Neural tube defects

The neural tube defects (NTD) are important malformations which are particularly useful for epidemiological research because they are both grossly obvious and severe. They are therefore far more readily ascertained than internal malformations and are often so lethal that they are likely to be present as a cause of death in archived mortality records. It is also unlikely that affected children dying with NTD will have another cause of death on the death certificate and thus there will be little diagnostic variation with time.

The lesion known as spina bifida was first described by Tulp in 1652. The association between hydrocephalus and spina bifida was reported by Morgagni in 1761. Between 1883 and 1894 the characteristic malformation of the brainstem responsible for the hydrocephalus was described (Cleland, 1883; Chiari, 1891, 1895; Arnold, 1894). Until the hydrocephalus could be controlled, the condition was often fatal despite attempts at surgical treatment such as puncturing or ligating the lumbar sac (Newbigging, 1834).

Mortality rates from spina bifida are available for England and Wales since 1848. There has been a gradual decline in this mortality rate throughout the twentieth century but, between 1969 and 1970, this decline reversed. It has been suggested that this national change was due to the introduction of selection prior to surgery (OPCS, 1976a). It is possible, however, that the change was due to other, non-medical factors. Because the local timing

of the changes in medical and surgical management of these children is known with some precision in Sheffield, it may be possible in the present study to answer this.

Following the thalidomide tragedy, each MOH was required to keep a register of all handicapped and malformed children. Prior to these registers, the overall incidence of children born with malformations had to be inferred from mortality rates. For serious lesions such as spina bifida, the incidence was often inferred to be the same as the mortality rate (OPCS, 1976b). In the present study it will be possible to examine both birth rate (i.e. incidence) and mortality rate after 1963 for Sheffield's children to see whether this assumption is valid.

The introduction of shunts to control hydrocephalus revolutionised management of spina bifida and, following the introduction of the Holter valve, attempts began at early corrective surgical closure of the back wound (Sharrard et al., 1963). From 1962 all children born in Sheffield with spina bifida were treated as surgical emergencies. If necessary, active resuscitation was carried out on any child regardless of the degree of paralysis or the presence of multiple abnormalities. Early hopes that this aggressive management would enable many of these children to lead a normal life were not realised. Among others, Lorber (1973) showed that not only were these hopes unfounded, but also that early closure of the back did not even improve muscle function. Additionally, many social and marital problems appeared to be aggravated by the survival of such severely

handicapped children and other children in the families were also suffering. Thus, after a few years, some of Sheffield's paediatricians considered the policy of surgery for all to be incorrect and selection prior to surgery was introduced in May 1971 (Lorber, 1973). This policy was not immediately accepted and, for some time afterwards, some children were referred for surgery regardless of severity of their lesion (Lister, 1973).

The discovery that alpha-fetoprotein (AFP) was a marker molecule suitable for the antenatal diagnosis of a child with an open neural tube defect allowed the possibility of a therapeutic abortion early in the pregnancy (Brock and Sutcliffe, 1972). Since January 1973, AFP levels have been measured in the amniotic fluid (AF-AFP) of all Sheffield mothers who had a family history of babies with NTD.

The discovery that elevated AFP levels could be detected in maternal serum (MS-AFP) allowed the introduction of a screening programme without the risks of amniotic puncture (Brock et al., 1973). Since January 1977 all pregnant women in Sheffield have been offered a serum-AFP screening test at 16-18 weeks gestation. This is repeated if elevated and followed by amniocentesis if both levels were greater than the 97.5 centile, the pregnancy known to be a singleton and the gestation confirmed by ultrasound scan. An abortion is offered to all women who have amniotic fluid AFP levels greater than six standard deviations about the mean for gestation.

The introduction of screening plus abortion would be expected to reduce the incidence of live born children with NTD. The present study will assess the effectiveness and efficiency of the screening programme. This screening programme, if effective, would also lower the mortality rate artefactually whereas selection prior to surgery might raise the mortality rate. However, selection prior to surgery was argued as being a reasonable procedure because the most severely affected infants, who were selected for no treatment, would have died regardless (Lorber, 1973). This study will be able to determine the effect on mortality rates and survival curves of all of the changes in management of spina bifida which were pioneered in Sheffield.

1.5.3 Studies of infectious disease mortality

At the beginning of the present century more children died from infectious diseases than from all other causes combined. Some of the major causes of these deaths in Sheffield were described in section 1.2.2. As more than half of all deaths occurred in children, infectious diseases were a major problem. There were many who argued that intervention to prevent these deaths would merely provide a burden for the future by permitting the unfit to survive. The argument was that unfit children became unfit workers and gave birth to more unfit children. These arguments were refuted by Chadwick and other reformers (see section 1.2.4). Drummond (1901) also showed the fallacy of these non-interventionist arguments by demonstrating that epidemics strike both the weak and the strong, not individuals but communities. He asked whether the nation was weaker

following the disappearance of smallpox and cholera and, if non-intervention selected out the fit, what were they fit for but slum dwelling? By the middle of the present century deaths from infectious diseases were uncommon and in the last quarter of the century such deaths have become rare. The reasons for this decline have been much debated and are probably multiple. Three possible reasons for the decline are: changes in the virulence of micro-organisms, changes in host resistance to infection and changes in treatment.

Each generation appears to have believed that their own panacea was the most important. Thus autogenous vaccines, phagocytic stimulation and serum treatments were all in turn reported as being most effective. Antibiotics were similarly regarded in the 1960s. However, the historical data show that mortality from infectious diseases had become relatively insignificant problems before many of these therapies could be introduced. They may have been important in the decline of morbidity but could have had only marginal effects on the decline of mortality.

Galbraith et al. (1980) in a review of the changing patterns of communicable disease in England and Wales argue that the conquest of diseases which have disappeared or declined was due to the introduction of effective vaccines. However, McKeown and Lowe (1974) consider that the decline in mortality began in the eighteenth century and was due to environmental and nutritional changes. Others (e.g. DHSS, 1976b) suggest that the conquest of at least some of these diseases was due to the

introduction of antibiotics. In the present study, available data on the time trends of deaths from infectious diseases among Sheffield's children will be correlated with the introduction of environmental controls and medical treatments.

Changes in the virulence of micro-organisms could result in a decline in mortality even if there were no changes in host resistance, antimicrobial measures or the infectivity of these organisms. Such changes have apparently occurred in certain diseases, especially scarlet fever. This disease was described as 'Hoc nomen morbi' (a disease only in name) by Sydenham and was often confused with mild measles (Singer and Underwood, 1962). Yet in the first half and at the end of the eighteenth century and also between 1830 and 1875, scarlatina was a fearful disease. In the intervening and subsequent periods the disease was common but mild. It now appears that the cutaneous, cardiac and renal consequences of beta-haemolytic streptococcal infection are due to an erythrogenic toxin which is only produced when the streptococci are lysogenised by a specific bacteriophage (see Hodes, 1979). It is possible that the changes in virulence of the streptococci were due to the presence or absence of the phage. Similarly, Corynebacterium diphtheriae may be present in the throat without causing clinical diphtheria and without secreting the toxin. Freeman (1951) showed that only strains of C. diphtheriae that were infected with a specific bacteriophage were able to produce toxin. Diphtheria has not shown the same historical cycles as scarlet fever but the decline in mortality may be due in part to the disappearance of the phage. Other infections may also have declined because of changes in the virulence of the causative organism.

To establish an infection, an organism must come into contact with the host and must have a certain infectivity. The host's resistance must also be sufficiently depressed to allow the organisms to multiply. This combination of factors may occur in overcrowded, malnourished populations. Toverud (1949) showed that the infant mortality rate of the European royal families in the early part of this century was less than 10 per thousand, notwithstanding the presence of a number of lethal hereditary diseases. In contrast, national rates were greater than 150 infant deaths per thousand. Increasing urbanisation which followed the Industrial Revolution brought large numbers of people together, creating the conditions for epidemics. It was widely assumed that these new town dwellers had left sylvan conditions. However, exposure to the elements, overcrowding, poor nutrition, sanitation and medical access may have been much worse in the countryside and there was much less security for agricultural workers - a bad harvest was far commoner than an economic recession. McKeown and Lowe (1974) argued that the agricultural and industrial revolutions were both beneficial factors in the decline of mortality.

There is evidence from studies of communities which have not yet been affected by the agricultural revolution that most deaths from infectious disease are, in reality, deaths from malnutrition (Anonymous, 1978a). Many of the late nineteenth century infection deaths were possibly predisposed by malnutrition and chronic infections. Somewhere, this cycle of deprivation had to be broken and there is a temporal association in this country with the sanitary reforms of the late nineteenth

century. Such associations do not necessarily imply a causal link. However, changes introduced after the decline in child mortality commenced are unlikely to have been causally effective. The present study will attempt to identify some of the reforms which could have been effective.

1.5.4 Sudden Infant Death Syndrome (SIDS)

Unexpected, inexplicable infant deaths have been known throughout history. The harlot's child involved in the judgement of Solomon (I Kings 3: 16-28) may have been a SIDS death although the evidence of one of the harlots suggests that the child died on the first postpartum night. For clarity and brevity, in this study all unexpected inexplicable deaths will be referred to as SIDS unless specified otherwise for historical or other reasons. It would appear that this mode of death was well known to the Bible translators in 1611. Their translation 'this woman's child died in the night; because she overlaid it' is a classical description of this condition.

The description by Templeman (1892) of overlaid infants is a valid description of what is now known as the sudden infant death syndrome: 'The external appearances presented by the body are chiefly of a negative character. There are no marks of violence to be found. As a rule there is no flattening of the nose and face from pressure. Post mortem lividity comes on early, and is specially well marked on that side of the body on which the infant has been lying; the face is placid and calm; the eyes sometimes slightly congested, but not staring; the lips are livid, the tongue not protruded. Frothy mucus, often tinged with blood, is generally seen about the mouth and nostrils.

The hands are sometimes tightly clenched'. He was not permitted to do a necropsy in many cases but in those examined he found 'a varying degree of congestion of the cerebral membranes - more or less engorgement of the internal organs, especially the lungs and kidneys, and the large thoracic veins, a fluid condition of the blood, which was not dark in colour; and generally a distended condition of the right side of the heart, while the left was nearly or altogether empty and contracted (in one case both sides of the heart were completely empty). In about half of the cases examined small punctiform haemorrhages were observed beneath the pleura and pericardium. The larynx, trachea and bronchi were, as a rule, congested, and contained some frothy, often blood-stained mucus'.

1.5.4(a) Development of the concept of SIDS

The concept of a sudden infant death syndrome is believed to have arisen when, following the disappearance of deaths from infectious disease, it was found that a sizeable proportion of infant deaths occurred in children who had apparently been completely well and in whom no significant pathological features could be found (Peterson, 1980). Such sudden deaths are dealt with by the Coroner and forensic pathologists, who are primarily concerned with the identification of unnatural deaths. Having excluded unnatural causes, the problem of labelling these deaths remained.

Emery (1976) argued that SIDS, cot death, crib death and other synonyms were invented for the convenience of considering these deaths as a single entity. However,

this ignores many earlier reports. The problem of sudden infant death was a major problem in the period of high infectious disease mortality (Templeman, 1892) and a natural aetiology for these deaths was proposed in preference to overlaying in 1834 (Fearn, 1834). The possibility of a multiple aetiology for sudden infant deaths has been known to forensic pathologists for years: Bowden (1952) showed how careful necropsy would reveal many natural pathologies in cases labelled by clinicians or parents as overlayings. Thus the medical 'discovery' of SIDS in the early 1960s was as much an artefact following the increasing evidence that these deaths could not be attributed to suffocation as the increasing clinical awareness that children were still dying despite the conquest of the epidemic infections (Werne, 1942; Simpson, 1947; Rabson, 1949).

1.5.4(b) Apparent increasing incidence of SIDS

Whatever its origins, the description and publicity of the sudden infant death syndrome in the early 1960s was followed by a rapid increase in the numbers of such deaths registered. Pharoah and Morris (1979), reviewing the causes of postneonatal mortality, comment on this increase as 'surely an artefact of registration; deaths which in the past would have been attributed to pneumonia or gastro-intestinal infection, for example, are now certified as sudden death, cause unknown'. Emery (1976) considered that the increase was genuine, although he was unsure whether the 'epidemic' was superimposed on a core of such

deaths which only emerged following the decline in infectious deaths or whether it was a new phenomenon.

By 1971, SIDS were identifiable on national statistics (OPCS, 1980c). Between 1971 and 1978, SIDS deaths in England and Wales increased in marked contrast to the overall decline in postneonatal mortality. To accept the argument of Pharoah and Morris (1979) that this was an artefactual increase is also to accept that there is a reciprocal artefact in the published figures of declining mortality from all other causes in the postneonatal period. It is possible that Pharoah and Morris (1979) are correct, but as Pilling (1976) states, 'to assume that doctors are increasingly using SIDS as a diagnosis without justification depends upon one's faith in the integrity of the medical profession in observing the regulations relating to death certification'.

Pilling (1976) noted that there had been a marked increase in the numbers of SIDS identified to the Sheffield Coroner after 1963. (Coincidentally, this was the year of the first Conference on Causes of Sudden Death in Infants (Peterson, 1980).) Pilling (1976) found that, until the mid-1950s, sudden infant deaths had all been regarded as due to overlaying or smothering by bedclothes and there were never more than 10 such deaths per year in Sheffield. In 1972 there were 31 such deaths. In an elegant study he re-examined the Coroner's records for Sheffield between 1961 and 1972 and confirmed that the SIDS mortality rate in the postneonatal age group rose

from 0.86 to 3.18 per thousand live births. But he also showed that this could not be entirely due to an artefact of registration because the overall postneonatal mortality rate also rose from 5.6 to 6.8 per thousand live births. He did not speculate on causal factors but observed the association of a rising postneonatal mortality rate with densely populated areas which also occurred elsewhere in the country. He has subsequently informed the present author that he believes the rise in Sheffield SIDS deaths might, in part, have been due to a transfer of registrations from pneumonia or gastro-enteritis deaths but that there was a genuine increase of SIDS deaths which he thought was due to some environmental change, possibly associated with feeding practices (Pilling, 1981, personal communication).

Pilling's study (Pilling, 1976) has a number of limitations. He did not check that the Coroner's records only related to Sheffield residents. Non-resident children may have died in the city while visiting relatives or have been brought to a Sheffield hospital by ambulance. Secondly, he made the reasonable assumption that all sudden infant deaths would be reported to the Coroner. This may be incorrect, e.g. if a family doctor wished to avoid an inquest to spare the family or himself (see Havard, 1960). Thirdly, he was unable to identify Sheffield children who died outside the city, as these would come under another Coroner's jurisdiction. This last criticism has been made of almost all population-based studies (Peterson, 1980).

To overcome these limitations, there would appear to be a need for a study where a single observer examined all records of postneonatal deaths among a population of children, regardless of where these deaths occurred, using a standard classification and while ignorant of the year of death.

1.5.4(c) Definitions of SIDS

Reviewing the literature on SIDS is difficult because of the variety of definitions used. There are essentially three groups of definitions: those which include all unexpected infant deaths (the clinical definition); those which include all inexplicable infant deaths (the pathological definition) and those which only include unexpected and inexplicable infant deaths (the author's definition).

An advantage of the clinical definition of SIDS is that it has forced into professional awareness that many childhood diseases do not have premonitory features and can be rapidly fatal. Thus, infants with a ductus-dependent congenital heart defect, a capillary bronchiolitis, or a meningococcal septicaemia may die within hours of the first sign of abnormality. However, a study of epidemiological features of fatal congenital heart disease would be of little value if it included deaths with meningococcal infections. Likewise, a study of SIDS must be based on a discrete entity (or as finite as possible, given present knowledge). On the other hand, a purely pathological definition of SIDS would include many neonatal and even adult deaths and is of equally limited value. Many

apparently inexplicable deaths are often readily explained from the clinical history and many were expected. Thus a pathological definition of SIDS is also unsatisfactory.

There appears to be a single discrete entity which presents as sudden death in infancy. The descriptions of sudden inexplicable deaths in the literature have a remarkable similarity despite geographical, temporal and cultural separations (see Fearn, 1834; Templeman, 1892; Simpson, 1947; Rabson, 1949; Werne and Garrow, 1953a, 1953b; Valdes-Dapena, 1967; Froggatt et al., 1971 and Peterson, 1980). All of these authors imply that there may be a single aetiology for the majority of these deaths although all stress that there is no evidence of a single disease. From a careful study of these reports it is apparent that there may be a single condition which affects young infants and which can be defined for meaningful epidemiological study as a death which is unexpected by history and in which a thorough necropsy failed to demonstrate an adequate cause of death. This definition is imperfect, especially as 'adequate' may be difficult to define, but an attempt will be made to overcome this in the present study.

1.5.4(d) Epidemiology of SIDS

Despite problems of definition, incidence rates of SIDS vary little from 0.6 to 3.0 per thousand live births in reports from all parts of the world and from primitive, rural and urban societies (Valdes-Dapena, 1967; Peterson, 1980). There are two features reported in all studies:

the lack of significant pathology and a peak incidence at three months of age. Other factors are: male preponderance; seasonal distribution (commoner in winter, correlating with peak incidence of viral infections); an excess in twins, in disadvantaged families, in crowded dwellings, among young mothers with high parity and possibly in premature infants; an apparent increase in minor illnesses shortly before death; a possible familial incidence but no specific inheritance pattern; an absence of significant space-time clustering; and no apparent peak on any particular day of the week (despite some early reports of a peak incidence on weekends).

The very large majority of these deaths occur at night and thus adult observation of the final events is limited. There are, however, well-documented case reports of infants dying while observed (Templeman, 1892; Rabson, 1949; Werne and Garrow, 1953b). These reports suggest that some children are awake immediately prior to death. There is no evidence of any terminal cry and parents are rarely wakened during the fatal night

1.5.4(e) Theories of SIDS aetiology

There has been much literature on the aetiology of SIDS. Many of the theories are merely speculations based on limited knowledge of the problem and with little factual support. Such theories are often difficult to test and will not be considered here.

The most persistent theory among both public and the medical profession is that of homicide or infanticide.

Suspicions of this aetiology were a major concern in the 1890s and are still advanced in the 1980s. The report of the Select Committee of the House of Lords on the Children's Life Insurance Bill, 1890 discussed the effect of such insurance on children's lives, infant mortality and the frequency of overlaying. Templeman (1892) showed that a financial motive could not explain many of the deaths in his series because few of the children had been insured and because it was not possible to take out a policy on an illegitimate child under three years. Simpson (1947), Rabson (1949), Bowden (1952) and Werne and Garrow (1953a) all discount an homicidal aetiology for these deaths. Werne and Garrow (1953a) found no evidence of poisoning in any case in their series. Kukull and Peterson (1977) suggested that few SIDS deaths could be due to infanticide because, despite the great increase in SIDS registrations in Washington State in the period 1954-1974, child homicide rates showed no reciprocal decline but also rose from 0.004 to 0.060 per thousand live births.

Despite this lack of evidence, suspicions of infanticide remain. This is understandable because of the lack of supporting evidence for any reasonable alternative explanation. The fear of the unknown leads to the desire for any explanation. Bowden (1952) reported that even some parents would accept infanticide if this could explain how their child died. There is an important legal difference between infanticide and accidental suffocation (see Bowden (1952) for summary of an 1897 judgement). This

legal difference is of little comfort to the parents however. There has always been a stigma attached to parents of children who die as SIDS. A sermon on this stigma stimulated Yeats to write his Ballad of Moll Magee (Yeats, 1889).

Until the 1950s, the commonest explanation for SIDS was overlaying and theories of aetiology revolved around the physical mechanism, including: pendulous breasts, carelessness, inebriation and suffocation by bedclothes. The concept of overlaying was gradually discarded by most forensic pathologists (Simpson, 1947; Rabson, 1949; Werne and Garrow, 1953a). Although the pathological findings are compatible with acute suffocation, it was the opinion of these pathologists that there was no evidence to support the theory of an extrinsic factor causing this suffocation. It was also the opinion of the majority of pathologists that a normal sleeping adult would be aroused by the struggles of an overlain infant before suffocation occurred (Valdes-Dapena, 1967).

With increasing prosperity following the second world war, it became fashionable for all parents to have cots for their children. Unexpected inexplicable deaths still occurred and could no longer be explained by overlaying. The public and medical profession were forced to the conclusion already accepted by forensic pathologists. This may partially explain the surge in SIDS registrations.

In the search for an internal factor causing suffocation, some adult pathologists observed the large infantile

thymus and proposed that status thymacticus could be the cause of death. However, these thymuses were of normal proportions for age and there was no evidence of airways obstruction (Simpson, 1947).

These deaths were next explained as being due to suffocation by the bedclothes or pillow. But this was unable to explain why many infants died on their backs with a clear airway nor could it explain observed deaths (Werne and Garrow, 1953b).

The discovery of vomitus in the airways offered an explanation of inhalation asphyxia for those infants who died on their backs. But there was often no inflammatory reaction which would suggest that the vomiting and inhalation were agonal events. An uncontrolled observation that many SIDS infants had raised levels of IgG antibodies against cow's milk protein led to the hypothesis that SIDS was due to an allergic/anaphylactic response to cow's milk in the inhaled vomitus (Parish et al., 1960). It was subsequently shown that guinea pigs sensitised to cow's milk would die suddenly with minimal pathology when challenged with intra-tracheal milk or intravenous injection of the antigen. However, controlled studies found that virtually all infants fed with cow's milk develop antibodies and that milk was found in the airways of children whose deaths were explicable (Valdes-Dapena, 1967). SIDS deaths also occur in children fed exclusively on human milk. Other theories of anaphylaxis have been subsequently proposed of which perhaps the most promising

was the suggestion that a second infection with a virus, following neonatal or intrauterine exposure, could produce an anaphylactic reaction. However confirmatory evidence for these theories is still lacking (Valdes-Dapena, 1967).

As far back as 1895 it was suggested that SIDS were due to a pulmonary infection (Brouardel, 1895 cited by Werne and Garrow, 1953a). This observation was overlooked until the 1940s when, with the decline in other deaths, it became possible to do more detailed necropsies on the SIDS deaths. Werne and Garrow (1953a, 1953b), came to the conclusion that the majority of these deaths were due to pulmonary infection. Simpson (1947) agreed with Hubble and Osborn (1941) that there is no doubt that bronchitis or bronchiolitis can develop in less than 18 hours but argued that during any asphyxiation the lungs became congested and excess mucus, oedema fluid and even blood will accumulate in the air passages. He also dismissed the practice of attributing such deaths to inhalation of vomit because the vomit may be an agonal reflection of the state of asphyxia already present (Simpson, 1947).

It has been suggested that some of these deaths are due to common infectious diseases which are in the invasive stage (Werne and Garrow, 1953a; Emery, 1976). Had the child survived long enough, he would have developed classical signs or symptoms and been seen by a doctor and possibly admitted to hospital. But, it is argued, by the time a child has developed classical signs, he has mounted a resistance which might be sufficient to effect a cure.

This could explain why so few SIDS occur in hospital. This hypothesis explains much of the puzzling data of SIDS, but is almost impossible to prove. The hypothesis could be tested by examining time trends of SIDS against fatal infectious disease trends in the same community of children. Presumably some of the factors responsible for the decline in infectious deaths would also produce a reduction in SIDS rate. Such an effect will be sought.

In the 1970s it was suggested that these infants may not die of any particular 'disease' but because they encounter some critical combination of factors while passing through a period of physiological vulnerability (Froggatt et al., 1971). Recent theories suggest that, possibly because of some immaturity or minor developmental handicap, a vital reflex may fail at a crucial period. The principal reflexes being investigated are: respiratory control (apnoea unresponsiveness and diving reflexes), control of cardiac rhythm and inhalation avoidance reflexes (see Peterson, 1980). There have been many other mechanisms proposed to explain these deaths, some of which have been reviewed by Valdes-Dapena (1967), Peterson (1980) and Moore (1981). None of these hypotheses to date has been consistently supported by data from controlled studies.

1.5.4(f) Hypernatraemic dehydration in SIDS and other infant deaths

Hypernatraemia has been recognised as a complication of infantile diarrhoea since 1911 (Salge, 1911). In 1972 it was suggested that hypernatraemia might be potentiated

by overconcentrated feeds (Taitz and Byers, 1972). Further studies showed that babies fed on cow's milk had a higher plasma osmolality and a higher urea concentration than breast-fed babies (Davies, 1973; Dale et al., 1975). Smith (1974) showed that 172 out of 302 (57%) Sheffield mothers prepared overconcentrated feeds. In 1974 it was shown that in 12 out of 25 unexpected infant deaths the vitreous fluid indicated severe antemortem electrolyte disturbance (Emery et al., 1974). It was postulated that the hypernatraemia was due to the infants being fed over-concentrated feeds and that high-solute feeding might predispose to unexpected infant deaths.

Following this postulation, a small working party in Sheffield began to publicise the possible dangers of over-concentrated infant feeds. The working party contacted every midwife, health visitor and family doctor to stress the potential danger of hypernatraemia and every mother who did not wish to breast feed was individually taught how to reconstitute powdered milks correctly. The Coroner commented publicly on some deaths in which hypernatraemia was found. The resulting public interest generated widespread press coverage. In 1974 the DHSS working party on infant feeding published their recommendations which included the removal of high-solute milks such as National Dried Milk (DHSS, 1974). The effect of these measures on infant mortality will be examined in the present study.

The widespread publicity that it was possible to kill a baby by giving overconcentrated feeds would be expected

to produce an increase in SIDS if the major aetiology is premeditated homicide. This study will examine whether this hypothesis is tenable - an increase in SIDS after 1974 would support it, a decrease would preclude it.

1.5.4(g) Attempts at prevention of SIDS

These deaths appear to occur so suddenly that resuscitative measures are likely to be unavailable. Therefore preventive efforts have been directed at pre-empting the terminal events. In the absence of an explanation of these terminal events, preventive measures have had to be empirical. Monitoring of respiration is used because it is argued that it is necessary for a child to stop breathing before death ensues. It is hoped that such monitors will detect terminal apnoea in time for some intervention. However, if terminal apnoea is consequent upon some other lethal event then such interventions may not be successful. If the terminal events are a combination of factors at a crucial period of development then it is hoped that the vulnerable child can be identified and protected through this period.

Other projects have sought a means of identifying infants at risk by seeking some abnormality (for example, of cardiac rhythm) in the neonatal period and yet others have sought to identify infants at risk by multivariate analysis of commonly available information (Peterson, 1980). Sheffield attempted this latter method by using a score derived from birth data and then directing specially trained health visitors to visit children with a high score. A control group of high score infants was identified but

not given any special attention. During 1974-1975, the years of the experiment, there was a reduction in the numbers of cot deaths in the city (Carpenter and Emery, 1977). But the difference in decline between the control group and those receiving extra visits was not significant.

This health visitor experiment might have coincided with a natural decline in SIDS. In this study, the annual incidence of SIDS in Sheffield between 1947 and 1979 will be examined to establish whether this might have been the case. The postneonatal mortality rate will also be examined in other towns which did not use health visitors in this way to see whether there was a similar decline in these towns (SIDS forms a significant proportion of the deaths in the postneonatal age group, OPCS, 1980). The changes in local government boundaries in 1974 significantly altered the structure of the population in many large towns. Therefore towns were chosen for this study if they had less than a 1% change in population following the 1974 reforms.

1.5.5 Malignant diseases

Malignant diseases are the second commonest cause of death among children aged 1-15 years. The epidemiology of childhood malignant disease has been reviewed by Marsden and Steward (1976) and Birch (1979). Leukaemia has been reviewed by Doll (1972) and OHE (1980). There have been very few community-based, histologically-consistent studies of childhood malignant disease. Birch et al. (1980) found very few other reliable population-based studies for which specific histology is

available. Additionally, changes in disease coding or changes in populations attending hospitals could lead to great artefactual changes in the reported incidence of malignant disease. The present study will endeavour to overcome these problems. Sheffield is especially suitable for such a study because, in addition to the features outlined in section 1.1.4, it has maintained detailed cancer registration data for a long time (Doll, 1972).

Malignant disease in childhood has a very different pattern from that of adults. The commonest malignancies in adults are of epithelial origin (e.g. bronchus, colon) whereas in children they are from a number of tissues. The commonest malignancies in children are, in descending order: leukaemias, central nervous system tumours, neuroblastomas, soft tissue sarcomas and nephroblastomas (Birch et al., 1980). Because of the variety of tissues from which childhood tumours may arise, classification by site is inappropriate and an histological classification is necessary.

Leukaemia is the commonest childhood malignancy, accounting for about one-third of all childhood malignancy deaths (Birch et al., 1980). Childhood leukaemias are most commonly of the acute form and are predominantly of the acute lymphoblastic type. The incidence of leukaemia mortality has apparently increased in this country since the beginning of the present century (see Doll, 1972). It is argued that much of this increase could be due to improved diagnosis or ascertainment. It is also possible that some deaths registered as pneumonias or other infections were predisposed to by an underlying malignancy. The present

study will attempt to avoid these problems by using detailed necropsy data.

The incidence of tumours of the CNS, neuroblastoma and nephroblastoma have all apparently increased in Scandinavia in the period 1954-1974 (see Birch et al., 1980). However, the Scandinavian studies did not report on ascertainment. Birch et al. (1980) found no change in the incidence of these tumours in the north west of England in a similar period. In the present study, the pattern of fatal incidence of these and other solid tumours will be examined.

1.5.6 Unnatural deaths

In contrast to deaths from natural causes, deaths from unnatural causes have shown no historical decline and are now the commonest mode of death in children aged 1-15 years in the developed world.

Unnatural deaths can be subdivided into intentional or accidental. Intentional deaths would include homicides (murder and manslaughter), infanticide and suicide. Murders are deaths which followed actions intended to cause death whereas manslaughter is a death caused by an action which, however premeditated, was not intended to cause death. Manslaughter is subdivided into deaths caused by an illegal act and those caused by criminal negligence where a legal action was done so carelessly as to cause death. These definitions have been somewhat altered in respect of motor accidents but the principles on which the definitions are based remain.

Infanticide is the unlawful killing of an infant (i.e. less than one year old) by its mother. It was introduced in the

Infanticide Act of 1938 for the humanitarian treatment of women who, by reason of the postnatal condition, might unlawfully cause the death of their child whether by an unlawful act or omission. Prior to 1938, the legal precedent for this special treatment of mothers had been established (see Bowden, 1952). Under the Act, a conviction of murder could be treated as manslaughter. Infanticide has become of less legal significance since the abolition of capital punishment. Deaths from infanticide have become less common since the 1967 Abortion Act (Weatherall, 1976). It is not known whether this is due to the termination of unwanted pregnancies or some other factor such as the change from reporting sudden infant deaths as overlayings.

Deaths from homicide and suicide are uncommon in children (Hollinger, 1979). The relative importance of such deaths to a community is not known because no report has been traced of a long-term study in a community.

In contrast to intended unnatural deaths, accidental deaths are an important cause of death in children aged 1-15 years. Accidents are the commonest cause of death in this age group in developed countries and follow only gastro-intestinal and respiratory infections in the third world (Dyson, 1977). Accidental deaths cover a wide range of conditions including: poisonings, falls, burns, drownings, road-traffic accidents and misadventures during medical care. Road-traffic accidents are the most important cause of death, accounting for about 40% of all unnatural deaths throughout the world (Marcusson and Oehmisch, 1977). There is a considerable excess proportion of male deaths in this category. Between 1951 and 1971 road

traffic fatalities world wide rose by such an extent that they masked the decline in all other accidental deaths (Marcusson and Oehmisch, 1977).

The second most important cause of accidental death is drownings which account for 23% of male and 17% of female fatal accidents world wide (Marcusson and Oehmisch, 1977). Other important causes of accidental death are ingestions, falls and deaths by fire (Jackson and Wilkinson, 1976). These accidental deaths occur principally in otherwise healthy children and it has been shown that they can be prevented (Marcusson and Oehmisch, 1977).

Considering the size of the problem it is surprising that there is a paucity of medical literature on non-fatal childhood accidents (Jackson and Wilkinson, 1976). As Jackson and Wilkinson (1976) state "accidents are as capable of analysis as many other phenomena", and may be predicted and prevented. In other countries, deaths by fire have been reduced to zero, drownings have been reduced significantly and deaths from motor vehicle accidents have also been reduced - all by preventive action (Marcusson and Oehmisch, 1977). The Transport and Road Research Laboratory (TRRL) have done outstanding work in preventing child road accidents and legislation has been introduced in an attempt to limit the risk of deaths by fire. But TRRL figures are based on police reports and the majority of child accidents are not reported to the police (Jackson, 1978). Building design has done little to control the climbing and exploring natures of children. In high-rise flats, especially, there are often tragic consequences (Jackson and Wilkinson, 1976).

Without medical studies of the impact of accidents in a community, legislators and research workers may be unaware of major hazards.

Accidental ingestions have been the subject of more research. Fraser (1980) has reviewed the literature and examined national fatal childhood poisonings between 1935 and 1977. He found that there was a rapid increase in the number of fatalities between 1935 and 1950 followed by a trough then another peak in 1964 and a subsequent decline. However, the numbers of children at risk varied greatly during this time hence the mortality rate may have shown little change. Fraser (1980) showed that drug prescribing habits might influence the types of drug ingested and hence the fatality rate. He found that, after 1970, tricyclic antidepressants replaced salicylates as the most commonly fatal poison. The increasing use of benzodiazepines might also have played a part in reducing fatalities by being a less lethal drug when taken in excess.

The introduction of child-proof packaging in 1976-77 has been claimed to have reduced significantly the number of hospital admissions following drug ingestion (Sibert et al., 1977). However, Fraser (1980) found that fatalities from aspirin and paracetamol (the drugs initially put into safety packs) had fallen to fewer than two per year before 1976. Thus he concludes that the introduction of safety packaging could be expected to have little effect on mortality from these drugs in childhood. Fraser (1980) also suggests that some of the mortality attributed to aspirin ingestion may have been due to the vigorous treatments for these ingestions.

Speizer et al. (1968a, 1968b) showed that there was a close correlation between the use (or abuse) of certain aerosols and mortality from asthma. As they stressed, a temporal correlation is a poor basis for drawing conclusions about cause and effect. However, it would appear that there might have been an increased number of deaths from accidental self-poisoning with these aerosols. For brevity and completeness, the incidence of asthma mortality will be examined under the heading of accidental death in the present study to determine whether the effect seen nationally could have been observed in this population of children.

1.6 Problems with time trend studies

In time-trend studies there are many confounding variables, some of which have been outlined in the preceding sections. It is therefore rarely possible to suggest causal associations. In contrast, such studies might show a negative association as it is unlikely that a change will be due to some measure introduced after that change has occurred. Medical advances or environmental controls introduced after a decline in mortality has begun are unlikely to have had a significant effect in initiating that decline. In the present study such negative associations will be sought.

Changes in the diagnosis or recognition of disease have occurred during the period studied. Prior to 1950, the fibrocystic changes in the pancreas and other organs of the disease now known as cystic fibrosis were thought to be due to congenital syphilis (Emery, personal communication). Thus the dramatic decline in congenital syphilis seen in the early 1950s may be

as much due to the recognition of a new disease as to the effectiveness of penicillin. Reciprocal changes also occurred. In the early 1950s, one Sheffield pathologist noted "cystic fibrosis" on one-third of necropsy reports on children under five including all leukaemias - a fashion which was ignored in the present study unless corroborative evidence was found. Changes in the classification of other diseases such as SIDS or leukaemias have already been discussed.

Although these problems limit the interpretation of time-trend studies, it is important to observe a time-trend before proceeding to analyse other factors. A study of changes in mortality associated with alterations in the class or sex structure of a population could be seriously misinterpreted if a secular trend has not first been identified. In brief, it is essential to observe what happened where and when before proceeding to study how or why and this is the aim of the present study.

1.7 Aims of the present study

The aims of the present study are:

1. To examine changes in child mortality since the inception of the National Health Service.
2. To obtain a representative sample which is of sufficient size for validity but which can be analysed by a single observer.
3. To obtain and examine all original available data relating to all child deaths in this sample.
4. To devise a flexible yet specific coding system applicable to all deaths which will overcome problems

inherent in archived records due to changes in coding systems.

5. To compare the certified causes of death with those given on necropsy reports.

6. To examine the validity of death certification as a measure and record of child mortality.

7. To examine changing patterns of fatal childhood disease with time.

8. To place the observed trends in mortality into an historical perspective.

9. To examine previous explanations for declines in childhood mortality to determine whether these explanations are compatible with the data from the present study.

SECTION 2

MATERIALS AND METHODS

2.1 Sources of data

Data were collected from three principal sources: death certificates, necropsy reports and the reports of the Sheffield Medical Officer of Health (MOH). For the major part of the study the sample or index population was defined as children aged less than 15 completed years who died between January 1st 1947 and December 31st 1979, and who had a permanent residence within the Sheffield city boundary at the time of death. Additionally, Sheffield child deaths between 1885 and 1946 have been examined.

2.2 Identification of the index population - the death certificate search

An application was made to the Registrar General for permission to view the original death certificate archives relating to every death in Sheffield between January 1st 1947 and December 31st 1979. A personal dispensation was granted on condition that the archives were viewed personally, in the presence of a member of the Register Office Staff, at their convenience, and that no particulars of any individual would be made known.

Between November 1979 and July 1980 every death certificate issued in Sheffield after December 1946 (approximately 250,000 certificates) was examined. Details were copied from the certificate of any person with a stated age of 15 years or less into a ledger of Sheffield child deaths.

A copy of the death certificate of any Sheffield child who dies outside the city is obtained by the MOH. These deaths average two or three a year, usually occurring while on holiday.

The home address given on each child's certificate was checked against the Official Index of Sheffield Streets which was in use at the time of death. Because of boundary changes this Index was revised in October 1946, March 1953, March 1968 and March 1974. Every child whose address was in the appropriate Index was included in the study.

Throughout the period studied, deaths in Sheffield were registered on the standard certificates issued by the Registrar General. These certificates conform to the model recommended by the World Health Organisation (WHO, 1977). In April 1969 the forms were changed slightly and the dates of birth and death recorded instead of the age and date of death.

Other details recorded on each child death were the name, age, sex, place of death, father's occupation and the cause(s) of death. These details were all transcribed into a ledger of Sheffield child deaths.

2.3 Validating the certified cause of death - the post-mortem search

Permission was obtained from all the pathologists in the city who perform necropsies on children to search their records. Details of these necropsies were recorded in a separate ledger from the death certificate copies and cross-referencing numbers attached.

The majority of the necropsies were performed at the Children's Hospital (SCH) where there has been one Consultant Paediatric Pathologist since 1947. At the Jessop Hospital for Women (JHW) there have been two pathologists dealing with

obstetric and perinatal pathology. The pathologist at the Northern General Hospital (NGH) covers all aspects of pathology. There was one pathologist at the NGH between 1947 and 1977 and his successor worked alone until early 1979. In the department of Forensic Pathology there have been two pathologists who conferred with the Children's Hospital pathologist on many paediatric cases. Other hospitals in the city performed necropsies on children at the request of the Coroner in deaths following trauma or surgery.

The detail of post-mortem examination varied. In most laboratories a routine necropsy was performed to verify a diagnosis or to explain why treatment was unsuccessful. These necropsies were done by busy pathologists with many adult necropsies to do each day in addition to surgical biopsies and other routine work. At the SCH detailed necopsies were always performed in which all tissues were examined methodically and all organs were routinely sampled in a standardised manner for histology and a minimum of 51 histological blocks taken from specified sites at each post mortem (see Appendix I). These blocks are stored and were re-examined in doubtful cases. Full post-mortem details were found in all of the Coroner's and almost all of the SCH cases. For some of the earlier years, only summaries of the NGH and JHW necropsies were available.

2.4 Digital coding of the data

All of the information gathered from the death certificates and necropsy reports was converted to digital form to facilitate computer analysis. The digital coding was done via coding sheets which were designed for this study (see Appendix II).

The components of this coding sheet were:

1. Serial Number: A unique identification number for each child.

2. Address: The home address given on the death certificate coded into electoral ward, with the aid of the Index of Sheffield Streets current for the time of death.

3. Sex: Male or female.

4. Place of death: The place of death given on the death certificate was coded into hospital, home or outside. Hospital deaths were further coded into the specific hospital.

5. Date of birth: After April 1969 the date of birth was given on the death certificate. Prior to this, death certificates recorded the age and date of death. The year of birth for these pre-1969 deaths was calculated by subtraction of the age from the date of death. For children under 1 year, this was accurate to the month of birth. For children over 1 year, the year of birth was estimated by the following method: if the death occurred after June 30th the year of birth was assumed to be: (year of death - age in years); if the death occurred before June 30th the year of birth was assumed to be: (year of death - {age in years + 1}). The results correlated well with available information on necropsy reports.

6. Date of death: This was given on all certificates.

7. Age: Prior to April 1969, all death certificates gave the age at death in completed days, weeks or years. For deaths after April 1969 the age was calculated by subtraction of the date of birth from the date of death.

8. Father's occupation: The stated parental occupation was coded into the Registrar General's Social Classes, the

World Health Organisation's Socio-economic group (SEG) and the International Labour Organisation's (ILO) Classification of Occupations (Registrar General, 1960). For consistency, one revision of these classifications was used although slight variations occurred between censuses. The revision nearest to the middle of the study (the 1960 revision) was used. In addition to the classifications given in the 1960 Classification of Occupations, unemployed fathers were allocated to an extra SEG; unmarried mothers were allocated to either the SEG of their male counterpart, or, if unemployed, to the extra SEG; unmarried working mothers were also placed in an extra social class (Class VI) and unmarried unemployed mothers into social class VII. Manual and non-manual workers in Class III were split into two sub-groups.

9. Indexed cause of death: The cause of death given on the death certificate was recorded. The method of assessing and coding will be described in item 13.

10. Date of death registration: Official statistics in Britain are analysed by the date of registration. The date of registration given in the Registrar's archives was coded to allow future comparison with national statistics.

11. Sudden or expected death: The deaths were coded into Coroner's cases, non-Coroner's sudden deaths and expected deaths. The Coroner's cases were coded according to verdict into natural, open, accidental, manslaughter or murder.

Sudden unexpected death in infancy (SIDS) was defined for this study as a death which was unexpected by history and in which a thorough necropsy failed to demonstrate an adequate cause

of death. A thorough necropsy would always include an examination of all organs and body fluids both macroscopically and microscopically (see Appendix I) together with a study of police statements or other history. An adequate cause of death has been defined, following discussions with senior paediatric and pathology colleagues, as any morbid process which was of sufficient severity as to preclude survival. Throughout the present study, the author determined such cases alone while ignorant of the year of death.

12. Post-mortem: This recorded the place where a post-mortem was performed - usually the hospital where the child died or the medico-legal centre. However, there were a number of cases where the body was removed to another centre for more detailed examination. Hence the numbers of post-mortems in a hospital could be greater than the number of deaths there.

13. Digital classification of the cause of death: A simplified classification of diseases was devised based on the Ninth Revision of the International Classification of Diseases 'ICD9' (WHO, 1977) but expanded where necessary to allow greater specificity. The logical format of this new three-digit classification was: the first digit gave the major disease category (1 = Epinatal Disease, 2 = Congenital Malformation etc.), the second digit gave the subdivisions of each category by organ or system (22 = Congenital heart disease) and the third digit gave the specific disease (222 = Transposition of the great vessels). The classification is given in Appendix III. The uniqueness of the classification ensured that the confident-iality of the records was maintained.

Each death certificate was examined individually without knowledge of the year of death by using an assistant who sat behind the author reading out the certificates. The author decided on the cause of death by the method described below and personally coded each disease using the classification devised for this study. Each necropsy report was then analysed in the same manner.

Having independently coded each death certificate and necropsy report all of the data for each child were brought together and the underlying cause of death determined with the author still ignorant of the year of death.

14. The underlying cause of death: The death certificate is divided into two parts. Part I deals with the causes leading directly to death and is in three sections, where section (a) the direct cause of death is due to (b) the antecedent cause which is due to (c) the underlying cause of death. Part II of the certificate deals with other conditions contributing to death but not related to the disease causing it.

The international rules for arriving at the underlying cause of death given in the ICD9 (WHO, 1977) were followed whenever possible. The general rule is that the underlying, indexed cause of death is the entry on the lowest line on the Part I i.e. (c) takes precedence over (b) which takes precedence over (a). During the archive search it became apparent that these rules had been ignored or misunderstood in approximately one-third of the cases. These certificates were corrected before coding, e.g. 'Prematurity due to hyaline membrane disease' was coded in the reverse order.

15. The validity of the certified cause of death - agreement between death certificate and necropsy report: The degree of agreement between the cause of death on the death certificate and necropsy report was categorised by the following criteria:

(a) If, regardless of word order, the underlying cause of death on the necropsy report was found in Part I of the death certificate then the certified cause of death was considered valid. For example, the death certificate 'Spina bifida due to meningitis due to hydrocephalus' was considered to agree with the necropsy report 'Neurospinal dysraphism (L2-5) with hydrocephalus secondary to Chiari deformity and E. coli meningitis'.

(b) The certified cause of death was considered invalid where major discrepancies were found. For example the death certificate 'Respiratory failure due to renal tuberculosis' was considered to disagree with the necropsy report 'Disseminated neuroblastoma, no acid-fast bacilli seen in any section'.

(c) Where the underlying cause of death on the necropsy report could not be found in Part I of the death certificate but was in Part II, then 'incorrect word order' was recorded. For example, one child's death certificate read: I(a) Cardiopulmonary failure (b) pneumonia; II cerebral palsy, whereas the necropsy report was 'Severe perinatal hypoxia, spastic quadriplegia, cerebral softening with scarring, pressure sores, hypostatic pneumonia (pneumonococcal)'.

(d) Where a necropsy report could not be found, even if the certificate suggested that a necropsy might have been done (e.g. 'cerebral embolism due to pulmonary atresia'), then 'don't know' was ascribed.

It was not assumed that the necropsy report was always correct. For example, a child who died aged 12 hours was certified 'Idiopathic respiratory distress syndrome due to prematurity'. The necropsy report read 'Congenital heart disease - patent ductus arteriosus'. The weight at death was 1800 g. As in a normal child the ductus may not have closed by 12 hours it appears that the clinician was correct. In such cases the cause of death in the certificate was entered on the coding sheet. The object of this part of the study was to validate the accuracy of the stated cause of death on death certificates therefore these deaths were not entered as disagreements because the death certificate did not require modification.

16. Summary of the death: Having examined the information on each death an overall summary was made. Deaths were categorised into those which were considered to be inevitable; those for which treatment was available; and those in which insufficient cause was found to explain the death adequately. The 'inevitable' deaths were subdivided into deaths among severely handicapped children, and other conditions for which no curative treatment was available at the time of death.

2.5 Reproducibility of the Coding

The reproducibility of the coding process was tested by recoding a systematic 2% random sample two months after the coding had been completed. The 2% sample was generated by re-examining every fiftieth death after choosing a death in the first month of 1947 by random number. Entry to the total series of deaths was dependent only on the date of death,

therefore this was a systematic random sample. After recoding the 2% sample, the original coding sheets relating to these deaths were checked manually against the punch cards as a check for transcription errors by the typist.

2.6 Computer handling of the data

The completed coding sheets were transferred to standard Fortran punched cards at the University of Sheffield Computing Centre, using IBM 029 punch typewriters (International Business Machines Ltd.). After punching, each card was verified on an IBM 059 verifier and any errors corrected by the typists.

The corrected cards were transferred to computer files on the University of Sheffield's ICL 1906S computer via an ICL 2101/2 card reader (International Computers Ltd.). The 1906S computer files were then transferred internally to the University of Sheffield's Prime 750 computer (Prime Computers Inc.). All the computer procedures were performed by the author alone on the Prime 750 computer using the Statistical Package for Social Scientists, Version M, Release 7.2, December 1977 (SPSS, 1975; Prime, 1979).

2.6.1 Errors in the computer file

Before analysing the data, the computer files were checked to ensure accuracy. Each of the cards was checked manually to ensure that it contained no more than and no less than 62 digits. Each variable was checked with the Prime Computer Editor to ensure that values lay within the ranges studied (e.g. the years studied were 1947 to 1979, but some deaths had been coded in 1906, 1995 and 1999). Missing or incorrect values were checked against the original ledgers and corrected.

2.7 Assessing the completeness and accuracy of death registration

To assess whether all Sheffield child deaths had been registered, 1500 consecutive necropsies (done between April 1961 and April 1966) in the SCH files were examined and the reports on Sheffield children matched against the death certificate ledger.

To assess the accuracy of the information given on death certificates, the personal details (age, sex, address etc.) given on the necropsy reports were checked against the same details on the corresponding death certificate.

2.8 Identification of the population at risk

The numbers of children born in each year varied during the period studied. The numbers of deaths were standardised to mortality rates by dividing by the number of children alive in each group. In the neonatal age group the risk population was the number of live births. The numbers of live-born Sheffield children are totalled weekly by the Sheffield Area Health Authority (Information Services) from midwives' returns and cross-checked against the returns of birth registrations from the Registrar of Births, Deaths and Marriages. The home address of each baby is coded into electoral wards with the aid of the current Index of Sheffield Streets. All children whose addresses are contained in the appropriate Index are included in the annual total of Sheffield live births.

The number of children at risk of dying in a specific age group after the first month was estimated by the following

formula (assuming no net migration):-

Number aged A to B years at risk in year Z = Sum of the survivors to A years in birth cohorts (Z - {B + 1}) to (Z - A)

e.g. the number of children aged 5 to 9 years alive in 1960 is the sum of live births between 1950 (1960 - {9 + 1}) and 1955 (1960 - 5) less the sum of deaths under 5 years in each birth cohort from 1950 to 1955.

This calculation was used for each quinquennial age group (1-4, 5-9, 10-14 years) for each of the 33 years in the study. The assumption concerning net migration was checked against intercensal projections, using the censuses of 1931, 1951, 1961, 1966 and 1971. No census was taken in 1941 because of hostilities.

The data were analysed in the age bands recommended by the World Health Organisation (WHO, 1977). These age bands are < 1 year, 1-4 years, 5-9 years and 10-14 years. To obtain samples of reasonable size the 5-9 and 10-14 year age groups were added together. Deaths under 1 year were subdivided into neonatal and postneonatal. Neonatal deaths were further subdivided into deaths under 1 week for some studies.

Because of the different age distribution of certain causes of death, analyses of overall mortality rates by cause are not readily interpreted. Therefore the major pathological groups were examined for those age groups where the majority of deaths occurred. Deaths from epinatal diseases under one month of age were examined by year of birth as a rate of the total live births. Deaths from malformations under two years of

age were analysed by year of birth per thousand live births. Deaths from infectious diseases and cot deaths among children aged one month to two years were examined by year of death per thousand children alive in the age group. Deaths from trauma and tumours among children aged more than two years were examined by year of death per thousand children alive in this age group.

2.8.1 Congenital abnormality births

A register of Sheffield children born with congenital abnormalities has been kept by the MOH since January 1963. A copy of this register was used to calculate case-fatality rates for certain congenital malformations.

2.9 First week deaths from prematurity in Sheffield's Maternity Hospitals

Children with the diseases of prematurity were not transferred for intensive care before late 1979. First week deaths from prematurity were analysed by maternity hospital. The numbers of live births in each maternity hospital were obtained from the records of the Sheffield MOH for the period 1947-1973 and from the Sheffield Area Health Authority (Information Services) for subsequent years.

The facilities for neonatal care are not the same in the three maternity hospitals. The Jessop Hospital for Women (JHW) has always had the best facilities in the city and relatively high staffing levels. The Nether Edge Hospital (NEH) was well equipped when modernised in 1969. The Northern General Hospital (NGH) has the least equipped special care baby unit (SCBU) and the most old fashioned equipment. Paediatric

staff at the NGH cover the general paediatric wards in the NGH as well as the SCBUs in NGH and NEH. The JHW staff work full time with neonates. Staffing in these hospitals has gradually increased since 1947 and in 1979 the staffing levels were:-

NGH: 2 consultants (1 with neonatal interest)
1 senior registrar, 1 registrar, 3 senior house officers;

NEH: the same consultants, senior registrar and registrar as NGH with 1 senior house officer on rotation from NGH;

JHW: 1 consultant neonatologist, 2 paediatric consultants with neonatal interest, 1 neonatal registrar, 4 neonatal senior house officers and additional cover provided by 3 lecturers/senior registrars.

2.10 Neonatal and postneonatal mortality in large English towns

The neonatal and postneonatal mortality rates in Sheffield between 1947 and 1978 were compared with the corresponding rates in other large English towns by examining the tables published by the Registrar General (1947-1973) and the Office of Populations, Censuses and Surveys (OPCS Series VS, 1974-78). Towns were chosen where the total population changed by less than 1% following the local government reorganisation in 1974. These towns were identified by comparing the estimated mid-year populations in 1973 (Registrar General's Annual Review 1973) with the mid-year population in 1974 (OPCS, local authority vital statistics). The large towns identified were: Bristol, Coventry, Derby, Hull, Plymouth, Southampton.

2.11 Comparison of Sheffield child mortality with national studies

The Sheffield mortality rates were compared with national rates for corresponding years given in the statistical reports

of the Registrar General (Registrar General, 1947-1974; OPCS, 1974-1978). The causes of early neonatal death were examined nationally twice during the period studied: in the 1958 and 1970 British Births Surveys (Butler and Bonham, 1963; Royal College of Obstetricians and Gynaecologists, 1975). The causes of death of the Sheffield children born in these cohorts were compared with the national cohorts to determine to what extent the Sheffield child population was representative of the national population.

2.12 Historical trends in Sheffield child mortality 1885-1979

To place the results of the main (1947-79) study in perspective, certain details of Sheffield child mortality prior to 1947 were obtained from the annual reports of the MOH (Sheffield 1874-1973). Details were also obtained from these reports of public health measures introduced in Sheffield.

SECTION 3

RESULTS

3.1 The sample population

A total of 7049 Sheffield children died between January 1947 and December 1979. Because of a fire at the Sheffield Register Office on 21st February 1947, some certificates were rendered unreadable. Certain details, including the cause(s) of death, were omitted from the replacements provided by the General Register Office. In some cases details were available from necropsy reports but there remain 14 children for whom the cause of death is unknown. The distribution of the remaining deaths by age and cause is shown in Table I.

Necropsy reports were traced by examining the relevant mortuary ledgers for children with names corresponding to the names given on the death certificates in the sample population. The surnames of both parents may be given on the certificates of children of Muslim parents and of illegitimate children. Where they were given, both names were sought. If only one name was given and the child was known to the mortuary by another name it is possible that a necropsy had been performed but this could not be linked with the child. Therefore the reported necropsy rate may be lower than the actual rate.

Necropsy reports were found for 4280 of the 7049 deaths. The distribution of necropsies by age and cause of death is shown in Table II. Copies of all necropsy reports for the Coroner were traced. Almost every necropsy report at the Sheffield Children's Hospital (SCH) was also traced. Because of shortage of storage space, at the Jessop Hospital for Women (JHW) and at the Northern General Hospital (NGH) reports more

than 15 years old are destroyed. Summaries of these reports were found but in some of these summaries histological details were missing. The distribution of the necropsies by age and hospital is shown in Table III.

3.2 Validation of the data

3.2.1 Reproducibility of the coding

The systematic 2% random sample of the coding sheets comprised 140 children. Recoding this sample involved 8680 digits in total. Comparison of the recoded sheets with the originals identified major errors in two cases: the age in one had been coded as 4 instead of 5 months and the date of death in another as the 20th instead of the 25th. The only other differences revealed by the recoding were 28 cases with minor variations in the coded cause of death (e.g. 'leukaemia not specified' instead of 'acute lymphoblastic leukaemia'). These variations were not corrected as only 140 of the 7049 cases were re-examined. The coding procedure therefore had a confidence limit of 0.02% (2 major errors in 8680 digits).

3.2.2 Coding errors

Manual examination of the 7049 coded sheets revealed 186 missing values where the code for 'don't know' had been omitted. These were corrected. Examination of the computer files using the Prime editor found 38 variables which had been given nonsensical values. They were all elementary coding errors e.g. the age of a child 14 months old had been coded as '14 months 00 years' instead of '02 months 01 year'. These errors were also corrected.

3.2.3 Errors in punch-card typing

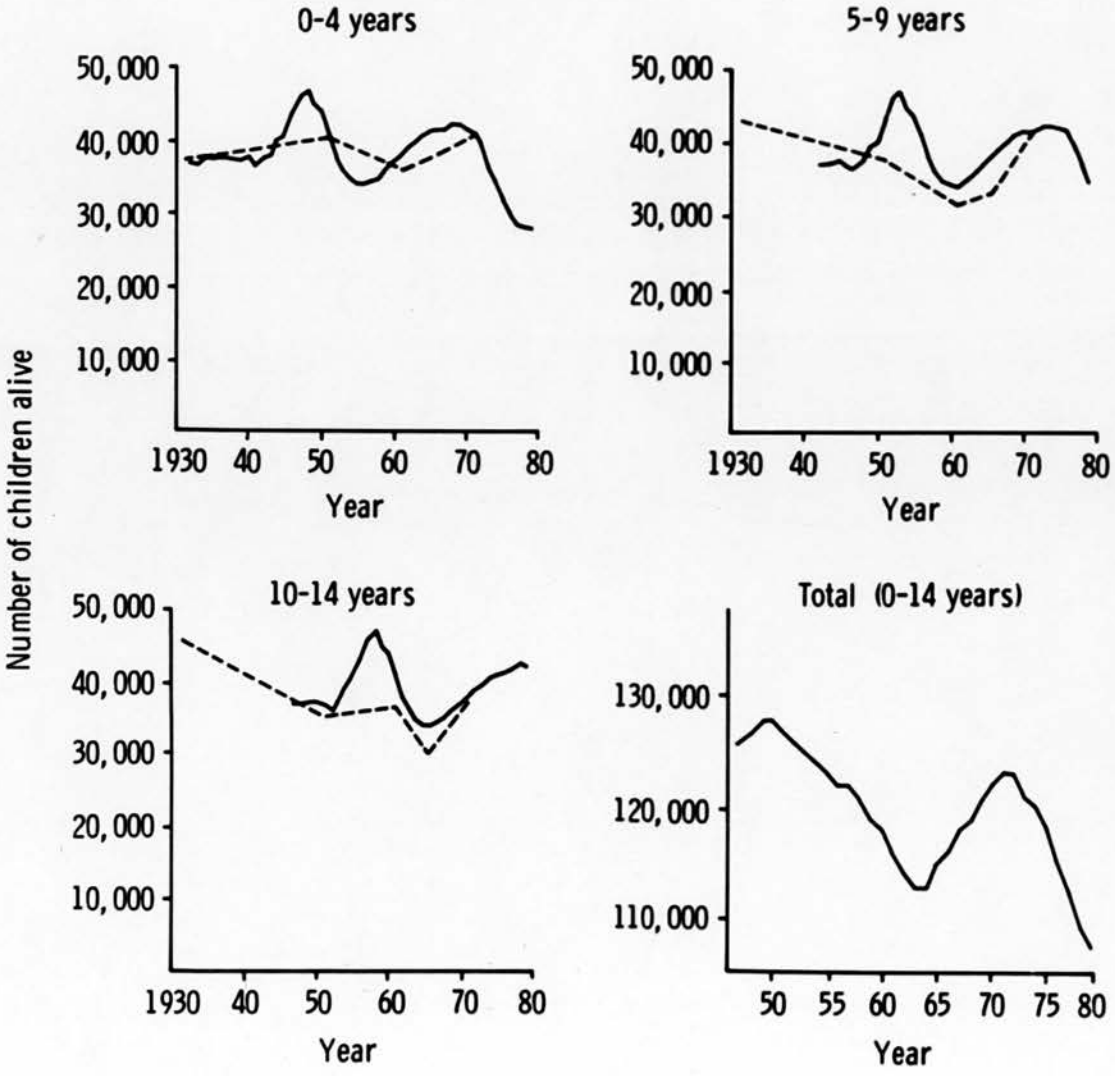
Manual examination of the 7049 punched cards revealed 7 with more than 62 columns of data. These were due to duplication of digits and caused all subsequent variables on the card to be meaningless. The Prime Editor identified another 10 variables with nonsensical values that resulted from typing errors (e.g. deaths coded in 1906, 1995, and 1999). These 17 errors were found despite the verification process which should guarantee no variation between the punched card and coding sheet. An additional 37 cases were found where typing errors had resulted from ambiguous or illegible writing. All of these errors were corrected. Examination of every digit on the punched cards for the 140 deaths in the 2% sample uncovered no errors.

3.2.4 The population at risk

The estimated numbers of children alive in each age group for each year are shown in Appendix IV. It can be seen from Fig. 4 that these estimated numbers approximated the census numbers but diverged greatly from the intercensal projections. There was some divergence between the estimated and census numbers of 5-9 year olds in 1951 and the 10-14 year olds in 1961. Both include the cohort of children who were born just after the second world war. It appears that there may have been some net emigration from the city in this period, probably to the dormitory suburbs which were built just outside the city boundaries after the last war.

Accurate figures of Sheffield births and deaths are available for the years 1851, 1861, 1871, 1873 and annually

ESTIMATED NUMBERS OF SHEFFIELD CHILDREN ALIVE IN QUINQUENNIAL GROUPS AND NUMBERS ENUMERATED IN THE NATIONAL CENSUSES



after 1885 (Sheffield, 1885-1979). The Sheffield birth rate from 1850 to 1979 is shown in Fig. 5.

3.3 Death certification as a measure of child mortality

3.3.1 Assessing the completeness of death certification

Among the 1500 necropsies performed at SCH between April 1961 and April 1966, there were 180 stillbirths, 557 children with an address outside Sheffield, 373 children for whom no address was given and 390 Sheffield children. The details of the 763 children in the last two groups were compared with the ledger of Sheffield child deaths and matching certificates found for 430 of them. Among the 390 children with a Sheffield address on the necropsy report there were 38 for whom a death certificate could not be found. A careful search through the archives of the Register Office uncovered certificates for 30 children with the same name, sex, age, date and cause of death, but with an address outside Sheffield on the death certificate. One illegitimate child's certificate was matched because both parents' names had been given. Four children died in the first few days and may have been registered as stillbirths. The remaining 3 children with unmatched necropsy reports were aged 5, 8 and 9 months.

