A CLINICAL STUDY OF POST PERINATAL DEATHS IN SOUTHERN DERBYSHIRE

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Based on work carried out in Southern Derbyshire Health District

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Jarman Index Related to Post perinatal Mortality.
Public Health 1992,106;163-165.

c) Newlands M, Emery J L.
Child Abuse and Cot Deaths.
Child Abuse & Neglect,1991,15;275-278.

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SUMMARY

This study was undertaken to investigate why in Southern Derbyshire, a district apparently representative of much of England and Wales, the Infant Mortality Rate since 1975 has been generally higher than the national rate.

Between 1981 and 1983 the use of national and small area statistics identified that rates varied significantly above the national averages (Relative Risk = 1.18 [95% C.I. 1.01 - 1.34] $X^2 = 7.01$ df = 1 $p < 0.01$) and that the problem lay in both the perinatal and post perinatal period.

This study is based upon a hypothesis that:- Elucidation of the determinants of infant mortality requires more than the collection of routine clinical and statistical data.

The study is presented therefore as a thesis constructed from a series of clinical and epidemiological investigations with the following objectives.

1. To establish the geographical location of all post perinatal deaths and to relate them to the demographic and socio-economic features of the District.
2. To define criteria for differentiating possibly avoidable and preventable from probably not preventable deaths.
3. To identify defects in clinical and community health services which could have contributed to avoidable and preventable deaths.
4. To investigate each death in detail to identify features which might have been contributory to the death and which, if avoided, could have influenced the outcome.
5. To investigate differences in causes and patterns of deaths between the north and the south of the County of Derbyshire.
6. To identify factors which may explain why, in the decade since 1981, death rates in Southern Derbyshire were higher, and those in North Derbyshire lower than the national averages.

As a background to the study, the history, geography and demography of Derbyshire was reviewed:

The Registrar General's censuses of populations 1971 and 1981 indicated that Southern Derbyshire was unremarkable to the national demographic status. The main differences between Southern Derbyshire and England and Wales were the proportions of ethnic

minorities, migration of families and home ownership. Local demographic and socio-economic features showed areas of deprivation within Derby City and areas of economic decline to the east and south of the District due to closure of coal mines and decline in the heavy engineering industries.

Historically, Derbyshire appears to have reached its zenith in early Georgian times when Darwin and Wedgwood made their mark. Derbyshire was a leader in the industrial revolution and enjoyed the notoriety of the group of intellectuals known as the Lunatic Society. Derbyshire has never been a leader in the field of medical progress yet in 1908 Sidney Barwise, the then County Medical Officer of Health, made the observation that infant deaths should be divided into two groups - preventable and not preventable. He was locally the first person to make this observation, which is again tested in the present thesis.

Two mortality studies had previously been undertaken in Southern Derbyshire. These were:-

1. A Derbyshire Area Working Party which was set up in 1978 following a request from the Secretary of State to the chairman of the Regional Health Authority in 1977.
2. The 1983/84 Trent Regional Confidential Enquiry into Child Deaths - 28 weeks gestation to one year after birth. This was a case control study and provided more detail.

My own studies which form the basis of this thesis were based on the following observations.

A similar scale of preventability rating was applied to trends in death registrations over a 15 year period in relation to changes in registered causes. In 1979 the group of diagnoses ascribed to trauma transferred into the Sudden infant death syndrome diagnosis, as did 25% of those deaths ascribed to infection. A data base of post perinatal mortality over a five year period confirmed that the local post perinatal death rates were high and that the profile of deaths was the same as elsewhere in the country.

Two further studies were undertaken using routine data collection. These comprised an evaluation of the effects of notifiable childhood infections on post perinatal mortality rates and evidence of workload and performance in the Primary Health Care Teams,

measured by standard indicators. These studies were limited in their contribution to the investigation of post perinatal mortality, but proved useful as management tools.

Concepts of preventability of death after birth were examined in the light of Barwise's principles and the findings from the 1983/84 study. The approach of grouping diagnoses by scale of preventability was the most pragmatic way of looking at the problems of post perinatal mortality. The study was concerned with the possible preventability of factors mitigating against the child after birth which were contributory to the child's death.

Having established a profile of death by scale of preventability by registrable cause, the first study of factors mitigating against the child was devised. This comprised confidential case review, and established a new data base within the District. Causal factors within the environment, medical and social care and adverse factors within the families were recorded for the first time. In a high proportion of the post perinatal deaths adverse medical and social factors were identified, some of which were actionable. Given the national prominence of Confidential Enquiries into Stillbirths and Deaths in Infancy (CESDI), evaluation of this method of enquiry was undertaken.

It was found that possibly preventable deaths presenting as sudden unexpected deaths accounted for 40.5% of all the post perinatal deaths (refer to Section 7, p46).

Dual epidemics of measles and pertussis did not appear to contribute significantly to the post perinatal mortality rates (refer to Section 8, p71).

The Department of Health and Social Security (DHSS) recommended Performance Indicators, showed that some aspects of efficiency were measured, but there was no indication of quality or effectiveness of care (refer to Section 9, p77).

Preventability ratings are valid parameters for discovering failures in care and developing intervention strategies (refer to Section 10, p87).

The information obtained by Confidential Review revealed General Practitioner case discussion to be essential for confidential information, feedback, factor identification and guidance for action (refer to Sections 11 & 12, p93 & 117).

The Jarman scores of deprivation indicate areas of high post perinatal mortality, and in particular apply to the possibly preventable deaths (refer to Section 13, p132).

There is a definite association between child abuse and 10% of children presenting as unexpected deaths in the District (refer to Section 14, p135).

It is clear, from these results, that information obtained by routine procedures and enquiries, including that information from registrations, notifications and statistical returns, would not in themselves have been sufficient to elucidate differences in the components of the Infant Mortality Rate between those of Districts and the Nation. The hypothesis upon which the study is based is therefore upheld, while alternatives for the pursuit of a more informative and active programmes are suggested.

Section 1

INTRODUCTION AND HYPOTHESIS

The general reasons for undertaking the present study were twofold. Firstly, high infant mortality rates had been a cause for concern in the District during the 1970's[1]. Conventional data collection had not revealed the reason for these high rates. No one in the District had been identified to investigate post perinatal mortality. This was an important omission within the child care services and presented an opportunity for study.

Secondly, study of social and medical demography uncovers areas of need within the local population, but does not identify the needs of individual families nor the reasons for failure in delivery of care[2,3,4,5,6,7,8]. To this end a system of confidential review into all post perinatal deaths was undertaken to seek out adverse factors which in some way had been contributory to the child's death and which, if remedied or removed, would safeguard the lives of other children in similar circumstances.

Post Perinatal Infant Mortality Rates have long been used as one of a series of indicators of the health of the nation. Thirty five per cent of children dying in the post perinatal period die as cot deaths. Distinctions were made between Sudden Unexpected Infant Deaths (SIDS) and explained deaths in the realisation from previous clinical studies that Sudden Infant Death Syndrome constitutes 5-15% of children presenting as Cot Deaths[9,10].

The term Cot Death was first described in 1954 by Dr A M Barrett[11], a Pathologist at Cambridge University. He wrote "The term Cot Death is used here to include all cases in which an apparently healthy infant is unexpectedly found dead in its sleeping quarters, whether in a cot, pram or in any other kind of bed". It was not until 1969 that Beckwith[12], an American Pathologist, described Sudden Infant Death Syndrome, "The sudden death of an infant or young child which is unexpected by history and in which a thorough post mortem examination fails to demonstrate an adequate cause". This was accepted as a registerable cause of death in England and Wales in 1971 by the Registrar General and the Coroners Society of England and Wales[13], and by the WHO classification of diseases, 8th Revision 1968, as SIDS (ICD 795). The definition of SIDS has lately been revised by the National Institute of Child and Human Development[14] and is as follows:

"The sudden death of an infant which remains unexplained after thorough case investigation, including performance of a complete autopsy, examination of the death scene and review of the clinical history".

In 1979 the International Coding of Disease was revised through the 9th Edition and SIDS became (ICD 798). SIDS (ICD 798.0) included those deaths where the underlying cause of death was given as Sudden and Unexpected Infant Death. The coding rules for selecting the underlying cause of death, where two or more causes appear on the death certificate, ensured that a more specific cause is selected in preference to the less specific one.

SIDS was only selected when it was the sole cause of death mentioned on the death certificate. Up to, and including, 1985 these figures had been recorded for sudden and unexpected deaths at all ages from birth upwards. But since 1986 these figures have been recorded for sudden and unexpected deaths at all ages from the age of 28 days onwards.

It is possible to divide Cot Deaths into three categories[10].

1. The explained which are due to gross congenital anomalies, rare disorders, overwhelming infection, accidents or non accidental injury.
2. The partially explained deaths where minor disease or abnormality is found as contributory factor.
3. The totally unexplained deaths.

Only the totally unexplained deaths or partially explained deaths are described as Sudden Infant Death Syndrome, which is not a disease but a diagnosis of exclusion where no explicit cause of death can be identified. Because of the manner of registration of such deaths it is difficult to distinguish between the explained, partially explained and unexplained deaths from death certification.

In Derbyshire a preliminary investigation into Infant Mortality Rates occurred in 1978 as a result of a directive from the Department of Health and Social Security[15]. The Area Medical Officer convened an Area Working Party under the chairmanship of one of the specialists in Community Medicine. This Working Group was followed by an initial fall in Infant Mortality Rates and led to a case control study of the deaths of infants aged 28 weeks gestation to one year after birth. This was part of a multicentre study in Trent Region[16] and presented the opportunity for a detailed enquiry into Southern

Derbyshire's high infant mortality rates. The result provided information unique to the District, but was not detailed enough to reveal the reasons for the high rates. To obtain more detail about the family circumstances, I set up the study by confidential case review to identify adverse factors which may have contributed to the child's death.

This thesis addresses the process of identification of these factors and the source of information to investigate the hypothesis that:- Elucidation of the determinants of infant mortality requires more than the collection of routine clinical and statistical data.

Section 2

THE GEOGRAPHY OF DERBYSHIRE

The Location

The administrative County of Derbyshire is situated in the Midlands region of England and occupies 650,000 acres stretching 60 miles from the north to the south and 45 miles from west to east. Its central location is complemented by the proximity to the national motorway network and its inclusion in the main line railway system. It is also well served by the East Midlands and Manchester international airports (Figure 2.1, p18).

Derbyshire has no major conurbation but it adjoins, and is influenced by, the larger cities of Manchester to the north west, Sheffield to the north, Birmingham to the south west and Nottingham to the east.

There is one local authority administration but in the 1982 reorganisation of the National Health Service, the County was divided into two main health districts - the North Derbyshire and the Southern Derbyshire Health Authorities which constitute two of 11 districts of the Trent Regional Health Authority. Population wise, Trent region is one tenth the size of England and Wales and for resource allocation, Southern Derbyshire is one eleventh the size of Trent region, being the largest non teaching district and the sixth largest health district within England.

Geographical Regions

Within Derbyshire contrasting geographical environments and landscapes are juxtaposed (Figure 2.2, p19). To the north west is Britain's first national park, the Peak District, with its wild moorland, exposed mountain plateau and deeply cut river valleys. Further south are the Derbyshire Dales, the green river valleys of the Rivers Wye, Lathkill and Dove which have been renowned for their fishing and walking potential for centuries. Isaak Walton described their beauty in his book "The Compleat Angler". Country houses such as Chatsworth, the home of the Dukes of Devonshire, and Haddon Hall are renowned. With approximately one third of Great Britain's inhabitants living within 50 miles of Derbyshire these landscapes are under pressure for recreational use. There are other conflicts of interest manifest between agricultural and forestry practices and between conservation and development. Special landscapes have now been defined

lying mainly in the west of the County where housing and agricultural development are controlled[4,17,18].

To the south and the west of the County lie the dairying areas of the red marls where Ashbourne, a county market town, is sited. To the east, the coal measures of the Erewash, Doe Lea and Rother valleys have until recently supported traditional heavy coal based industries. Landscape scars still remain despite the high priority of reclamation of derelict land in the County's approved structure plan. The acres of derelict land are the landscape evidence of the general process of de-industrialisation where the heritage of derelict railways and canals, old sewage works and landfill sites still remains. Derbyshire is still the leading mineral producing county in the country being a major producer of limestone, coal and dolomite. There are considerable reserves of sand and gravel and recently an interest has been shown in oil and gas exploration. Socially and economically a decline in these industries is reflected in high unemployment rates (up to 15% in 1987).

North Derbyshire District (Figure 2.3, p20)

Rykneld Way, a Roman road, runs north and south dividing the district with two thirds of the territory to the west of the district where only one third of its inhabitants reside. Chesterfield, with a population of 97,000 persons sits astride a Roman road and is famous for its church spire which was built about 1400 to a height of 228 feet and is renowned for leaning from west to the east. The economy of Chesterfield is based on coal mining, foundries and heavy engineering, all of which are in a long term decline. It is described as a poor environment with dereliction. Since 1983 due to contraction of the traditional large industries unemployment has risen above the county and national levels.

North East Derbyshire - in the hinterland of Sheffield

The north east of the County comprises flatlands where mining towns are depressed by pit closures. Closure of the large colliery in Shirebrook district in 1986 caused major unemployment problems offset to some degree by employment in quarrying, heavy engineering and steel production. Also the district is still occupied in the mining industry.

North West Derbyshire

North West Derbyshire comprises small market towns by trout fishing rivers in the southern foothills of the Pennines. The subterranean caves in this district are renowned

for the pretty crystals of Blue John. The small rural communities are isolated and still have need for dispersal of maternity, emergency and out-patient facilities. The population are engaged in farming, quarrying and tourist industries. Well dressing is a custom carried out in west Derbyshire. The materials used in each well dressing are dictated by tradition. Well dressing was said by some to be a thanksgiving from 1615 when during a severe drought the wells never ran dry.

Matlock

Matlock is at the centre of North and Southern Derbyshire and is the site of the gorge where the river Derwent cuts through the limestone. It was not only a spa but was the centre where water power was developed in the Industrial Revolution. The town was previously a fashionable resort for the middle classes in the 19th century and has now emerged as a tourist centre and the administrative centre for the County Council services. The rather restricted outlook of Derbyshire people as perceived by Sir John Betjamin, was described in his poem "Matlock Bath" (Figure 2.4, p21).

Derby

Derby, with a population of 210,000 is England's newest City having gained status by Royal Assent in 1977, Jubilee Year. It was a leader in the Industrial Revolution, having a long history of manufacturing industry in textiles, ceramics and in engineering. The economy accounts for almost one third of the County's total employment. At present Rolls Royce Aerospace equipment is the dominant employer but there is diverse employment in railway engineering, research and development. Growth of employment in business, administration, service, tourist and distribution trades has compensated in some degree for the job losses in manufacture. The future is more secure given the boost to the area gained by the location of a Toyota car manufacturing plant 5 miles to the south of the City and by the enhanced accessibility associated with the Stoke to Derby motorway link to be completed in the early 1990s. Socially within the inner city areas of multiple deprivation have been highlighted[4] but these have been the target for positive discriminatory investment by both public and private sectors.

Alfreton, Ilkeston and Long Eaton

These smaller towns of eastern Derbyshire are all facing a period of readjustment economically and socially following the long term decline in the traditional industries of coal mining and textiles. Growth in service employment and high technology industries has occurred but a mismatch of skills has caused further unemployment. These areas seem destined to become commuter settlements for Nottingham and Derby.

Ashbourne, Mackworth and Wirksworth Districts

These are major tourist areas and were known for their textile and mineral industries. Ashbourne is a major tourist centre and is famous for its Shrovetide football which was introduced during the reign of Elizabeth I.

Swadlincote

Swadlincote is the largest rural community to the south of the County and is situated on the Southern Derbyshire coalfield. It is an area in long term decline resulting in employment loss and environmental decay and dereliction. The forthcoming closure of another colliery compounds the problems of employment in this area.

The County of Derbyshire thus comprises an area of sharp geological and economic contrasts, varying from the beautiful and conserved west and the isolated villages of the north west of the county to the heritage of dereliction and decay from declining industry in the east of the County.


DERBYSHIRE
Location
N
0
100
200 km
Leeds
Manchester
Sheffield
DERBYSHIRE
Derby
Nottingham
East Midlands
Airport
Birmingham
M1
LONDON

Figure 2.1


DERBYSHIRE
Geographical Features
West Yorkshire
TAMESIDE and GLOSSOP
HEALTH
AUTHORITY
Greater
Manchester
South Yorkshire
HIGH PEAK
NORTH DERBYSHIRE
HEALTH AUTHORITY
Cheshire
Buxton
BOLSOVER
CHESTERFIELD
DERBYSHIRE
DALES
NORTH EAST
DERBYSHIRE
River Derwent
Nottinghamshire
Staffordshire
Ashbourne
Ilkeston
EREWASH
DERBY
Long
Eaton
SOUTHERN
DERBYSHIRE
HEALTH
AUTHORITY
SOUTH
DERBYSHIRE
River Trent
Swadlincote
Leicestershire
Land over 200 metres
River
Boundary of Health Authority
County or District boundary
Selected town
0
10 km

Figure 2.2

Location Map

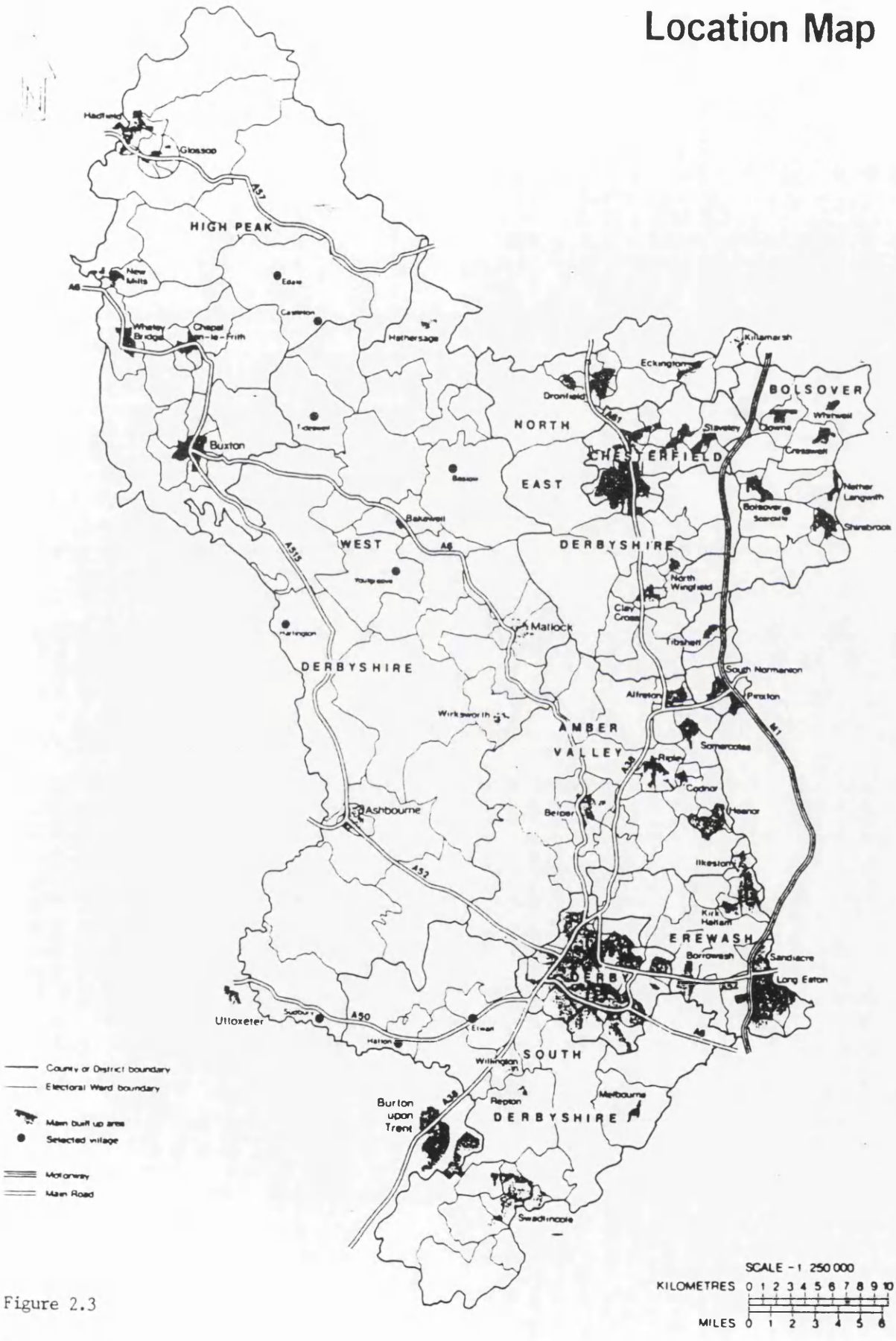


Figure 2.3

Matlock Bath

From Matlock Bath's half-timbered station
  I see the black dissenting spire—
Thin witness of a congregation,
  Stone emblem of a Handel choir;
In blest Bethesda's limpid pool
Comes treacling out of Sunday School.

By cool Siloam's shady rill—
  The sounds are sweet as strawberry jam:
I raise mine eyes unto the hill,
  The beetling HEIGHTS OF ABRAHAM;
The branchy trees are white with rime
In Matlock Bath this winter-time,

And from the whiteness, grey uprearing,
  Huge cliffs hang sunless ere they fall,
A tossed and stony ocean nearing
  The moment to o'erwhelm us all:
Eternal Father, strong to save,
How long wilt thou suspend the wave?

How long before the pleasant acres
  Of intersecting LOVERS' WALKS
Are rolled across by limestone breakers,
  Whole woodlands snapp'd like cabbage stalks?
O God, our help in ages past,
How long will SPEEDWELL CAVERN last?

In this dark dale I hear the thunder
  Of houses folding with the shocks,
The GRAND PAVILION buckling under
  The weight of the ROMANTIC ROCKS,
The hardest Blue John ash-trays seem
To melt away in thermal steam.

Deep in their Nonconformist setting
  The shivering children wait their doom—
The father's whip, the mother's petting
  In many a coffee-coloured room;
And attic bedrooms shriek with fright,
For dread of *Pilgrims of the Night.*

Perhaps it's this that makes me shiver
  As I ascend the slippery path
High, high above the sliding river
  And terraces of Matlock Bath:
A sense of doom, a dread to see
The *Rock of Ages cleft for me.*

Figure 2.4

Section 3

DEMOGRAPHY

National demographic and social changes between the 1971 and 1981 censuses[5,6]. showed in the increasing proportion of the population in the older age groups and a declining share in the youth. Family and household arrangements were attended by declining marriage rates, later marriages, more divorces and fewer children per couple. There was a wider entry into the population of youth, women in paid work and two career couples. There was a growth in the ethnic minority groups and wider disparities between the rich and the poor. All this was also true of Derbyshire[4], although the proportion of the population from the ethnic minority group remained less than the national and there were proportionally more owner-occupied homes and less unemployment[6,8].

A summary of the 1981 census of the population is shown in Figure 3.1 and denotes comparisons between North and Southern Derbyshire, Trent Region and England and Wales. Cells of the 1981 census used by Jarman[19,20,21] to define variables for underprivileged area scores in England, Wales and Scotland are included It was recognised that these scores denoted urban rather than rural deprivation, but their universal usage in Great Britain enabled comparison on a constant basis.

The county of Derbyshire comprises 208 electoral wards. These were used as units to measure variation in key social and economic indicators across the County[7,18,21]. The most densely populated areas in the County were Derby City, which contained 49% of the population of Southern Derbyshire, Chesterfield to the north of the County and the small urban conurbations along the eastern borders of the County. Population numbers indicate that Southern Derbyshire comprises 1.1% of the total population of England and Wales, and 11.4% of Trent Region of which it forms a part.

North Derbyshire is approximately two thirds the population size of Southern Derbyshire, having a population of 357,773 persons[6]. Apart from the size of the populations of North And Southern Derbyshire the main differences lie in the proportion of ethnic minority groups within the two districts, home ownership and the proportion of unemployed (Figure 3.1, p24). In 2.4% of the population of Southern Derbyshire the head of the household was born in the New Commmonwealth or Pakistan, compared

with 0.1% in North Derbyshire. Eighty two per cent of the ethnic minority population of Derbyshire reside in Derby City.

Jarman deprivation scores[19,20,21] were calculated using the information shown in Figure 3.1 for both North and Southern Derbyshire. The deprivation score for North Derbyshire was -14.22 compared with -4.22 in the South, neither District being deprived compared with the national averages[22,23]. When deprivation scores were calculated by electoral ward, in the South there were eight electoral wards with deprivation scores greater than 20, while in North Derbyshire there were none.

1981 Census of Populations Comparison - Derbyshire with Trent Region and England and Wales

1981 Census	England & Wales	Trent Region	County of Derbyshire	N. Derbys. Health District	S. Derbys. Health District
Total Population (Numbers)	45771956	4520612 (9.8%)	901831 (2%)	357773 (40% of Derbys.)	515472 (11% Region/ 1% E and W
0-4 yrs %	6%	5.9%	5.9%	5.6%	6%
0-15 yrs	22.2%	22.6%	22.3%	21.8%	22.5%
Over 65's	17.5%	17.1%	17.2%	16.8%	17.4%
New Commonwealth and Pakistan	4.5% #	2.3%	2.5% #	0.1% *	2.4%*
% Migrants 1980/81	9.2%	9%	8.1%	7.9%	8.5%
% 1 parent families	2.1%	2.8%	1.7%	1.5%	1.8%
% 16+ econ active	61%	61%	60.6%	59.5%	61.2%
% Econ active men unemployed	13.9%	9.65%	11.2%	8.5% *	11.4% *
% Econ active women unemployed	5.8%	6.1%	4.2%	5.8% *	4.2% *
% Unemployed	9.3% #	8.3%	7.7% #	7.5% *	8.9% *
% House owner occupied	55.9% #	56%	60.1% #	55.3% *	63% *
% Council house	31.3%	32.2%	28.8%	33.1% *	25.7% *
% Privately rented	12.8%	11.8%	11.1%	10.5%	9.4%
All amenities	95.6%	95.4%	95.1%	96.2%	94.3%
% 1.5 persons 1/more room	0.8%	0.42%	0.3%	0.3%	0.4%
Household no car	38.6%	41%	39.4%	39.9%	39.1%
Jarman Depriv.	0			-14.22	-4.22

* denotes differences between North and Southern Derbyshire
# denotes differences between the County of Derbyshire and England and Wales.

Figure 3.1

Section 4(a)

RELEVANT GENERAL HISTORY OF INFANT MORTALITY

"The Infant Mortality Rate is the most sensitive subtle index of all social welfare and of sanitary administration especially under urban conditions." Sir George Newman[24].

Infant mortality was first recorded by the pastors of parishes in England during the latter half of the 16th century. Wrigley and Schofield[25] researching these registers, deduced infant mortality rates from 1550-1799. They observed that the infant mortality rate had increased by 25% during this time and that the highest rates were in urban parishes. These demographic trends were confirmed when official figures became available from all over the country in the 19th century[1,26,27]. Laslett[25] writes about the Pastor of Clayworth in Nottinghamshire who had visited all his parishioners on two occasions 1676 and 1688 and recorded the poor state of nourishment, housing and "medically ignorant and disease exposed people". There had been much remarrying and many one parent families. The average number of children per family was 7, yet Louis Henry[25] had diagnosed limitation of birth as far back as the late 17th century when researching Geneva records. Wrigley's findings were similar for the parish of Colyton in Devon[25]. The infant mortality rate slowly declined in the early part of the 19th century and fluctuated between 145-156 per thousand live births in 1841-1900[26,27].

Maud Pember Reeves[28], in whose home the Fabian Women's Group was founded in 1908, described the finding of a survey undertaken by the Fabian Group 1909-1913. The Fabian survey was primarily concerned with infant mortality and in some respects is a precursor of the confidential review undertaken in this District (refer to Section 11, p93).

The findings by the Fabian Women's group in this small study were as follows:-

1. There was no doubt that the healthy infant at birth was less healthy at three months and less healthy still at a year. By the time the child was old enough to go to school it had developed rickets or lung trouble through entirely preventable causes.
2. The outstanding fact about the children was their puny size and damaged health. The health of those who lived upstairs was better than those who lived on the ground floor and decidedly better than those who lived in the basements.

3. The first baby of the family enjoyed superior health. Was this due to (a) more food, (b) less overcrowding, (c) less exposure to infection, or a combination of all three?
4. The all embracing cause of sickly children is poverty. The proportion of infantile death rate of Hampstead to that of Hoxton was 18 to 140 per thousand live births.
5. The babies in the study, with the exception of a child who died of gastroenteritis and a child who died as sudden unexpected death, survived their first year, but did not fulfil their first promise. By the age of one their environment had put its mark upon them.

The Fabian study showed where many of the problems lay.

Maternal health and nutrition had effect on perinatal and neonatal mortality rates, but environmental improvements pointed to better survival of young children after birth.

Peaks in infant mortality[29] occurred in 1911, 1915 and 1918. These coincided with epidemics of whooping cough, measles and meningitis.

Regional variations in Infant Mortality Rates were apparent, the rates being higher in the North of England than in the South[29,30].

Areas where overcrowding in the home were most severe showed the best improvements in mortality rates, while Hope[25] found that extra domestic female labour was not responsible for excess infant mortality although this had previously been blamed.

During the 1914-18 war the birth rate declined throughout Europe[29,30,31], but it was only in England and Wales where the infant mortality rates fell. The birth deficit was thus unlikely to be the cause of the declining rates[30,31]. The most striking improvements were in the urban areas, for example, in Wigan the infant mortality rate 1901-1903 was 181 and declined to 119 per thousand live births by 1919. Similarly in Bethnal Green the infant mortality rate fell from 150 to 93 per thousand live births and in Coatbridge there was a decline of 19% compared with 13% for the whole of Scotland[25,30].

Winter[30], in his book "The Great War and the British People" describes the effects of war on the civilian health and population trends. His opinions support the argument that improved standards of living were responsible for the decline in infant mortality rates

rather than public health measures or medical services during the war. However, Henry Williamson[32] describes the deprivation suffered by families while their fathers were at Ypres.

Between 1900 and 1930 the infant mortality rates in England and Wales fell by one half and in Scotland by one quarter. In 1934 Sir George Newman[24] reported "No spectacular improvement can be anticipated as the lower the rate the nearer one approaches the irreducible minimum which must always remain." He had compared the years 1921 and 1933[26]. "The factors that were formerly held to be productive of much ill health and mortality in infants have been eliminated to such an extent that there must be other factors to be taken into account. There is improved housing with personal and domestic cleanliness, increased attention to sanitation, disposal of refuse, storage and better preparation of food and relative freedom from dust in the public thoroughfares. There is also home visiting, medical consultation and treatment centres with personal nurturing in infancy"[24].

In 1941 the birth rate was the lowest recorded from 1900-1970, 13 per thousand population compared with 17.6 in 1938. Thereafter the birth rate began to rise sharply[26]. Many women became pregnant to avoid being conscripted for war work and pregnancy became known as "the prevalent disease". Longmate[33] describes how people lived during the Second World War. Many babies were born by candlelight at home without the help of a midwife. The war had cured unemployment, making it possible for people to feed their families. The tighter rationing became, the larger the share that went to the priority classes. Lord Woolton, who had started his career as a Social Worker in the Liverpool slums, was instrumental in the production of the green ration book for children under the age of five. Early in the war infant mortality showed a sharp rise, but by 1942 fell below the previous figures[26,27].

At the time of the reorganisation of health services in 1974[34,35] the infant mortality rates were falling slowly, and England and Wales had fallen behind other countries[3,26,27].

The Seebolm report[36] and the 1970 Social Services Act recommended that Social Workers should be able to deal with interrelated problems within the family context. Social services became visible and accessible to the local community, but they recognized that they could do virtually nothing to influence factors such as unemployment, low income, poor housing and educational under-attainment, all of which were causal factors to the problems of the families they were trying to help.

Social services could not make a major contribution unless there was a corporate strategy for ameliorating the problems of multiple deprivation.