3.3.2 Comparison of personal details on death certificate and necropsy record

Whenever a necropsy report contained an age or date of birth, this was checked against the stated age on the corresponding death certificate. It was found that many arithmetical errors were present in the necropsy reports (e.g. a child born on 30th December 1972 who died on 3rd January 1975 was given

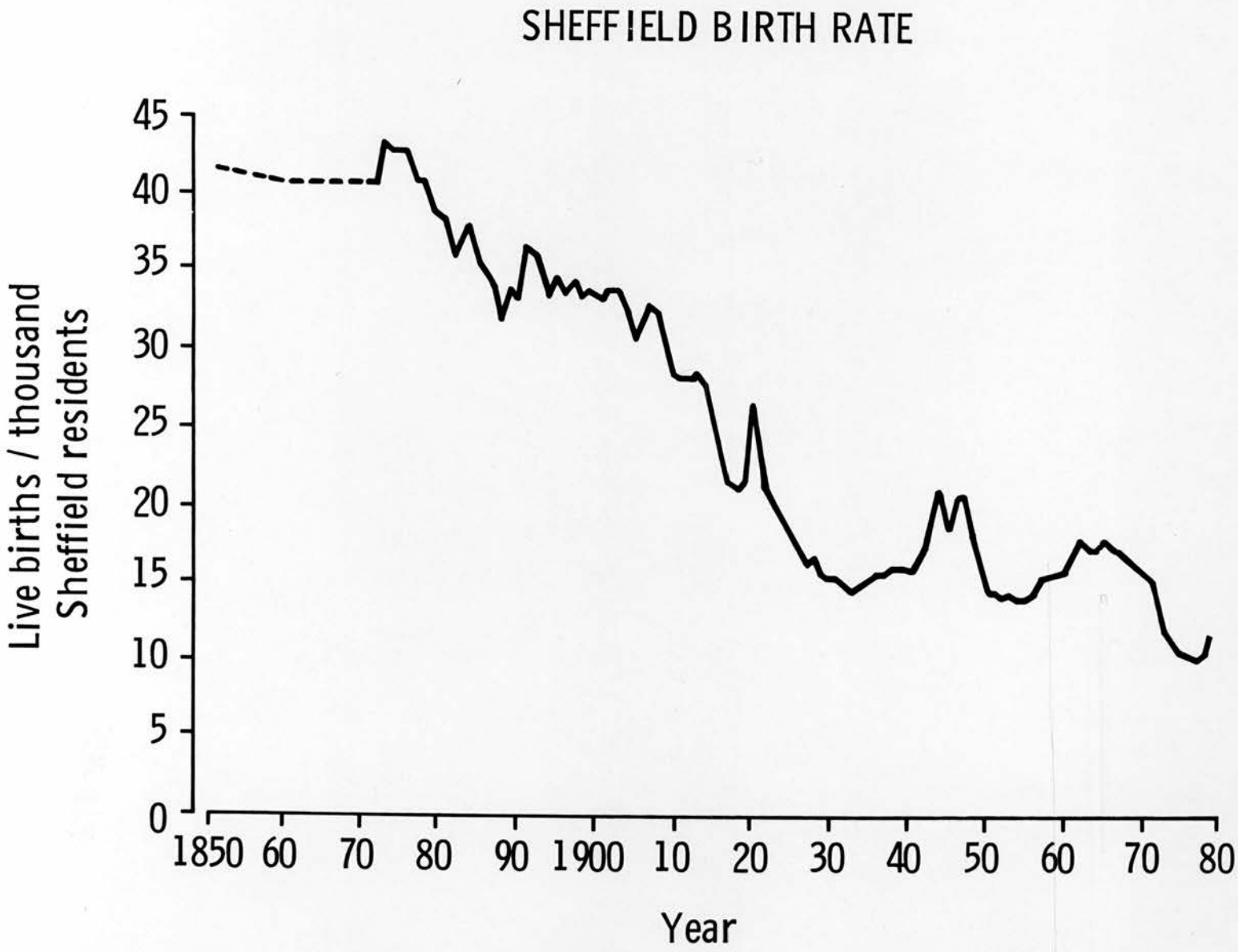

SHEFFIELD BIRTH RATE
Live births / thousand
Sheffield residents
45
40
35
30
25
20
15
10
5
0
1850 60 70 80 90 1900 10 20 30 40 50 60 70 80
Year

Fig. 5

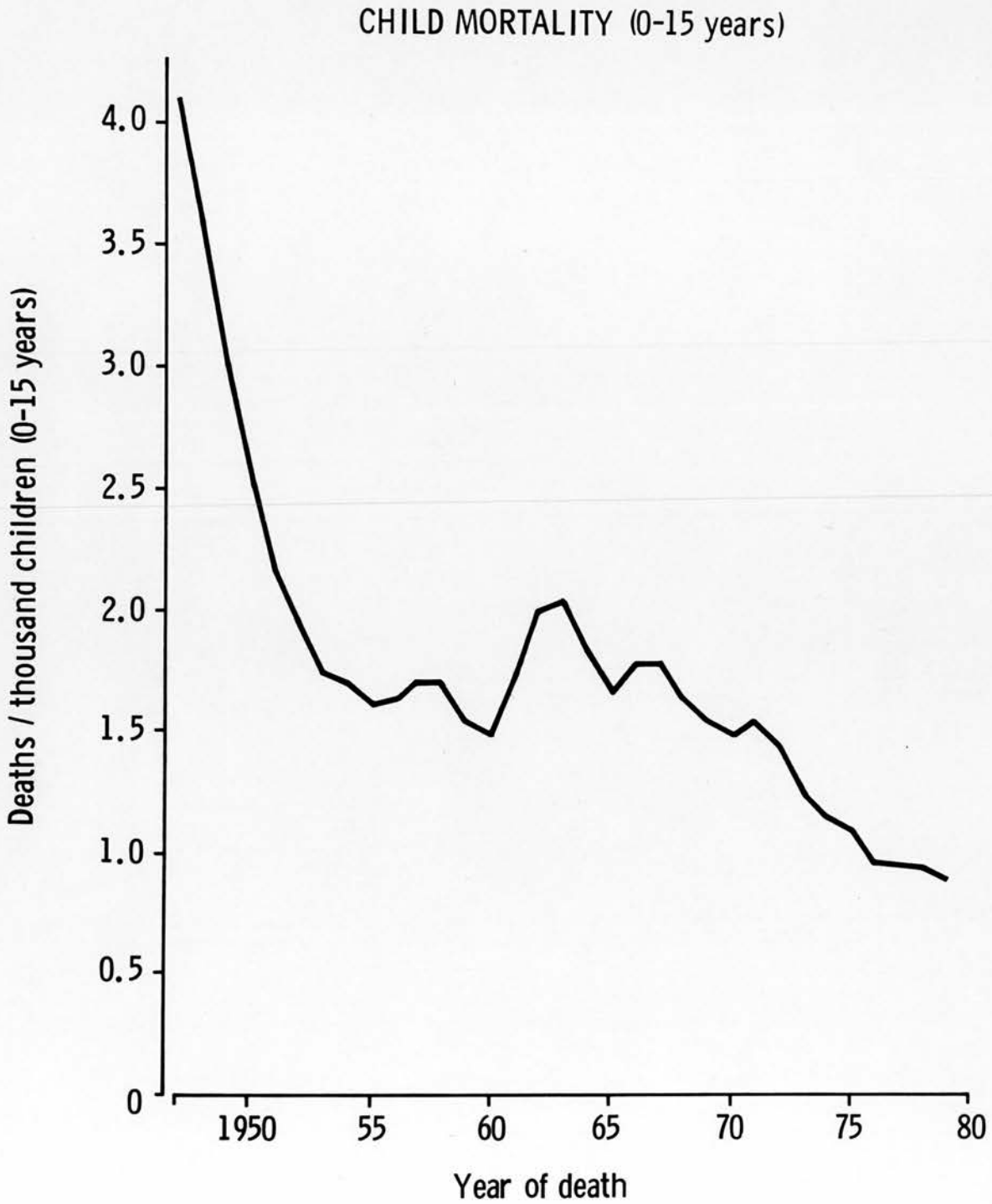
the age of two completed years by the Registrar, but three completed years by the pathologist). There were many variations in spelling of names and occasionally different addresses. There were also nonsensical errors (e.g. a child apparently born on 10.11.68 who died on 7.5.67 aged 5 months). It was found in all cases of disagreement that the Registrar's facts were the more credible.

3.3.3 Validity of the certified cause of death

Histological reports were found in 4007 of the 4280 reports. The accuracy of the certified cause of death was determined by comparison with these histologically verified reports. The distribution of the certified cause of death by agreement is shown in Table IV. The distribution of accuracy by age is shown in Table V and by social class in Table VI. Social class was not entered for 46 of these children. Analysis by specific parental occupation found a strikingly uniform proportion of inaccurate certificates, ranging from 7.6% in children of unskilled workers to 10.0% in children of service workers. Among doctor's children, 8.9% of the certified causes of death were found to be inaccurate. Of the major disagreements, 85% were found at SCH where detailed necropsies with exhaustive histology (Appendix I) were always done.

3.4 Mortality trends I - Death by age

The overall child mortality rate fell from over 4 to less than 1 death per thousand children aged 0-15 between 1947 and 1979 (Fig. 6). To analyse this fall further the deaths have been standardised by age and by cause. Standardisation by age gives a high degree of consistency allowing comparison with


CHILD MORTALITY (0-15 years)
Deaths / thousand children (0-15 years)
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0
1950
55
60
65
70
75
80
Year of death

other studies but poor specificity. Standardisation by disease gives great specificity allowing meaningful study of associated factors but, because of the variability of diagnoses, may give poor consistency for international or historical comparisons. The age at death has not been standardised for gestation. The proportionate distribution by age of the 7049 deaths is summarised for the 33 years in three sector diagrams, each representing an 11-year period (Fig. 7). Each diameter is proportional to the overall child mortality rate in that 11-year period.

Age-specific mortality rates for both England and Wales (E & W) and Sheffield are shown in Figs. 8 and 9. The rate of change of these age-specific Sheffield rates is shown in Figs. 10 and 11.

3.4.1 Neonatal mortality

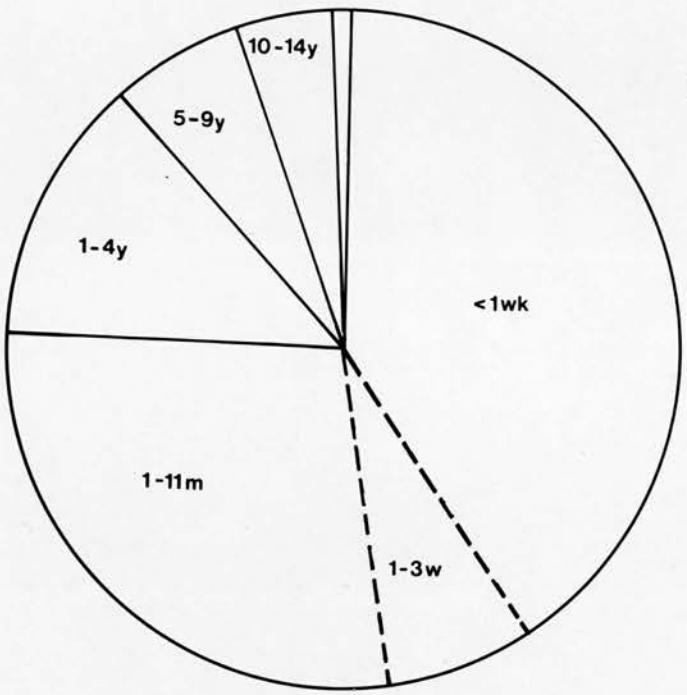
There were 3507 deaths under one month of age. The proportionate distribution of these deaths by cause is shown in Fig. 12. The subgroup 'Prematurity not otherwise specified' includes all those children where the certified cause of death was prematurity and no weight or histology was available. All 43 children who died with intra-ventricular haemorrhages are included in the Idiopathic Respiratory Distress Syndrome (IRDS) group, because they all had hyaline membrane formation in the lung and it was not known whether all brains in these IRDS deaths had been examined.

3.4.1(a) Changing pattern of fatal neonatal pathology

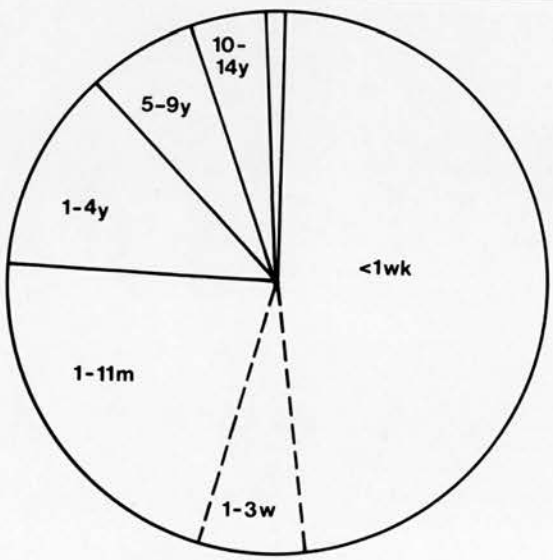
Nearly all of the 3507 neonatal deaths fell into three pathological groups: malformations, prematurity or 'obstetric' (Fig. 12). The changing pattern of each pathological group's contribution to the neonatal mortality

PROPORTIONATE DISTRIBUTION OF DEATHS BY AGE

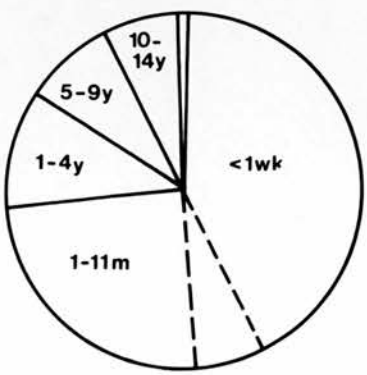
○ = 1 death / 10, 000 children



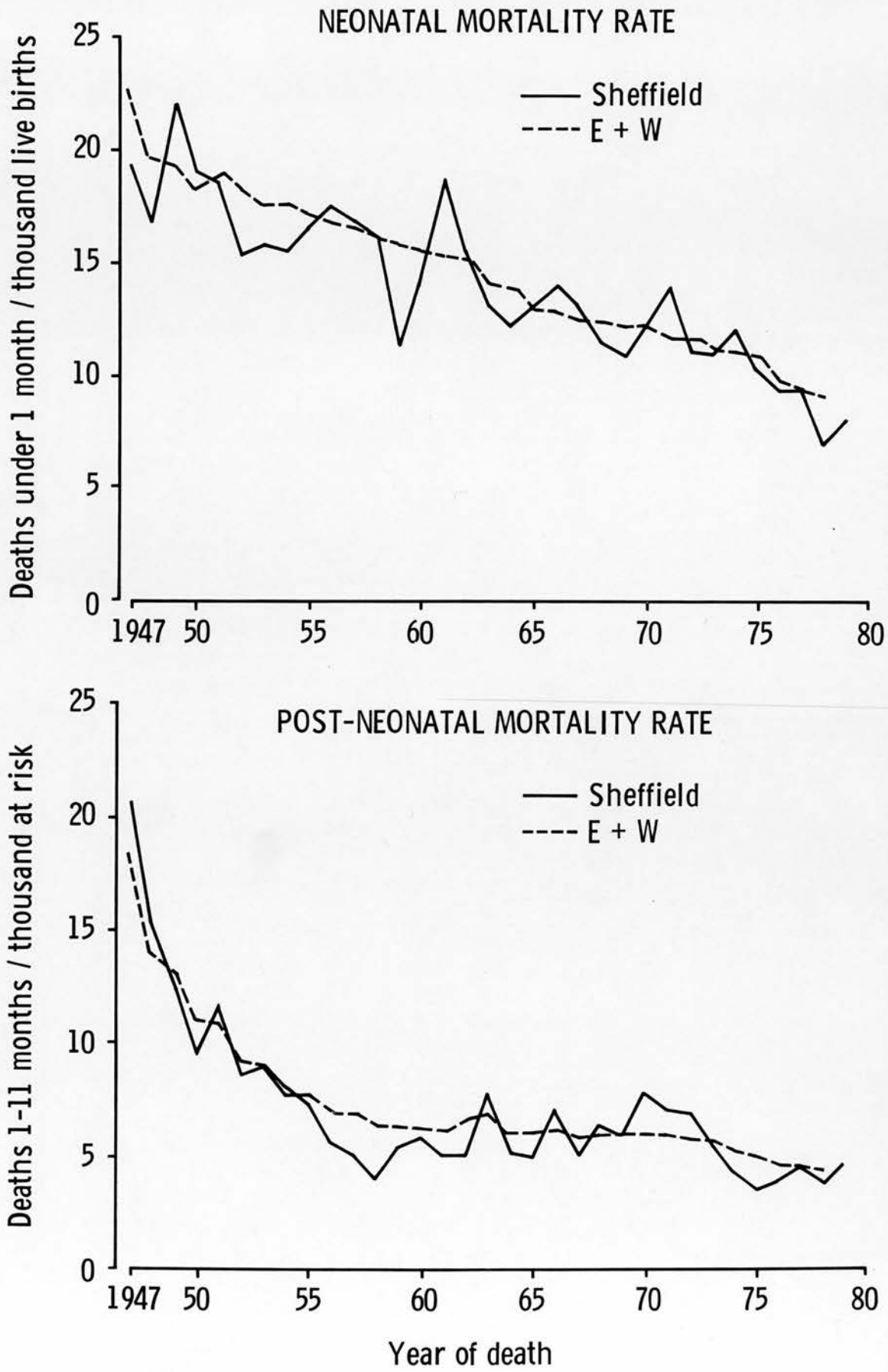
1947-57



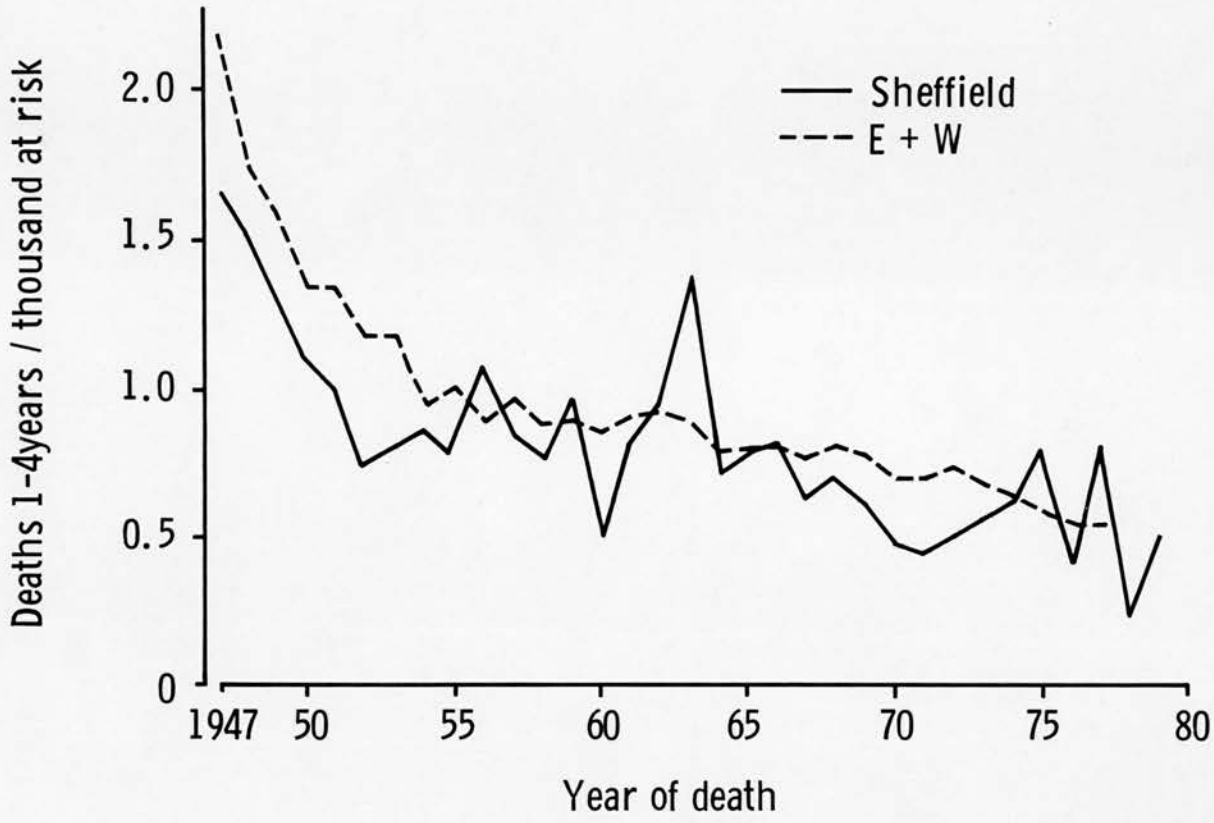
1958-68



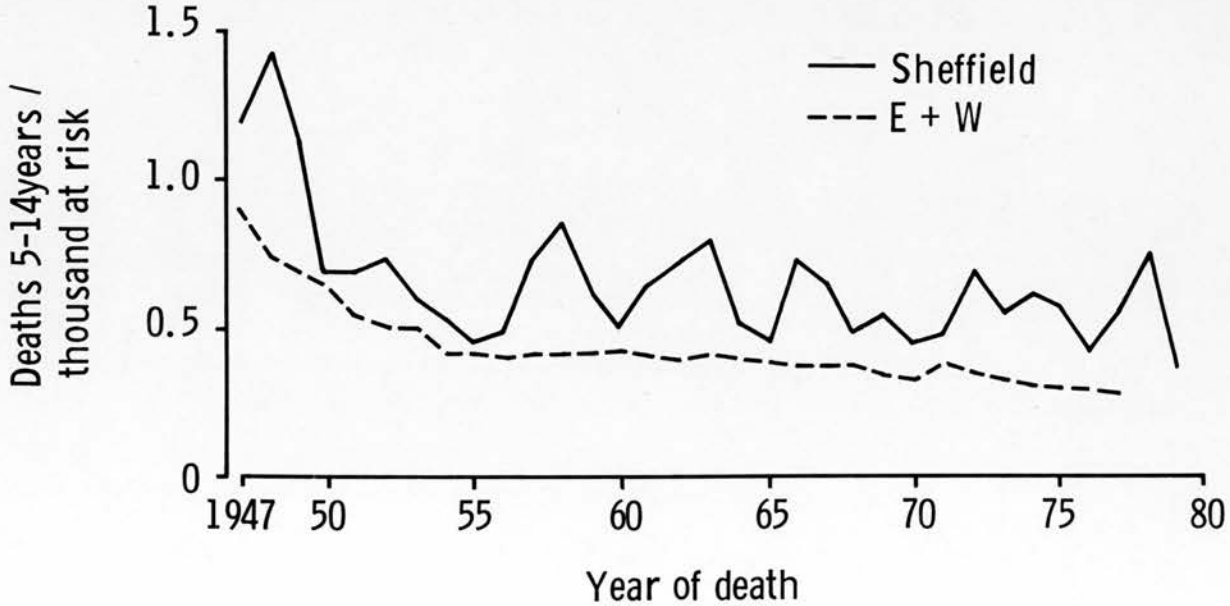
1969-79

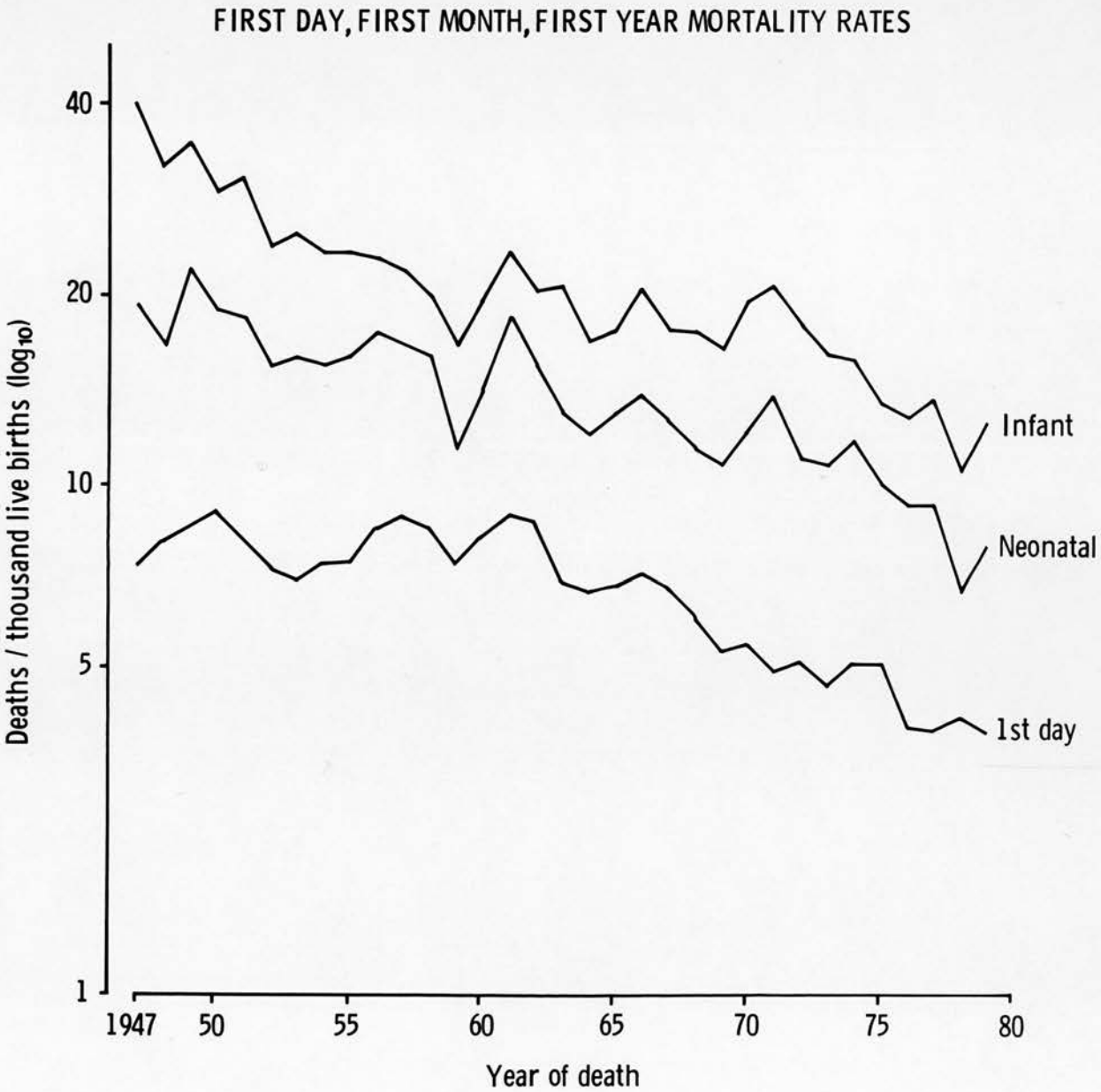

NEONATAL MORTALITY RATE
Sheffield
E + W
Deaths under 1 month / thousand live births
25
20
15
10
5
0
1947
50
55
60
65
70
75
80
POST-NEONATAL MORTALITY RATE
Sheffield
E + W
Deaths 1-11 months / thousand at risk
25
20
15
10
5
0
1947
50
55
60
65
70
75
80
Year of death

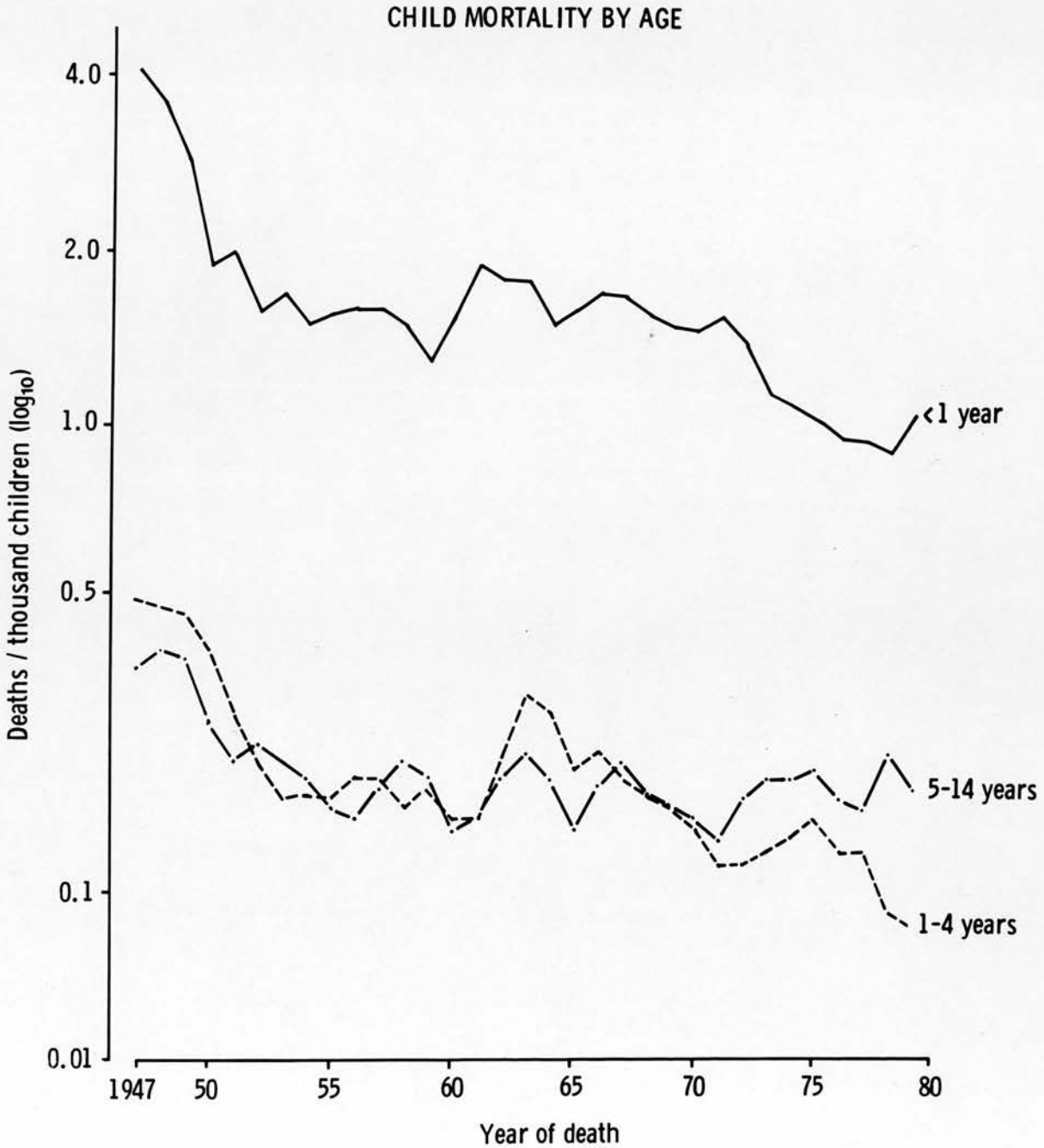
PRE-SCHOOL MORTALITY RATE



SCHOOL CHILD MORTALITY RATE




FIRST DAY, FIRST MONTH, FIRST YEAR MORTALITY RATES
Deaths / thousand live births (log_{10})
40
20
10
5
1
1947
50
55
60
65
70
75
80
Year of death
Infant
Neonatal
1st day


CHILD MORTALITY BY AGE
Deaths / thousand children (log_{10})
4.0
2.0
1.0
0.5
0.1
0.01
<1 year
5-14 years
1-4 years
1947
50
55
60
65
70
75
80
Year of death

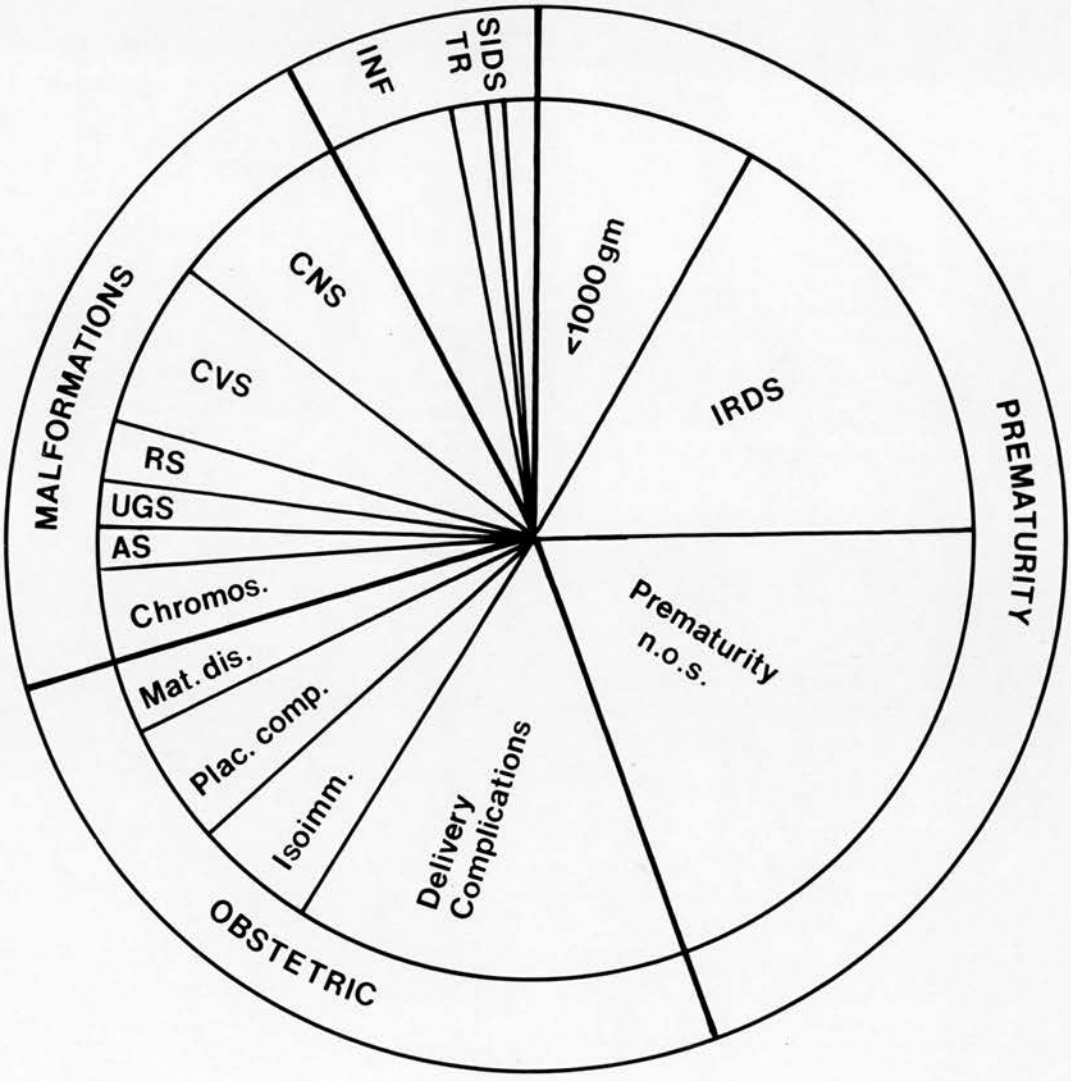
rate is shown in Fig. 13. The use of the word 'obstetric' to describe a group of pathologies does not reflect upon the work of obstetricians or midwives but is used as a generic term for those diseases particularly associated with the work of an obstetric department. Similarly the term 'birth trauma' may include iatrogenic trauma, but encompasses that group of traumatic conditions which are associated with birth (see Appendix III).

In Fig. 13 it will be seen that the prematurity mortality rate has declined steadily. The dips in this rate which occurred in 1952-1954 and 1958-1960 are associated in time with an approximately equal rise in the 'obstetric' mortality rate. To examine for a possible diagnostic fashion the obstetric rate has been subdivided into its constituent pathological subgroups (Fig. 14). There appears to be some temporal association between the dips in prematurity mortality rate and the surges in fatal placental complications. The disease 'placental insufficiency' was responsible for these surges.

3.4.1(b) Early neonatal deaths from prematurity by maternity hospital

Mortality rates from the diseases of prematurity in each of Sheffield's maternity hospitals are shown in Fig. 15. Children who died at home or in private nursing homes have been omitted for clarity. In the preceding section the possibility of a diagnostic fashion causing the dips and surges in the neonatal prematurity mortality rate was examined. If there was a diagnostic fashion then it would

NEONATAL MORTALITY (n = 3507)



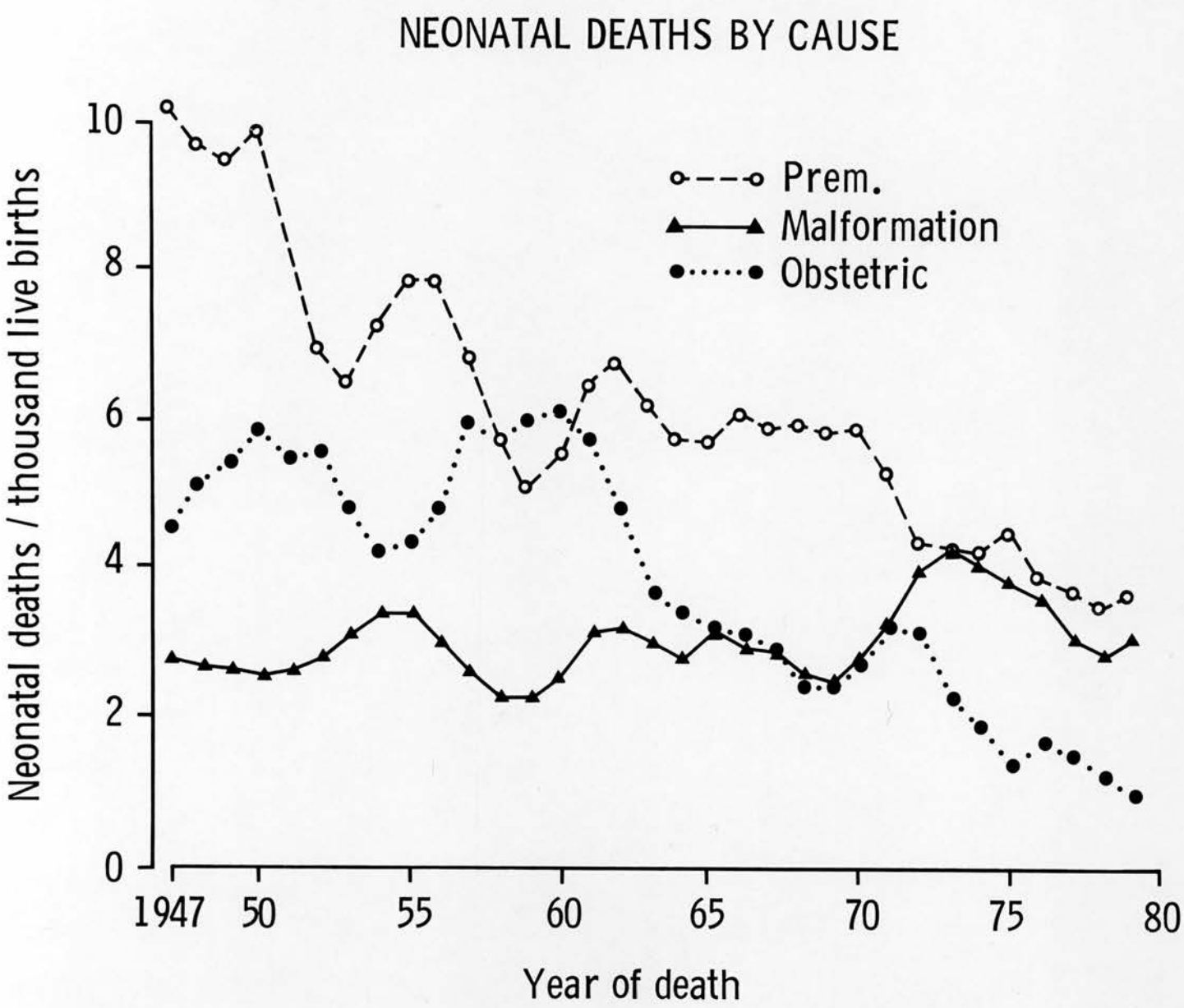
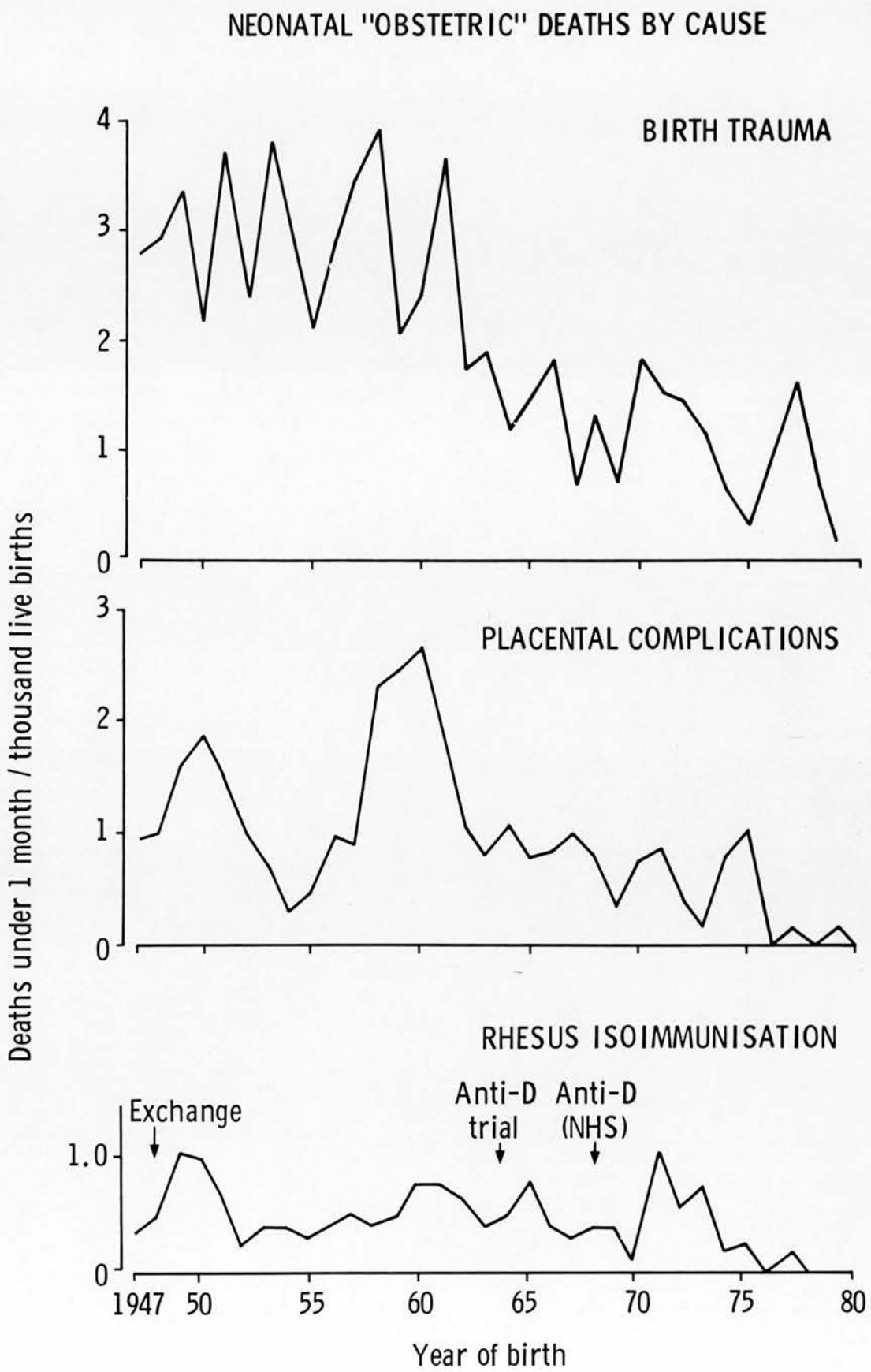

NEONATAL DEATHS BY CAUSE
Neonatal deaths / thousand live births
10
8
6
4
2
0
1947
50
55
60
65
70
75
80
Year of death
Prem.
Malformation
Obstetric

Fig. 13


NEONATAL "OBSTETRIC" DEATHS BY CAUSE
BIRTH TRAUMA
PLACENTAL COMPLICATIONS
RHESUS ISOIMMUNISATION
Exchange
Anti-D trial
Anti-D (NHS)
Deaths under 1 month / thousand live births
Year of birth
0
1
2
3
4
1.0
1947
50
55
60
65
70
75
80

FIRST WEEK DEATHS FROM PREMATURITY BY MATERNITY HOSPITAL

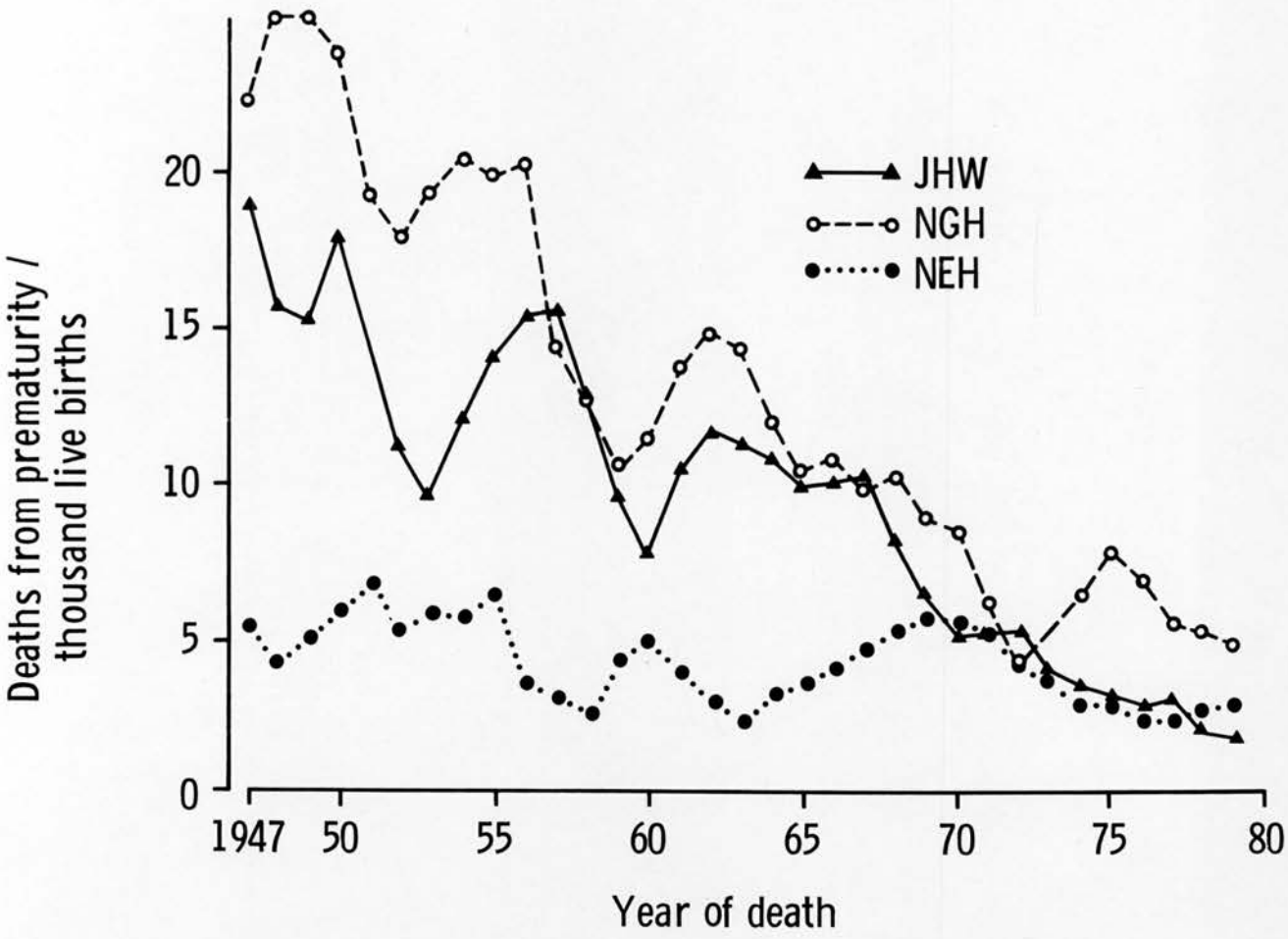


Fig.15

be expected to occur at the same time in certificates from NGH and NEH as these hospitals have the same paediatric staff. There is no formal rotation between these hospitals and JHW.

The hospital mortality rate from the diseases of prematurity would be expected to fall if an increasing number of low risk births occurred in hospital. Fig. 16 shows the changing distribution of the place of Sheffield live births (births in private nursing homes have been grouped with the home deliveries).

3.4.1(c) First day deaths

The time trends of the major causes of death in the first 24 hours are shown in Fig. 17. The reasons for the overall fall in this death rate appear to be primarily the decline in deaths from birth trauma and secondly an increased rate of decline of deaths from prematurity.

The trends in stillbirth and first day death rates are shown on a logarithmic scale in Fig. 18 so that the rate of decline of each can be compared directly. If the rate of decline of the first day mortality rate which began in the early 1960s were due to an increasing practice of registering such deaths as stillbirths then the rate of decline of the stillbirth curve would be expected to slow.

3.4.1(d) Foundlings

There were 23 infant deaths between 1947 and 1979 which were determined by a coroner's inquest to have been due to some form of inattention at birth. The distribution

PROPORTION OF SHEFFIELD LIVE BIRTHS DELIVERED AT HOME

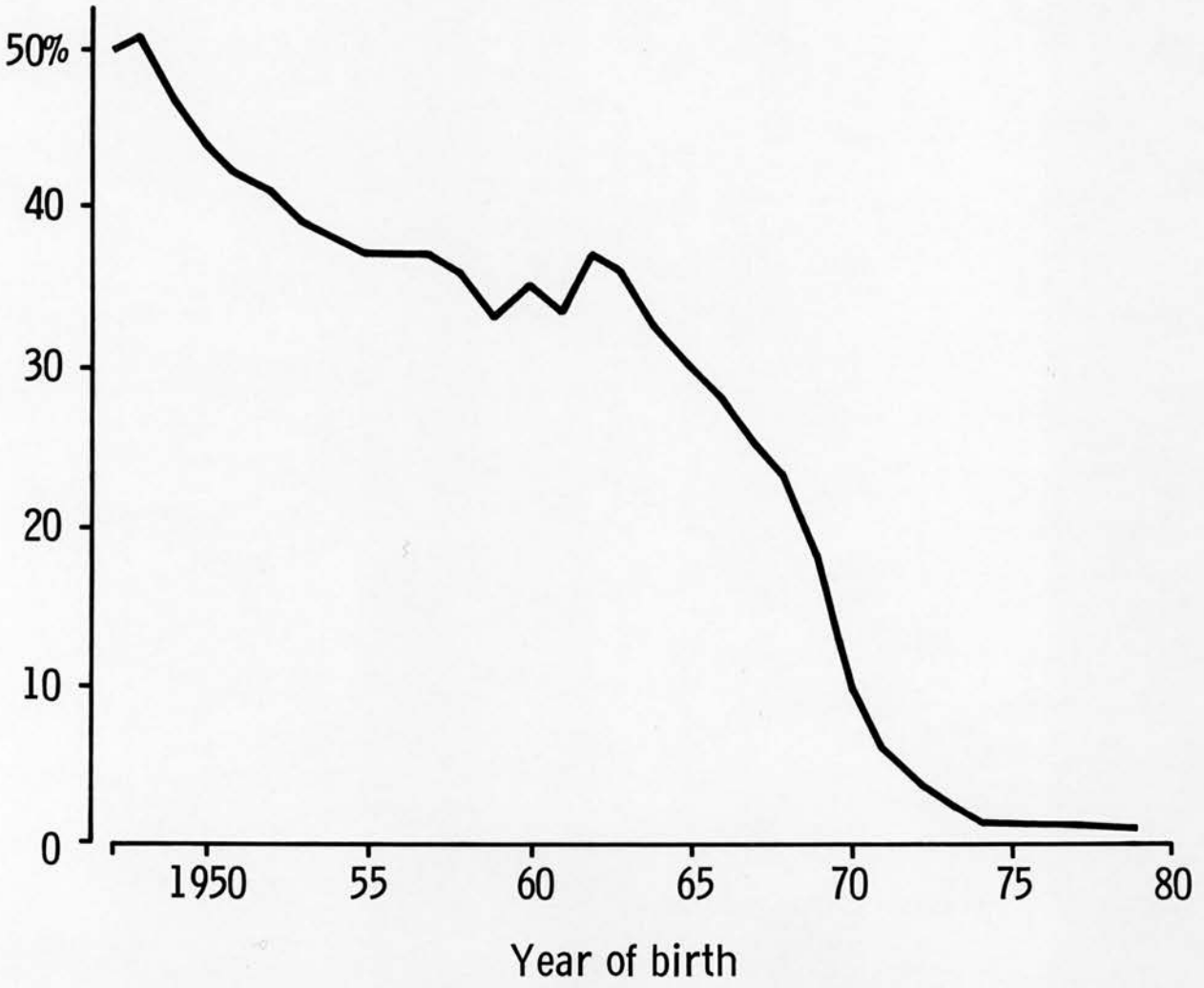
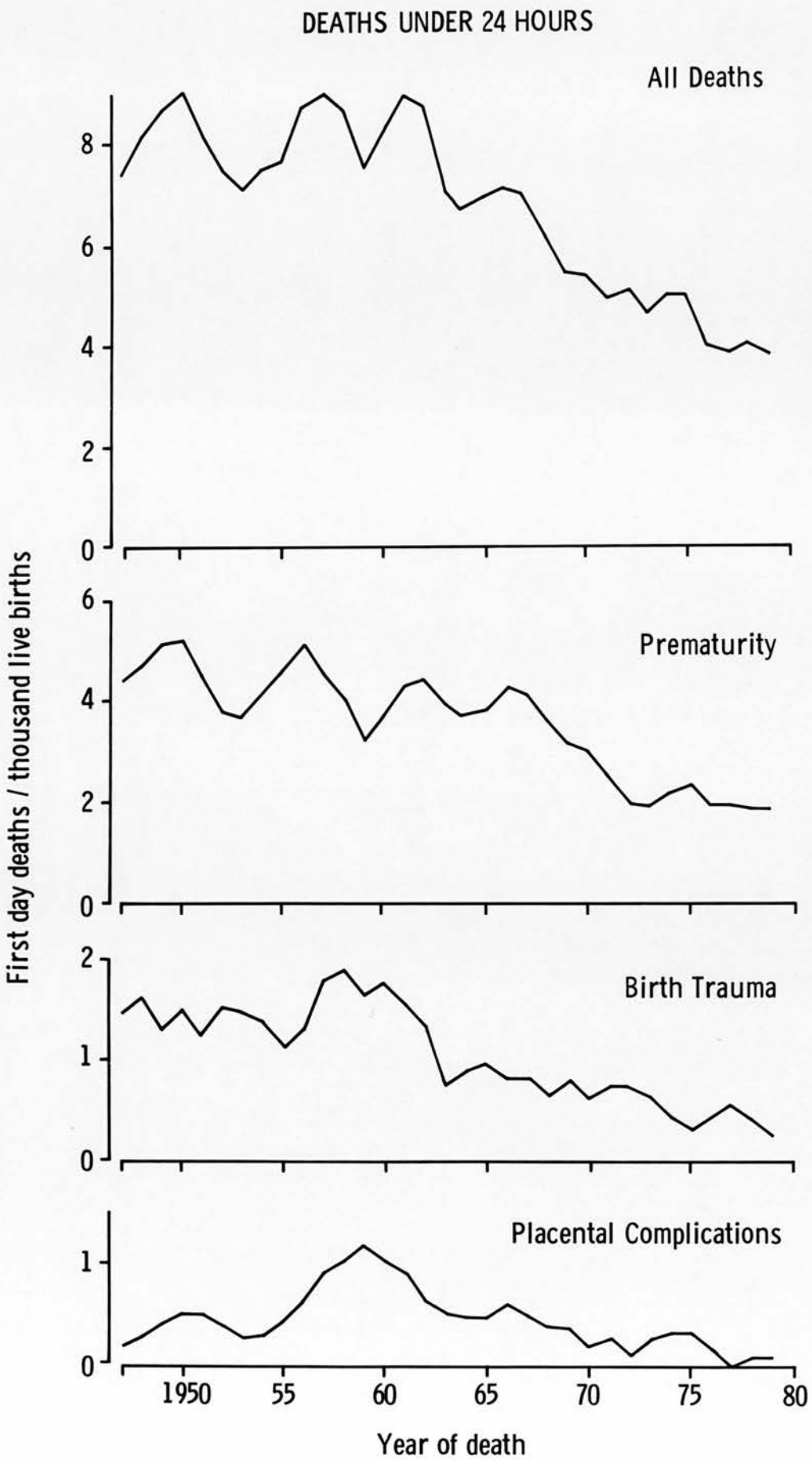
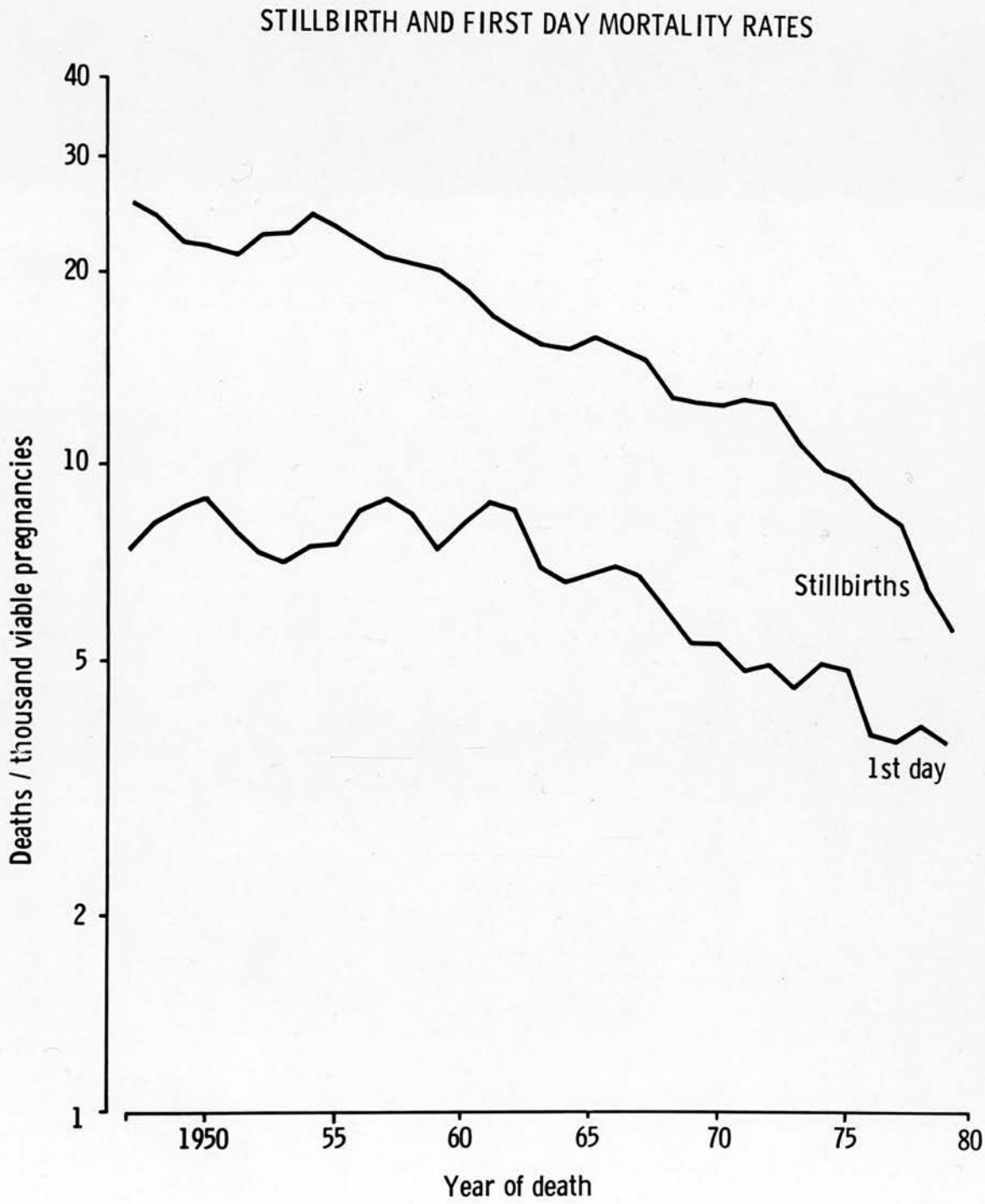


Fig.16


DEATHS UNDER 24 HOURS
All Deaths
Prematurity
Birth Trauma
Placental Complications
First day deaths / thousand live births
0
2
4
6
8
1
1950
55
60
65
70
75
80
Year of death


STILLBIRTH AND FIRST DAY MORTALITY RATES
40
30
20
10
5
2
1
Deaths / thousand viable pregnancies
Stillbirths
1st day
1950
55
60
65
70
75
80
Year of death

of these deaths by year of birth and social class is shown in Table VII. During the 12 years prior to the Abortion Act there were 13 foundlings; in the next 12 years there were four. This decline cannot be accounted for solely by the fall in the numbers of live births (see Appendix IV).