The Court Committee's report "Fit for the Future" 1976[3], reviewed the existing health services for children. They found that environment and demographic features of the population played a large part in the improvement of infant mortality rate from 1948-1973 (rates 34 per thousand live births to 17 per thousand live births). Preventive measures, such as family planning, immunisation and better health education, improved the lot of our children, while more effective treatments, higher standards of medical and nursing care reinforced these improvements. He noted that our infant mortality rates had fallen behind those of other countries and within our own country there were disturbing regional and district differences, and differences between social classes; children were still dying from 19th century causes. The Court Committee looked at the changing pattern of family and social life, the changing pattern of disease and health care and at the training of professionals. The report advocated a combined approach for local authority services and health services and in the interim period the health services should respond by an improved type of health care. Despite the upsurge of political concern for children during the 1960s and 70s, the demographic and environmental changes on families had not been addressed and the prevalence of emotional and psychiatric disorders was unknown. After 30 years of the National Health Service there was still a marked class gradient in standards of health.

The Committee chaired by Black[2] submitted a report in 1980 in an endeavour to change administration within governmental social policies and identified proven health experience in the lower occupational groups. The report increased public and political concern but drew a negative response from the Government. One of the recommendations of this report was that there should be a shift of resources to community care. Studies by Wynn[37] and Morris[38] found that children had the greatest inequality of access to the health services.

The reorganisation of health services in 1982[39] was followed by a new management structure in 1984[40]. During this period the infant mortality rates declined at a very slow rate and deaths were more and more attributed to "preventable causes". In 1989 Hall[41], having taken account of the Black Report, stressed the importance of measuring the health of children in relation to social and environmental conditions.

Since the 1970s means of medical advancement have been increasingly challenged. Preventive medicine and core primary services are now the focus of attention. The Alma Ata declaration of 1977[42] placed primary health care at the centre of the health care system, and in 1981 the World Health Assembly adopted a global strategy of "Health for All" by the year 2000[43] and 38 targets were set. Target number 10 was the reduction in infant mortality rates.

Section 4(b)

THE LOCAL HISTORY OF DERBYSHIRE RELATING TO INFANT MORTALITY

Most of the information for this section has come from the reports of the Medical Officers of Health for Derbyshire 1897-1973.

The live birth rates in Derbyshire have followed the national averages since the turn of the century, as had the Infant Mortality Rates[25,26,27] (Figures 4.1 & 4.2, p32), yet Derbyshire trailed in the advancement of medical progress.

In 1897 the County Medical Officer of Health applauded the Public Health Amendment Act of 1890 and the Infectious Diseases Prevention Act of 1890 and was at pains to name the districts which had not yet adopted these Acts. There were still many deaths in the County from diarrhoea. Urban districts were most unsatisfactory, particularly the mining districts. Over the next few years the employment of women in factories, improper feeding and unnecessary exposure to cold and damp were postulated as the causes infant mortality. By 1905 the local infant mortality rate was five times higher in the urban and mining districts than in the rural areas.

In 1908 Dr. Barwise[44], County Medical Officer of Health, observed that infant deaths followed maternal malnutrition, infant mismanagement and neglect. In one borough where 88% of the mothers breastfed their children, the death rates in 0-1 year olds halved. He concluded that infants' deaths could be divided into two groups, preventable and not preventable. He calculated the Infant Mortality Rate should never exceed 75 per 1,000 live births. Barwise stressed the concept of groups of preventability of infant deaths and directed his efforts to the group of deaths he deemed preventable. Deaths due to prematurity and immaturity he recognised as due to factors beyond his control. He encouraged efficient water and sewage services. The districts which did not notify their births were chastised. He advocated the teaching of home economics and care of infants at schools and depots for the care of infants where babies were regularly weighed. The School Medical Officer was to call at the depots to give advice.

The fall in infant mortality rates continued. In Derbyshire they fell from 99 to 70.7 per 1,000 live births between 1911 and 1921, just below the national averages. The improved environmental conditions and the introduction of home visiting and medical

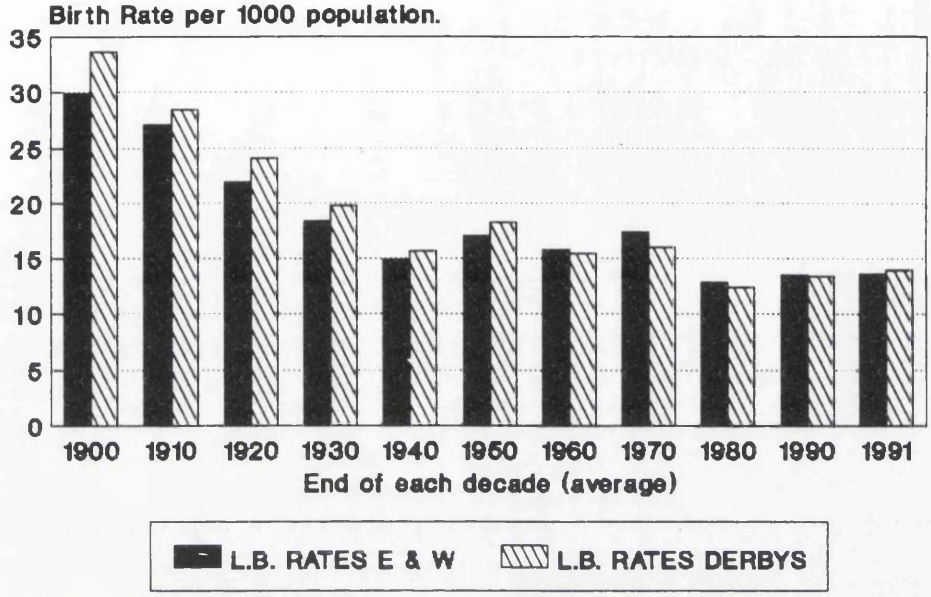
consultation and treatment centres, were the reasons given by the County Medical Officer of Health.

In 1937 Derbyshire promoted a local health week. This year also saw the first home helps in the County and home improvements were started under the Housing Act of 1936, Section 88. In 1941 whooping cough was a notifiable disease and the local public had at last accepted that diphtheria could be prevented. Immunisation was the major activity of the year (Infant Mortality Rates were 49.8 and 45.6 per thousand live births in England and Wales and Derbyshire respectively).

By 1944 there had been a large influx of evacuees into Derbyshire and there was a great increase in births. Illegitimacy had increased from 4.2% of live births in 1939 to 9.7% in 1944. Adoption work and social welfare committees were set up. Extra milk and vitamins were supplied in line with national policy. Wartime day nurseries were very busy.

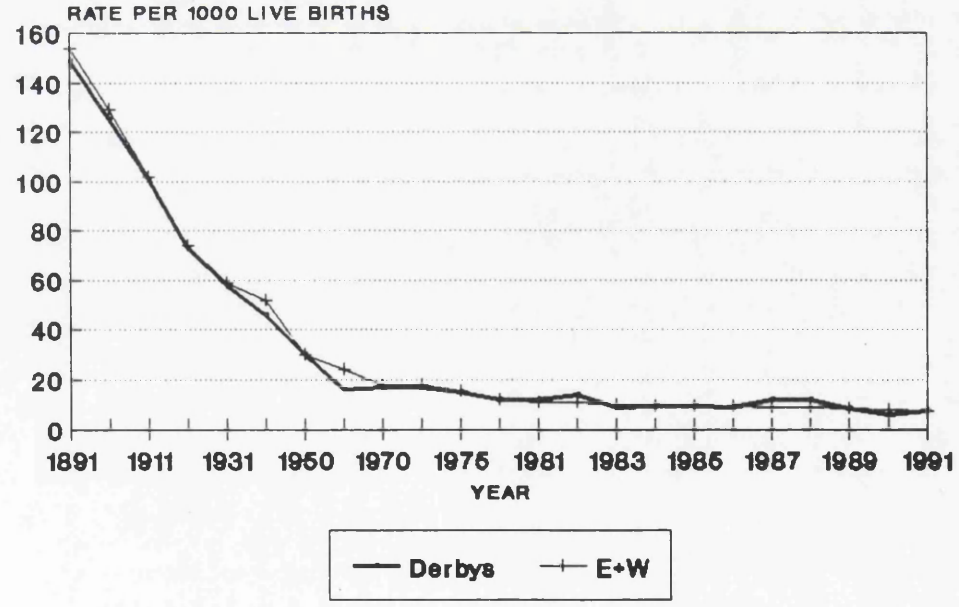
During the years of the Second World War premature birth, congenital anomalies and birth injuries were the commonest causes of death, followed closely by deaths ascribed to pneumonia or diarrhoea. From 1900 to the early 1970s, in line with the national experience, there had been an 88% reduction in the infant mortality rates which was attributed to general improvements in the environment and advances in obstetric care. In 1972 the infant mortality rates in England and Wales and Derbyshire were equal at 17 per thousand live births, but in the late 1970's the local infant mortality rates increased above the national averages (Figure 4.3, p33). In 1978 Derbyshire was instructed by the Regional Medical Officer to undertake the first of the local major investigations into infant mortality. This study led to the eventual development of investigation by detailed confidential case review.

LIVE BIRTH RATES PER 1000 POPULATION
E&W and DERBYS



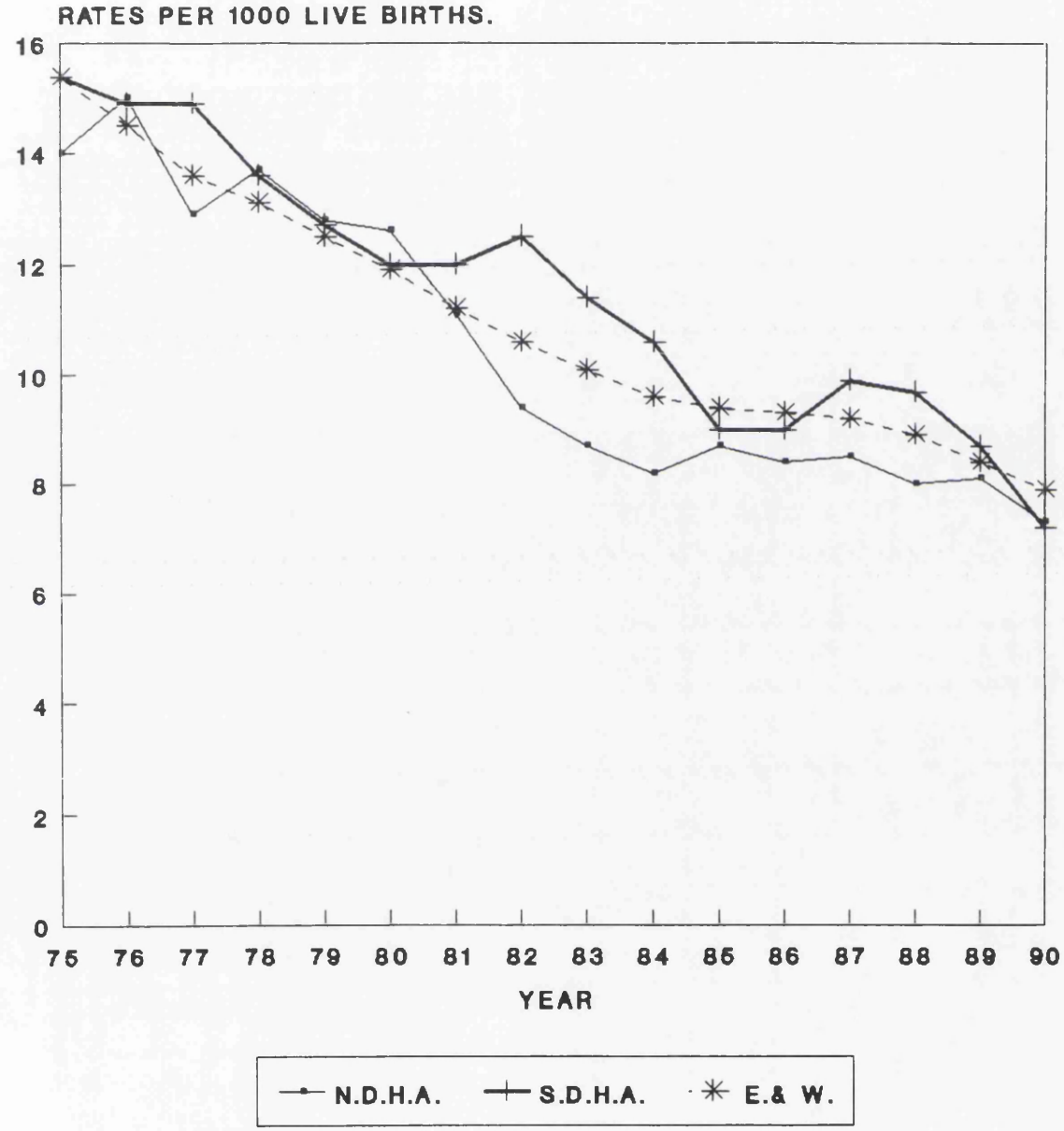
Source Reg.Gen.Statistical Review.
Fig 4.1

INFANT MORTALITY RATES E&W AND DERBYS
1891 - 1991



Source: OPCS VS1 Mortality Statistics
Fig 4.2

INFANT MORTALITY RATES.
Comparison between Nth and Sth Derbys and E&W.



3 year rolling averages.
Source: OPCS VS1, SD25.
Fig 4.3

Section 5

THE DERBYSHIRE AREA WORKING PARTY 1978

In August 1978 the Chairman of the Trent Regional Health Authority was requested by the Chief Medical Officer[15] to report on what steps were being taken within his Area Health Authorities to reduce infant mortality. Derbyshire was asked to set up a working group because the 1976 and 1977 area infant mortality rates had risen while the regional and national averages had fallen (Figure 5.1, p38).

The objectives of this group were to analyse the trends in infant mortality in the area, review record keeping systems, investigate hospital and community service provision and to make recommendations for improvement in these areas.

They set about their task by asking three questions:

1. How many of these deaths were avoidable?
2. How could prevention of these deaths be achieved?
3. At what cost would prevention be achieved?

It will be noted later in this section that none of these questions were answered.

The Study

The group obtained data for National, Regional and Local vital statistics from the Office of Population Censuses and Surveys (OPCS) forms SD.52, VS.1 and SD.25. They then identified those children resident in the district who had died during the post perinatal period (one week - one year) during the years 1976, 1977 and 1978. The registered causes of death were categorised by the International Coding of Disease (ICD 8) system.

General Practitioners allowed the investigators access to their records (EC6) if they were still available. These records also incorporated some information from the District Midwives. Further information was extracted from the Hospital Obstetric Records and the Health Visitors' Child Health Records. The data was tabulated for analysis. The deaths were then grouped into categories of avoidability after birth, in an effort to identify circumstances which could be improved for the future.

The results are depicted in Figures 5.1, p38 & 5.2, p39.

A total of 190 post perinatal deaths occurred in the area 1976-1978. In 69 cases there was insufficient or no information to analyse (n = 121). Seventy eight of the 121 cases were categorised as "unavoidable" as the registered cause of death had been congenital malformation or perinatal causes. There was no further analysis of this group. The remaining 43 deaths were analysed further by geographical location, age at death, obstetric details and social conditions. These deaths were categorised into the "avoidable group". In 14 of the 43 "avoidable" deaths no avoidable factors were determined. There remained a group of 29 deaths from a total of 190 where any avoidable factors were identified, only five of which had definitely avoidable factors.

Thirty six per cent of the children were excluded from the study. Sixty four per cent of those included in the study had not been investigated beyond death registration cause, leaving a total of 23% of the original sample to be investigated.

Conclusions of the Working Party[2,3,37,45,46,47,48,49,50,51,52,53,54,55,56,57,58,59,60,61,62,63,64,65,66]

By using data from the "avoidable death" group the Working Party concluded that parental social factors and faults in service provision had been factors which had influenced the babies' deaths. A series of recommendations were made upon these conclusions.

Recommendations of the Working Party

1. **Health Visitors:** The area did not have sufficient Health Visitors to meet the needs of families with young children.

   The Working Party concluded that extra support was needed for some families, but that a minimum pattern of health visiting should be established for all families.

2. In 1974 there were two Consultant Paediatricians and one Senior House Officer. The Working Party took the opportunity to say that this level of staffing was insufficient.

3. **Social Work** integration with medical services was unsatisfactory and the quality and nature of social work involvement was uncertain. There were no formal criteria for referral to Social Workers in the antenatal or maternity units where problems should have been identified and dealt with.

4. **Health Education:** The Health Education programme was haphazard. The public were not being educated in the importance of early antenatal attendance. They were unaware of the dangers of hyperthermia in young babies and the importance of good diet. The teaching of child care and related subjects in schools was not universal and it was speculated that most children leaving schools were not informed, either about good practice during pregnancy or how to care for young children.

5. **Documentation:** The study had identified inadequacies in record keeping and lack of availability of many records. These details had seriously limited the scope of the study.

6. **It was concluded** that the study should not continue in its present form but that all infant deaths should be reviewed as part of routine childhood services and the appropriate information collated annually.

Criticisms of the Study

When 95% confidence intervals were subsequently calculated for the infant mortality rates in Derbyshire Area Health Authority 1976-78, there was no significant difference between the local and national rates (Figure 5.1, p38).

The study had included 190 post perinatal deaths the group which contained some degree of "avoidability" was reduced to 43 deaths. In 14 deaths in this 'avoidable' grouping no avoidable factors were found. The conclusions of the Working Party were drawn on the findings from the deaths of 29 children, only 15% of the total sample. No-one is able to say why and in what circumstances 36% of children died and in a further 41% only death registrations were available and no further background information. The classification of avoidable factors was crude. There was no definition of parental social factors and service failure. Data collection had posed a major problem. Health Visitor records were variable in quality. General Practitioner records were incomplete.

It is easy to criticise the study in retrospect but the Working Party could not have made such a comprehensive list of recommendations from the data available. The study revealed failure to register and implement the Court Committee's Report[3], but the recommendations provided a useful basis for improving services despite the failure to obtain a critical data collection.

In mitigation, this working group was the first within the area to promote a multidisciplinary approach in the investigation of childhood death. They had recognised that the death of an infant should be investigated, not solely in medical or legal isolation, but also by factors in the child's environment. There was little evidence of interdisciplinary work or counselling of the bereaved and no reference to any effort made to help parents with the care of their next child.

There were no pointers as to who should have the responsibility for further investigation of child death or what information should be collected and analysed. There was no record of the financial implications of the recommendations. Most of the recommendations from the study were implemented over the following four years. These were in line with the recommendations made by the Parliamentary Committee chaired by Mrs. Renée Short (1980)[66].

Preparation for the further reorganisation of the area into two District Health Authorities in 1982 had distracted senior workers' interests from their day to day work into re-applying for their own jobs and coping with the stress of management change. No-one had been assigned to the monitoring of infant mortality and in 1982, the infant mortality rates were again higher than national averages (Figure 4.3, p33).

None of the three original questions from the study had been answered necessitating a more detailed study which was undertaken in 1983/84.

COMPARATIVE INFANT MORTALITY RATES RELATED TO SOUTH,

CENTRAL AND NORTH DERBYSHIRE DISTRICTS

	1974	1975	1976	1977	1978	1979
England and Wales	16	16	14.2	13.8	13.2	12.8
Trent Region	15.5	15.6	14.7	13.9	13.2	12.5
Derbyshire Area Health Authority	15.3	14.6	15.3	15.6	13.9	11.9
South District	16	14	19	14.9	16	14
Central District	16	15	12.1	14.1	14.6	9.6
North District	13	14	14.3	16.6	13.4	11.9

DERBYSHIRE AREA HEALTH AUTHORITY. INFANT MORTALITY RATES (per 1000 Live Births)

1976 15.3 (95% ci 2.9, 29.7)

1977 15.6 (95% ci 0.4, 30.8)

1978 13.9 (95% ci 0.5, 27.3)

Fig 5.1

DEATHS GROUPED BY CATEGORY OF AVOIDABILITY 1976 1978

1978 AREA WORKING PARTY DERBYSHIRE AREA HEALTH AUTHORITY

TOTAL NUMBER % POST-PERINATAL DEATHS	INADEQUATE INFORMATION	NUMBER OF DEATHS INVESTIGATED	NUMBER OF UNAVOIDABLE DEATHS	NUMBER OF AVOIDABLE DEATHS	FACTORS * POSSIBLY AVOIDABLE	PROBABLY AVOIDABLE	DEFINITELY AVOIDABLE	NO AVOIDABLE FACTORS
190	69 (36)	121	78	43	16	8	5	14
	Excluded from study		Congenital malformation = 64 + Perinatal causes = 14	FURTHER ANALYSIS AVOIDABLE FACTORS				
			No further investigation	BIRTH INJURY = 1	1	0	0	0
				SERVICE FAILURE = 23	1	1	1	0
				PARENTAL SOCIAL FACTORS = 23	12	7	4	0
				INDICATION OF CHILD ABUSE = 2	2	0	0	0
				Total = 29				

* only 1 avoidable factor had been associated with a case
There was no indication where there may have been more than 1 avoidable factor

Figure 5.2

Section 6

THE TRENT CONFIDENTIAL ENQUIRY INTO PERINATAL AND INFANT MORTALITY 1983-1984

Introduction

In 1982 following the report of the Parliamentary Committee, chaired by Renée Short (1980)[66], Trent Regional Health Authority set up a Regional Perinatal and Infant Mortality Review Committee under the chairmanship of one of the Regional Community Physicians. For the first time a structured study was set up in 1983 comprising investigation into all deaths from 28 weeks gestation to one year after birth by confidential enquiry using age matched live children as controls. The main objectives were:

1. To identify the possible areas of deficiency.
2. To identify points requiring further study.

Methods

The study took place from December 1983 to November 1984 inclusive. A register of all post perinatal deaths was compiled in the Department of Child Health and the 10th next live birth notified to the District was used as an age matched control to the baby who had died. Community and hospital data was collected for each child included in the study. A post mortem examination summary report was completed by the Pathologist if permission had been given by the parents. An assessment conference was held for each child, arranged in a local hospital. The assessors were the study coordinator, the Community Physician and a senior midwife who had also been involved. The family Health Visitor and midwife were invited as appropriate. The deaths were then grouped into categories of preventability taking into account the registered cause of death. The presence or absence of any failure on behalf of the medical/nursing profession was noted, and on behalf of the parents, factors thought to have contributed to the cause of death. The presence or absence of social contributory causes was considered as well as the completeness or otherwise of the information obtained[16].

Results

The results are depicted in Figures 6.1, 6.2, p43 & 6.3, p44.

The infant mortality rate fell during the period of study, the only other occasion of decline being during the investigation of the Area Working Party in 1978 (Figure 4.3,

p33). During the 12 months of the study there were 6,659 births and 33 post perinatal deaths (rate 4.9 per 1,000 live births). Twenty three of these were grouped into a possibly preventable category (Figures 6.1 and 6.2, p43). This included all 21 children registered as cot deaths (rate 2.4 per 1,000 live births). The children presenting as cot deaths were subdivided into groups indicating the presence of adverse factors that may have played a role in producing death and which, if prevented or dealt with, might conceivably have influenced the outcome (Figure 6.3, p44). Ten deaths fell into the probably not preventable after birth category (rate 1.35 per 1,000 live births), (Figure 6.1, p43). Adverse social factors were present in only three of these cases and in none were they thought to be contributory to the cause of death. This compared with the possibly preventable group of 23 deaths where adverse social factors were thought to have made a major contribution to the death in six cases (Figure 6.2, p43).

Discussion

The total number of post perinatal deaths is small in one district. Even so, the presence of adverse factors appear to have played a considerable role in the deaths of six of the possibly preventable deaths. When the control data was analysed the prevalence of adverse social factors was the same as that in the probably not preventable group of deaths. Where they were present these social irregularities were not deemed contributory to the deaths of the children. It was obvious to the conference members that the social data were insufficient to draw firm conclusions and that any psychological problems that could have been present had not been investigated in any detail. In the children registered as cot death, by far the largest group was the multifactorial group where the presence of major adverse factors was overwhelming, but there was incomplete knowledge of the psychological state of the parents and of the adverse social factors present. These frustrations pointed the way to the need for ongoing but more detailed study of all post perinatal deaths in the District.

The study as a multi-district study had been extremely difficult to organise and the lack of funding for participation prohibited the collection of full data sets in most districts. Southern Derbyshire Health Authority had managed to compile a full data set according to the requirements of the study, but there were difficulties in arranging case conferences at regular intervals, and the general practitioners were not present.

Conclusions

The main value of the study was the revelation to the researchers of the insufficiency of the social, psychological and family information[16]. Despite this limitation, the study

identified social factors which had contributed to the deaths of six children. The final report drew attention to the differences in Infant Mortality rates in Southern Derbyshire as compared to North Derbyshire (Figure 6.4, p45).

The collection of data for examination outside the District was not satisfactory, in-district analysis would have been easier. Factors vary in different areas thus local assessment is essential. This level of enquiry revealed that a more detailed study by in-depth confidential review was required and that a parallel study should be undertaken with North Derbyshire. These studies are described in Sections 11 and 12, p93 and p117.

DEATHS CATEGORISED BY POSSIBLE PREVENTABILITY AFTER BIRTH

(n=33) RATE 4.9/1000 LIVE BIRTHS

DEATHS PROBABLY NOT PREVENTABLE AFTER BIRTH (n=10) RATE 1.35/1000 LIVE BIRTHS

CAUSES	NUMBER OF CHILDREN	DEFECT IN MEDICAL SERVICE	SEVERE SOCIAL DEFECT	CONTRIBUTING TO DEATH
MALIGNANCY	0	0	0	0
DEGENERATION	1	0	0	0
METABOLIC ERROR	1	0	0	0
PERINATAL CONSEQUENCES	3	2	2	0
CONGENITAL DEFORMITY	5	0	1	0
TOTAL	**10**	**2**	**3**	**0**

Fig 6.1

DEATHS POSSIBLY PREVENTABLE AFTER BIRTH (n=23) RATE 3.45/1000 LIVE BIRTHS

CAUSES	NUMBER OF CHILDREN	DEFECT IN MEDICAL SERVICE	SEVERE SOCIAL DEFECT	CONTRIBUTING TO DEATH
MENINGOCOCCAL INFECTION	2	0	0	0
CHILDREN REGISTERED AS COT DEATHS (n=21) RATE 3.15/1000 LIVE BIRTHS				
ACUTE INFECTION	1	1	0	0
TRAUMA	2	0	2	2
NOT KNOWN/ QUERY ACCIDENTAL	2	0	2	2
SUDDEN INFANT DEATH	16	0	2	2
TOTAL	**23**	**1**	**6**	**6**

Fig 6.2

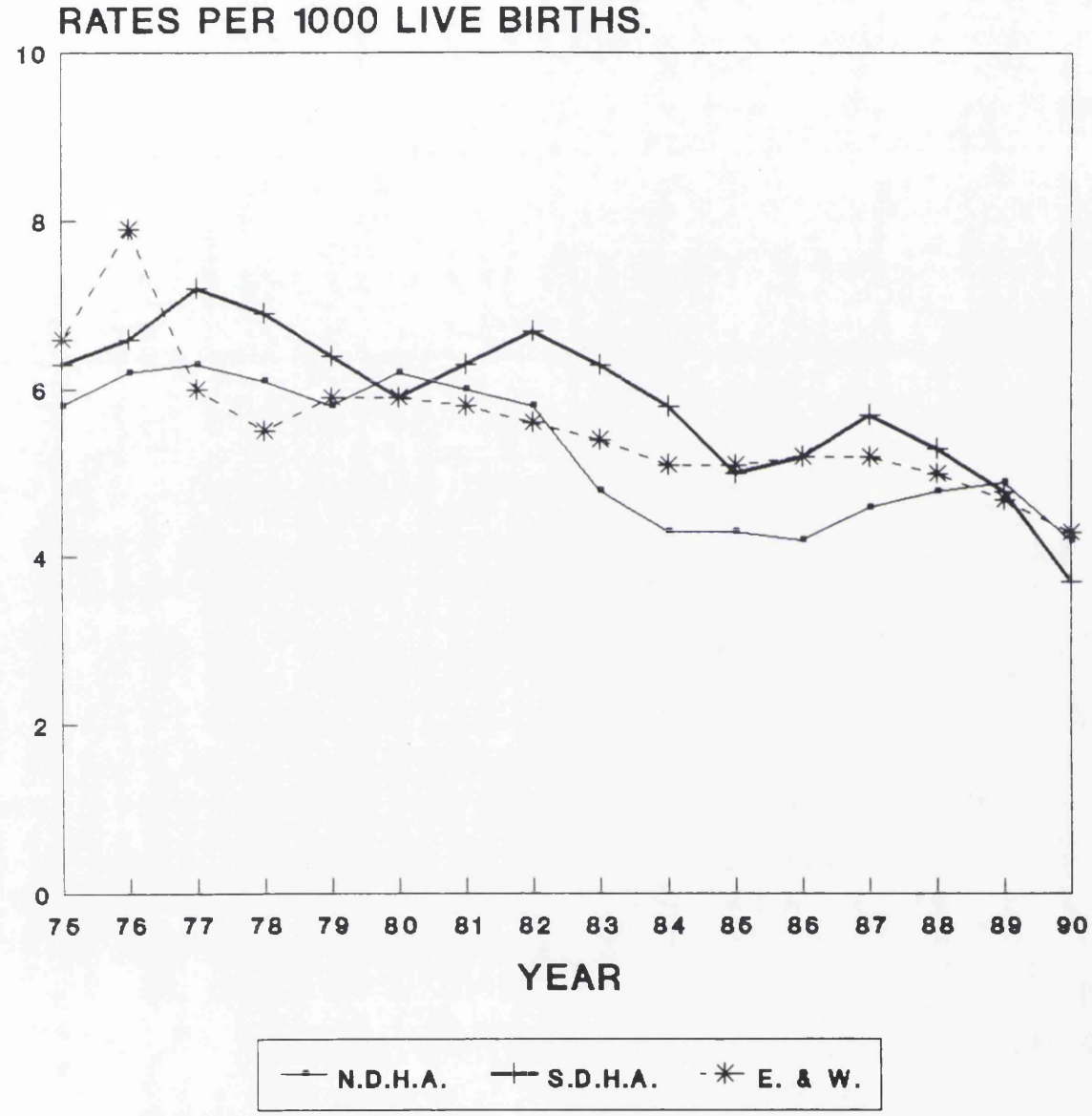
CHILDREN REGISTERED AS COT DEATHS CATEGORISED INTO GROUPS SHOWING THE PRESENCE OF SOCIAL DEFECTS WHICH MAY HAVE CONTRIBUTED TO DEATH

GROUP CATEGORY	NUMBER OF CHILDREN	PRESENCE OF MAJOR SOCIAL FACTORS	
MULTIFACTORIAL			
MOST PROMINENT FACTOR BEING:			
a) ACUTE INFECTION	2		
b) PSYCHOLOGICAL STATE OF THE PARENTS	1	9	(100%)
c) PREVIOUS PREMATURE BIRTH	1		
d) SOCIAL FACTORS	5		
POSSIBLE CHILD ABUSE	3	3	(100%)
METABOLIC DISEASE	1	0	(100%)
ACCIDENT	1	1	(100%)
NOT KNOWN	7	1	(14%)
TOTAL	**21**	**14**	**(66.6%)**

Fig 6.3

POST PERINATAL MORTALITY RATES.

Comparison between Nth and Sth Derbys and E. & W.



3 year rolling averages.
Source: OPCS VS1, SD25.
Fig 6.4

Section 7

DATA BASE AND TRENDS IN POST PERINATAL MORTALITY

Introduction

A data base of post perinatal mortality was maintained in the Department of Child Health from 1984-1988 to identify the pattern of death in the District. The data have since been updated to 1991.

Method

Each death was recorded by date and registered cause and was related to the birth data and the Community Child Health Record. The birth weights of the dead children were recorded by 500 gram bandings to ascertain how many children of low birth weight had died in the post perinatal period and the ages at death were categorised by four week age bands. The month when the child died was recorded to obtain the seasonal pattern. The deaths were plotted on a map to show the geographical distribution. The Jarman indices[19,20] of deprivation were calculated for each electoral ward and ranked by high, medium and low deprivation scores. The deaths were grouped into two categories of preventability after birth[67], using the listings of the International Code of Disease (ICD9).