3.4.1(e) Comparisons of early neonatal deaths in Sheffield with the British Births surveys

The distribution of the causes of death in the first week among the Sheffield babies born in March 1958 and 5th-11th April 1970 are shown in Tables VIII and IX together with the distribution for England and Wales. The distribution of fatal early neonatal pathologies for Sheffield in the whole of 1958 and 1970 is also shown.

3.4.1(f) Comparison of early neonatal mortality among early spring births with overall neonatal mortality

Because of the small number of deaths among the Sheffield samples in March 1958 and 5th-11th April 1970 it is difficult to make comparisons with the national samples. To increase the numbers and test whether the method of sampling by season of birth might produce representative groups for studying early neonatal mortality, the distribution of the causes of early neonatal deaths among children born in March and the first week of April have been compared with the annual cohort for all 33 years. The results shown in Table X suggest that comparisons of proportional distributions of causes of death are reasonable. However, the numbers (and proportions) of early neonatal

Sheffield deaths analysed by month of birth for 1958, 1970 and the whole period 1947-1979 (Table XI) show a strong seasonal pattern.

3.4.1(g) Infant and perinatal mortality by cohort analysis

To obviate any significant effect due to variation in the recording of the age at death, stillbirths, infant and child deaths were analysed by birth cohort. This is illustrated in two ways in Fig. 19 which shows survivors in each birth cohort per thousand viable pregnancies (i.e. pregnancies which have completed 28 weeks gestation). This figure shows the effect of the dramatic declines in stillbirth and infant mortality rates. The relative importance of survival to the first birthday in determining the "reproductive efficiency" of a community is also shown. After the first birthday, there appears to be relatively little risk of dying in the rest of childhood.

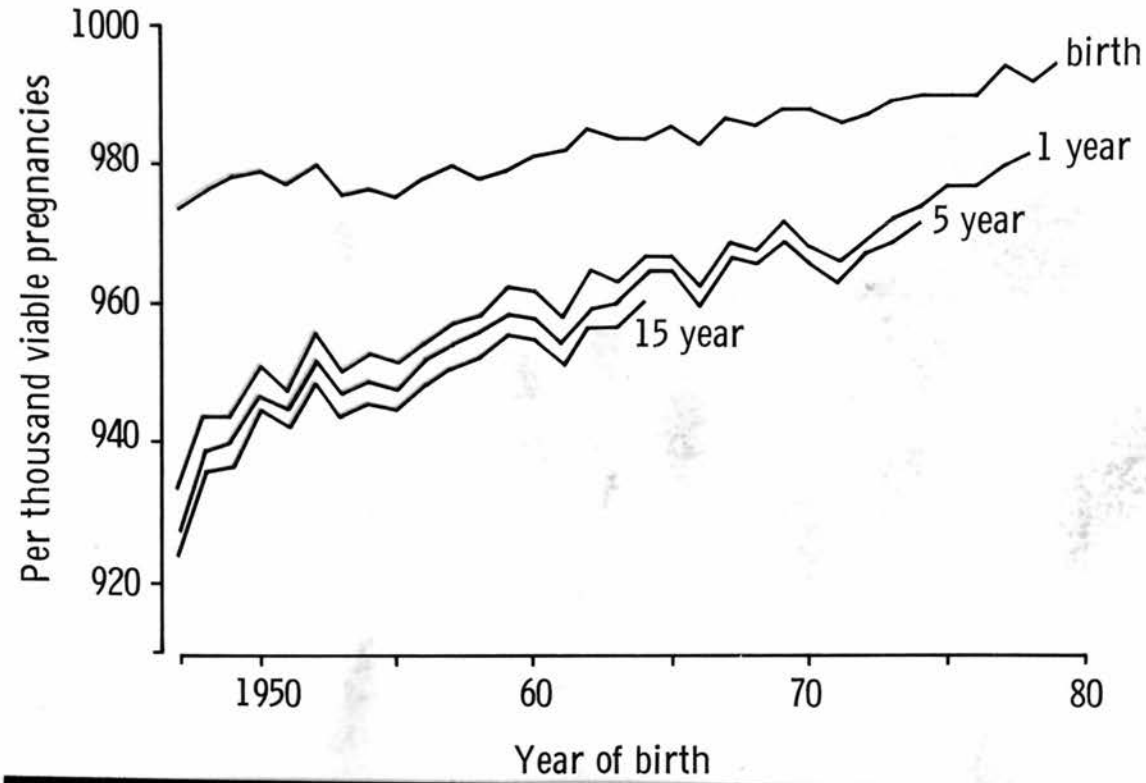
3.4.2 Postneonatal mortality

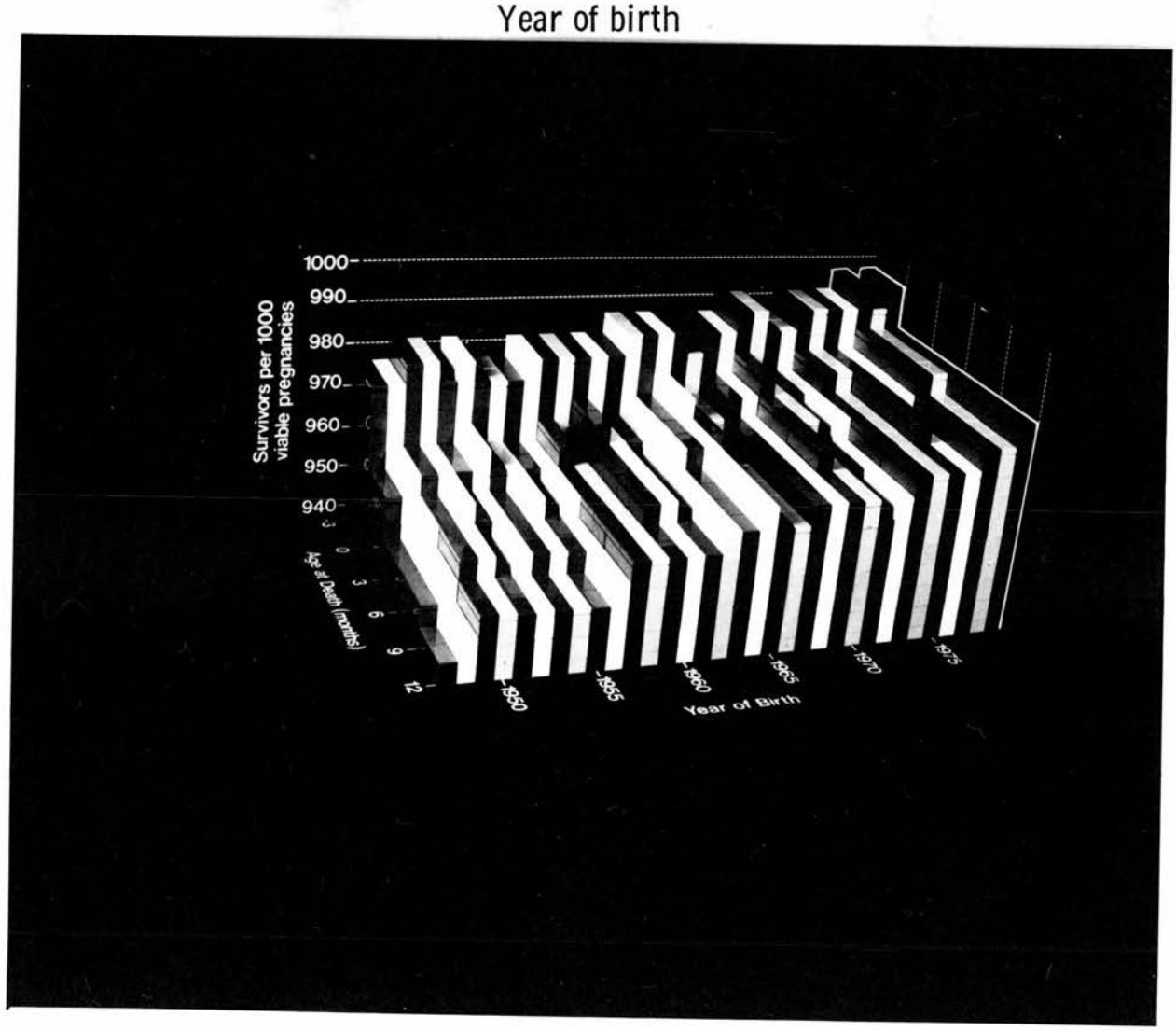
There were 1775 deaths in the postneonatal age group. The changing postneonatal mortality rate is shown in Fig. 8 and the proportionate distribution of these deaths by cause is shown in Fig. 20. The time trend of major causes of death in the postneonatal age group is shown in Fig. 21. The primary reason for the rapid decline in the postneonatal mortality rate (Fig. 8) is the decline in deaths from infectious diseases.

3.4.2(a) Apparent disappearance of hypernatraemia from Sheffield postperinatal deaths

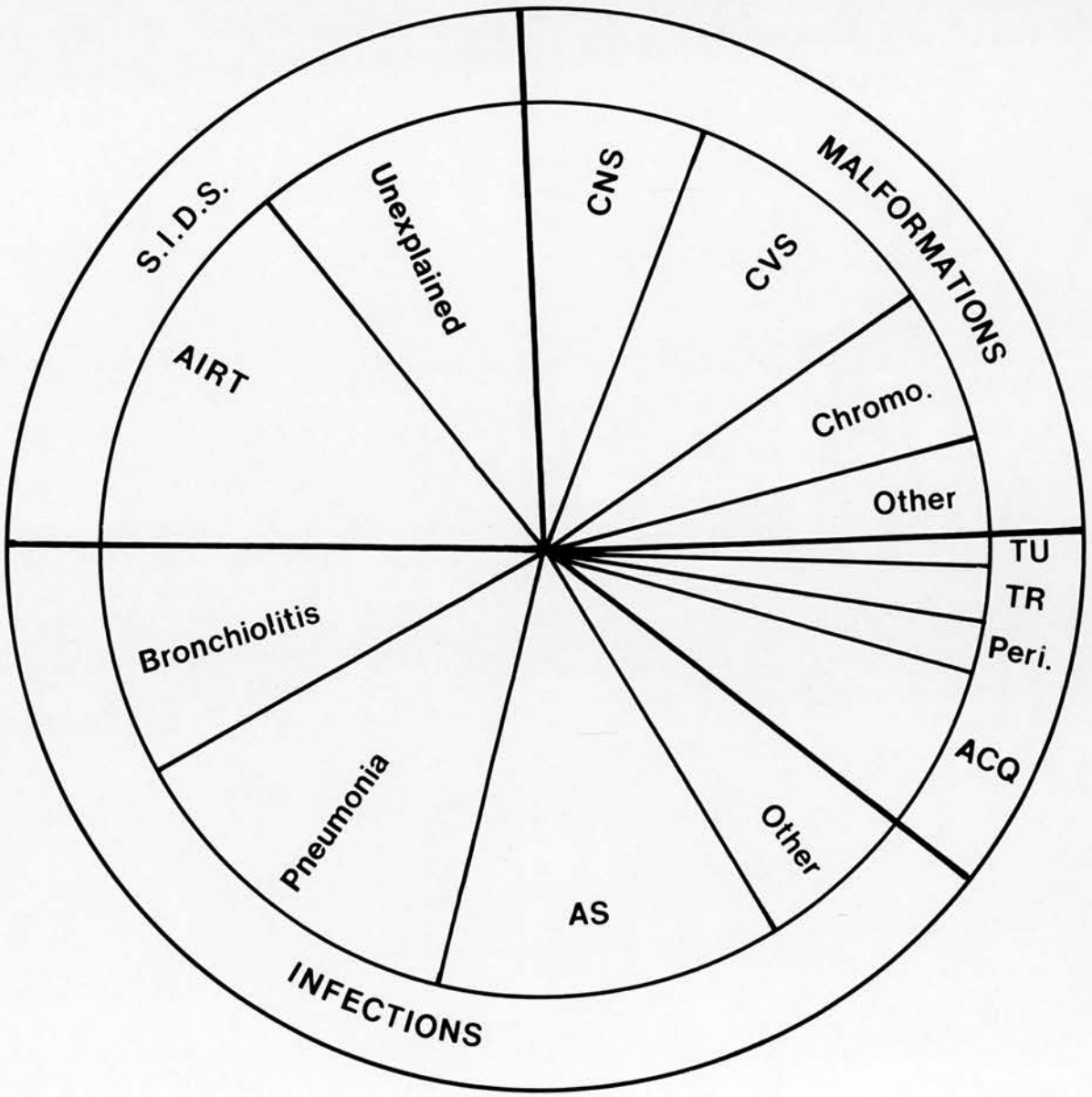
Table XIII shows the complete figures obtained by this part of the study. Columns six and seven are not mutually

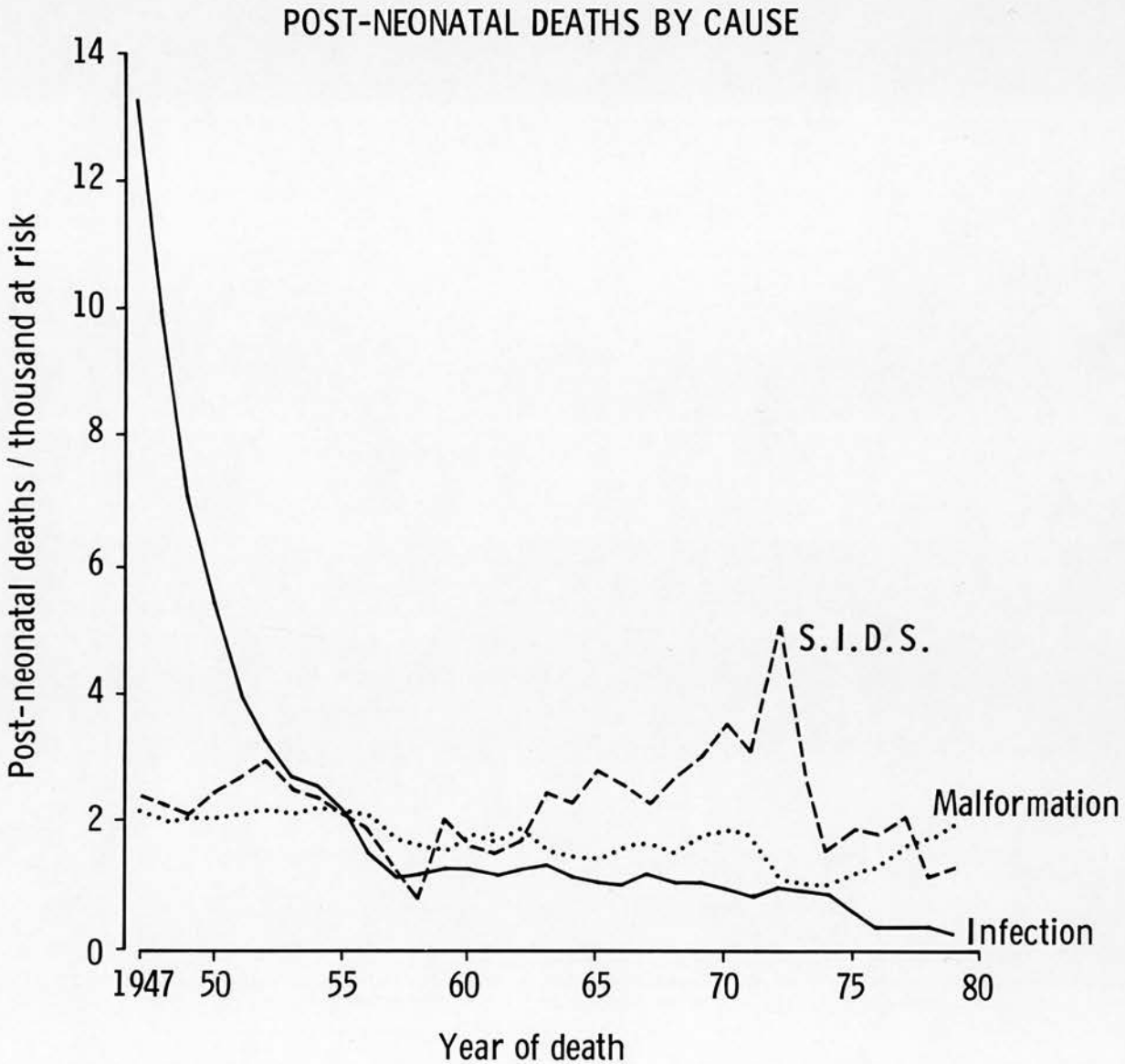
SURVIVAL RATES





POSTNEONATAL MORTALITY (n = 1775)




POST-NEONATAL DEATHS BY CAUSE
Post-neonatal deaths / thousand at risk
14
12
10
8
6
4
2
0
1947
50
55
60
65
70
75
80
Year of death
S.I.D.S.
Malformation
Infection

exclusive as infants who died from gastroenteritis and hypernatraemia are recorded in both columns. The variation in the proportion of postperinatal deaths in which a vitreous examination was performed is accounted for by deaths from major congenital malformations.

No Sheffield infant died with hypernatraemia between December 1976 and December 1980. Between October 1972 and December 1979, 238 necropsies were done at SCH on Sheffield infants aged between 1 week and 2 years. Twenty-three of these infants had vitreous hyperelectrolytaemia. The underlying causes of death of these children as given on the necropsy record are listed in Table XII.

Feeding histories were obtained from the mothers of 20 of the 23 infants who died with hypernatraemia and from age-matched living controls drawn from the Sheffield birth register. Equal numbers of cases and controls received unmodified (high solute) milks but four controls had been breast fed beyond six weeks whereas none of the infants who died was. There was no difference between cases and controls in the introduction of cereals or solids into the diet in the last three weeks of life nor was there any difference in the number receiving solids by the date of death. The most striking difference was that seven of the infants who died had complete changes of milk formula whereas none of the controls did.

3.4.3 Preschool mortality

There were 845 deaths in the 1-4 year age group. The decline of the preschool death rate was shown in Fig. 9. The

proportionate distribution of these deaths by cause is shown in Fig. 22.

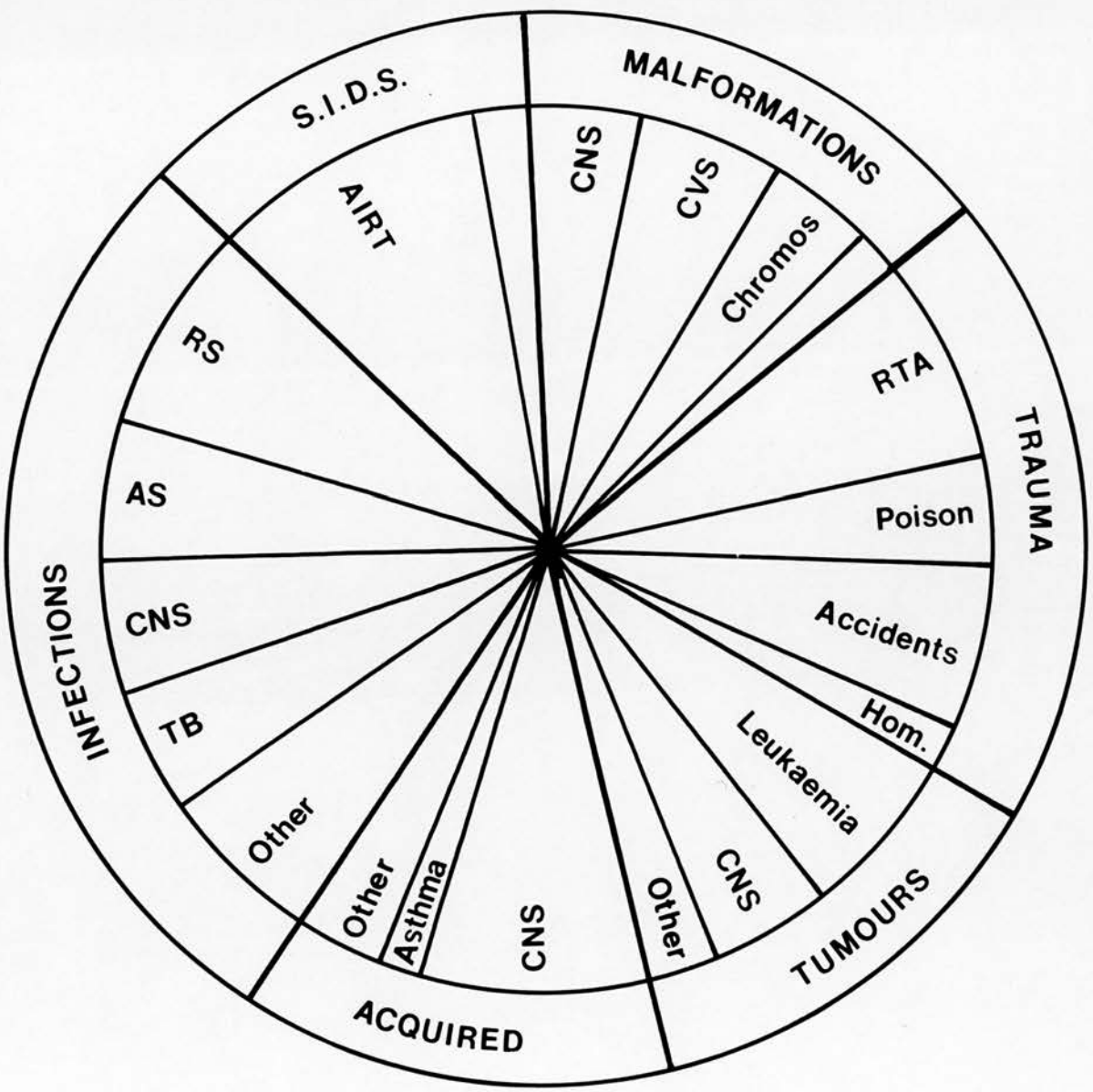
Mortality falls rapidly with increasing age, therefore to obtain a reasonable sample this age group covers four times the combined age spread of the previous two. The number of children alive in this group has also increased hence the vertical scales on the graphs are ten times greater than in graphs referring to infant deaths.

The distribution with time of the deaths which contributed to Figs. 9 and 22 are shown in Fig. 23. In each of the constituent graphs of Fig. 23, the principal contributory subgroup has been shown as a dotted line. The interplay of accidental falls with road traffic accidents (RTA) has been shown under the trauma curve; another important condition in this group of deaths is self-poisoning (Table XIV) which peaked in 1958-61 and 1974-77. The increased separation between leukaemia and all tumours in 1951-54 and 1974-79 is due to an increased number of CNS tumours. The increased separation between congenital heart deaths and all malformations in the 1960s was primarily due to the rise in spina bifida deaths, partly due to greater survival beyond infancy. The later separation of total malformations from congenital heart deaths is due to an increased number of respiratory and renal malformations and possibly to increasingly successful corrective cardiac surgery.

3.4.4 Schoolchild mortality

There were 837 deaths in the 5-14 year age group. The decline in the schoolchild mortality rate was shown in Fig. 9.

PRESCHOOL (1-4 years) MORTALITY (n = 845)



PRE SCHOOL MORTALITY BY CAUSE

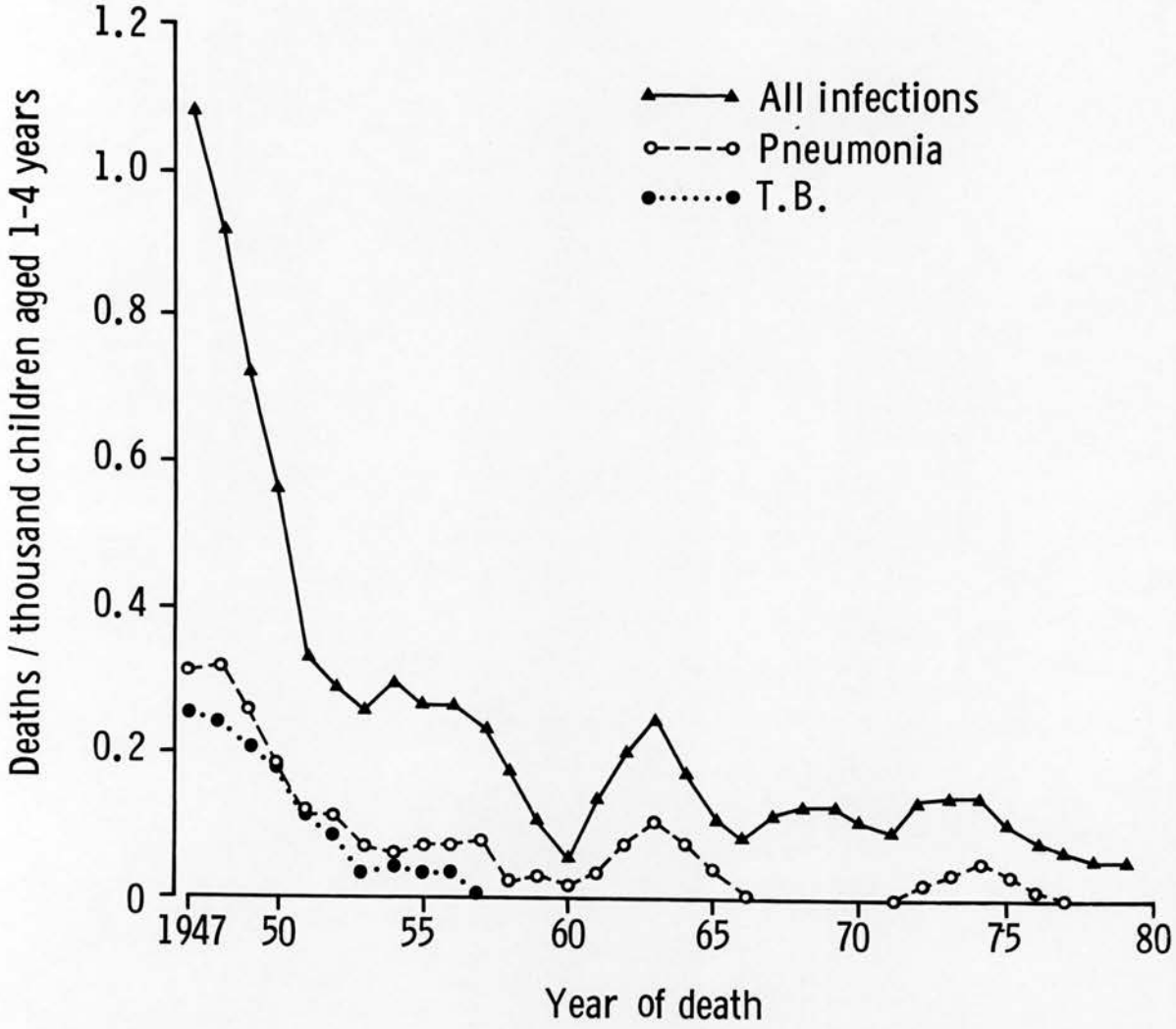
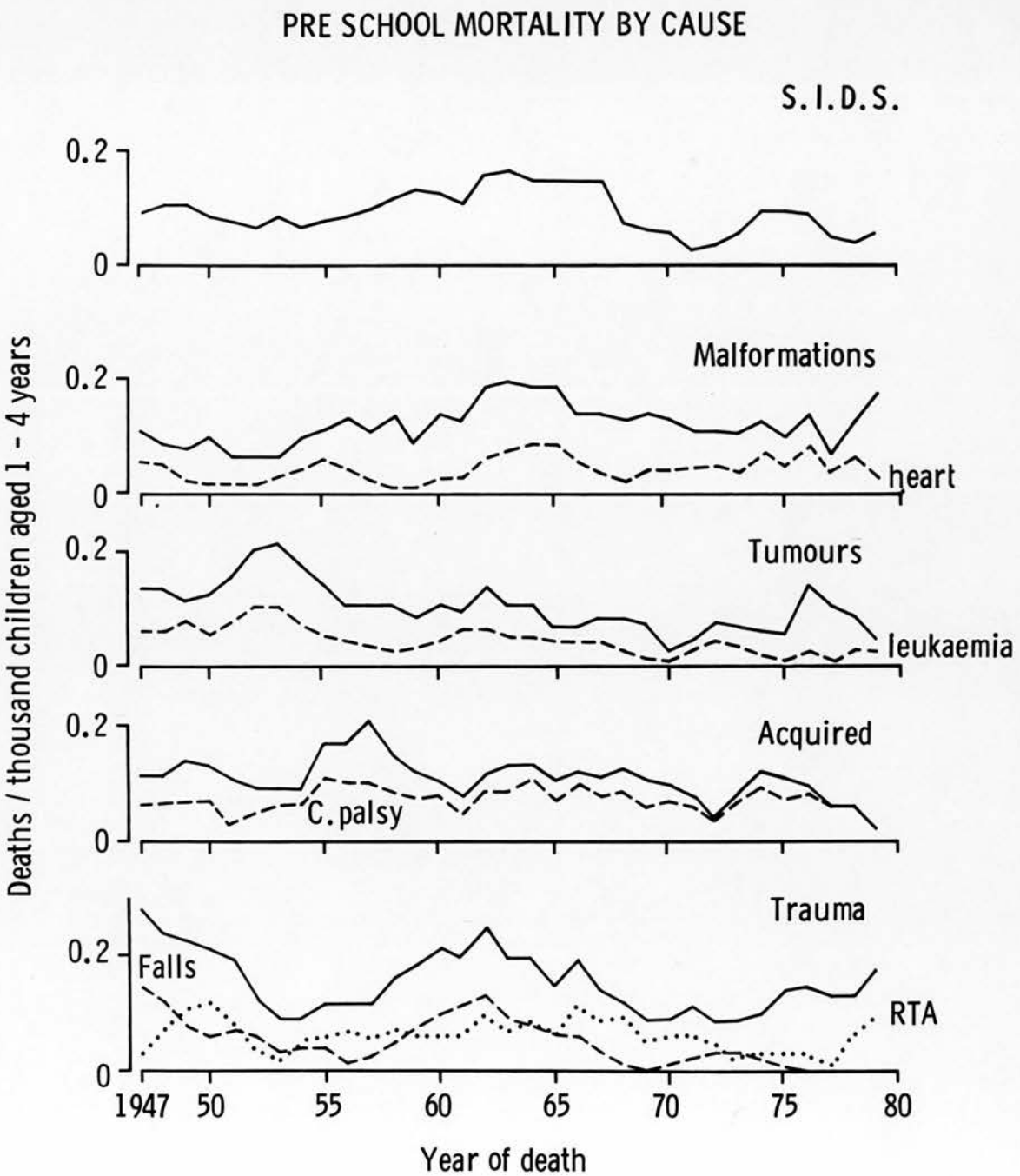


Fig. 23a


PRE SCHOOL MORTALITY BY CAUSE
S.I.D.S.
0.2
0
Malformations
heart
Tumours
leukaemia
Acquired
C. palsy
Trauma
Falls
RTA
1947
50
55
60
65
70
75
80
Year of death
Deaths / thousand children aged 1 - 4 years

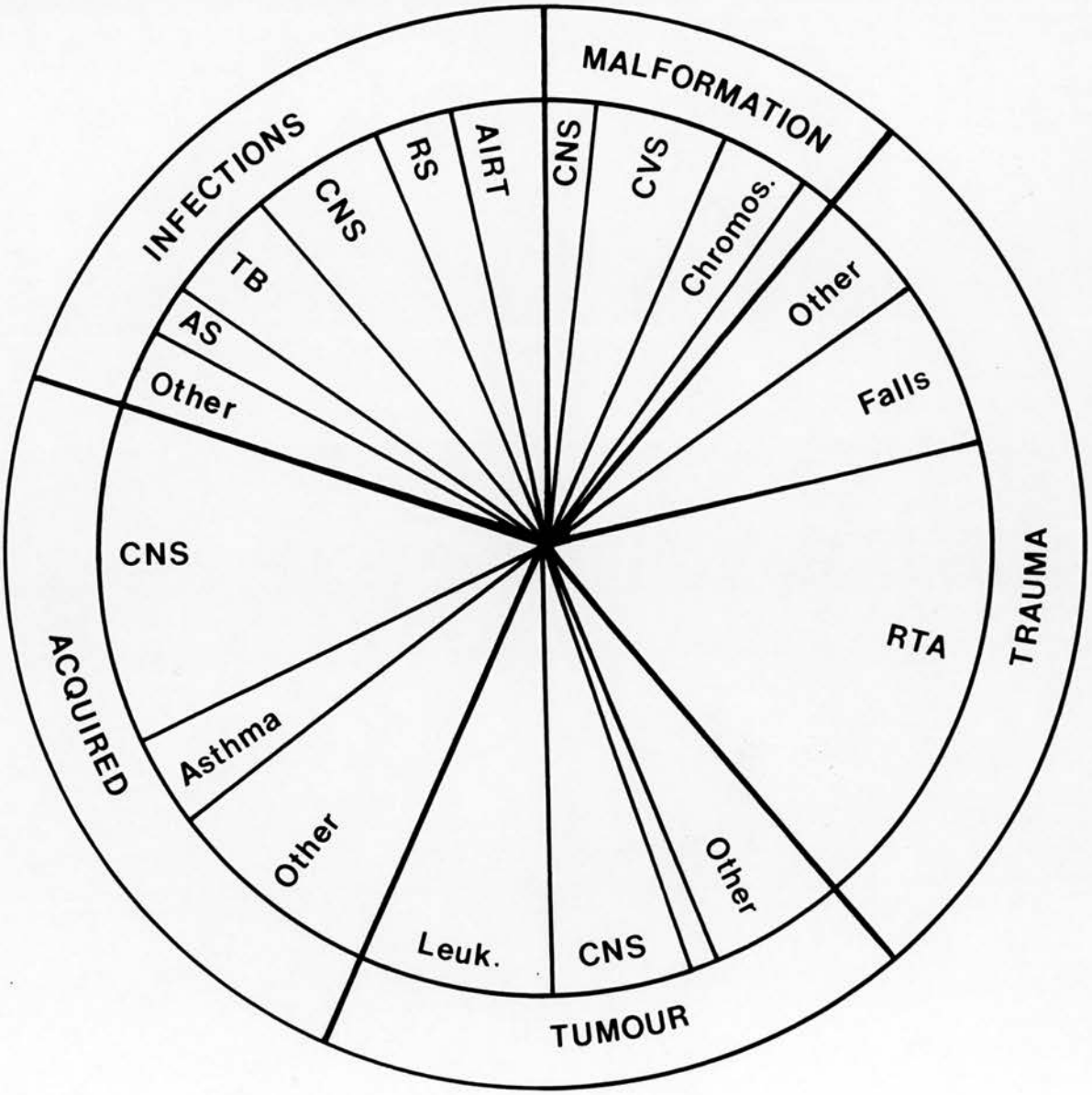
The proportionate distribution of these deaths by cause is shown in Fig. 24. This age group contains approximately twice as many children as the preschool age group and ten times as many children as the infant groups. The mortality rate is so low that such a combination is necessary to obtain a reasonably sized group. The proportion of deaths in the 5-9 year group remained approximately equal to the proportion in the 10-14 year group throughout the period studied (Fig. 7).

The distribution of the deaths which contributed to Figs. 9 and 24 are analysed by cause in Fig. 25. The principal contributory disease to each major pathological group is shown as a dotted line. No single solid tumour gave a persistent trend in fatal incidence although, as in the preschool age groups, CNS tumours are the second commonest malignancy. The decline of the miscellaneous 'acquired' groups in the 1940s was due to the disappearance of deaths from rheumatic heart disease and a decline in asthma deaths. The decline in infection deaths was due to a decline in pneumonia, gastroenteritis and meningitis as well as the decline in tuberculosis deaths illustrated.

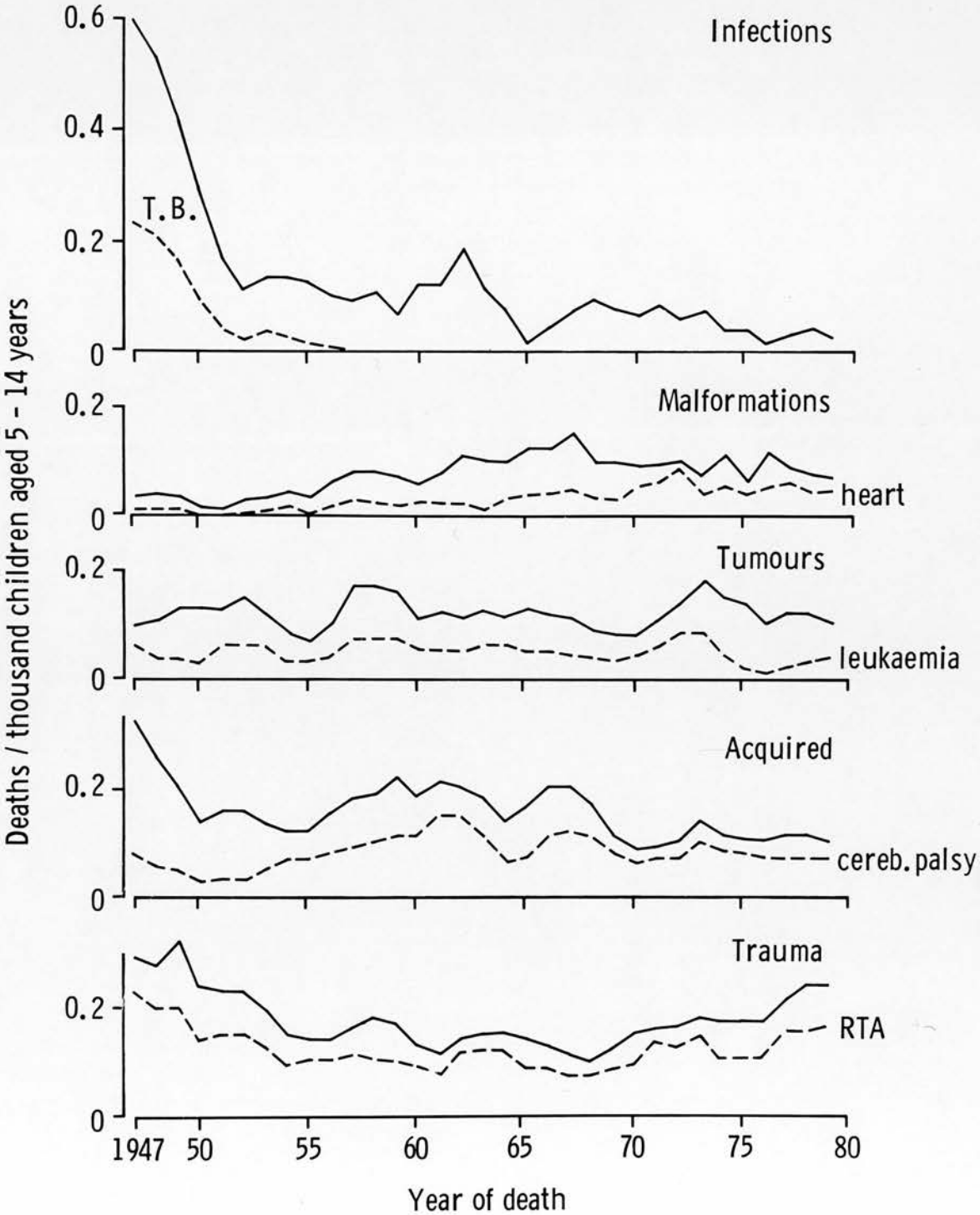
3.4.5 Simultaneous analysis of cohort and annual age-specific mortality trends

Age-specific mortality rates were examined by year of birth (cohort analysis) and year of death (annual analysis) simultaneously. The results of this study are summarised in a model made of children's building bricks (Sunderland, 1980) in Fig. 26. The cohorts move diagonally across the figure and have been shaded for clarity. Because of the disparity of scale between early deaths (stillbirth and first year) and later deaths, the mortality

SCHOOLCHILD (5-14 years) MORTALITY (n = 837)



SCHOOL CHILD MORTALITY BY CAUSE



rates have been presented on a logarithmic scale.

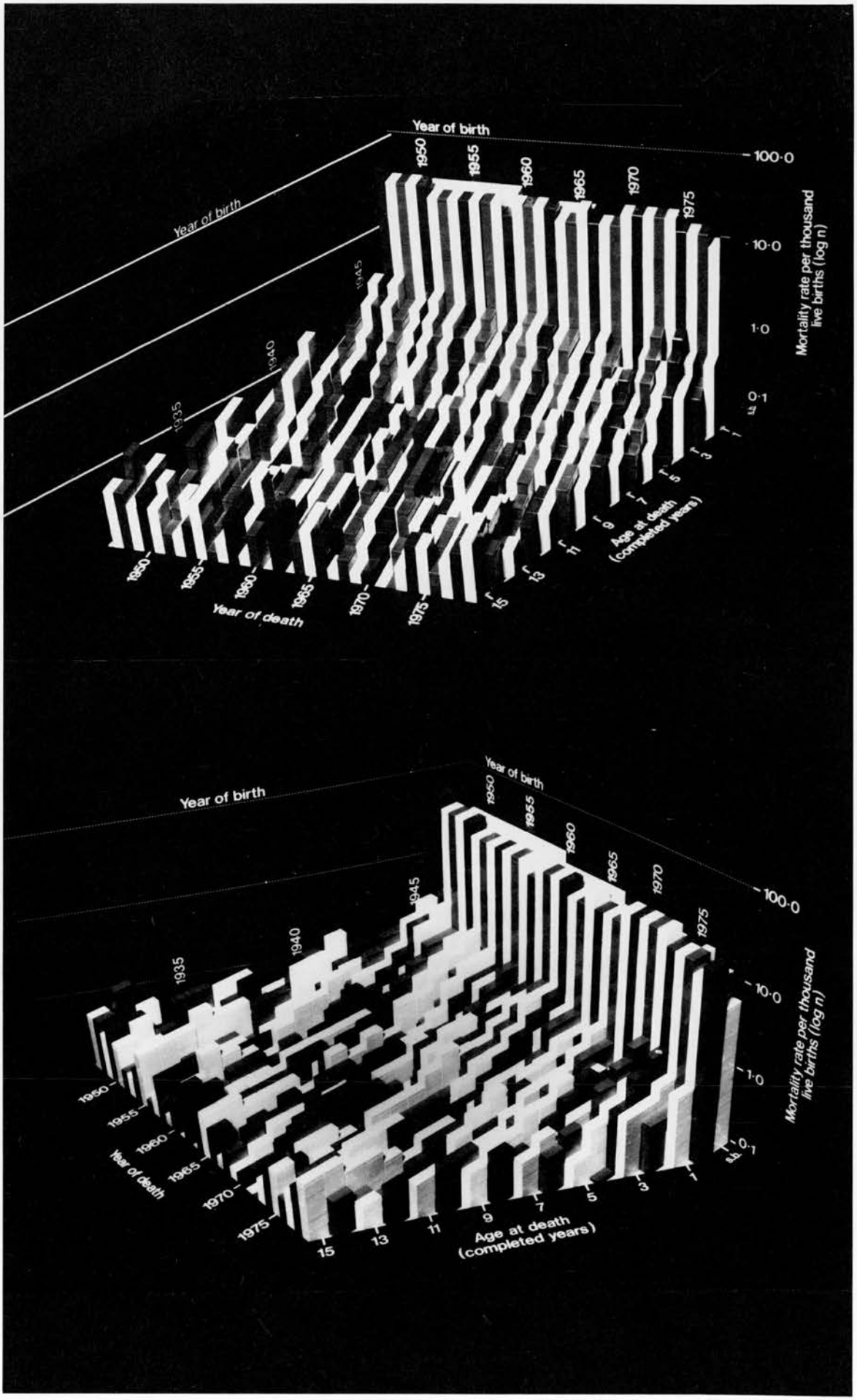
A series of curves is shown in Fig. 26b which represent contours of sections taken across the year of birth axis of the model in Fig. 26a. These curves show the proportions of children in each cohort who died in a particular age group.

3.4.6 Neonatal and postneonatal mortality rates in selected large English towns

Six towns were identified where the population did not alter by more than 1% following the 1974 local government re-organisation. They were Bristol, Coventry, Derby, Hull, Plymouth and Southampton. The changing neonatal and post-neonatal mortality rates in these towns together with those of Sheffield and the rates for England and Wales are shown in Fig. 27. Among these seven towns, the changes in these age specific mortality rates in Sheffield have most closely reflected the national average.

3.5 Mortality trends II - Death by cause

Because of the Sheffield Register Office fire on 21st February 1947, the cause of death was unknown for 14 of the children. The underlying causes of death of the remaining 7035 children were grouped into seven major categories: Epinatal (Epi), Malformation (Malf), Infection (Inf), Trauma (Tr), Tumours (Tu), Acquired (Ac) and Unexplained (SID). The constituent subgroups of each category are given in Appendix III. These groups follow approximately the ICD9 (WHO 1977) groupings with minor modifications (e.g. meningitis is coded under 'Infection' rather than under 'Nervous System').


Year of birth
1950
1955
1960
1965
1970
1975
100·0
10·0
1·0
0·1
Mortality rate per thousand live births (log n)
Year of birth
1945
1940
1935
1950
1955
1960
1965
1970
1975
Year of death
15
13
11
9
7
5
3
1
Age at death (completed years)
Year of birth
1950
1955
1960
1965
1970
1975
100·0
10·0
1·0
0·1
Mortality rate per thousand live births (log n)
Year of birth
1945
1940
1935
1950
1955
1960
1965
1970
1975
Year of death
15
13
11
9
7
5
3
1
Age at death (completed years)

AGE-SPECIFIC COHORT MORTALITY RATES

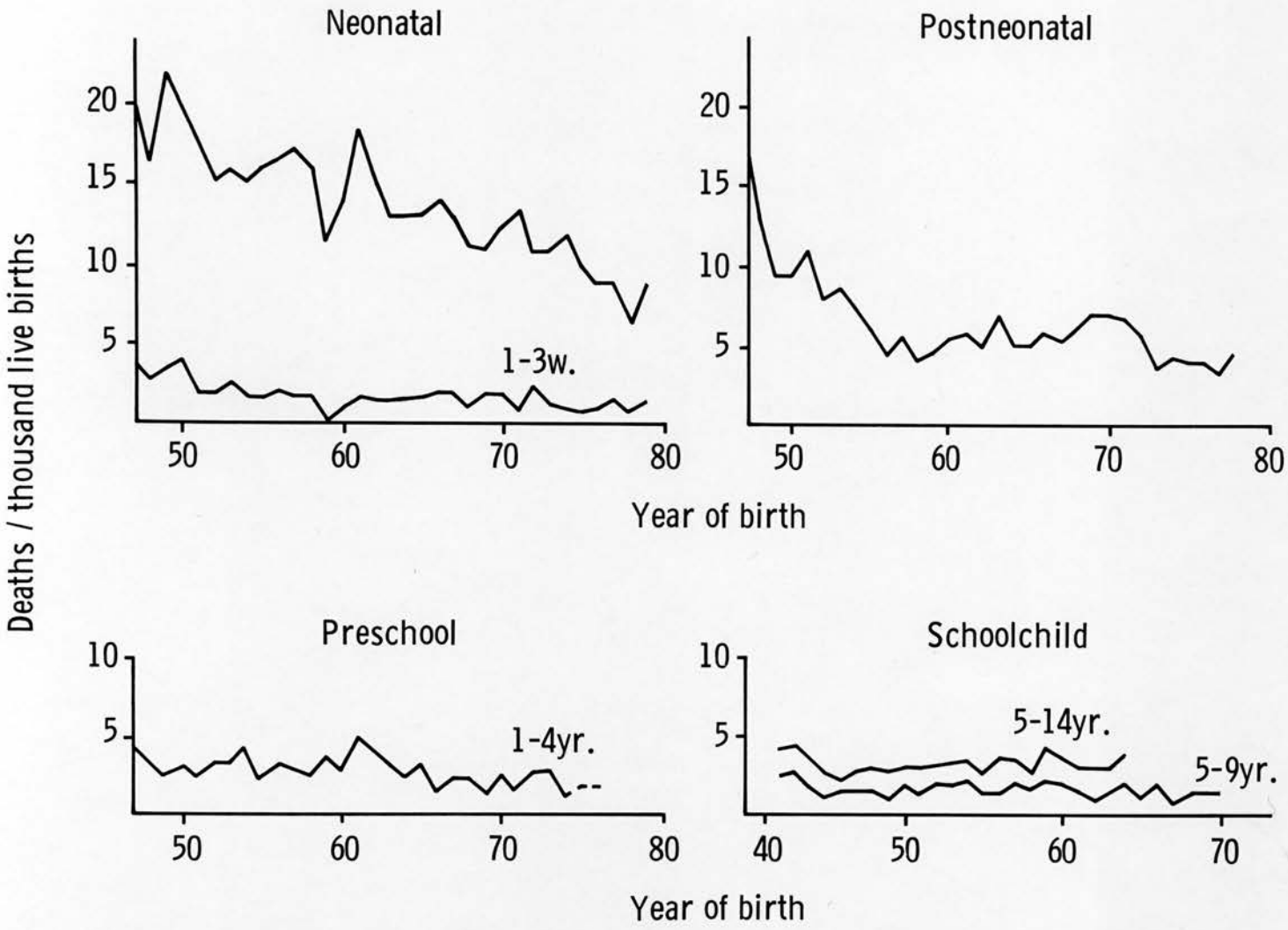


Fig. 26b

NEONATAL MORTALITY RATES IN SELECTED ENGLISH TOWNS

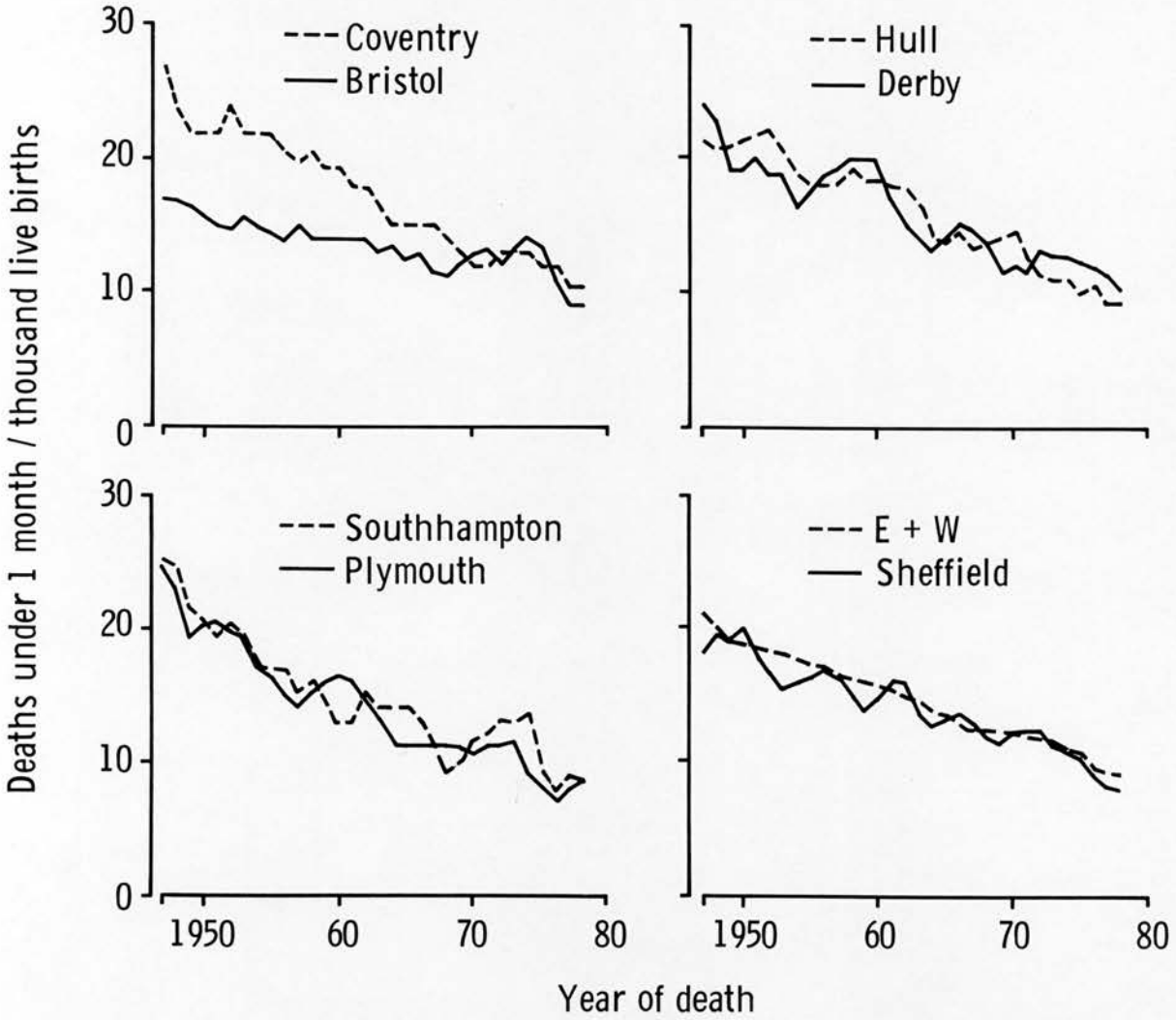


Fig. 27a

POST-NEONATAL MORTALITY RATES IN SELECTED ENGLISH TOWNS

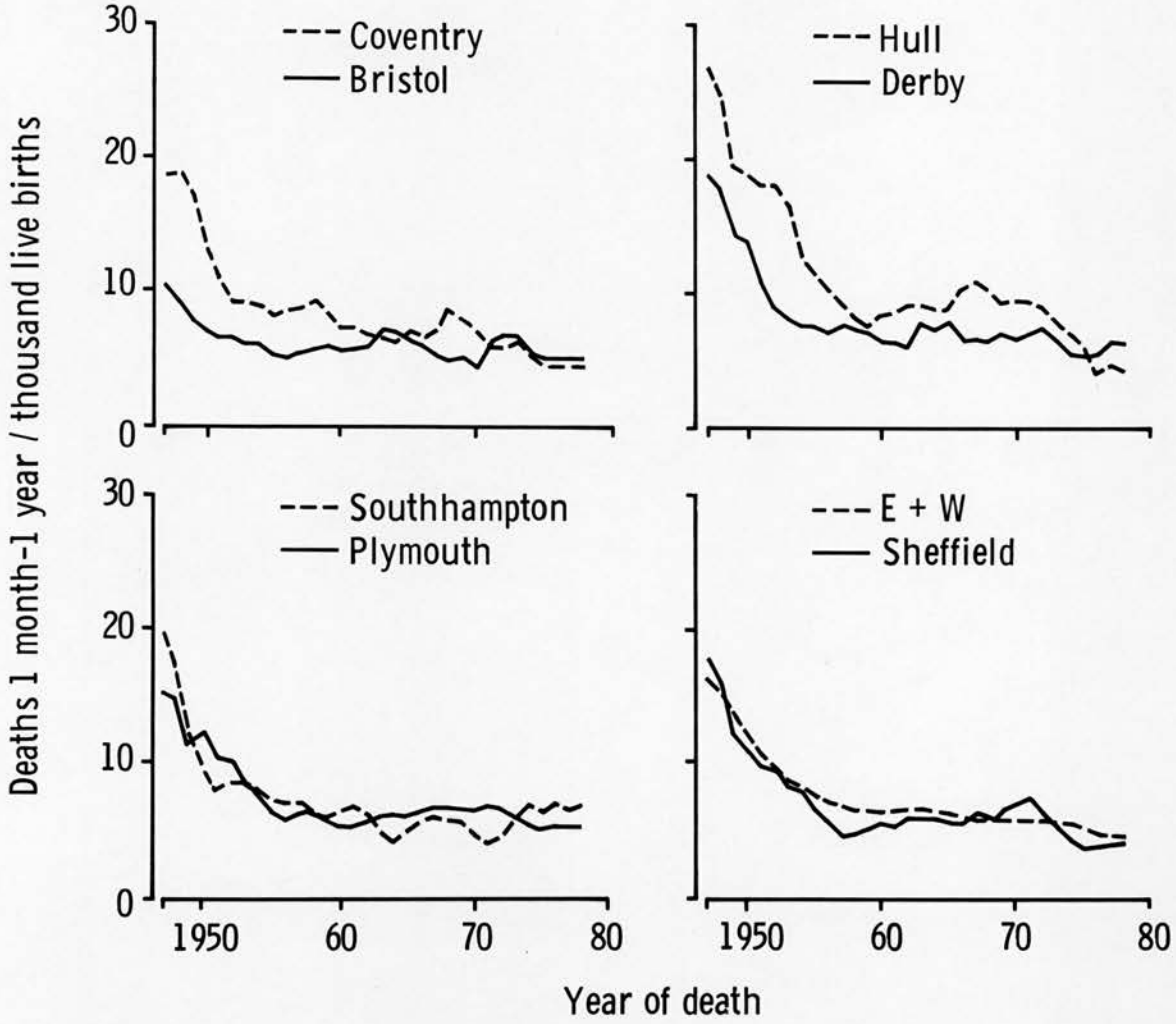


Fig. 27b

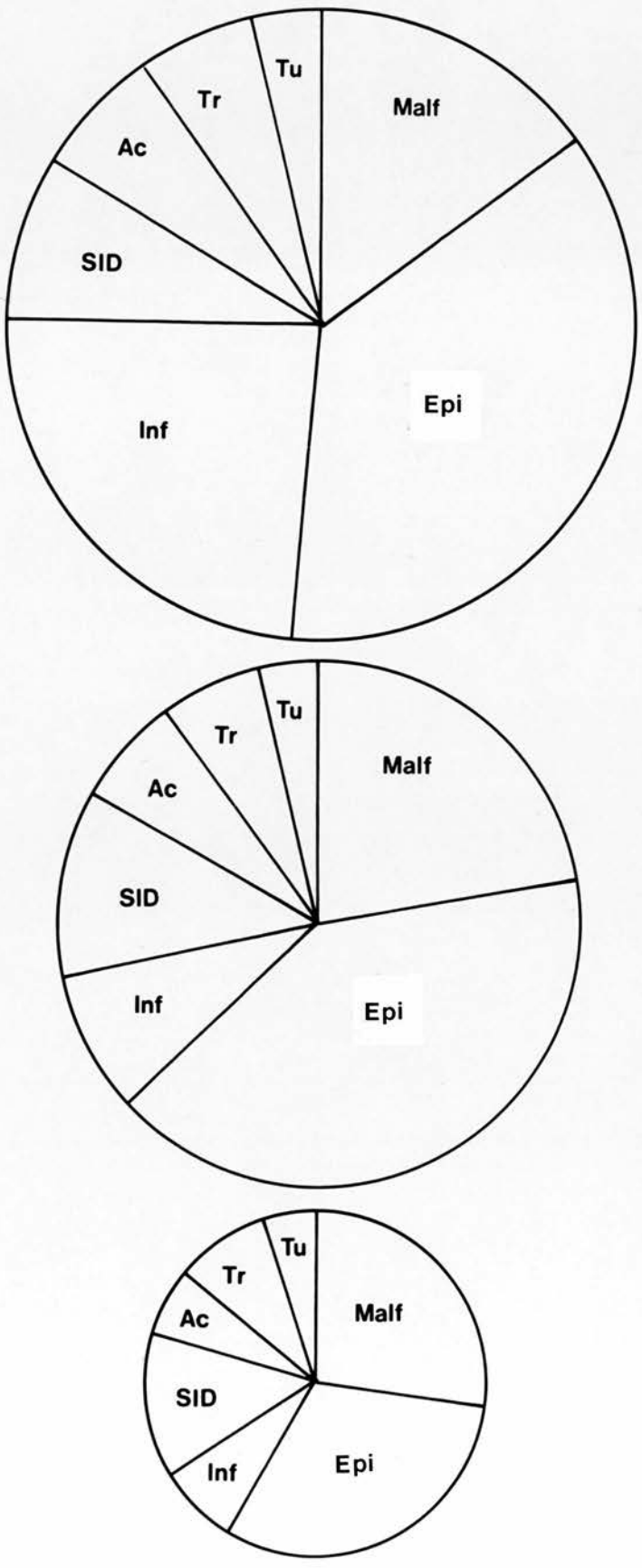
The distribution of these deaths by age and cause is shown in Table I. The proportionate distribution of the deaths by cause is summarised in consecutive 11-year periods in Fig. 28 where each diameter is the same as in Fig. 7, representing the overall child mortality rate in each 11-year period. The proportionate distribution of the pathologies which make up each major class of deaths is shown in Fig. 29 where each diameter is proportional to the mortality rate for each group. The changing pattern of the causes of death with time is shown in Fig. 30 which has been split for clarity into deaths from those diseases which might be considered preventable and those which might be considered inevitable. The miscellaneous groups of 'acquired' conditions has been omitted from Fig. 30 for clarity. The composite mortality rate for this miscellaneous group of conditions fell from 3.1 deaths per thousand children aged 0-15 years in 1947 to 0.8 in 1955 and then fluctuated about this level with no significant variation (the rate in 1979 was 0.8).