Results

During the period of study there was a total of 34,412 live births and 187 post perinatal deaths (rate 5.4:1,000 live births from 1984-1988), (Figure 7.1, p55).The numbers of births increased slowly by 500 births and numbers of deaths increased annually except in 1986. The post perinatal mortality rate increased from 4.7 to 7.42 per 1,000 live births.

Season (Figure 7.2, p56)

Fifty six per cent of the deaths occurred during the winter months November to March and only 14% during the months of June, July and August.

Deaths by Age (Figure 7.3, p56)

The median age of death was 15.35 weeks. Age at death ranking showed 4-7 weeks as the commonest age of death with 40-47 weeks the least common. Seventy point five per cent of deaths occurred before the 20th week.

Deaths by Birth Weight (Figures 7.4, 7.5 & 7.6 p57)

Two thousand four hundred and fifty seven (7.1%) of the live births weighed less than 2.50 kilograms, 57 of these babies died in the post perinatal period (rate 23.2 per 1,000 low birth weight babies). One hundred and twenty babies died who had weighed more than 2.50 kilograms at birth (rate 3.75 per 1,000 births greater than 2.50 kilograms). The birth weight of eight babies who died had not been recorded. These deaths occurred within 1,000 live births. Babies of low birth weight (less than 2.50 kilograms) died at six times the rate of those weighing more than 2.50 kilograms at birth.

Deaths by Place (Figure 7.7, p58)

Fifty eight per cent of all post perinatal deaths occurred within the home setting. Twenty four per cent died whilst in hospital and 8% died in the Special Care Unit. The rest of the children were found to be dead on arrival at the Accident and Emergency Department.

Deaths by Residence (Figure 7.8, p58)

Each death was recorded by District Council, there being five District Councils in Southern Derbyshire. Derby City which contains 49% of the population contained 56% of the deaths.

Deprivation

The post perinatal death rate in the group of electoral wards with high Jarman scores of deprivation was 8.4 per 1,000 live births compared with 4.2 in the group with low deprivation scores. The proportion of low birth weight babies born was 7.22 and 6.33 using the same comparison (Figure 7.9, p59).

Deaths by Preventability (Figures 7.10 p60 and 7.11 p61)

61 (33%) of the 187 deaths were grouped in the probably not preventable category (rate 1.8:1,000 live births) and (67%) in the possibly preventable after birth group (rate 3.7:1,000 live births).

Results From Updated Data Base 1984-1991

The low increase in live births continued but the post perinatal mortality rate fell and remained below the national average from 1989 onwards (Figure 7.3, p56).

There was no shift in the distribution of low birth weight babies who died ($X^2=4.54$ $df=7$ $p=0.72$). The risk of a low birth weight baby (<2500 gm) dying in the post perinatal period relative to those babies of birth weight > 2500 gm remained high (Relative Risk = 5.19 [95% C.I. 4.00 - 6.75] $X^2=188$ $df=1$ $p<0.00001$).

In 1990, for the first time since 1984, more post perinatal deaths occurred in hospital than at home. (Figure 7.7, p58). The number of deaths in 1990 was small and the post perinatal mortality rate was the lowest recorded since 1975. The difference between the death rates in the possibly preventable and probably not preventable death groups was the smallest recorded.

Discussion

Difficulties were encountered in being informed of post perinatal deaths of the children resident in the District. The Coroners serving the five District Councils were asked to notify the Community Physician of all child deaths that came to their notice. Similarly the Police Force was asked to supply the same information. General practitioners, hospital doctors and health visitors agreed to notify the Department of Child Health of the deaths of child patients. The local Children's Hospital was contacted on a daily basis and an agreement was reached with the hospitals in Nottingham, Burton-on-Trent, Leicester and North Derbyshire to quickly inform the Community Physician of deaths of children normally resident in Southern Derbyshire Health Authority. In addition, staff at the Department of Child Health scanned the local newspaper for reports of infant deaths. Despite all these precautions it was sometimes many weeks before notification of death reached the Department of Child Health, resulting in anguish for many bereaved parents when clinical or hospital appointments were sent out in error. Despite repeated reminders difficulties still remain to this day.

The numbers and rates of post perinatal mortality were above the national average except for 1984 and 1986 but the period of study was too short to show trends.

The seasonal variability[10,67,68,69,70,71,72,73,74,75,76,77,78,79] and the average age of death were common to those found elsewhere[1,10,67,71,75,80,81,82,83]. Although there appeared to be a shift in distribution in age at death, to the younger age group, this was due to random fluctuation ($X^2=10.4$ $df=7$ $p=0.16$).

There appeared to be a shift in the distribution of deaths in the low birth weight group[1,10,84,85,86,87,88] when the numbers of deaths were high, particularly in 1988. This proved to be random fluctuation ($X^2 = 2.53$ df = 4 p = 0.64).

Deaths analysed by place were similar to findings elsewhere[1,10,67,71,89]. Place of residence at death was unremarkable in that 56% of deaths occurred in the City of Derby where 49% of the population resides. The post perinatal mortality rate was significantly higher in the areas of the County where the Jarman scores of deprivation were high[1,19,20,74,89,90], as was the proportion of low and very low birth weight babies[79,91,92]. No child of the ethnic minority group died as a cot death, although more than 80% of the ethnic minorities resided in parts of the City where the Jarman deprivation scores were high. All the babies dying in this ethnic community sub-group were of very low birth weight or had severe congenital malformations[1,10,67,69,70,74,82,92] and had been categorised into the probably not preventable after birth grouping[10,67,70,72,73,93].

In the two groups of preventability, the rates of death in the possibly preventable group were almost double those in the probably not preventable group. The numbers of deaths due to congenital malformations showed no particular trend 1984-1988 but those due to perinatal causes increased in number, perhaps due to improved care or neonatal special care units. The majority of these babies were of very low birth weight and the registered cause of death was extreme prematurity. Perinatal causes and congenital malformations were the cause of about two thirds of the probably not preventable group[69].

Deaths in the possibly preventable groups occurred at twice the rate of those deemed probably not preventable. Nearly all these babies were found dead in their cots at home and accounted for 40.5% of all the post perinatal deaths[10,67,70,72,79]. Many of these cases presented with findings at post mortem examination and in many cases there was recorded evidence of clinical disease before death[79,94,95]. In this group of preventability, infection accounted for 25% of the deaths and SIDS for a further 36%[10,67,70,72,73,93]. The consistent presence of high rates of possibly preventable deaths 1984-1988 was the reason for the examination of death registration trends for a further period of time. If this pattern of death registration had prevailed over a long term then there was much work to be done and many improvements to be made, but those registered as SIDS averaged a rate of 1.9:1,000 live births, lower than the national rate.

This data base was used as a management tool as areas of high post perinatal mortality were identified; in particular those areas where possibly preventable deaths were found. This resulted in subsequent deployment of Health Visitors to the more deprived areas.

Having established a pattern of post perinatal mortality, death registrations were investigated back to 1975 to formulate a more accurate picture of local trends. Over the fourteen year period these identified how each group of preventability had contributed to the post perinatal mortality rate[1,67,93], and showed changes in death registration within these two groups.

Local birth rates and post perinatal death rates were compared with national averages. Figures 6.4 p45 and 7.12 p62). The revised International Code of Diseases (ICD9) was used throughout, as ICD10 was not yet in general use. As described on p133, Jarman scores were used as a measure of deprivation as the possibly preventable deaths had implications for primary health care. Comparison of the census data 1971 and 1981[5,6,96,97] showed that the population profiles had changed in parallel with the national trends (Figure 3.1, p24). It was thus justifiable to compare local data with the national data base.

From 1975-1988 there were 92,510 births and 570 post perinatal deaths in Southern Derbyshire (rate 6.2 per 1,000 live births) (Figure 7.1, p55). The birth rates showed a steady rise in line with national rates (Figure 7.12, p62). Infant mortality rates showed a gradual downward trend in line with the national average[1,22,68,69,72,73,81,97,98,99,100,101,102,103,104,105,106,107,108,109,110,111,112,113,114,115,116,117,118,119,120](Figure 4.3, p33). Using three year rolling averages, the infant mortality rate only fell below the national average on two occasions between 1975 and 1988. Peaks of infant mortality occurred in 1977, 1982 and 1988.

Between 1981-1983 the infant mortality rates differed significantly from the national averages, but this gap diminished by 1986-88 when no statistical difference was demonstrated.

Infant Mortality Rates SDHA vs England & Wales 1986-1988

	England and Wales	Southern Derbyshire Health Authority
Infant mortality rates	9.3	10.2

Relative Risk = 1.08 [95% C.I. 0.95 - 1.24] $X^2 = 1.23$ Yates corrected $p = 0.27$
Source: OPCS Series VS 1.

Deaths 1986-1988

	England and Wales	Southern Derbyshire Health Authority
Perinatal death rate	4.0	4.3
Post perinatal death rate	5.2	5.7

Relative Risk = 1.01 [95% C.I. 0.86 - 1.19] $X^2 = 0.01$ Yates corrected $p = 0.93$
Source: OPCS Series VS1

When the infant mortality rates were calculated for 1989-1991 the situation had improved further. The relative risk of dying as an infant death in Southern Derbyshire compared with England and Wales was then = 0.92 [95% C.I. 0.79 - 1.07] $X^2 = 1.11$ Yates corrected $p = 0.3$.

Locally the post perinatal death rates followed the same trends as the infant mortality rates, but only fell below national averages in 1985 (Figures 4.3 p33 and 6.4 p45). Of the 570 deaths in this age group 207 (36.3%) were grouped in the probably not preventable grouping and 361 (53.7%) in the possibly preventable group (rates 2.2 and 3.9 per 1,000 live births respectively) (Figures 7.10 p60 and 7.11 p61). The rates of deaths in both groups varied annually, but the probably not preventable group gradually decreased as a proportion of all post perinatal deaths. In the possibly preventable grouping the rate of deaths showed a gradual increase (Figure 7.13, p63).

Probably Not Preventable After Birth Grouping

The rates of live births in children born with congenital malformation[103] (per 1,000 live births) were similar to the rates in England and Wales[111,121] (Figure 7.14, p64). The death rates of children dying of congenital malformation decreased by over one third[64,72,73,107], (Figure 7.15, p65) and the proportion of all post perinatal deaths ascribed to congenital malformations decreased by half (Figure 7.16, p65)[64,72,73,81,102,107,108].

The data base of 1984-1988 showed that in the majority of babies dying of perinatal causes the death certification had included extreme prematurity. These babies were of low birth weight and/or low gestational age[85,90,102,104,112]. The proportion of low birth weight babies of all live births in Southern Derbyshire showed a slight increase from 6.8% to 7.4% (Figure 7.17, p66). The proportion of very low birth weight babies (less than 1500 grams) was average to both Trent Region and England and Wales (Figure 7.18, p66).

Figure 7.19 shows that the majority of low birth weight babies weighed 2000-2499 grams. The proportion of very low birth weight babies varied very little between 1986 and 1988. Pharoah and others[64,70,72,73,107] have observed that improved neonatal care has improved small babies' chances of surviving birth and the neonatal period, only to die at a later date. Some deaths are thus dislocated from the perinatal period into the post perinatal period, decreasing perinatal mortality rates at the expense of an increase in post perinatal mortality rates. Figure 7.20 shows the increasing death rate of low birth weight babies in the post perinatal period 1978-1988. The proportion of all post perinatal deaths in the perinatal causes grouping doubled during the period of study (Figure 7.21, p68). It was noted that in 1980 there were no deaths ascribed to perinatal causes, yet there were babies of low birth weight who had died. The most likely explanation of this is that the deaths were incorrectly coded[94].

Probably Preventable After Birth Group

There was a general decrease in numbers of deaths ascribed to infection throughout the period of study. Prior to 1979, when "Sudden Infant Death Syndrome" had become a popular registerable cause of death, the general trend of deaths ascribed to infection appeared to be increasing[1,10,67,72,73,83,107,119,122]. Post 1979 this trend appeared to be reversed and the proportion of deaths ascribed to infection fell[70,94] (Figure 7.22, p68). Regression analysis and the F test of coefficient showed there was no significant trend during either period of time.

During 1975-1978, 52 post perinatal deaths (54%) were certified as due to infection. This proportion fell in 1980-1988 to 41%. When "trauma" and "not known" causes were examined over the same time spans, "not known", which had accounted for 16% of post perinatal deaths, disappeared completely after 1979. The diagnosis of trauma accounted for 27 (28%) of deaths 1975-1978 being virtually absent for three years, only to re-emerge and account for 10% of all post perinatal deaths 1980-1988 (Figure 7.23, p69). During the years 1975-1978 there were only two deaths ascribed to Sudden Infant Death Syndrome. In 1979 when the diagnosis became fashionable the numbers of deaths ascribed to SIDS rose steadily[115,116,123].

During the period 1980-1988, 54% of all post perinatal deaths were ascribed to SIDS, the proportion almost doubling during this time (Figure 7.24, p69).

Figure 7.25 shows how the changing classifications of death causes altered from 1975/78 to 1980/88[64,70,73,81,94,107].

Change in classification

	1975-1979 (%)	1980-1988 (%)
Respiratory causes	52 (53.6)	91 (41.2)
Trauma	27 (27.8)	10 (4.5)
SIDS	2 (2.1)	120 (54.3)
Not known	16 (16.5)	0 (0.0)
Total	97 (100)	221 (100)

$X^2 = 118.2$, $p < 0.00001$

Post perinatal deaths

	1975-1979	1980-1988
Respiratory causes	52	91
Trauma, Not known, SIDS	45	130

$X^2 = 4.21$, $p < 0.05$

If the distribution pattern had remained the same as in 1975-79 the expectation would have been 119 deaths ascribed to respiratory causes, 62 to trauma, 4 SIDS and 35 "not known" 1980-1988. Of the 116 extra SIDS in 1980-88, 28 came from the respiratory group, 52 from the trauma group and 35 from the not known group. Thus 25% of the respiratory group transferred to the SIDS diagnosis and 75% from trauma and not known groupings. The increase in SIDS was due to a reduction in "not known", trauma and respiratory causes.

The cot death grouping comprised those deaths ascribed to Sudden infant death syndrome, trauma and not known, but excluded those that had died suddenly and unexpectedly from infection. There were 192 cot deaths (rate 2.1 per 1,000 live births) as shown in Figure 7.11. Between 1980 and 1988 the cot death rate doubled and was above national averages on five occasions. In 1986 when the national cot death rate was at its highest[72,73,107], the rate in Southern Derbyshire was at its lowest during the 14 years of study (Figure 7.26, p70).

Concerning deprivation; Southern Derbyshire was not regarded as a deprived District, having a Jarman score of -4.22. However, there were some highly deprived wards in the District, the highest Jarman scores being within the inner City of Derby. The association between generalised deprivation and post perinatal mortality is noted in Figure 7.9.

Conclusions

In Southern Derbyshire, against the background of falling infant mortality and post perinatal mortality rates, the rates of deaths classified as possibly preventable remained the greatest proportion of all post perinatal mortalities and this included the group defined as cot deaths.

DEATHS IN SOUTHERN DERBYSHIRE

YEAR	1975	1976	1977	1978	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991	TOTAL
No of births	6349	6071	5890	6127	6604	6777	6815	6620	6696	6622	6832	6801	7172	7134	7382	7650	7558	115100
Postperinatal deaths (7 days - 1 year)	37	47	40	46	42	38	39	49	45	30	39	31	34	53	27	25	31	653
Rate (per 1000 live births	6.5	7.6	6.7	7.7	6.6	5.7	5.7	7.6	6.5	4.7	6	4.55	4.7	7.4	3.7	3.3	4.1	5.7

Future Years of Life Lost SDHA due to postperinatal mortality 1988 = 3922
This compares to FYLLs due to All Accidents (4740), Lung Cancer (3320), Cerebrovascular Disease (2400), Coronary Heart Disease (10,155).

Source: OPCS VS3 Mortality Statistics

Fig 7.1

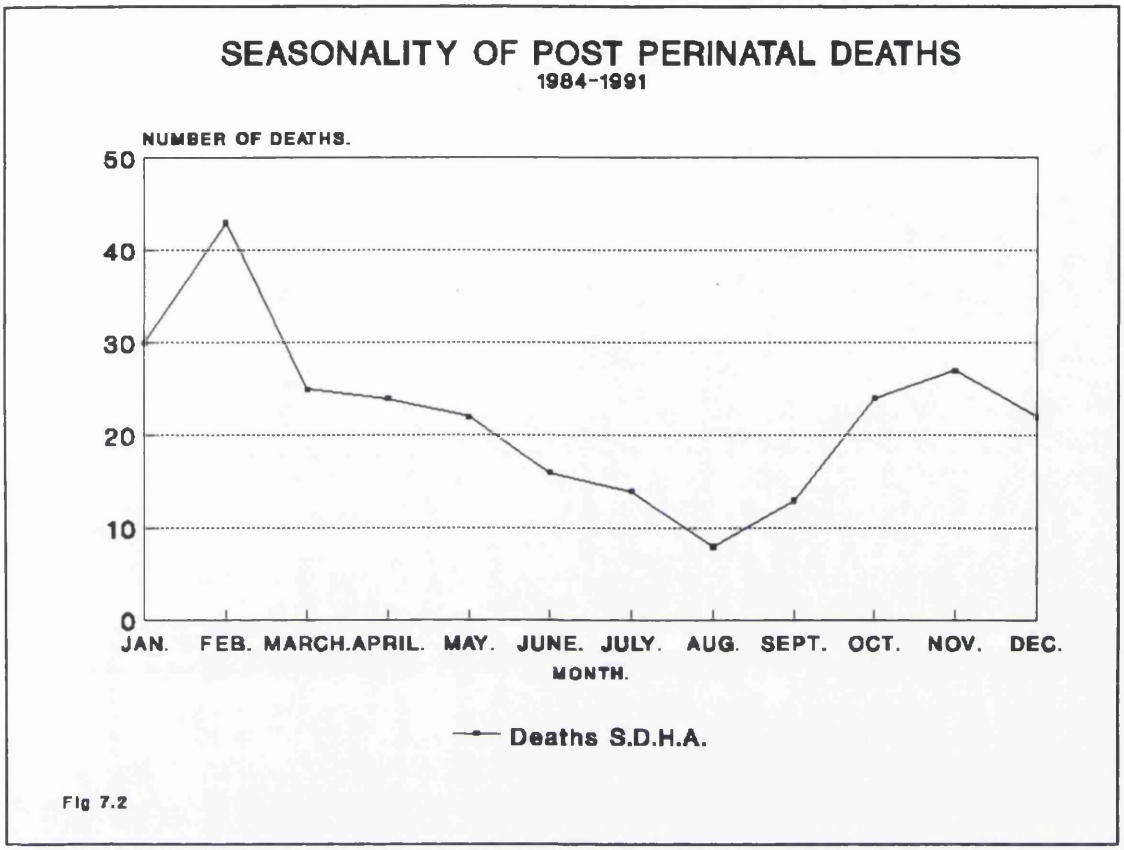


Fig 7.2

DEATHS BY AGE

DEATHS IN WEEKS	1984	1985	1986	1987	1988	1989	1990	1991	TOTAL	%
1-3 WEEKS	4	3	4	8	9	5	5	5	**43**	16
4-7 WEEKS	2	6	6	7	8	5	4	7	**45**	17
8-11 WEEKS	2	4	5	5	9	6	5	7	**43**	16
12-15 WEEKS	5	5	6	4	8	5	2	4	**39**	15
16-19 WEEKS	5	5	2	3	6	2	4	3	**30**	11
20-23 WEEKS	1	3	1	1	2	0	1	1	**10**	4
24-27 WEEKS	2	4	2	1	4	2	0	0	**15**	6
28-31 WEEKS	2	1	1	2	1	0	1	0	**8**	3
32-35 WEEKS	2	2	2	0	1	1	2	0	**10**	4
36-39 WEEKS	2	0	0	1	2	1	0	3	**9**	3
40-43 WEEKS	1	0	0	0	0	0	0	0	**1**	0.4
44-47 WEEKS	0	1	1	2	2	0	1	0	**7**	2.6
48-52 WEEKS	0	5	1	0	1	0	0	1	**8**	3
TOTAL	**28**	**39**	**31**	**34**	**53**	**27**	**25**	**31**	**268**	

Chi square = 10.4 with 7 d.f. : p = 0.1654

Although there appeared to be a shift in age at death in the younger age groups, this proved to be no more than random fluctuation.

Fig 7.3

POST PERINATAL DEATHS BY BIRTHWEIGHT

WEIGHT	1984	1985	1986	1987	1988	1989	1990	1991	TOTAL
<1000g	1	2	0	2	7	1	2	2	**17**
1000-1499g	2	1	3	1	1	2	0	3	**13**
1500-1999g	1	2	2	2	7	1	2	0	**17**
2000-2499g	2	6	5	5	5	3	4	1	**31**
2500-3000g	5	5	6	11	7	7	7	6	**54**
>3000g	16	20	13	11	26	12	10	19	**127**
N/K	1	3	2	2	0	1	0	0	**9**
TOTAL	**28**	**39**	**31**	**34**	**53**	**27**	**25**	**31**	**268**

Figure 7.4

$\chi^2 = 4.54$ with 7 df : $p = 0.72$

There appeared to be an increased trend in the numbers of LBW babies (<2.5gms) dying but this was due to random fluctuation.

NUMBERS OF LOW BIRTH WEIGHT BABIES AND BABIES WEIGHING >2500 GMS

	1984	1985	1986	1987	1988	1989	1990	1991	TOTAL
LBW Babies < 2500 gms	462	441	483	549	522	526	545	554	**4082**
Babies > 2500 gms	6160	6391	6324	6575	6540	6624	6933	6897	**52444**
Weight NK	1	3	2	2	0	1	0	0	**1000**
Total Births	6622	6832	6807	7124	7062	7150	7478	7451	**52626**

Figure 7.5

RISK OF A BABY OF LOW BIRTH WEIGHT (< 2500 GMS) DYING IN THE POST PERINATAL PERIOD

RELATIVE TO THOSE BABIES OF BIRTH WEIGHT > 2500 GMS

	NUMBER OF BABIES DIED	NUMBER OF BABIES BORN	POST PERINATAL MORTALITY RATE
LBW Babies < 2500 gms	78	4082	19.1
Babies > 2500 gms	190	52444	3.6

Figure 7.6

Risk of dying as LBW (< 2500 gms) baby relative to > 2500 gm baby is 5.19 (C.I. 4.00, 6.75)

$\chi^2 = 188$, df = 1 $p < 0.00001$

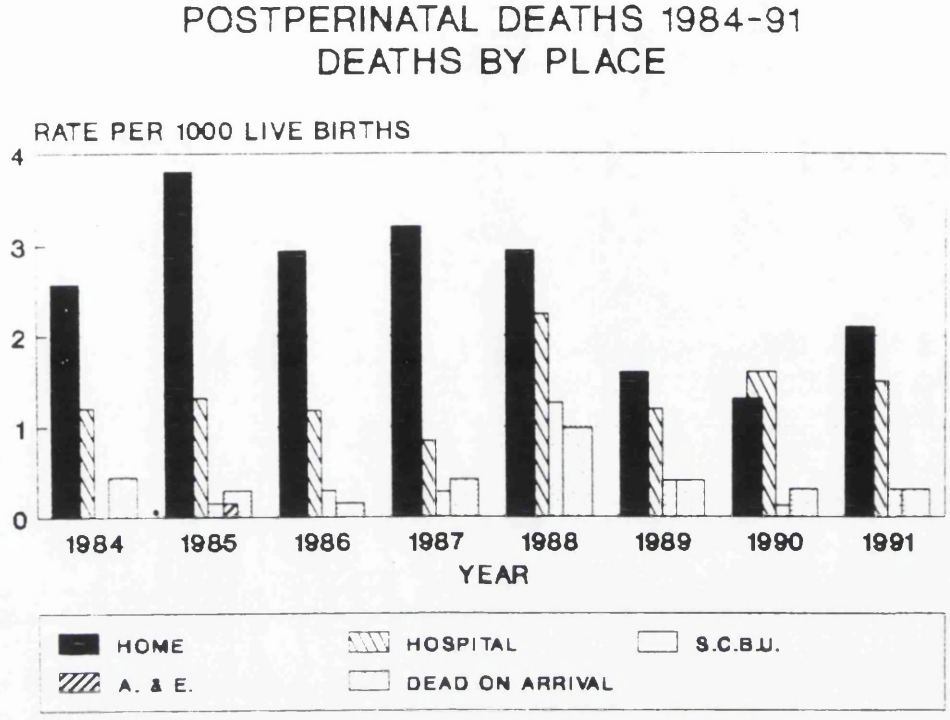

POSTPERINATAL DEATHS 1984-91
DEATHS BY PLACE
RATE PER 1000 LIVE BIRTHS
4
3
2
1
0
1984
1985
1986
1987
1988
1989
1990
1991
YEAR
HOME
HOSPITAL
S.C.B.U.
A. & E.
DEAD ON ARRIVAL

Fig 7.7

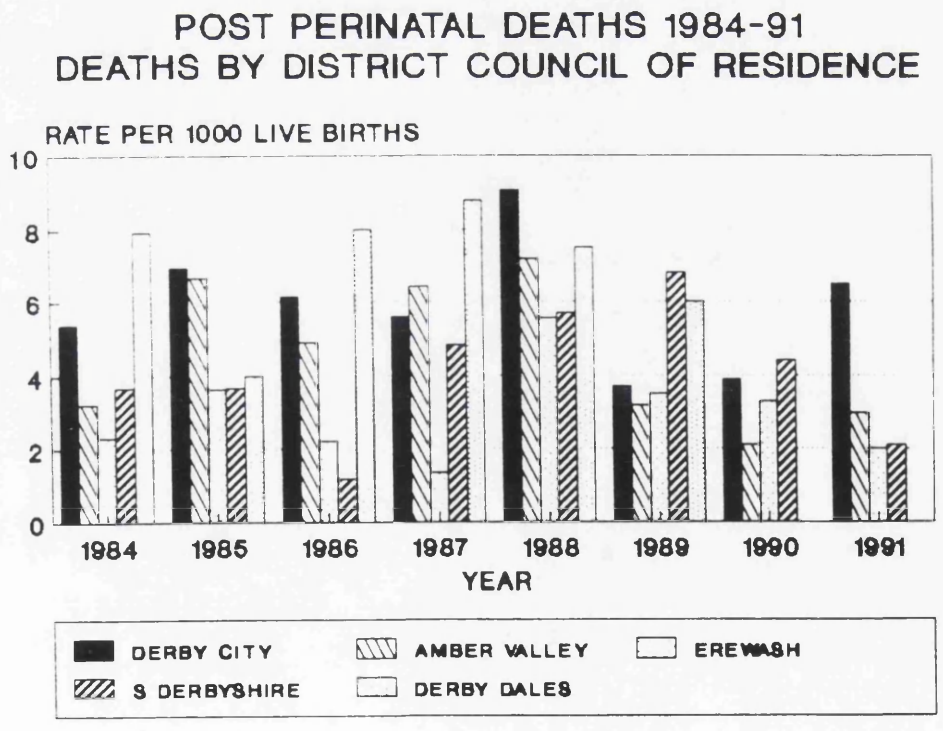
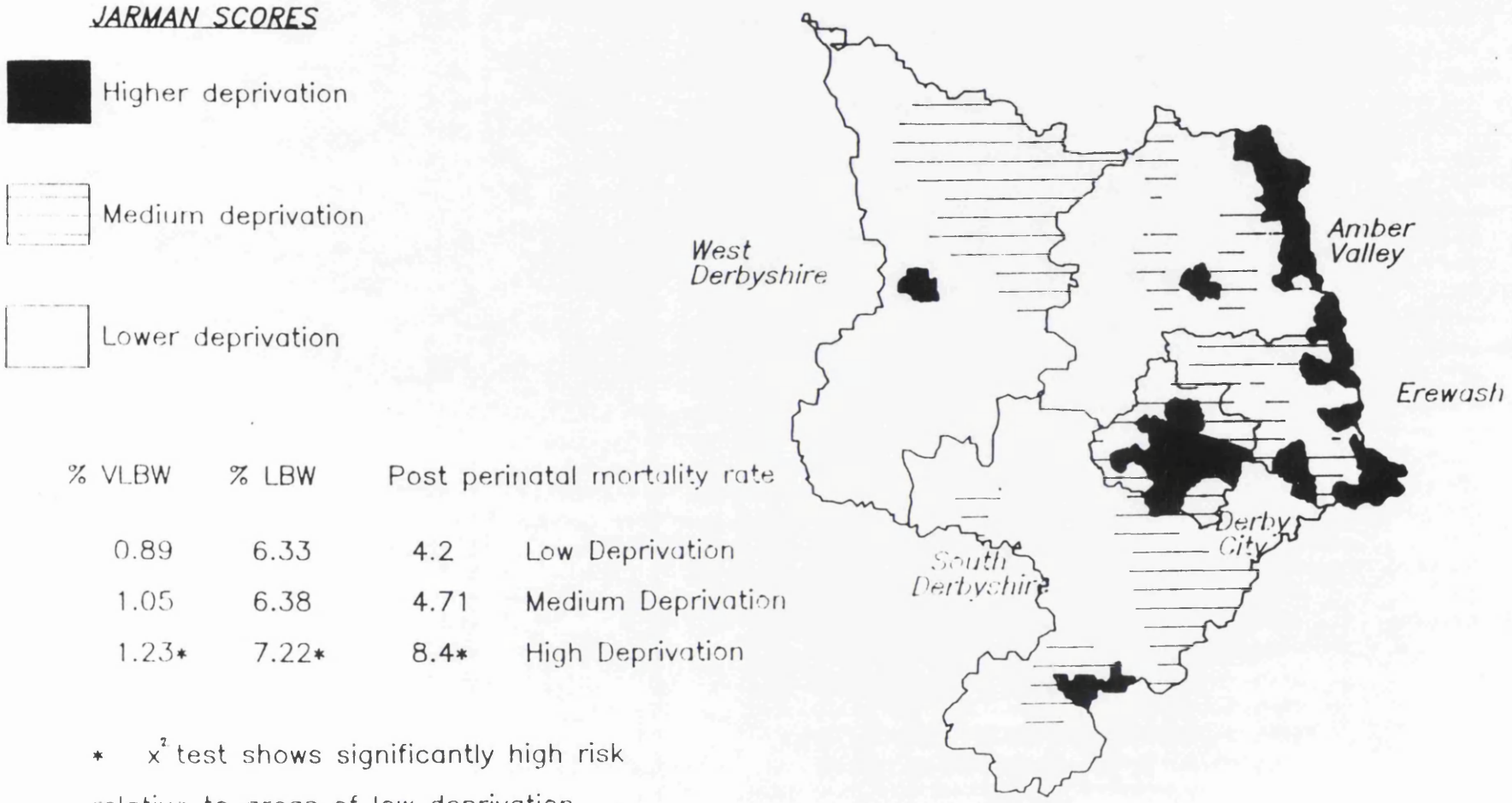

POST PERINATAL DEATHS 1984-91
DEATHS BY DISTRICT COUNCIL OF RESIDENCE
RATE PER 1000 LIVE BIRTHS
10
8
6
4
2
0
1984
1985
1986
1987
1988
1989
1990
1991
YEAR
DERBY CITY
AMBER VALLEY
EREWASH
S DERBYSHIRE
DERBY DALES

Fig 7.8

AREAS OF HIGHER, MEDIUM AND LOWER SOCIAL DEPRIVATION

SDHA 1981–1988



% VLBW	% LBW	Post perinatal mortality rate	
0.89	6.33	4.2	Low Deprivation
1.05	6.38	4.71	Medium Deprivation
1.23*	7.22*	8.4*	High Deprivation