3.5.1 The epinatal diseases

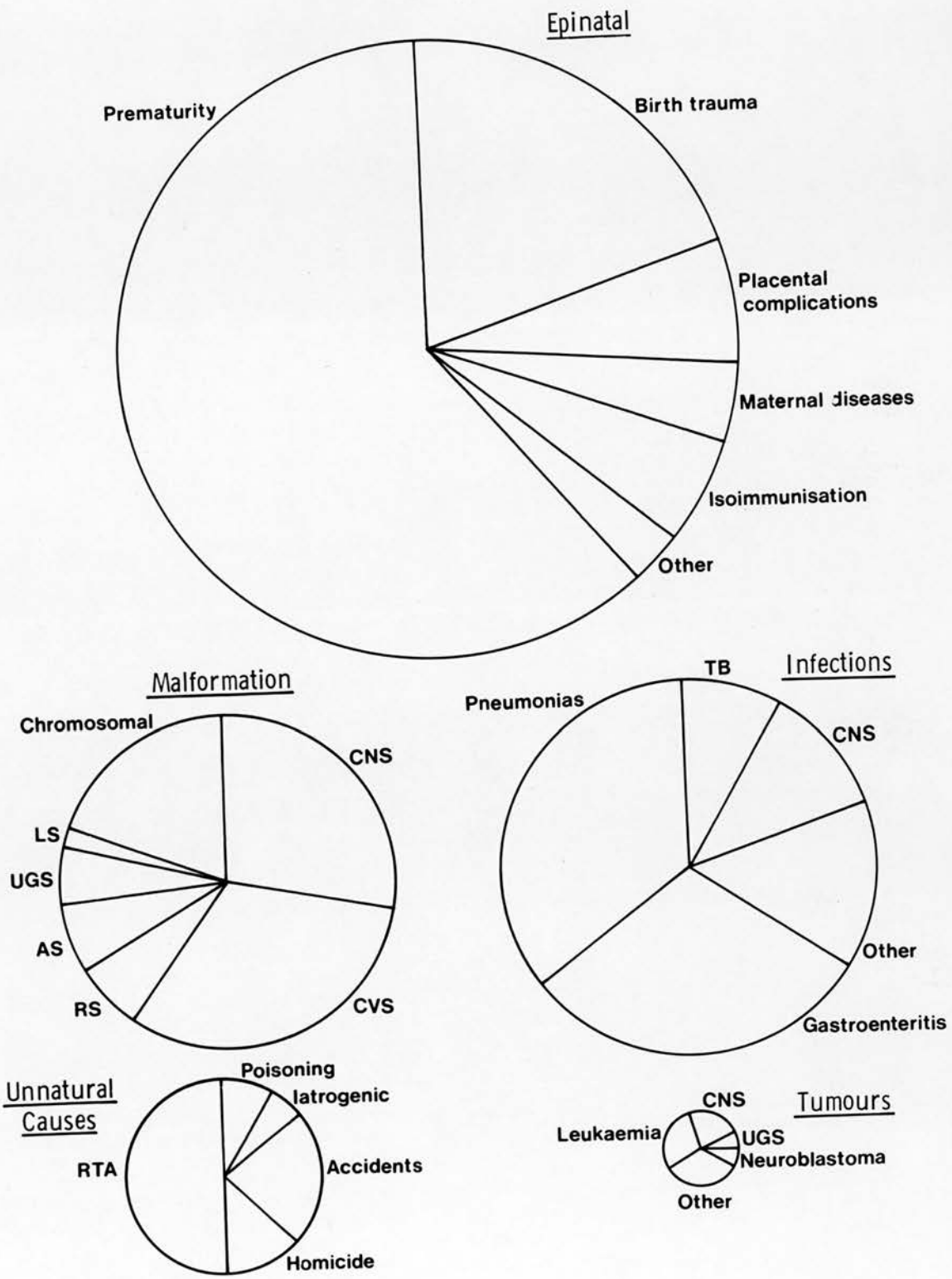
Epinatal diseases were the underlying cause of death in 2589 (37%) of the 7035 children. Almost all of the deaths from epinatal disease occurred within seven days of birth (Table I). The 38 deaths which occurred among children more than one month old were all in mentally handicapped children where there was an unequivocal statement referring to a perinatal insult. The distribution with time of the 2551 neonatal deaths from epinatal disease is shown by cause in Figs. 13 and 14.

PROPORTIONATE DISTRIBUTION OF DEATHS BY CAUSE

○ = 1 death / 10,000 children



CHILD DEATHS 1947-1979 BY CAUSE

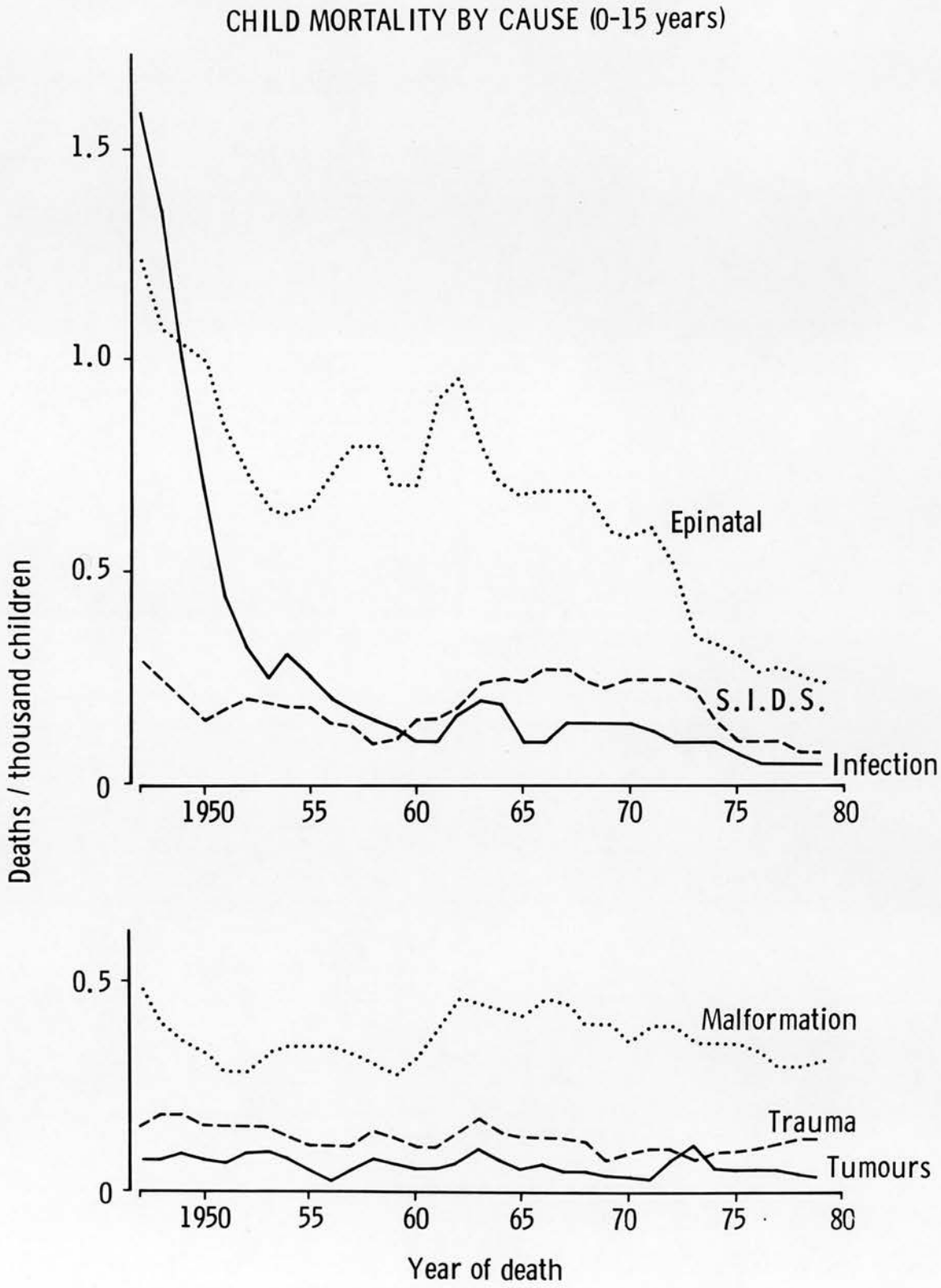


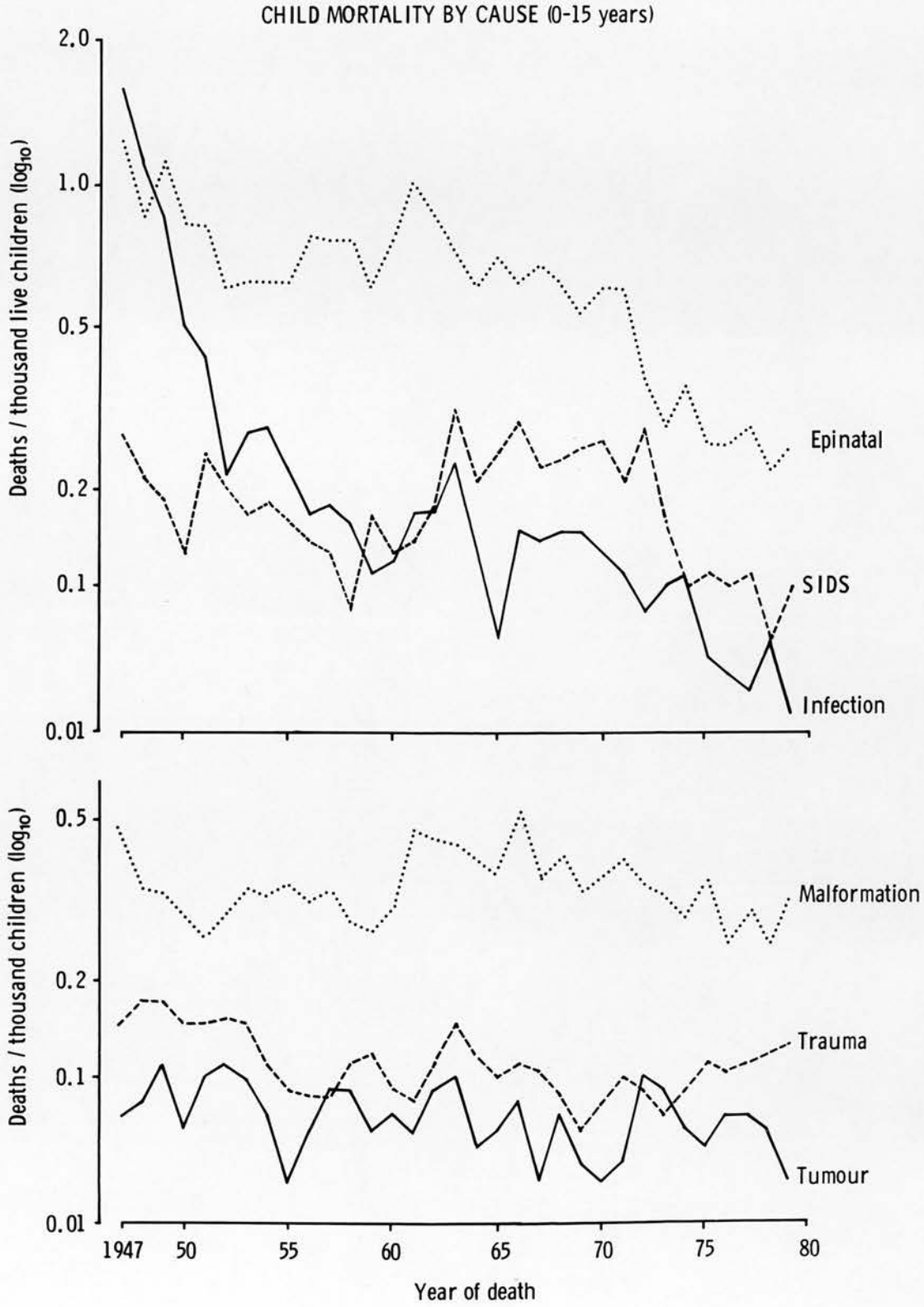
Prematurity not otherwise specified accounted for 709 of the 2588 deaths. Extreme immaturity implying a birth weight of less than 1000 g accounted for 321 deaths. The idiopathic respiratory distress syndrome accounted for 168 deaths and hyaline membrane disease a further 170; some pathologists appear to prefer the term 'primary atelectasis' which was given as the cause of death of 133 children. Pneumothorax as a consequence of immature lung disease was the given cause of death in 12 children. Intra-ventricular haemorrhage was the given cause of death in 38 children. Additional causes of death were pulmonary haemorrhage 20, meconium aspiration 12, post-ventilation syndrome 5 and other diseases of prematurity 2.

Birth trauma was constituted from eight categories: prolonged rupture of the membranes (7 deaths), precipitate delivery (7 deaths), prolapsed cord (16 deaths), intrauterine asphyxia (215 deaths), trauma on delivery of the head (83 deaths), intracerebral haemorrhage (94 deaths), tentorial tear (90 deaths), and subdural haematoma or skull fractures (7 deaths).

Placenta praevia accounted for 51 deaths; other forms of placental haemorrhage for 78 deaths, placental abruption 16 deaths and placental dysfunction or insufficiency 11 deaths. Other placental abnormalities were responsible for 2 deaths.

Rhesus isoimmunisation accounted for 120 deaths, ABO incompatibility and other non-rhesus isoimmunisation for 6 deaths. Kernicterus not otherwise specified was the cause of death in 4 children. It has not been possible to determine the numbers of live born isoimmunised children in Sheffield throughout the


CHILD MORTALITY BY CAUSE (0-15 years)
Deaths / thousand children
1.5
1.0
0.5
0
Epinatal
S.I.D.S.
Infection
1950
55
60
65
70
75
80
0.5
0
Malformation
Trauma
Tumours
1950
55
60
65
70
75
80
Year of death


CHILD MORTALITY BY CAUSE (0-15 years)
Deaths / thousand live children (log10)
2.0
1.0
0.5
0.2
0.1
0.01
Epinatal
SIDS
Infection
Deaths / thousand children (log10)
0.5
0.2
0.1
0.01
Malformation
Trauma
Tumour
1947
50
55
60
65
70
75
80
Year of death

period studied. Figures were available for the outcome of pregnancies of Sheffield women who attended Welfare Clinics but not for the remainder of Sheffield women (Sheffield 1947-1973).

Haemorrhagic disease of the newborn was responsible for 28 deaths. The mortality rate remained steady between 1947 and 1966, when it fell to zero.

Perinatal infections accounted for 66 deaths; there were 9 with necrotising enterocolitis, 42 with other bacterial infections and 15 with non-bacterial infections. There were 4 deaths with congenital syphilis.

There were 88 deaths where maternal disease was the underlying cause of death. Included in these deaths were 52 with pre-eclamptic toxaemia, 8 with maternal hypertension, 5 with maternal infections, 8 with chronic maternal disease, 1 with hyperemesis and 14 deaths in children of diabetic mothers. These maternal disease deaths showed no significant variation with time.

3.5.2 Congenital malformations

Congenital malformations were the underlying cause of death in 1402 (20%) of the 7035 deaths.

Malformations of the central nervous system accounted for 390 deaths, 337 being neural tube defects (254 of which were spina bifida, 51 anencephaly and 32 encephalocoele). There were 21 deaths with microcephaly and 19 deaths with neuromuscular dystrophy, including 12 with Werdnig-Hoffman disease.

There were 455 deaths with congenital heart disease including 153 with unspecified congenital heart disease. There were 152 bulbus cordis and septal anomalies including: 21 with common truncus, 48 transposition of the great vessels, 33 Fallot's tetralogy, 5 common ventricle, 29 ventricular septal defect, 7 endocardial cushion defect and 9 secundum atrial septal defect. There were another 149 deaths in the group of congenital heart diseases considered to be acquired after formation of the heart and great vessels, including: 19 with pulmonary valve stenosis or atresia, 15 tricuspid stenosis or atresia, 22 aortic valve stenosis or atresia, 6 congenital mitral stenosis, 31 hypoplastic left heart, 8 patent ductus arteriosus, 28 coarctation of the aorta, 6 pulmonary artery anomalies and 14 total anomalous pulmonary venous drainage.

Malformations of the respiratory tract accounted for 95 deaths, including 3 with choanal atresia, 33 trache-oesophageal fistula, 6 cystic or honeycomb lung, 3 pulmonary hypoplasia with no renal malformation, 6 cleft palate and 39 with diaphragmatic anomalies.

Anomalies of the alimentary system were responsible for 92 deaths. These deaths include 12 deaths from exomphalos, 21 atresias of the small bowel, 14 large bowel anomalies (including anus and cloaca), 14 Hirschsprung's disease, 12 malrotations or abnormal mesentery, and 8 with biliary atresia. There were 8 deaths with hypertrophic pyloric stenosis.

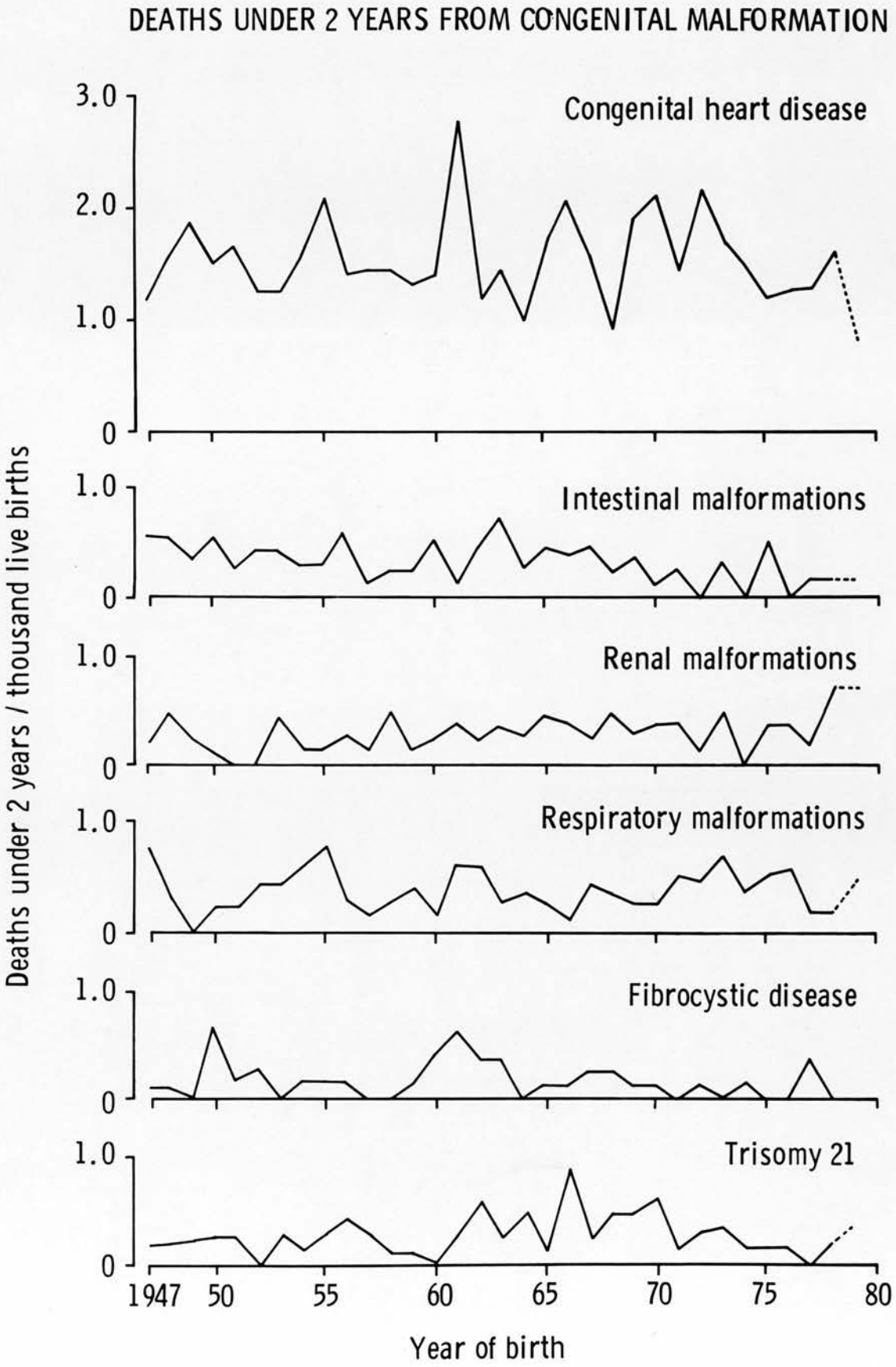
There were 77 deaths with renal anomalies including 37 with renal agenesis, 12 cystic kidneys of all types, 7 hydronephrosis or hydroureter, 10 urethral abnormalities including

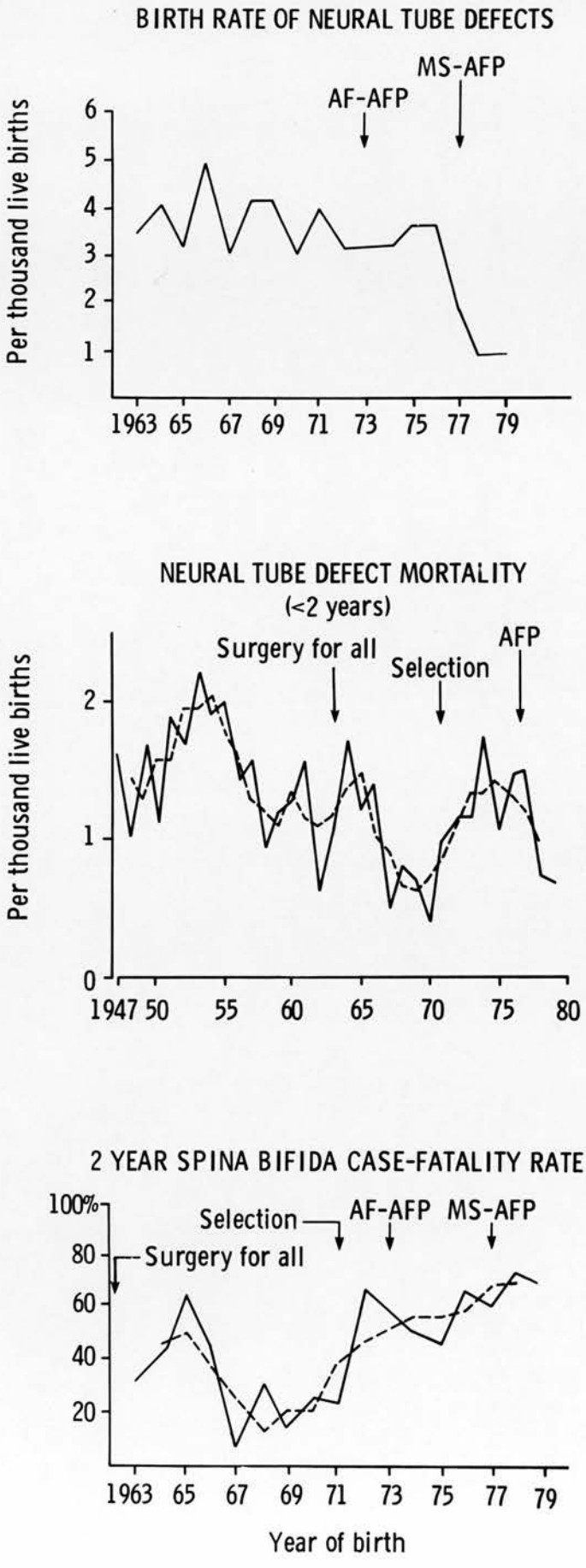
posterior urethral valves and 3 with bladder extrophy.

There were 79 deaths with Down's syndrome and 30 with other known chromosomal defects. There were 62 deaths registered as 'multiple congenital malformation' and 2 pairs of conjoined twins sharing vital organs.

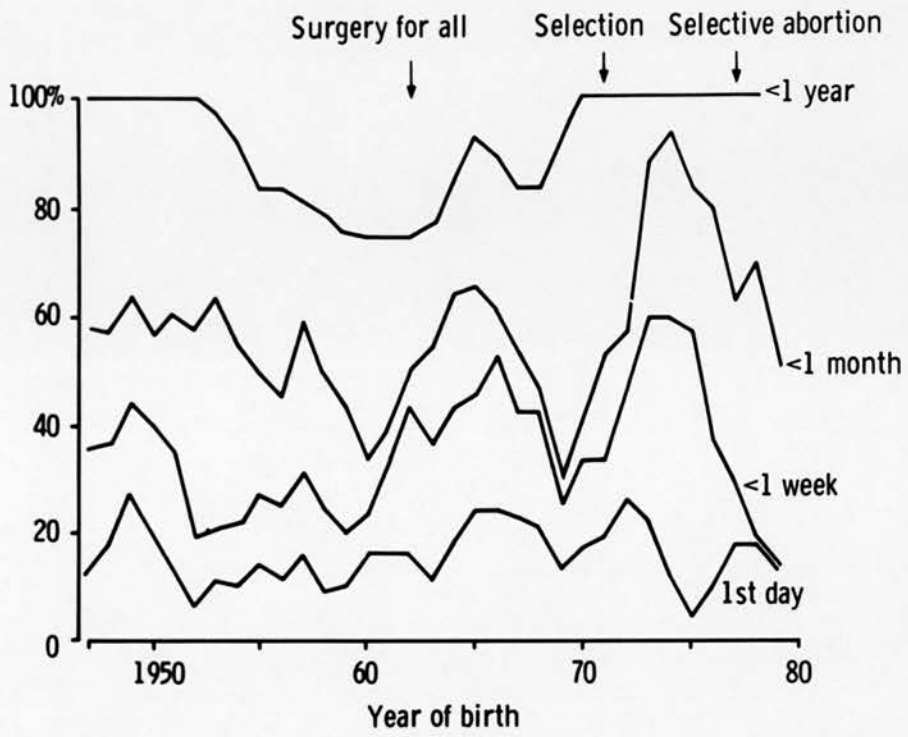
There were 61 deaths with fibrocystic disease, 15 deaths with inborn errors of carbohydrate metabolism and 11 with other inborn errors of metabolism. There were 12 deaths with achondroplasia, 6 deaths with osteodystrophies and 3 deaths with other limb anomalies. There were no deaths with obvious stigmata of thalidomide teratogenesis, although some might have been certified as multiple congenital anomaly and not had a necropsy. There were 5 deaths with adrenal hypoplasia and 2 deaths with other congenital endocrine anomalies.

The distribution of deaths under two completed years with a congenital malformation is shown in Fig. 31 by year of birth, standardised to the number of live births in each year. Fig. 32 shows the annual proportions of Sheffield children born with a neural tube defect and the fatal incidence of neural tube defects under two years (per thousand live births and per hundred affected children). Three-year moving means are shown as broken lines for these latter curves. The proportionate distributions of spina bifida deaths and survivors by age are shown in Fig. 33. The congenital anomaly register was found to be of limited value in identifying malformations which were not detectable at birth because of the voluntary notification. Therefore birth and case fatality rates for other malformations have not been shown.

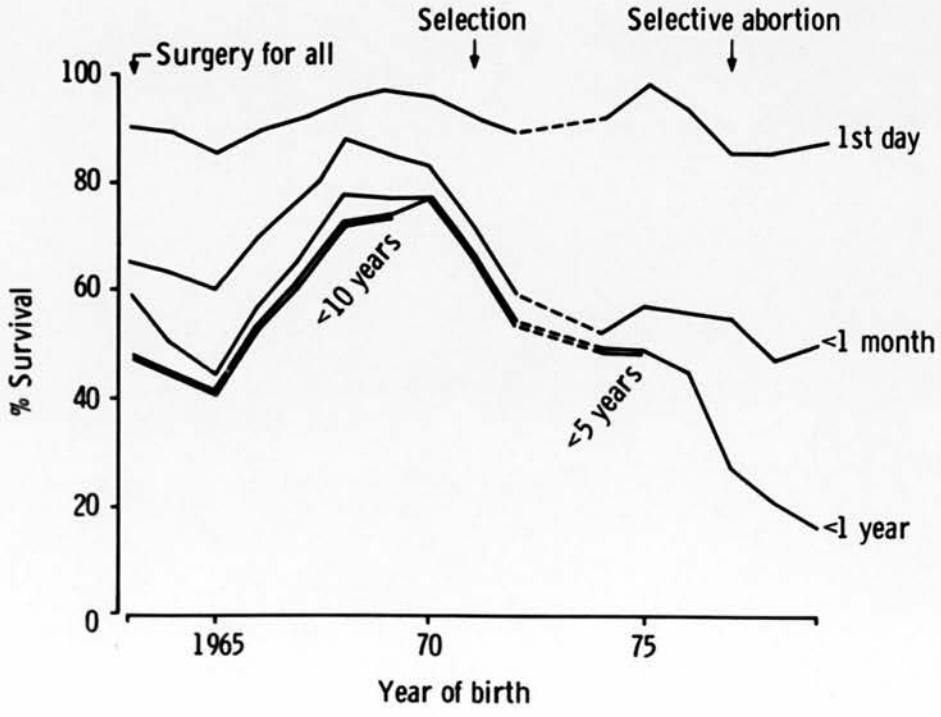

DEATHS UNDER 2 YEARS FROM CONGENITAL MALFORMATION
Congenital heart disease
3.0
2.0
1.0
0
Intestinal malformations
1.0
0
Renal malformations
1.0
0
Respiratory malformations
1.0
0
Fibrocystic disease
1.0
0
Trisomy 21
1.0
0
1947
50
55
60
65
70
75
80
Year of birth
Deaths under 2 years / thousand live births


BIRTH RATE OF NEURAL TUBE DEFECTS
MS-AFP
AF-AFP
Per thousand live births
6
5
4
3
2
1
1963 65 67 69 71 73 75 77 79
NEURAL TUBE DEFECT MORTALITY
(<2 years)
Surgery for all
Selection
AFP
Per thousand live births
2
1
0
1947 50 55 60 65 70 75 80
2 YEAR SPINA BIFIDA CASE-FATALITY RATE
100%
Selection
AF-AFP
MS-AFP
Surgery for all
80
60
40
20
1963 65 67 69 71 73 75 77 79
Year of birth

PROPORTIONATE DISTRIBUTION OF SPINA BIFIDA DEATHS BY AGE
(100% = up to 15 years)



SURVIVORS WITH SPINA BIFIDA



3.5.3 The infection deaths

Children dying with an infection who had any other disease (e.g. a congenital malformation, cystic fibrosis or cerebral palsy) were coded into those other disease groups. Thus the 1060 children dying with infectious diseases were otherwise normal children.

There were 93 deaths with tuberculosis (TB), 53 with TB meningitis, 27 miliary TB, 6 pulmonary TB, 2 intestinal TB, 2 bone or joint infection and 3 with renal TB.

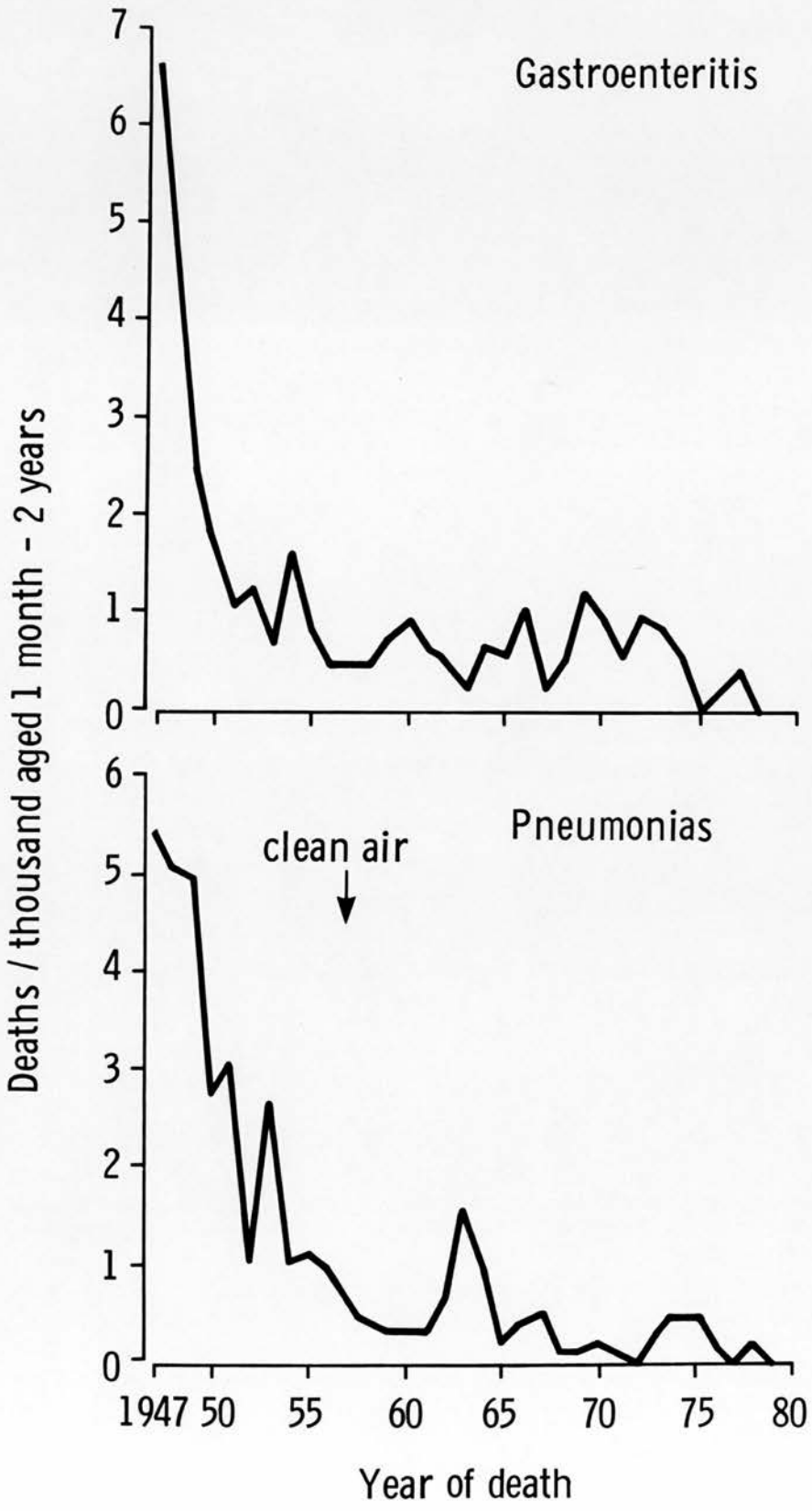
There were 123 deaths with non-tubercular infections of the central nervous system including: 29 polio, 25 encephalitis, 58 bacterial meningitis, 9 cerebral or spinal abscess and 2 with acute infectious polyneuritis. There were 61 deaths with meningococcal septicaemia.

There were 317 deaths with infections of the intestinal tract, 285 being indexed as gastroenteritis and not otherwise specified. No supportive histological or bacteriological evidence of gastroenteritis was uncovered by necropsy in these deaths. There were 10 deaths with typhoid or paratyphoid fever, 18 deaths with appendicitis and 4 with peritonitis.

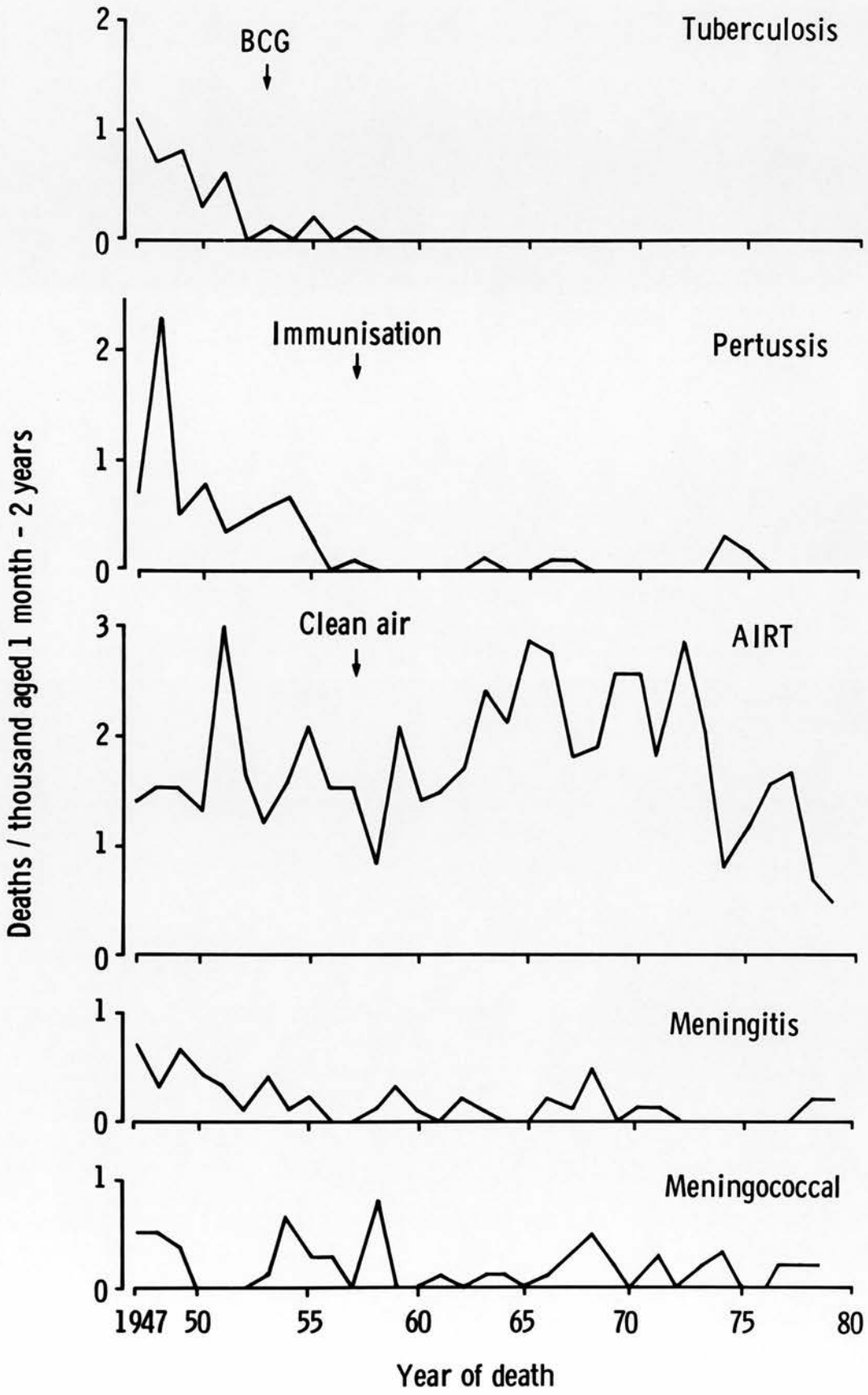
There were 526 deaths with acute upper respiratory tract infections, and 375 deaths with lower respiratory tract infections; 258 of the respiratory deaths were due to unspecified pneumonia, 7 to influenza, 2 to diphtheria, 67 to pertussis, 11 to bronchiectasis, 29 to empyemas or pulmonary abscesses and 1 to epiglottitis.

There were 152 other infection deaths including: pericarditis 1, myocarditis 4, bacterial endocarditis 4, tetanus 1,

DEATHS AMONG CHILDREN AGED 1 MONTH - 2 YEARS



DEATHS BETWEEN 1 MONTH & 2 YEARS FROM INFECTIONS



other septicaemias 29, pyelonephritis 8, osteomyelitis 2, hepatitis 11, measles 29 and chickenpox 2.

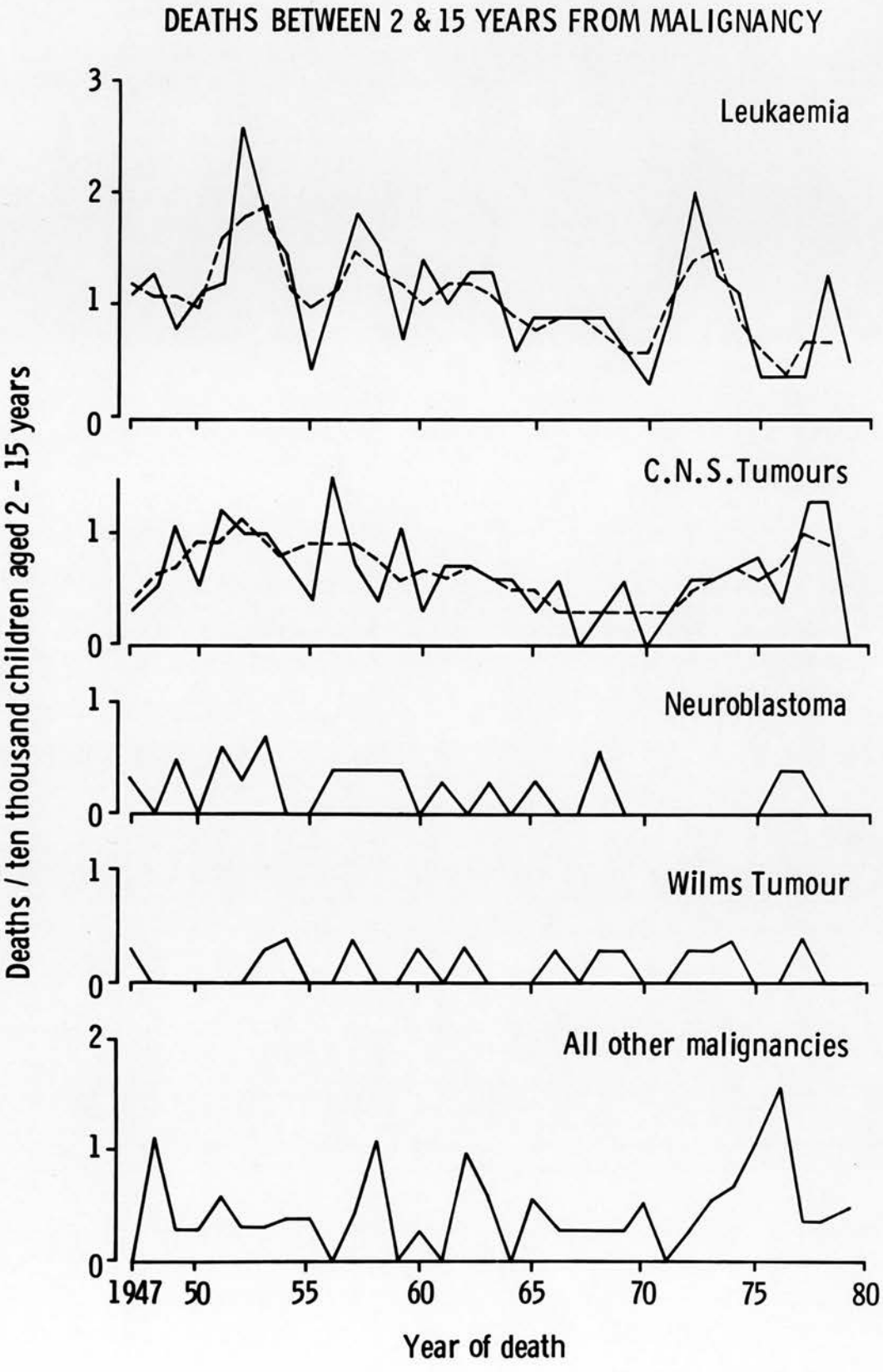
The distribution with time of the infection deaths among children aged more than 1 month and less than 2 completed years is shown in Fig. 34.

3.5.4 Sudden Infant Death Syndrome (SIDS)

Among the 4013 Sheffield child deaths which occurred after the first week of life between 1947 and 1979, there were 2724 where an antemortem history was available; 1737 (64%) of these children died suddenly. According to the clinical definition of SIDS (section 1.5.4(a)) these children might be considered as SIDS. However, 647 of these children had a previous history of ill health or obvious gross pathology to account for their deaths and 432 died violent deaths. This left 658 deaths which were defined as SIDS in the present study. There were an additional 93 deaths with incomplete histories but no significant necropsy findings which were also indexed as SIDS.

3.5.5 Malignancies

There were 293 deaths from malignant disease: 118 leukaemias (54 unspecified, 42 acute lymphoblastic, 19 acute monoblastic, 1 acute monocytic, 2 chronic myeloid), 19 Wilms' tumour, 69 CNS tumours, 25 neuroblastomata and 62 other malignancies including 12 lymphosarcomata, 6 histiocytoses, 18 unspecified sarcomata, 4 embryomata and 4 teratomata. The changing distribution with time of these deaths among children over 2 years of age is shown in Fig. 35. The broken lines on the leukaemia and CNS tumour curves show the 3 year moving mean.


DEATHS BETWEEN 2 & 15 YEARS FROM MALIGNANCY
Leukaemia
C.N.S.Tumours
Neuroblastoma
Wilms Tumour
All other malignancies
Deaths / ten thousand children aged 2 - 15 years
0
1
2
3
1947
50
55
60
65
70
75
80
Year of death

3.5.6 Acquired diseases

There were 121 deaths with cerebral palsy, 15 with epilepsy, 32 cerebral degenerations and 64 hydrocephalus (7 post-meningitic). Five children died with cerebral thrombosis, and 25 with cerebral haemorrhage. The time trend of cerebral palsy deaths is shown in Fig. 36.

Twenty-six children died with rheumatic heart disease and 14 children with cardiomyopathy. There were 24 deaths with asthma. The distribution of these deaths with time is shown in Fig. 36.

There were 32 deaths with intestinal obstruction (hernia, volvulus and intussusception), 5 with other intestinal diseases and 35 with malabsorption.

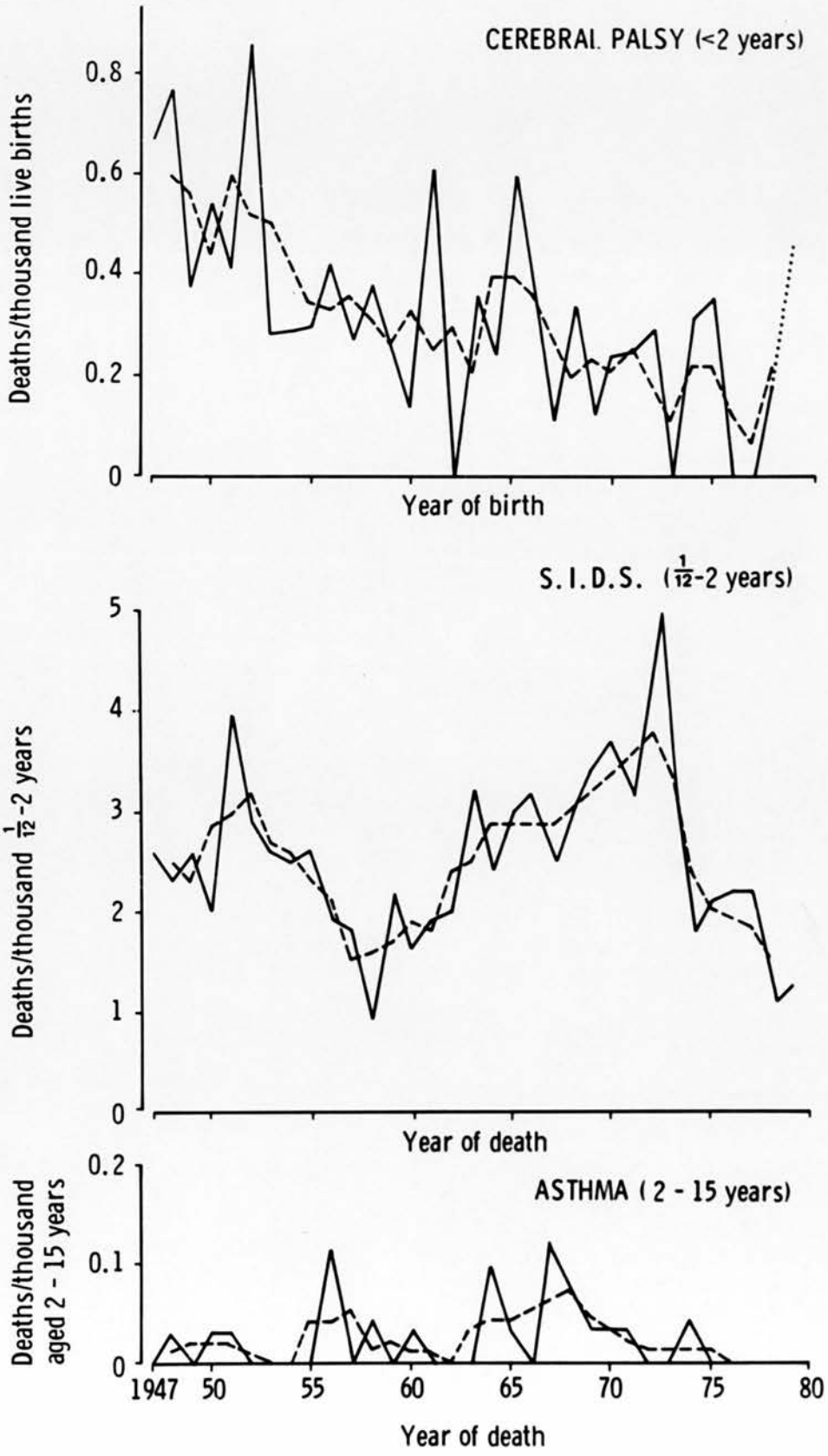
There were 27 deaths with chronic nephritis, 6 glomerulonephritis, 3 with nephrotic syndrome and 3 nephrocalcinosis.

There were 2 deaths with polyarteritis, 1 with Still's disease and 1 with dermatomyositis. There were 4 deaths with diabetes and 3 with other endocrine disorders.

3.5.7 Unnatural deaths

Unnatural causes accounted for 471 of the deaths, 76% being over 1 year of age and 52% over 5 years (Table XIV).

There were 238 accidents involving road traffic: 25 specified as cycle accidents and 169 specified as pedestrian accidents. Following six of these pedestrian deaths the drivers were convicted of dangerous driving. In most of the other accidents the inquest recorded accidental deaths as evidence was presented that the children ran into the path of the vehicle.

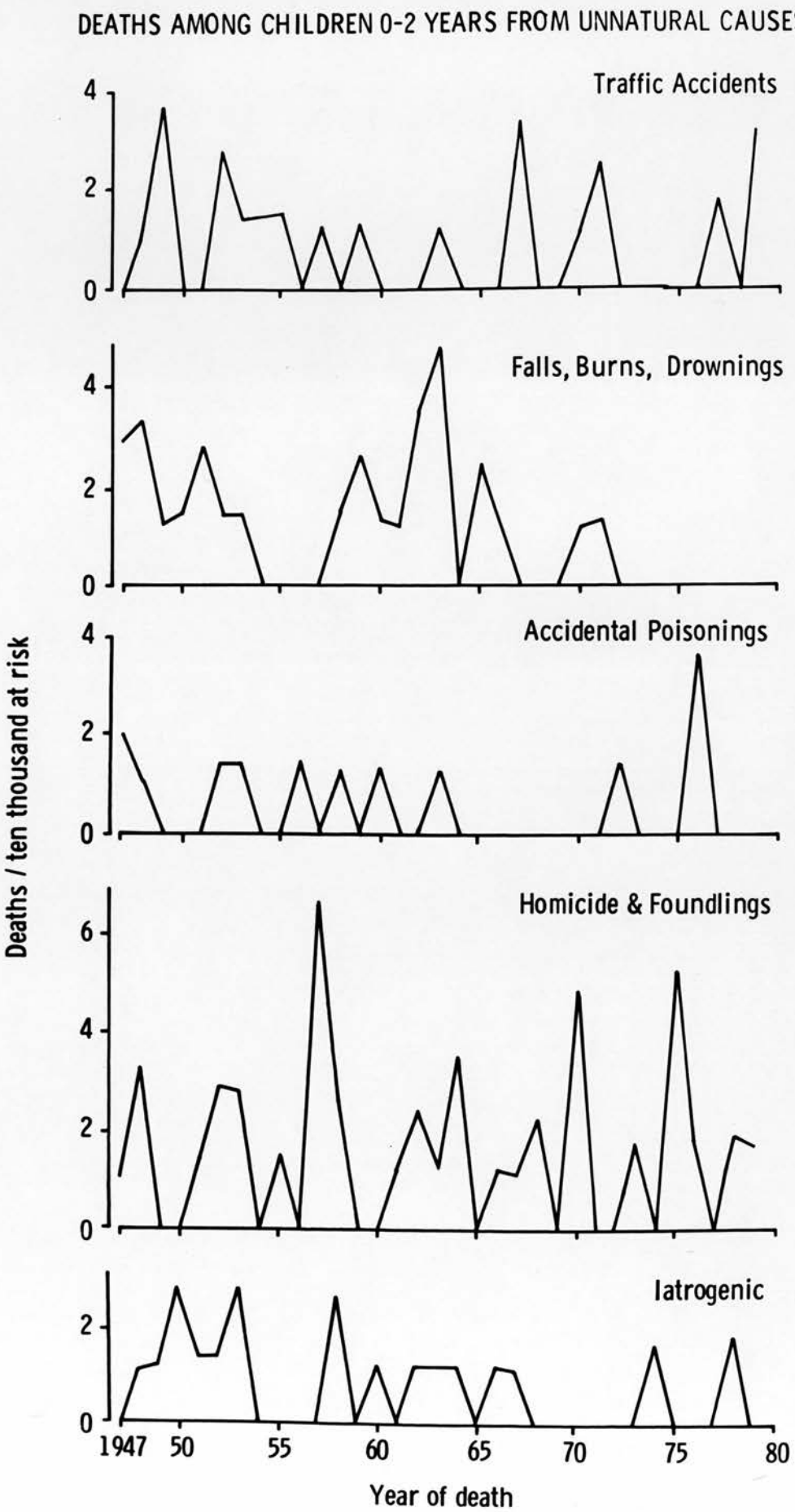

CEREBRAL PALSY (<2 years)
Deaths/thousand live births
0.8
0.6
0.4
0.2
0
Year of birth
S.I.D.S. (1/12-2 years)
Deaths/thousand 1/12-2 years
5
4
3
2
1
0
Year of death
ASTHMA (2 - 15 years)
Deaths/thousand aged 2 - 15 years
0.2
0.1
0
1947
50
55
60
65
70
75
80
Year of death

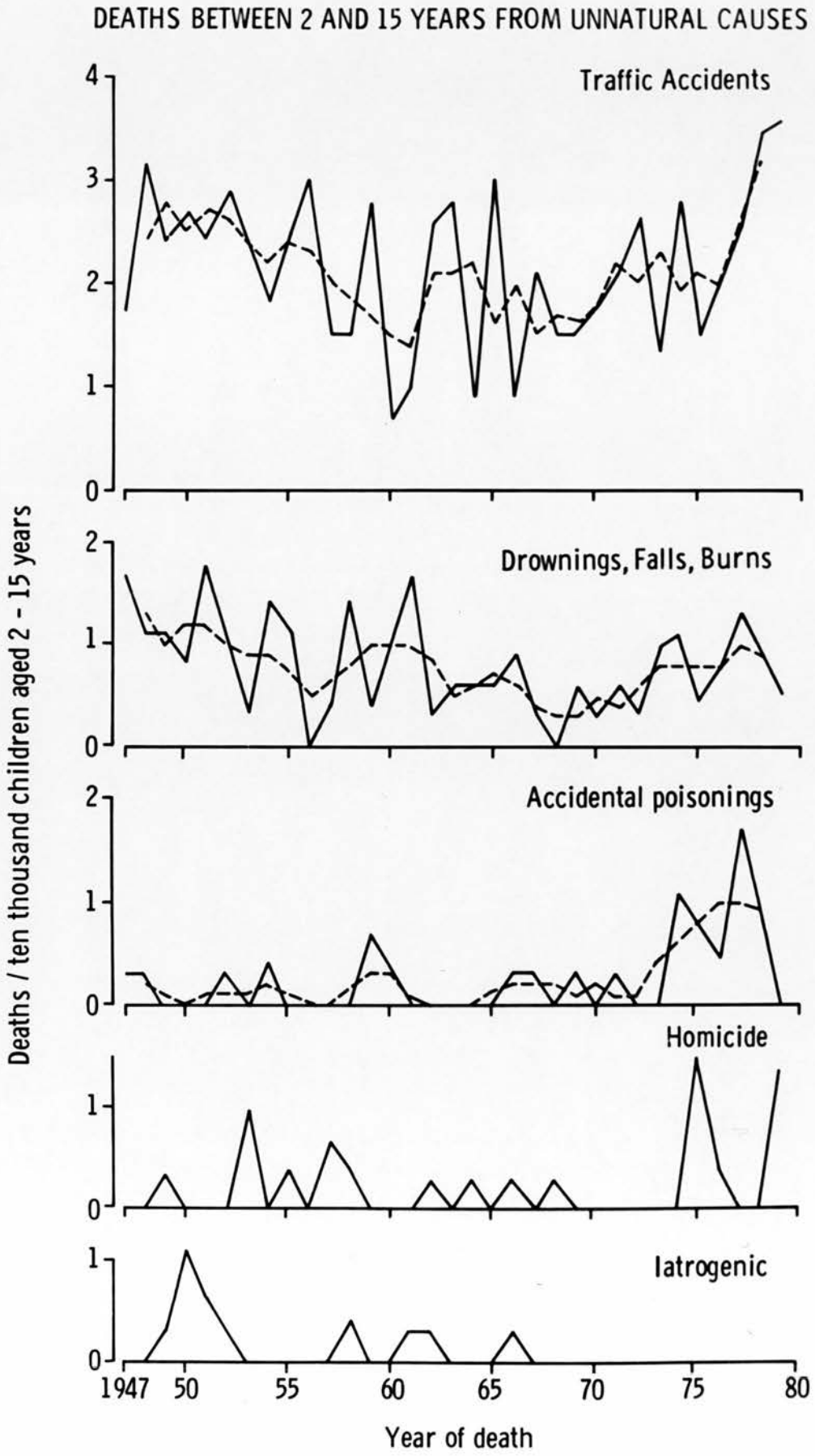
There were 9 deaths from accidental drug poisoning, 20 gas poisonings and 5 electrocutions. There were 30 deaths following medical misadventure (anaesthetic accidents, post-tonsillectomy haemorrhage, other surgical accidents and one accidental drug overdose). Deaths following possible overdose of cytotoxic drugs were indexed under the respective tumours and not considered here.

There were 13 deaths following falls, 45 due to burns or scalds, 45 drownings and 13 deaths following inhalation of a foreign body (other than terminal inhalation). Eight children died by hanging (three toddler accidents and five suicides). There were 2 drug suicides and one death with anorexia nervosa.

There were 55 violent deaths where a second party was involved: 5 proven battering deaths, 2 ritual murders, 2 strangulations, 4 suffocations (one attributed to a 5 year old sibling), 5 convictions of neglect with intent to kill and 14 other homicides. There were 23 children who died because of inattention at birth (foundlings).

The time trend of deaths from unnatural causes among children under two years of age is shown in Fig. 37a and over two years in Fig. 37b. The three-year moving mean of some of these trends is shown as a broken line. The distribution of the numbers of foundlings in three year periods is shown in Table VII. The distribution of these unnatural deaths by age and cause is shown in Table XIV.