* x^2 test shows significantly high risk relative to areas of low deprivation

Figure 7.9

DEATHS PROBABLY NOT PREVENTABLE AFTER BIRTH

	1975	1976	1977	1978	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991	TOTAL
Malignancy ICD Code 140-239.9	2	0	0	0	1	0	0	2	0	0	0	0	0	1	0	0	0	6
Degenerative ICD Code 581	1	0	0	0	0	0	0	0	0	0	0	0	1	1	2	1	0	6
Cerebral ICD Code 345-348	0	1	2	0	0	0	0	0	0	0	0	1	0	0	0	0	0	4
Metabolic Error ICD Code 269-273 and 584	1	2	0	0	1	0	0	0	0	1	1	0	0	0	0	0	1	7
Intestinal Surgical ICD Code 530-560	1	1	1	0	0	0	1	1	1	0	1	0	0	0	0	0	0	7
Perinatal ICD Code 760-779.9	3	3	2	3	1	0	3	4	8	3	2	5	3	9	3	3	2	57
Congenital deformity ICD Code 740-759.9	7	15	13	11	7	10	14	8	9	4	6	7	4	7	5	6	7	140
Inevitable(mixed) ICD Code 335-343 and 425	0	0	0	0	0	2	2	4	0	0	1	0	0	3	0	0	0	12
TOTAL	**15**	**22**	**18**	**14**	**10**	**12**	**20**	**19**	**18**	**8**	**11**	**13**	**8**	**21**	**10**	**10**	**10**	**239**
RATE	**2.6**	**3.5**	**3**	**2.3**	**1.5**	**1.5**	**2.9**	**2.8**	**2.7**	**1.2**	**1.6**	**1.8**	**1.1**	**2.9**	**1.4**	**1.3**	**1.3**	**2.1**

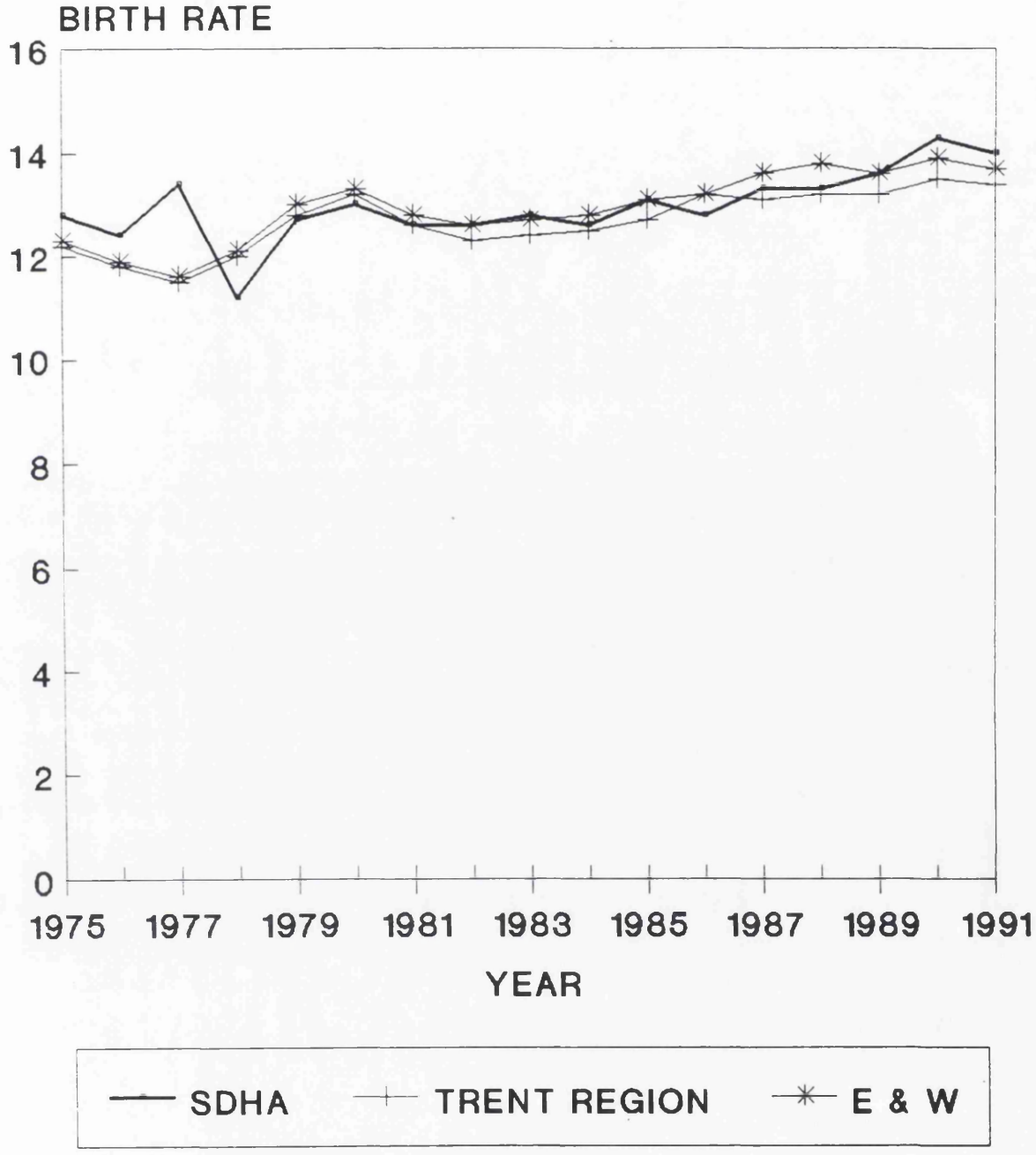
Fig 7.10

DEATHS POSSIBLY PREVENTABLE AFTER BIRTH

	1975	1976	1977	1978	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991	TOTAL
Infection(Resp & Intestinal) ICD Code 1-139.8 and 464-519	13	13	8	18	15	15	10	11	9	11	11	11	2	11	8	8	3	**177**
Trauma ICD Code 819-913	5	6	10	6	4	0	1	0	1	1	2	2	1	2	1	2	1	**45**
Poisons ICD Code 968-988	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	**1**
Not Known ICD Code 788-795	1	3	4	8	0	0	0	0	0	0	0	0	0	0	0	0	0	**16**
SIDS ICD Code 798	1	1	0	0	13	11	8	19	15	9	15	4	21	18	6	5	16	**162**
Meningoccocal ICD Code 320-322	2	2	0	0	0	0	0	0	2	0	0	1	2	1	2	0	1	**13**
TOTAL	**22**	**25**	**22**	**32**	**32**	**26**	**19**	**30**	**27**	**22**	**28**	**18**	**26**	**32**	**17**	**15**	**21**	**414**
RATE (per 1000 live births)	3.8	4	3	5.3	5	3.2	2.7	4.4	4	3.3	4	2	3.6	4.5	2.3	2.0	2.8	**3.6**
CAUSES: SIDS & TRAUMA & NOT KNOWN	7	10	14	14	17	11	9	19	16	11	17	6	22	20	7	7	17	**224**
RATE (per 1000 live births)	**1.2**	**1.6**	**2.3**	**2.5**	**2.7**	**1.6**	**1.3**	**2.8**	**2.4**	**1.65**	**2.5**	**0.9**	**3.1**	**2.8**	**1.0**	**1.0**	**2.2**	**2.0**

Fig 7.11

COMPARATIVE TRENDS IN NATIONAL, REGIONAL AND DISTRICT LIVE BIRTH RATES



Source: VS1, SD25 OPCS
Fig 7.12

POST PERINATAL DEATH RATES IN SOUTHERN DERBYSHIRE.

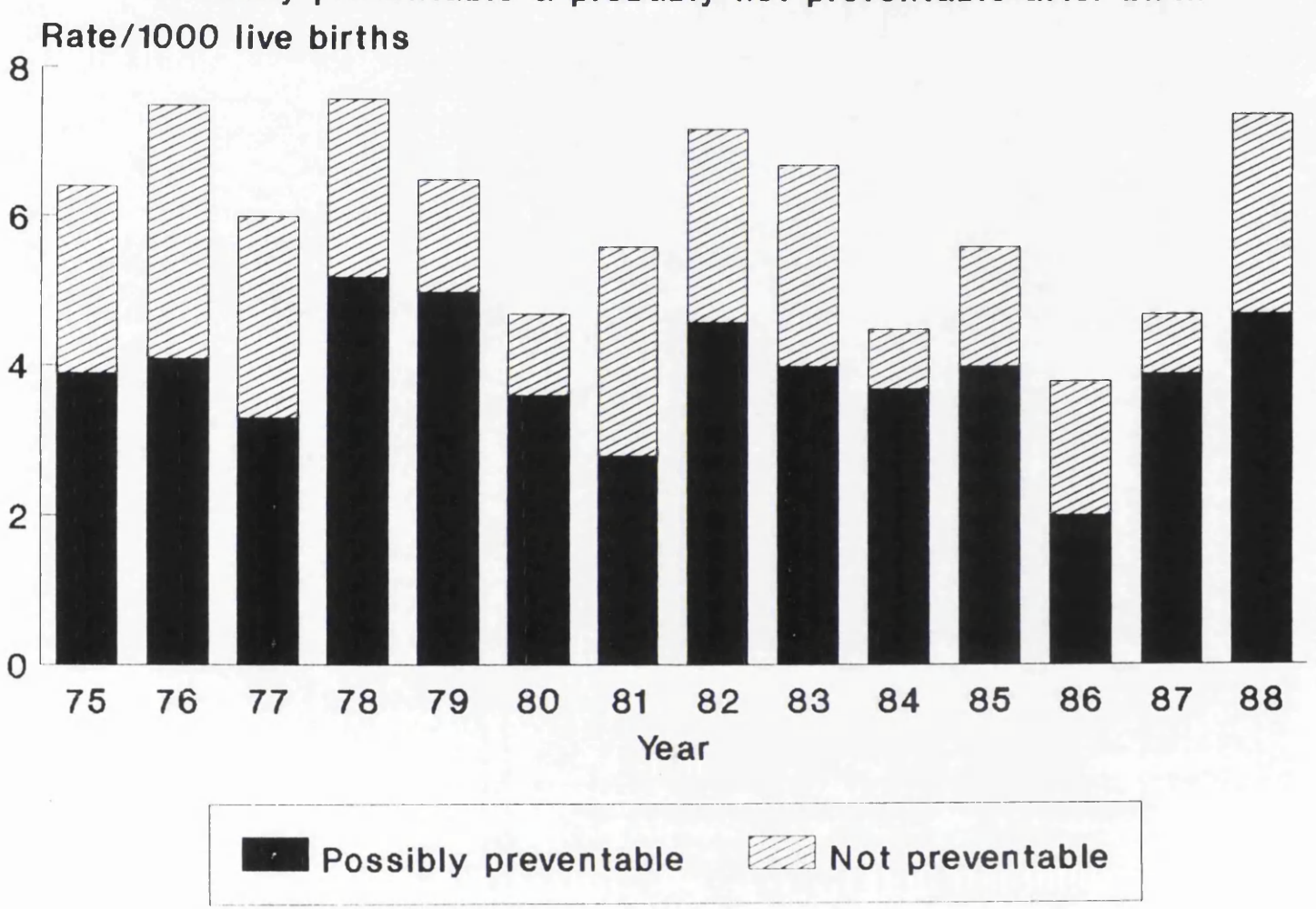
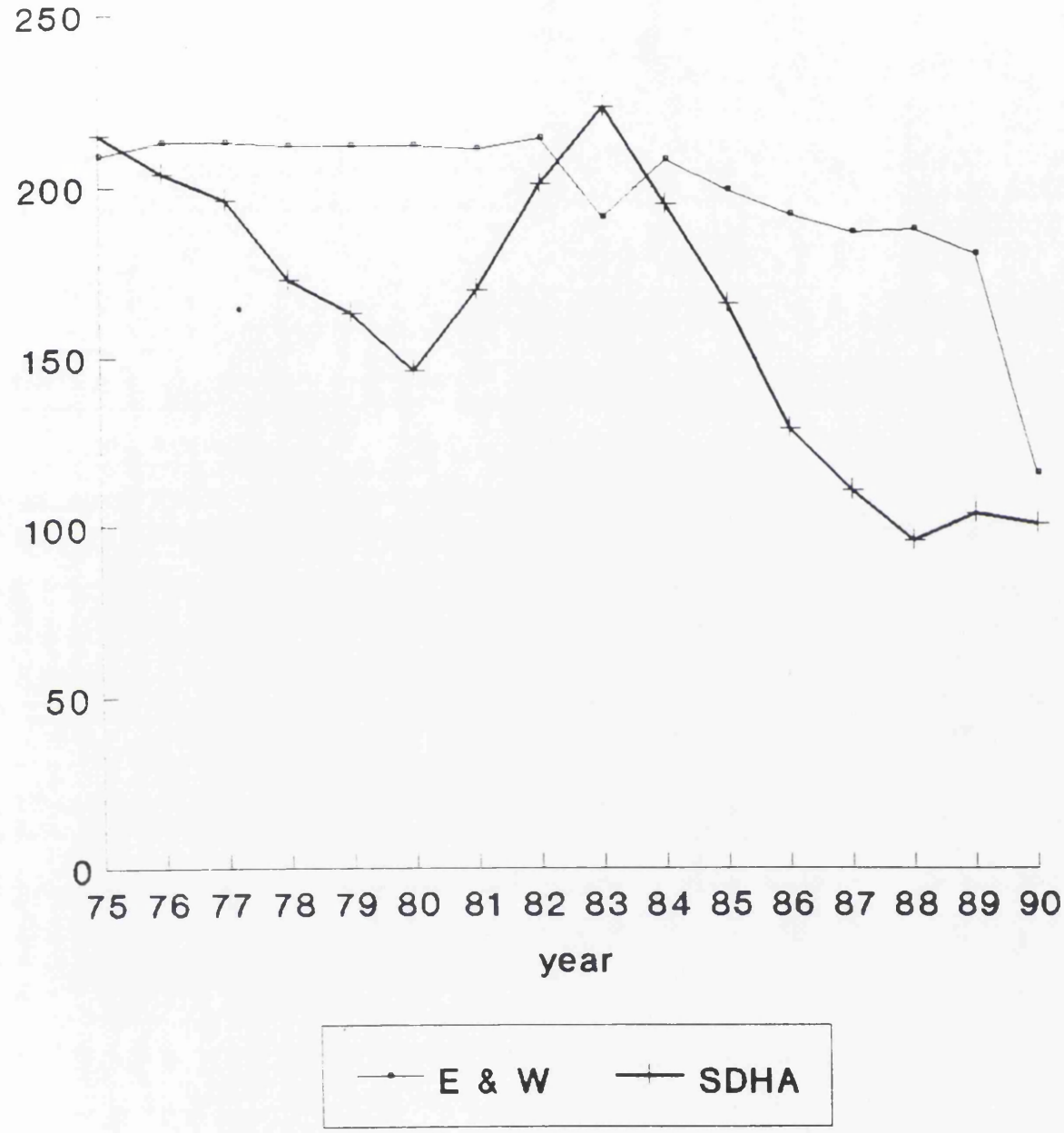


Fig 7.13

Rate of Congenital Malformations per 10000 Live Births, National and District



Source: MB3 OPCS
Three year rolling averages
Fig 7.14

RATES OF POST PERINATAL MORTALITY
ASCRIBED TO CONGENITAL MALFORMATIONS

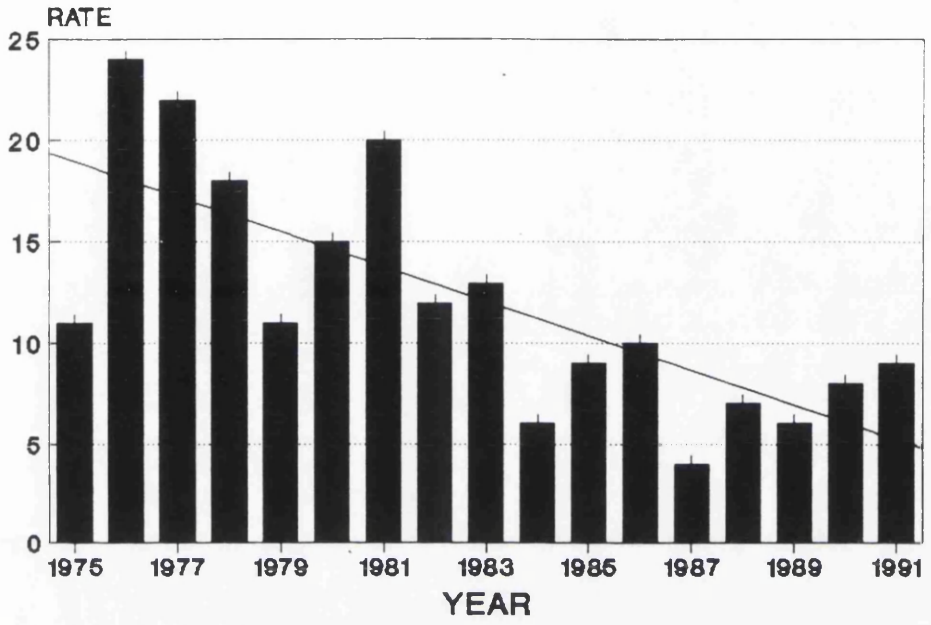


Fig 7.15

PERCENTAGE OF POST PERINATAL DEATHS
ASCRIBED TO CONGENITAL MALFORMATIONS

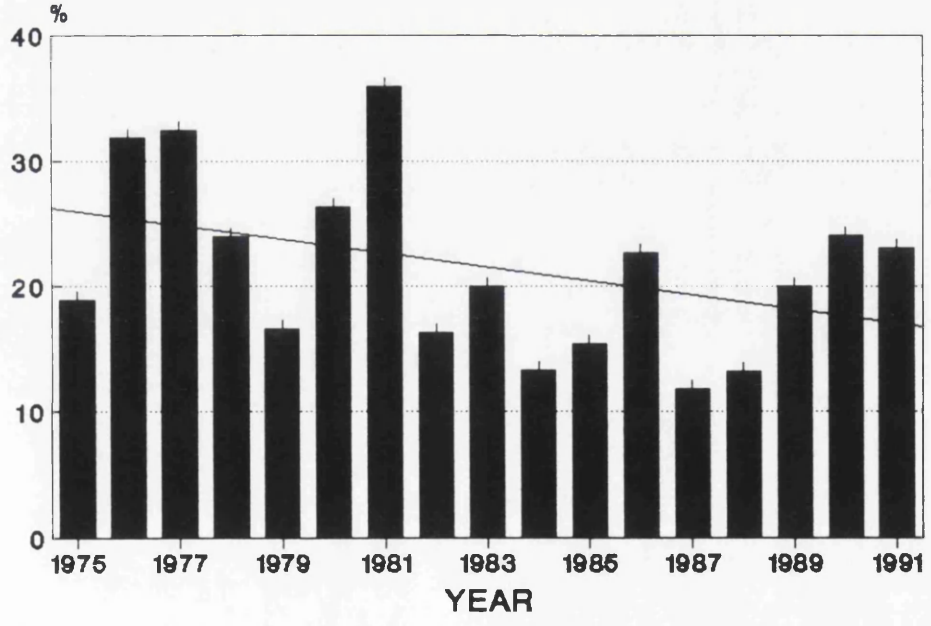
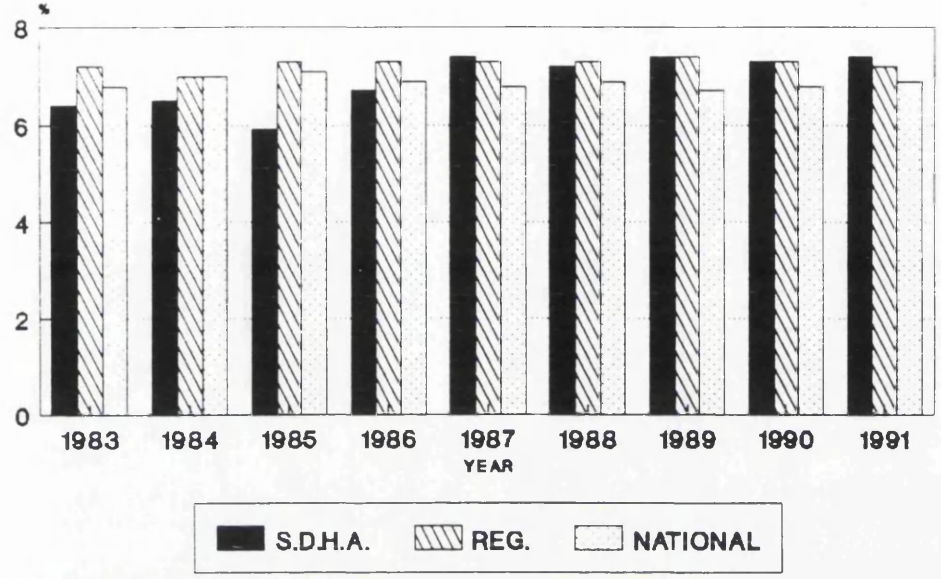


Fig 7.16

PROPORTION OF LBW BABIES AS A PERCENTAGE
OF ALL LIVE BIRTHS COMPARING SDHA,
REGION AND ENGLAND & WALES



Source: VS2 OPCS
Fig 7.17

LOW BIRTH WEIGHT BABIES 1987
AS A PERCENTAGE OF ALL LIVE BIRTHS

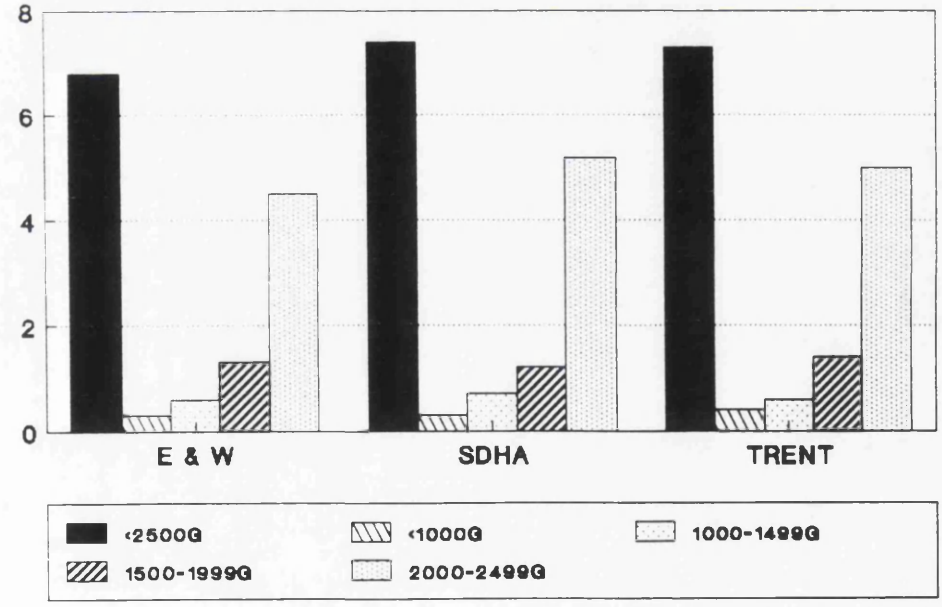


Fig 7.18

PROPORTION OF LOW BIRTH WEIGHT & VERY LOW BIRTH WEIGHT BABIES IN SDHA 1986-1991

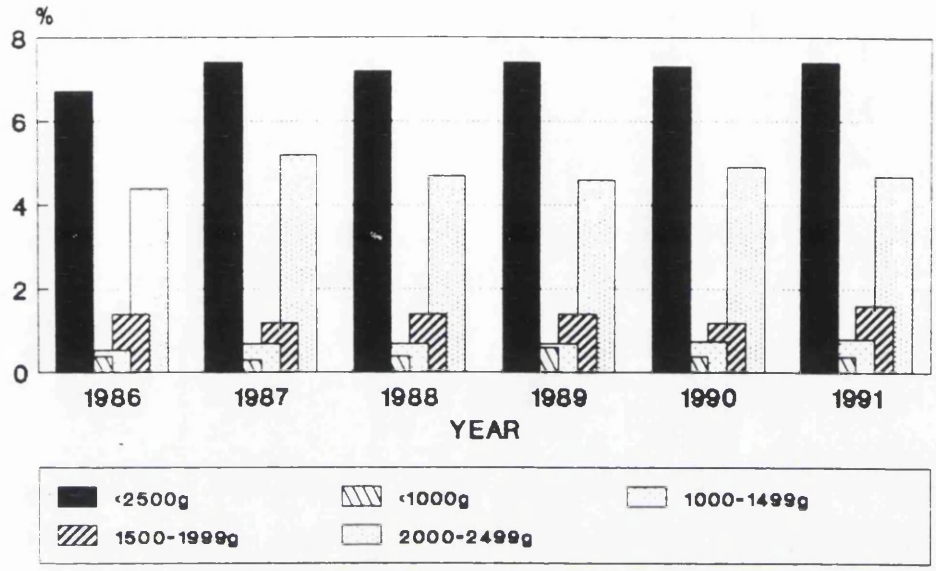


Fig 7.19

POST PERINATAL DEATHS BY CAUSE
SDHA 1979-1991
LOW BIRTH WEIGHT BABIES

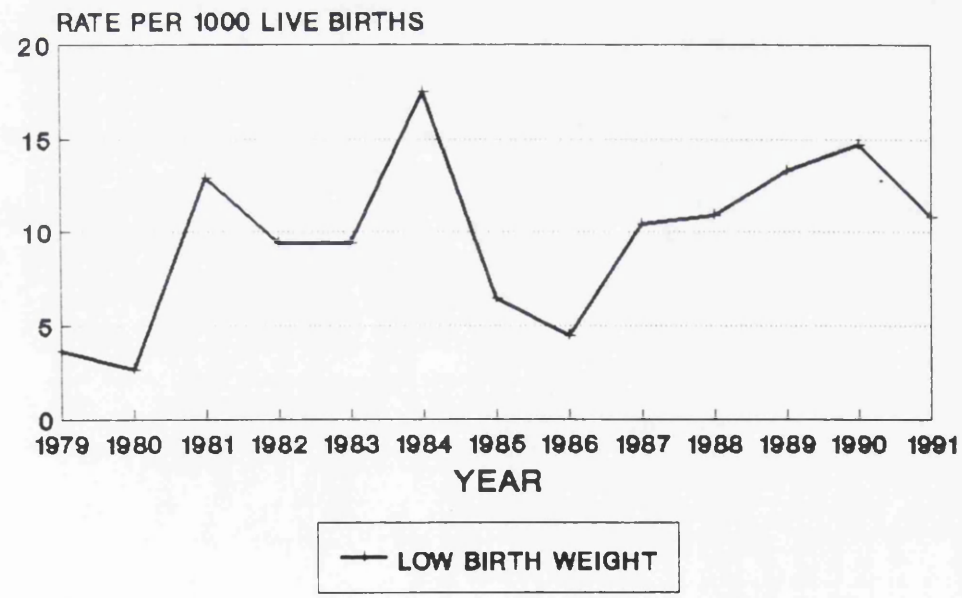


Fig 7.20

PROPORTION OF POST PERINATAL DEATHS
ASCRIBED TO PERINATAL CAUSES
SDHA 1975-91

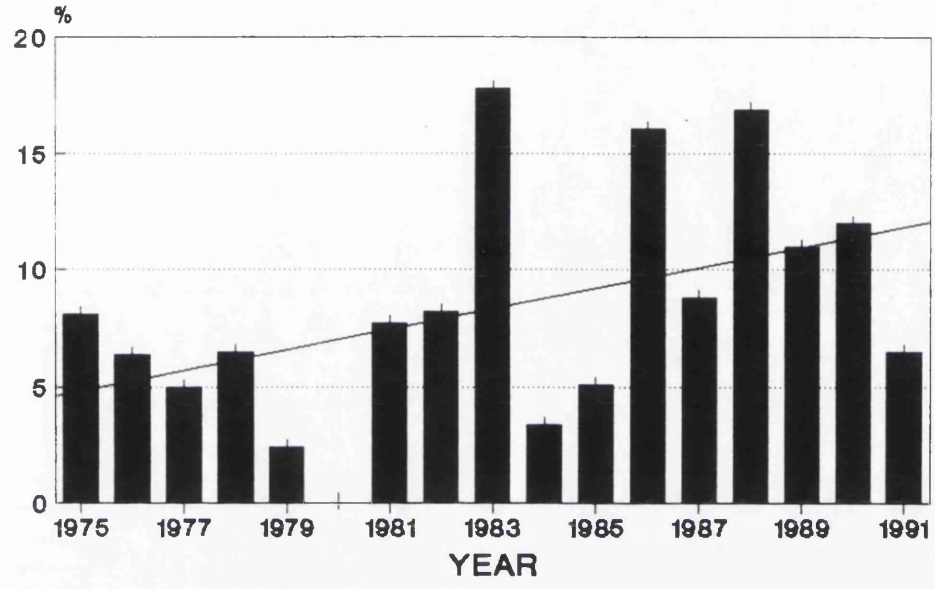


Fig 7.21

PROPORTION OF POST PERINATAL DEATHS
ASCRIBED TO RESPIRATORY & INTESTINAL
INFECTION

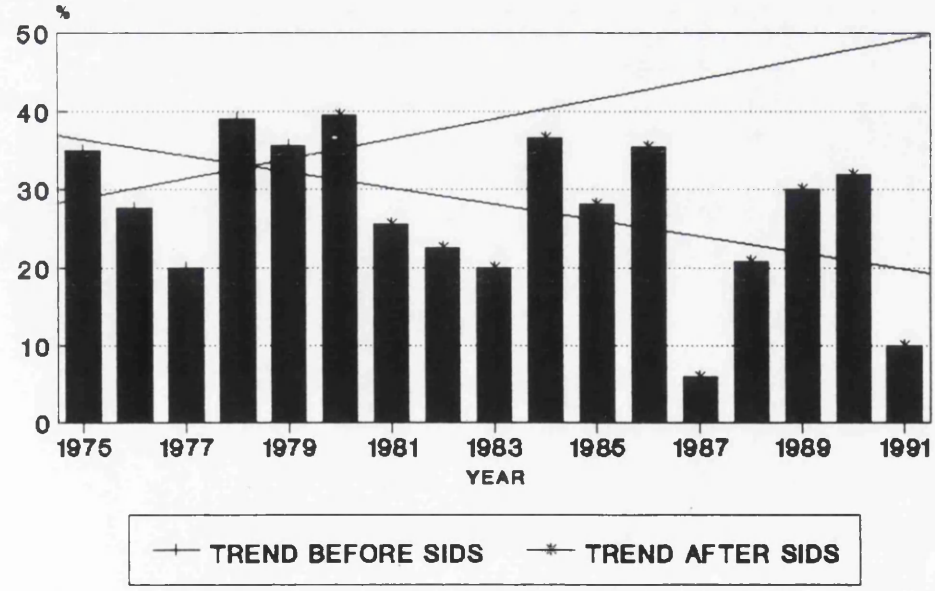
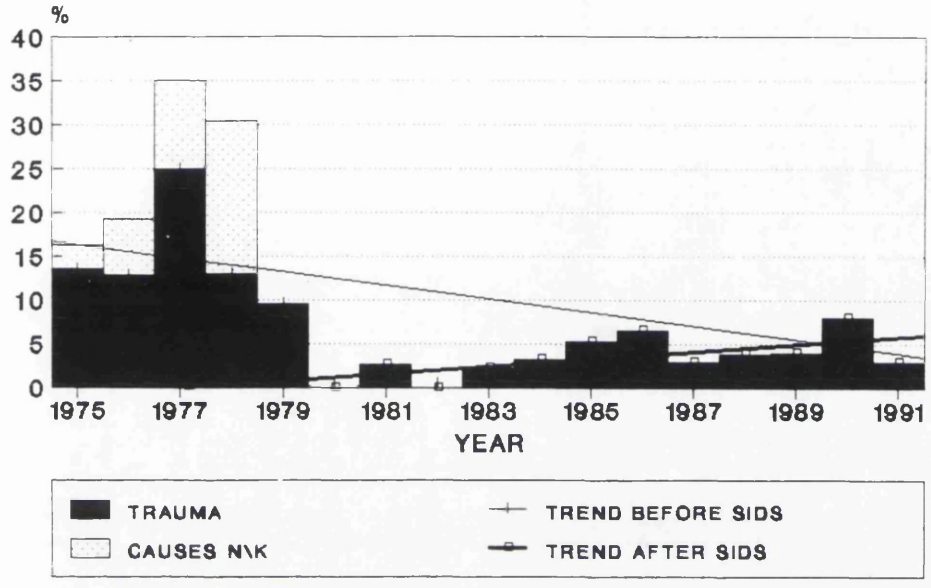


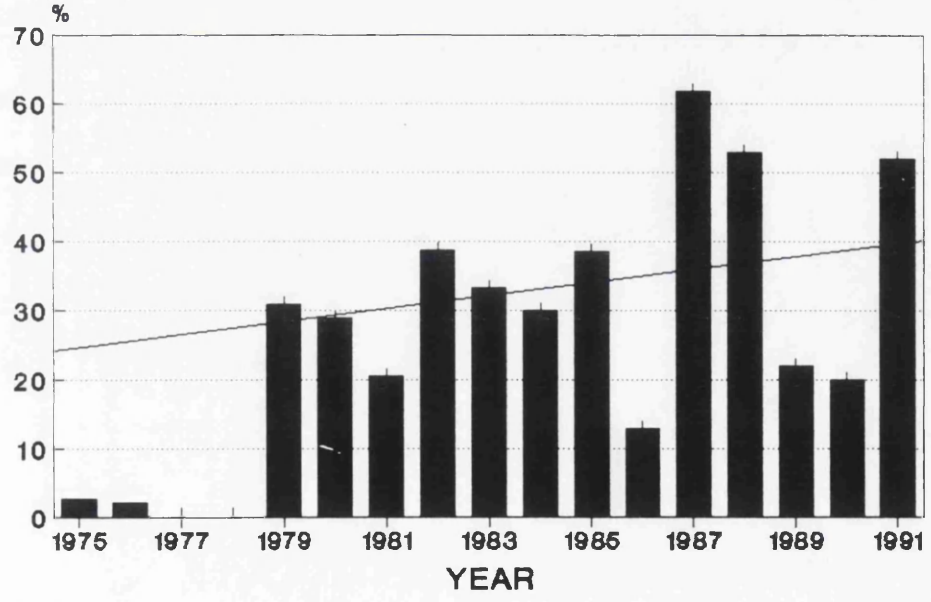
Fig 7.22 PERCENTAGE OF ALL POST PERINATAL DEATHS

POST PERINATAL DEATHS ASCRIBED TO TRAUMA AND CAUSES NOT KNOWN



PERCENTAGE OF ALL POST PERINATAL DEATHS
Fig 7.23

PROPORTION OF POSTPERINATAL DEATHS ASCRIBED TO SUDDEN INFANT DEATH SYNDROME



PERCENTAGE OF ALL POSTPERINATAL DEATHS
Fig 7.24

THE EFFECT OF CHANGES IN CAUSE OF DEATH CLASSIFICATION IN 1979

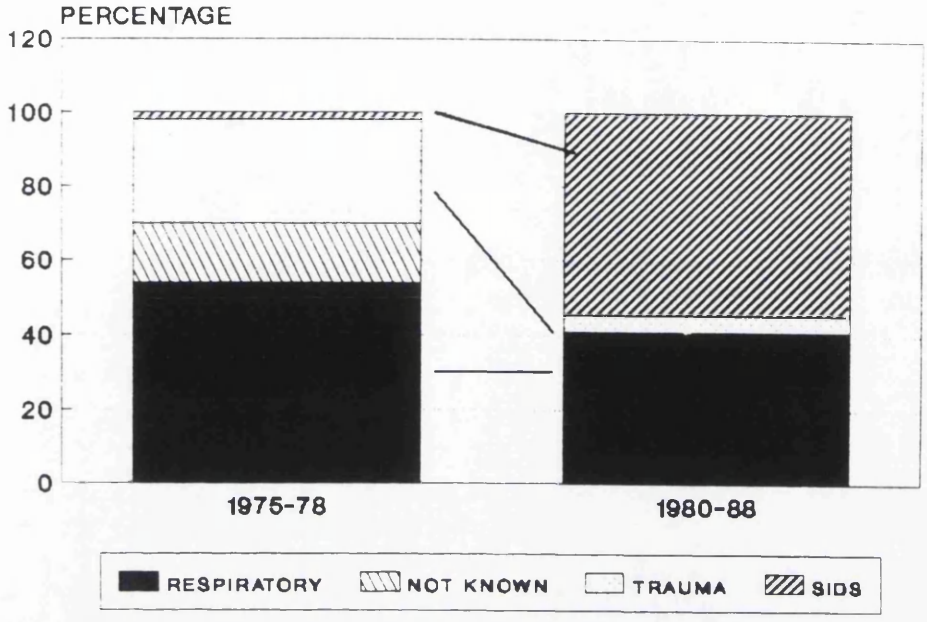


Fig 7.25

COT DEATH RATES IN SDHA COMPARED WITH ENGLAND & WALES

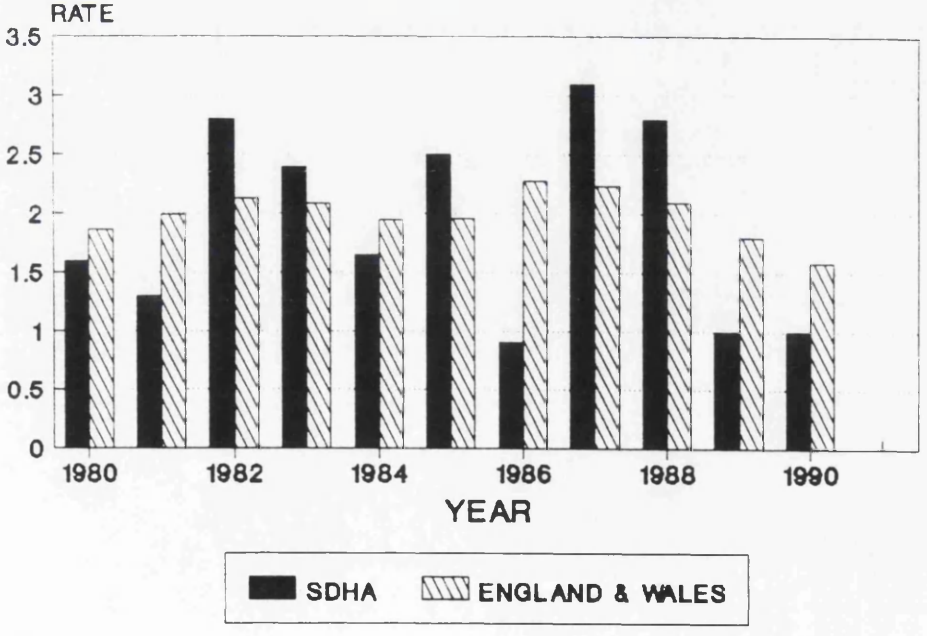


Fig 7.26

Section 8

EPIDEMICS AND POST PERINATAL MORTALITY

Introduction

Previous studies have associated infection with sudden unexpected death in infancy[67,79,95,124,125,126,127,128,129,130]. While it is recognised that many infectious diseases are so associated. The data presented here is limited to pertussis and measles which are statutorily notifiable diseases and for which information is available by local data collection.

The aim was to see if there was any association between epidemics of measles and pertussis and peaks in post perinatal mortality rates in Southern Derbyshire.

Methods

Measles and pertussis are routinely notified to the Department of Public Health Medicine by General Practitioners and hospital doctors for inclusion in the OPCS Communicable Disease Statistics Series MB2. Local notifications were recorded for the years 1977-1988 and related to the annual post perinatal mortality rates by group of preventability.

Results

The numbers of locally notified cases of pertussis and measles are depicted in Figure 8.1. Locally, epidemic years for measles were 1977/78, 1980, 1982/83 and 1986 (Figure 8.2, p76). Each successive epidemic was smaller in size either by notification numbers or by timespan. The numbers of notified cases of measles in the 1986 epidemic were approximately one quarter of those recorded in the 1977/78 epidemic.

Epidemic years for pertussis were 1978/79, 1982/83 and 1986/87, each successive epidemic recording fewer notified cases than the previous one. The peak in notified cases in the 1986 epidemic was approximately one third the size of that in the 1978/79 epidemic (Figure 8.3, p76). Dual epidemics of pertussis and measles occurred in 1977/78, 1982/83 and 1986 (Figure 8.4, p76).

Local post perinatal death rates fluctuated from year to year, peaking in 1978, 1982 and 1985, the general trend being downwards. The peaks in mortality coincided with

the dual epidemics of measles and pertussis 1977/78 when pertussis notification numbers were at their highest but measles notifications were on the decline.

In the 1982/83 dual epidemic the peak in post perinatal mortality rates again coincided with the greatest number of pertussis notifications (Figure 8.4, p76), but the numbers of measles cases were still increasing. In 1986 post perinatal mortality rates were at their lowest level during the decade of the study. This coincided with a peak in the dual epidemic of measles and pertussis.

Peaks in the death rates in the probably not preventable after birth grouping did not coincide with high numbers of notifications of either measles or pertussis, but in the possibly preventable grouping, peaks in mortality occurred in 1978/79, 1982/83 and again in 1985, the latter peak did not coincide with high notifications of pertussis or measles.

The single epidemic of measles in 1980 was at a time when the post perinatal mortality rate had fallen below the national average for the first time in many years and possibly preventable deaths were declining.

Discussion

Notified cases of measles and pertussis, while indicating epidemic years of these diseases in the District, do not reflect the true incidence of these diseases within the community. Although doctors who clinically diagnose measles and pertussis have a statutory requirement to notify the Director of Public Health, it has been accepted and understood that not all the overt cases are reported. Also, children who contract measles or pertussis but do not present with the recognised clinical manifestations are not notified and their numbers are not known[124].

Nationally and locally, as immunisation uptake rates increased, epidemics of measles and pertussis decreased in size[131]. During the dual epidemic of 1986 there was no local peak in post perinatal mortality which may support conventional opinion that smaller epidemics are associated with lower morbidity and mortality but does not guarantee it[124,129,132,133,134]. Many cases of Sudden Infant Death Syndrome have respiratory tract infection included on their death certificates[79,93,134,135,136], the symptoms of which may be compatible with undiagnosed whooping cough or measles, yet post mortem examination does not reveal a causal organism.

In Southern Derbyshire since 1983, Bordetella pertussis has been isolated in only one case at post mortem examination. This was a male child with three older siblings. He was born by spontaneous vertex delivery at full term weighing 3,060 grammes. There were no neonatal complications and no reported illnesses during his life. In December 1986 he was found dead in his cot at 11 a.m. on a Monday morning aged three months. He had been seen alive and apparently well by his father earlier in the day. There had been no recently recorded illnesses in the family in the weeks before the child's death. None of the children in this family had had pertussis immunisation. The certified cause of death was Sudden Infant Death Syndrome. At this time there was no system for case review in the District: nothing was known about the home circumstances of this child or the events leading to his death.

The seasonality of the occurrence of cot death has been recorded and high rates in the winter months is undisputed[74,75,76,77,136,137,138]. It is not only epidemics of pertussis and measles that occur during the winter and spring months. Epidemics of non notifiable disease[67,128,129,139,140,141,142] such as respiratory syncytial virus, influenzae virus, adenoviruses and rotaviruses all contribute to high morbidity in young children[140]. There are also non seasonable outbreaks of disease which are not associated with increases in cot deaths[74,137,138,139,142]. Other investigators have suggested that an infectious process during the cold weather might precipitate cot death in otherwise compromised children[74,78,126,127,136]. The child who has been compromised at birth may be susceptible to rapid progression of infection which may only result in minor or symptomless illness in his more robust siblings.

Other factors considered were the effects of passive smoking[143,144] on respiratory disease and the combination of hyperthermia and infection in young children.. Studies of respiratory tract flora in SIDS are inconclusive and usually cannot confirm that organisms found at post mortem examination are responsible for the child's death[139] as these organisms are found in a proportion of the general population. The numbers and types of organisms isolated by microbiological examination do not reflect the incidence of a disease in a population and neither do the isolates from SIDS conclusively give the diagnosis of death[142]. In Southern Derbyshire, as the SIDS rates increased the deaths ascribed to respiratory infections fell (Figure 7.11, p61). This was due in part to transference of diagnosis from the respiratory grouping to the SIDS group.

Conclusions

The findings only partly support the claim that post perinatal mortality is associated with dual epidemics of measles and pertussis in Southern Derbyshire.

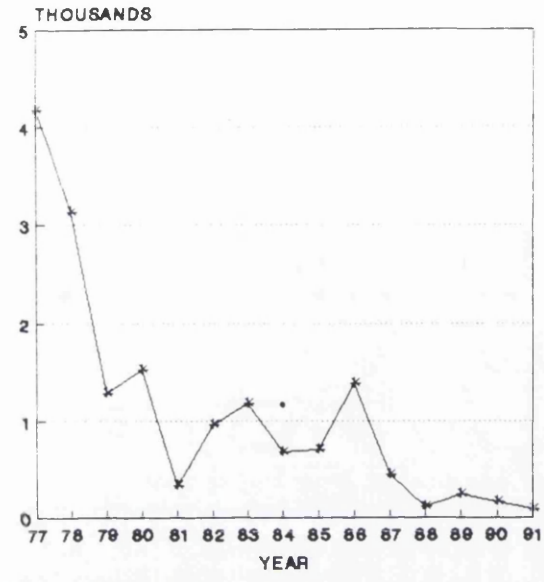
It was recommended that specimens for microbiological examination should be taken prior to death or immediately following death. This is now done in Southern Derbyshire. Even so, many deaths are still registered as SIDS when a history and/or findings of infection are present. A full history of events leading to the child's death should be available to the pathologist. This rarely occurs. A brief summary of the circumstances surrounding the child's death is all that the pathologist has to work on and he is usually unaware of any aberration in the child's life in the weeks preceding death.

SDHA IMMUNISATION UPTAKE RATES AND NUMBERS OF NOTIFIED CASES OF MEASLES AND PERTUSSIS 1977-1991

YEAR	1977	1978	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991
Measles notifications SDHA	4185	3127	1285	1521	329	961	1183	671	701	1377	445	113	250	158	94
Measles notifications E&W	173361	124067	77363	139487	52979	94200	103703	62080	97408	82061	42165	86001	26222	13302	9680
% Measles immunisation uptake SDHA	60	57	65	68	70.72	73.7	70.25	76.7	82.25	75.53	83.59	82	85	89	89
% Measles immunisation uptake E&W	50	48	51	53	55	58	60	63	68	71	76	78	80	84	87
Pertussis notifications SDHA	196	966	478	349	147	790	230	17	111	373	126	33	87	162	37
Pertussis notifications E&W	17475	65957	30816	21131	19385	65812	19340	5517	22046	36506	15203	5117	11646	15286	5201
% Pertussis immunisation uptake SDHA	44	32	42	48	60.4	62.9	69.83	70.47	76.06	74.46	72.3	79	84	86	86
% Pertussis immunisation uptake E&W	40	31	35	41	46	53	59	65	65	67	73	73	75	78	84

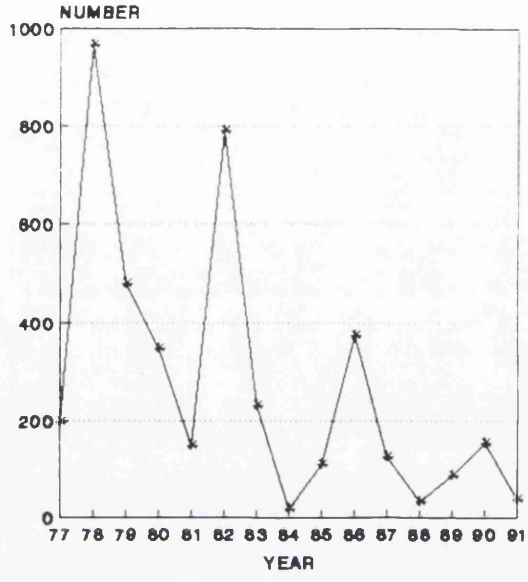
Figure 8.1
Source: OPCS Communicable Disease Statistics Series MB2

NOTIFICATIONS OF MEASLES
IN SDHA



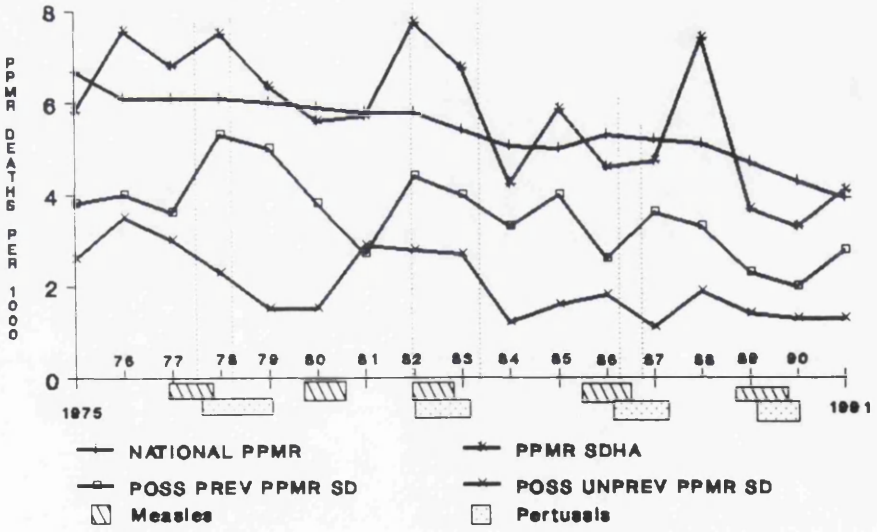
Source: OPCS Communicable Disease
Statistics MB2
Fig 8.2

NOTIFICATIONS OF WHOOPING COUGH
IN SDHA



Source: OPCS Communicable Disease
Statistics Series MB2
Fig 8.3

POST PERINATAL MORTALITY RATES
SDHA 1975 - 1991



Source: OPCS SD52, VS1 MB2
Fig 8.4

Section 9

EVIDENCE OF PRIMARY CARE WORKLOAD AND PERFORMANCE 1978-1988

Introduction

Health Service indicators provide information on the work done by Health Authorities, the staff they employ and the resources they use[146]. These indicators are derived from the readily available data submitted annually by Health Authorities. The use of the indicators by the Griffiths management structure of 1984[40] introduced a new approach to the attempts to improve health services to the population within budgetary restrictions. To this end data on well established criteria in primary health care efficiency was analysed.

Methods

Case loads: The numbers of General Practitioner Principals in Southern Derbyshire were obtained from the Family Health Services Authority for the years 1978-1988 and were related to the numbers of children aged 0-4 years resident in the District. The numbers of Health Visitor whole-time equivalent on the payroll was supplied by the District Nursing personnel services 1982-1988, previous information having been lost during the process of the 1982 reorganisation of the Health Services.

The annual average caseload of 0-4 year olds was related to the local General Practitioner Principal and Health Visitor.

Vaccination and Immunisation: District immunisation uptake rates of pertussis and measles were compared with the regional and national figures. In 1985 data was obtained from the local child health computer and immunisation uptakes of the primary diphtheria, pertussis and tetanus course (DPT) and measles immunisations were calculated by individual General Practice or Child Health Clinic. These were displayed graphically in the form of histograms[146]. Each General Practitioner was sent a copy of the histograms with the practice uptake rate denoted, to enable the General Practitioner to see how the practice uptake rates compared with all other practices in the District.

Child Protection Register: The numbers and rates of children (0-17 years) were obtained from the Derbyshire Child Protection Register, maintained by the Area Social Services Department.

Results

The data obtained are presented on p79 and in Figures 9.1-9.9, p81-86.

Caseloads: Between 1978 and 1988 the numbers of children aged 0-4 years in Southern Derbyshire increased by 5,517. The number of General Practitioner Principals increased by 61, their caseload of children 0-4 years remained virtually the same over the period. The number of Health Visitors increased by 9 whole-time equivalent 1982-88; despite this their caseload for 0-4 year olds showed a slight increase (Figure 9.1 p81 and 9.2 p82).

Immunisation Uptakes: Immunisation uptakes in Southern Derbyshire Health Authority were consistently above regional and national averages (Figures 9.3-9.5, p83-84). When uptakes of individual practices were examined the pattern of uptake differed widely between practices and also between individual immunisations. Figure 9.6 shows the distribution of practices having high and low uptakes for DPT and polio and Figure 9.7 shows the picture for measles. Both the figures depict the performance of an individual general practice in the context of all general practices in the District.

Child Abuse: The number of children aged 0-17 years registered on the Child Protection Register increased by 150% since the inception of the Child Protection Register in 1980 (Figure 9.8, p86). The rates increased by 3.7 per 1,000 children 0-17 years (Figure 9.9, p86).

Discussion

Court[3] in his report "Fit for the Future" 1976 identified problems in the field of child care, especially in the inner urban areas. Other studies have suggested that increased health visiting and easy access to services are instrumental in reducing post perinatal mortality[62,147,148,149,150,151,152,153,154,155,156,157].

The series of Health Service indicators devised by the Department of Health and Social Security[145,158,159] were designed to help involve clinicians in the management of their work. In the Community Child Care Package various factors were measured as shown overleaf.

Sources **DHSS Community Package 1986/87 Performance Indicators**

	District % Rank	Performance Indicator Value	Region Averages	England Averages
1 **Post Neonatal Mortality Rate**	40	3.8	3.9	4.2
2 a) Child Clinic Attendance Rate	25	719.0	740.0	821.0
b) % Nurse referrals to Doctors	71	8.3	5.6	5.8
c) Clinical Medical Officers/resident population	41	5.6	7.4	10.0
3 **Community Child Care Health Visiting**				
a) HV contact rate 1-4 years	36	658.0	700.0	703.0
b) HV/1000 under 5 yrs	8	2.7	3.3	3.5
c) HV staff/1000 under 5 years	8	2.8	3.5	3.7
d) % HV staff - HV's	57	97.0	94.0	93.0
e) % HV staff - Trained	58	3.2	5.1	5.0
f) % HV staff - Auxiliary	33	0.0	0.6	2.0

4 **Vaccination and Immunisation Uptake Rates** (Figures 9.3-9.5, p83-84).

Although these performance indicators are used as a tool in health management the Department of Health and Social Security book of indicators[145,158,159] did not mention the significance of variation of the District from regional and national norms. These indicators do not cover all aspects for which a Health Authority is responsible. They can only highlight issues for further discussion and investigation in the light of local knowledge of the characteristics of individual Districts.

The General Practitioner caseload of 0-4 year olds[160,161] remained steady but was above that of the Trent Region (average 104 0-4 year olds per General Practitioner Principal) (personal communication Trent Regional Health Authority). The Health Visitor child

caseload increased marginally. The need for health visiting[162,163,164] services had increased during this period as demonstrated by the increasing numbers of children on the Child Protection Register, which involves more time consuming multi-disciplinary case conferences. There was also pressure on the Health Visitors to increase their commitment to the elderly. The Health Visitors were apparently working very hard as they only ranked 8% in numbers per 1,000 0-4 year olds, but ranked 36% in their contact rate. This was a relatively high contact rate per Health Visitor, but it did not show the quality of performance.

Regarding child abuse[161], the rising numbers and rates of registered children paralleled an increasing social awareness, thus the parameters of assessment of the situation were changing. It is difficult to assess whether the rise in child abuse was an apparent or true rise, but Southern Derbyshire has one of the highest registration rates in Trent Region. Using the 1988 Department of Health and Social Security Local Authority Statistics, Southern Derbyshire Registration Rates 0-16 years compare with Districts like Bradford and Kensington and are much higher that the National Registration Rate of 1.9 per 1000 0-16 years.

Immunisation Uptake Rates: An assessment of uptake rates by different general practices showed a variation from 0-100% across the District, but practices with low immunisation rates did not always relate to areas of high child morbidity and mortality[165,166,167,168,169,170,171,172,173,174,175]. The District uptake rates of measles and pertussis increased by 26% and 47% respectively over the decade[168,169,170,171].

In general, the District performance value was poor compared with Trent Region and England, but these indicators provide information on the quantity of manpower and possible time availability and make no assessment of the quality of care provided.

Conclusions

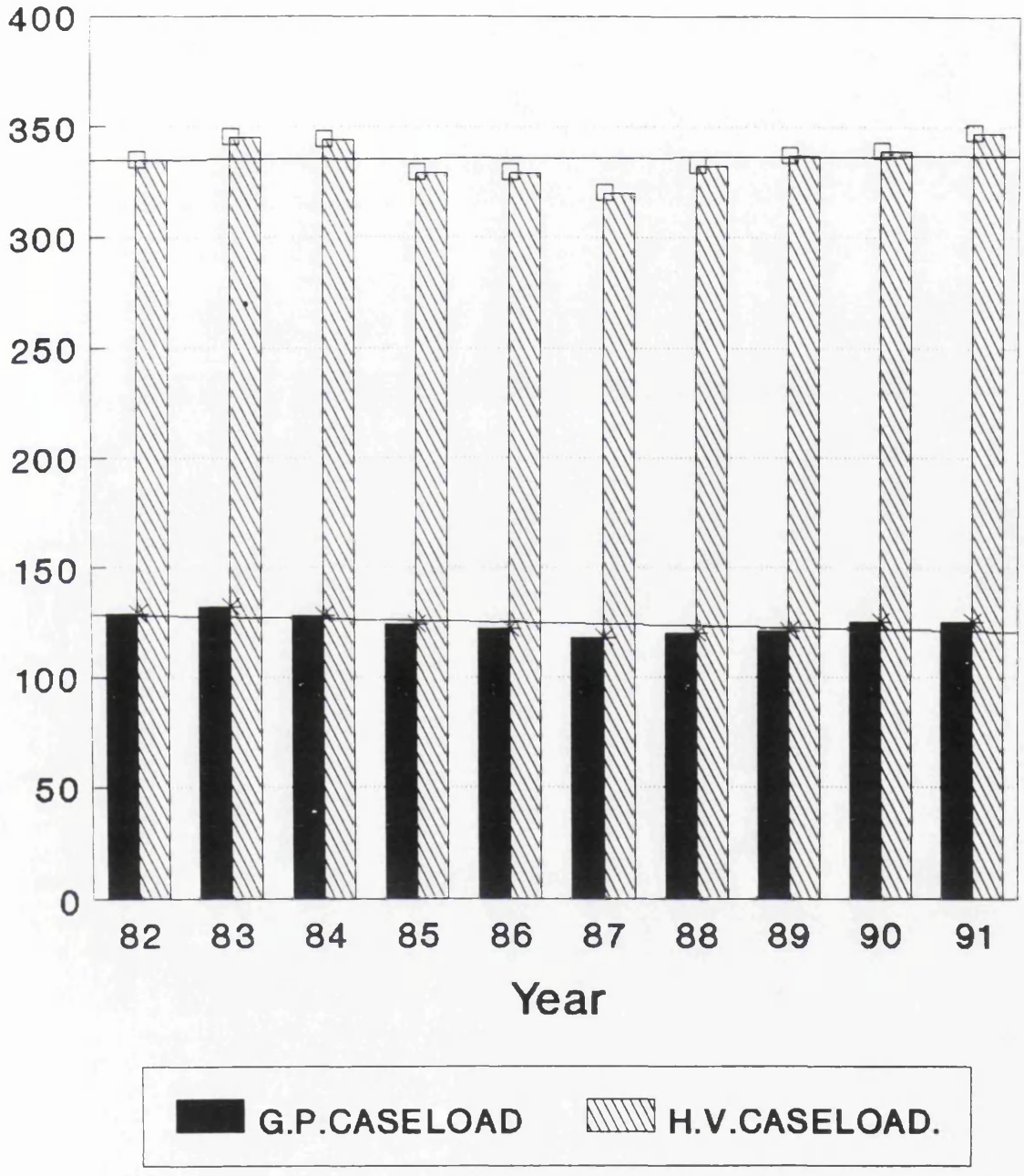
Some aspects of efficiency are measured by these performance indicators, but they do not give any idea of the quality or effectiveness of care provided.