DEATHS AMONG CHILDREN 0-2 YEARS FROM UNNATURAL CAUSES
Traffic Accidents
Falls, Burns, Drownings
Accidental Poisonings
Homicide & Foundlings
Iatrogenic
Deaths / ten thousand at risk
0
2
4
6
1947
50
55
60
65
70
75
80
Year of death

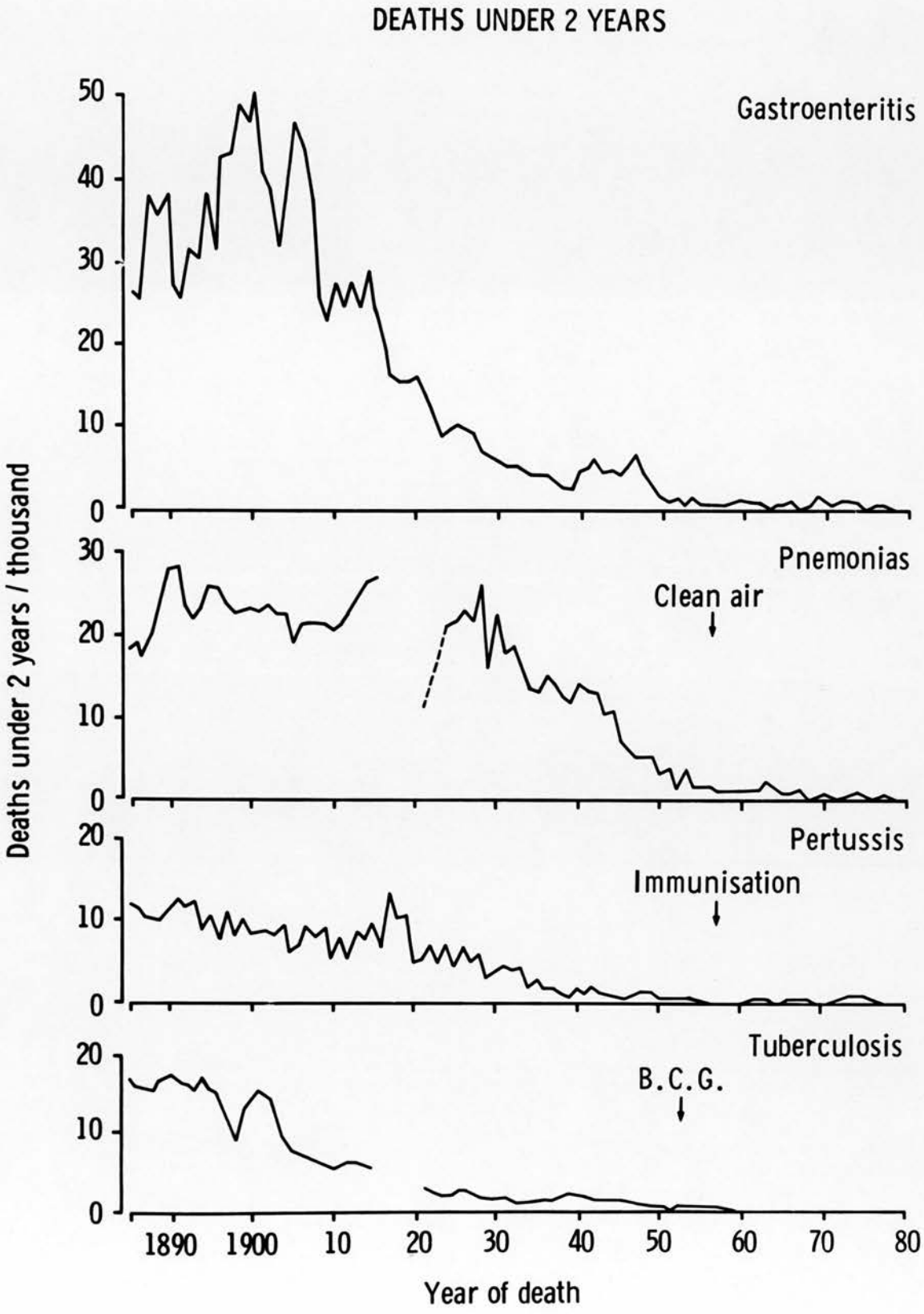

DEATHS BETWEEN 2 AND 15 YEARS FROM UNNATURAL CAUSES
Traffic Accidents
Drownings, Falls, Burns
Accidental poisonings
Homicide
Iatrogenic
Deaths / ten thousand children aged 2 - 15 years
Year of death
1947
50
55
60
65
70
75
80

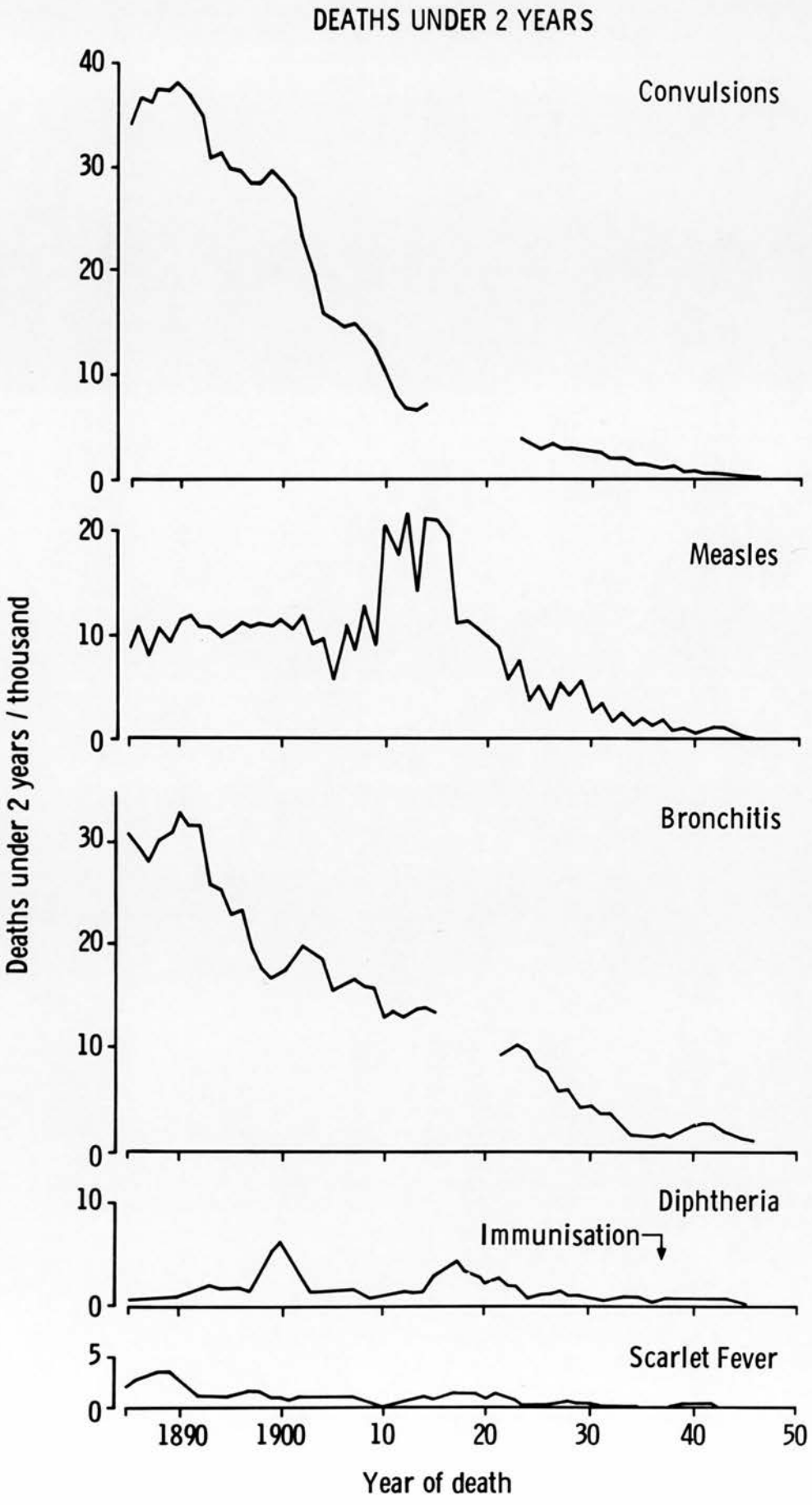
3.6 Mortality trends 1874-1946

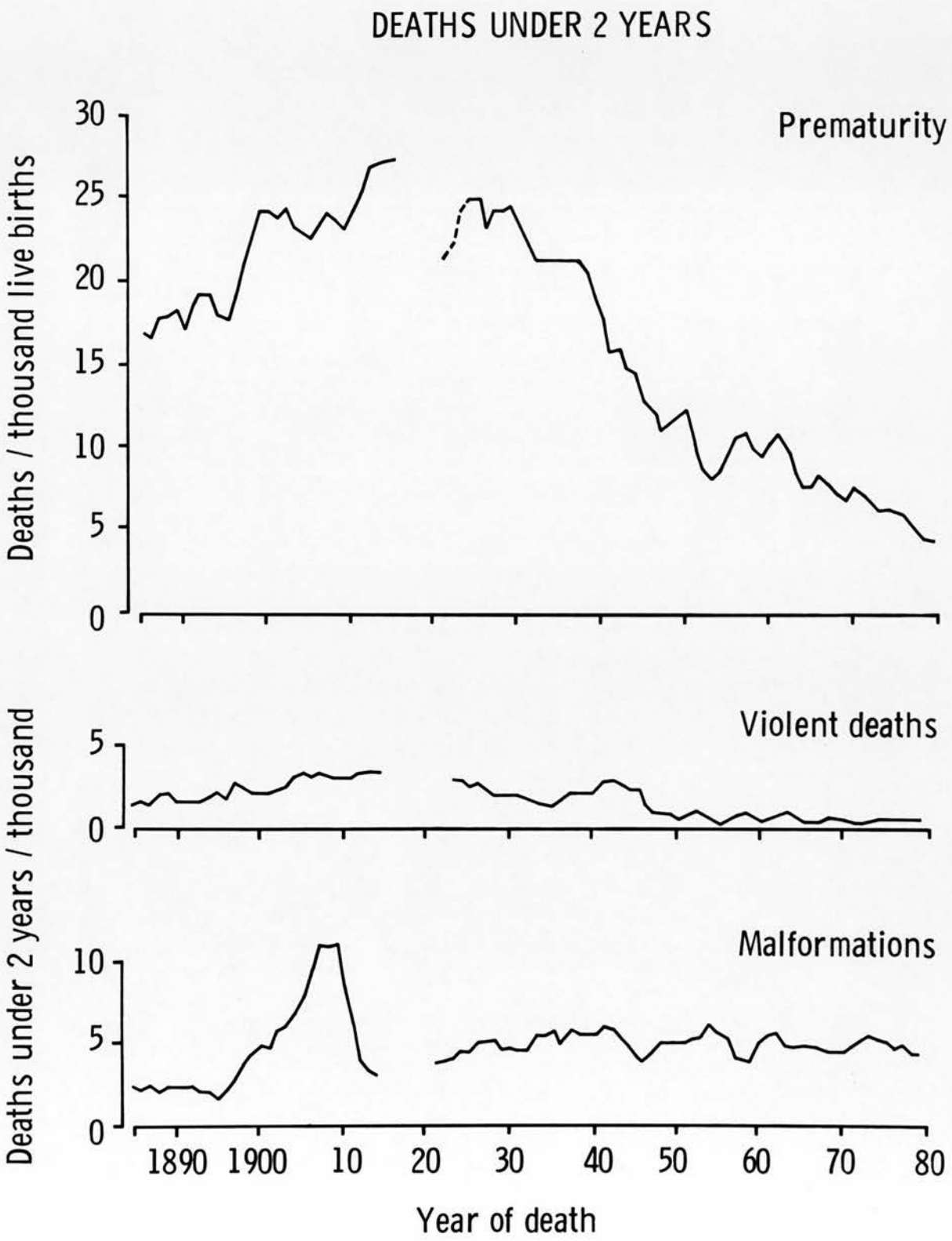
The annual totals of births, deaths by age and child deaths from certain causes were obtained from the MOH reports (Sheffield 1874-1973). These data were address-standardised but their validity could not be verified. Changing classifications of disease were overcome as far as possible by reference to old medical dictionaries and textbooks. Thus tubercular diseases were found under scrofula, tabes mesenterica and phthisis as well as tubercular hydrocephalus and other tubercular diseases.

In the earlier years there were up to 200 infant deaths indexed as "convulsions" which were separate and in addition to deaths from epilepsy or meningitis. There were four such deaths in the major (1947-1979) study aged 4, 8, 11 and 24 months. Two died at home, two in hospital. Necropsies had been done on the hospital deaths. An adequate explanation for these deaths could not be found and they were indexed as sudden infant death syndrome.

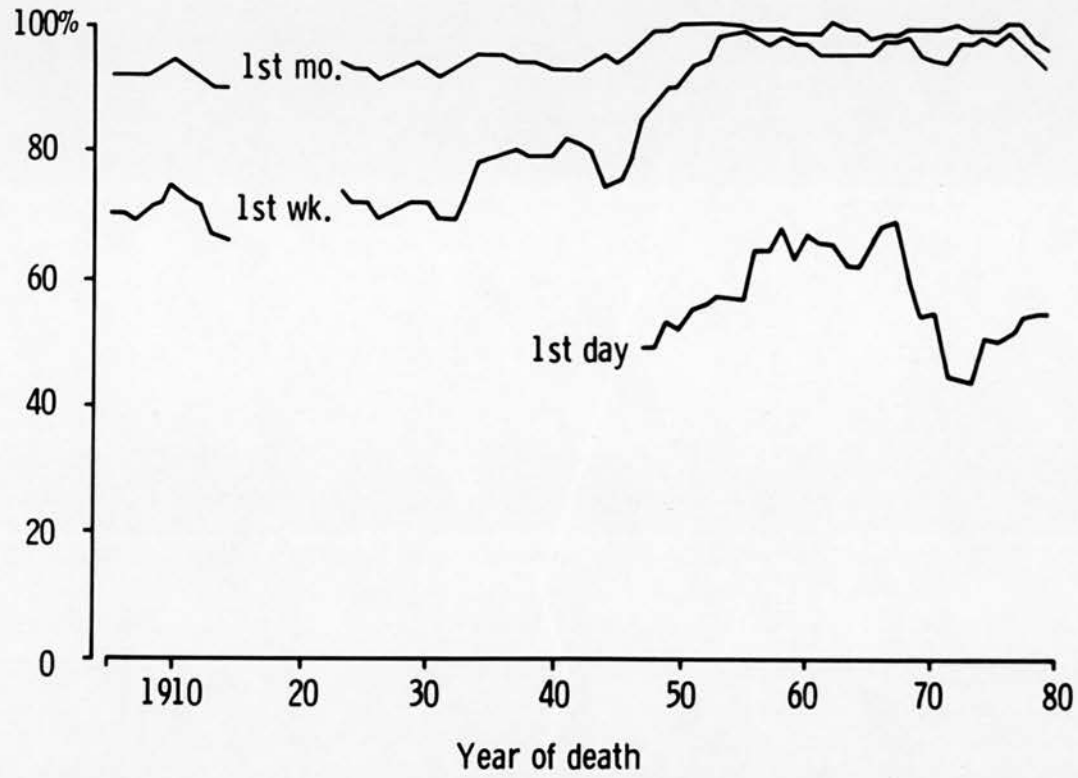
Between 1914 and 1923 the data collected by the MOH were severely limited. Available data on the distribution of disease-specific mortality rates for certain infections are shown in Figs. 38 and 39, mortality rates for prematurity, congenital malformations and violence are shown in Fig. 40. The proportionate distribution by age of infant deaths from prematurity and malformations are shown from 1905 (the earliest record) in Fig. 41. Crude infant, neonatal and first week mortality rates are shown in Fig. 42.


DEATHS UNDER 2 YEARS
Gastroenteritis
Pnemonias
Clean air
Pertussis
Immunisation
Tuberculosis
B.C.G.
Deaths under 2 years / thousand
Year of death
1890
1900
10
20
30
40
50
60
70
80

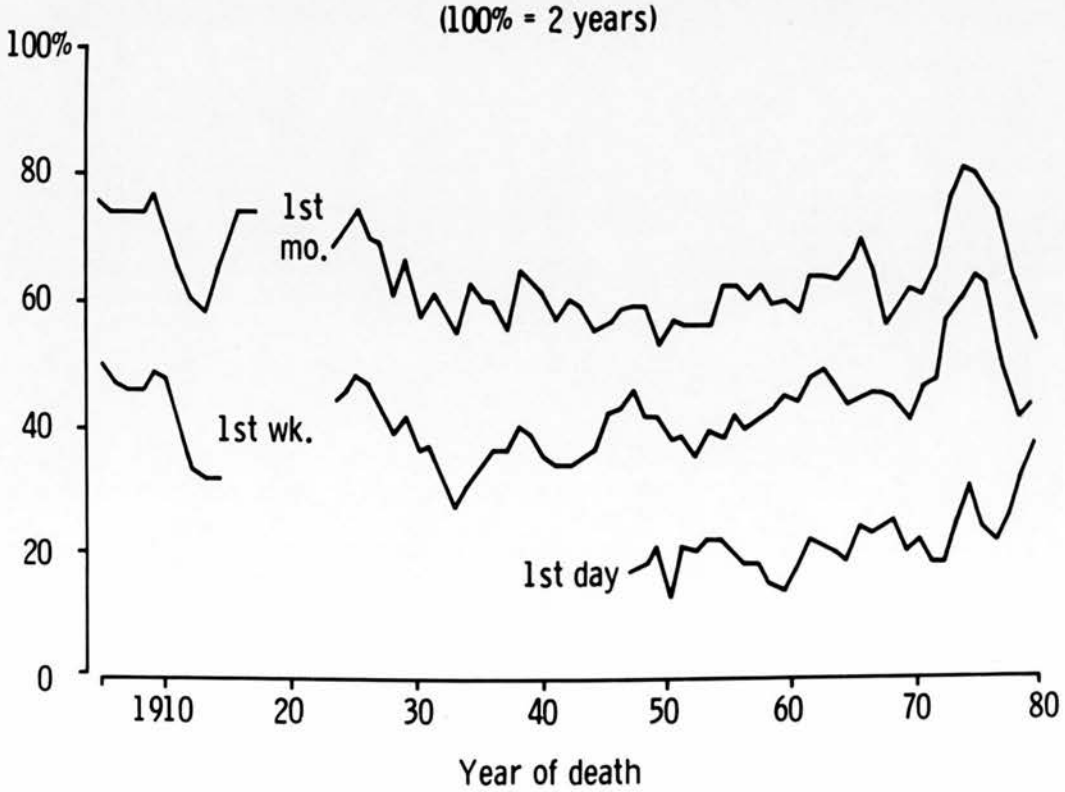

DEATHS UNDER 2 YEARS
Convulsions
Measles
Bronchitis
Diphtheria
Immunisation
Scarlet Fever
Deaths under 2 years / thousand
Year of death
1890
1900
10
20
30
40
50

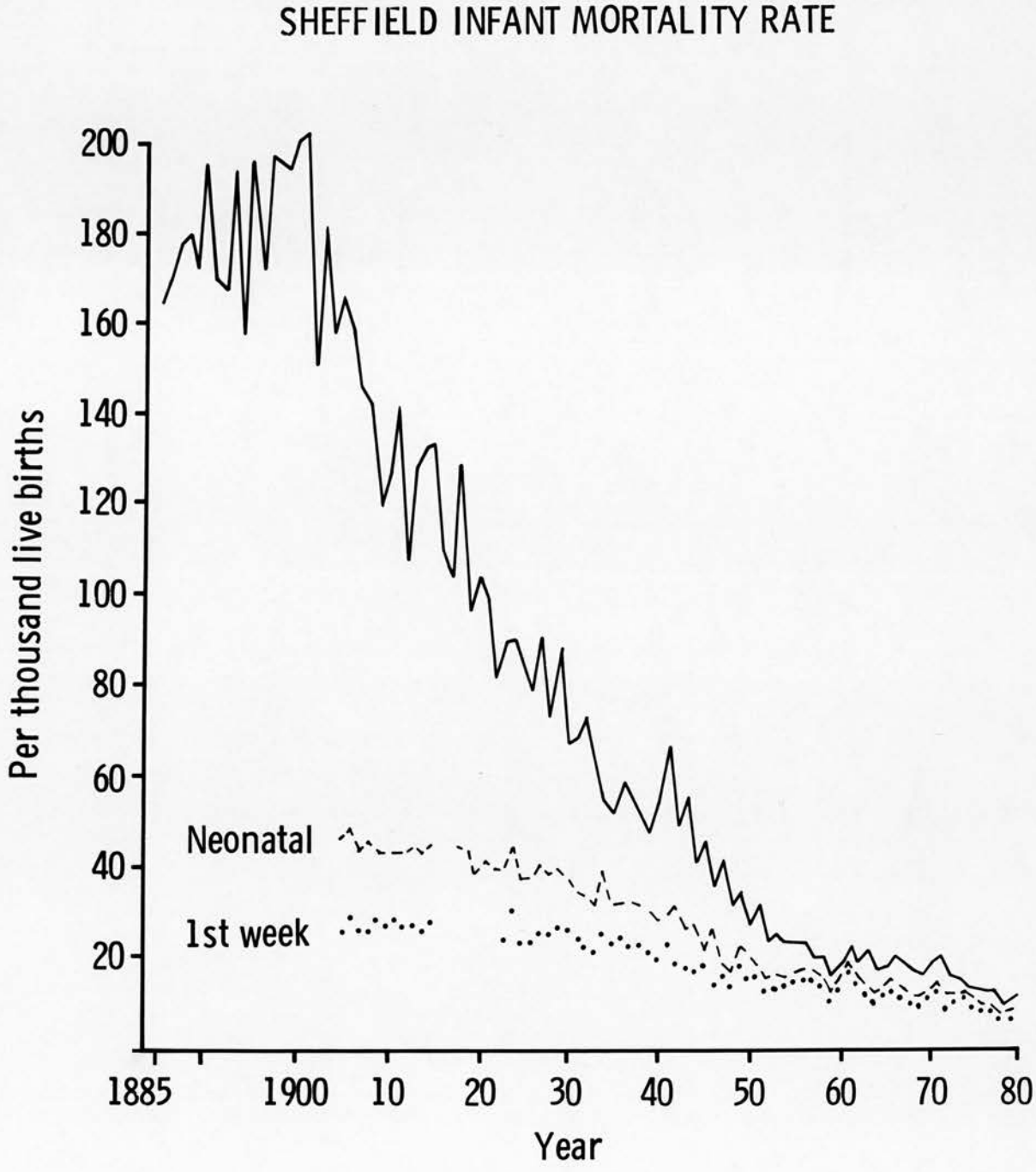

DEATHS UNDER 2 YEARS
Prematurity
Deaths / thousand live births
30
25
20
15
10
5
0
Violent deaths
5
0
Malformations
10
5
0
Deaths under 2 years / thousand
1890
1900
10
20
30
40
50
60
70
80
Year of death

PROPORTIONATE DISTRIBUTION OF PREMATURITY DEATHS BY AGE
(100% = 2 years)



PROPORTIONATE DISTRIBUTION OF MALFORMATION DEATHS BY AGE
(100% = 2 years)

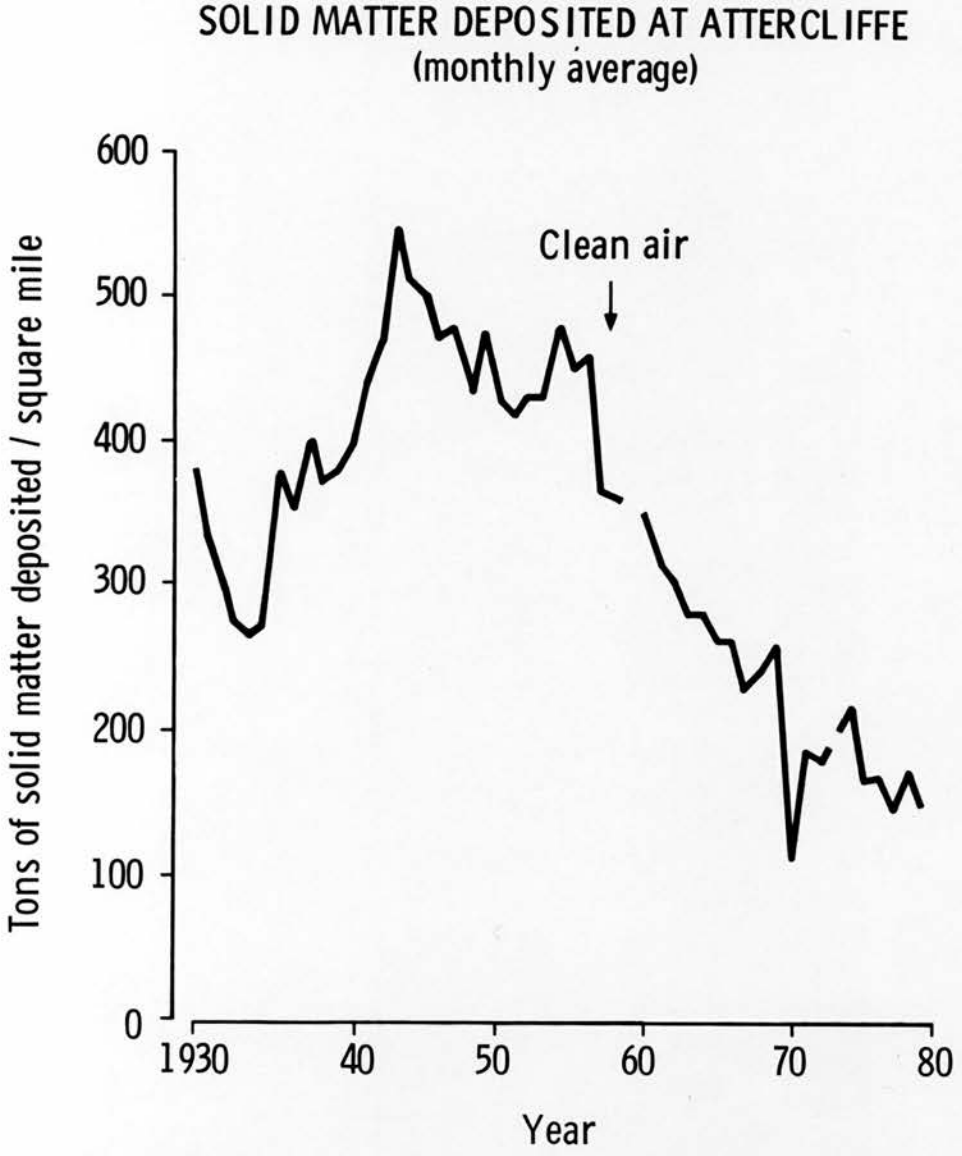



SHEFFIELD INFANT MORTALITY RATE
Per thousand live births
200
180
160
140
120
100
80
60
40
20
Neonatal
1st week
1885
1900
10
20
30
40
50
60
70
80
Year

3.7 Public Health measures

Standardised records of atmospheric soot pollution have been kept since 1929 and for sulphur dioxide since 1946. The records of soot and sulphur dioxide concentrations at the Attercliffe sampling station are shown in Fig. 43. The Attercliffe gauge is situated in the heavy industrial area downwind of the middle of the city and the railway steaming-up yards. These figures are probably worse than those for any other area of the city and most areas of Sheffield now have much lower levels than those shown in Fig. 43.

The proportion of Sheffield's housing stock built since 1885 is shown in Fig. 44 together with a housing index devised for this study which shows the numbers of new houses completed each year per thousand residents. This standardises the number of new houses to the growing population and shows the peak periods of slum clearance. The proportion of houses served by privy middens is shown in Fig. 45. As the midden clearance began in the most populated areas, the fall in the proportion of persons using middens will have been even sharper.


SOLID MATTER DEPOSITED AT ATTERCLIFFE
(monthly average)
Tons of solid matter deposited / square mile
600
500
400
300
200
100
0
Clean air
1930
40
50
60
70
80
Year

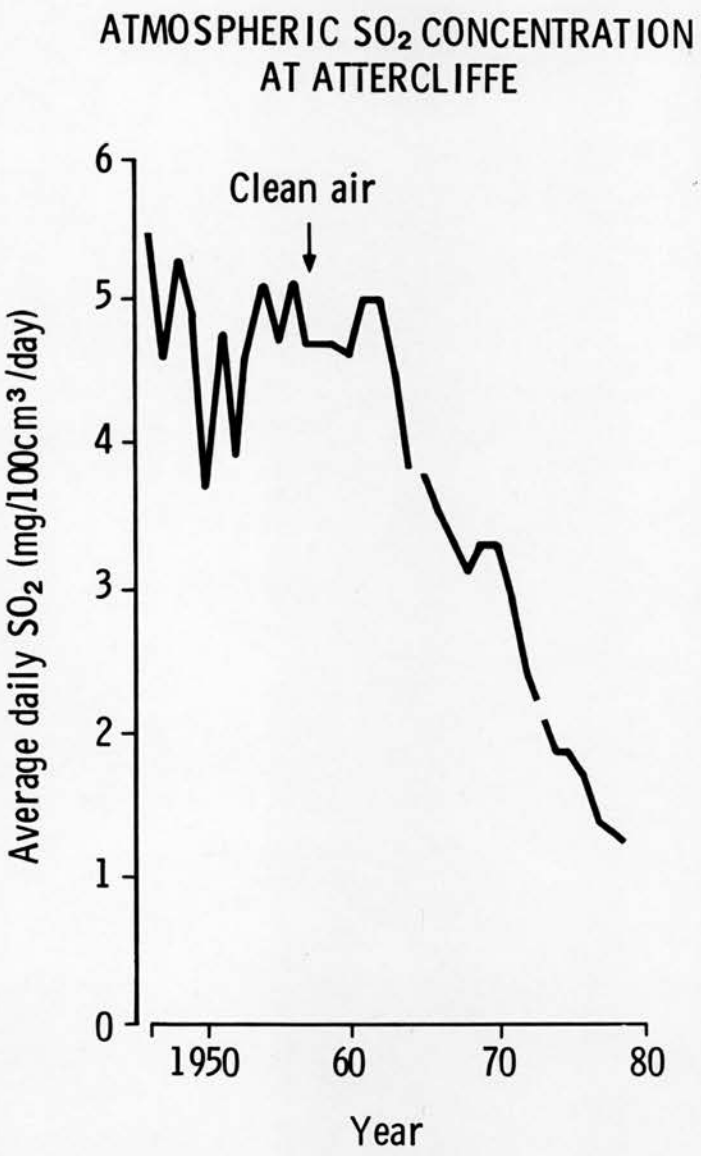
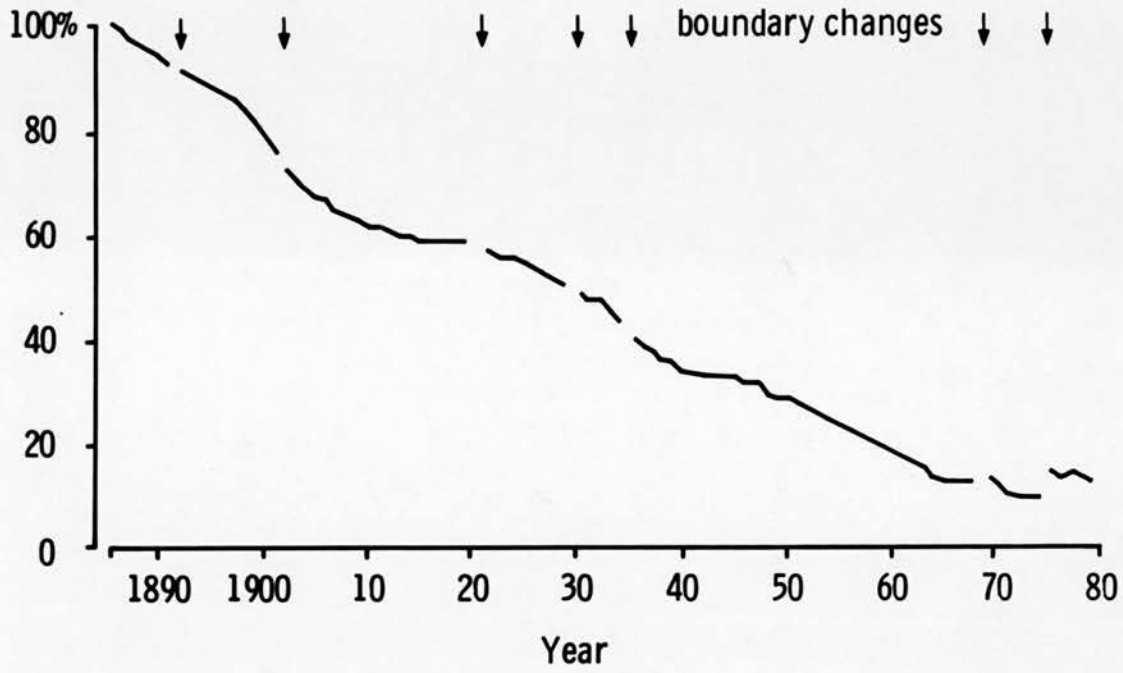
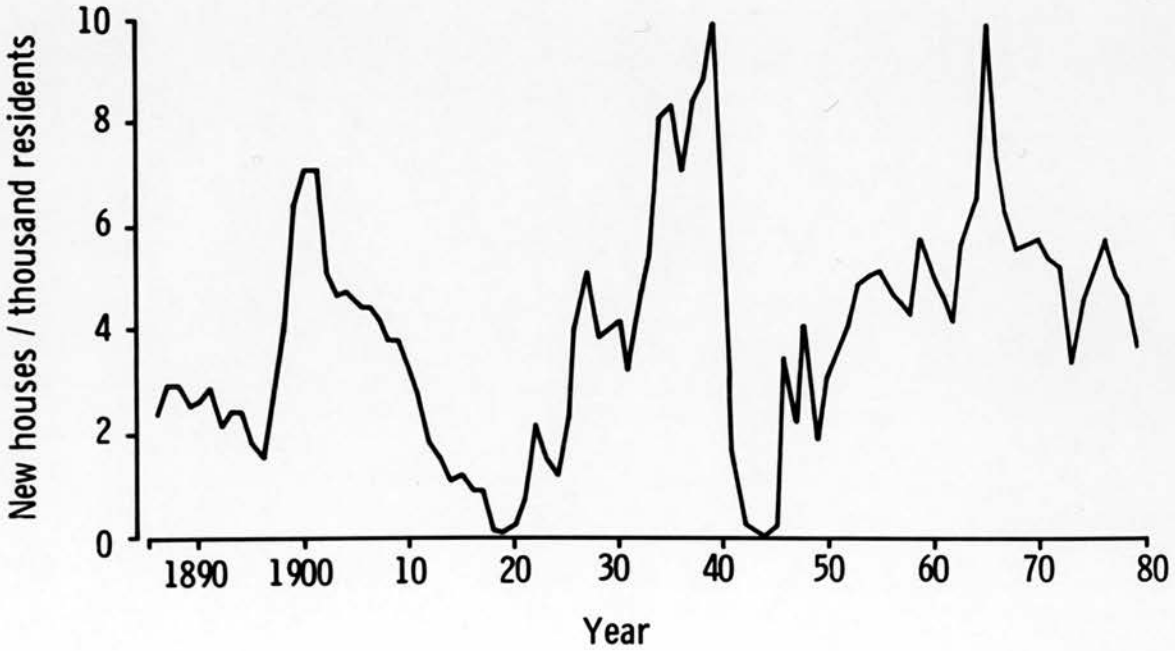

ATMOSPHERIC SO_2 CONCENTRATION
AT ATTERCLIFFE
Average daily SO_2 (mg/100cm³/day)
6
5
4
3
2
1
0
Clean air
1950
60
70
80
Year

Fig. 43

PROPORTION OF SHEFFIELD HOUSING STOCK BUILT BEFORE 1885



GROWTH AND REPLACEMENT OF SHEFFIELD HOUSING STOCK



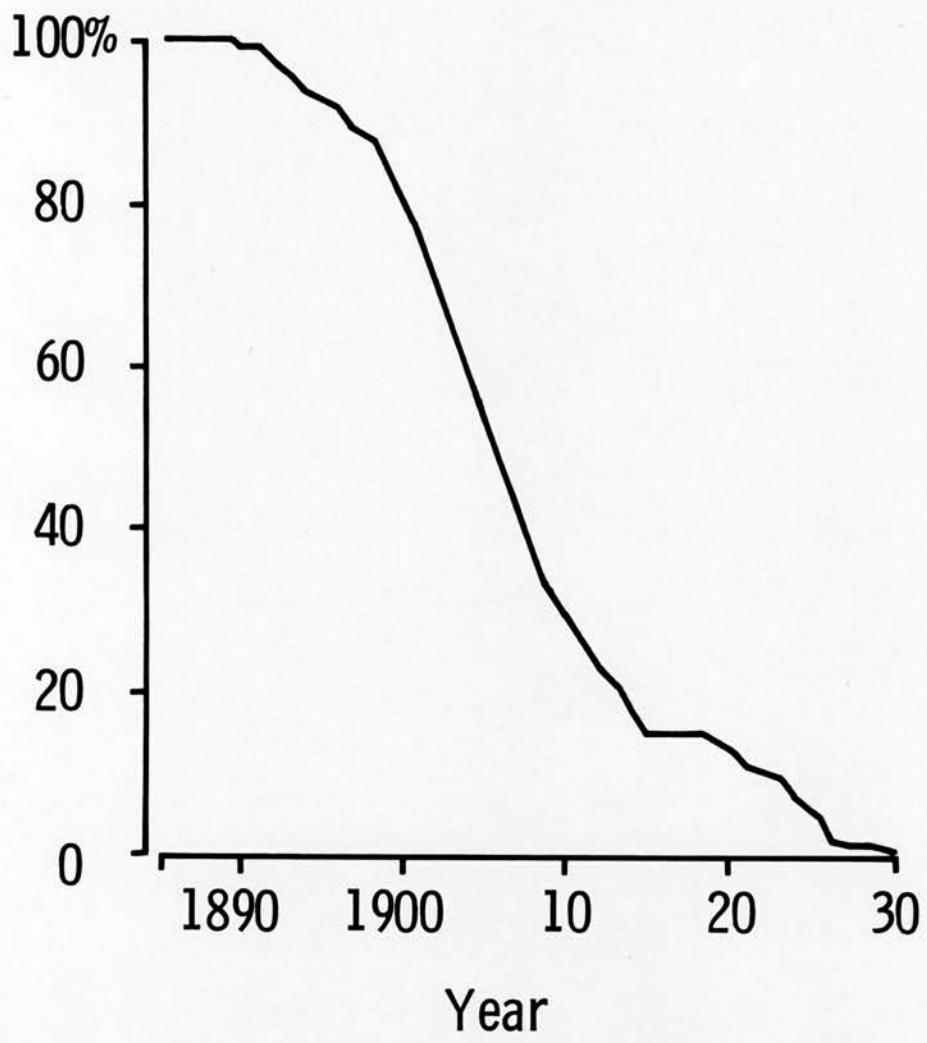

% OF SHEFFIELD HOUSES WITH PRIVY MIDDENS
100%
80
60
40
20
0
1890
1900
10
20
30
Year

Fig. 45

SECTION 4

DISCUSSION

4.1 Methodology

4.1.1 The study population

The population for the major study (1947-1979) was identified by examination of every death certificate issued in Sheffield in these years. This was necessary because some of the central computer records of death certificates do not contain the home address. The causes of death in these records were coded according to the classifications of causes of death current at the time of death. These classifications have been revised a number of times during the period studied. Therefore time-trend studies which are based on computer records might show changes in the incidence of certain fatal diseases which are due to a change in classification. For example, leukaemias were classified with anaemias and disorders of blood until 1947 when the sixth revision of ICD reclassified them as malignancies. An apparent rise in childhood deaths from malignancy may be a consequence of this (Tait and Boog-Watson, 1962). The original certificates were examined and recoded personally in the present study to avoid such problems.

During the archive search a number of duplicate entries were discovered. In most cases the death had been reported to the Coroner and a death certificate issued by the doctor. In these cases the doctor's certificate had been deleted when the Coroner issued his certificate. One case was found where two doctors in the same hospital had apparently issued certificates for the same child. The particulars were identical in all respects except that the forename was spelt Shane and Shaun respectively. These may have been twins although only one

necropsy was found which contained no mention of a twin. Other duplications may have occurred but were assumed to be twins in the absence of evidence to the contrary. In these cases the deaths were notified by different persons, usually the father and a grandparent.

In some computer records of death certificates, only the last entered cause of death has been indexed. The present study would suggest that this could result in serious distortion of the pattern of fatal diseases because of the large proportion of incorrectly completed certificates. The method of examining all entered causes of death greatly increased the yield of data and helped to avoid these potential errors.

Permission to examine the death certificate archives was originally refused. It is understood that this was because the certificates are bound under strict confidentiality and the provisions of the Official Secrets Act might apply. Alternative methods of identifying the sample population were pursued. Burial records were searched but not all children have a church burial and parish boundaries do not conform to the city boundaries. The counterfoils of all death certificates issued in Sheffield hospitals were traced but all of the counterfoils of certificates issued by family doctors could not be found. Weekly returns of death certificates of all Sheffield child deaths were made to the MOH before 1974 and to the Sheffield Area Health Authority (Information Services) subsequently. Copies of these weekly returns are circulated widely among Town Hall staff without apparent breach of confidentiality. A complete series of these MOH returns for the period 1963-1979 was found in the Town Hall

but no records earlier than 1963 could be traced.

The Church of Latter-Day Saints (Mormon) is establishing microfilm archives of many vital statistics but before an approach was made to them, further requests were made to the Registrar of Births, Deaths and Marriages. The initial reply was that copies of death certificates would be made available - at £3.50 per copy, provided that the Registrar was furnished with the name of each child. As the archive search was necessary to identify the children it was not possible to provide these names. It then emerged that copies of every certificate issued in Sheffield could be obtained - but at the same price per copy. From published mortality figures it was calculated that there had been some 250,000 deaths in Sheffield between 1947 and 1979. The expense was thus prohibitive. Finally, with the support of the Sheffield Registrar, a direct application was sent to the Registrar General for permission to view the archives. Permission was then given to search the archives for the years 1947-1962, on the understanding that no particulars of any individual entry or name of any person would be made known and that the searching would be done in the presence of a member of the Register Office staff, at their convenience.

4.1.2 Defining the city boundary

The population at risk was defined as the number of children who had a permanent residence in the city. The city boundaries have changed during the period studied and it was therefore necessary to scrutinise the home address on each death certificate. The city boundaries are defined in the Index of Sheffield Streets which is regularly updated but with each boundary revision the

old Index was destroyed and only a few copies of certain Indexes (no complete series) have been kept in the city libraries. Eventually, with the assistance of a retired Elections Officer, a complete series of Indexes was found by searching through cupboards in the Town Hall. It is recommended that one copy of each revision of the Index should be lodged in the local studies library.

4.1.3 Defining child death

The age at death was found to be more accurately recorded by the Register Office staff than by any other source and therefore the study population has been defined by the age on the death certificate. However, some very early neonatal deaths may have been registered as stillborn. If such a practice were widespread it would artificially lower the neonatal and infant mortality rates without affecting the perinatal mortality rate. A possible reason would be the great difference between the cost of burying a stillbirth and an infant. In Sheffield in autumn 1980 the costs were £9-£50 for a stillbirth and £200-£300 or even more for an infant (Sunderland, 1981a).

The extent of this practice cannot be assessed directly. It would be expected to affect only first day deaths irrespective of birth weight. Such a trend has been identified for first day deaths in England and Wales since 1961 but which was interpreted as indicating the effective introduction of neonatal intensive care units (Pharoah and Alberman, 1981). This trend could be due to a decline in deaths from birth trauma if the national experience were the same as Sheffield's (Fig. 17). In Sheffield, the first day mortality rate also began to fall in the early 1960s

but there was no slowing of the decline in the stillbirth rate (Fig. 18). This suggests that if some early neonatal deaths were registered as stillborn, this was not widespread although it is possible that the rate of fall of the stillbirth rate could have been greater but was slowed by this practice.

An increasing tendency for obstetricians to prematurely deliver babies believed to be at high risk of intrauterine death may have balanced out the numbers of early neonatal deaths incorrectly registered as stillborn but again it is not possible to test this. In the present study, one pathologist in Sheffield recorded all deaths in the first 24 hours as stillbirths in the 1950s although, in the cases registered by the clinicians as infant deaths (i.e. live born), there was irrefutable histological evidence of post partum life; in these cases the pathologist's statement was ignored.

The effect of the Abortion Act on reducing the numbers of children at risk (Sunderland, 1981b) cannot be measured in this study.

4.1.4 Defining the cause of death

A grave and little-appreciated problem for students of mortality records is the method of deciding what a person died from - as opposed to what he dies with. The death certificate lists the diseases which a person died with and the conventional practice is to take the last entered disease as the underlying cause of death - what he died from. This study has shown that such a practice can lead to serious errors. An example may clarify the problem:

A female baby was born at term by caesarean section on 26.4.80. The mother had had eight previous pregnancies, the first two miscarried, six other children are alive and well. There were no antenatal problems but passage of dark blood per vaginum led to emergency caesarean delivery. The baby weighed 3180 g and did not feed well. She was kept in hospital for seven weeks, weighing 3290 g on discharge. She was seen in outpatients two weeks later and readmitted because of failure to thrive. She remained in hospital for a further five weeks when she weighed 3630 g. Three days after this discharge from hospital she appeared well but at 4 p.m. that day, while on the settee beside her mother, she developed 'breathing trouble' and her eyes were rolling. There were no signs of vomiting but she felt very hot. The doctor arrived at 4.35 p.m. and certified her dead.

After preliminary investigations and pending necropsy, the police considered that this was a cot death - no suspicious history or evidence suggested a violent death. At necropsy, evidence of acute inflammation of the respiratory tract was found in a child who had evidently failed to thrive. Additionally, both kidneys were found to be disproportionately small but normally formed, their combined weight being less than half the weight of one normal kidney from a child of this age. The provisional report to the coroner gave the cause of death as 'Acute infection of the respiratory tract (Unexpected death in infancy) in a child with small kidneys who had failed to thrive'. At this stage many necropsies would have been closed. Histological examination revealed upper respiratory tract inflammation, moderately severe

enteropathy, punctate cerebral and pituitary calcification (which might have interfered with vital reflexes), renal hypoplasia and renal arteriolar hypertensive changes, greatly elevated vitreous sodium (161 mEq/l) and urea (60 mmol/l) and neuroblastomata in both adrenals. When these findings were presented to a group of consultant paediatricians there was considerable discussion as to the mechanisms leading to death but they were unable to agree on the underlying cause of death. Another example of this dilemma has been given by Emery (1962). The solution for the present study was for the author to consider all the evidence available from the death certificate and necropsy report and to make a decision based on clinical and pathological knowledge.

Considerable doubt still remains as to why a given child will die with a collection of pathologies whereas many other children in apparently identical circumstances will remain alive. The present study could not examine this problem.

The use of multiple coding greatly increases the data yield in studies of this kind (Fedrick and Butler, 1972) and was considered but discarded for this study. In multiple coding, a search is made for the presence of a disease anywhere in the death certificate or necropsy report. Thus a mongol child in the present study with a congenital heart defect and early pneumonia who was killed following a vehicular collision was indexed as a trauma death. By multiple coding he would be considered under all four diseases (congenital heart disease, chromosome anomalies, trauma and infection). There are two problems with such a multiple coding analysis. There is an

implicit assumption that every condition was consistently recorded by every doctor on every certificate or necropsy. This is probably an erroneous assumption. The long period considered in this study greatly increases the chance of artefactual omissions as the level of reporting is dependent upon the perceived severity of the condition by the medical attendant. Secondly, interpretation of such an analysis may be misunderstood to imply that the diseases considered would all have been fatal. This is patently incorrect - the mongol child had not died from his chromosomal problem or his heart disease before the accident and it is not certain that he would have died from the pneumonia.

4.1.5 Defining the population at risk

The Sheffield birth rate has fallen steadily since 1851 (Fig. 5). It would therefore be expected that the numbers of child deaths would decline by at least the same amount. Because of this changing population at risk, the results have been presented as proportions of the numbers of children alive.

Studies of mortality based on central records usually take the population at risk from figures in census returns with approximations for inter-census years. Such approximations can take no account of intervening changes in the population. It would appear that the estimated populations calculated in this study are more accurate than intercensal projections for Sheffield because of the great fluctuation in the birth rate. The assumption regarding net migration (section 2.8) would appear to have been reasonable as the calculated population approximates the census population for each census year (Fig. 4).

Census enumerations contain inaccuracies. The U.S. Bureau of Census has shown that errors in the census vary by as much as 14% overcounting and 8% undercounting in different areas (see Lillienfeld, 1976). The 1966 (10% sample) census appears to have underestimated the numbers of Sheffield children (Fig. 4).

The population of Sheffield has been stable at approximately 500,000 since 1920 (Fig. 1). Therefore the birth rate between 1920 and 1979 (Fig. 5) is a close representation of the numbers of live births. As only 15% of the population in 1851 and 1951 were born outside Yorkshire (Pollard and Hunt, 1956) and as 97% of all Sheffield births in 1973-1977 could be traced to a Sheffield address throughout their first two years of life (Sheffield Child Development Study, unpublished data) it would appear that the great majority of the children whose deaths were examined in this study were Sheffield-born.

Although the birth rate has been declining since 1851, the rate of one-year-old survivors remained steady between 25 and 30 per thousand residents until 1914. It may be that parents spontaneously reduced their family size as they became confident that their offspring would survive. As fewer children will almost invariably lead to fewer grandchildren in a stable society, a spontaneous reduction in "community replacement" rate (one year survivors per thousand residents) would appear as a self-perpetuating reduction in the birth rate until the family size became stabilised at a lower rate. The birth rate would then appear to rise until the parent generations (the denominator) had passed on. This "community replacement" rate may be a more useful index of fertility patterns than the birth rate.

The decline in birth rate from 40 births per thousand residents in 1851 to 10 per thousand in 1977 followed a sigmoid curve with peaks associated with each of the world wars (Fig. 5). The peak between 1920 and 1922 is comprised of some 7000 'extra' births and the birth bulge after the second world war contains some 20,000 'extra' births (Fig. 5). The children of the 1920s were themselves having children in the early 1940s which merge into the second world war births. This generation had their children in the 1960s and, given Sheffield's stability of population, this could be the explanation for the observed increase in births in the 1960s (Fig. 5). If this were the case, then there would appear to be approximately 20 years between birth and childbearing in Sheffield. The children born in the 1960s might be expected to have their children in the 1980s and another 'birth bulge' might be expected. This seems to have commenced (Fig. 5).

However, parents do not have all of their children on their twentieth birthday. Therefore the next birth bulge may be spread over a longer period and the birth rate will then not rise as high as previously. Extrapolations of the trends shown in Fig. 5 suggest that the peak birth rate will not exceed 15 births per thousand but that the increased birth rate may last into the next century. Despite these birth bulges, it appears that the Sheffield birth rate is going to stabilise at about 10 births per thousand residents.

Fertility rates and child mortality rates both increase with decreasing parental social class (Bone, 1978; OPCS, 1979a). A rise in birth rate is probably not distributed uniformly

through the community with a greater increase occurring in the lower social classes. If so, a rising birth rate might show a rising child mortality rate which would principally be due to deaths among children of lower social class parents. Such an effect has been noted for cot deaths (Peterson, 1980). If such a class effect exists, it cannot be examined in the present study which is concerned with disease-specific and age-specific mortality rates.

Throughout the period covered by the present study, medical efforts have been directed towards reducing mortality. Success of these efforts will have resulted in more children surviving. There would then be an increased number of children at risk of developing other diseases. This has particular relevance to studies of early child mortality because obstetricians' efforts are directed principally towards reducing stillbirths. It is now possible to predict a likely stillbirth with some certainty and to deliver such babies prematurely to effect a live birth. A decline in stillbirth rate may therefore be at the expense of increased neonatal mortality. Similarly, a decline in neonatal mortality will result in an increased number of babies at risk of succumbing to cot death; or an increased number of children surviving to school age (coupled with an increased number of cars on the roads) may be followed by an increased mortality from road accidents. To standardise this potential artefactual change, the 'number of children at risk' has been used as denominator in calculating age-specific disease rates.

4.1.6 Data preparation and analysis

Manual examination of large numbers often introduces arithmetical errors (Butler and Alberman, 1969). To reduce the possibility of clerical errors and to reduce inter-observer variation, the alternative of computer handling of the data was chosen. Both the three-digit and four-digit ICD codings of disease (WHO, 1977) were found to be too vague in many areas for this study while in other areas (e.g. infections) far too much detail was given. It may be necessary to have the facility for one hundred divisions of tuberculosis and ten subdivisions of glanders or anthrax; however, there would appear to be some imbalance when this only allows thirty subdivisions of congenital heart disease or ten subdivisions for congenital respiratory diseases. These possibilities have been multiplied one hundred-fold to overcome the problem of classifying congenital malformations (Weatherall, 1980) which in consequence requires that space is theoretically created for ten thousand divisions of tuberculosis. In the present study, because of the limitation of computer storage space, a new disease code was devised which followed the general pattern of ICD9 but expanded or restricted codes as necessary. This classification (Appendix III) was flexible, comprehensive and efficient. Only three digits were necessary and an allocated fourth digit was used for a summary code of the severity of each disease.

4.1.7 Errors in data preparation

In transferring the data to coding sheets 186 digits were omitted. These were all "don't know" values and if undetected would not have affected the analyses. They would have

represented an error rate of 0.04% (186 omissions among 437,038 digits). Lay coders might have produced a higher error rate.

Thirty-eight nonsensical values were coded which were due to lapses of concentration. Such 'outliers' have been described as flamboyant manifestations of variability (Armitage, 1970). In the present study they were all elementary coding errors.

Errors which lie outside the studied ranges are easily detectable. Coding errors within the ranges can only be detected by examination of each digit. Most published studies do not report examining for this error although it is possible that the errors reported in studies of disease codings could be in part due to such mistakes (Fedrick and Butler, 1972; WHO, 1967). In the present study two major coding errors were found in the 8680 digits re-examined in the 2% recoding study.

Accurate transfer of coded data to punch cards is so vital that the procedure is usually done by trained secretarial staff using verification processes developed by computer manufacturers to exclude all such errors. However, these processes are done by humans. It is worrying that at least seventeen major errors occurred at this 'infallible' stage. If these errors had not been sought and corrected, the information on those children would have been seriously distorted. It is not known if any typing errors were committed within the defined ranges although no such errors were found in the 140 cards in the 2% recoded sample.

4.1.8 Errors in computer analysis

The computer programme used for analyses - the Statistical Package for Social Scientists (SPSS 1975) - is widely used and

reliable. During analyses up to two million variables were cross-tabulated. Apart from the physical difficulty of conducting these analyses manually, it is also virtually impossible to add such large series of numbers without mistake (Butler and Alberman, 1969). Despite public scepticism and predilection to blame the computer for administrative mistakes, the computer is a most efficient adding machine. Most mistakes in computer analyses are due to human errors in programme writing or data input.

To test for accuracy, two large computer analyses were repeated and the tabulations compared. Although almost every figure in every cell was identical, in both studies there were two figures which differed by one unit. As the programme of instructions was identical, it would appear that the computer may be inaccurate when handling such large numbers.

4.1.9 Security of the computer files

It was a condition of access to the death certificate archives that no details of any individual would be made known. A coding system was devised to ensure that no child could be identified from the computer files should the double password security be breached. The unique classification of diseases used afforded additional security because, for example, the mothers of children dying with congenital syphilis could still be alive.

It was found possible to gain access to the computer file-stores while intentionally using incorrect passwords. Although it was not possible to obtain printouts or to manipulate the files by such access, it was possible to read the data stored in

the files on a display unit. More seriously, it was found that passwords could be altered and this not only allowed unauthorised and unlimited access to the files but also debarred the owner of the file because he would not know the new password. For security reasons this method cannot be described but it is achieved by discovering how a computer 'remembers' each user's password and then altering this memory. The University of Sheffield computers form part of a European network and there are plans to link up with American machines. It is therefore theoretically possible for anyone with a linked terminal to gain access to the files of these children: two 13 year old boys in a Manhattan School totally destroyed the intricate and confidential business records of a number of Canadian businesses from a computer terminal at their school (Anonymous, 1980). Traditionally, confidential records are kept in individual files. These files were far more readily accessed by the author than the computer files, without any security check. The only safeguards on the contents of these files were their geographical location and the legibility of the handwriting whereas the computer files were protected by passwords, specific computer language and digital coding of information.

4.1.10 Control of observer variation

There are five tests of quality control for any epidemiological survey: only positive cases may be included and all positive cases must be included; subject variation, inter-observer variation and intra-observer variation must all be controlled. In the present study, the first three tests are controlled in that only dead children were included and, as far

as is known, all dead children were identified and there is no subject variation in a mortality study.

All studies which use death certificates as the basic data will incorporate an uncontrollable inter-observer variation because the death certificates were written by a large number of doctors in a variety of hospital and family practices. In the present study, the use of pathology reports to verify the cause of death limited this inter-observer variation by reducing the number of observers. Four of the pathologists involved are still working and generously assisted in the standardisation of their reports and in explaining how their diagnoses might have varied with time. An additional advantage of studying a single city was that senior colleagues, who wrote many of the certificates years before, were able to clarify any ambiguities among those deaths where no necropsy was done.

Most published studies of mortality do not report the level of inter- or intra-observer variation in the data collection and processing. Fedrick and Butler (1972) examined the cause of death coded in the British Perinatal Mortality Survey with the coding by the General Register Office on the same records. They found major discrepancies between the codes, caused in part by ICD groupings but state that even a perfect coding system could not achieve greater accuracy than 90% for easily recognised malformations (e.g. anencephalus) and for internal malformations (e.g. renal agenesis) the best accuracy to be hoped for would be less than 60%. They found that neonatal pneumonias diagnosed at autopsy were correctly coded in less than 30% of the cases. They showed that a child with anencephalus was coded as microcephalus and 13 out of the 120 children with spina bifida were

coded as dying of conditions unrelated to any congenital deformity.

The use of a computer in the present study reduced to one the number of persons involved with data handling and hence reduced the possibility of clerical and arithmetical errors. Apart from revisions of ICD codings, there may have been some intra-observer coding error during the 33 years that the 7049 certificates were issued. By recoding all of these certificates in a six-week period and using a single classification of disease, the possibility of these errors was reduced and the possibility of inter-observer coding errors was removed. Coding the information on the 7049 deaths was an exhausting process. The possibility of some human error remains and during the coding process some intra-observer error could have crept in. Any such errors were randomised by examining the data without knowledge of the year of death. These errors were sought by recoding 2% of the sample. It would appear from the result of that study that there is a confidence limit of 99.8% in the consistency and accuracy of the coding procedure. Coding of the causes of death and any inherent bias in the observer both appear to have been consistent.

The discrepancies found by Fedrick and Butler (1972) could in part be due to the use of lay coders. In a study to determine the degree of agreement between European coders, a sample of 1000 completed death certificates were copied and sent to six European centres for coding. The results were compared with the codings of staff at the World Health Organisation Centre for Classification of Disease in London. Discrepancies between the centres ranged from 15 to 34%. On no occasion did any centre completely

agree with another (WHO, 1967). It would therefore appear that studies which have used central records and studies which have not examined the degree of inter- and intra-observer coding errors should be interpreted with caution.

4.2 Death certification as a measure of child mortality

4.2.1 The completeness of death registration

It is an assumption of mortality data that every death is registered. It is not readily possible to identify any under-registration of deaths from routinely gathered statistics. Rogers et al. (1961) suspected that there had been a degree of underregistration of neonatal deaths in North Carolina when they noted that, among babies weighing less than 1500 g at birth, the neonatal mortality rate was lower for illegitimate and non-white babies than for legitimate white babies. They found that among the 1484 babies weighing less than 1500 g who were born in 1958, 459 did not have a matching death certificate. Among these apparent survivors, 69 were dead and there were an additional 30 infants who could not be traced. This gives a figure of at least 15% (69 out of 459) unregistered deaths. Following this, McCarthy et al. (1980) in a careful hospital survey showed that 21% of neonatal deaths between 1974 and 1977 in Georgia were unreported.

Scott and her colleagues (1981) in a twelve-month study of 19 maternity hospitals in Ulster found that 31 liveborn infants, known to them to have died in the first week, had not been registered dead within the permitted period. They also found 16 stillborn infants of over 28 weeks gestation who had not been registered. However, death certificates for 26 of the 31

babies and registration for 13 of the stillbirths were subsequently traced. They found that among 440 perinatal deaths there were initially 69 (subsequently 8) unregistered deaths. In contrast, the Register Office identified another 96 perinatal deaths of which Scott et al. were unaware. It thus appears that a sizeable proportion of perinatal deaths among hospital delivered babies may be unregistered.

None of the above studies commented on the possibility that there may have been many more such deaths among infants who had not been registered as births or were born outside hospital. The authors did not include foundlings in their figures. Puffer and Serrano (1976), in a study of maternity hospitals throughout America, found that 207 out of 2762 neonatal deaths (7.6%) were unregistered and, in Santiago, 56% of the neonatal deaths identified in the mortuary had neither been registered as births nor deaths. A recent case in Yorkshire of two children who were buried in the back garden only came to light because the parents continued to claim welfare benefits.

In the present study, 1500 consecutive necropsy reports issued between April 1961 and April 1966 included 390 Sheffield children, 38 of whom had apparently not been registered dead. Certificates were found for 30 of these children with a non-Sheffield address. A possible explanation for this is that there may have been a mistaken belief that it is necessary to have a Sheffield address to obtain treatment in a Sheffield hospital. The apparently large number of such cases is biased

because many children are brought to Sheffield for surgery and their parents stay with relatives or in rented accommodation.

There remain eight Sheffield children who had a necropsy without apparently being registered dead, four of whom were early neonatal deaths. There are three possible explanations for the inability to pair these necropsy reports with a death certificate: some of these children may have been illegitimate - the mother's name must be given on the death certificate but the father's name is often given to the hospital; secondly, some may have been registered as stillbirths; thirdly, some may have been disposed of without informing the authorities. This last explanation does not apply to the children described here as these bodies were all recorded as being released to an undertaker. The converse, where newborn infants are disposed of illicitly (foundlings) has not been considered here as such deaths, if identified, are notified to the coroner and will be included in the death registrations. Among the four remaining children, one was found to be illegitimate and registered under both parental names. The other three could also have been illegitimate. No other explanation for these unmatched certificates has been found.

These findings are based on necropsies at SCH. It will be seen from Table III that only a proportion of early neonatal necropsies are performed at SCH. The proportion of unregistered deaths at maternity hospitals could be higher and there may be additional babies born outside hospital and buried illicitly who are neither registered as births nor deaths. While death certificate archives contain the most complete record of child deaths in the city, some doubt must remain as to whether all deaths have been included, especially in the very young.

4.2.2 Comparison of personal details on death certificate and necropsy record

The personal details (age, sex, address etc.) given on a death certificate are verified and, if necessary, corrected by the Register Office staff examining the informant at the time of registration of the death. Further details from the Coroner or police enquiries are included subsequently. In contrast, information in the necropsy record is usually obtained second-hand or from hospital notes. Details on the necropsy record are rarely checked. It is therefore not surprising that errors were entered in the necropsy records and it seems reasonable to assume that the death certificate details are the most consistently accurate records available.

The accuracy of the stated age at death given on death certificates was examined by the Registrar General (1968) in which the National Health Service records were traced of a 12% sample of the 5300 patients who were registered dead in March 1966. The age of these patients was calculated by subtracting the date of birth (on the National Health Service record) from the date of death (on the death certificate). This calculated age was compared with the stated age on the death certificate. There were 68 children under 15 years but only one infant in the sample. Errors were found in the stated age which increased with increasing age but were rarely greater than one year too much or too little. It was calculated that errors in the stated age of children would be expected in 3% of under-10s and 1% of child deaths aged 11-15 years. However, the study was biased in that the first three decades were over-represented

(although infants were conspicuously absent), and the season was chosen when a low number of deaths might be expected.

During the archive search it was noted that parental occupation stated on the death certificate showed an increasing number of 'engineers'. The Classification of Occupations (Registrar General, 1960) contains a code in the skilled manual worker category for this. However, from details in the necropsy records it would appear that the designation 'engineer' may now refer to a union member who need have no skill or trade. The Registrar General's coding was followed and a note made of the quality of the paternal handwriting in such certificates. Qualified engineers are usually identified by an adjective e.g. structural, civil, naval. If the necropsy record contained parental details then these were used for clarification whenever necessary.

4.2.3 Validity of the certified cause of death

The finding in the present study that approximately 50% of the last-entered causes of death on children's death certificates disagreed with the pathologist's opinion as to the cause of death is in keeping with the majority of other studies on accuracy of certification of the cause of death (Gittelsohn and Senning, 1979). However, as the principal aim of this part of the study was to define the validity of death certificates rather than the extent of disagreement, the methodology of the present study was different from other studies.

Heasman and Lipworth (General Register Office, 1966) compared clinician's diagnoses before necropsy with the pathologist's afterwards. Among their findings they showed that spina bifida

and meningocoele appeared to be overdiagnosed by the clinician and congenital hydrocephalus underdiagnosed; whereas the pathologists, who were assumed to be correct, did the reverse. It seems unlikely that clinicians would overdiagnose such a lesion. If the pathologists preferentially put hydrocephalus last on the death certificate while the clinicians placed spina bifida last then the 'disagreement' is created. In Heasman and Lipworth's data the sum of hydrocephalus plus spina bifida is the same for clinicians and pathologists. It seems more reasonable to accept that if spina bifida occurred anywhere on either certificate to consider this to be agreement, which was the procedure used in the present study.

The much lower rates of inaccurate certificates found in the present study (15% overall, Tables IV-VI) are due to this different methodology rather than to greater local care in certification. By assuming that the pathologist could be wrong as well as the clinician, the accuracy rate of certified causes of death has been greatly improved. Though it may seem reasonable to assume that the pathologist is correct as he is able to examine the whole body and has available the clinical findings, the clinician may be in a better position to describe the mortal process because he has observed the final course of events.

As the clinician can attend the necropsy before completing a death certificate and as the pathologist studies the clinical history before doing the necropsy there should be no discrepancies. 'Consistency in certification is biased in favour of a persisting diagnosis' (Emery, 1962). The discovered errors in certified cause of death can be partly explained by the greater

yield of new findings from microscopic studies in child deaths. However, as the certifying doctor is recorded as witnessing the necropsy in some of the more outstanding errors it would appear that some errors arise from lack of care when completing certificates. In the study of perinatal mortality in Ulster (Scott et al., 1981), the recommended WHO certificate of perinatal death was completed as well as the usual certificate for the Registrar General. Among 344 paired certificates only 202 (59%) gave an identical cause of death and 24 (7%) gave totally different causes of death although each pair of certificates was said to have been completed by the same person.

Other studies of accuracy of the certified cause of death usually use coded records and many studies merely examine for congruence of coding lists. The coding procedures used to identify the underlying cause of death lead to many misunderstandings in addition to the previously discussed practice of using the last-entered cause of death. Heasman and Lipworth (General Register Office, 1966) showed that 'hypertensive heart disease' would disagree with 'hypertensive heart failure' because they had different ICD codes. Hook et al. (1977) showed that 'aortic stenosis' is entered as a chronic rheumatic disease although in their experience all of these cases in childhood were due to malformation. It has also been shown that 'aortic aneurysm' should be coded as syphilitic unless otherwise specified but that there was no uniform practice among European lay coders, many of whom coded it into one of the arteriosclerotic categories (WHO, 1967). In addition to these varying coding practices, revisions of ICD will also affect

studies based on central archives (see section 4.1.1 and Lillienfeld, 1976). These problems were avoided in the present study by using one professional instead of many lay coders, by using one disease coding, by performing all of the coding in a short period and by using the reports of a small group of pathologists to verify the causes of death.

Emery and Irvine (1958) examined the necropsy records and clinical notes on 150 consecutive deaths aged from birth to 12½ years in a children's teaching hospital. They found that in only 15% of the cases was there full agreement between pathologist and clinician. In 40% of cases the certified cause of death was incorrect and in 18% they considered that the pathology findings necessitated a complete revision of the death certificate which is similar to the figure of 15% found in the present study. They found no correlation with age or length of stay in hospital, in keeping with earlier findings of the Registrar General (1958).

Table IV shows that examination of death certificates without detailed necropsies will give unreliable and inconsistent results, with the possible exception of the diseases included under Violent Death. Studies of the miscellaneous acquired group will contain about 11% gross inaccuracies; while studies of infections, tumours and malformation will contain about 15% inaccuracies. The proportion of disagreement in the epinatal group (18%) may be an underestimate because of the low necropsy rate (Table II, see Kane, 1964). The high rate of 'incorrect word order' among the acquired group is because very many of the cerebral palsy deaths were registered as pneumonia with cerebral palsy entered in part II of the certificate.

Disagreements are greatest among children less than 1 year (Table V) but there is remarkably little variation of disagreement with age among these infants (from 16.7% to 18.3%). The level of disagreement decreases rapidly after the first birthday.

The social class distribution of disagreement shows very little variation (Table VI) which is contrary to the suggestion of Adelstein and White (OPCS, 1976c) who speculated that inaccurate certificates would be more common among the lower social classes and that the children of doctors are likely to have the most detailed diagnoses. Both of these suggestions were found to be incorrect for Sheffield with inaccurate certification ranging from 12.8% in class V to 17.6% in class IIINM. (Examination by socioeconomic group found a similar distribution and has not been presented.) The strikingly uniform distribution of disagreement by class extended to specific parental occupations with the range of 7.6% to 10.0%; 8.9% of doctor's children's certificates were inaccurate.

The process of deductive reasoning by which a doctor arrives at a diagnosis is highly personal, little studied and less understood. The possibility of diagnostic error is little appreciated and, unless doctors apply some form of internal audit, cannot be detected. Many of the studies on the accuracy of death certification fail to appreciate the nature of this diagnostic process and underestimate the problem of deciding upon a single cause of death. Such studies also appear to assume that pathologists are less fallible than clinicians. Studies of the reliability of diagnosis have shown that doctors (including pathologists) not only disagree over the interpretation of signs, symptoms, investigations and treatments with their colleagues, but also

with their own previous diagnosis. Koran (1975a, 1975b) reviewed 38 of these studies and found that, when a group of doctors are given the same information in a different order or after a short period, they will disagree with their own previous diagnosis and with their colleagues in at least 10% of cases and often in more than 20%. The present study which has found a disagreement of 15% between information given by clinicians with that given by pathologists is therefore in keeping with these studies. The numbers of doctors involved in the present study over 33 years is much greater than the numbers in these other studies. It would appear that there is a confidence limit of 85% on such diagnoses.

A higher level of agreement has been achieved in the present study by allowing for minor variations in diagnostic label and ignoring all of the technical disagreements caused by variation in the coding of the last-entered diagnosis. Cameron and McGoogan (1981) modified the method of Heasman and Lipworth (General Register Office, 1966) so that clinicians' diagnoses were classed as "agreed" if confirmed by necropsy, "overdiagnosed" if disproved and "underdiagnosed" where a disease revealed at necropsy had not been anticipated clinically. Their findings supported those of Heasman and Lipworth (General Register Office, 1966). The extra work involved in filling in dummy certificates before the necropsy may preclude widespread adoption of this method but these results confirm the importance of a high necropsy rate if valid mortality information is to be obtained. Even by the lenient rules adopted for the present study, overall agreement was only 77% of the deaths which had a necropsy. This suggests

that studies of mortality based on death certificate alone have an unsound foundation.

Necropsies are only done at the request of a clinician or the coroner. Understandably a clinician, having devoted his energies to preventing a child's death, sometimes finds it distasteful to have to ask parental permission for a necropsy. It seems improper that a pathologist's workload should be determined by others - would clinicians in turn accept a pathologist deciding which patients they should see? There is no apparent ethical objection to all dead patients being referred to the pathologist in the same way as doctors refer living patients for a specialist opinion. The pathologist may then decide, in consultation with his clinical colleagues, as to which patients should have a necropsy with the pathologist obtaining consent from the relatives. In this way the pathologist could develop closer contacts with bereaved relatives and may, for example, establish a follow-up or bereavement counselling service.

The inaccuracy of these certified causes of death is not surprising as death certification completion is usually delegated to the most junior member of staff and little instruction is given in medical schools. As death certification remains the most consistent, albeit indirect, method of assessing a nation's health, attention to this detail is long overdue.

Kane (1964) stated that rates of incidence based on unautopsied material were not worth collecting, let alone publishing. Because there is no available method for validating the certified cause of death in the 39% of these Sheffield children who did not have a necropsy, it could be argued that the data is too

severely limited for meaningful analysis. However, Graunt (1676) did not wait for better material to produce his analyses and by using available material he provided a much stronger case for improvements. His conclusions in 1676 were extended but not refuted as more accurate data became available.

4.3 Mortality Trends I - Deaths by Age

In 1900 there were 8292 deaths among Sheffield's population; 4382 (53%) occurred under 15 years of age and 2511 (30%) were in infants. In 1947 the proportions were 8% and 7% respectively and in 1979 they were 1.7% and 1.3%. The Sheffield infant mortality rate is available from 1885, the neonatal and first week rates from 1905 and the total child mortality rate from 1947. The changes in these rates with time are shown in Figs. 6 and 42.

The decline of the total child mortality from 4.1 deaths per thousand in 1947 to 0.9 in 1979 (Fig. 6) is the resultant of two major trends: a rapid decline before the 1950s superimposed on a more gradual but continuous decline throughout. The rapid decline occurred among all children over one month old, expecially in the postneonatal age group (Figs. 8-11). The continuous decline occurred among all children under 5 years and appears to be continuing (Figs. 8-11). This decline also emerges in cohort analysis (Figs. 19 and 26) which suggests that each successive cohort is healthier than the last.

Examination of the mean distribution of deaths by age in three 11-year cohorts (Fig. 7) shows that there has been little change in the proportionate distribution with time. The relative

importance of deaths among the very young is shown in Fig. 7 where, throughout the period 1947-1979, three-quarters of all deaths in childhood occurred in the first year and about half of all childhood deaths occurred in the first month. Fig. 7 also shows that the majority of neonatal deaths occur in the first week and Fig. 10 shows that most of these deaths occurred in the first day. The death rate among children aged 1-4 years was approximately the same as that among all children aged 5-14 years (Fig. 7). The proportionate distributions of deaths by age of these Sheffield children are similar to those found for South Warwickshire children in 1962-1963 (Dunn and MacGregor, 1964), and for children in Malmö in 1972 (Östberg, 1973). However, the value of such proportionate distributions is limited because of the changing pattern of deaths by age with time (Figs. 8-11).

4.3.1 Comparisons between Sheffield and England and Wales

The Sheffield birth rate (Fig. 5) was 4.5 births per thousand greater than the rate for England and Wales until 1916 and (except for the period 1937-1947) has been slightly lower than the national rate since 1923 (Sheffield 1874-1973).

The decline in age-specific mortality rates in Sheffield has closely reflected the decline in England and Wales from 1947 to 1979 although the mortality rate among 5-14 year olds in Sheffield has been slightly above the national average throughout (Figs. 8 and 9). The neonatal and postneonatal mortality rates in Sheffield were much closer to the national average than in some of the other large English towns studied (Fig. 27). Some other town might have shown a closer reflection of the national

trend but it has not been possible to study other towns because of the changes in population which followed the 1974 local government reorganisation.

The decline in first day mortality rate in Sheffield (Fig. 10) occurred at the same time as the observed change in the national rate (Pharoah and Alberman, 1981). The implications of this have been discussed in Section 4.1.3.

It would appear that mortality rates increase in proportion to the distance from the South-East of England (data in Registrar General 1839-1973; OPCS 1974-1980; Butler and Bonham, 1963). Thus the neonatal mortality rate in Cornwall is similar to that in Wales and the north of England, all of which are higher than that in the Home Counties and lower than those in Scotland or Ulster. Sheffield lies in the middle of this national mortality gradient and it would appear that Sheffield's children have closely reflected the overall national trend.

On the strength of these age-specific mortality rates, Sheffield's children may be a representative national sample. If this is the case then the changes in fatal diseases among Sheffield's children might be extrapolated to explain national trends. However, without corroborative evidence such extrapolations should be treated with caution.

4.3.2 Comparisons of neonatal spring deaths in Sheffield and England and Wales

National studies of perinatal mortality were made among the children born in the month of March 1958 and the seven days 5th-11th April 1970 (Butler and Bonham, 1963; Royal College of

Obstetricians and Gynaecologists, 1975). The causes of neonatal death in Sheffield in March 1958 were similar to the national pattern (Table VIII). The distribution of the causes of early neonatal death in Sheffield in 1970 was also similar to the national pattern in the week 5th-11th April 1970 (Table VIII). However, comparisons of the Sheffield deaths in these short spring cohorts with the deaths in Sheffield in the whole study year suggest that samples based on one week's births may be unrepresentative (Tables VIII and IX).

One reason for the difference in mortality pattern between the spring and whole year cohorts is that there may have been too few deaths in the Sheffield spring samples for statistical validity (Tables VIII and IX). Examination of the aggregate distribution of Sheffield mortality pattern of early neonatal deaths in March, 5th-11th April and the whole year for the period 1947-1979 shows that by increasing the sample size, a similar distribution of mortality emerges for the aggregate spring cohorts as is found in the whole year (Table X). This 33-year aggregate distribution of Sheffield early neonatal mortality shows a similar distribution to the national pattern (Table IX, Butler and Bonham, 1963). It is noteworthy that the proportional distribution of principal causes of early neonatal deaths has shown little variation in this period.

Table XI gives one reason why the spring cohorts are unrepresentative of annual rates - the numbers of deaths vary according to the season of birth. If these deaths had been evenly distributed throughout the year then approximately one-twelfth (8.3%) of the deaths would have occurred in each

month. In Sheffield in March 1958 and April 1970 the proportion of deaths was greater than would have been expected had they been distributed evenly. Taking the aggregate distribution for the period 1947-1979 does not even out this distribution (Table XI) probably because of the uneven distribution of live births by season. Crude birth rates vary by season and there is an even greater fluctuation of illegitimate birth rate by season (Registrar General, 1969). The season of birth has also been observed to have some effect on the incidence of stillbirth, congenital malformations and the subsequent experience of a number of psychiatric disorders (Slatis and de Cloux, 1967; Janerich and Garfinkel, 1970; Sandahl, 1978; Anonymous, 1978b).

It is not possible to compare directly the children in the present study with these other studies but some of the children considered in Tables VIII to XI were also tabulated by Butler and Bonham (1963) and the Royal College of Obstetricians and Gynaecologists (1975). The studies reviewed above would all suggest that the season of birth does have some effect on subsequent experience of diseases. From Tables VIII to XI it can be seen that samples of children chosen by birthdate may not be representative. Unless the implicit assumption concerning date of birth and subsequent experience of disease has been tested, extrapolations from studies which were based on samples selected by birth period should be treated with caution. It is suggested that future studies should be based on samples in which all birth cohorts and preferably several years are adequately represented.

4.3.3 Changes in infant mortality rate

The national infant mortality rate has declined steadily since the beginning of the present century. This national decline began after 1903 and has been attributed to alterations in obstetric practices following the Midwives Act of 1902 (OHE, 1979). However, Fig. 42 shows that in Sheffield the fall occurred principally in the postneonatal group and Figs. 38-40 show that the fall was primarily due to a decline in infectious deaths, especially gastroenteritis. The fall in infant mortality was closely associated in time with the slum clearances at the turn of the century and especially with the removal of privy middens (Figs. 44 and 45). The provision of piped water in the Public Health reforms at the turn of the century would have had a less dramatic effect in Sheffield because of the abundance of pure running water in the area and because piped water had already been available for some time because of the requirements of the forges and foundries (Holland, 1843). The changes in mortality among Sheffield infants between 1947 and 1979 (Fig. 8) appear to be part of this overall trend and the analyses of this study have been unable to show any appreciable alterations in these age-specific rates which could be attributed to recent advances in medicine.

During the late 1970s, public attention was drawn to the relatively high infant mortality rate in this country, especially when compared with Scandinavia (see section 1.4.1). Although comparisons between a heavy industrial city and a largely rural country may not bear close examination, it is noteworthy that the infant mortality rate in Sheffield fell from 200.0 in 1900 to 18.5 in 1959 (Fig. 42) while the Swedish rate fell from 94.0

to 18.5 in the same period (Lancet, 1980), i.e. Sheffield achieved twice the fall that Sweden did. International comparisons such as those done by Wynn and Wynn (1976, 1977, 1979) over relatively short time periods may be misleading.

4.3.4 Fatal diseases in each age group

4.3.4(a) Neonatal deaths

Three groups of diseases were responsible for the large majority of the 3507 neonatal deaths (Fig. 12). Almost half of these deaths were due to diseases of prematurity and many of the infants whose deaths were indexed as due to maternal diseases or placental and delivery complications were born prematurely. The indexed cause of death in the very large majority of these neonatal deaths is descriptive rather than aetiological because very little is known about their aetiology. Mortality from these conditions (including malformations which account for approximately one-quarter of all neonatal deaths) is more likely to be reduced by changes in the antenatal or pre-pregnancy period than by changes in postnatal care.

Fig. 12 shows the mean distribution of all neonatal deaths by cause. The value of this figure is limited because of changes with time and Fig. 13 shows that in any particular year the relative proportions will vary. The fatal incidence of the obstetric and prematurity diseases has declined while malformation mortality has risen slightly. The diseases of prematurity were the most important cause of neonatal death throughout and have shown the greatest overall decline. If the distribution of causes of neonatal

death was the same in other large towns then the declines in neonatal mortality rates observed in other large towns (Fig. 27) could also be due to a decline in prematurity deaths. Every town examined has shown a decline in neonatal mortality rate. Such secular trends should be sought before attributing success solely to medical intervention.

4.3.4(b) Postneonatal deaths

Between 1947 and 1979, the number of Sheffield deaths in the postneonatal period (1775) is about half of the number of neonatal deaths (3507) although the age group is eleven times larger. Three groups of diseases account for most of the postneonatal deaths (Fig. 20). One-quarter of these deaths were due to malformations. Another quarter of the deaths are due to sudden inexplicable deaths (SIDS). In half of the SIDS deaths there was evidence of mild acute inflammation of the respiratory tract (AIRT) which was deemed insufficient to account for the death and is a very common finding in otherwise healthy children at this age (see Peterson, 1980). More than one-third of the postneonatal deaths were due to infectious diseases and almost one quarter of all postneonatal deaths in 1947-1979 were attributed to respiratory infections.

The fatal incidence of these diseases has changed substantially in the 33 years studied (Fig. 21). While the malformation death rate has remained steady, the infectious death rate has almost disappeared. Infection and SIDS mortality rates both declined between 1952 and 1958, SIDS incidence then gradually rose to 1972 while the

infection death rate continued to decline. This observation would contradict the opinion of Pharoah and Morris (1979) that the rise in SIDS in the postneonatal period was due to an artefact of registration associated with a reciprocal decline in infection mortality rate. The possibility for some artefact of registration remains, because the decline in postneonatal SIDS rate in the early 1970s was greater than the decline in the crude postneonatal rate (compare Figs. 8 and 21).

The decline in crude postneonatal mortality rate in Sheffield in the early 1970s (Fig. 8) would appear to be primarily due to a decline in SIDS rate. Similar declines in crude postneonatal mortality rate occurred at different times in Coventry, Bristol, Derby and Hull; Southampton may be entering the cycle which these other towns have passed through (Fig. 27). If the declines in these other towns were due to a decline in SIDS rate, then the hypothesis that the decline in Sheffield postneonatal rate was due to the deployment of specially trained health visitors (Emery, 1976) becomes untenable because these other towns did not use health visitors in this way at the time of their observed declines. However, the close association in time between the decline in SIDS rate with the introduction in 1973 of the prospective health visitor (HV) study in Sheffield suggests that some important change occurred at this time. Whether this was a real decline or an artefact of registration cannot be fully answered by the present study as all of these infants were examined in one department which had an

interest in the outcome of the HV study and the present study was based in part on this department's necropsy reports. There is some support for the artefact theory in that evidence was found during the archive search of retrospective changes in other classifications e.g. the subjective A-D classification of severity of disease (Emery, 1962) was changed twice; and Pilling (1976) showed that the reporting of SIDS soared after 1963, the year of the first conference on SIDS. However, those who support the artefact theory will have to explain the reduction in the crude postneonatal mortality rate which occurred in Sheffield at the same time (Fig. 8).

4.3.4(c) Preschool deaths

The 834 deaths in the preschool period are only one-sixth the number of infant deaths although the period covered is four times as great. The mortality rate in this preschool period is principally in the second year of life (Fig. 26). There is a greater proportion of unnatural deaths and tumours in the preschool group than in infancy (Fig. 22). Malformations and SIDS together account for a quarter of the preschool deaths, most occurring in the second year. Infections were the largest single disease group and showed the most marked decline with time principally due to a decline in pneumonias and tuberculosis (Fig. 23a). In contrast to infant deaths, there has been a less striking decline in non-infectious mortality rates over the 33 years studied (Fig. 23b).

There was an increase in the malformation death rate in this age group during the early 1960s (Fig. 23b), possibly due to an increasing survival from younger age groups. The commonest single group of fatal malformations was congenital heart disease. There was a slight decline in tumour death rates due primarily to a decline in leukaemia death rates. Of the two principal factors in unnatural (trauma) deaths, accidental falls have declined but road traffic accidents (RTA) have remained relatively constant. Among the miscellaneous (acquired) group there has been a steady incidence of cerebral palsy deaths; the majority of the other deaths in this acquired group were due to asthma.

These patterns and trends of fatal disease incidence in preschool children are in keeping with other studies (Mair and Tait, 1953). There have been very few studies of mortality in this age group. Mair and Tait (1953) suggest that this may be because both the community and medical authors are distracted by infants: they state that 'with the arrival of a new baby the young child has to move out of his mother's arms and begin to fend for himself ... Perhaps it is this background place in the family life that is reflected in the paucity of medical literature'. Whatever the reason, there is virtually no literature on long term community studies of the mortality or morbidity of this group of children.

4.3.4(d) Schoolage deaths

The 839 Sheffield deaths in the 5-14 year group between 1947 and 1979 are similar in number to the preschool deaths

but cover more than twice the age group. One quarter of these schoolage deaths were due to unnatural causes (labelled 'trauma' in Fig. 24) and the single commonest cause of death in this age group was road traffic accidents (RTA). Another quarter of the deaths were due to the miscellaneous (acquired) conditions of which cerebral palsy and asthma were the most important. Infections, malformations and tumours make up the other half of these deaths (Fig. 24).

The incidence of fatal infections showed a striking decline by the mid 1950s (Fig. 25) as it has done for all age groups after the neonatal period (Figs. 21 and 23a). The major reason for the decline in schoolage infection deaths was the disappearance of fatal tuberculosis. The incidence of malformation deaths shows a gradual increase (Fig. 25) approximately five years after the increase in the preschool group (Fig. 23) which would support the suggestion of an increased survival with these conditions from infancy. The trauma fatality rate (Fig. 25) shows a similar pattern to that in the preschool group (Fig. 23b). In both the trend in RTA deaths is rising in the most recent years. The mortality rate from tumours shows little variation with time which is surprising in view of the reported success of the new antimitotic treatments. The numbers of tumour deaths have declined but the decline in the number at risk produces a steady rate. The numbers of children with tumours may be increasing which would balance out successful treatment (see Birch et al., 1980). This study has examined for survival to fifteen years of age rather than for a five

year survival from diagnosis so results from other studies may not be comparable.

The trends in these fatal disease rates are in keeping with other studies for school child mortality (Tait and Boog-Watson, 1962) but, as with the preschool group, there are very few long term community studies of mortality in this age group.

4.3.4(e) Patterns of pathology in deaths by age

Apart from the neonatal period, there is no characteristic pattern of fatal pathology specific to any age group. The data in this study on deaths by age allows standardised comparison with other communities and the patterns of fatal pathology may help to explain the trends in crude rates observed elsewhere. However, extrapolations from Sheffield to some other community should be treated with caution. Analysis of trends in fatal pathology by age will possibly overlook important factors because of variations in age at death. Therefore discussions of these trends has been deferred to section 4.4 where the incidence of fatal pathology has been analysed for the age groups at greatest risk.

4.3.5 Analyses of age-specific mortality by year of birth

Changes in a mortality rate can be due to changes in the numerator or in the denominator. It has been shown in Fig. 4 that the total Sheffield child population in any year fluctuated considerably during the period studied, ranging from 128,097 to 106,633 children (Appendix IV). Thus mortality rates would be expected to show a similar fluctuation even if the numbers of

deaths remained constant. To control this potential artefact, the age at greatest risk has been identified and only deaths in these high-risk age groups have been considered in calculating disease-specific mortality rates (sections 2.8 and 4.4). Cohort analyses, by following all of the children born in each year throughout their childhood, avoid the problem of a changing denominator because, in a cohort analysis, the risk population can be determined from the number of live births.

Epinatal and malformation deaths (discussed in sections 4.4.2 and 4.4.3) have been presented by year of birth as it is considered that events preceding or closely related to the time of birth are more important in understanding these deaths even if the death is delayed. An estimate of the date of conception can also be made from cohort study dates. Such calculations cannot be used in year of death studies. Events operating around the time of death were considered to be of more importance in the infection, SIDS, trauma and tumour deaths and these conditions have therefore been analysed by year of death.

Fig. 19 shows the proportion of survivors per thousand viable pregnancies for each year and demonstrates that there has been a gradual increase in the numbers of survivors in each cohort. There appears to have been no significant transfer of early neonatal deaths to stillbirths (see also Fig. 18). There has been no apparent overall increase in deaths among older children which might have been expected because of the greater number of survivors in each cohort (Fig. 19). There is no dramatic improvement in any particular period to suggest any significant change which may be attributed to medical or environmental advances (Fig. 19).

There is some variation in survival between cohorts. For no known reason, almost every fifth year (1951, 1961, 1966 and 1971) has an unusually poor survival record (Fig. 19). Other cohorts experience low mortality at most ages. For example, Fig. 26b shows that the 1959 cohort had a neonatal mortality rate of 11.5 which was lower than any subsequent cohort until 1968 (11.0) and was lower than the cohorts of 1970 (12.3), 1971 (13.5) and 1974 (12.0). This 1959 cohort also had a lower postneonatal mortality rate (4.2) than any other cohort until 1973 (3.6) and the late neonatal rate of 0.3 has not been approached since (the 1978 cohort being nearest with 0.7). By contrast, the 1961 cohort had a higher neonatal mortality rate (18.5) than any cohort after 1950, a higher postneonatal rate (7.0) than any cohort since 1954 and the highest preschool rate (5.0) of any cohort in the 33 years. There may be factors operating which influence the outcome of such cohorts despite medical intervention. There appear to be good and bad 'harvests' among these children and such effects should be considered when analysing the effectiveness of medical interventions.

Fig. 26a also shows age-specific mortality rates for each year of age, each year of birth and each year of death. The model illustrates the complex integration of the effects of age, year of birth and year of death upon each other. It confirms that certain cohorts had higher mortality rates than others and shows that analysis by year of death alone might erroneously attribute an increasing mortality to some local cause when it could be due to some predisposing factor carried since birth. The timing and spacing of pregnancies varies for each parent

couple. Therefore changes in the genetic stock will only become evident gradually and may be imperceptible on year-by-year analysis. In contrast, medical and environmental changes such as penicillin or clean air control are introduced relatively quickly and would be expected to effect a sudden decline in mortality. No such effect has been observed in this study.

All of the observations from these cohort analyses are compatible with the hypothesis that the major factor in the decline of child mortality in Sheffield during the period 1947-1979 was a gradual improvement in the constitution of Sheffield's children (see section 4.6).

4.4 Mortality Trends II - Deaths by Cause 1947-1979

This section will refer to the 7035 children for whom a cause of death was found. The 14 missing children are those whose death certificates were destroyed in the Sheffield Register Office fire on 21st February 1947 where no necropsy data could be traced to indicate a cause of death (see section 3.5).

4.4.1 Overall mortality patterns

The distribution of these 7035 deaths by cause in three 11-year cohorts (1947-1958; 1958-1968; 1969-1979) is shown as a proportion of the numbers of children alive in Fig. 28. These sector diagrams are drawn to the same scales as Fig. 7. In contrast to deaths by age (Fig. 7), the proportionate distribution of deaths by cause shows significant variations in time in the relative distribution of the major fatal pathologies. There was a considerable decline in infection deaths and a proportionate increase in malformation deaths. The proportions

of tumour, trauma and acquired deaths remained approximately steady and SIDS shows a slight overall increase in proportion (Fig. 28).

The distribution of each subgroup of diseases which make up the major pathology groups are shown in Fig. 29. Each diameter is proportional to the number of deaths from each disease per thousand Sheffield children alive between 1947 and 1979. The area of each segment in Fig. 29 therefore gives the relative contribution of each condition to overall child mortality.

The fatal incidence of each major pathology is shown in Fig. 30 as a rate per 1000 children alive in each year. The overall mortality rate shown in Fig. 6 was drawn to the same scale as Fig. 30; thus the decline shown in Fig. 6 can be seen to be primarily due to declines in infection and perinatal disease rates (see also section 4.3). The value of Fig. 30 is limited because the denominator is the total number of children alive whereas many fatal pathologies occur in limited age groups (Table I). For example the numbers of deaths in a year from prematurity cannot be closely related to the numbers of 15 year old children alive in the same year. Therefore the denominator has been limited to the population at greatest risk for each group of pathologies in the following sections. However, examining mortality as a proportion of all children alive enables relative priorities to be identified. It will be seen from Figs. 29 and 30 that the epinatal diseases are a more urgent problem to this community than, say, the malignant diseases.

4.4.2 Epinatal diseases

The fatal incidence of the epinatal diseases declined during the period studied. It can be seen from Table I that almost all of the epinatal disease deaths occurred in the first month. Deaths indexed under epinatal diseases in older ages were all children with cerebral palsy where there was an unequivocal comment relating to an aetiology following a perinatal event. The majority of these late deaths were certified as pneumonias with cerebral palsy and the perinatal event entered in part II of the death certificate. Such certificates are a major component of the 'incorrect word order' column in Table IV.

The epinatal disease deaths which occurred in the first month of life have been analysed by year of birth as a rate per thousand live births in each year. For consistency with other analyses in Section 4.4, the causal incidence of neonatal deaths is shown in Fig. 13 by year of death. Analysis of these data by year of birth produced an almost identical graph and has not been reproduced. In Fig. 13, the prematurity and obstetric curves showed reciprocal dips and surges in the years 1950-1953 and 1955-1961. To examine for possible diagnostic fashions, the constituent diseases in the obstetric group were analysed separately (Fig. 14). The rises in placental complications mirror in time the falls in the prematurity mortality rate shown in Fig. 13. The disease label principally involved in the surges of placental complications was 'placental insufficiency, cause unknown'; mortality from other placental complications such as placenta praevia showed no such increases. There has possibly been some interchange between the diagnosis of

prematurity and placental insufficiency, although why this should have emerged as a fashion at some times is unknown and if there was such a fashion, why it occurred in JHW one year after NGH (Fig. 15) is also unknown as there was no formal rotation of staff between these hospitals.

The decline in neonatal mortality from birth trauma since 1963 (Fig. 14) occurred principally in first day deaths (Fig. 17). This decline is primarily responsible for the overall decline in first day mortality (section 4.3.1). If this decline in birth trauma occurred nationally, it would refute the suggestion that the national decline in first day mortality rate since 1963 could be associated with an assumed introduction of special care baby units (Pharoah and Alberman, 1981). The reduction in Sheffield first day mortality rate may relate to the increasing proportion of hospital births after 1963 (Fig. 16). An association seems likely between a reduction of deaths between birth trauma deaths (especially intracranial haemorrhages) and increased hospital supervision of deliveries. If so, it may have serious import in the 'home-or-hospital delivery' debate.

4.4.2(a) Rhesus isoimmunisation

This section deals only with deaths from isoimmunised babies that were live born. No data were available on stillbirths or survivors. Fatal rhesus disease among live born babies showed a relatively steady incidence which was little affected by the introduction of exchange transfusion in Sheffield in 1948-1949 nor the subsequent introduction of other therapies (Fig. 14). The rise in death rate in the first few years after the introduction of exchange

transfusion might be due to the acquisition of experience (see below), but the numbers involved are small. This increase in death rate might equally be due to increasingly vigorous obstetric management resulting in the premature delivery of affected babies who might otherwise have been stillborn.

Anti-D would not be expected to prevent the birth of isoimmunised babies to women who were already sensitised because anti-D works by preventing sensitisation. Thus a lag period would be expected until the majority of sensitised women cease bearing children before the effect of anti-D could be appreciated in the community. Meanwhile no more women should become sensitised. In Sheffield the principal reproductive years are from 19 to 34 (OPCS, 1979b); thus the expected lag period would be 15 years. It is therefore significant that the fatal incidence of rhesus disease began to decline eight years (and fell to zero 15 years) after the introduction of anti-D (Fig. 14). The prophylactic use of anti-D appears to have been more effective in reducing mortality among live-born Sheffield children than exchange transfusion or other treatments.

Other studies of anti-D efficacy have examined the reduction of the numbers of affected infants, including stillbirths and survivors (see Clarke and Whitfield, 1979). Such study designs are preferable for examining the efficacy of a treatment but the present study's primary objective was to understand the pattern of death in childhood. A problem with interpreting studies of affected infants is

that many have used central indexes. Clarke and Whitfield (1979) showed that there is some inaccuracy in the recording of Rhesus disease and many other non-Rh isoimmunised children may be included. Anti-D cannot be expected to prevent such conditions.

In 1947 Diamond reported that repeated transfusions of Rh-negative blood reduced the case fatality rate in rhesus isoimmunised babies from 40% to 30%. Delivery at 37-38 weeks gestation together with Rh-negative transfusions lowered the case fatality rate to about 20% and, following the introduction of exchange transfusion, the case fatality rate dropped to about 10% (see Clarke, 1975). Case fatality rates cannot be calculated for Sheffield children because there is no address-verified record of the numbers of affected children born. Figures are available for the period 1948-1972 for the outcome of pregnancies of Sheffield women who attended Welfare clinics but not for the remaining Sheffield women (Sheffield 1947-1973). The numbers are available of rhesus sensitised pregnant women delivered in Sheffield hospitals after 1975 via the Trent Regional Health Authority Hospital Activity Analysis. Correction factors are also available to estimate the numbers of such women who were Sheffield residents. These data are collected and recorded by untrained clerical staff. The recorded numbers of rhesus isoimmunised women delivered in NGH rose sharply between 1977 and 1978 (from 30 to 722 per year), in sharp contrast to the numbers in NEH and JHW (16 to 34 and 54 to 43 per year respectively). There is some centralisation of

rhesus affected pregnancies in NGH, but not to this extent. The estimated number of NGH deliveries of Rh-positive pregnant women (who cannot become rhesus isoimmunised) was about 750 in 1978. A clerk may have recorded these Rh-positive women in mistake for rhesus sensitised women. Whatever the cause and even if an artefactual error were proved, it is understood that these records will not be altered. Such factors must be considered when interpreting studies which used centrally indexed records.

Central indexes can be corrected for false positives by re-examination of hospital notes (see Clarke and Whitfield, 1979) but studies based on central indexes will be unable to identify false negatives. The present study was able to identify such cases if a necropsy had been done.

There were a number of neonatal deaths registered under rhesus isoimmunisation where serious medical accidents occurred which were not mentioned on the death certificate. There were deaths due to hypothermia following prolonged exposure during exchange transfusion or the use of blood straight from a fridge. Some children died with massive air embolism, others were given blood which had been heated so rapidly as to cause lysis (in one a potassium level of 34 mg/l was found). There were no deaths due to transfusion 'reactions' or inappropriate transfusions but one child died following transfusion with six-week-old blood. All of these children were registered as dying from rhesus disease and the medical accidents were not mentioned; the

misadventures emerge from the necropsy report. It is not known if any babies who did not have a necropsy suffered similar accidents. It could be argued that such deaths should be indexed under medical misadventure (or unnatural causes). However, because of the uncertainty of completeness of necropsy reporting and because these accidents would not have occurred had the children not been isoimmunised, these deaths have been indexed for the present study under rhesus disease. This could explain why the death rate rose following the introduction of exchange transfusion (Fig. 14).

Reports from California, Australia and Finland have all shown that increasing experience of exchange transfusion had little appreciable effect on the mortality rate from rhesus disease whereas the introduction of anti-D gamma-globulin was rapidly followed by a reduction in the number of affected women and subsequently in the mortality rate (see Clarke, 1975). Sheffield's experience would support these reports.

4.4.2(b) First week deaths from prematurity

The argument in favour of intensive neonatal care facilities (SCBU) is powerful and emotive; tiny premature babies are fighting for life but dying or becoming brain-damaged because of their immaturity. They need warmth, food and oxygen and the technology is available to fulfil these needs. These arguments and claims have been advanced for the past 20 years (see Dunn and MacGregor, 1964; Pharoah and Alberman, 1981). The argument against SCBUs is that these expensive units have so far failed to live

up to their promise (see Davies, 1980). All studies of the effectiveness of these units have been hospital-based and problems inherent in such studies have been discussed in Section 1.1. However, it has been shown that the handicap rate among survivors of very low birth weight (VLBW) did not alter in 15 years in one hospital (Jones et al., 1979); that the crude mortality rates among VLBW babies in a hospital with no expensive facilities were at least as good as those in a superbly equipped teaching hospital SCBU (Hughes-Davies, 1979); and that outcome in survival or handicap rates at school age among VLBW children in a hospital where the baby unit was staffed only by nurses was as good as those from a London teaching hospital (Steiner et al., 1980). Each of these studies has been criticised by neonatologists whose arguments have been reviewed in Section 1.5.1(a). The studies quoted above may have been done before adequate technologies had become available. But whether that technology has yet been perfected is debatable and the above studies have all examined outcome after the claims for SCBUs had been advanced in the 1960s. In the meantime, vast monies have been expended on units which have yet to show conclusive evidence of benefit to the communities who have paid for them.

Death rates from prematurity in Sheffield maternity hospitals are shown in Fig. 15, standardised for hospital deliveries because of the changes in place of birth (Fig. 16). Home-delivered babies showed a very low mortality rate throughout but this will have been biased by the

numbers of multiparous births so they have been omitted from Fig. 15 for clarity. The causes of death and addresses of the dead children were standardised by the method described in Section 2. The diagnosis 'prematurity' is not specific, but covers adequately changing diagnostic fashions throughout the 33 years and conveys to most doctors the concept of a specific condition. It has been found to be a reasonable label in the present study but the diagnostic limitations discussed in Section 1.3.2 should be borne in mind.

The decline in prematurity mortality rate began in JHW before a SCBU was established in 1948 (Fig. 15) and the overall decline shows no appreciable change following the provision and upgrading of this unit. The prematurity mortality rate at NGH has shown an even greater overall decline although this hospital has never had the staff or the facilities to compare with JHW. Unfortunately information on ethnic and social class distribution of Sheffield live births is not available although it is not doubted locally that NGH has always served the most disadvantaged and would therefore be expected to have the highest prematurity, VLBW and mortality rates. The rise in NGH prematurity mortality rate in 1973-1974 (Fig. 15) is associated in time with the arrival of the babies of Asian immigrants, most of whom are delivered at NGH. The NEH was upgraded from a GP maternity unit in 1969 when a purpose-built maternity unit was opened. Therefore the low mortality rates in NEH in the period 1947-1968 may be a

reflection of the low-risk pregnancies delivered and the subsequent rise during the 1970s is probably due to an increase in the numbers of high-risk pregnancies being delivered.

The comparisons of prematurity mortality rates by Hughes-Davies (1979) and Steiner et al. (1980) between their hospitals and a London hospital could have been biased by some unknown geographical factor - Fig. 27a shows that neonatal mortality rates differ and fall at different times in different parts of the country. The present study avoided this problem by examining a single community. The differences between maternity units (Fig. 15) cannot be due to a concentration of high-risk babies at any one centre as Sheffield's obstetricians and paediatricians did not transfer such babies between units. It is therefore disappointing to find that the hospital equipped and staffed to provide the best service for premature babies (JHW) has not produced a more striking fall in prematurity mortality rate than the other hospitals. Hopefully the new intensive care SCBU at JHW (opened late in 1979) will produce a further fall in the prematurity mortality rate but such an effect cannot be monitored by the present method because sick neonates are now transferred to JHW from the other hospitals. Before any further reduction can be attributed to these new facilities it will have to be shown that the rate of decline in the citywide prematurity mortality rate is greater than that which might have occurred had the established trend continued (Fig. 40).

The present study has been unable to standardise for parity, birth weight, ethnic origin or social class yet the biasing effect of such factors can be seen in Fig. 15 which shows that NEH with no neonatal facilities in 1947 achieved a lower mortality rate than JHW did in 1972 although both hospitals are ostensibly serving the same community. The hospital-specific mortality rates (Fig. 15) could have fallen as fewer babies were delivered at home (Fig. 16) because these were presumably low-risk pregnancies. However, this would not explain why the citywide prematurity mortality rate fell (Figs. 13 and 40). It could be argued that even the facilities in JHW were suboptimal during the period studied and that comparisons between this and other hospitals are invalid. If so, there is then no obvious medical explanation for any of the decline in the Sheffield prematurity mortality rate shown in Fig. 13. This decline may have begun as early as 1914 (Fig. 40) and no synthesis of medical factors has been found to explain this. In Section 4.6 an hypothesis will be developed which suggests that the decline in prematurity mortality rate may be due to insidious biological changes in succeeding generations of Sheffield's children.

An important biological factor which could explain the decline in prematurity mortality rate in Sheffield is the decline in the numbers of live born VLBW infants which has been observed at least from 1962 (Gordon, 1977a). There has been a disproportionately large fall in the numbers of live-born infants weighing less than 1500 g who have always

made an excessive contribution to the neonatal mortality rate. Gordon (1977a) gives no explanation for this finding but it may be due to a general improvement in the health of Sheffield's mothers, similar to that predicted by Kermack et al. (1934) and observed elsewhere by Baird (1980). These findings are in keeping with an interpretation of the present cohort studies (Section 4.3.5) that a major factor in the decline of Sheffield's child mortality could be a gradual improvement in the constitution of Sheffield's children which may be due to a previous improvement in the intrauterine environment. There would appear to be factors operating in this community which are far more important than medical interventions. This need not mean that SCBUs are unimportant, merely that social, biological and other changes are so powerful that the effect of the medical technologies cannot be appreciated and such non-medical factors should be sought before attributing improvements solely to medical advances.

It is not possible to use the present data to argue against the further provision of SCBUs without explaining also why coronary care units (CCU) have been maintained. Mather et al. (1971) showed that there was no evidence to suggest any medical gain in admission to hospital with a CCU compared with treatment at home. The cost to the community of both SCBUs and CCUs should be balanced against their benefits. A health service with limited resources will have to limit facilities in some area and SCBUs and CCUs are provided to the possible detriment of geriatric, mental handicap or other services. With limited funds, the

maximum community benefit should override individual needs. Statistics of the mortality or handicap rate for a hospital are of value to parents seeking the best treatment for their child and are justifiable where these parents pay for these services but have no place in a scientific assessment of resource allocation.

If the relative merits of SCBUs and CCUs were to be debated, SCBUs have one advantage over CCUs in that any factor which enhances the quality of life before child-bearing may be expected to produce an improvement in the health of the next generation (Baird, 1980). Therefore relatively short-term studies of benefit from SCBUs may be examining the wrong outcome. However, inflated claims of benefit should be treated with caution. At the time of writing the conclusions of Davies (1980) and Gordon (1977b) still hold true for Sheffield: "frequently repeated statements that modern methods of perinatal intensive care are leading to a striking reduction in handicap must at present be considered uncritical" (Davies, 1980) and "There is no evidence to suggest that superintensive neonatal care has improved survival for very lightweight babies" (Gordon, 1977).

There would appear to be an urgent need for a population-standardised controlled trial of the outcome of a highly equipped and fully staffed SCBU compared with a premature baby unit staffed only by trained nurses. Objections to such a trial could be advanced by both sides of the SCBU debate. However, objections concerning

mortality could be unfounded in the light of the present study.

4.4.2(c) Cerebral palsy deaths

The aetiology of cerebral palsy is multifactorial; some are due to congenital defects, some to perinatal accidents and the majority unknown. Analysis of the trend in deaths under two years with cerebral palsy shows a steady and significant decline (Fig. 36). This decline does not show any change which might be associated with changes in obstetric or neonatal management. However the apparent rise in the late 1970s may be a consequence of increasing management of premature infants who might otherwise have died but it could equally be an artefact of small numbers. The trend in the decline in cerebral palsy mortality is similar to the rate of decline of prematurity mortality. Both could be due to improvements in the general health of Sheffield's infants and mothers.

4.4.3 Malformation deaths

Congenital malformations were the third commonest cause of Sheffield child deaths between 1947 and 1979 (Fig. 29) but, in contrast to the slightly commoner infection deaths, showed little change in fatal incidence with time (Fig. 30). About one-third of these malformation deaths were due to congenital heart disease, a quarter to neural tube defects and one-fifth to chromosomal defects. The remaining fifth are approximately equally divided between alimentary, respiratory and renal malformations (Fig. 29). Surgical correction is possible for the majority of alimentary and cardiovascular malformations. Surgery is also possible for spinal defects although it is now debatable whether the outcome of such surgery may lead to normal life. At the time

of writing there is no curative treatment for the majority of chromosomal defects but hopes of genetic manipulation with bone-marrow transplantation may prove successful. Treatments are available for some of the renal and respiratory malformations but there has been no successful surgery for renal agenesis or pulmonary atresia to date; transplant surgery may hold hope for the future but such operations are not without cost to the donor.

The relative incidence of deaths from malformations as a proportion of all child deaths has increased in the 33 year period (Fig. 28). This is more due to a decline in infection deaths than a true rise in the rate of malformation deaths (Fig. 30). The great majority of these malformation deaths occurred in the first two years of life (Table I). Therefore the number of children alive each year in this period has been taken as the population at risk and the two-year mortality rate from malformations calculated for each year. Having taken the two-year period it is possible to examine mortality rates by year of birth up to the 1978 cohort. Had all malformation deaths under 15 years been scrutinised, apart from problems with the varying risk population (Fig. 4), the trend could only be examined until 1964 because subsequent cohorts have yet to reach 15 years. It is reasonable to assume that children dying in the first two years are almost all Sheffield born (see Section 1.1.3) whereas there is no evidence available to extend this assumption to older children.

4.4.3(a) Two year death rates from specified malformations

With the exception of neural tube defects (which will be discussed separately), all congenital malformations show

a steady death rate in the first two years of life (Fig. 31). As explained above, these trends can be followed to 1978; children born in 1979 had not reached their second birthday at the time of writing so the final mortality rate for this cohort may be higher.

The mortality rates for the conditions examined show no apparent effect of the medical and surgical advances which have occurred in the past 30 years. This is initially puzzling because many clinicians report that these advances have been associated with a reduction in the number of deaths (see Anonymous, 1981). In the present study, the numbers of deaths have fallen but so have the numbers of children born hence there is a steady mortality rate. The present study has examined deaths in a defined community, not hospital deaths. A serious distorting factor which can affect all hospital-based studies is that there is no control over the population from which patients may be drawn (see Section 1.1). Thus a hospital which develops a successful treatment may draw patients from an increasingly large area and, depending on the severity of the condition of these new patients, the mortality in the hospital may alter without any further change in treatment. If another hospital then drains off some of these patients, the original hospital's mortality rate may again alter without any change of treatment.

The present study cannot examine survival or quality of survival. The new treatments may have affected both. For example a child with congenital heart disease born in

1947 who developed an Eisenmenger complex and died in the later teens or early adulthood would not have been identified in the present study. A similarly affected child born in 1979 might be successfully treated and lead a normal life.

No information on the causes of stillbirth were available to the present study. Thus the apparent slight increase in lethal renal malformations (Fig. 31) may be due to changes in registration. However, this study has found a five-fold increase in incidence of renal agenesis among liveborn Sheffield babies in the period 1975-1979. There is some experimental evidence to implicate environmental teratogens in the aetiology of this condition, some of which are produced in steel manufacture. The numbers of babies involved are small therefore further studies including all deaths and in collaboration with other areas will be needed to determine whether this trend is significant.

The low incidence of lethal fibrocystic disease are mostly deaths with meconium ileus. Throughout the study period there were few such deaths from respiratory or cardiac complications under 15 years. This study could not determine the numbers of children crippled by respiratory infections or cor pulmonale and thus the effect of antibiotics or pancreatic extract cannot be examined. There was no significant rise in incidence during the 1950s due to increasing recognition of the disease therefore the method employed to control this potential artefact appears to have been effective (see section 1.6). A separate analysis of the age at death from fibrocystic disease for cohorts born

in 1947-1964 (who had completed 15 years by the end of the study period) showed no increase in the mean age at death. These results have not been presented because it is not known how many affected children were born in each year nor is it certain that children dying in their teens were Sheffield born. It would appear that any study which proposes to show a benefit of recent advances in the treatment of cystic fibrosis will have to examine older children or use morbidity data.

4.4.3(b) Problems with the congenital abnormalities register

The Sheffield Congenital Abnormalities Register (CAR) was consulted in the hope of identifying all children with malformations so that disease-specific case fatality could be calculated. However, with the single exception of gross abnormalities recognisable at birth, it was found impossible to use the CAR to calculate birth or fatality rates because of considerable underreporting of the numbers of affected children. There was even some underreporting of such gross malformations as anencephalus: there are death certificates for two infants with this condition born in 1979 yet the CAR records none live or stillborn. The CAR is also of limited value in calculating an overall birth rate of malformed infants because of great variation in the severity of the conditions recorded: suspected systolic murmurs and clicking hips are documented as carefully as Fallot's tetralogies and dislocated hips; squints, skin tags and respiratory distress syndrome have been recorded along with absent kidneys, spina bifida and anal agenesis. Internal malformations are

difficult to detect and the staff who maintain the CAR have done an excellent job in scouring medical records for the details obtained. It is perhaps unfortunate that they have been so successful.

Following the 1974 local government reorganisation, the CAR staff were allocated additional administrative duties. This may explain the as yet unsubstantiated reports that the numbers of abnormalities registered nationally fell by half after 1974. In the present circumstances it seems that these valuable registers should be treated with caution for conditions which cannot be recognised at birth.

4.4.3(c) Birth and death rates from neural tube defects

The Sheffield birth rate for neural tube defects remained steady at about 3.5 affected live born babies per thousand live births from 1963 (the earliest record) until 1976 (Fig. 32). The introduction of amniotic fluid alpha foetoprotein (AF-AFP) screening and subsequent abortion for previously affected families in 1973 had no significant effect on the birth rate. This may be because of poor uptake of the test or because previously affected families contribute only 5-10% of the total number of affected pregnancies. The introduction of screening all pregnant mothers (MS-AFP) with subsequent abortion if positive in 1976 is associated with a dramatic decline in the birth rate of neural tube defects (Fig. 32). This would suggest that uptake of the test is high in Sheffield. There is a residual birth rate of affected children which could be due to two factors: there will be a continuing incidence of

closed lesions which cannot be detected in utero and, because the confidence limits for AFP levels are chosen to avoid the tragedy of aborting a healthy foetus, it is likely that a small proportion of open lesions with low AFP levels will not be identified. There may also be occasional laboratory errors. The procedure of screening and abortion has not produced a true reduction in the incidence of these lesions but merely transferred the delivery of the foetus to an earlier age where it is not notifiable and thus become unidentifiable. Interpretations of historical data suggest that the incidence of these lesions is declining (OPCS, 1976b). If this is so, it is important that detailed records are kept of the lesions in the abortuses lest a stage is reached where more harm is done to normal foetuses by the screening procedures than the benefit gained by this eugenic procedure.

Prior to the introduction of a CAR, the incidence of these lesions was inferred from mortality figures. This assumed that all affected children died in infancy. Rogers and Weatherall (OPCS, 1976b) took this assumption to the extreme of publishing a graph of incidence of spina bifida calculated from the numbers of deaths per thousand live births (i.e. the mortality rate). Comparison of the neural tube defect mortality and birth rates in Sheffield after 1963 (Fig. 32) shows that there is a very poor correlation which suggests that the assumption that all affected children die in infancy may be erroneous.

The neural tube defect mortality rate (Fig. 32) shows considerable variation which contrasts with most other malformation mortality rates (Fig. 31). The neural tube defect mortality rate declined after 1953 until 1970 (Fig. 32). The new surgical techniques may have increased mortality while experience was gained or children born in 1963 and 1964 may have had more lethal lesions. The introduction in May 1971 of selection prior to surgery with no treatment for severely affected infants is associated with a reversal of the mortality rate trend which then continued to rise until AFP screening and abortion artefactually reduced the numbers of affected children. Lister (1973) reported that some Sheffield doctors objected to selection prior to surgery and referred affected children directly to surgeons who continued to offer surgery-for-all. He showed that selection was not introduced for all children for some time after 1971. This must cast doubt on a causal association between the introduction of selection and the reversal of the mortality rate shown in Fig. 32.

Because of the artefactual effects of a changing risk population and because the numbers of liveborn Sheffield children with spina bifida are known with some certainty since 1963, two-year case fatality rates have been calculated for each subsequent cohort (Fig. 32). This shows an increase from 30% to 65% in the proportion of affected children dying after the introduction of surgery-for-all in 1963; a decline to 6% by 1967 and then a subsequent steady rise which has not been appreciably influenced by selection

or AFP screening. Because of the small numbers involved, it is preferable to consider the three year moving mean (broken line in Fig. 32). One might expect selection to have increased the proportion dying as severely affected babies would not be considered suitable for surgery; conversely one might expect screening to have removed the most severely affected foetuses and those born alive to be less likely to die.

Cusum analysis (Johnson and Leone, 1964) of both mortality and case-fatality rates using deaths under 1 week, 3 months, 6 months, 1 year and 2 years identify that significant changes in spina bifida death rates occurred at all of these ages in 1967 and 1972. Chi-squared testing confirms that the differences in the case fatality rates are significant at the 0.1% level on both occasions with the higher proportion surviving in 1967-1971. There was no significant alteration in case fatality rate in 1971 (the year selection was introduced) compared with previous years, nor after the introduction of either screening programme. The statistically significant changes which occurred after 1972 could be associated with selection prior to surgery if there was irregular local application of selection as suggested by Lister (1973). But if this were the explanation one would expect a gradual transition, not the sudden change observed between 1971 and 1972. No satisfactory explanation has been found for the change which occurred in 1967. The case-fatality curve in Fig. 32 could be read as a square wave with high rates in 1963-1966, low rates in

1967-1971 and high rates in 1972-1979; this is in keeping with the statistical analysis. If so, it may be that once the initial surgical problems had been overcome there was a low mortality rate until selection was widespread. However, this interpretation is speculative and not consistent with the dates of changing management. It remains possible that all of the observed changes in spina bifida mortality rates could be due to other unknown, non-medical factors.

Rogers and Weatherall (OPCS, 1976b) show that the spina bifida mortality rate for England and Wales has shown three periods of increase (1930-1940; 1945-1949; after 1969) superimposed on a general downward drift. They suggest that the national change after 1969 was due to widespread introduction of selection although this is not consistent with published dates. The weight of evidence reviewed by Rogers and Weatherall (OPCS, 1976b) supports the view that the aetiology of these deformities is probably due to environmental influences acting on a genetically susceptible subsection of the population. The changes in mortality rates in the previous periods are as great as those seen after 1969. No medical factors were identified to explain the earlier changes and all could be due to unknown factors.

Examination of the proportionate distribution of the spina bifida deaths by age (Fig. 33) shows that there has been little change in the proportion dying on first day. The proportion of deaths in the first week and first month remained relatively steady until 1969 and then rose rapidly. This may be due to increasing deaths following selection.

There was another small peak of increasingly young deaths between 1960 and 1970 which could be due to a number of operative deaths in early life with decreased mortality after the first year. There were no deaths in the 1-15 year age group between 1947 and 1952 which might support the assumption of Rogers and Weatherall (OPCS, 1976b) that all affected infants died in infancy. The cohorts born after 1964 have not yet reached their sixteenth birthday thus limiting interpretation of the second part of this figure. This figure is also limited by variations in the numbers of children born each year and by the artefactual effects of screening and abortion.

The survival curves shown in the bottom half of Fig. 33 cannot be extended earlier than 1963 because the numbers of affected babies born are not known. These survival curves show that 90% of the liveborn affected babies survive their first day whereas the introduction of selection was followed by a rapid decline in the proportions surviving beyond one month. Screening and abortion (coupled with selection) did not affect the proportions surviving to one month, although the numbers were sharply reduced (see Fig. 32). Affected babies surviving the AFP screen are not likely to survive their first year because selection is still applied.

The management of spina bifida was revolutionised by advances which were pioneered in Sheffield. The timing of these changes in management are known accurately. An attempt has been made to determine precisely the effect these changes had on mortality from this terrible condition.

It has not been possible to show with confidence any definite effect. Although it is probable that surgery-for-all reduced mortality and that selection increased it, other non-medical factors cannot be excluded. As has been discussed already, the present study can make no comment on the quality of life enjoyed by survivors.

4.4.4 Infection deaths

In 1947, 203 (38.7%) of all 524 Sheffield child deaths were due to infections. In 1957 the figure was 23 (10.6%) of 216; in 1967, 17 (8.3%) of 205 and in 1977 4 (3.4%) of 116 deaths. Except in the neonatal period, infections formed a major proportion of Sheffield child deaths (Figs. 12, 20, 22 and 24). Infectious diseases were the single most common cause of child death in 1947 (Fig. 30) yet fell so rapidly that, averaged over the 33 year period, they are only slightly more important than malformations as a cause of death (Fig. 29). The dramatic fall in infection mortality rate was so rapid that by 1960 these deaths had become nearly as uncommon as tumour deaths and less important than all other causes of childhood mortality (Fig. 30).

Semilogarithmic analysis of the mortality rates shown in Fig. 30 reveals that there are two components to the rate of fall of the infection curve: a rapid fall from 1947 to 1952 and a more gradual but steady fall from 1953 to 1979. The rate of fall of infection mortality since 1953 has been as great as, but with smaller numbers than, the overall fall of the epinatal disease mortality rate (although the latter has fallen more rapidly since 1962).

The decline in infection deaths was the main reason for the declines observed in all non-neonatal age-specific mortality rates (Figs. 21, 23a and 25). The most important fatal infections were gastroenteritis and pneumonias (Fig. 29). The majority of these infection deaths occurred in the age group 1-23 months and, for the reasons already discussed, only this high risk group will be considered in further detail.

4.4.4(a) Fatal infection trends among children aged 1-23 months

Among this age group all fatal infection rates show some reduction during the 33 years studied. Acute infections of the respiratory tract (AIRT) will be discussed in the section on SIDS because they were deemed to be an inadequate explanation for death. With the exception of AIRT and meningococcal infections, all fatal infection rates showed a decline early in the period studied (Fig. 34).

The largest and most rapid fall in mortality rate was in gastroenteritis infections with pneumonias also showing a large rapid fall (Fig. 34a); other infections showed less dramatic falls (Fig. 34b). There are many possible explanations for these falls in mortality rate but closer examination reveals that few are tenable. It is unlikely that the introduction of antibiotics played much part in the decline in fatal gastroenteritis. The development of NHS hospitals with paediatrically trained staff administering intravenous fluids may have been responsible but the decline appears to have begun before the advent of the NHS (Fig. 38). Antibiotics may have played a part in the decline in fatal

pneumonias but, although they were available in hospitals after 1947, they were not freely available on general practitioner's prescription until 1957 (Lunn, personal communication, 1980). As many of the pneumonia deaths in this study occurred at home, the antibiotics may not have been reaching these children and, in any case, the decline in pneumonia mortality appears to have begun long before this (Fig. 38). The decline in fatal pneumonias (Fig. 34a) occurred at a time of high atmospheric pollution (Fig. 43). Any beneficial effect of clean air came too late to have any significant effect on pneumonia mortality, although it may be associated in time with an increase in AIRT mortality (Fig. 34b).

BCG and pertussis immunisation cannot be the sole explanation for the declines in fatal tuberculosis and whooping cough (Fig. 34b). It would be true to say that there have been almost no deaths after the introduction of these vaccines but it is not possible, in this community, to say that the decline in mortality rate was due to the introduction of vaccination. Tuberculosis affects the very young as miliary TB or as tuberculous meningitis and then, for unknown reasons, it is not a major disease until the late teens or early adulthood. The present study could not examine mortality trends in this older age group nor could it examine morbidity. Specific chemotherapy and BCG vaccination may have had profound effects on the health of Sheffield's children but this could not be shown in the present study. The observed decline in TB mortality rate

appears to have begun before 1947 (Fig. 38) and could be due to some powerful non-medical factor.

The benefits and risks of pertussis immunisation have been much discussed following the suggestion of an association between this immunisation and infantile spasms (see Dick, 1974 and Robinson, 1981). The lay press pursued this claim uncritically and gave it wide publicity and subsequently immunisation rates fell dramatically (McKendrick et al., 1980). The present study has discovered two facts of value to the debate: pertussis mortality rate in this community fell before the introduction of immunisation and, secondly, deaths reappeared in 1963, 1966, 1967 and (following the immunisation decline in 1974) there was a minor epidemic of fatal pertussis in 1974-1976 (Fig. 34b). Pertussis vaccines in use in this country are not identical and have been modified since their introduction (Griffith, 1978). The deaths in the 1960s could have been among incorrectly or unimmunised children. The 1974-1976 deaths were all in young unimmunised children, many of whom had unimmunised siblings. Pertussis immunisation would appear to afford protection to these young children.

The slight steady decline in bacterial meningitis mortality rate (Fig. 34b) shows no sudden fall which might be expected following the introduction of antibiotics. Almost all of these children died in hospital where antibiotics were available in small quantities even in 1947. The organisms involved were of all types with Haemophilus and Pneumococci predominating (tuberculous meningitis deaths were included in the TB curve).

The meningococcal mortality rate also shows only a slight decline overall with no apparent antibiotic effect (Fig. 34b). A possible reason for this is that meningococcal septicaemia can be so fulminating that an affected child may die before receiving medical attention. Children who survive long enough to manifest non-purpuric signs may be biologically different from those who succumb.

The declines in fatal infection rates observed in this study (Fig. 34) are both dramatic and highly significant. These declines are usually attributed to the introduction of antibiotics or effective vaccines. However, examination of the trends suggested that these declines had begun before 1947. This was one reason for extending the 1947-1979 necropsy-verified study to include unverified historical data on earlier Sheffield child deaths. These historical mortality rates are discussed in Section 4.5. If the observed declines were due to the introduction of antibiotics, it would be expected that the mortality rates would not fall until after their discovery in the 1940s. If they were due to vaccines or environmental controls then the declines would not be expected to begin until after their introduction. It has already been shown that the decline in pneumonia death rate occurred before clean air legislation and the declines in fatal TB and pertussis occurred before the introduction of vaccination. It remains possible that all of these observed declines may have been due to other, non-medical factors.