PRIMARY HEALTH CARE CASE LOADS AND VACCINATION AND IMMUNISATION UPTAKE

	1978	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991
% Measles immunisation uptake	57	65	68	70.72	73.7	70.25	76.7	82.25	75.53	83.59	82	85	85	89
% whooping cough immunisation uptake	32	42	48	60.4	62.9	69.83	70.47	76.6	74.46	72.3	79	84	86	86
GP Principals	224	229	232	239	249	250	259	267	273	283	285	286	281	288
Numbers of children 0-4 years	28000	27000	27000	31100	32000	33000	33100	33100	33400	33500	34200	34700	35200	36100
Average number 0-4 years per GP	117	118	116	130	129	132	128	124	122	118	120	121	125	125
Health Visitors WTE					95.62	95.62	96.12	100.72	101.57	104.57	103.05	103.05	103.84	104.02
Average number 0-4 yr olds per HV					335	345	344	329	329	320	332	337	339	347
Children on CPR (Child Protection Register)			289	344	416	288	372	477	630	588	697	725	786	706
0-17 years (Rates)			(2.2)	(3.2)	(3.8)	(2.6)	(3.4)	(3.8)	(5.1)	(4.7)	(5.9)	(5.3)	(5.7)	(5.3)

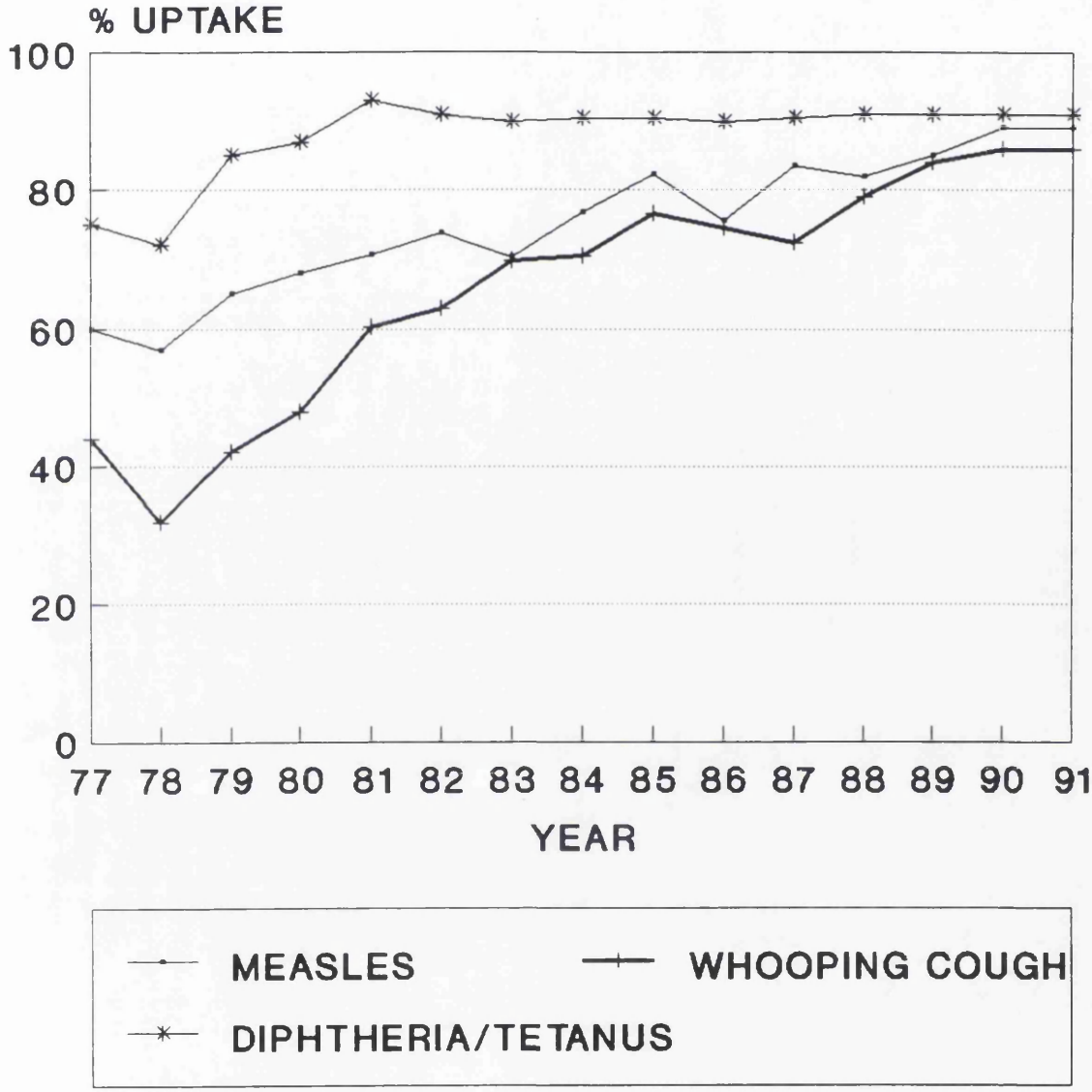
Figure 9.1

GP AND HEALTH VISITOR CASE LOADS
0-4 YEAR OLDS



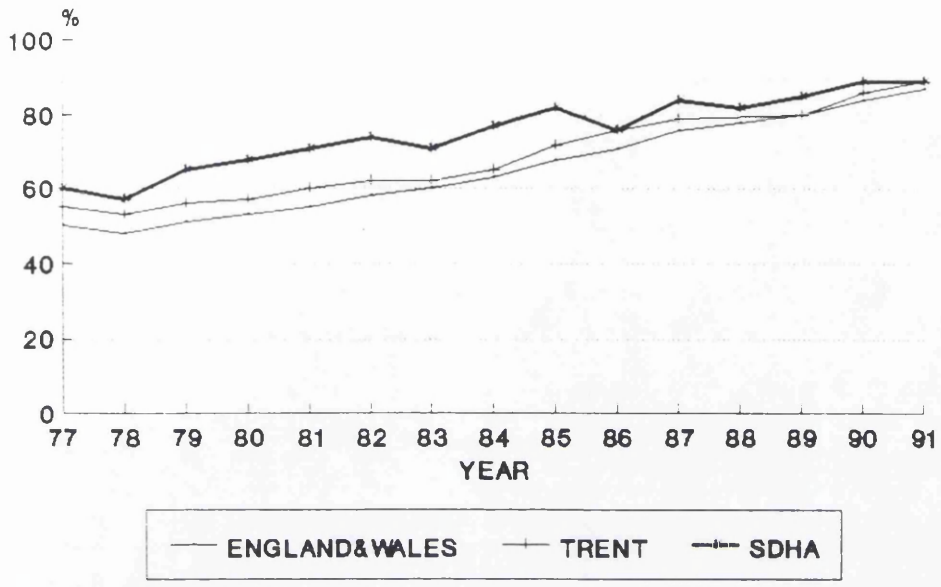
Source: Child Health Dept SDHA
Fig 9.2

IMMUNISATION % UPTAKE SOUTHERN DERBYSHIRE 1977-1991



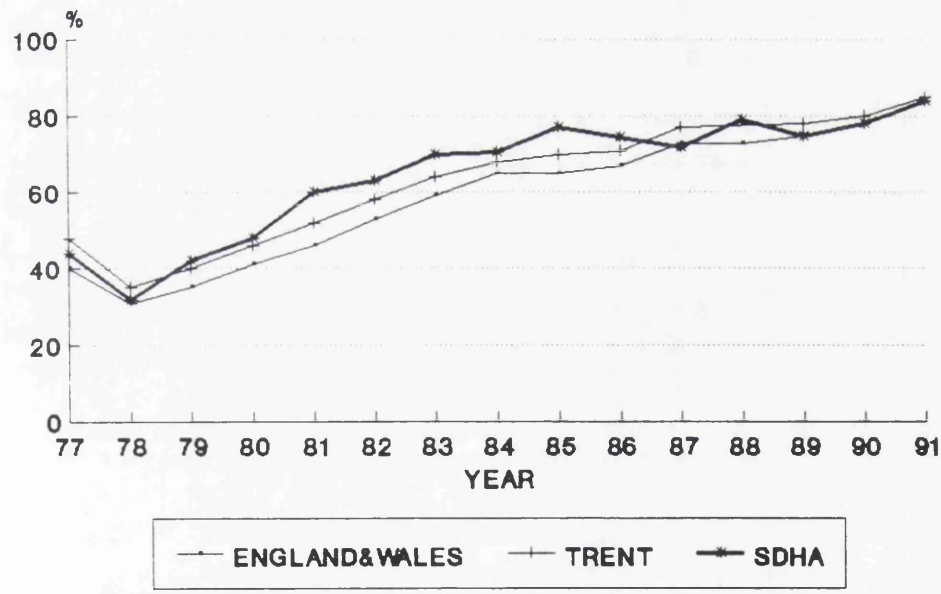
Source: SBL607 OPCS
Birth Registrations
Fig 9.3

IMMUNISATION RATES OF MEASLES IN ENGLAND & WALES, TRENT AND SDHA



Source: Form SBL607 OPCS
Birth Registrations
Fig 9.4

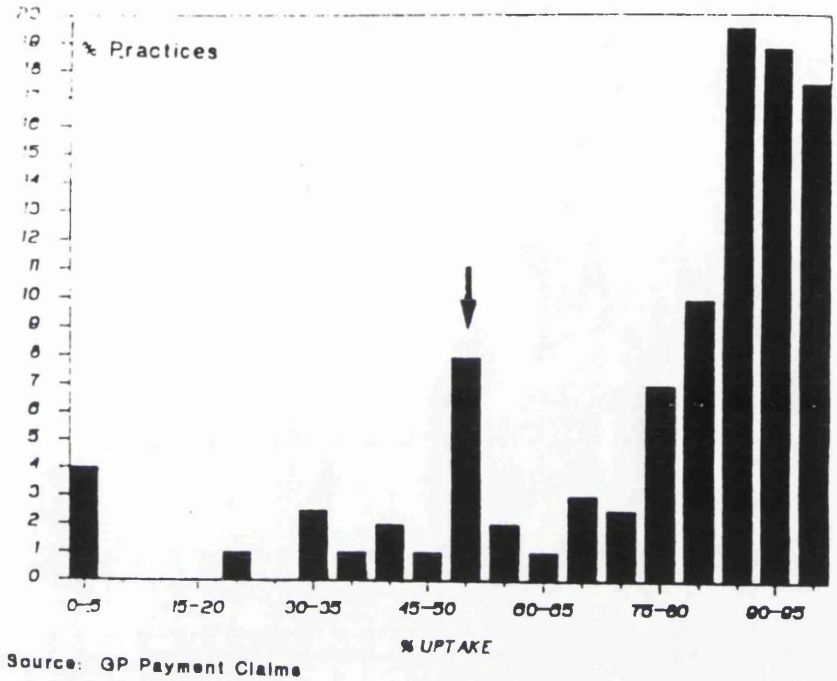
IMMUNISATION RATES FOR WHOOPING COUGH IN ENGLAND & WALES, TRENT AND SDHA



Source: Form SBL607 OPCS
Birth Registrations
Fig 9.5

COMPARING UPTAKE OF IMMUNISATION BY GENERAL PRACTICE. DIPHTHERIA, PERTUSSIS, TETANUS & POLIO

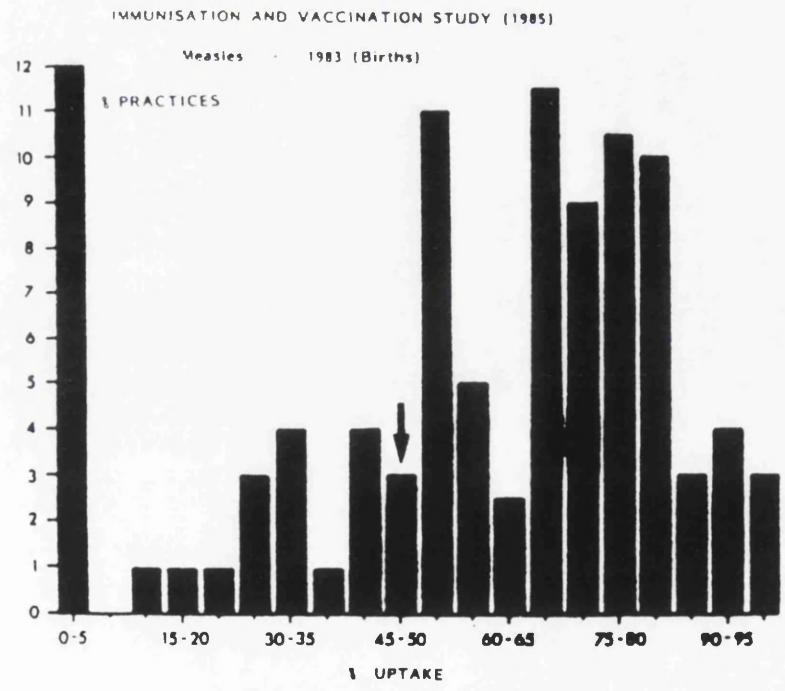
1983 - BIRTHS



Source: GP Payment Claims

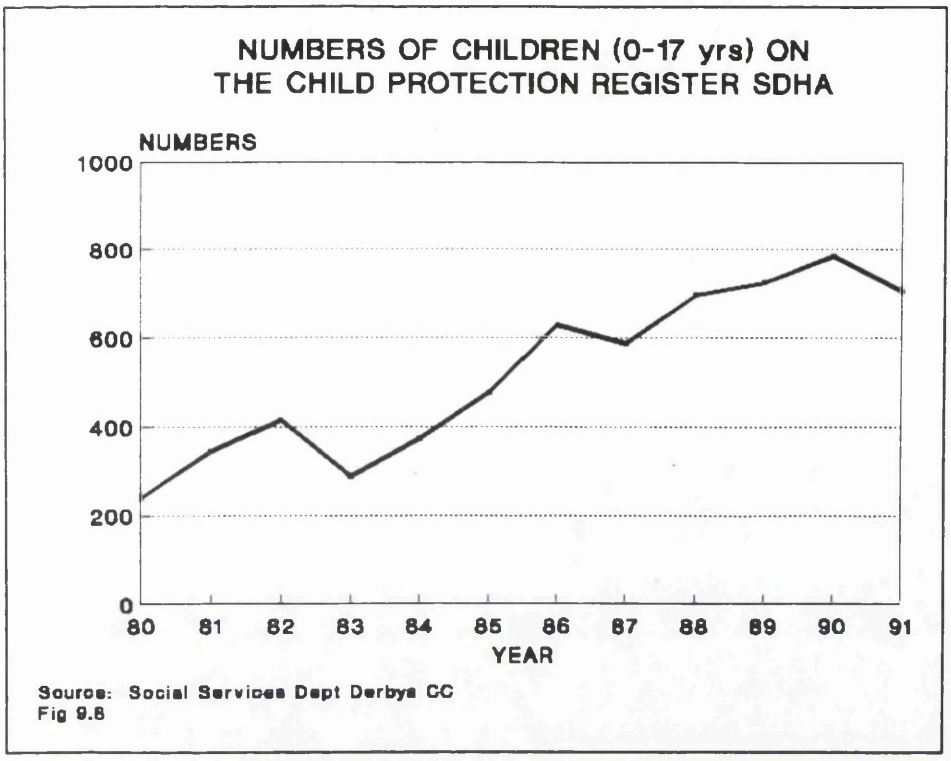
Fig 9.6

COMPARING THE MEASLES UPTAKE OF IMMUNISATION BY GENERAL PRACTICE BASED ON GP PAYMENTS

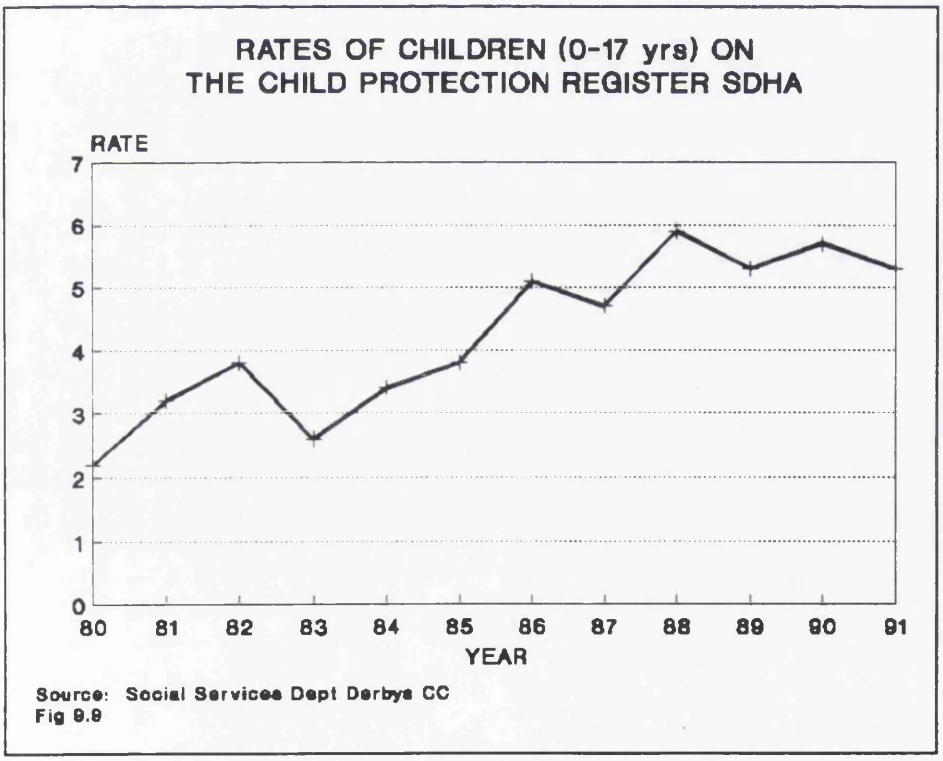


Note: ↓ used to indicate to the GP where his own performance lay

Fig 9.7



Number of children on Child Protection Register in England & Wales in 1989 was 41200. Number of children on Child Protection Register in England & Wales in 1990 was 43600.



Rate of children on Child Protection Register in England & Wales 1989 was 3.8.
Rate of children on Child Protection Register in England & Wales 1990 was 4.02.
(Rate per 1000 children)
Source: Children and Young Persons on Child Protection Registers. Dept of Health.

Section 10

CONCEPTS OF PREVENTABILITY

Mediaeval medicine developed the art of prognosis. Forms of therapy were developed later and involved the concept of preventing the natural course of a disease. Thus the concept of therapy and preventability are closely interlinked.

Concerning the possibility of preventing death in young children, in 1908 Dr Sidney Barwise[44] (who was then the Medical Officer of Health for the County of Derbyshire) observed in his Annual Report that infantile mortalities should be considered in two groups, "preventable" and "not preventable". In the "not preventable" group he included prematurity and immaturity which he attributed to ante-natal causes and thus outside his endeavours. In the "preventable" group he included diarrhoeal, respiratory infections and tuberculous diseases. Some of these deaths were attributed to an interaction of injudicious or contaminated food, neglect, poor nourishment and exposure to infection. Suffocation, syphilis and rickets were also considered preventable. Because he regarded these maladies as preventable after birth, he directed his efforts into measures to prevent and minimise the effects of infection, and to generally improve the home circumstances and environment of the population of Derbyshire. These factors he saw as the main causes of "preventable" deaths.

Gregory[176] in 1770 made the observation that the threefold purpose of medicine is (1) to preserve health, (2) to cure disease, and (3) to prolong life. Every last effort is made to sustain life rather than make a critical assessment of the option of death[1,84,86,86,177,178,179,180]. This observation assumes that we have the power of prolonging life, but does not mention prevention of disease as a major purpose in the practice of medicine. It is in this belief that levels of child care services might have played a part in keeping infant mortality rates lower than they might otherwise have been.

Infant mortalities vary between areas where living conditions are good but access to specialised paediatric services is poor, and death rates in inner cities where the reverse applies[181,182,183]. One hundred years ago, parents expected some of their children to die before the age of five, but there is now an increasing belief that modern science and technology should be able to prevent death. At birth and during the first month after birth, twice as many babies of unskilled manual parents die compared with those of

social class I[3,68,69,72,73,97,99,100,101]. Inequalities[2,3,147,184,185,186,187] amongst children are very obvious when whole populations are studied, but why one child dies and another survives in the same population is not so obvious. Most epidemiological and socially derived data is inadequate to explain this. To any one General Practitioner, working in the isolation of his own practice, disparities and unequal risks are not as obvious[54,56,57,70,96,149,150,188,189,190] and the individual Practitioner is, in most respects, dependent on his own experience and clinical acumen for the detection of overt and hidden risks.

Concepts of preventability of death have changed through time and have been dependent on improved living conditions, advancing technology, improved prescriptive and preventive measures[27,70,81,191,192,193,194]. Preventability is also dependent on problems of time. In political terms, throughout the history of the century, wars have heightened the awareness of various governments to the fact that our children are the replacement of those lost in the course of conflict[30]. Politicians surmised that if Great Britain was to survive as a viable nation, her children needed extra care and attention. In this consideration war was good for children[30,31].

Although the criteria have changed, the concept of groups of preventability is a pragmatic way of grouping diagnoses together to study large enough numbers to be meaningful in statistical terms for analysis[67,70,72,73,74,100,101,112,113,115,122,195]. Every clinician believes that deaths caused by illnesses in which he specialises must have a degree of preventability[81,85,86,93,99,119,120,126,196,197,198,199,200,201], if not in the present knowledge and circumstances, then at some time in the future. Without this belief his credibility would be in question and his work would seem pointless.

Concepts of preventability differ in degree in each diagnostic category[64,70,72,73,81,93] and values given on a preventability scale are always relative. At one end of the scale is the condition of anencephaly where death could not be prevented after birth and prevention must be directed before conception or during pregnancy. At the other end of the scale are deaths due to accidents, such as road traffic accidents and accidental drowning. These deaths are regarded as fully preventable. Between these two extremes lies the whole range of morbidity, where it is more difficult to balance views on the possible preventability after birth and the probably not preventable deaths[70,93,197,201,202].

Among Dr Barwise's "preventable" diseases, gastroenteritis and other infectious diseases still cause child deaths, but when these occur in this country they are usually

due to late or inadequate treatment and related to social class[2,3,64,67,70,73,147,148] rather than to the virulence of an infection. It is, therefore, legitimate to classify most of these deaths as possibly preventable on more than one score.

A different situation arises in the case of meningococcal infection where the mortality rate is similar in the different social classes. Early diagnosis and treatment usually prevent death, and this has been the case in Southern Derbyshire where General Practitioners carry parenteral Penicillin at all times, but the rate of disease onset and disease progression can be so rapid that death is inevitable, thus the Waterhouse-Friederichsen syndrome is on the borderline of preventability. Here a more conservative line was taken and meningococcal disease was grouped with the probably not preventable deaths.

Children dying as cot deaths were included in the possibly preventable group, not because it was felt that all the deaths of babies dying suddenly and unexpectedly were preventable, but because of three other major considerations:-

1. There is a considerable and continuing difference in the rate of cot deaths in different communities, both in this country and internationally[2,3,64,67,70,72,96,115,148,189,190,193,195].

2. There is a relationship of high numbers of deaths to social groupings, for example as seen in the USA in the black and white populations[74,203]

3. Confidential enquiries into each death have uncovered avoidable adverse factors that could well have contributed to a large proportion of cot deaths[67,71,93,113,150,151,179,203,204,205,206].

In the "probably not preventable" after birth grouping were those deaths where community care could do little to prevent the outcome. Influences in the preconceptual and ante-natal period are major contributory factors to these deaths[2,3,83,86,88,93,98,102,104,121,197,200,201,205]. Modern day technology and prescriptive medicine would be unlikely to save the lives of babies in this grouping. As this study is concerned with post-natal care and post perinatal death, the groupings of preventability are related to children presenting for health care and not to the ultimate preventability for all deaths.

Anencephaly, one of the extreme examples of non preventability of death, has been mentioned, but in other cases life may be initially prolonged although death is the most probable outcome. Many babies with severe congenital malformations survive birth and the perinatal period only to die in the early post perinatal period[121]. Spina bifida is one such example. Although detection of spina bifida is possible during the ante-natal period and the outcome of affected pregnancies is dealt with, there are still some children born with severe spina bifida which could have been prevented[201]. When these children survive into the post perinatal period and then die their deaths are usually not preventable.

Approximately one in 48 babies is born with a congenital malformation[121]. Babies born with minor congenital malformations who subsequently died were not included in the not preventable grouping as their conditions did not affect the outcome of death. The proportion of live babies born with severe congenital malformations has decreased, but many of the points relating to these births are not relevant to the age grouping of the study. Once a baby has been born with congenital malformations, some may be amenable to correction either by surgical means or by dietary adjustments. These babies usually survive and die later of other causes. Those babies who die with anomalies which are not at present compatible with life, were justifiably grouped in the probably not preventable group.

Perinatal causes, another major group of diagnoses in the probably not preventable grouping, comprised in the majority of babies of very low birth weight. There are many causes of low birth weight and its prevention lies in the ante-natal field. Prevention of death in liveborn low birth weight babies lies in good obstetric and neonatal care. In line with national experience, some of these babies have survived the perinatal period only to die at a later date[67,70,72,73,86,179], many of them before being discharged from the Special Care Unit[84,85,86]. The prevention of death in this grouping lies before the period where we would be providing care, thus in this study deaths due to perinatal causes were categorised in the probably not preventable grouping.

Preventability cannot be ascertained by death registration alone. The final facts of the course of the disease leading to individual death must be ascertained before any true judgement of its preventability can be made. Dr Barwise's observations on preventability, and to a large extent my own, are of necessity simplistic. It is postulated that under the circumstances the following grouping is the most practical way of classifying and investigating the problem.

Groups of preventability following full case review

Group A: Conditions with poor prognosis where deaths are apparently inevitable with the present knowledge and facilities.

1. Very gross deformities, some of known Mendelian determination.
2. Obstetric complications (prematurity/dysmaturity).
3. Obstetric complications related to delivery.
4. Tumour.
5. Hereditary degenerative disease e.g. Myotonia Congenita, Wernig Hoffman disease.
6. Acquired untreatable disease e.g. Waterhouse Friederichsen Syndrome.

Group B: These deaths could possibly be prevented. These are deaths where there is a potentially treatable condition present, either as a major cause or a possible contributing factor for death. They occur in diagnosable and treatable conditions where ideal circumstances occur.

1. The condition is recognised, documented and the treatment has been initiated.
2. The condition has not been recognised but there have been specific symptoms present before death.
3. There have been major non specific symptoms present, indicative of disease.

Group C: These deaths occur during the course of a minor condition not normally requiring treatment, i.e. during an apparently minor disease.

1. The condition is recognised and under treatment.
2. The condition has not been recognised but symptoms are present.
3. There have been minor non specific symptoms present.

Group D: No terminal illness is reported. Nothing is found clinically. These are the deaths as described by Beckwith[12] as Sudden Infant Death Syndrome.

Group E: Accidental death. These are deaths due to ordinary accidents.

Group F: Non accidental death. These deaths are due to non accidental injury either due to violence or to unapparent battering, deaths where filicide is suspected or proven.

Group G: These are children who present as unexpected deaths which have been insufficiently investigated to enable the children to be categorised into any of the A to F groupings. Group G includes cases where no necropsy has been undertaken or where necropsy is inadequate. If however, a confident clinical diagnosis has been made, for

example congenital deformity of the heart, then these cases can often be categorised without necropsy.

Section 11

STUDY BY CONFIDENTIAL REVIEW

Introduction

In 1987/88, having realised the limitations of the Trent Study (refer to Section 6, p40), all post perinatal deaths in the District were investigated in a study by detailed confidential case review. This system was introduced by me in January 1987.

The objective of the confidential review was to obtain sufficient information about each child death to answer the families' questions. All the people involved in the care of the case participated, in order to determine the causal and contributory adverse factors to death.

Methods

Before setting up the mechanisms for case review, wide consultation took place. A consultation document was presented to the Divisions of Child Health, Obstetrics and Pathology. A similar consultation document was presented to the Local Medical Committee, to gain approval from General Practitioners within the District. Once Divisional approval had been obtained the consultation document went through the Medical Executive Committees to the Medical Advisory Committee and with their approval, to the District Medical Committee. Finally, the case for confidential reviews was presented to the District Health Authority. This procedure took almost one year to achieve. Permission was obtained from the Director of Social Services for access to their case records. Similar permission was granted by the Police Department with the proviso that information was kept completely confidential and that the case notes were not taken from the police offices.

The Sheffield system of enquiry by questionnaire[161] was adopted, with the addition of a local questionnaire for the Hospital and Community Social Services Departments.

All deaths were routinely notified to the Department of Child Health and pending appointments for the dead child were immediately cancelled. Death notifications came from many sources and delay in notification sometimes occurred (refer to p48). On receipt of a death notification, the family doctor was contacted to ask if he would participate in the review and keep the child's records at the surgery instead of returning them to the Family Health Services Authority.

At one of the routine bereavement visits following the death of a child, the Health Visitor asked the family if they would be willing to participate in a confidential review of their child's death. It was explained that this would entail a visit from a senior Health Visitor and that they would be able to discuss events surrounding their child's life and death. This discussion would be completely confidential. Written parental consent was then obtained. Once parental permission had been obtained, a date was arranged for the senior Health Visitor to visit and complete the questionnaire concerning the child. This home interview was normally planned to take place approximately one month after the death of the child, but varied from family to family, as some could not cope with this type of interview until many months had passed. Each home interview was arranged according to parental wishes.

Questionnaires are also completed by:-

1. The family doctor
2. The family Health Visitor
3. The obstetrician
4. Accident and Emergency (if any contact)
5. Consultant Paediatrician (if any contact)
6. Social Worker (hospital and/or community if any contact)

Reports were obtained from the Coroner and the Pathologist including microbiology and virology reports.

All information was collated and a case discussion was arranged. This was usually held in the family doctor's surgery. The chairman was the same person throughout the study and had never been directly involved in the care of any of the children. The other core members were:

1. The family doctor
2. The Senior Health Visitor
3. The family Health Visitor

If the Paediatrician or a Social Worker had been involved with the case, they were invited, but the numbers present were always kept to a minimum and there were no observers. The case discussions were held with the General Practitioner as host, usually in his surgery. The General Practitioner always spoke first followed by the Health Visitor, then the Paediatrician or the Social Worker. The Senior Health Visitor who completed the home questionnaire was last to present her findings. The pathological

findings were always presented after all the clinical and social details. The named paediatric Pathologist chose not to take part in the case discussions, but was amenable to discussion of his findings. During the case discussion a summary chart of the details of the conference was completed and the General Practitioner was asked if the investigating team could be of any help with the care of the next child in the family. He was also asked if there were any recommendations that he could make as a result of the case review. The main points and the recommendations of the conference were agreed by all participants. The Senior Health Visitor and the Chairman summarised the conclusions of the case discussion and listed the main features. The deaths were classified into clinico-pathological groupings as defined by the Sheffield enquiries[93]. The recommendations made by the review participants were summarised.

Information available from case review of all deaths 1987/88 was analysed by epi info, and the source of each new item of information was identified. This was done to assess the importance of each step in a full case review. The sources of information were:-

1. Routine recorded information and questionnaires.
2. Home interview.
3. Case discussion in the General Practitioner's surgery.

All adverse factors mitigating against the child, which in some way may have contributed to death, were then related to each death in the clinico pathological groups. A final assessment of preventability was made: not preventable after birth, deaths with elements of preventability, and "do not know".

Results

There was a total of 14241 live births and 87 post perinatal deaths (Rate 6.1 per 1000 live births) (Figure 7.1, p55). A list of the post perinatal deaths, their causes and age group at death is shown in Figure 11.1. The ratio of male:female deaths was 1.1:1 (Figure 11.2, p108) 39% of all deaths were classified in the 'A' "inevitable" clinico pathological group, while the 'B' group contained the next largest proportion (22%). The male:female death ratio varied in the different clinico pathological groups. In the "A" and 'D' groups male and females were equally affected. In the 'B' 'C' and 'F' groups, males were more affected than females (1.7:1). Both deaths ascribed to accident 'E' deaths were male children.

Figure 11.3 shows the age group at death and clinico Pathological group. 40% of 'A' deaths occurred in the first month of life, but 50% survived to die > =4.3-<26 weeks.

The reverse applied in the 'B', 'C', 'D', 'G' groupings, where the majority died aged > =4.3-<26 weeks. In the 'F' group, 37% of the deaths occurred after 26 weeks. A different profile of clinico pathological death groups was shown in each age group. (Figure 11.4, p109). In the early deaths 'A' deaths accounted for 70% of deaths in this age group. 'B' deaths were not found. Of infants dying > =4.3 and < =26 weeks 26% had overt signs of treatable illness before death ('B' deaths). In the late deaths, >26 weeks, there were no idiopathic cot deaths ('D' deaths), but the proportion of 'F' deaths increased to 25%.

Analysis of case review status

Fifty two (61%) of the post perinatal deaths underwent full case review, including a case discussion in the General Practitioner's surgery. Routine information was available for all 87 deaths. Home interview was allowed in 69 (79%) of cases (Figure 11.5, p110).

The reasons for incomplete case review are shown in Figure 11.6. Parental permission was not given in 22% of cases and in a further 18% of cases the General Practitioner did not participate at every stage of the review. Figure 11.7 shows the case review status by clinico pathological group. Only 9(26%) of 'A' deaths had a full review, but in the 'B' and 'C' groups, 90% were reviewed by the full team.

Only adverse factors thought to have contributed to death in some way were listed during the process of case review. A total of 325 factors was identified, 183 were found in recorded routine data, a further 45 new factors at home interview and 97 new items of information emerged for the first time at case discussion (Figure 11.8, p112). The teams participating in the case reviews judged which adverse factors should have been amenable to action. The greatest proportion of adverse factors which were actionable, were found at case discussion (65%).

As the adverse factors emerged they were categorised into 12 convenient groups. These are listed in Figure 11.9. There were 45 items of information indicative of the child's medical condition during its life. These were all recorded in routine information and none were judged actionable. Professional factors, including delivery of care, were found at all three sources of information. Fifty eight per cent of those found in routine records were actionable compared with 81% of those disclosed at case discussion and 100% found at home interview.

In the group with parenting problems, only one out of eleven adverse factors from routine information was actionable compared with over half those emerging during home interview and case discussion. Lifestyle included heavy tobacco and alcohol usage and included two families with drug problems. Unsupported mother emerged at all stages of the review, but it was only at case discussion that the professionals acknowledged that they could have taken action.

Psychiatric problems were well recorded but seven psychological factors were revealed at case discussion. Family stress was recorded in the case notes and also appeared at both home interview and case discussion.

Poor housing and socio economic status was judged non actionable. This information was mostly from routine records.

Poor bonding was difficult to judge. There had to be consensus agreement by all those involved in the care of the family. It was found in three cases and at three different sources of information and never thought to be actionable.

Family involvement with other agencies was known from routine notes and was acknowledged as a trigger for increased awareness by the teams of carers. Factors relating directly to the child or previous siblings were available from routine information except where the mother disclosed that she had found the baby face down.

When adverse factors were related to deaths by clinico pathological groups (Figure 11.10, p114), the actionability ratio varied by different group, being twice as high in the 'B', 'C', 'D' groups as in the 'A' group of deaths.

After relating adverse factors to the clinico pathological groupings, the deaths were divided into three final groups of preventability. Figure 11.11 and the actionability ratio was calculated for each group. This showed at 0.6 for the group of deaths with elements of preventability (Figure 11.12, p114).

Cases where there had been a full review (n=52) were analysed by the number of adverse factors found for the first time at case discussion. A total of 97 adverse factors were associated with 37 children and 63 of these factors were judged to have been actionable by the care teams at case discussion. Forty two of the actionable factors applied to 22 babies in the 'B', 'C', 'D' groups (Figure 11.13, p115). Figure 11.14

identifies the source of information of the adverse factors and the proportion which were actionable. The actionability case ratio was four times greater when the source of information was the case discussion. In Figure 11.15, the final preventability grouping, 37 actionable factors were associated with 19 babies where there were known elements of preventability (Figure 11.15, p110).

Thirty seven (71%) of the 52 babies who had had a full case review had actionable adverse factors revealed for the for the first time at case discussion.

Discussion

The number of post perinatal deaths in a district is small, each death being a sentinel event[207]. When conventional data collection does not answer the families' questions, a more detailed investigation is warranted. Although every death is ascribed a terminal causal factor, adverse events during the child's life maybe contributory to death[82,83,181,182,191,192,193,195,198,199,200,202,203,205,206,208,209,210,211,212]. This study by case review identified adverse factors which if remedied, might have altered the outcome of death. The source of each item of information was identified to routine information, home interview or case discussion.