4.4.5 Sudden infant death syndrome

A purely clinical definition of SIDS is dependent upon parental and professional perception and such a definition, based only on history, would embrace many malformations and possibly many florid infectious diseases which were not perceived or had not become manifest. This might explain variations in the reported incidence of SIDS (Peterson, 1980). Much medical diagnosis is of necessity expedient. However, only the consistent application of reasonably defined criteria will make it possible to identify features common to a disease which may then allow preventive or therapeutic action. It is therefore necessary to identify as clearly as possible the criteria for definition of a disease. The clinical defintion of SIDS will fail to do this.

A pathological definition of SIDS would include many deaths which are explicable from the history because many fatal diseases (e.g. diabetic ketoacidosis) may not be manifest in the body after death. All studies based purely on necropsy reports may also be confounded by the fact that death is not instantaneous - some cells take many hours to die after the cessation of heart or brain stem activity. Apparently significant changes found at necropsy may have occurred terminally but secondary to the lethal event. Such findings could be misinterpreted as the cause of death (see Simpson, 1947). Autolytic processes also produce changes which may be misinterpreted as antemortem pathology and minor pathological changes could occur antemortem but after an unrelated lethal

event has occurred. It is therefore not surprising that Froggatt et al. (1971) and others have found special necropsy studies disappointingly unhelpful in elucidating an aetiology for SIDS. Such necropsy studies are essential for a consistent definition but the aetiology (or aetiologies) of this condition seem more likely to be identified from studies of pathophysiological mechanisms in the living.

The definition used in the present study (section 2.3.11) is not perfect because there would be different interpretations as to what is an adequate explanation of death. Most clinicians can agree as to what diseases might be expected to be fatal. Most pathologists likewise can agree as to what might be acceptable as an explanation for death. But the lists of diseases agreed by the clinicians and the pathologists may not be congruent. This problem was overcome in the present study by a single observer with clinical, pathological and forensic paediatric experience considering each death. All data on the 7049 deaths were classified in a six-week period (section 2.3) in an attempt to reduce intraobserver variation. There are few other studies where only one department examined all children with SIDS (see Kukull and Peterson, 1977). Studies of SIDS involving large numbers of pathologists will have potentially large interobserver variations as to the definition of SIDS. Because of these variations in definition, the results of the present study may not be exactly comparable with other work. No other report has been traced which studied the time-trend of SIDS over such a length of time and very few

reports have been found where SIDS was defined by a single observer examining all of the data on each death (see Kukull and Peterson, 1977). In the present study, the incidence of SIDS was found to be 2-4 such deaths per thousand children at risk (Figs. 21 and 36). This is similar to the incidence in Belfast in 1965-1967 (Froggatt et al., 1971) and other international studies (see Peterson, 1980).

The results of the present study have been shown standardised for the postneonatal age group (Fig. 21) and for the 1-23 month age group (Fig. 36) because of variations in the risk age group in other published work (see Froggatt et al., 1971; Peterson, 1980).

Some of the studies reviewed by Peterson (1980) would exclude deaths with AIRT because these studies defined SIDS as the absence of all pathology. Valdes-Dapena (quoted in Peterson, 1980) discusses this problem and states that such findings are present in at least 50% of sudden deaths of seemingly well infants. Clinical experience shows that the great majority of three month old children may have an AIRT without ill effect (Sheffield Child Development Study, unpublished data). The death rate among children in the present study where AIRT was the only pathology found is shown in Fig. 34b on the same scale as the SIDS rate in Fig. 36. Removal of the AIRT deaths from the SIDS curve would produce a steady incidence throughout of about 0.8 deaths per thousand at risk which is very similar to the lowest reported incidence in the world (0.6, see Peterson, 1980). SIDS may be a single

condition which affects children world wide. It seems probable that variations in the reported incidence of SIDS are due to variations in definition. Variations in the time trend of SIDS in Sheffield (Fig. 36) may also be due to variations in ascertainment (see section 4.3.4(b)).

The fluctuations in the Sheffield SIDS rate between 1947 and 1979 (Figs. 21 and 36) were almost entirely due to fluctuations in AIRT mortality rate (Fig. 34b). The dramatic reversal of the upward trend in SIDS rate after 1972 has been related to the Health Visitor project already discussed (section 4.3.4(b)). Another reversal in SIDS rate occurred in 1957-1958 which remains unexplained. If SIDS were due to fulminating infections which were so rapidly fatal that classical signs did not become manifest, it might be expected that the SIDS and the overall infection mortality rates would show similar patterns with time in a defined community. Both rates showed similar declines in the mid 1950s (Fig. 21; and compare Figs. 34b and 36). However, the overall trends in SIDS and infection mortality rates show very different patterns which suggests that some other aetiology applies to SIDS.

The rise in SIDS rate in the 1960s might be due to a change in diagnostic label from gastroenteritis or pneumonia (Pharoah and Morris, 1979). The mortality rates for gastroenteritis, pneumonia, AIRT and SIDS (Figs. 34 and 36) show that there are no major reciprocal variations which would be expected if Pharoah and Morris (1979) were correct. It is

possible (but unlikely from the necropsy reports) that what was once called pneumonia might have become labelled AIRT. The other diseases in the SIDS category (Appendix III) are unlikely to have been mistaken for infection deaths.

The risk of SIDS increases with decreasing parental social class (Peterson, 1980). Fertility rates also increase with decreasing social class. If the predicted increase in birth rate occurs in the 1980s (section 4.1.5), there will be a disproportionate increase of births to parents of low social class. If this occurred, the SIDS rate would be expected to increase. This class effect could partially explain the observed variations in the SIDS rate (Figs. 21 and 36). It has not been possible to examine this further in the present study because the class distribution of Sheffield live births is not available.

Almost all of these Sheffield SIDS deaths occurred at home and it is not surprising that hospital-based clinicians were unaware of the scale of the problem for some time as these bodies were usually examined by forensic departments. It has been calculated that SIDS accounts for a similar number of child deaths as the number of adult deaths from bronchogenic carcinoma (see Peterson, 1980). SIDS accounted for as many Sheffield child deaths as did all childhood tumours and violent deaths together (Table I). The condition has received far less public attention or research funding to date.

4.4.5(a) Apparent disappearance of hypernatraemic dehydration from Sheffield infant deaths

There are many possible explanations for the disappearance of hypernatraemia from Sheffield infant deaths (Table XIII) none of which are mutually exclusive. Four possible factors are: the gradual reduction in fatal gastroenteritis (Fig. 34a); the removal of unmodified high sodium milk preparations; an increased incidence of breast feeding; and the local campaign to increase awareness of the dangers of feeding overstrength feeds (section 1.5.4(g)).

The fall in incidence of fatal gastroenteritis cannot wholly explain the fall in the number of hypernatraemic deaths because gastroenteritis was not present in 15 of the 23 infants who died with hypernatraemia (Table XII). The fall in hypernatraemic deaths in Sheffield began before the DHSS working party had published its findings in 1974 (DHSS, 1974); before unmodified milks, especially National Dried Milk, were removed in 1975; and before the Sheffield working party had brought to public awareness the dangers of overstrength bottle feeds. Breast feeding in Sheffield is increasing gradually (Pursall et al., 1978) and, by 1977, 44% of Sheffield's babies were receiving breast milk at one month of age - 32% wholly breast fed and 11.5% being part breast, part bottle fed (Sheffield Child Development Study, unpublished data). However, the majority of Sheffield babies are still wholly

bottle fed. Biering-Sorensen et al. (1978) in a detailed study of infant feeding and SIDS in Denmark found no evidence that infants who suffered unexpected death got more concentrated feeds than paired living control infants. None of the four factors identified can therefore have had much effect in initiating the reduction in hypernatraemic deaths and although changes in feeding practices may have helped to maintain the decline the identified changes were introduced too late to have begun it.

The decline in hypernatraemia deaths in 1972-1979 occurred at the same rate and is of the same magnitude as the decline in SIDS deaths (Table XIII). But hypernatraemia was not found in many SIDS deaths and was found in many explicable deaths (Table XII). Thus the declines cannot be directly related. There may be some common factor linking the declines in these deaths but it is probably not related to changes in feeding practice which were not advocated until 1974 in Sheffield or 1975 nationally. If there is a common factor linking these declines then it is at present unidentified.

If SIDS were due to premeditated homicide then the widespread local publicity that feeding overstrength feeds could lead to death (which occurred in Sheffield in 1974), could have led to an increase in SIDS and an increase in hypernatraemia found at necropsy. The reverse occurred (Table XIII). This supports the conclusion of Kukull and Peterson (1977) that SIDS is not an euphemism for infant murder. This study has been unable to exclude the

possibility that SIDS is due to a sudden impulsive suffocation. The reasons for discarding that hypothesis have already been discussed (section 1.5.4(e)).

4.4.6 Death from malignant disease

Malignant diseases are the least common of the six groups of fatal diseases in this study (Table I). Their relative importance compared with other diseases can be seen in Fig. 29. Fatal malignant diseases among Sheffield children showed little change over the 33 years studied and are not a major cause of child death (Fig. 30).

Birch et al. (1980) found that the relative incidence of malignant disease in childhood in the Manchester region in the period 1954-1977 was: leukaemia one-third, CNS tumours one-quarter, neuroblastoma 6.5%, Wilms' tumour 5% and soft tissue sarcomas 6%. The fatal incidence of malignancies in Sheffield between 1947 and 1979 was very similar (Fig. 29). A study of incidence of malignancies is preferable to a study of mortality because it is unlikely that such serious diseases will not present to medical care and thus the ascertainment of a cancer register should be high. However, accurate cancer registers have not been established long enough to provide time trends comparable to the present mortality study and variations in diagnosis have led to suspicions of the accuracy of historical records (Doll, 1972). Also, these registers are dependent on the voluntary and consistent reporting of all children with tumours. This problem does not occur with a necropsy-verified mortality study.

New anti-cancer treatments have been reported to lead to an improvement in survival and possible cure for some children with

tumours (OHE, 1980). Thus a mortality study may not reflect accurately the disease incidence. However, reports of improved survival could in part be due to artefact. For example, if a new test could diagnose bronchogenic carcinoma three years earlier than any other test, then patients diagnosed by the new test would be expected to have an 8-year survival comparable to the previous 5-year survival without any improvement in treatment and, as mortality increases with time, use of this new test would result in an apparent increase in 5-year survival. The present study avoided such artefactual problems by examining mortality under 15 completed years. Only if children survived beyond their fifteenth birthday would they not be included.

The cost of anti-cancer treatment appears initially to be prohibitive. However, the treatment of malignant disease in children may be highly cost effective. A course of anti-cancer drugs in 1981 cost about £200 although antibiotics, medical salaries and other requirements would inflate these costs per child. Courses of treatment rarely last for more than 18 months and few survivors have any mental or physical handicap as a consequence of treatment. There are failures but as they all die they do not drain resources. This outcome contrasts sharply with, say, successes in treatment of spina bifida or cystic fibrosis.

There were very few deaths from malignancy under two years of age (Table I). Therefore the risk group for these diseases has been taken as children aged over two. The numbers of children in this risk group have varied considerably during the

period studied (Fig. 4). Thus variations in the mortality rates described could be due to either variations in the numbers of deaths or the numbers at risk.

The incidence of leukaemia in childhood has been reported to have risen in Britain since about 1970 (Birch et al., 1979; Geary et al., 1979). Such a change could balance out the reported improvement in survival (see OHE, 1980) and might result in little change in the mortality rate. The Sheffield leukaemia mortality rate rose in 1970-1972 but has subsequently settled. It is not possible in the present study to determine whether this is due to changes in incidence, survival or death. There has been a slight downward trend in the incidence of fatal childhood leukaemias in Sheffield during the period studied (Fig. 35) but no sudden change which might be attributed to a successful new treatment. This finding is surprising in view of reports of cures with new anti-cancer therapies (OHE, 1980). This discrepancy could be because a decline in mortality from these new treatments has not yet become appreciable or because of an increasing incidence of leukaemia. It could also be due to the different methodology of the present study discussed above. This methodology would not be affected by the artefact of earlier diagnosis leading to an apparent increase in 5-year survival. There are at least two disturbing reports of relapse in children with acute lymphoblastic leukaemia who had been presumed cures where the new leukaemia appears to be identical to the previous one (Madden, 1981; Wyld and Lilleyman, 1981). The depressing finding of the present study together with these reports suggests that the future may hold some unpleasant surprises.

The fatal CNS tumour rate showed a similar decline to leukaemias between 1951 and 1967 but the subsequent increase has produced a relatively stable trend overall (Fig. 35). The mortality rates from neuroblastoma and Wilms' tumour in this age group show little trend with time but the numbers are small. The curve for 'all other malignancies' is comprised of a heterogeneous group of conditions (Appendix III) which were too few for individual time trend analyses. The increase in this group in 1971-1979 (Fig. 35) was due to three deaths with Letterer-Siwe disease and two with retinoblastomas superimposed on a steady incidence of fatal sarcomas. The present study found little evidence of increases in the fatal incidence of solid tumours to support Scandinavian reports of a recent increase in incidence of neuroblastoma, Wilms' tumour and CNS tumours (see Birch et al., 1980). The Scandinavian reports may be reflecting changes in ascertainment and diagnosis. Birch et al. (1980) found no comparable increase in solid tumours in Manchester. There were a few deaths which, in the pathologist's opinion, were due to an overdosage of cytotoxic drugs. These deaths occurred when the drugs were relatively new and, from available notes, the large doses were used in a desperate attempt to save the child's life. However, some of these deaths could have been due to previously unappreciated toxic side effects of these drugs. A high necropsy rate will have to be maintained if such effects are to be recognised promptly in the future.

4.4.7 Deaths from unnatural causes

Sheffield child deaths from unnatural causes occurred at a steady rate throughout the period studied (Fig. 30). This group of 471 deaths was the second least common cause of death overall

(Fig. 29) but, following the decline in fatal infectious diseases, unnatural deaths became the most important cause of death in children aged 1-15 years (Figs. 23b and 25). This is in keeping with the finding of Jackson and Wilkinson (1976) based on the numbers of child deaths in England and Wales in 1950-1972. The proportionate distribution of the causes of all unnatural deaths is shown in Fig. 29 (the constituents of the groups of causes shown are given in Appendix III).

A morbidity study would be preferable to a mortality study of accidents because, in contrast to natural causes of death, there is little disagreement on the diagnosis of accidents. However, any child can have any number of non-fatal accidents any number of times. These accidents can vary in severity from a scratched finger to a fractured skull. Therefore, without some standardisation or index of severity, morbidity studies of accidents could vary from area to area according to availability of services. Mortality studies are not ideal for this problem because, for example, a car could hit a child with equal force on the leg or the skull. Irreparable damage to the leg is of different significance than the same damage to the brain. Thus accidents with apparently identical factors could have very different outcome. However, a mortality study does have the ultimate standardisation of severity and records only one such accident per child.

The distribution by cause and age of the numbers of unnatural deaths is shown in Table XIV. It is evident that infants and toddlers will have the majority of their accidents where they spend most of their time - in the home - whereas older children,

having learned to survive the home environment, meet their accidents outside. Thus the causal pattern of fatal accidents varies with age (Figs. 37a, 37b and Table XIV).

4.4.7(a) Fatal traffic accidents

The trend in fatal traffic accident rates among Sheffield's children aged 2-15 years is the most worrying of all the trends discovered in the present study because it has not only increased since the mid-1960s but the rate of increase appears to be accelerating (Fig. 37b).

On Christmas Day 1977 the author was in a large casualty department in the Midlands. There was a new notice 'Skateboard injuries queue here' with a special register for these injuries. By midnight the register was empty. On the same day, 25 children were injured by falling off their new bikes. The medical and lay press gave massive coverage to the potential hazards of skateboarding and many parents provided their children with protective clothing. There is no injury to a child using a skateboard that may not be more seriously acquired on a cycle yet no such protective clothing, advice or warnings are given to new cyclists. Skateboards are banned from public highways yet cyclists are allowed to wobble along any road except a motorway. The present study found no death where a skateboard was involved; in contrast there were 35 where a bicycle was mentioned on the death certificate and there may have been more which were simply registered as traffic accidents. There are many more non-fatal child accidents caused by, or involving, bicycles.

Official statistics of such accidents are compiled from police records. These records only report accidents brought to police notice and the estimate of severity may be inaccurate. A comparison between hospital and police records of 400 children's cycle accidents in Newcastle showed that only 11% of those known to the hospital were known to the police (see Jackson, 1978).

The majority of fatal traffic accidents involved pedestrians of whom the majority were previously healthy children. The vast majority of these deaths are thus in children unknown to the medical profession. The insidious eruption of this 'epidemic' of deaths has therefore been primarily observed by the police, coroners and pathologists with casualty staff and surgeons looking on. These children do not get referred to large teaching hospitals. There is as yet no national pressure group of parents who have lost children in this way. Public and professional ignorance needs to be urgently rectified if this carnage is to be controlled.

This 'epidemic' of accidents is not limited to Sheffield. Jackson and Wilkinson (1976) showed that accidents are the major cause of death throughout Britain in children over one year and Marcusson and Oehmisch (1977) reported that accidents are rapidly becoming the major world cause of post-infant death. The increase in traffic accidents between 1951 and 1971 worldwide was so great that it masked the declines in all other unnatural causes of death. The present study confirms this among Sheffield children (Fig. 25).

In part, the problem is due to an increased number of vehicles travelling at higher speeds on roads not designed for children. Yet the majority of fatal child accidents occur in suburban areas. There is a limited but valuable literature on factors common to many child traffic fatalities (see Jackson, 1978). There are at least three variables in all such accidents: the child, the driver, the environment. Children are physically disadvantaged for coping with traffic. Their height may make it impossible for them to see or hear oncoming traffic from behind parked cars; they are less able to localise sounds than adults and have difficulty in concentrating, sequencing and co-ordinating all information in traffic; right-left discrimination is limited and understanding of road signs is often poor (Sandels, 1975). Children involved in accidents are more likely to have experienced recent emotional stress such as parental illness or separation and to come from overcrowded housing (see Jackson, 1978).

Adults often have poor understanding of children's capabilities in traffic. Sandels (1977) found that there is an over-representation of adolescent men without a family of their own among car drivers who have knocked children down and an enquiry of mother's expectations of their children's behaviour in traffic found that over half would allow their 5-year old to cross the main road alone and 13% of mothers of children aged 2 would also do so (Sadler, 1972).

A macabre but realistic approach has been taken by many vehicle designers who, assuming that accidents will continue,

have designed the front of cars to scoop pedestrians onto the bonnet and then retain them there thus limiting the severity of their injuries. Mascots have been removed from bonnets, bumpers lowered and smoother surfaces incorporated. Less changes have occurred with vans and lorries although there are now designs to close in the gap between the trailer and road and to enclose the rear wheels. The interior of cars have also been designed to minimise serious injuries to children and compulsory seat belt wearing for children may be introduced. Traffic engineering is increasingly (and often inexpensively) reducing the exposure of children to accidents by siting pedestrian crossings at points of maximum safety and redesigning road alignments to improve vision. Some areas also are able to physically separate pedestrian and vehicular traffic but this is more expensive. There will always be other hazards such as poor lighting and poor weather but drunken driving, poor vehicle maintenance and poor road surfaces can be limited or removed. Sandels wrote in 1975 'Children are unreliable in traffic until childhood has matured out of them. What we can do however is to adapt the traffic environment to the children' and in 1977 'In the end, adults are always to blame for traffic accidents involving children' (Sandels, 1975, 1977). More research is needed but much is already known. It is already too late for the children in this study.

4.4.7(b) Accidental poisoning deaths

Despite much medical and public alarm, accidental drug poisonings were not a major cause of death among Sheffield's

children (Table XIV). Far more common were deaths due to poisoning by gases or liquids and carbon monoxide was the most important single poison. A very large proportion of these deaths was in the infant/toddler age range (Table XIV).

These poisonings are almost all true accidents, a consequence of curiosity; only eight suicides were found in the whole series. The increase in fatal poisonings among children aged 2-15 (Fig. 37b) was not due to the increasing potency of modern drugs or household chemicals, but to an increasing number of carbon monoxide poisonings. There was one death following inhalation of trichloacetylene vapour and one death due to 'Distalgesic' ingestion in the period 1977-1978.

Fraser (1980) examined the numbers of fatal accidental poisonings in Britain in 1958-1977 but excluded poisonings by gases and vapours. He showed that the annual numbers of deaths peaked in 1964 and fell steadily thereafter. It would have been preferable had he used rates rather than numbers (see section 1.5.6(b)). He found that drugs caused 81% of the 598 fatalities, non-medicinal products 18.5% and plants 0.5%. He also found that there was a changing pattern of drugs ingested which might follow prescribing habits e.g. tricyclic antidepressants replaced salicylates as the most commonly fatal poison after 1970. He did not comment on whether the drugs were prescribed for parents or children. Part of the decline documented by Fraser (1980) is probably due to a decline in the numbers of children at risk; part may be due to the prescribing of

drugs with a higher therapeutic ratio e.g. benzodiazopines instead of tricyclic antidepressants; and part may be due to improved medical services. However, Fraser (1980) showed that the child resistant packages were introduced too late to have much effect on fatal poisonings. The present study has found that poisonings by gases far outweighed all other causes which puts Fraser's (1980) findings into perspective.

During the mid and late 1960s there was an increase in the number of deaths from asthma. It has been shown that this increase correlated with the introduction of antispasmodic drugs delivered by aerosol and it has been suggested that these deaths could be due to cardiac side-effects of these drugs (Speizer et al., 1968a, 1968b). There was a similar rise in asthma mortality rate among Sheffield children (Fig. 36). Necropsy studies in these children did not identify any other cause for the deaths and available histories record the use of bronchodilator inhalers.

4.4.7(c) Iatrogenic deaths

The 30 deaths indexed as unnatural iatrogenic deaths were all in ill children but where the illness was unlikely to be fatal or where the death was a direct consequence of some medical misadventure. Thus the deaths which followed accidents during exchange transfusion for rhesus disease (Section 4.4.1) have not been included here. All such deaths were indexed under the condition which necessitated the operation or treatment as it was argued that death would

probably have occurred from that condition had the operation not been attempted. Similarly deaths following possible overdoses of cytotoxic drugs in children with terminal malignant disease were indexed under the malignant disease. This reasoning may be questioned with the consequent increase in the iatrogenic deaths rates shown in Fig. 37. No deaths were found which could have been attributed to over-vigorous medical treatment of accidental overdosage, suggested by Fraser (1980).

Deaths following operations were all reported to the coroner. All were found to be due to misadventure and none to malpractice. The decline in this fatality rate is in part due to improved anaesthetic and surgical techniques and in part due to a decline in tonsillectomy. It is not possible to fault the doctors involved because it is not known whether these children might have died without the operation or treatment. The last death in this series (a 13-day old premature child who died of a thrombotic occlusion of the aorta after a hind-quarter amputation for gangrene of one leg) might have died of respiratory distress syndrome although it is debatable whether the intra-aortic probe which was associated with the thromboses was an essential part of his treatment. All operations entail a hazard and the separation of these deaths from the conditions necessitating operation may be artificial. It is salutary that no comparison could be made with the experiences of other professions because data is unavailable.

4.4.7(d) Fatal burns, drownings, falls

These were the second most important group of unnatural deaths (Table XIV and Fig. 37). These diseases show different age distributions with burns and scalds predominant among toddlers and drownings among older children. The second most important cause of accidental death worldwide is drowning (Marcusson and Oehmisch, 1977). It is a little surprising that this is also the second commonest cause of unnatural death in a land-locked city (Table XIV). The majority of these Sheffield drownings occurred in the local canal although there were a few in swimming pools and some of the toddler drownings occurred in garden ponds.

The decline in mortality rate from burns and scalds is due to a number of factors including the introduction of flame-retardant materials for children's clothing; a possible increased use of fire guards; changes in the design of kitchenware; and the availability of antibiotics to control the infections which were often the terminal insult. The continuing incidence of these deaths is probably due to the fact that children are inquisitive and their explorations will occasionally have fatal consequences. The majority of these deaths could have been prevented had simple preventive measures been employed. Many potentially fatal factors are to be found in modern homes and many design features, especially in modern housing, take no account of children (Jackson and Wilkinson, 1976). Successful preventive measures will only follow attempts to view the world through children's eyes.

4.4.7(e) Violent, homicidal deaths

Violent deaths are composed of a series of apparently unrelated sporadic incidents and the numbers of deaths involved are small so that the apparent increase in the late 1970s (Fig. 37b) is an artefact of small numbers. It is not known whether any of these children had been considered previously for abortion.

The introduction of a liberal abortion law in 1968 (Abortion Act 1967) appears to be associated with the decline in numbers of foundlings (Table VII). Weatherall (1976) found a similar trend nationally and suggested that the decline in such deaths may be due to improved health services, increased contraceptive usage, the introduction of the Abortion Act, increased supervision and hospital delivery of pregnancies, or an increasing ease of disposal of a baby's body - particularly in large towns where mechanical handling and compression of rubbish wrapped in opaque plastic bags is commonly practised.

Other homicidal deaths appear to be unrelated incidents. Because accidents are low-frequency events, epidemiological methods are of limited value in the study of aetiological factors. Detailed investigations of each accident are likely to produce more valuable data.

4.5 Historical trends

As the observed declines in the incidence of fatal diseases among Sheffield children may have begun before the period

studied (1947-1979) historical trends in Sheffield childhood mortality rates have been examined using records collected by the Sheffield Medical Officers of Health. The causes of death given in these records may not have been verified by necropsy. For brevity, only deaths in the first two years of life will be examined as this age group accounts for over three-quarters of all childhood deaths. The diseases have been grouped under generic headings using modern diagnostic terms (see sections 2.10 and 3.6).

Doubts may remain about the validity of this historical data. Historical records may be less accurate than modern ones because of improvements in diagnosis. These historical data must be interpreted with caution but medical records are of at least as high quality as the parliamentary and legal records on which most of our history texts are based.

4.5.1 Deaths from prematurity

The trend in fatal incidence of the diseases of prematurity (Fig. 40) shows a gradual increase until the mid-1910s and then a steady decline. Data were not collected or were very limited during and after the first World War. The great majority of these prematurity deaths occurred in the first week and about half on the first day (Fig. 41).

The increase in incidence of prematurity deaths before 1915 could be due to changing ascertainment or to diagnostic fashions or to a genuine trend. The MOH records appear to have used the diagnosis of 'prematurity' for otherwise

inexplicable deaths in newborn infants weighing less than 5 lbs (2270 g) and excluded birth injuries, maternal diseases, infections and other diseases peculiar to infancy (Sheffield 1874-1973). In the present study, deaths due to primary atelectasis, respiratory distress, hyaline membrane disease or other synonym were grouped with those registered as due to prematurity or immaturity.

Kermack et al. (1934) suggested that infant mortality rates might relate to conditions enjoyed by the mother during her own conception and childhood. Baird (1980) has shown that this hypothesis could explain trends in certain malformation mortality rates, certain class effects on perinatal mortality and also certain features of stillbirth and perinatal mortality rates. Baird (1980) also showed that this cohort effect could explain the lack of any social class gradient in maternal height in Sweden and suggested that British infant mortality rates will not approach the Swedish rates until at least one generation after women are reared in an environment comparable to that in Sweden. This hypothesis could explain why the prematurity mortality rate in Sheffield leapt up in 1896-1899 and 1910-1912 (Fig. 40) as these periods are approximately one generation after the economic recessions of the 1870s and 1890s which severely affected the artisan and labouring classes in Sheffield. The trend in prematurity mortality rate (Fig. 40) reversed approximately one generation after the beginning of the fall in infant mortality rate (Fig. 42).

There may have been a gradual improvement in the general health of Sheffield's mothers which probably began in their own childhood. If so, these changes began long before the Welfare State or the National Health Service. The hypothesis of Kermack et al. (1934) has stood the test of time and their predictions have proved surprisingly accurate (see Section 4.6 and Greenwood, 1936). Their comments are therefore relevant: 'care of children during their first 10-15 years of life is of supreme importance' as 'each generation after the age of 5 years seems to carry along with it the same relative mortality throughout adult life, and even into extreme old age'. The benefits to accrue from the expenditure and care of premature infants may not become apparent for some time.

4.5.2 Deaths from malformations

Deaths from malformations among Sheffield children aged less than two years have occurred at a steady rate since 1920 (Fig. 40). The fluctuation in malformation mortality rate before 1920 could be due to changes in classification or registration although Baird (1980) and others (e.g. McKeown and Lowe, 1974; OPCS, 1976b) accept these historical figures as accurate. If the incidence of lethal malformations relates to the health of a mother during her own conception (i.e. when the malformed infant's grandmother was pregnant, see Section 4.6) then the peak in malformation mortality rate between 1902-1911 (Fig. 40) could relate to the poverty in Sheffield during the economic recession of the 1890s. However, no such peak followed the slump of the 1870s or the 1930s. It may be that the Welfare relief was effective in securing the health of children during these years

but it is also possible that Baird's (1980) hypothesis is incorrect.

It appears from Fig. 40 that about five children per thousand live births will die in the first two years from a malformation. Fig. 41 shows that the majority of these deaths occurred in the first month of life; the changes in the 1970s can be explained by changes in the management of neural tube defects (see section 4.4.3(c) and Fig. 33). Fig. 40 shows no apparent effect on this overall malformation mortality rate from medical advances or surgical techniques. Unless there is some breakthrough, this figure of five deaths per thousand live births may be the irreducible minimum below which this community's neonatal and infant mortality rates will not fall.

4.5.3 Deaths from infections

Infections accounted for so many early child deaths that the vertical scale in Figs. 38 and 39 is half that of Fig. 40.

4.5.3(a) Gastroenteritis

The observed trend in gastroenteritis mortality rate (Fig. 38) shows that the trend from 1947-1979 (Fig. 34a) is part of an overall decline which began about 1905. None of the factors discussed in section 4.4.4 can explain this overall decline and, while it is possible that some of these factors may have contributed to this decline, it would be surprising if they were all to coincide to produce the steady decline observed.

No indication is given in the MOH reports as to why this decline occurred. In retrospect there is a temporal association between the fall in fatal gastroenteritis rate

and the replacement of middens with water closets (Fig. 45). This temporal association could be causal but this cannot be confirmed. The only other measure found to have a temporal association with the fall in gastroenteritis deaths in Sheffield was the replacement of housing stock (Fig. 44). These new houses were all built with water closets, adequate ventilation and free running water; they were built away from the polluted river and were used to rehouse the poorest citizens. This may have had a beneficial effect on the raising of the general health of children and thus their resistance to gastroenteritis as the majority of these deaths occurred in the poorer quarters of the city and especially in areas polluted by sewer gas rising from the river (Sheffield 1874-1973). Other public health reforms would be less likely to have had a causal association in Sheffield because they were introduced either before or after this critical period (see Section 1.2.4).

It is possible that no single factor was responsible for the decline in gastroenteritis mortality but that a series of beneficial improvements each in turn reduced mortality. Thus the replacement of middens with water closets in 1900-1930, followed by new housing in 1920-1940 and intravenous fluid treatment in the NHS after 1950 could all have contributed to the decline. Modern experience from underdeveloped countries suggests that nutritional and environmental factors probably play a major role in the aetiology of fatal gastroenteritis (Anonymous, 1978a). There is no identifiable bacterial or viral cause of most

gastroenteritis deaths; diarrhoea epidemics are commonest in the rainy season (which is also the hungry season) and the peak incidence of kwashiorkor follows the peak incidence of diarrhoea. Furthermore, malnutrition was probably the underlying cause of death in at least half of all child deaths in Latin America (Anonymous, 1978a).

Ballard, in a classic epidemiological study (Local Government Board 1889) showed that the peak age incidence of fatal gastroenteritis was 3 months to 2 years. Infants who were wholly breast fed were remarkably exempt from fatal diarrhoea regardless of social class; conversely, infants wholly bottle fed suffered most heavily and bottle feeding was decidedly more dangerous than other artificial feeding. He showed that epidemics of fatal gastroenteritis were likely to occur when the atmospheric temperature was raised but more especially when the soil temperature four feet from the surface was in excess of $56^{o}F$. He also found that calm weather promoted mortality whereas high winds tended to lessen it and that buildings sheltered from prevailing winds or with impediments to the free circulation of air were associated with higher mortality. Rainfall exerted some effect on diarrhoea mortality but this was not consistent and could be explained by an indirect effect such as preventing the rise in soil temperature. Similarly, elevation above sea level had some effect on mortality but not an apparent specific effect on diarrhoea mortality. The structure of the soil had certain associations in that where houses had a foundation on solid rock or clay,

diarrhoea mortality was low. Conversely, permeable or sandy soils especially those containing organic matter were favourable to a high diarrhoea mortality. Excessively wet and completely dry soils were protective but damp soils (especially those habitually soaked by slops or leaking drains and cesspools) were asscciated with a high mortality. Crowded, dark, dirty and poorly ventilated dwellings had a strong association with diarrhcea mortality which might have explained the social class effect he observed.

These findings could be explained by indirect mechanisms - the soil temperature could be associated with the life cycle of flies or merely reflect changes on the moors where the water supply was gathered; again in warm weather children might play on contaminated porous ground and thus become infected. Whatever the reasons, Ballard (Local Government Board 1889) recommended that liquid and solid filth should be removed regularly; that persistently damp ground should be drained and the ground about dwellings should be made impermeable or paved; and that water closets and sewers should replace middens. Introduction of these measures in Sheffield shows a close temporal association with the decline in fatal infant gastroenteritis (Sheffield 1874-1973).

Ballard (Local Government Board 1889) found that polluted drinking water was associated with epidemics of diarrhoea irrespective of season but found no evidence to associate water pollution directly with the annual summer epidemic of fatal gastroenteritis. This is consistent with

Sheffield's experience where annual epidemics occurred although clean running water was available (Holland, 1843). Walker and Walker (1978) found little improvement in gastroenteritis incidence or mortality following a water purification project in Africa and suggested that good general nutrition may be the primary requirement to break out of the malnutrition-gastroenteritis cycle. Tomkins (1981) investigated the influence of pre-existing malnutrition on the severity of diarrhoea among children in Nigeria and found that the attack rate was not increased among underweight or stunted children (defined by centile charts) although children who were wasted experienced more episodes than those who were not wasted and underweight, stunted and wasted children had more prolonged attacks.

In the winter of 1890-1891 there were two serious epidemics of enteric fever in the Tees valley (Local Government Board 1893). The incidence and mortality rate was strikingly in excess among those whose water supply was drawn from the river Tees, but only among those inhabitants who consumed this water (who did not differ from other inhabitants in any other way). The river received 'either directly or indirectly the drainage of some 20 villages and hamlets as well as that of the town of Barnard Castle (population 4341); besides washings of land heavily manured, at times with night soil, together with the drainage of graveyards and farmhouses ... almost everything was so contrived as to ensure, to the fullest, the fouling of the river by every conceivable form of filth'. Despite

evidence of heavy pollution downstream of these townships, there was no evidence of pollution just upstream of the pumping stations which served those affected by enteric fever. Many of the weirs on the river were broken so that much of the excrement accumulated on the river banks and was only washed downstream when the river was in flood. There had been such floods before each of the unseasonal epidemics of enteric fever. Similar incidents might occur with sewage entering drinking water in other parts of the world today whose drinking water is drawn from rivers but this is unlikely to have been a factor in the Sheffield gastroenteritis epidemics because of the abundance of clean moorland water.

The decline in infant mortality rate in Sheffield was primarily due to the decline in gastroenteritis. Similar trends occurred nationally at the beginning of the twentieth century. British records at this time were of very high quality. Gastroenteritis is the major cause of death in childhood in the world today (Marcusson and Oehmisch, 1977; Tomkins, 1981). The likely benefit of measures introduced in Britain could be assessed from these records before being introduced into countries which still have high infant mortality rates.

4.5.3(b) Pneumonias

The incidence of fatal pneumonias among Sheffield children increased steadily until at least 1915 and declined after 1925 (Fig. 38). The incidence appears to have been unaffected by the Public Health reforms at the turn of the

century (Section 1.2.4) but the fall may be associated in time with the rapid housing development in 1920-1940 (Fig. 44). The decline shows no effect of the introduction of antibiotics (not discovered until the 1940s) or clean air (introduced in 1957) or other interventions.

The decline in incidence of fatal pneumonias began approximately one generation after the decline in the infant mortality rate. If the decline in infant mortality rate was also associated with an increase in general health of all children then the reduction in pneumonias may be a consequence of healthier children being born to these healthier survivors who were born at the turn of the century (see Section 4.6).

4.5.3(c) Tuberculosis

The trend in fatal tuberculosis (TB) among Sheffield's infants showed a slight early decline and a steady decline from about 1903 (Fig. 38). There have been no deaths from tuberculosis in Sheffield children under two years after 1959. The decline appears to have been unaffected by antibiotics or immunisation both of which were introduced too late to have much effect on the downward trend. The results of the present study are consistent with the suggestion of McKeown and Lowe (1974) that the decline in fatal tuberculosis may be associated less with medical advances of the twentieth century than with other factors such as changes in nutrition which began in the seventeenth century. This need not mean that medical advances have been ineffective but the non-medical factors have produced such changes that

the full power of medical factors cannot now be appreciated from mortality studies.

4.5.3(d) Pertussis, Diphtheria, Scarlet Fever

These three conditions had a much lower fatal incidence among young Sheffield children than the other conditions examined (Figs. 38 and 39). For unknown reasons scarlet fever had a much lower mortality in Sheffield than the national average (Sheffield 1874-1973). The disappearance of fatal scarlet fever (Fig. 39) could relate to the introduction of antibiotics but could equally be due to other factors such as an increase in host resistance or diminution in virulence of the streptococcus (possibly because of changes in the bacteriophage - see Section 1.5.3). There is no apparent effect of serum treatments or other historically 'successful' therapies on the time trend of fatal scarlet fever incidence.

Fatal diphtheria had a low incidence among Sheffield children throughout (Fig. 39). This trend shows no appreciable effect which could be attributed to immunisation but the final disappearance might relate to the introduction of antibiotics. However, other factors such as phage mutation (see Section 1.5.3) may also have contributed to the decline. This observed pattern of fatal diphtheria is different from that of notified cases of diphtheria nationally (see Galbraith et al., 1980) which show an apparent dramatic change in incidence after the introduction of immunisation. One reason for this may be differences in the age ranges examined; another may be that notifications of cases are far more susceptible to diagnostic

fashions than are registrations of deaths. Alternatively, immunisation may have effected a decline in the number of cases but a proportion of children might still die regardless of the number infected if morbidity and mortality are unrelated. If so, this would suggest that host resistance may be an important factor in determining whether an infected child died or not. Unfortunately, such changes could not be examined in the present study. A number of reports purporting to show the effectiveness of immunisation show the numbers of children notified each year. The fallacy of such an argument when the numbers of children at risk are also changing has already been discussed (see Section 1.1).

The mortality rate from pertussis has declined since at least 1885 (Fig. 38) and had all but disappeared before antibiotics or immunisation could be introduced. This need not mean that either were ineffective, merely that other factors had already been successful and thus the full power of these medical factors cannot be tested. Any of these infectious diseases may have recurred but for the presence of these measures. The recurrence of fatal pertussis in the late 1970s following public abandonment of immunisation suggests that this is so and also that pertussis immunisation may be more beneficial than alternative medical therapies for this condition (see section 4.4.3).

4.5.3(e) Measles

The mortality rate from measles in Sheffield was higher than the national average - in contrast to the incidence of

fatal scarlet fever (Sheffield 1874-1973). No evidence was found to suggest a diagnostic artefact. Measles mortality rate showed a steady trend from 1885 to 1910, a surge between 1911-1918 and a steady decline thereafter (Fig. 39). The reasons for this pattern are unknown and the incidence of measles infection (measured by MOH notifications) remained relatively steady throughout (Sheffield 1874-1973). No medical or environmental change was found to account for these changes in mortality. No reciprocal change was found in conditions which complicate measles and which might have become the registered causes of death. These changes in mortality could reflect changes in the constitution of Sheffield's children.

4.5.3(f) Bronchitis

The steady decline in bronchitis mortality rate between 1885 and 1946 (Fig. 39) occurred principally among children aged 1-11 months. This condition may be the same as that defined as AIRT in the present study (Fig. 34b). Bronchitis mortality declined steadily before the introduction of clean air or antibiotics. It is not clear whether the nineteenth century public health reforms were associated with this decline because it could have begun before 1885 when figures are unavailable.

4.5.3(g) Convulsions

The diagnosis 'infantile convulsions', 'teething convulsions' or 'non-epileptic childhood convulsions' was given as the cause of death for over 200 children per year at the turn of the century. The majority of these deaths

occurred at home and, while some occurred in the first few weeks of life, most were in the postneonatal period. Clues as to an aetiology can be obtained from death certificates issued by the coroner following necropsy e.g. a three-month old boy who died in March 1882 whose death was registered as 'an unknown but natural cause, presumably convulsions'. There were four deaths registered as convulsions in 1947 which were included in the major (1947-1979) study. These children were aged 4 to 24 months, three were boys and two had necropsies - which failed to reveal an adequate cause of death. There was no suggestion of epilepsy or predisposition such as meningitis. They were indexed as SIDS. Templeman (1892) and Werne and Garrow (1953b) reported that some children dying inexplicably convulse terminally. Some SIDS occur in teething children. The condition referred to as convulsions has many features in common with (and may be the same as) SIDS.

If convulsions and bronchitis are equivalent to the conditions grouped under SIDS in the major (1947-1979) study, then backward projection of the incidence of SIDS (Fig. 36) would rise to 70 deaths per thousand at risk at the turn of the century (see Fig. 39). This would be greater than the aggregate total of gastroenteritis and pneumonia mortality rates (see Fig. 38).

4.5.4(h) Violent deaths

Unnatural deaths show a steady trend of 2-3 deaths per thousand at risk (Fig. 40). This group is an aggregate of many different causes including: foundlings, battering,

overlayings and accidents (burns, drownings, falls etc.). Overlayings were removed after 1947 because of necropsy evidence that they should be considered as SIDS. This could account for the observed decline (Fig. 40) and supports the suggestion by Jackson and Wilkinson (1976), Pilling (1976) and Pharoah and Morris (1979) that some earlier SIDS deaths might have been registered as unnatural deaths.

4.6 Towards an understanding of child mortality patterns - The generation-cohort effect

In the preceding sections, explanations based on medical, technological or environmental improvements have been examined and found unable to account fully for the observed declines in the incidence of fatal diseases among Sheffield's children. This does not mean that these advances have been ineffective or irrelevant but they may have been introduced too late to demonstrate their potency if other non-medical factors had already effected the decline or disappearance of deaths from the diseases studied. Throughout the discussion, reference has been made to 'other non-medical factors' which might have contributed to the observed declines in mortality. Such factors would include birth rate, maternal age and health, parental social class and legitimacy (Knox and Mackintosh, 1958; Elwood et al., 1974) which have not been analysed in the present study but there must be other factors as well.

There are many areas of the world where child mortality is as high today as it was in Sheffield at the end of the nineteenth century and the historical part of the present study (Section 4.5)

showed that highly significant declines in child mortality occurred without modern technology. If any causal factors can be identified then it may be possible to achieve similar reductions in child mortality elsewhere in the world, possibly at considerably less expense than is presently envisaged. The possible effect of such success on population pressures and food shortages is beyond the scope of the present study.

Examination of the mortality trends in the present study for evidence of successful medical or non-medical intervention proved disappointing. The decline in child mortality rates began at about the time of the sanitary reforms at the end of the last century which produced enormous social changes. However, it is unclear how such social changes could be solely responsible for the continuing declines in child mortality. It is also difficult to correlate the many medical advances of the present century to the steady decline in child mortality, even allowing for some staggering of the introduction of these measures.

A plausible explanation would be that there is a previously unappreciated effect, termed here the generation cohort effect (GCE), which postulates that there may be a full generation delay between an improvement in health and the decline in a fatal disease rate. Such a mechanism would become self-perpetuating in a stable community and could explain the observed trends in the present study. The basic mechanism postulated is that an improvement in the general health of a community of children will be sustained at least until they reach childbearing

age and will then result in an improvement in the health of their children. Such beneficial effects could be cumulative, resulting in increasingly healthier succeeding generations and would be manifest by biological changes in these new generations such as those observed in the birth weight distribution of Sheffield's children (Gordon, 1977a).

Kermack et al. (1934) showed that adult health was principally determined by the physical condition which had been achieved by the age of 15 (possibly by the age of 5) which supports the first supposition of the GCE hypothesis. Kermack et al. (1934) were hampered by the lack of accurate disease-specific mortality rates but showed that other workers, approaching the problem from different directions, had all been forced to the same conclusion. Writing in 1934 with no awareness of the future 'epidemics' of coronary artery disease, bronchogenic carcinoma or road traffic accidents, Kermack et al. (1934) calculated expected mortality rates for 1941 and 1951. Their predictions subsequently proved surprisingly close although they were a little pessimistic about the 1951 rates and incorrect in their supposition that people would live longer in each generation. They observed a striking delay of approximately one generation between the decline in relative mortalities by years of birth and the decline in infant mortality rate. However, they discounted the explanation that this could be due to the birth of a more healthy race of children in each successive decade because of 'the inherent improbability of any substantial improvement in this respect taking place' in such a short space of time.

The calculations of Kermack et al. (1934) were based on the assumption that the inherited vitality or constitution remained statistically constant. The GCE hypothesis suggests that this improves if the health of the parent generation had improved during childhood. Kermack et al. (1934) observed that before birth and for the first year of life the welfare of a child depends in large measure on the general health and vitality of the mother. Baird (1980) showed that death rates from CNS deformities and certain other conditions can be related to the period in which the mother herself was born and reared. Thus the increased death rate from anencephalus in the 1940s and 1950s could be attributed to women who were born during the depression of 1926-1937. He suggested that, while advances in obstetric care will probably continue to reduce the perinatal mortality rate, it is unlikely that rates similar to those in Sweden can be achieved until at least one generation after women have been reared in an environment comparable to that in Sweden where, for example, social class differences in stature have disappeared.

The GCE hypothesis was derived independently and was based on observations of the disease-specific trends between 1947-1979, in contrast to Kermack et al. (1934) who examined age-specific rates. It was formulated before the analyses of the historical data were done (in part they were done to test it). The hypothesis is able to explain satisfactorily many of the observed trends and it is difficult to think of any equally satisfactory substitute.

It is unlikely that the mothers of 1947 were still bearing children in 1979. It is also unlikely that each new generation began simultaneously. Because of the freedom of choice of spouse and because all couples do not have all their children at a certain age, it is not possible to determine exactly the generation interval. However, most Sheffield residents bear their children between 19 and 29 years of age (OPCS, 1979b). This is in keeping with the historical cyclical pattern observed in Sheffield's birth rate (Fig. 5). If this generation interval was the same throughout this century then there may be significance in the time interval between the fall in infant mortality rate (Fig. 42) or gastroenteritis rate (Fig. 38) compared with the falls in pneumonia mortality rate (Fig. 38) or prematurity mortality rate (Fig. 40). If an improvement in general health among survivors occurred as the infant mortality rate fell in 1900-1910 then the GCE would predict healthier parents giving birth to healthier children in 1920-1930. These healthier children would weigh more and hence be less likely to die of prematurity. Their mothers would also be more likely to carry more of them to term. These children would have increased resistance to infections and hence the pneumonia, pertussis and diphtheria mortality rates might be expected to fall as has been observed (Figs. 38-40). Once initiated this would become self-perpetuating without other intervention. The factors associated with the beginnings of this decline could have had far greater effects than originally envisaged.

There is considerable interest in the uneven distribution of disease and mortality in Britain (DHSS, 1976a; Barker, 1981).

Census data suggest that migration falls with social class. These lower social classes also have higher fertility, morbidity and mortality rates. If the migration patterns span generations then the GCE would suggest that the distribution of disease across the country could be explained by each locality's heritage of disease pattern from the last century and will not alter until at least one generation after social inequalities between regions have disappeared. This explanation seems more plausible than the suggestion that these regional differences may not be just a legacy of past social inequalities but due to differences still present in the environment (Barker, 1981).

If correct, this generation-cohort effect could have far reaching implications for the health of the world's children and especially for those communities which treat female children as second-class citizens. If correct, then the GCE would predict continuing high childhood mortality rates among such communities until at least one generation after such practices cease because all of their children are being handicapped in utero. If correct, the GCE would suggest that (far from reducing paediatric care because mortality can be reduced cheaply in other ways) paediatric care should become the major recipient of any community's health resources and that if any spending cuts are to be made they should be made in that section of the community after childbearing has ceased. Thus, if resources are limited, it may be advisable to transform coronary care units into special care neonatal units. As Kermack et al. (1934) showed 'death rates of the adolescent and adult depend on the constitution acquired during the first 15 years or so of life' and 'care of

children during their first 10-15 years of life is of supreme importance ... the improved physique built up during this period would seem to be of decisive effect at all later ages.'

4.7 General discussion and conclusions

This would appear to be the largest published study where child death in a stable community has been studied over a length of time; where all available information on each death has been collected and assimilated by a single observer; where all possible steps have been taken to control variation and where all information on each death has been standardised to the most recent international recommendations. These measures allow comparisons to be made across time within this community and control most of the problems encountered in time-trend studies although the problem of differing diagnoses with time could not be completely controlled by the use of necropsy reports. Because of these measures it will not be possible to make direct comparisons with historical mortality rates in other communities which used other disease classifications but the results of the present study can be compared with others based on ICD9. The standardisation by age allows comparisons with any other study which follows the recommendations of the World Health Organisation (WHO, 1977).

Comparisons between the present study and national figures of age-standardised mortality rates together with available disease-specific rates suggest that Sheffield's children may be a representative sample of the average national trend. However, unsubstantiated extrapolations of associations found in the Sheffield figures should be treated with caution.

Mortality rates are composed of two elements: the numerator and the denominator. The present study has shown that variations in a mortality rate may be due to changes in either and could depend upon the choice of denominator. Thus apparently significant differences in mortality rates between two communities may be as much due to artefactual differences in the populations chosen as to real differences between these populations (see also section 4.8.2).

The studies on validity of death certification as a measure of child mortality appear to be the largest such series where a single observer has compared the registered cause of death against the necropsy reports of such a small group of pathologists. The study method was different from other published series in that the present study did not set out to discover the extent of disagreement between clinician and pathologist, nor did it assume that the pathologist was always right. Rather, the present study set out to discover the validity of certified causes of death when there was open access to a necropsy if so desired. It is appreciated that many of the registered causes of death could have been different from what the clinicians originally intended before the necropsy. As the present study could only compare diagnoses where a necropsy was done, it probably under-represents inaccuracy. It is, however, surprising to find that even where a necropsy was done, the registered causes of death should contain so many invalid diagnoses. The present study has found that one reason why some other studies found high levels of inaccurate recording of causes of death may be because they examined only the last-entered cause of death. The listing of

the order of causes of death appears to have been haphazard in some of the death certificates of the children studied. The present study also found that the death certificate record may not be a complete record of the numbers of child deaths but that there is no better alternative.

Temporal associations are not necessarily causal. The findings of the present study do not necessarily mean that another community might obtain similar declines in their child mortality rates if they introduced the changes which occurred in Sheffield prior to the observed declines. Also, some of the factors introduced in Sheffield after fatal diseases had disappeared could have been beneficial but a mortality study would be unable to show this.

A decline in mortality rate does not necessarily imply a fall in the prevalence of a disease, merely that deaths from the disease have decreased. The fall could be due to a diminution in the number of affected children, a decline in the severity of the disease, or an increased survival among affected children due to improved resistance or improved treatment. In general terms, a decline in mortality rate may be related to morbidity in a manner analagous to the way that melting of an iceberg observed above the surface relates to the decline in the mass of ice below the surface. But with small numbers of deaths this analogy may be inappropriate. The declines in mortality rates observed in the present study could be due to many factors. This study cannot determine with confidence the relevant priorities among these factors. However, it would appear that non-medical factors have been the more influential.

This study cannot comment on the effectiveness of medical factors in shortening or preventing diseases among children who did not die.

Many of the findings of the present study were unexpected and may be partly due to the methodology employed. This study has examined the experience of a community of children which is contrary to the method of most doctors who deal with numbers (not rates) of patients and treat them regardless of their place of residence. This numerical approach is relevant to most doctors who have to think in terms of numbers - numbers of beds, of operations, of treatments. The conclusions of the present study are not relevant to planning care for individual patients. However, a numerical approach may be seriously misleading if attempting to understand a community's needs and a clinic- or hospital-orientated approach may direct resources to inappropriate priorities (e.g. many would be surprised to find that SIDS accounted for as many deaths as bronchogenic carcinoma - see Peterson, 1980). A hospital-based approach is only relevant for comparing outcome between hospitals and may be of limited relevance if parents or patients choose where to be treated and themselves pay for that treatment. But patient experience is strongly influenced by social factors and such economic considerations are likely to produce highly selected hospital populations which cannot be compared with hospitals serving different populations. In a society where the community pays for treatment it is far more relevant to examine outcome on a community basis. It is also more scientific and objective to examine a stable population at risk rather than a continuously

varying hospital population. There is a dearth of literature based on communities so the conclusions of the present study cannot be compared with other communities. There is an urgent need for further studies in other communities.

The trends in fatal childhood disease in this community cannot be wholly explained by medical actions. Other non-medical factors appear to have been operating with profoundly beneficial effects. Further improvements might be achieved if such factors could be identified. Such future improvements may be far greater and cost far less than interventions based on some current medical thinking. Improvement in the health of children may produce a generation-cohort effect which could extend the benefit into subsequent generations. Such an effect will have far greater impact than would developments of treatments for patients who are past childbearing. The role of much modern medicine has increasingly moved from 'care' into 'cure'. This study would suggest that the move has been inappropriate. There is ample scope for the development of 'caring' medicine.

4.8 Recommendations and suggestions for further work

4.8.1 Potential improvements in the collection of mortality data

During the major (1947-1979) part of this study, some quarter of a million death certificates were examined. The information given on these certificates could have been more fully and more readily obtained if there were minor changes in the certificate layout. The present British death certificate conforms to the WHO model certificate (WHO 1977). Before

introducing such changes, any potential benefits should be balanced against the possible loss of consistent international reporting. Information on the cause of death as presently collected is too frequently presented inappropriately and incorrectly, resulting in errors by lay coders. It is suggested that:

1. The section of the death certificate concerned with the cause of death be redesigned with three columns to collect data on (a) the underlying condition(s) which led to death; (b) precipitating condition(s) which immediately preceded death but which of themselves may not have caused death or which were secondary to the lethal process; and (c) predisposing conditions which could have caused death and may have predisposed to it but which were not relevant to the actual lethal process. These lists of conditions should be in descending order with the most serious condition in each column at the top.

2. A separate space should be made for the certifying doctor to enter the disease which he believes was <u>the</u> cause of death. This will allow continuity of current demographic studies. There is often no single cause of death and with increasingly long courses of disease and the multiplicity of diseases in the elderly this section may be difficult to complete. However, this method is to be preferred to the use of lay coders and a multiplicity of coding rules to determine the underlying cause of death (see WHO 1977). The doctor attending the deceased remains the best-informed person to decide on the cause of death. That doctor is not necessarily the most junior member of the firm.

3. Both maternal and paternal dates of birth; and parental and grandparental occupations would be useful to studying generation effects and movements between generations across social class boundaries. The occupations of single, divorced or widowed women should be recorded if there is no consort or if the female partner is the principal breadwinner.

4. Whenever possible the birthweight of the child and the mother should be obtained. Maternal height has also been found useful (Baird, 1980).

The redesigned section on cause of death should encourage fuller recording of all diseases present and may thus allow more consistent multiple coding analyses. It should also provide fuller documentation of the prevalence of diseases in a community.

4.8.2 Considerations of the definitions of early childhood mortality rates

4.8.2(a) Abortion and perinatal mortality rates

The perinatal mortality rate (PMR) is defined as the number of stillbirths plus first week deaths per 1000 total births (OPCS 1980a). Thus, a decrease in the numbers of babies at risk of dying in the perinatal period without any alteration in the number of pregnant women would be expected to produce a fall in PMR.

In 1978 there were 11,851 legal abortions, 596,418 live births, and 5108 stillbirths to residents of England and Wales - i.e. one out of every six known pregnancies among residents of England and Wales ended as a legal abortion (OPCS 1980a, 1980b). Less than 3% of these

abortions were done for foetal or maternal diseases (OPCS 1980b). All babies are at risk of dying in the perinatal period but the risk is increased among babies born to unmarried and young women (OPCS 1979b). Sixty-three per cent of the abortions were in unmarried women and 27% were in women aged less than nineteen years (OPCS 1980b), so a liberal abortion law might reduce the PMR by the early removal of high-risk pregnancies without there being any change in the standard of perinatal care. The distortion produced by a liberal abortion policy cannot readily be assessed because we do not know how many of the aborted foetuses would have survived beyond the first week of life. However, the introduction of a liberal abortion law in Oregon was associated with significant declines both in foetal deaths over 20 weeks' gestation and in premature births to women over the age of 20 years (Quick, 1978).

In perinatal statistics the denominator '1000 total births' is taken to be the sum of live births plus still-births (OPCS 1980a), disregarding all early foetal deaths and legal abortions. Both numerator and denominator in a proportion ought to relate to the same population; the numerator should be derived solely from the denominator and all individuals represented by the denominator must be at risk of entering the numerator. However, the numerator and denominator in British perinatal statistics are based on subtly different populations because the numerator excludes all babies born before 28 weeks while the denominator will include those live born before this date. If live

birth before 28 weeks' gestation was unusual this may not have mattered but with the legalisation of late termination of pregnancy and the increasing skill of neonatal intensive care units the discrepancy becomes important. The difficulty could be avoided by redefining the denominator as 'per 1000 viable pregnancies' or as 'per 1000 known pregnancies' (i.e. legal abortions, still-births, and live births). The first option is the better but viability will have to be defined.

In Britain the 1953 Births and Deaths Registration Act defines a stillbirth as 'a child which has issued forth from its mother after the twenty-eighth week of pregnancy and which did not, at any time after being completely expelled from its mother, breathe or show any other sign of life'. Foetuses delivered dead before 28 weeks' gestation are thus not registered (regardless of birthweight) while an intrauterine death before 28 weeks in which the dead foetus is retained and delivered after this date must by law be registered as a stillbirth. There is no gestational age criterion for live births: 'any child which has breathed or shown other sign of life, such as beating of the heart, pulsation of the umbilical cord, or definite movement of voluntary muscles, is considered live-born for registration purposes'. Thus in maternity hospitals tiny macerated foetuses retained to 28 weeks or more are registered as stillborn, large 27 week abortuses are unregistered, and tiny 25 week babies on ventilators are registered as live born. Current British registration practice cannot comply

with the international recommendations that national perinatal statistics should include all foetuses and infants delivered weighing at least 500 g or, when birthweight is unavailable, the corresponding gestational age (twenty-two weeks) or body length (25 cm crown-heel) (WHO 1977). There is room for discussion, but legal, pathological, and obstetric opinion would not appear to disagree with the definition of a viable pregnancy as any pregnancy where a gestation of 22 weeks or the corresponding weight (500 g) or length (25 cm crown-heel) was attained.

Comparison of perinatal mortality statistics between countries with differing availability of abortion should be treated with caution. There would seem to be a need to standardise the recording of British perinatal statistics.

4.8.2(b) Depressing effect of burial charges on vital statistics

Despite clear definitions to distinguish live and stillbirth, "a proportion of the deaths which occur within a few days after birth are incorrectly registered as stillbirths, thereby inflating the stillbirth rate and lowering the neonatal mortality rate" (Barker and Rose, 1976). One effect of this shortfall could be inaccuracies in birth, fertility, and childhood mortality rates that depend on the accurate recording of the number of live births.

In Britain, a doctor who knowingly registers a birth or death incorrectly risks prosecution for perjury, so the

scale of the practice of registering early neonatal deaths as stillbirths would be difficult to measure. If the practice were common, early childhood mortality rates would be depressed but deaths after the first day would be unlikely to be affected. The decline in first day mortality in England and Wales since 1963 was attributed to the introduction of intensive care nurseries (Pharoah and Alberman, 1981) but the rate of decline in deaths in the 1-27 day age group showed no improvement, which might favour some artefactual change in the first day deaths.

It is possible, though unlikely, that doctors might use this device artificially to lower neonatal and infant mortality rates. Alternatively, doctors might believe that distressed parents would find it easier to accept a stillbirth than the death of a liveborn baby. Another possible explanation is an awareness of the great difference in cost between burying a stillborn foetus and an infant. Stillborn babies can be cremated or buried in unmarked graves at the expense of the health authority. Stillbirths or neonates can be buried privately in a marginal grave for about £25, but this means no mourners, no cars, no church service, and a rough wooden coffin. Even a simple burial with two cars, a church service, a marked grave, and a small gravestone will cost £280: the current death grant for a child under 3 years is £9. Neonatal deaths are more common among young parents of low social class (OPCS, 1979a). Such parents often have considerable financial problems even before preparing for the arrival of

a baby, yet they may be unwilling to have a common grave for their child. An awareness of these problems may have encouraged some doctors to register some early neonatal deaths as stillborn, unaware that this is perjurious.

Thus burial charges might have had an indirect depressant effect on many vital statistics, and this potential artefact should be considered when mortality rates are compared. Dividing mortality into perinatal and postperinatal periods (i.e. putting the major division after the seventh day rather than the birth) might produce more consistent statistics.

4.8.2(c) Restructuring the classification of diseases

The Manual of the International Statistical Classification of Diseases, Injuries and Causes of Death (ICD) is based on a classification adopted in 1893 and originally devised by a compromise between classifications suggested by William Farr and Marc d'Espine in 1855 (WHO 1977). The classification is still based on the general philosophy of classifying diseases according to their aetiology rather than a particular manifestation and shows traces of Farr's original arrangement of five groups of diseases: Epidemic diseases, Constitutional (general) diseases, Local diseases arranged according to anatomical site, Developmental diseases, and diseases that are a direct result of violence. Medical knowledge of the aetiology of many diseases has advanced since 1855 and the value of the classification is becoming limited. Infectious diseases and tumours do not

readily fit into a systematic classification and the recording of tumours by site of origin is of very limited value in childhood (section 4.4.6); it may also lead to misinterpretation of many aetiological factors in adult tumours.

The multiplicity of codes allows increasing scope for misclassification. It also introduces the need to group various diseases to obtain sufficient numbers for study or to ensure approximate identification of all patients with a common condition. Other workers may disagree with the grouping chosen and produce results which cannot be compared.

The original use of ICD as a classification of causes of death has become overshadowed by the extension of ICD in 1920 to include statistics of morbidity; the extension in 1977 to allow 'the use of ICD for indexing and retrieval of records and for statistics concerning the planning, monitoring and evaluation of health services' and future plans to 'render it useful for Health Insurance Statistics ... and as a basis for central payment for medical services' (WHO 1977). The majority of changes introduced in ICD9 came about as a result of clinical pressures. However, these revisions have left a classification which gives as much potential space to Tularaemia, or Glanders, or Melioidosis as it does to 'Congenital anomalies of the ear, face, and neck' or to 'Chromosome anomalies'. The former conditions may be important in parts of the world

but there are unlikely to be as many subdivisions of them as there are of the latter. The enormous expense for any country of modifying its record system to a new revision of ICD can only be justified if there is some major advantage. It is suggested that the inclusion in one classification of all known diseases and injuries is wasteful and unnecessary for much biostatistical work. There is a need for a new simplified classification of causes of death which is based on a more relevant format. The classification used in the present study has proved efficient, flexible and practical. It would require considerable expansion, especially of the "acquired" section, if it were to include adults but is suggested as one possible alternative to ICD9.

4.8.3 Suggestions for future work

1. The results presented in the present work have been limited to analyses of the validity of death certification and time trends in the corrected causes of death. The 1947-1979 study of Sheffield childhood mortality has also data on the year or date of birth of these children, all necropsy findings, the area of residence, the sex, age, parental occupation and legitimacy. Data have been collected on the distrubution of Sheffield live births between 1947 and 1979 by residence and sex and data could be available on this distribution by parental occupation. Future studies into social and biological factors in these child deaths are proposed which could unravel some of the powerful non-medical factors which appear to have been working in this community.

2. The studies of Sheffield child mortality trends between 1885 and 1979 found dramatic changes in the fatal incidence of certain conditions which are still common in other parts of the world. Further studies to identify the factors responsible for these declines may identify some factor which can be readily and cheaply introduced into those parts of the world.

3. The historical studies have touched on many unexpected or forgotten observations relating to child mortality. Further historical studies could be of value.

4. Many of the findings from the present study were unexpected. There is a need for similar studies in other communities to verify or challenge the methodology and findings of this study.

5. There is a serious lack of community-based studies of diseases. There is an urgent need for a re-examination of much medical teaching against community-based studies.

6. The generation-cohort effect hypothesis should be examined in another community.

APPENDIXES

APPENDIX I

THE SHEFFIELD PAEDIATRIC NECROPSY

All tissues are examined routinely with the exception of the facial structures and normal limbs (both of which are examined by X-ray). All organs are removed and stored in 10% formalin for at least four weeks and are then dissected in detail. Blocks are taken for histological examination from any abnormal tissue and standard blocks are taken routinely from the following sites:

1. Supero-medial aspect of the cerebral cortex
2. Corpus callosum
3. Ammon's horn
   (Blocks 1-3 are taken from a mid-coronal slice of the right cerebral hemisphere)
4. Cerebellar cortex and dentate nucleus (right)
5. Mid-brain and aqueduct
6. Pons
7. Medulla
8. Mid-cervical spinal cord
9. Mid-thoracic spinal cord
10. Mid-lumbar spinal cord
11. Pituitary
12. Nasal septum
13. Parotid
14. Cervical lymph node
15. Thyroid
16. Carotid artery
17. Palate and lymphoid ring
18. Vocal cord and epiglottis
19. Mid trachea and oesophagus
20. Thymus
21-25. Blocks from each lobe of lung
26. Heart (anterior portion of inter-ventricular septum) and left anterior descending coronary artery

27-28. Both lobes of liver

29. Portal tract

30. Pancreas, ampulla of Vater and duodenum

31. Spleen

32. Mesenteric lymph node

33-38. Proximal and distal jejunum, terminal ileum, appendix, mid-colon and rectum

39-40. Both adrenals

41-42. Both kidneys and pelvis of ureter

43. Urinary bladder

44. Both gonads

45. Sartorius muscle (right)

46. Fifth right rib

Each of these blocks is embedded in paraffin wax and stained with haematoxylin and eosin or Masson's trichrome. Additionally, blocks are taken from the right corpus callosum, the heart and coronary artery, the right adrenal and both lobes of the liver for frozen section and fat staining. All of these histological slides are stored in the tissue bank at the Children's Hospital.

Samples of body fluids are taken by aseptic technique from the vitreous humour, the spinal cisterna and the right ventricle of the heart for biochemical and bacteriological analysis. Bacteriological swabs are taken from all cavities and organs for culture and samples of lung and intestine are examined for viruses.

APPENDIX II

Sheffield Childhood Mortality Study

		Columns
Serial Number		1 – 4
Address		5 – 6
Sex		7
Place of Death		8
Date of Birth		
	Day (1 – 31)	9 – 10
	Month (1 – 12)	11 – 12
	Year (47 – 80)	13 – 14
Date of Death		
	Day (1 – 31)	15 – 16
	Month (1 – 12)	17 – 18
	Year (47 – 80)	19 – 20
Age		
	Completed days (0 – 6)	21
	Completed weeks (1 – 4)	22
	Completed months (1 – 12)	23 – 24
	Completed years (1 – 15)	25 – 26
Social class of father		27 – 29
Index cause of death		30 – 33
		34 – 37
Date registered		
	Day (1 – 31)	38 – 39
	Month (1 – 12)	40 – 41
	Year (47 – 80)	42 – 43
Post mortem	Coroners'/Sudden death	44
	Where performed	45
	Cause of death	46 – 49
	Subsidiary causes	50 – 53
		54 – 57
	Agreement with death certificate	58
	Category (A – D)	59

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APPENDIX III

CLASSIFICATION OF FATAL DISEASES

1. EPINATAL

Complication of pregnancy and delivery

01 Premature rupture of membranes
02 Multiple pregnancy
05 Forceps trauma
07 Precipitate delivery
06 Traumatic cerebral haemorrhage
08 Intracranial haemorrhage n.o.s.
09 Tentorial tear
11 Subdural
10 Prolapsed cord
12 Intrauterine hypoxia, asphyxia
13 Other

Complication of placenta

20 Placenta praevia
21 Other forms of placental haemorrhage
23 Placental dysfunction, insufficiency, infarction
24 Other placental abnormality

Dysmaturity

30 Light for dates
31 Extreme immaturity (implies < 1000 g)
32 Prematurity n.o.s.
39 Hyaline membrane disease
40 Respiratory distress syndrome
44 Primary atelectasis, pulmonary immaturity
41 Intra ventricular haemorrhage
42 Pneumothorax, air embolism
43 Pulmonary haemorrhage
45 Meconium aspiration
46 Congenital pneumonia
47 Post-ventilation syndrome
50 Perinatal infections, bacterial
54 Congenital syphilis
51 Perinatal infections, non-bacterial
52 Sclerema neonatorum
53 Cold injury
60 Haemolytic disease n.o.s.
61 Haemolytic disease Rhesus
62 Haemolytic disease ABO
63 Haemolytic disease other
66 Kernicterus
64 Haemorrhagic disease of newborn
65 Other haematological disorders

Maternal conditions

70 Maternal hypertension, pre-eclamptic toxaemia
71 Maternal infections
72 Other chronic maternal conditions
73 Maternal injury
74 Maternal operation
76 Maternal diabetes
77 Other maternal endocrine disturbance
78 Hyperemesis
80 Other ill-defined perinatal disease

2. CONGENITAL ABNORMALITY

Central nervous system

01 Anencephaly
02 Spina bifida
03 Encephalocoele
12 Microcephaly
10 Muscular dystrophy
11 Other CNS deformity

Cardiovascular system

20 Congenital heart n.o.s.
21 Common truncus
22 Transposition of great vessels
23 Fallot's tetralogy
24 Common ventricle
25 Ventricular septal defect
26 Endocardial cushion defect
27 Atrial septal defect (secundum)
30 Aortic valve stenosis/ atresia
31 Mitral valve stenosis (congenital)
32 Hypoplastic left heart
33 Patent ductus arteriosus
34 Coarctation
35 Pulmonary artery anomalies
36 Tricuspid atresia
38 Total anomalous pulmonary venous drainage

Respiratory system

40 Choanal atresia
41 Anomaly of larynx, trachea or bronchus
42 Trache-oesophageal fistula
43 Cystic lung, honeycomb lung
44 Agenesis, hypoplasia of lung
45 Cleft palate and lip
46 Diaphragm hernia, defect, absence
47 Other

Alimentary system

50 Exomphalos
51 Congenital pyloric stenosis
52 Atresia small intestine
53 Atresia large intestine and anus
54 Hirschsprung's disease
55 Meckel's diverticulum
57 Bile duct atresia
58 Coeliac disease
56 Other

Urogenital system

60 UGS anomalies n.o.s.
61 Renal agenesis, dysgenesis
62 Cystic kidneys
63 Hydronephrosis, hydroureter
64 Bladder extrophy
65 Anomalies of urethra
66 Congenital nephrotic

Locomotor system

70 Anomalies of limbs
71 Achondroplasia
72 Osteodystrophies

Endrocrine system

75 Adrenal hypoplasia
76 Other congenital endocrine

Chromosomal

80 Down's
81 Other

Other

82 Multiple congenital abnormalities
84 Conjoined twins
85 Fibrocystic disease
86 Other metabolic
87 Gargoyle
88 Cystic hygroma

3. INFECTION

Tuberculosis

01 Miliary TB
02 Pulmonary
03 CNS, Meningeal
04 Intestinal
05 Bone and joint
06 Genitourinary
07 Other organs
08 Late effect of TB

Central nervous system

10 Polio
11 Encephalitis
12 Meningitis, bacterial
13 Meningitis, non-bacterial
14 Cerebral spinal abscess
15 Phlebitis, thrombosis, cerebral sinuses
18 Acute infectious poly-neuritis

Cardiovascular system

21 Acute pericarditis
22 Acute endocarditis
23 Acute myocarditis
24 Other

Alimentary system

30 Gastroenteritis n.o.s.
31 Typhoid and paratyphoid fever
34 Acute appendicitis
35 Peritonitis
80 Hepatitis

Respiratory system

40 Suppurative otitis media, mastoiditis
43 Acute pharyngitis, tonsillitis
45 Croup
46 Laryngitis and tracheitis
48 Laryngotracheobronchitis
49 Acute infection of respiratory tract
41 Bronchitis
42 Bronchiolitis
52 Pneumonia due to unspecified organisms
55 Other bacterial pneumonia
56 Bronchiectasis
57 Empyaema, pulmonary abscess
58 Influenza
59 Diphtheria
60 Whooping cough
61 Epiglottitis

Other infections

72 Septicaemia n.o.s.
70 Streptococcal infections
71 Tetanus
73 Meningococcal infection
74 Measles
75 Chickenpox
76 Rubella
77 Toxoplasmosis
78 Candidiasis
79 Syphilis
92 Infections of kidney
95 Cellulitis
96 Osteomyelitis
97 Infected eczema

4. UNNATURAL CAUSES

Traffic accidents

01 Motor vehicle accident
02 Motor vehicle accident with pedestrian
03 Pedal cycle accident
04 Multiple injuries n.o.s.
05 Other road vehicle accident

Poisonings

10 Accidental poisoning by drugs
11 Accidental poisoning by solids, gases, liquids
12 Accidental electrocution
13 Mercury poisoning
14 Lead poisoning

Iatrogenic

20 Misadventures during surgical/medical care

Falls, burns etc.

30 Accidental falls/subdural
31 Fire and flames/burns/ scalds
32 Drowning
34 Inhalation of foreign body

Homicide

42 Strangulation
44 Hanging, suicide
43 Neglect with intent to kill
45 Foundling
46 Battering

5. MALIGNANCY

Leukaemia

01 Lymphoid acute
02 Lymphoid chronic
03 Myeloid acute
04 Myeloid chronic
05 Monocytic acute
06 Monocytic chronic
07 Unspecified acute
08 Unspecified chronic

Kidneys

11 Kidney n.o.s.
10 Wilms' tumour

Central nervous system

20 Cerebrum - malignant
21 Cerebellum
22 Brainstem
23 Other
25 Meninges
26 Benign CNS tumour

Other

30 Neuroblastoma
48 Ganglioneuroblastoma
40 Teratoma
41 Reticulosarcoma
42 Lymphosarcoma
50 Rhabdomyosarcoma
53 Sarcoma n.o.s.
43 Hodgkin's
49 Retinoblastoma
44 Letterer-Siwe
45 Skin n.o.s.
46 Bone and Cartilage
47 Connective tissue
52 Nasopharynx tumour n.o.s.
56 Gonads n.o.s.
54 Benign tumour
55 Unspecified

6. UNEXPLAINED

01 Cot death n.o.s.
12 Obstruction of external orifices
13 Terminal inhalation
14 Overlaying
03 Vocal cord necrosis
07 Hypernatraemia n.o.s.
09 Minimal fibroelastosis
16 Possible neglect/gentle battering
21 Debility, defective vitality
22 "Convulsions"
23 "Teething"

7. MISCELLANEOUS, ACQUIRED, UNCERTAIN AETIOLOGY

Central nervous system

01 Cerebral palsy
02 Epilepsy n.o.s.
03 Cerebral degeneration
04 Cerebral haemorrhage
05 Hydrocephaly

Cardiovascular system

10 Cardiomyopathy
11 Rheumatic heart disease
12 Other
15 Air embolism

Respiratory system

20 Asthma
21 Pneumothorax

Alimentary system

30 Intestinal obstruction, hernia, volvulus
31 Liver disease
32 Malabsorption, marasmus
35 Peptic ulcer
36 Vomiting n.o.s.
34 Hiatus hernia, achalasia
47 Other

Urogenital system

40 Nephritis (chronic)
41 Nephrotic syndrome
42 Renal failure n.o.s.
43 Nephrocalcinosis
44 Nephritis n.o.s.

50-59 Locomotor system and collagen disorders

Endocrine system

60 Diabetes
61 Other

Haemopoietic system

70 Anaemias
71 Coagulation defect
72 Other

Other

80 Eczema

81 Failure to thrive n.o.s.

82 Angioneurotic oedema/ anaphylaxis

83 Psychiatric

APPENDIX IV

RISK POPULATIONS OF SHEFFIELD CHILDREN

	Live Births	1-11m	1-4y	5-9y	10-14y	Total Children
1947	10522	10132	35982	36475	36720	125612
1948	9107	8838	37985	37114	36708	127541
1949	8087	7833	37163	38994	36841	128098
1950	7370	7154	36764	40013	36942	127792
1951	7233	7026	33957	43108	36394	127141
1952	7005	6842	30851	45712	36385	126184
1953	7055	6882	28855	46463	37033	125095
1954	6867	6710	27904	44575	38925	123770
1955	6756	6604	27460	43556	39942	122824
1956	7040	6886	27038	40747	43042	122387
1957	7519	7351	27082	37550	45650	121948
1958	7656	7498	27551	35622	46403	120991
1959	7709	7585	28339	34490	44509	118628
1960	7829	7677	29320	33948	43494	117828
1961	8157	7958	30111	33805	40685	115912
1962	8612	8437	30718	34315	37486	114002
1963	8396	8228	31657	34934	35558	113291
1964	8400	8259	32300	35812	34424	113604
1965	8505	8350	32882	36879	33877	114739
1966	8291	8127	33274	37934	33733	115797
1967	8876	8713	32964	39005	34248	117668
1968	8874	8722	33449	39737	34867	119487
1969	8465	8312	33912	40419	35747	121085
1970	8214	8058	33874	41086	36821	122543
1971	7875	7716	33805	41281	37870	123378
1972	6944	6827	32808	41566	38945	122803
1973	6011	5923	30913	42066	39675	121158
1974	6180	6080	28524	42128	40358	119629
1975	5740	5661	26546	41843	41019	117540
1976	5537	5464	24491	41432	41226	114920
1977	5457	5388	23128	39567	41511	111765
1978	5619	5556	22593	36753	42024	108988
1979	6045	5979	22069	34525	42082	106633

TABLES

TABLE I

Distribution of deaths by age and cause

Age	Epinatal	Congenital Deformity	Infection	Violent Death	Tumours	Unexplained	Acquired	Total
< 1 week	2425	527	18	32	2	12	20	3036
1-3 weeks	126	219	55	1	2	54	14	471
1-11 months	32	429	591	33	20	557	113	1775
1-4 years	3	122	247	158	110	98	107	845
5-9 years	1	55	65	156	77	18	104	476
10-14 years	2	41	71	71	74	10	92	361
15 years	0	9	13	20	8	2	19	71
Total	2589	1402	1060	471	293	751	469	7035

Number of missing observations = 14

TABLE I

TABLE II

Distribution of necropsies by age and cause

Age	Epinatal	Congenital Deformity	Infection	Violent Death	Tumours	Unexplained	Acquired	Total
< 1 week	1284	359	13	32	2	10	9	1709
1-3 weeks	71	161	36	1	2	53	8	332
1-11 months	26	279	240	33	14	530	71	1193
1-4 years	2	80	129	157	50	90	50	558
5-9 years	1	22	26	155	27	10	39	280
10-14 years	0	14	29	71	19	5	32	170
15 years	0	1	7	18	0	1	11	38
Total	1384	916	480	467	114	699	220	4280

TABLE II

TABLE III

Distribution of deaths by age and place of necropsy

Age	SCH	JHW	NGH	Coroner	Other	None	Total
< 1 week	905	409	332	49	14	1331	3040
1-3 weeks	212	35	64	16	5	141	473
1-11 months	924	9	130	98	32	588	1781
1-4 years	334	0	56	142	26	288	846
5-9 years	115	0	22	136	7	197	477
10-14 years	62	0	20	78	10	191	361
15 years	3	0	6	24	5	33	71
Total	2555	453	630	543	99	2769	7049

TABLE III

TABLE IV

Validity of certified cause of death by agreement with death certificate

(Row percentages shown in brackets)

Cause	Agreement	Major Disagreement	Incorrect word order	Total
Epinatal	955 (77.6)	217 (17.6)	58 (4.7)	1230
Congenital Deformity	643 (69.7)	131 (14.2)	149 (16.1)	923
Infection	280 (76.5)	57 (15.5)	29 (7.9)	366
Violent Death	447 (97.2)	12 (2.6)	1 (0.2)	460
Tumours	87 (81.3)	16 (15.0)	4 (3.7)	107
Unexplained	541 (78.7)	134 (19.5)	12 (1.7)	687
Acquired	131 (56.0)	25 (10.7)	78 (33.3)	234
Total	3084	592	331	4007

TABLE V

Validity of certified cause of death by age

(Row percentages shown in brackets)

Age	Agreement	Major Disagreement	Incorrect word order	Total
< 1 week	1191 (77.0)	259 (16.7)	97 (6.3)	1547
1-3 weeks	226 (73.9)	56 (18.3)	24 (7.8)	306
1-11 months	836 (73.4)	202 (17.7)	101 (8.9)	1139
1-4 years	428 (80.3)	58 (10.9)	47 (8.8)	533
5-9 years	239 (83.3)	9 (3.1)	39 (13.6)	287
10-14 years	136 (83.4)	7 (4.3)	20 (12.3)	163
15 years	28 (87.5)	1 (3.1)	3 (9.4)	32
Total	3084 (77.0)	592 (14.8)	331 (8.3)	4007

TABLE VI

Validity of certified cause of death by parental social class

(Row percentages shown in brackets)

Class	Agreement	Major Disagreement	Incorrect word order	Total
I	97 (78.9)	19 (15.4)	7 (5.7)	123
II	236 (75.4)	51 (16.3)	26 (8.3)	313
IIINM	187 (73.3)	45 (17.6)	23 (9.0)	255
IIIM	1456 (76.2)	284 (14.9)	172 (9.0)	1912
IV	418 (77.4)	83 (15.4)	39 (7.2)	540
V	477 (79.4)	77 (12.8)	47 (7.8)	601
Unemployed or Illegitimate	176 (81.1)	31 (14.2)	10 (4.6)	217
Total	3047 (76.9)	590 (14.9)	324 (8.2)	3961

Number of missing observations = 46

TABLE VII

Numbers of foundlings by year of birth and social class

Class	1947-1949	1950-1952	1953-1955	1956-1958	1959-1961	1962-1964	1965-1967	1968-1970	1971-1973	1974-1976	1977-1979	Total
I-II	-	-	-	-	-	-	-	-	-	-	-	-
IIINM	-	-	-	-	-	-	-	-	-	-	-	-
IIIM	-	-	-	2	-	-	1	-	-	-	-	3
IV	-	-	-	-	-	-	-	-	-	-	-	-
V	-	-	1	-	-	-	-	-	-	-	-	1
Illegitimate - employed	-	1	-	1	1	1	-	-	-	1	-	5
Illegitimate - unemployed	-	-	-	1	-	1	-	-	1	1	-	4
Unknown	2	2	-	3	-	1	1	1	-	-	-	10
Total	2	3	1	7	1	3	2	1	1	2	-	23

TABLE VII

TABLE VIII

Neonatal deaths among British and Sheffield babies born in March 1958 with Sheffield January-December 1958

	< 1 week			1-3 weeks		
	Britain (March)	Sheffield (March)	Sheffield (Jan-Dec)	Britain (March)	Sheffield (March)	Sheffield (Jan-Dec)
Malformation	169 (21.6)	1 (9.1)	16 (14.4)	70 (45.3)	-	6 (42.9)
Isoimmunisation	33 (4.2)	-	3 (2.7)	2 (1.3)	-	1 (7.1)
Asphyxia	109 (14.0)	3 (27.3)	29 (26.1)	1 (0.6)	-	-
Cerebral birth trauma	44 (5.6)	4 (36.3)	17 (15.3)	1 (0.6)	-	1 (7.1)
Pulmonary infection	104 (13.3)	-	-	40 (25.8)	-	1 (7.1)
Hyaline membrane disease	117 (15.0)	-	24 (21.6)	1 (0.6)	-	1 (7.1)
Intraventricular haemorrhage	50 (6.4)	-	3 (2.7)	4 (2.6)	-	1 (7.1)
Pulmonary haemorrhage	46 (5.9)	-	4 (3.6)	8 (5.2)	-	-
Other infection	8 (1.0)	1 (9.1)	1 (0.9)	16 (10.3)	-	2 (14.3)
No histological lesion	68 (8.7)	-	10 (9.0)	2 (1.3)	-	-
Miscellaneous	12 (1.6)	2 (18.2)	4 (3.6)	7 (4.5)	-	1 (7.1)
No histology	21 (2.7)	-	-	3 (1.9)	-	-
	781	11	111	155	-	14

TABLE IX

Early neonatal deaths in Britain and Sheffield, 5-11 April 1970 with Sheffield Jan-Dec 1970

	Britain (April 5-11)	Sheffield (April 5-11)	Sheffield (Jan-Dec)
Asphyxia	30 (18.6)	-	12 (11.5)
Malformation	39 (24.2)	-	16 (18.4)
Isoimmunisation	4 (2.5)	-	1 (1.1)
Atelectasis	51 (31.7)	1 (25.0)	32 (36.8)
Pneumonia	2 (1.2)	-	-
Pulmonary haemorrhage	2 (1.2)	-	-
Other infection	2 (1.2)	-	1 (1.1)
Cerebral birth trauma	4 (2.5)	-	10 (11.5)
Intraventricular haemorrhage	6 (3.7)	-	3 (3.4)
Miscellaneous	4 (2.5)	-	1 (1.1)
Immaturity	17 (10.6)	3 (75.0)	11 (12.6)
Total	161	4	87

TABLE X

Early neonatal deaths in Sheffield 1947-1979

	March	Jan-Dec	5-11 April
Malformation	52 (17.6)	540 (17.8)	10 (16.9)
Isoimmunisation	11 (3.7)	125 (4.1)	4 (6.8)
Asphyxia	45 (15.2)	458 (15.1)	9 (15.3)
Cerebral birth trauma	30 (10.1)	283 (9.3)	7 (11.9)
Pulmonary infection	-	13 (0.4)	-
Hyaline membrane disease	105 (35.5)	1108 (36.5)	21 (35.6)
Intraventricular haemorrhage	5 (1.7)	36 (1.2)	-
Pulmonary haemorrhage	1 (0.3)	17 (0.6)	-
Other infection	5 (1.7)	53 (1.7)	-
No histological lesion	34 (11.5)	316 (10.4)	6 (10.2)
Miscellaneous	8 (2.7)	87 (2.9)	2 (3.4)
Total	296	3036	59

TABLE XI

Numbers (and percentage of annual total) of early neonatal deaths by month born - 1958, 1970 and 1947-79

	Jan.	Feb.	Mar.	Apr.	May	Jun.	Jul.	Aug.	Sep.	Oct.	Nov.	Dec.	Total
1958	12 (10.8)	7 (6.3)	11 (9.9)	3 (2.7)	3 (2.7)	9 (8.1)	12 (10.8)	8 (7.2)	12 (10.8)	14 (12.6)	15 (13.5)	5 (4.5)	111
1970	12 (13.8)	4 (4.6)	6 (6.9)	11 (12.6)	2 (2.3)	5 (5.7)	5 (5.7)	9 (10.3)	10 (11.5)	9 (10.3)	5 (5.7)	9 (10.3)	87
1947-1979	316 (10.4)	238 (7.8)	296 (9.7)	234 (7.7)	250 (8.2)	247 (8.1)	245 (8.1)	224 (7.4)	225 (7.4)	261 (8.6)	229 (7.5)	275 (9.0)	3040

TABLE XI

TABLE XII

Indexed cause of death of infants dying with hypernatraemia

(October 1972 - December 1979)

	Year of death						Total
	1972†	1973	1974	1975	1976	1977-79	
Gastroenteritis	1	4	2		1		8
Meningitis		1					1
CNS abnormality		1	1				2
Laryngotracheobronchitis	2	2	1				5*
Malabsorption		1					1*
Endocardial fibroelastosis	1		1				2*
Unexplained sudden death	2	1		1			4*
Total	6	10	5	1	1	0	23

* Presented as unexpected infant deaths.

† October to December only.

TABLE XII

TABLE XIII

Child deaths under 2 years in Sheffield County Borough 1969-74 and Sheffield Metropolitan Borough 1974-79

Year	Total live births	Total deaths (0-2 yr)	Postperinatal deaths (1-103 wk)	% of postperinatal deaths with vitreous examination	No. of deaths with hypernatraemia (per 1000 live births)	No. of deaths with gastroenteritis (per 1000 live births)	No. of cot deaths (per 1000 live births)
1972	6994	127	66			8 (1.1)	30 (4.3)
†	1551†	34†	19†	63†	6† (3.9)	1†	9†
1973	6100	104	46	79	10 (1.6)	7 (1.1)	20 (3.3)
1974	6810	105	39	63	5 (0.7)	6 (0.9)	11 (1.6)
1975	5739	82	30	59	1 (0.2)	2 (0.3)	13 (2.3)
1976	5525	77	31	74	1 (0.2)	1 (0.2)	11 (2.0)
1977	5450	78	37	51	0	1 (0.2)	12 (2.2)
1978	5536	58	25	56	0	2 (0.4)	7 (1.3)
1979	6045	80	41	42	0	0	9 (1.5)

† Oct-Dec 1972 only

TABLE XIII

TABLE XIV

Unnatural deaths by cause and age in completed years

	< 1	1	2-4	5-9	10-14	15	Total
Traffic accidents	10	11	53	103	28	8	213
Traffic with cycles	-	-	1	6	13	5	25
Poisonings (drugs	0	4	2	0	2	1	9
Poisonings (other	1	7	10	4	4	0	26
Burns	5	16	16	3	5	-	45
Drownings	-	2	9	27	6	1	45
Falls	2	4	4	5	4	-	19
Iatrogenic	15	3	4	4	2	2	30
Foundlings	23	-	-	-	-	-	23
Violent deaths	10	6	6	4	7	3	36
	66	53	105	156	71	20	471

TABLE XIV

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REFERENCES

Alderson, M. R. and Meade, T. W. (1967) Accuracy of diagnosis on death certificates compared with that in hospital records. Brit. J. Prev. Soc. Med. 21: 22-29.

Anonymous (1978a) Child mortality - nutrition - gastroenteritis - water contamination. Lancet (ii): 616.

Anonymous (1978b) Seasonality of birth in schizophrenia. Lancet (i): 481-482.

Anonymous (1979) Reducing perinatal mortality (editorial). Brit. Med. J. (1): 971.

Anonymous (1980) To err is ... Lancet (ii): 580.

Armitage, P. (1970) In Data handling in epidemiology. Ed. W. W. Holland. Oxford University Press, London.

Arnold, J. (1894) Myelocyste, transposition von Gewebskeimer und Sympodia. Beitr. Path. Anat. 16: 1-28. Cited by Frank, J. D. and Fixsen, J. A. (1980).

Baird, D. (1980) Environment and reproduction. Brit. J. Obstet. Gynaecol. 87: 1057-1067.

Ballantyne, J. W. (1892) Studies in foetal pathology and teratology. II Clinical notes of four cases and description of two specimens on general dropsy of the foetus. Edin. Med. J. 38: 57-67 and 142-160.

Barker, D. J. P. (1981) Geographical variations in disease in Britain. Brit. Med. J. (ii): 398-400.

Barker, D. J. P. and Rose, G. (1976) In Epidemiology in medical practice. Churchill Livingstone, Edinburgh.

Bevis, D. C. A. (1952) The antenatal prediction of haemolytic disease of the newborn. Lancet (i): 395-398.

Biering-Sorensen, F., Jorgensen, T. and Hilden, J. (1978) Sudden infant death in Copenhagen 1956-1971. I Infant feeding. Acta Paediatr. Scand. 67: 129-137.

Birch, J. M. (1979) The epidemiology of childhood tumours. In Topics in paediatrics. I Haematology and oncology. Ed. P. M. Morris-Jones. Pitman Medical, Tunbridge Wells.

Birch, J. M., Marsden, H. B. and Swindell, R. (1979) Acute lymphoid leukaemia of childhood in North West England. Lancet (ii): 854-855.

Birch, J. M., Marsden, H. B. and Swindell, R. (1980) Incidence of malignant disease in childhood: a 24-year review of the Manchester Children's tumour registry data. Brit. J. Cancer, 42: 215-223.

Bjerre, I. and Östberg, G. (1974) Infant Mortality. Causes of death during the first year of life in a five-year series. Acta Paediatr. Scand. 63: 49-58.

Bone, M. (1978) Family planning services: changes and effects. H.M.S.O., London.

Bowden, K. M. (1952) Overlaying of infants. Med. J. Aust. 2: 609-611.

Brock, D. J. H. and Sutcliffe, R. G. (1972) Alpha-foetoprotein in the antenatal diagnosis of anencephaly and spina bifida. Lancet (ii): 197-199.

Brock, D. J. H., Bolton, A. E. and Monaghan, J. M. (1973) Prenatal diagnosis of anencephaly through maternal serum alpha-foetoprotein measurement. Lancet (ii): 923-924.

Butler, N. R. and Bonham, D. G. (1963) In Perinatal mortality. Churchill Livingstone, Edinburgh.

Butler, N. R. and Alberman, E. D. (1969) In Perinatal problems. Churchill Livingstone, Edinburgh.

Cameron, H. M. and McGoogan, E. (1981) A prospective study of 1152 hospital autopsies: I Inaccuracies in death certification. J. Path. 133: 273-283.
A prospective study of 1152 hospital autopsies: II Analysis of inaccuracies in clinical diagnoses and their significance. J. Path. 133: 285-300.

Campbell, A. C. P., Gaisford, W., Paterson, F. and Steward, J. K. (1981) Tumours in children. A survey carried out in the Manchester region. Br. Med. J. (i): 448-452.

Carpenter, R. G. and Emery, J. L. (1977) Final results of study of infants at risk of sudden death. Nature 268: 724-725.

Carr, D. H. (1967) Chromosome anomalies as a cause of spontaneous abortion. Am. J. Obstet, Gynecol. 97: 283-293.

Central Health Services Council (1961) Report of the sub-committee on the prevention of prematurity and the care of premature infants. H.M.S.O., London.

Chalmers, I., Dauncey, M. E., Verrier-Jones, E. R., Dodge, J. A. and Gray, O. P. (1978) Respiratory distress syndrome in infants of Cardiff residents during 1965-75. Br. Med. J. (2): 1118-1121.

Chiari, H. (1891) Uber veränderungen des kleinhirns, des pons und der medulla oblongata in folge von congenitaler hydrocephalie des grosshirns. Deutsch, Med. Wschr. 27: 1172-1175. Cited by Frank, J. D. and Fixsen, J. A. (1980).

Chiari, H. (1895) Uber veränderungen des kleinhirns, des pons und der medulla oblongata in folge von congenitalen hydrocephalie des grosshirns. Deutschr. Akad, Wiss Wien, 63: 71-116. Cited by Frank, J. D. and Fixsen, J. A. (1980).

Clarke, C. A. (1975) In Rhesus haemolytic disease. Selected papers and extracts. University Park Press, Baltimore.

Clarke, C. A. and Whitfield, A. G. W. (1979) Deaths from rhesus haemolytic disease in England and Wales in 1977: accuracy of records and assessment of anti-D prophylaxis. Br. Med. J. (i): 1665-1669.

Cleland, J. (1883) Contribution to the study of spina bifida, encephalocoele and anencephalus. J. Anat. Physiol. 17: 257-292. Cited by Frank, J. D. and Fixsen, J. A. (1980).

Cochrane, A. L. (1972) In Effectiveness and efficiency. The Nuffield Provincial Hospitals Trust, London.

Combined Study (1966) Prevention of Rh-haemolytic disease: results of the clinical trial. A combined study from centres in England and Baltimore. Br. Med. J. (2): 907-914.

Combined Study (1971) Prevention of Rh-haemolytic disease: final results of the "high-risk" clinical trial. A combined study from centres in England and Baltimore. Br. Med. J. (2): 607-609.

Dale, G., Goldfinch, M. E., Sibert, J. R. and Webb, J. K. G. (1975) Plasma osmolality, sodium and urea in healthy breast-fed and bottle-fed infants in Newcastle upon Tyne. Arch. Dis. Child. 50: 731-734.

Darrow, R. R. (1938) Icterus gravis (erythroblastosis) neonatorum. An examination of etiologic considerations. Arch. Pathol. 25: 378-417.

Davies, D. P. (1973) Plasma osmolality and feeding practices of healthy infants in the first three months of life. Br. Med. J. (2): 340-342.

Davies, P. A. (1980) Perinatal mortality. Arch. Dis. Child. 55: 833-837.

Dean, G. and McLoughlin, H. (1980) The registration and certification of deaths in the west of Ireland. Ir. Med. J. 73: 269-270.

Department of Health and Social Security (1974) Present day practice in infant feeding. H.M.S.O., London.

Department of Health and Social Security, Department of Education and Service and Welsh Office (1976a) Fit for the future: report of the Committee on Child Health Services (Court, S. D. M., Chairman). H.M.S.O., London.

Department of Health and Social Security (1976b) Priorities for health and personal social services in England: a consultative document. H.M.S.O., London.

Department of Health and Social Security (1976c) Prevention and health: everybody's business. H.M.S.O., London.

Diamond, L. K. (1947) Erythroblastosis foetalis or haemolytic disease of the newborn. Proc. roy. Soc. Med. 40: 546-550.

Dick, G. (1974) Reactions to routine immunisations in childhood. Proc. roy. Soc. Med. 67: 371-372.

Doll, R. (1972) In The epidemiology of leukaemia. Leukaemia Research Fund, London.

Donne, J. (1624) Devotions. Cited by Tripp, R. T. (1976) In The international thesaurus of quotations. Penguin, London.

Drummond, W. B. (1901) In The Child. J. M. Dent and Sons, London.

Dunn, P. M. and MacGregor, M. E. (1964) Infant and child mortality in South Warwickshire, 1962-1963. Arch. Dis. Child. 39: 492-495.

Dyson, T. (1977) Levels, trends, differentials and causes of child mortality - a survey. World Health Stat. Rep. 30: 282-311.

Ehrat, R. (1948) In Die missbildungen der neugeborenen an der Universitatsfrauenklinck Zurich 1921-1944. Buchdruckerei Jak. Villiger & Cie - Wädenswil, Zurich.

Elwood, J. H., MacKenzie, G. and Cran, G. W. (1974) Observations on single births to women resident in Belfast, 1962-1966. J. Chronic Dis. 27: 517-562.

Emery, J. L. (1962) Accuracy of death certification. Certification of death by the pathologist. Proc. roy. Soc. Med. 55: 738-740.

Emery, J. L. (1976) Unexpected death in infancy. In Recent advances in paediatrics. No. 5. Ed. D. Hull. Churchill Livingstone, Edinburgh.

Emery, J. L. and Irvine, K. A. (1958) Death certification of children. Br. Med. J. (2): 1510-1512.

Emery, J. L., Swift, P. G. F. and Worthy, E. (1974) Hypernatraemia and uraemia in unexpected death in infancy. Arch. Dis. Child. 49: 686-692.

Fearn, S. W. (1834) Sudden and unexplained death of children. Lancet (i): 246.

Fedrick, J. and Butler, N. R. (1972) Accuracy of registered causes of neonatal deaths in 1958. Br. J. Prev. Soc. Med. 26: 101-105.

Frank, J. D. and Fixsen, J. A. (1980) Spina Bifida. Br. J. Hosp. Med. 24: 422-437.

Fraser, N. C. (1980) Accidental poisoning deaths in Britain 1957-77. Br. Med. J. (1): 1595-1598.

Freeman, V. J. (1951) Studies of the virulence of bacteriophage-infected strains of Corynebacterium diphtheriae. J. Bacteriol. 61: 675-688.

Froggatt, P., Lynas, M. A. and MacKenzie, G. (1971) Epidemiology of sudden unexpected death in infants ('cot death') in Northern Ireland. Br. J. Prev. Soc. Med. 25: 119-134.

Galbraith, N. S., Forbes, P. and Mayon-White, R. T. (1980) Changing patterns of communicable disease in England and Wales: Part I - newly recognised diseases. Br. Med. J. (2): 427-430.
Part II - disappearing and declining diseases. Br. Med. J. (2): 489-492.
Part III - increasing infectious diseases. Br. Med. J. (2): 546-549.

Geary, C. G., Benn, R. T. and Leck, I. (1979) Incidence of myeloid leukaemia in Lancashire. Lancet (ii): 549-551.

General Register Office (1966) Studies on medical and population subjects No. 20. Accuracy of certification of cause of death (Heasman, M. A. and Lipworth, L.) H.M.S.O., London.

Gittelsohn, A. and Senning, J. (1979) Studies on the reliability of vital and health records. I Comparison of cause of death and hospital record diagnoses. Am. J. Public Health, 69:680-689.

Goodfellow, A. W. (1956) The city of Sheffield: the development of communications. In Sheffield and its region. A scientific and historical survey. Ed. D. L. Linton. British Asssociation for the Advancement of Science, Sheffield.

Gordon, R. R. (1977a) Disproportion in the falling birth rate. Br. Med. J. (2): 939-940.

Gordon, R. R. (1977b) Neonatal and "perinatal" mortality rates by birth weight. Br. Med. J. (2): 1202-1204.

Graunt, J. (1676) In Natural and political observations, mentioned in a following index, and made upon the bills of mortality. John Martyn, London.

Greenwood, M. (1936) English death rates, past, present and future. J. Roy. Stat. Soc. 99: 674-713.

Griffith, A. H. (1978) Reactions after pertussis vaccine: a manufacturer's experiences and difficulties since 1964. Br. Med. J. (1): 809-815.

Harvey, P. (1976) In Up the hill to Western Bank. A history of the Children's Hospital, Sheffield, 1876-1976. The Centenary Committee, Children's Hospital, Sheffield.

Havard, J. D. J. (1960) In The detection of secret homicide. Macmillan, London

Havard, J. D. J. (1962) Accuracy of death certification. Medico-legal aspects. Proc. roy. Soc. Med. 55: 736-738.

Hippocrates, Of Airs, Waters, and Places. Translated by Jones, W. (1931). Heinemann, London.

Hodes, H. L. (1979) Diphtheria. Pediatr. Clin. North Am. 26: 445-459.

Holland, G. C. (1843) In The vital statistics of Sheffield. Robert Tyas, London.

Hollinger, P. C. (1979) Violent deaths among the young: recent trends in suicide, homicide, and accidents. Am. J. Psychiatry, 136: 1144-1147.

Hook, E. B., Farina, M. A. and Hoff, M. B. (1977) Death certificate reports of cardiovascular disorders in children: comparison with diagnoses in a pediatric cardiology registry. J. Chronic Dis. 30: 383-391.

House of Commons (1980) Perinatal and neonatal mortality. Second report from the Social Services Committee (Short, R., Chairman). H.M.S.O., London.

Hubble, D. and Osborn, G. R. (1941) Acute bronchiolitis in children. Br. Med. J. (1): 107-110.

Hughes-Davies, T. H. (1979) Conservative care of the newborn baby. Arch. Dis. Child. 54: 59-61.

Jackson, R. H. (1978) Hazards to children in traffic. A paediatrician looks at road accidents. Arch. Dis. Child. 53: 807-813.

Jackson, R. H. and Wilkinson, A. W. (1976) Why don't we prevent childhood accidents? Br. Med. J. (1): 1258-1262.

Janerich, D. T. and Garfinkel, J. (1970) Season of birth and birth order in relation to prenatal pathology. Am. J. Epidemiol. 92: 351-356.

Jewkes, J., Sawers, D. and Stillerman, R. (1969) The prevention of Rhesus haemolytic disease. In The sources of Invention. Macmillan, London.

Johnson, N. L. and Leone, F. C. (1964) In Statistics and experimental design in engineering and the physical sciences. John Wiley and Sons Inc., New York.

Jones, R. A. K., Cummins, M. and Davies, P. A. (1979) Infants of very low birth weight: a 15 year analysis. Lancet (i): 1332-1335.

Kane, S. H. (1964) Significance of autopsies in premature infants. J.A.M.A. 187: 865.

Kermack, W. O., McKendrick, A. G. and MacKindlay, P. L. (1934) Death rates in Britain and Sweden. Some general regularities and their significance. Lancet (i): 698-703.

Kitchen, W. H. and Campbell, D.G. (1971) Controlled trial of intensive care for very low birth weight infants. Pediatrics, 48: 711-714.

Knox, G. and MacKintosh, J. (1958) Postneonatal infant mortality in Birmingham between 1947 and 1956. Br. J. Prev. Soc. Med. 12: 131-134.

Koran, L. M. (1975a) The reliability of clinical methods, data and judgements. N. Engl. J. Med. 293: 642-646.

Koran, L. M. (1975b) The reliability of clinical methods, data and judgements: II Interpreting diagnostic procedures. N. Engl. J. Med. 293: 695-701.

Kukull, W. A. and Peterson, D. R. (1977) Sudden infant death and infanticide. Am. J. Epidemiol. 106: 485-486.

Lancet (1979) Letters to Editor, Lancet 1979 (ii): 254-255 and 362-363.

Lancet (1980) Better perinatal health. Collected reprints of a series published in The Lancet between November 17, 1979 and January 12, 1980. The Lancet, London.

Landsteiner, K. (1900) Zur Kenntnis des antifermentativen, lytischen und agglutiniesenden Wiskungen des Blutserums, und der Lymphe. Zentralb. f. Bakteriol. 27: 357. Cited by Wiener, A. S. and Peters, H. R. (1940).

Landsteiner, K. and Wiener, A. S. (1940) An agglutinable factor in human blood recognised by immune sera for rhesus blood. Soc. Exp. Biol. N.Y., 43: 223.

Leck, I. and Record, R. G. (1963) Sources of variation in the reporting of malformations. Dev. Med. Child Neurol. 5: 364-369.

Levine, P. (1943) Serological factors as possible causes in spontaneous abortions. J. Hered. 34: 71-80.

Levine, P., Burnham, L., Katzin, E. M. and Vogel, P. (1941) The role of isoimmunisation in the pathogenesis of erythroblastosis fetalis. Am. J. Obstet. Gynecol. 42: 925-937.

Liley, A. W. (1963) Intrauterine transfusion of foetus in haemolytic disease. Br. Med. J. (ii): 1107-1109.

Lillienfeld, A. M. (1976) In Foundations of Epidemiology. Oxford University Press, New York.

Lister, J. (1973) Selection for treatment in spina bifida cystica (letter). Br. Med. J. (4): 355.

Local Government Board (1889) Diarrhoea and diphtheria. Supplement in continuation of the Report of the Medical Officer for 1887. H.M.S.O., London.

Local Government Board (1893) Enteric fever in the Tees valley. Supplement in continuation of the Report of the Medical Officer for 1891. H.M.S.O., London.

Lorber, J. (1973) Early results of selective treatment of spina bifida cystica. Br. Med. J. (4): 201-204.

Lunn, J. E., Knowelden, J. and Handyside, A. J. (1967) Patterns of respiratory illness in Sheffield infant schoolchildren. Br. J. Prev. Soc. Med. 21: 7-16.

Lunn, J. E., Knowelden, J. and Roe, J. W. (1970) Patterns of respiratory illness in Sheffield junior schoolchildren, a follow up study. Br. J. Prev. Soc. Med. 24: 223-228.

McCarthy, B. J., Terry, J., Rochat, R. W., Quave, S. and Tyler, C. W. (1980) The underregistration of neonatal deaths: Georgia 1974-77. Am. J. Public Health, 70: 977-982.

McDonald, A. D. (1961) Maternal health in early pregnancy and congenital defect: Final report on prospective inquiry. Br. J. Prev. Soc. Med. 15: 154-166.

Macfarlane, A., Chalmers, I. and Adelstein, A. M. (1980) The role of standardisation in the interpretation of perinatal mortality rates. Health Trends, 12: 45-50.

McKendrick, W., Gully, P. R. and Geddes, A. M. (1980) Protection against pertussis by immunisation. Br. Med. J. (2): 1390-1391.

McKeown, T. and Lowe, C. R. (1974) In An introduction to social medicine, 2nd ed. Blackwell Scientific Publications, Oxford.

McKeown, T. and Record, R. G. (1960) Malformations in a population observed for five years after birth. In Ciba foundation symposium on congenital malformations. Eds. G. E. W. Wolstenholme and C. M. O'Connor. Churchill, London.

Madden, M. (1981) Late relapse after presumed cure of acute lymphatic leukaemia. Lancet (ii): 306.

Mair, J. M. and Tait, H. P. (1953) Child deaths in Edinburgh 1901-51. Edinb. Med. J. 60: 212-222.

Marcusson, H. and Oehmisch, W. (1977) Accident mortality in childhood in selected countries of different continents. World Health Report Statistics, 30: 57-92.

Marsden, H. B. and Steward, J. K. (1976) In Tumours in Children. Springer-Verlag, Berlin.

Mather, H. G., Pearson, N. G., Read, K. L. Q., Shaw, D. B., Steed, G. R., Thorne, M. G., Jones, S., Guerrier, C. J., Eraut, C. D., McHugh, P. M., Chowdhury, N. R., Jafary, M. H. and Wallace, T. J. (1971) Acute myocardial infarction: home and hospital treatment. Br. Med. J. (3): 334-338.

Mollison, P. L. and Walker, W. (1952) Controlled trials of the treatment of haemolytic disease of the newborn. Lancet (i): 429-433.

Moore, A. (1981) The sudden infant death syndrome. Br. J. Hosp. Med. 26: 37-45.

Morgagni, J. B. (1761) The seats and causes of disease investigated by anatomy (translated by Cook, W. 1882). London, Longman. Cited by Frank, J. D. and Fixsen, J. A. (1980).

Neel, J. V. (1958) A study of major congenital defects in Japanese infants. Am. J. Hum. Genet. 10: 398-445.

Nelson, M. M. and Forfar, J. O. (1969) Congenital abnormalities at birth: their association in the same patient. Dev. Med. Child Neurol. 11: 3-16.

Nevanlinna, H. R. and Vainio, T. (1956) The influence of mother-child ABO incompatibility on Rh immunisation. Vox Sang. 1: 26.

Newbigging, P. S. K. (1834) Spina Bifida: probationary essays, vol. VI. Royal College of Surgeons, Edinburgh. Cited by Frank, J. D. and Fixsen, J. A. (1980).

Nishimura, H., Takano, K., Tanimura, T., Yasuda, M. and Uchida, T. (1966) High incidence of several malformations in the early human embryos as compared with infants. Biol. Neonate, 10: 93-107.

O.H.E. (1979) Perinatal mortality in Britain: a question of class. Office of Health Economics, London.

O.H.E. (1980) Leukaemia towards control. Office of Health Economics, London.

O.P.C.S. (1976a) Child Health: a collection of studies. Studies on Medical and Population Subjects, No. 31. H.M.S.O., London.

O.P.C.S. (1976b) Anencephalus, spina bifida and congenital hydrocephalus. England and Wales, 1964-1972. Studies on Medical and Population Subjects, No. 32. H.M.S.O., London.

O.P.C.S. (1976c) Causes of children's deaths analysed by social class (1959-63). In Studies on Medical and Population Subjects No. 31. H.M.S.O., London.

O.P.C.S. (1979a) Infant and Perinatal Mortality (O.P.C.S. Monitor DH1 79/2). H.M.S.O., London.

O.P.C.S. (1979b) Infant and Perinatal Mortality. Supplement Trent. (O.P.C.S. Monitor DH1 79/1). H.M.S.O. London.

O.P.C.S. (1974-78) Series VS. H.M.S.O., London.

O.P.C.S. (1974-1980) Mortality statistics: series DH1: review of the Registrar General on deaths in England and Wales. H.M.S.O., London.

O.P.C.S. (1980a) Mortality statistics, series DH3, No. 5. H.M.S.O., London.

O.P.C.S. (1980b) Abortion statistics, series AB, No. 5. H.M.S.O., London.

O.P.C.S. (1980c) Sudden infant death syndrome, 1971-1978 (O.P.C.S. Monitor DH3 80/3). H.M.S.O., London.

Östberg, G. (1973) Child mortality in Malmö. Arch. Dis. Child, 48: 159.

Parish, W. E., Barrett, A. M., Coombs, R. R. A., Gunther, M. and Camps, F. E. (1960). Hypersensitivity to milk and sudden death in infancy. Lancet (ii): 1106-1110.

Peterson, D. R. (1980) Evolution of the epidemiology of sudden infant death syndrome. Epidemiol. Rev. 2: 97-112.

Pharoah, P. O. D. and Morris, J. N. (1979) Postneonatal mortality. Epidemiol. Rev. 1: 170-183.

Pharoah, P. O. D. and Alberman, E. D. (1981) Mortality of low birthweight infants in England and Wales 1953 to 1979. Arch. Dis. Child. 56: 86-89.

Pilling, H. H. (1976) Sudden and unexpected deaths in infancy and the pattern of postneonatal deaths in Sheffield 1961-72: the Coroner's view. Med. Sci. Law 16: 25-28.

Plater, F. (1614) Observationum, lib. iii: 748. Basileae. Cited by Ballantyne, J. W. (1892).

Pollard, S. and Hunt, A. J. (1956) The city of Sheffield. The growth of population. In Sheffield and its region. A scientific and historical survey. Ed. D. L. Linton. British Association for the Advancement of Science, Sheffield.

Prime (1979) S.P.S.S. on Prime computers. Prime Computers Inc., 500 Old Connecticut Path, Framingham, Massachusetts 01701.

Puffer, R. R. and Serrano, C. V. (1976) The inter-American investigation of mortality in childhood. World Health Stat. Rep. 29: 493-520.

Pursall, E. W., Jepson, M. E., Smith, B. A. M. and Emery, J. L. (1978) Breast-feeding and mother's education (letter). Lancet (ii): 734-735.

Quick, J. D. (1978) Liberalised abortion in Oregon: Effects on fertility, prematurity, fetal death and infant death. Am. J. Public Health 68: 1003-1008.

Rabson, S. M. (1949) Sudden and unexpected natural death. IV Sudden and unexpected natural death in infants and young children. J. Pediatr. 34: 166-173.

Race, R. R. and Sanger, R. (1950) In Blood groups in man. Blackwell Scientific Publications, Oxford.

Rawlings, G., Reynolds, E. O. R., Stewart, A. and Strang, L. B. (1971) Changing prognosis for infants of very low birth weight. Lancet (i): 516-519.

Reed, D. M., Bakkteig, L. S. and Nugent, R. P. (1978) The epidemiology of respiratory distress syndrome in Norway. Am. J. Epidemiol. 107: 299-310.

Registrar General (1839-1973) Annual Report of the Registrar General. H.M.S.O., London.

Registrar General (1958) The Registrar General's Statistical Review for 1956 Part III: 182-207. H.M.S.O., London.

Registrar General (1960) Classification of Occupations 1960. H.M.S.O., London.

Registrar General (1968) Registrar General's Statistical Review for 1966, Part III: 73-77. H.M.S.O., London.

Registrar General (1969) Registrar General's Statistical Review for 1967, Part III. H.M.S.O., London.

Robinson, R. J. (1981) The whooping-cough immunisation controversy. Arch. Dis. Child. 56: 577-580.

Rodeck, C. H., Kemp, J. R., Holman, C. A., Whitmore, D. N., Karnicki and Austin, M. A. (1981) Direct intravascular fetal blood transfusion by fetoscopy in severe rhesus isoimmunisation. Lancet (i): 625-627.

Rogers, P. B., Council, C. R. and Abernathy, J. R. (1961) Testing death registration completeness in a group of premature infants. Public Health Rep. 76: 717-724.

Royal College of Obstetricians and Gynaecologists and the Population Investigation Committee (1948) Maternity in Great Britain. (Douglas, J. W. B.). Oxford University Press. London.

Royal College of Obstetricians and Gynaecologists (1975) British Births 1970: a survey under the joint auspices of the National Birthday Trust and the Royal College of Obstetricians and Gynaecologists. Vol. I: The first week of life (Chamberlain, R., Chamberlain, G., Howlett, B. and Claireaux, A.). Heinemann Medical, London.

St. Leger, A. S., Cochrane, A. L. and Moore, F. (1978) The anomaly that wouldn't go away. Lancet (ii): 1153.

Sadler, J. (1972) Children and road safety: a survey among mothers. H.M.S.O., London.

Salge, B. (1911) Die physikalischen Eischeinungen des Blutes beim gesunden und kranken Sängling: der osmotische Druck. Zeit. für Kinderheilk. 1: 126-138.

Sandahl, B. (1978) Seasonal birth pattern in Sweden in relation to birth order and maternal age. Acta Obstet. Gynecol. Scand. 57: 393-396.

Sandels, S. (1975) Children in traffic. Elek, London.

Sandels, S. (1977) An overall view of children in traffic. In Children, the environment and accidents. Ed. R. H. Jackson. Pitman Medical, Tunbridge Wells.

Saunders, G. L. (1860) In Town and country: being a brief sanitary investigation into the causes of the difference of the death-rate in urban and rural districts; also an enquiry into the health of Sheffield. Harrison, Sheffield.

Scott, M. J., Ritchie, J. W. K., McClure, B. G., Reid, M. McC. and Halliday, H. L. (1981) Perinatal death recording: time for a change? Br. Med. J. (1): 707-710.

Sharrard, W. J. W., Zachary, R. B., Lorber, J. and Bruce, A. M. (1963) A controlled trial of immediate and delayed closure of spina bifida cystica. Arch. Dis. Child. 38: 18-22.

Sheffield (1874-1973) The Health of Sheffield. The annual report of the Medical Officer of Health.

Sibert, J. R., Craft, A. W. and Jackson, R. H. (1977) Child-resistant packaging and accidental poisoning. Lancet (ii): 289-290.

Simpson, K. (1947) Pathology of sudden death. Lancet (ii): 745-747.

Singer, C. and Underwood, E. A. (1962) In A short history of medicine. Oxford University Press, London.

Slatis, H. M. and de Cloux, R. J. (1967) Seasonal variations in stillbirth frequencies. Hum. Biol. 39: 284-295.

Smith, B. A. M. (1974) Feeding overstrength cow's milk to babies. Br. Med. J. (4): 741-742.

Speizer, F. E., Doll, R. and Heaf, P. (1968a) Observations on recent increase in mortality from asthma. Br. Med. J. (1): 335-339.

Speizer, F. E., Doll, R., Heaf, P. and Strang, L. B. (1968b) Investigations into use of drugs preceding death from asthma. Br. Med. J. (1): 339-343.

Spence, J. C., Walton, W. S., Miller, F. J. W. and Court, S. D. M. (1954) A thousand families in Newcastle-upon-Tyne. Oxford University Press, London.

S.P.S.S. (1975) Statistical Package for the Social Sciences (Nie, H. H., Hull, C. H., Jenkins, J. G., Steinbrenner, K. and Bent, D. H.) 2nd edition. McGraw Hill, New York.

Starfield, B. (1981) Patients and populations: necessary links between the two approaches to pediatric research. Pediatr. Res. 15: 1-5.

Steiner, E. S., Saunders, E. M., Phillips, E. C. K. and Maddock, C. R. (1980) Very low birth weight children at school age: comparison of neonatal management methods. Br. Med. J. (2): 1237-1240.

Stewart, A. L. (1977) The survival of low birth weight infants. Br. J. Hosp. Med. 18: 182-190.

Stewart, A. L., Reynolds, E. O. R. and Lipscomb, A. P. (1981) Outcome for infants of very low birthweight: survey of world literature. Lancet (i): 1038-1040.

Sunderland, R. (1980) Children's building bricks for three-dimensional illustrations. Lancet (ii): 863.

Sunderland, R. (1981a) Depressing effect of burial charges on vital statistics. Lancet (ii): 309.

Sunderland, R. (1981b) Abortion and perinatal mortality rates. Lancet (i): 896.

Sunderland, R. and Emery, J. L. (1981) Febrile convulsions and cot death - an hypothesis. Lancet (ii): 176-178.

Sunderland, R., Carpenter, R. G. and Gardner, A. (1982) Are all born equal? Incidence of febrile convulsions by season of birth. Brit. Med. J. (in press).

Tait, H. P. and Boog-Watson, W. N. (1962) Deaths among Edinburgh school children (1901-61). Scott. Med. J. 7: 425-430.

Taitz, L. S. and Byers, H. D. (1972) High calorie/osmolar feeding and hypertonic dehydration. Arch. Dis. Child. 47: 257-260.

Taylor, J. (1873) In An enquiry into the causes of the mortality of Sheffield. William Townsend and Son, Sheffield.

Templeman, C. (1892) Two hundred and fifty-eight cases of suffocation of infants. Edinb. Med. J. 38 (2): 322-329.

Tomkins,, A. (1981) Nutritional status and severity of diarrhoea among pre-school children in rural Nigeria. Lancet (i): 860-862.

Toverud, K. (1949) Den Profylaktiske Pediatris Berettigeske og Novendighet. Nord. Med. 41: 922-928.

Tulp, N. (1652) Observationes Medicae. Amsterdam. Cited by Frank, J. D. and Fixsen, J. A. (1980).

Valdes-Dapena, M. A. (1967) Sudden and unexpected death in infancy: a review of the world literature 1954-1966. Pediatrics, 39: 123-138.

Waldron, H. A. and Vickerstaff, L. (1977) In Intimations of quality; antemortem and postmortem diagnosis. Nuffield Provincial Hospital Trust, London.

Walker, A. R. P. and Walker, B. F. (1978) Pure water and infections in Africa (letter). Lancet (ii): 639.

Warkany, J. (1971) In Congenital Malformations. Year Book Medical Publishers Inc., Chicago.

Weatherall, J. A. C. (1976) Foundlings. In Studies on Medical and Population Subjects No. 31 (see O.P.C.S. 1976a). H.M.S.O., London.

Weatherall, J. A. C. (1980) Eurocat guide to eponyms and synonyms. E.E.C. Concerted Action Project, Brussels.

Werne, J. (1942) Postmortem evidence of acute infection in unexpected death in infancy. Am. J. Pathol. 18: 759-761.

Werne, J. and Garrow, I. (1953a) Sudden apparently unexplained death during infancy. I Pathologic findings in infants found dead. Am. J. Pathol. 29: 633-675.

Werne, J. and Garrow, I. (1953b) Sudden apparently unexplained death during infancy. II Pathologic findings in infants observed to die suddenly. Am. J. Pathol. 29: 817-831.

W.H.O. (1967) The accuracy and comparability of death statistics. W.H.O. Chronicle, 21: 11-17.

W.H.O. (1977) Manual of the International Statistical Classification of Diseases, Injuries and Causes of Death. Ninth Revision. World Health Organisation, Geneva.

Wiener, A. S. and Peters, H. R. (1940) Hemolytic reactions following transfusions of blood of the homologous group, with three cases in which the same agglutinogen was responsible. Ann. Intern. Med. 13: 2306-2322.

Wyld, P. J. and Lilleyman, J. S. (1981) Late relapse after presumed cure of childhood lymphoblastic leukaemia. Lancet (ii) : 86.

Wynn, M. and Wynn, A. (1976) Prevention of handicap of perinatal origin: an introduction to French policy and legislation. Foundation for Education and Research in Child-Bearing, London.

Wynn, M. and Wynn, A. (1977) The prevention of preterm birth: an introduction to some European developments aimed at prevention of handicap. Foundation for Education and Research in Child-Bearing, London.

Wynn, M. and Wynn, A. (1979) Prevention of handicap and the health of women. Routledge and Kegan Paul, London.

Yeats, W. B. (1889) The Ballad of Moll Magee. In The collected poems of W. B. Yeats (1971) Macmillan, London.