The Deaths

While 34 (39% of all deaths) were due to inevitable causes, 40% of these occurred in the first month of life, due to congenital malformation or gross immaturity. Fourteen (25%) survived to die in the 1-6 month period reflecting the findings of Pharoah and others[1,10,70,72,73,84,85].

Thirty (35%) of all deaths showed varying signs and symptoms of treatable disease. These were the partially explained cot deaths[1,10,83,93,118]. Twenty three (77%) of these deaths occurred between one and six months[1,72,73,75,78]. One occurred in the first month of life and 20% occurred as late cot deaths.

The ratio of male to female children dying was 1.1:1, but in the inevitable group and the idiopathic cot deaths, males and females were equally affected. In the clinico pathological groups of deaths with the highest preventability, the male to female ratio was 1.7:1[3].

When deaths were categorised by clinico pathological group and age group, the age pattern was very different between the 'A' deaths and the 'B' to 'G' deaths[93]. The age

at death in the 'A' group was evenly distributed in the early and common cot death age groups. In the 'B', 'C','D' and 'G' groups, there were very few deaths before one month and after six months. The 'E' deaths occurred in the early cot death age group but over one third of the suspected filicides occurred in the late cot death group.

Status of Case Review

Although full case review was offered to all cases, 52 (61%) of cases completed case review with the case discussion in the general practitioner's surgery. In 35 cases the review was completed by the chairman and other primary carers without the General Practitioner being present. A total of 69 (79%) families allowed a home interview. The reasons for incomplete case review depended on decisions taken by the parents or the general practitioners. In the 'A' group of deaths, the proportion of full case review was much smaller than the other deaths and was related to what was seen as an acceptable clinical diagnosis by the carers. In ten cases the general practitioner did not participate in any part of the review and in a further six cases, although they had completed the questionnaires, the general practitioners elected not to attend the case discussion, implying that they had more urgent things to attend to. In 19 cases, mostly 'A' deaths, parental consent was withheld, the majority being satisfied with the cause of death given, although in one case the mother was too ill to participate in the enquiry. In two other cases the mothers had disappeared from the district, one with three children on the Child Protection Register. This method of selection made internal comparisons between the different groups of deaths difficult as in the pre-diagnosed inevitable group of deaths, it is likely that only families with other problems were fully studied. The lack of response by parents and general practitioners to the call for detailed information on the pre diagnosed inevitable deaths could be a matter for concern in the proposed case control study in the National Confidential Enquiry into Stillbirths and Deaths in Infancy (CESDI)[213], as it suggests that this detail will not become available for healthy infants used as controls[214].

There was a high proportion of complete case review in the death categories 'B' and 'C' where children showed varying degrees of signs and symptoms of treatable illness before death. These children had died suddenly and unexpectedly and the diagnosis at death had left many unanswered questions. A full case review was undertaken in all but three of these deaths. Two of the three cases did not have a case discussion due to general practitioner default, the third case did not have a home interview due to lack of parental consent. Over 90% of the cases which were potentially the most amenable to intervention had a full case review.

Case Review

The post perinatal death reviews were used to monitor health care delivery in Southern Derbyshire[215,216,217,218,219,220,221]. Adverse factors within the delivery of the Health and Social Services as well as in individual family's social and environmental circumstances, were used as a trigger as many of these factors were amenable to action[221]. Only factors which were thought to have contributed to death were selected. In theory, all adverse factors should be amenable to action but pragmatically, action taken to remedy adverse factors is dependent on the awareness, capabilities and facilities of the carers to react to their presence[221,222,223,224]. These abilities vary making it difficult to identify criteria for setting protocols for professional intervention[224]. Quality of care is an individual responsibility and the majority of carers were able to identify factors where their action had fallen short of their own expectations of their standard of care. It was this acknowledgement that determined whether intervention could have taken place.

Adverse factors

1) Demographic factors. These included socio-economic information and were for the most part, routinely recorded.
2) Psycho-social factors. These included childhood experiences, attitudinal problems and ability to cope under stress. These particular factors determine why in identical environmental circumstances, one family can cope and another cannot.

There were 325 adverse factors attributed to the 87 deaths, 183 of these were found from routine case information and only 20% of these were actionable. This was the least sensitised information and comprised mainly demographic features, many of which were common to families whose children do not die and therefore, were least likely to trigger increased awareness and action by the professionals. At home interview, 45 new factors emerged and 58% of these were actionable. The revelation of these adverse factors depended upon the skill of the senior health professional undertaking the home interview. Once the mother's trust was gained she was able to disclose her private feelings of self doubt, guilt and loss of confidence in professional services. At case discussion a further 97 adverse factors were revealed, 65% of which were actionable. The adverse factors revealed were highly sensitised information which had not been recorded but appeared to be more amenable to action than information found from routine case notes or home interview.

At case discussion those participating were only present with the general practitioner's approval. The General Practitioner was host and those invited were made welcome.

In this environment the participants were able to examine the appropriateness of their actions on adverse factors in the child's life. The case discussions were chaired by the same person throughout the study so that any bias introduced during the case discussion applied equally across the district and comparisons could be made on a more objective basis. The chairman was independent of the direct care of any of the cases. This was crucial in maintaining an objective, non-judgemental atmosphere to encourage constructive criticism and discourage negative remarks or the apportionment of blame to individuals.

The chairman's role was to facilitate discussion within the teams and allow agreement and implementation of new standards of care. Difficulties in validating value judgements are acknowledged but should be countered by the objectivity of the chairman. Similarly, the chairman should redress balance when one member of the case discussion appears to try and influence the other contributors. These skills enabled consensus agreement to be reached in this study.

Adverse factors by clinico pathological group

The list of adverse factors obtained during full case review was long and varied but not exhaustive. As the study progressed, it was found convenient to group the factors under 12 headings, some factors were repeated in more than one case and many cases had more than one factor. As more cases are reviewed, factors common to different groups of death will become more apparent. The grouping of factors enabled a more objective approach to the review[222,223].

Some adverse factors found during the case review were general service faults which could not be remedied by the primary carers. Action was dependant on change of policy by provider units. The chairman recorded these recommendations and was charged with the responsibility of ensuring that action was taken. Outcomes are monitored by the Director of Public Health on an annual basis.

The type of information emerging at each stage of the review was consistent across the different groups of death. Socio-economic and lifestyle information was recorded in the case notes in all groups of deaths including the 'A' deaths. The family functioning, attitudes and relationships with professionals, and failure in delivery of care were not recorded, but were revealed at case discussion. These were the factors judged most amenable to action.

In the inevitable deaths, if the adverse factors had been actioned by the teams, it is suggested that the quality and length of remaining life could have been improved. In this group of deaths, most of the actionable factors were communication defects from professionals to parents. In two cases the child had been allowed home, but no written instruction or telephone contact number had been given. At home interview some mothers felt they had received inadequate support and information from professionals.

The greatest proportion of actionable factors related to the 'B' deaths. Frequent features in this group were the known incompetence of the mother and instability in the functioning of the family[225,226,227]. The teams realised that they had not acknowledged the significance of these factors and as a consequence their response had been inappropriate. In one case a child had been seen by five different doctors who had not discussed the case with each other. No-one had taken responsibility. In other cases, documented weight loss and other signs of illness had not triggered response from the General Practitioner or Health Visitor. Lack of information to the hospital concerning appalling home conditions resulted in two babies being discharged home when prolonged hospital care would have been more appropriate.

Adverse factors in the 'C' group of deaths were mainly concerned with inadequate support and immature mothers[225,226,228]. Although these facts were recorded in the case notes, no action had been taken.

In the 'D' Group of deaths, the idiopathic cot deaths, very little new information was revealed at the case discussion which would be expected under the Beckwith rubric[12]. It was recorded however, in the case notes that most of these mothers were heavy smokers[229,230].

At case discussion in the one 'E' Death, subject to full case review, no actionable factors were found.

The 'F' deaths revealed interagency failure[231,232,233]. In every case the parent had recorded psychological/psychiatric problems and had received treatment[227,234], but the needs of the rest of the family had not been met. This group contained a family where there had been a previous cot death of a sibling[235].

The actionable factors in the 'G' group were professional default by inadequate histology and in one case undiagnosed illness in the Mother. Inadequate histology was

a matter for concern and triggered action to ensure that the job description for a newly appointed consultant pathologist contained responsibility for paediatric necropsies. A protocol for paediatric necropsy has been adopted. In a further case the baby came from a family with known multiple social problems. The Health Visitor had an impossible case load in a deprived area. She identified that she had not been able to deliver the quality of care that she would have done in other circumstances. This was an instance where the Chairman of the case discussion took action on the Health Visitor's behalf which resulted in an ongoing review of Health Visitors case loads[3,155,231,233,236,237,238,239,240,241,242,243].

When the adverse factors were related to the clinical pathological groups of death the actionability/case ratio for 'A' deaths was 0.6 compared with 2.2 for the 'B', 'C' and 'D' deaths. Forty six per cent of factors being actionable in the 'B', 'C', 'D' group compared with 27% in the 'A' group.

After applying all applicable adverse factors to each death, the deaths were grouped into final groups of preventability. In 33% of deaths, intervention may have altered the outcome but in a further 26% it was not known whether intervention would have made any difference. The actionability/case ratio was two in the deaths with elements of preventability and in the 'do not know' group the actionability/case ratio was 1.8, but this group contained the 'G' deaths where the pathology was incomplete and not all the facts were known.

When the source of actionable factors was related to home interview and case discussion, the actionability factor per case was four times greater for information obtained at case discussion. The 63 actionable factors related to 37 cases (71%), which had had a full case review.

Examples of adverse factors which trigger action[221]

The case discussion enabled the teams to identify groups of adverse factors in each clinico pathological group of death. These factors acted as trigger points to take action to improve their quality of care. Communication problems were common and cot death is now part of the multidisciplinary training programme. The annual monitoring of case reviews by the Director of Public Health has identified that communication failures no longer feature as actionable adverse factors.

Babies who live in cold damp houses and whose parents smoke have alerted the participants in case reviews to the need for early response to illness[244,245,246].

The staff at the Special Care Baby Unit now routinely give telephone contact points and an information leaflet to Mothers whose babies have been under their care. There have been no further reports of this type of communication failure.

Non compliance with medical services including non attendance for appointments, and no access for the Health Visitor were identified as characteristics of poor parenting[2,3,150,168,231,247]. Following case discussions the teams realised that if these characteristics were identified, more vigilance and support were required. There is evidence from the Director of Public Health's annual review that these problems persist and are the main group of recorded adverse actionable factors.

Conclusions

The new information that became available from these case reviews was found to be of such value to the primary health care teams and to the managers of the child health services, that the system was adopted as an ongoing measure of individual family need, service delivery and change in standards of child care.

The difference between this study and the Department of Health and Social Security 1000 Death Study[67] was the transfer of the case discussion from the hospital to a venue of the general practitioner's time and choice. The source of information in the 1000 deaths study was not recorded and I was, therefore, unable to estimate the amount of extra information made available by this change of venue.

The recent introduction of the National Confidential Enquiry into Stillbirths and deaths in Infancy (CESDI)[213] appears to follow the pattern of enquiry used in the Maternal Mortality Enquiry[248] and the National Confidential Enquiry into Perioperative Deaths[249], which has a case discussion in hospital premises. The Southern Derbyshire study shows that case discussion in the General Practitioners surgery is an essential component in the process of case review into post perinatal deaths, as it is a major source of unrecorded information which is amenable to intervention.

Such case discussion allows immediate feedback of all information to the participants and acts as an educational exercise to improve the quality of care for infants, including the care of the next child in the family[221,250,251,252].

Summaries of some cases (Appendix 1) demonstrate some remediable factors and illustrate medical and social details of the families involved which would not have been available without the willing cooperation of the case discussion members and the families.

Figure 11.1

Registered Cause of Death by Age Group at Death

Early Cot Deaths (ie. < 4.3 Weeks Old)

Case No 3 - SIDS.
Case No 5 - Cornelia de Lange syndrome.
Case No 9 - Acute pyelonephritis. Fallot's tetralogy.
Case No 12 - SUDI.
Case No 16 - Gross prematurity.
Case No 18 - SUDI.
Case No 21 - Severe prematurity.
Case No 31 - Acute tracheo-bronchitis. SUDI.
Case No 33 - Severe prematurity. Idiopathic Respiratory Distress synd.
Case No 39 - Extreme prematurity.
Case No 50 - Extreme prematurity.
Case No 51 - Acute congestion of the lungs. SUDI.
Case No 56 - Heart failure. Pulmonary infection. Prematurity. Convulsions.
Case No 58 - Necrotising enterocolitis. Prematurity.
Case No 59 - Hypoplastic left heart.
Case No 61 - Acute congestion of the lungs. SUDI.
Case No 64 - Complex heart lesion.
Case No 65 - Prematurity. Respiratory Distress syndrome.
Case No 74 - Spinal muscular atrophy.
Case No 87 - SUDI.

Common Cot Deaths (ie. ≥ 4.3 and ≤ 26.0 Weeks Old)

Case No 2 - SUDI.
Case No 6 - Haemophilus influenzae meningitis.
Case No 7 - Acute tracheo-bronchitis. SUDI.
Case No 8 - Acute pulmonary oedema. SUDI.
Case No 10 - Edward's syndrome. Severe cardiac defect.
Case No 11 - Acute congestion of the lungs. SUDI.
Case No 14 - SUDI.
Case No 15 - Acute congestion of the lungs. SUDI.
Case No 20 - SUDI.
Case No 22 - Acute congestion of the lungs. SUDI.
Case No 23 - Manslaughter.
Case No 24 - Acute congestion of the lungs. SUDI.
Case No 25 - Acute congestion of the lungs. SUDI.
Case No 26 - Acute congestion of the lungs. SUDI.
Case No 27 - SUDI.
Case No 28 - SUDI.
Case No 29 - SUDI.
Case No 30 - Gastro-enteritis. SUDI.
Case No 32 - Trisomy 13.
Case No 34 - Broncho-pneumonia. SUDI.
Case No 35 - Acute congestion of the lungs. SUDI.
Case No 36 - Spina bifida. Hydrocephalus.
Case No 37 - Acute tracheo-bronchitis. SUDI.
Case No 38 - SIDS.
Case No 40 - Zellweger's syndrome.
Case No 41 - Severe prematurity. Hyaline Membrane Disease.
Case No 42 - Genetic disorder - immune deficiency.
Case No 43 - Acute tracheo-bronchitis. SUDI.

Figure 11.1 (continued)

Case No 44 - SUDI.
Case No 45 - SUDI.
Case No 46 - Manslaughter.
Case No 47 - Acute broncho-pneumonia.
Case No 48 - Acute congestion of the lungs. SUDI.
Case No 53 - Congenital immune deficiency.
Case No 54 - Sub-aortic stenosis.
Case No 55 - SUDI.
Case No 57 - Renal failure. Oxalosis.
Case No 60 - SUDI.
Case No 62 - Meningococcal septicaemia. Waterhouse Friederichsen synd.
Case No 63 - Acute congestion of the lungs. SUDI.
Case No 66 - Congenital abnormality of the brain. Epilepsy.
Case No 67 - Atrial septal defect. SUDI.
Case No 68 - Prematurity. Broncho-pulmonary dysplasia.
Case No 70 - SUDI.
Case No 71 - Acute congestion of the lungs. SUDI.
Case No 72 - Acute tracheo-bronchitis. SUDI.
Case No 73 - Acute tracheo-bronchitis. Hypernatraemic dehyd. SUDI.
Case No 76 - Down's syndrome. Congenital heart defect.
Case No 77 - Acute lymphoblastic leukaemia.
Case No 78 - Meningococcal septicaemia. Waterhouse Friederichsen synd.
Case No 79 - Acute congestion of the lungs. SUDI.
Case No 80 - Acute congestion of the lungs. SUDI.
Case No 81 - Upper respiratory tract infection. SUDI.
Case No 82 - Extreme prematurity. RSV. Broncho-pulmonary dysplasia.
Case No 85 - Extreme prematurity. Broncho-pulmonary dysplasia.

Late Cot Deaths (ie. > 26.0 Weeks Old)

Case No 1 - Acute congestion of the lungs. SUDI.
Case No 4 - Bacterial meningitis. Broncho-pneumonia.
Case No 13 - Pneumococcal meningitis.
Case No 17 - Wernig Hoffman syndrome.
Case No 19 - SUDI.
Case No 49 - Down's syndrome. Congenital heart defect.
Case No 52 - SUDI.
Case No 69 - Cardio-respiratory failure. SUDI.
Case No 75 - Acute tracheo-bronchitis. SUDI.
Case No 83 - Hyperpyrexia. SUDI.
Case No 84 - SUDI.
Case No 86 - Multiple congenital deformities.

Clinico-Pathological Group by Sex of Child

All Deaths n=87 (% of all deaths)

Clinico-Pathological Group	Male (% in each clinico-pathological group)	Female (% in each clinico-pathological group)	Total
Group A	17 (50%)	17 (50%)	34 (39%)
Group B	11 (58%)	8 (42%)	19 (22%)
Group C	7 (54%)	4 (46%)	11 (13%)
Group D	3 (50%)	3 (50%)	6 (7%)
Group E	2 (100%)	0	2 (2%)
Group F	5 (63%)	3 (37%)	8 (9%)
Group G	0	7 (100%)	7 (8%)
TOTAL	**45 (52%)**	**42 (48%)**	**87 (100%)**

Ratio of male to female is 1.1:1

Fig 11.2

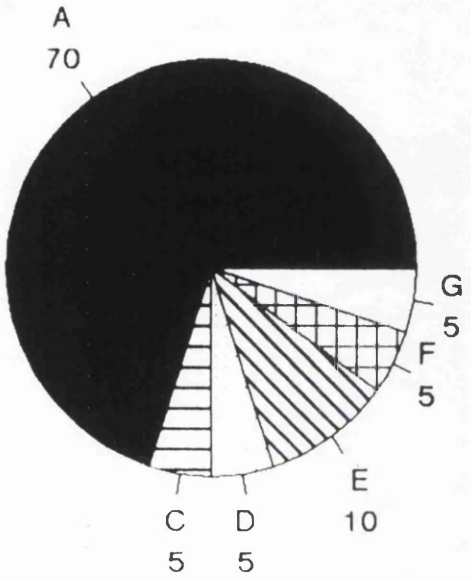
All deaths by clinico-pathological group and age group (% in each group)

Clinico-pathological group	Total Deaths	<4.3 weeks	≥4.3- <26 weeks	≥26 weeks
Group A	34	14 (40%)	17 (50%)	3 (9%)
Group B	19	0	14 (74%)	5 (26%)
Group C	11	1 (9%)	9 (82%)	1 (9%)
Group D	6	1 (17%)	5 (83%)	0
Group E	2	2 (100%)	0	0
Group F	8	1 (12.5%)	4 (50%)	3 (37.5)
Group G	7	1 (14%)	6 (86%)	0
TOTAL	**87**	**20 (23%)**	**55 (63%)**	**12 (14%)**

Fig 11.3

CLINICO PATHOLOGICAL DEATHS BY AGE GROUP

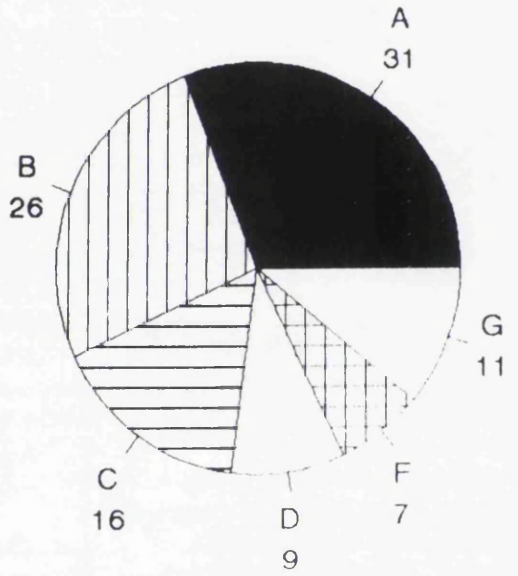
Early Deaths
(< 4.3 weeks)



% in each group

n = 20

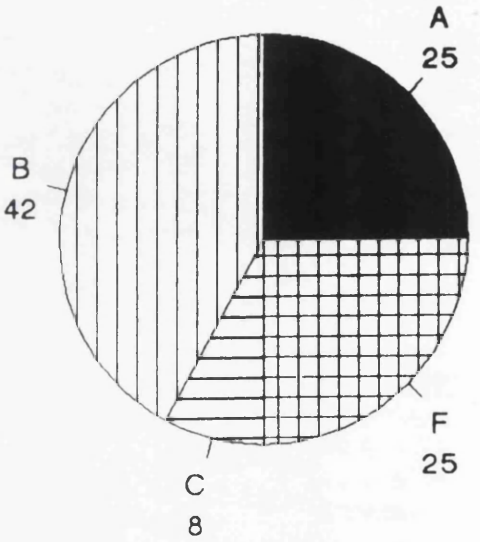
Common Age of Death
(>= 4.3 and <= 26 weeks)



% in each group

n = 55

Late Deaths
(>= 26 weeks)

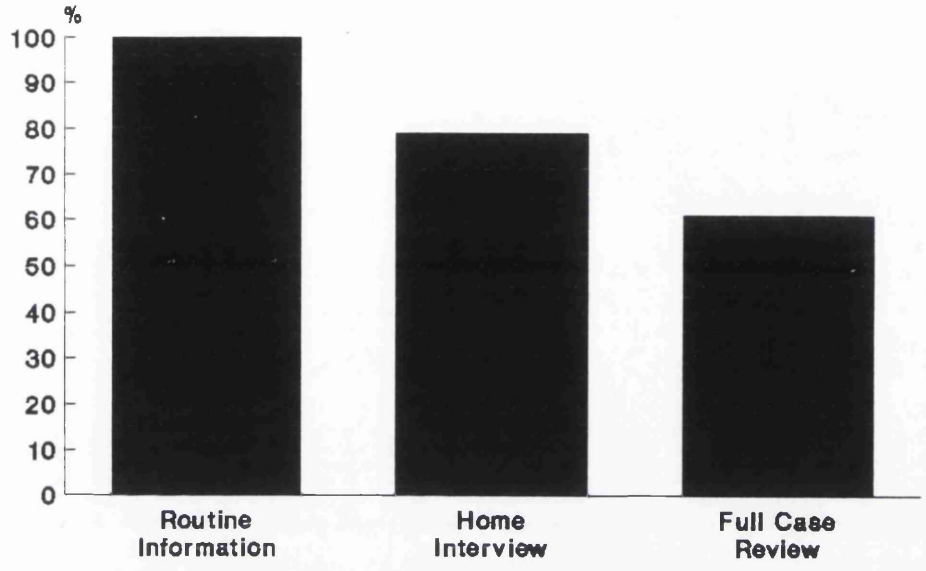


% late deaths

n = 12

Fig 11.4

Case Review Status (n=87)
Source of Information



Note: 87 = 100%
Fig 11.5

Reasons for Incomplete Case Review
n = 87

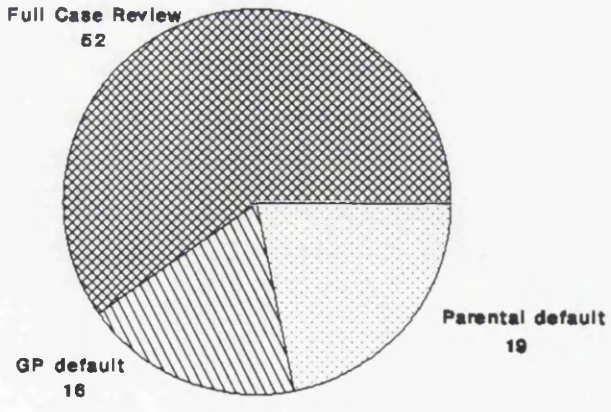


Fig 11.6

Primary case data. Total Deaths (n = 87)
Case review status (%)

Clinico Pathological Group of Death	Total Number of Deaths	Case Notes Available	Case Notes and Home Interview Available	Full Case Review Available
A	34	34	22 (65%)	9 (26%)
B	19	19	19(100%)	17 (89%)
C	11	11	10 (91%)	10 (91%)
D	6	6	5 (83%)	4 (67%)
E	2	2	1 (50%)	1 (50%)
F	8	8	6 (75%)	5 (62.5%)
G	7	7	6 (86%)	6 (86%)
Totals	**87**	**87**	**69 (79%)**	**52 (60%)**

Fig 11.7

Adverse Factors - Source of Information

All Deaths (n = 87)

	Source: Routine Information	Source: Home Interview	Source: Case Discussion	TOTAL
Non actionable adverse factors	147	19	34	200
Actionable adverse factors	36	26	63	125
ALL ADVERSE FACTORS	**183**	**45**	**97**	**325**

$\chi^2 = 63.14$

2 degrees freedom

$p = 0.000$

Fig 11.8

ANALYSIS OF ALL ADVERSE FACTORS AND SOURCE OF INFORMATION IN THE CONFIDENTIAL REVIEW INTO POST PERINATAL DEATHS (ACTIONABLE FACTORS)

ALL DEATHS (n=87)

ADVERSE FACTOR		ROUTINE INFORMATION	HOME INTERVIEW	CASE DISCUSSION	TOTAL ADVERSE FACTORS (actionable)
I	Medical History (severe)	45	0	0	45(0)
II	Defects (professional)	5(5)	1(1)	7(7)	13(13)
	Defects (inter-agency)	1(1)	3(3)	15(15)	19(19)
	Defects (hosp-to-home)	1(1)	3(3)	5(5)	9(9)
	Defects (GP-to-hosp)	0	0	2(2)	2(2)
	Defects (home-to-hosp)	0	2(2)	1(1)	3(3)
	Defects (follow-up)	0	1(1)	4(4)	5(5)
	Defects (DNA appts)	9(1)	0	0	9(1)
	Defects (no PM report)	7(6)	1(1)	1(0)	9(7)
	Uncooperative (with service)	2(0)	1(1)	3(0)	6(1)
	Uncooperative (with GP team)	1(1)	3(3)	4(0)	8(4)
III	Parent (inexperienced/low IQ)	2(0)	2(2)	5(3)	9(5)
	Parent (incompetent/low expect.)	2(1)	6(4)	8(5)	16(10)
	Parent (mum was in care/deprived)	5(0)	4(0)	1(1)	10(1)
	Parent (dad was in care/deprived)	2(0)	0	0	2(0)

Fig 11.9 continued overleaf

	ADVERSE FACTOR	ROUTINE INFORMATION	HOME INTERVIEW	CASE DISCUSSION	TOTAL ADVERSE FACTORS (actionable)
IV	Lifestyle (XS alcohol)	4(0)	1(0)	0	5(0)
	Lifestyle (XS tobacco)	12(12)	0	2(2)	14(14)
	Lifestyle (uses drugs)	2(0)	0	1(0)	3(0)
V	Unsupported (mum)	5(0)	3(0)	6(4)	14(4)
	Unsupported (by co-habitee)	2(0)	1(0)	4(0)	7(0)
VI	Mum (anxiety/depression)	4(0)	0	4(1)	8(1)
	Mum (personality disorder)	0	0	1(0)	1(0)
	Mum (overdose history)	2(0)	0	1(1)	3(1)
	Mum (psychiatric history)	5(0)	0	0	5(0)
VII	Dad (anxiety/depression)	1(0)	0	1(0)	2(0)
	Dad (personality disorder)	0	0	0	0
	Dad (overdose history)	0	0	0	0
	Dad (psychiatric history)	1(0)	0	0	1(0)
VIII	Family stress (acute)	3(0)	5(1)	4(3)	12(4)
	Family stress (chronic)	11(0)	1(0)	2(1)	14(1)
IX	Demography (poor housing)	10(4)	1(0)	6(3)	17(7)
	Demography (poor finances)	5(1)	0	0	5(1)
	Demography (unemployed)	6(0)	0	0	6(0)
X	Bonding (poor)	1(0)	1(0)	1(0)	3(0)
XI	Family (police involved)	4(0)	0	1(0)	5(0)
	Family (child protection register)	1(0)	0	0	2(1)
	Family (violent household)	3(0)	0	2(0)	5(0)
	Family (Social Services involved)	6(0)	0	0	6(0)
XII	Twins	6(0)	0	0	6(0)
XIII	Previous Child Death (of mother)	3(1)	0	0	3(1)
	Previous Child Death (of 2nd deg.)	0	1(0)	0	1(0)
XIV	Apnoea Attacks (previous)	2(0)	0	1(1)	3(1)
XV	Position at Death (face down)	2(2)	4(4)	1(1)	7(7)
	Position at Death (in mother's bed)	0	0	2(2)	2(2)
	Nothing Significant Found	N/A	N/A	N/A	5(4)
	Incomplete Survey	N/A	N/A	N/A	16(21)
TOTAL ADVERSE FACTORS		183(36)	45(26)	97(63)	325(125)

Fig 11.9

Analysis of all deaths (n=87) by clinico-pathological group.

Clinico-Pathological Group	Total Deaths	Non-Actionable Adverse Factors	Actionable Factors	Actionability Ratio
A	34	51	19	0.4
BCD	36	93	78	0.8
EFG	17	56	28	0.5
TOTAL	**87**	**200**	**125**	**0.6**

Fig 11.10

Clinico-Pathological Group by Final Preventability Grouping

Clinico-Pathological Group (No)	Inevitable	Elements of Preventability	Do Not Know
A deaths (34)	34		
B deaths (19)		17	2
C deaths (11)	1	1	9
D deaths (6)			6
E deaths (2)		2	
F deaths (6) (+2 subjudice)		7	1
G deaths (7)		2	5
Total 87	**35**	**29**	**23**

Fig 11.11

Preventability Grouping and Actionable Factors

	Total Deaths	Non-Actionable Adverse Factors	Actionable Factors	Actionability Ratio
Inevitable	35	55	20	0.4
Elements of Preventability	28	99	60	0.6
D/K	24	46	45	1
TOTAL	**87**	**200**	**125**	**0.6**

Fig 11.12

Full Case Review (n = 52). Actionability Ratio - Clinico-Pathological Group

Clinico-Pathological Group	Total Deaths	Total Deaths Full Case Review	Non-Actionable Adverse Factors Source Case Discussion	Total Actionable Factors Source Case Discussion	Numbers of babies with Actionable Factors Source Case Discussion
A	34	9	4	8	7
BCD	36	31	25	42	22
EFG	17	12	5	13	8
Total	**87**	**52**	**34**	**63**	**37**

Fig 11.13

Fig 11.14 (part 1)

The Source of Adverse and Actionable Factors at Home Interview and Case Discussion (%)

Source of Information	Number of Cases	Number of Previously Unknown Adverse Factors Found	Number of Previously Unknown Actionable Factors (% of new adverse factors)	Actionable Factor/Case Ratio
Home Interview	69	42	23 (55)	0.3
Case Discussion	52	97	63 (65)	1.2

Fig 11.14 (part 2)

The Source of Adverse and Actionable Factors at Home Interview and Case Discussion (%)

	Source Case Discussion	**Source Home/Interview**
Actionable Adverse Factors	63 (65)	23 (55)
Non Actionable Adverse Factors	34	19

Odds ratio of finding new actionable factors at case discussion is 1.53 (95% ci, 0.69, 3.41)
$\chi^2 = 1.28$, $p = 0.26$

Full Case Review (n = 52). Actionability Ratio - Final Preventability Group

Preventability Group	**Total Deaths**	**Total Deaths Full Case Review**	**Non-actionable Adverse Factors - source Case Discussion**	**Total Actionable Factors - source Case Discussion**	**Numbers of babies with Actionable Factors - source Case Discussion**
Inevitable	35	10	4	8	7
Elements of preventability	29	24	24	37	19
D/K	23	18	6	18	11
Total	**87**	**52**	**34**	**63**	**37**

Fig 11.15

Section 12

NORTH AND SOUTHERN DERBYSHIRE - A PARALLEL STUDY BY CONFIDENTIAL REVIEW 1987-88

Introduction

The differences in post perinatal mortality rates between North and Southern Derbyshire were a matter of curiosity and concern (Figure 6.4, p45). Court[3], Black[2] and others have reported regional differences and in Derbyshire the differences within the County.

The Trent Study[16] (refer to Section 6, p40) confirmed the differences but there was insufficient detail to explain why the post perinatal mortality rates in Southern Derbyshire were significantly above the North Derbyshire and national averages. Those in the North remained below the National averages from 1982 (Figure 6.4, p45). I set up a parallel study by confidential review into all post perinatal deaths which was undertaken by North and Southern Derbyshire over a period of two years.

Each review was undertaken using the method described in Section 11, p93.

The aims of the study were:-

1. To compare the deaths in each District by clinico-pathological diagnosis.
2. To compare the deaths using a standardised scale of preventability.
3. To relate each death to service, environmental, psychological and social factors which may have influenced the events leading to death.
4. To establish the geographical location of each death by electoral ward.
5. To identify each death by electoral ward relating to the Jarman scores of deprivation of that ward.

Methods

This study was set up in collaboration with the Community Paediatrician in North District. The protocol for the study was discussed and agreed (refer to Section 11, p93). To enable the comparisons to be made under standard test conditions the following points were agreed:-

1. The study would take place over a two year period 1987/88.
2. A senior doctor, independent of the care of any of the cases, would be coordinator of the study.

3. Professor J L Emery would be adviser to the study.
4. In Southern Derbyshire a named Senior Health Visitor or a Senior Clinical Medical Officer would undertake home visits to the bereaved families.
5. In North Derbyshire a named Senior Clinical Medical Officer or Clinical Medical Officer would undertake home visits and data collection.
6. A Senior Clinical Medical Officer from Southern Derbyshire would attend all case discussions in both Districts as an independent observer to ensure that the reviews had been undertaken in the same fashion in both Districts.
7. All cases would be reviewed at the end of the study and the clinico-pathological findings would be verified by the team of investigators, the coordinator and the adviser to the study.
8. The results would be made available to both District Health Authorities.

The method of review was based on the Sheffield model[204,253] and was the same as that devised in Southern Derbyshire (refer to p93). The classification of deaths was similar[93,253] (Figures 7.10 p60 and 7.11 p61). The deaths were noted by registrable cause and using the International Coding of Diseases (ICD9) were grouped by diagnosis. The deaths were further categorised by scale of preventability into seven clinico pathological groups (p91).

The study commenced in both Districts with the first death registered in 1987 and finished with the last registered death for 1988. The case reviews were instituted following direct notification of death.

Parental consent was obtained before the review procedure commenced. If this was not obtained, all available case notes were reviewed by the investigating team. In North District a home visit was arranged and parental consent to conduct the review was obtained at the time of the visit. In the South the family Health Visitor during the course of her bereavement visits to the family, explained why the review was being undertaken and obtained parental consent when she was sure the parents understood the implications. The completed questionnaires were returned to the coordinator in the south and to the investigating Senior Clinical Medical Officer in the north. To complete the review process, case discussions were arranged. If the family doctor did not participate, the case discussion was undertaken by the coordinator and the other professionals who had been involved with the case. Each case discussion was chaired by the coordinator in the South and by the Senior Clinical Medical Officer in the North. The independent observing Senior Clinical Medical Officer attended all the case

discussions. The parents were not invited. The sequence of events during case discussion was the same as previously described, p94. At the end of the case discussion the chairman and the observing Senior Clinical Medical Officer completed the summary matrix of adverse factors present and classified the death into one of the clinico-pathological groups A-G.

On completion of the review the findings were discussed with the families. At the end of the two year study all the deaths were reviewed by the teams and the clinico-pathological classifications were verified. A listing of all adverse medical and social factors associated with each case was made as in the Exeter model of confidential enquiry[178]. The Jarman scores of deprivation[19,20,21,254] were calculated for each electoral ward in both Districts and each death was related to the ward where the family had resided at the time of the child's death.

Finally, when all the facts had been analysed the deaths were further classified into five groups of preventability after birth.

1. Inevitable
2. Probably inevitable
3. Possibly preventable
4. Probably preventable
5. Not known

Results

The groups of preventability are summarised on p91. 'A' deaths were probably not preventable after birth. 'B-G' deaths were those deaths which in their clinico pathological groupings appeared to have some degree of preventability. During the series under study there were 8,802 births in North Derbyshire and 14,241 in the South (Figure 12.1, p127). In North Derbyshire there were 42 post perinatal deaths, 18 presenting as cot deaths. In Southern Derbyshire there were 87 deaths with 47 presenting as cot deaths. In Southern Derbyshire there were nine cases where there was insufficient information to categorise deaths by possible preventability. In North Derbyshire there were no such cases.

The total number of post perinatal deaths was higher in Southern Derbyshire than in the North there but was no significant difference between the two Districts 1987/1988.

	Post perinatal Deaths	Live children
North Derbyshire	42	8,760
Southern Derbyshire	87	14,154

Relative Risk = 0.78 [95% C.I. 0.55 - 1.13] $X^2 = 1.52$ $p = 0.22$

The case review status varied between the South and the North. General Practitioner attendance at case discussion being 61% in the South and only 36% in the North. Home interviews were completed in 79% of cases in the South and in 98% in the North.

When the deaths were categorised into their clinico-pathological groupings the profile of deaths was different in the two Districts, the main difference being in the deaths where potentially treatable conditions had been present prior to death ('B' deaths), (Rates 0.46 per 1,000 live births in the North compared with 1.3 in the South). Further analysis of all babies who had died as cot deaths emphasised the differences between North and South, the proportion of 'B' deaths being 17% and 34% respectively (Figure 12.1, p127).

Adverse Social Factors

The factors mitigating against a child were analysed under 34 headings and subdivided further into 12 subgroups (Figure 12.2, p128). These factors were related to the clinical groupings on the 'A-G' preventability scale (Figure 12.3, p129).

Adverse medical factors were confined to deaths in categories 'A' and 'B' which were the only deaths where there had been direct medical involvement. In 13 of these cases there had been delay in medical treatment.

Non cooperation with medical services was seen in all deaths except in group 'A'.

Inexperienced parenting predominated in the 'F' deaths, while defective modelling for parenting was evenly distributed amongst the possibly preventable groups of 'B-G'. Heavy drinking and smoking was associated with category 'D' deaths, the so called "idiopathic" cot deaths which contained one of the three families where there were drug problems. This family had received a carefully designed package of care but the mother had changed her family doctor three times and the medical care of the child was supervised by the hospital paediatrician.

Unsupported mothers occurred in 29 of 129 families, but only in one of the 'A' deaths. Overdose in a parent was found in one of the 'A' deaths and five of the other deaths which were categorised in the possibly preventable after birth grouping 'B-G'.

Depression and possible psychological disturbance was a common group of adverse factors, particularly in the mothers of the 'F' group of deaths where filicide was suspected. Poor housing was common to all groups except 'E', similarly financial problems. The numbers of unemployed parents in this series was remarkably small.

In the group where poor bonding had been observed, half the cases came under the category 'F' deaths which also contained the three families where there had been a previous cot death. Social Services involvement was prevalent in both 'B' and 'F' groups.

The presence of twins (four baby deaths) was only associated with the cot death groups of 'C' and 'D'.

Adverse factors/Case ratio

Figure 12.3 shows the numbers of adverse factors per case. The factor/case ratio was worked out for each District and for each clinico-pathological category of death. Taking both Districts together, in the A grouping there were 0.29 adverse factors per case, both Districts having a similar factor/case ratio (0.35 in the North and 0.25 in the South). In the possibly preventable after birth grouping ('B-G' deaths) in the 'B', 'E' and 'F' groupings, the adverse factor/case ratio was higher in Southern than in North Derbyshire. The greatest concentration of adverse factors was found in groups 'E' and 'F'.

Preventability

At the final review of each case an assessment was made to determine whether there were any points where intervention might have prevented death. All the cases were divided into five groups of preventability (Figure 12.4, p129).

1. Inevitable death
2. Probably inevitable death
3. Possibly preventable death
4. Probably preventable death
5. Do not know

From a total of 129 deaths it was uncertain whether 33 of them had any element of preventability, whereas 40 deaths had avoidable factors.

There were considerable differences in the clinico-pathological groups. In the 'A' group no element of preventability after birth was detected. In the 'B' grouping nearly all the deaths had elements of preventability, the reverse was true of the 'D' group, the so called idiopathic cot deaths. In the 'E' and 'F' groups all deaths, where the information was complete, were judged preventable. In the 'G' group, although the enquiries had not been complete, in two of these nine deaths avoidable factors had been recorded. The 'B', 'E' and 'F' group of deaths showed a similar pattern of avoidable factors.

Figure 12.5 compares North with Southern Derbyshire by groups of preventability. In 'A' deaths, which were judged inevitable after birth, the absence of adverse factors was the same in both Districts (81% and 83% respectively). Where deaths had been judged to have some element of preventability ('B-G' groupings) Southern Derbyshire showed a greater proportion with definite adverse factors (64% compared to 36%) but the types of adverse factors were similar in both Districts.

The distribution of Jarman deprivation scores by electoral ward is depicted in Figure 12.6. In both Health Authorities the most prevalent range of scores are in the ranges of -10 to 0, but Southern Derbyshire contains a wider range of deprivation scores than does North Derbyshire. In Southern Derbyshire there were eight electoral wards with Jarman scores greater than 20, while in North Derbyshire there were none (Figure 12.6, p130). The average Jarman score for all electoral wards in North Derbyshire was -14.2 and that in Southern Derbyshire -4.2, North Derbyshire having less deprivation than Southern Derbyshire as calculated by Jarman score (Figure 3.1, p24). There was an association of post perinatal mortality with high Jarman score (Figure 12.7, p131).

Discussion

The Department of Health has produced a series of performance indicators for Districts[145,159] but does not indicate what is a permissible deviation from the National average before enquiry and action should take place. The post neonatal mortality rate is used as one such indicator of performance in the field of child care (refer to p79).

Over the past decade the post perinatal mortality rate in Southern Derbyshire has been higher than the national average and that in North Derbyshire (Figure 6.4, p45). In the two years under study the post perinatal mortality rate in North Derbyshire was 4.77

per 1000 live births and that in Southern Derbyshire 6.1 per 1000 live births. The differences were not statistically significant (p120) but had been present for ten years. The initiative for this study was taken by Southern Derbyshire because of these relatively high post perinatal mortality rates.

When the study commenced, the general practitioners were not reporting their cot deaths immediately to the Department of Child Health, but as it progressed, early reporting improved.

The time interval between gaining parental consent and home interview varied from one month to several months after the child's death depending on when the parents felt able to cope. The method of obtaining consent appeared to be more effective in the North; in only one case home interview was refused as the family had left the district. There were more complete case reviews in the South where the coordinator was well known. General practitioners attended 61% of the case discussions compared to 36% in the North.

In both districts two reports were received from the local pathologists but they did not attend the case discussions in either district.

When all the deaths were grouped into the clinico-pathological groupings 'A-G', the deaths in the probably not preventable group (A group) were similar in both the North and the South and appeared to be randomly distributed throughout the population (Rates 2.16 and 2.3 per 1,000 live births respectively). This distribution occurred despite all the non caucasian deaths occurring in Southern Derbyshire[4,70,103,255] (Figure 3.1, p24).

The overall differences in mortality rates between the two Districts lay in the possibly preventable groups of deaths (B-G), and in particular in the B group of deaths which are those deaths which appear to have the most elements of preventability[151]. The 'B' death rate was 1.3 per 1,000 live births in Southern Derbyshire and 0.46 in the North District. The differences in the 'D', 'E' and 'F' groups of death were less marked. Statistical analysis was not undertaken between the clinico-pathological groups of death as the numbers were small and testing would not have ruled out occurrence by chance.

When the 'A' cases were compared with all other clinico-pathological groups from the point of view of the presence or absence of adverse medical and social factors, many differences were noted both within the clinico-pathological groups and between the two

Districts. The adverse factors found were similar in both Districts, including the medical factors where there had been delay in medical treatment. In these cases either the parent had been slow to recognise the severity of the condition, or the professionals involved had not referred the child for further investigation/treatment[62,150,233]. The difference between the death groups and the two districts indicated that it was not a particular group of adverse factors which had resulted in more deaths in Southern Derbyshire. The differences lay in the numbers of children who had adverse factors mitigating against them.

At confidential review a wide range of adverse factors was found (Figure 12.3, p129). Overall the factor/case ratios were similar in the North and the South, but different clinico-pathological groups had different ratios, the highest factor/case ratios being in the E and F group (Figures 12.4, 12.5 & 12.6, p129-130). The A deaths were associated with 0.35 adverse factors in North Derbyshire and 0.25 in the South, an overall ratio of 0.29 factors per case.

The adverse factors were related to individual deaths in each District. This was achieved by analysis of the in-depth confidential review into each case, these reviews being the only source of such detailed information. In many instances the confidential review revealed incidences where happenings in the child's life were possibly amenable to intervention.

The findings of some of the case reviews are described in Appendix 1, p166.

The Court Committee[3] acknowledged that the disadvantages of birth and early life cast "long shadows forward". It was noted that children still died from 19th century reasons because demographic and environmental changes on families had not been addressed, and the prevalence of emotional and psychiatric disorders in families was not known. Further studies have indicated that child mortality is related to many factors[2,67,228]. This parallel study by confidential review into child death supports this. There are no other reported studies to the depth of social detail of this parallel study by confidential enquiry.

The Jarman scores[19,20,21,264] were devised to identify underprivileged areas by considering factors which are thought to affect a General Practitioner's workload (refer to Section 13, p132). This study shows the association of post perinatal death with high Jarman scores of deprivation. Because post perinatal deaths in the possibly

preventable grouping are closely concerned with effects of medical care in the community[70,150,154], this association was expected. The death rate in the possibly preventable group of deaths was more than twice as high in the electoral wards with high Jarman scores greater than 30.

The probably not preventable deaths ('A') may also be related to general underprivilege as consanguineous marriages are related to some congenital malformations[103,255]. This particularly applies to Asian families. In Derbyshire over 80% of the Asian families live in Derby City and within four of the electoral wards where the Jarman scores were greater than 30. Other factors such as heavy smoking and the use of alcohol[143,144,256] commonly relate to underprivilege and stress. These factors are known to affect intrauterine growth and consequently the baby's chance of survival.

The indices measured for calculating Jarman scores for underprivilege are not directly causal to death. Adverse social factors revealed by the in depth confidential review into each case showed instances where such factors were directly contributory to some children's deaths. The numbers involved in this study were too small to say which death groups ('A-G') were associated with Jarman scores greater than 30. The present data would need to be expanded over a six year period or more for these results to have statistical significance. However, Jarman scores can be taken into consideration when investigating local post perinatal mortality rates as they have directed medical care to areas where post perinatal mortality was high.

At the final assessment, different clinico-pathological groups showed different levels of preventability in the group of children dying as sudden unexpected deaths (Figure 12.5, p130). The deaths where no preventable factors were apparent were those in group 'D', the group where there had been no significant pathology or clinical history, the so called "idiopathic" cot deaths. These deaths appeared to be randomly distributed in the general population, the rates were similar in the North and South and the adverse factor/ case ratios the same in each District.

The greatest numbers of possibly preventable deaths were in the 'B', 'E' and 'F' groups. In the 'B' deaths there were treatable factors present and these deaths should be most amenable to preventive measures. This group of deaths largely explained the different death rates in North and Southern Derbyshire.

The 'E' and 'F' deaths constituted over 10% of the deaths in the series. The preventable factors identified in these families are those associated more with the work of Social Services than with the Health Services, but at enquiry there were many instances where improved liaison between the two services would have ensured more coordinated care for the families.

In the population of Southern Derbyshire a high rate of cot death has been demonstrated in the siblings of families on the Child Protection Register[257,258] (refer to Section 14, p135). Their identification and prevention appears to lie in the multi-disciplinary field of child abuse[258,259,260,261,262,263], but further study in this field is required to establish which type of abuse in siblings is associated with cot death.

Conclusions

1. There was no difference in the rates of inevitable (A) deaths or the idiopathic (D) deaths in the two Districts.
2. The differences between the two Districts lay in the partially explained cot deaths (Group 'B' and 'C').
3. Attempts to reduce post perinatal mortality in Southern Derbyshire should focus on the partially explained cot deaths[10].
4. This study demonstrates five main uses of confidential reviews:
   i) Provides a source of data unique to each District.
   ii) Identifies the medical and social needs of individual families.
   iii) Gives a review of standards of quality in the child care services[264].
   iv) Provides the means for those involved with direct family care to be instrumental in affecting management decisions.
   v) Supplies supplementary information that is necessary for taking action on information such as that presented by Pharoah and Alberman[72,73].
   vi) Provides data based guidelines for national quality based performance indicators.

Postperinatal Deaths

North and Southern Derbyshire 1987 & 1988

		N. Derbys.	S. Derbys.
Births		8802	14241
Deaths (Rate/1000 live births)		42 (4.77)	87 (6.10)
Cot Death (Rate/1000 live births)		18 (2.04)	47 (3.30)
Total Deaths (Rate/1000 live births)			
Categories of Deaths	A	20 (2.27)	35 (2.45)
	B	4 (0.46)	19 (1.3)
	C	2 (0.23)	9 (0.63)
	D	8 (0.9)	6 (0.42)
	E	3 (0.34)	1 (0.07)
	F	5 (0.57)	8 (0.56)
	G	0	9 (0.63)
Unexpected Home Deaths (Cot Deaths)		18 (%)	47 (%)
	A	0 (0)	1 (2)
	B	3 (17)	16 (34)
	C	2 (11)	9 (19)
	D	8 (44)	6 (13)
	E	2 (11)	1 (2)
	F	3 (17)	6 (13)
	G	0	8 (17)
Other Deaths		24 (%)	40 (%)
	A	20 (83)	34 (85)
	B	1 (4)	3 (8)
	C	0	0
	D	0	0
	E	1 (4)	0
	F	2 (8)	2 (5)
	G	0	1 (3)

Figure 12.1

		Number of cases in the clinical groups							
		A	B	C	D	E	F	G	Total
		55	23	11	14	4	13	9	129
Communication defects									
1	Hospital - Home	3	2	-					5
2	GP - Hospital	1	-	-					1
3	Hospital - Home	-	1	-					1
4	Home - Hospital	-	6	-					6
5	Non cooperation with medical services		2	2	2	1	3	2	12
6	Very inexperienced parents or low IQ	1	2	1		2	6	2	14
7	Parents low/defective standard of health		3	3			2	0	8
8	In care as a child (broken home) Mother		2		1	1	2		6
9	In care as a child (broken home) Father				1	1	1		3
10	Heavy drinker (either parent)			1	4	1	1		7
11	Heavy smoker (either parent)			2	5		2	1	10
12	On drugs		1		1			1	3
13	Single unsupported mother	1	5		1	2	5	1	15
14	Co-habitating unsupported mother		4	4	3	2		1	14
15	Mother depressed/anxious	1	3	2			1	1	8
16	Personality disturbance		1		2		2		5
17	Overdose or other method		1		1		2		4
18	Has been under psychiatrist			1			2		3
19	Father Depressed/anxious						1		1
20	Personality disturbance						1		1
21	Overdose or other method	1	1						2
22	Has been under psychiatrist						1		1
23	Stress acute		1		2			1	4
24	Stress chronic (chronic illness)	1	2	3				1	7
25	Poor housing	4	5	2	0		2	2	15
26	Financial problems	2	1	1	4		3		11
27	Out of work		1	1	0			2	4
28	Poor bonding		1	1			3	1	6
29	Family involved with police	1	1	1	0		3	1	7
30	Under S.S as problem family		2	1	2	1	4	1	11
31	Member of family on N.A.I Register		2	1	1		2		6
32	Violent household		1		1	1	5	2	10
33	Twin			2	2				4
34	Previous cot death						3		3

Fig 12.2

Giving the presence of all adverse family and social factors present in North and South Derbyshire relating to death grouping

		All A	All B	All C	All D	All E	All F	All G	Total B-G
North	Total	20	4	2	8	3	5	0	22
	Adverse Factors	7	7	4	21	3	17	0	52
	Factors/ Case	0.35	1.7	2	2.6	1	3.4	0	2.3
South	Total	35	19	9	6	1	8	9	52
	Adverse Factors	9	40	23	13	9	40	24	149
	Factors/ Case	0.25	2.1	2.5	2.1	9	5	2.6	2.9
North & South	Total	55	23	11	14	4	13	9	74
	Adverse Factors	16	47	27	34	12	57	24	201
	Factors/ Case	0.29	2.0	2.4	2.4	3	4.4	2.6	2.7

Fig 12.3

The possible preventability of deaths related to death grouping.

	Inevitable	**Probably Inevitable**	**Possibly Preventable**	**Probably Preventable**	**DK**	**Total**
Group A	38	16			1	55
Group B			19	2	2	23
Group C		1	2		8	11
Group D				0	14	14
Group E			2	1	1	4
Group F			6	6	1	13
Group G			2		7	9
Total B-F		**1**	**29**	**9**	**26**	

Fig 12.4

Preventability and Adverse Social Factors

Preventability of death	Adverse Social Factors	North Derbyshire No.	%	Southern Derbyshire No.	%	Total No	%
	Absent	17	81	29	83	46	82
INEVITABLE OR PROBABLY INEVITABLE	Possibly	1	5	3	9	4	7
	Definite	3	14	2	6	5	9
	Not known	0		1		1	
	Total	**21**		**35**		**56**	
	Absent	2	18	4	14	6	15
PROBABLY OR POSSIBLY PREVENTABLE	Possibly	4	36	5	18	9	23
	Definite	4	36	18	64	22	56
	Not known	1		1		2	5
	Total	**11**		**28**		**39**	

Fig 12.5

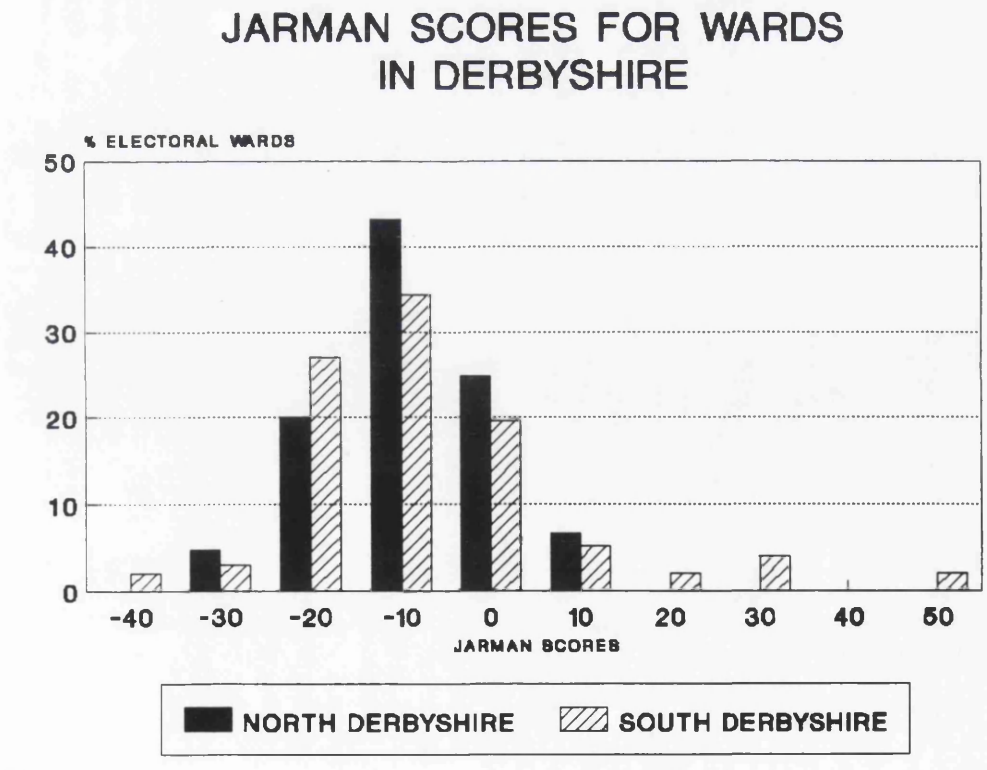


Fig 12.6

Post Perinatal Deaths related to Jarman Index in North and South Derbyshire

Social Deprivation Banding Jarman Scores	North Derbyshire			Southern Derbyshire		
	Births	Deaths	(Rate/1000)	Births	Deaths	(Rate/1000)
>50	-	-	-	950	11	(12.0)
40 - 49.99	-	-	-	-	-	-
30 - 39.99	-	-	-	1589	14	(8.8)
20 - 29.99	-	-	-	544	4	(7.4)
10 - 19.99	654	5	(7.6)	925	5	(5.4)
0 - 9.99	2788	20	(7.2)	2620	10	(3.8)
-0.01 - -10.0	3828	10	(2.6)	5128	28	(5.5)
-10.01 - -20.0	1274	5	(3.9)	1997	13	(6.5)
-20.01 - -30.0	262	1	(3.8)	238	1	(4.2)
> -30.01	-	-	-	59	1	(17.0)
Totals	**8806**	**41**	**(4.7)**	**14050**	**87**	**(6.2)**

Mortality Related to Jarman Score

Jarman Scores of Deprivation	Total live births	Total deaths	Deaths probably unpreventable after birth	Deaths possibly preventable after birth
> 30	2539	25 (9.9)	9 (3.5)	16 (6.3)
29 <	20317	104 (5.1)	46 (2.3)	58 (2.8)
	22856	129 (5.6)	55 (2.4)	74 (3.2)

Relative risk of a baby dying in an electoral ward with a Jarman deprivation score >30 is 1.93 (C.I. 1.22, 3.05) $\chi^2 = 8.99$ $p < 0.005$

Fig 12.7

Section 13

RELATION OF DEPRIVATION TO POST PERINATAL MORTALITY

Introduction

When trends in post perinatal mortality were studied (refer to Section 7, p46) an association of deprivation with post perinatal mortality was identified in Southern Derbyshire (Figure 7.9, p59).

The aim of this study was to see whether there was an association between high Jarman deprivation scores and the possibly preventable after birth death grouping.

Methods

Jarman scores of deprivation were calculated for each electoral ward in Southern Derbyshire. Numbers and rates of post perinatal deaths were calculated by electoral ward. Post perinatal deaths were categorised into the clinical groupings of preventability by death registration[93,265] cause as described on page 91. The death rates in each group of preventability were calculated by high (>30) and low (<30) Jarman scores.

Results

The data collected is depicted in Figures 13.1 and 13.2, p134.

Over the period of study there were 41,878 live births and 212 post perinatal deaths (rate 5.1 per 1,000 live births). Both the numbers and rates of death were twice as high in the possibly preventable grouping compared with the probably not preventable after birth grouping.

When the deaths in the two preventability groupings were examined in relation to Jarman under-privilege scores, the death rates in the probably not preventable after birth groupings were very similar in both high and low scoring areas. The deaths in the possibly preventable groupings showed a marked difference between the higher and lower Jarman scores.

Deaths in the deprived wards occurred at 2.3 times the rate of the lower deprived wards (Figure 13.2, p134).

Discussion

The Jarman scores were devised by considering factors which were thought to influence the General Practitioner's workload[19,20,21,266]. These scores had been adopted by Family Health Service Authorities throughout the country to weight salaries of General Practitioners working in deprived areas in an effort to compensate them for perceived extra workload and to entice them to work in difficult circumstances.

Other methods of deprivation have been devised, each method being based on different social and material factors[4,186,187,267,268,269,270]. The association of Jarman scores with medical care in the community encouraged their use in this study. It had been expected that high Jarman scores would be associated with the possibly preventable group of deaths[2,62,98,147,191,211,230,271,272,273]. This association showed consistency over time ($X^2 = 6.77$, $p = 0.009$).

Conclusion

Jarman scores can be taken into consideration where local post perinatal mortality rates are being investigated and identify areas where there are increased numbers of possibly preventable deaths.

Figure 13.1

Post Perinatal Deaths - Southern Derbyshire 1984-1989
(Groups of Preventability)

	Total Live Births	Total Post Perinatal Deaths (rate)	Possibly not preventable A deaths (rate)	Possibly preventable B-G deaths (rate)
1984	6,622	28 (4.7)	7 (1.2)	21 (3.3)
1985	6,832	39 (6.0)	11 (1.6)	28 (4.0)
1986	6,801	31 (4.6)	13 (1.8)	18 (2.0)
1987	7,172	34 (4.7)	8 (1.1)	26 (3.6)
1988	7,069	53 (7.4)	21 (2.9)	32 (4.5)
1989	7,382	27 (3.7)	12 (1.5)	15 (2.0)
TOTALS	41,878	212 (5.1)	72 (1.7)	140 (3.5)

Figure 13.2

Deaths by Preventability versus Jarman scores >30
Jarman scores <30

	Births Jarman >30	A deaths (rates) Jarman >30	B-G deaths (rates) Jarman >30	Births Jarman <30	A deaths (rates) Jarman <30	B-G deaths (rates) Jarman <30
1984	1,202	1 (0.8)	3 (2.5)	5,420	6 (1.1)	18 (3.3)
1985	1,235	1 (0.8)	13 (10.5)	5,597	10 (1.8)	15 (2.7)
1986	1,242	3 (2.4)	6 (4.8)	5,559	10 (1.8)	12 (2.2)
1987	1,250	2 (1.6)	9 (7.2)	5,922	6 (1.0)	17 (2.5)
1988	1,284	5 (3.9)	10 (7.8)	5,785	16 (2.8)	22 (3.8)
1989	1,250	0 (0.0)	6 (4.8)	6,132	12 (2.0)	9 (1.5)
TOTALS	7,463	12 (1.6)	47 (6.3)	34,415	60 (1.7)	93 (2.7)

Odds Ratio = 0.40 [95% C.I. 0.18 - 0.85] $X^2 = 6.77$ $p = 0.009$

Section 14

ASSOCIATION BETWEEN CHILDREN ON THE CHILD PROTECTION REGISTER AND SUDDEN INFANT DEATH SYNDROME (SIDS) IN SOUTHERN DERBYSHIRE

Introduction

The Area Social Services Department maintains a Child Protection Register on behalf of the Area Child Protection Committee[267] and a post perinatal death register is maintained in the Department of Child Health. Many family names appeared on both registers.

The aims of this study were to see if there was an association between child abuse and Sudden Infant Death in Southern Derbyshire[258,259,261,262,263,274,275,276].

Methods

The study was undertaken over a period of two years. The Post Perinatal Death Register was examined and the names of all the children who were born had died and registered as Sudden Infant Death Syndrome were noted 1987/88. The Child Protection Register was examined on a monthly basis. Change in the registration status of children was observed. Name changes, aliases and changes of addresses were noted for all children born and registered at any time during the period of study. The numbers of siblings and half siblings in the abusing families were noted.

Children who had been born and died registered as Sudden Infant Death Syndrome were related to those who had been born and registered on the Child Protection Register during 1987/88. By this cross referencing it was ascertained which children dying registered as Sudden Infant Death Syndrome came from abusing families.

Results

The data collected are displayed in Table 14.1 and 14.2.

There were 14,334 births 1987/88. One hundred and twenty seven children 0-2 years (rate 8.9 per 1,000 0-2 years) were registered on the Child Protection Register and there were five post perinatal deaths in children born during this period. Three of these deaths were registered as Sudden Infant Death Syndrome.

There were 39 deaths registered as Sudden Infant Death Syndrome (rate 2.7 per 1,000 live births). A quarter of these came from abusing families. A further seven of the 127

children on the Child Protection Register came from families where there had been a sudden infant death prior to 1987.

The numbers of siblings and half siblings of the 127 children on the Child Protection Register was 191.

Discussion

A child who is abused or is at serious risk of abuse is registered on the Child Protection Register maintained by the Social Services Department, after a multi-disciplinary case conference[257,277]. Each case conference is chaired by a Senior Social Worker and those present always include the Social Worker involved with the case, his/her line manager, the paediatrician, the Health Visitor/school nurse and her line manager, and the community doctor. The family doctor is always invited to attend[278]. Others attending may include a senior officer from the Police department, a school teacher and a representative from the legal department.

Parents are informed of the proceedings and possible outcome, but are not invited to attend. When the case has been discussed the decision is made whether the child should be registered. On registration a key worker is appointed from the Social Services department and recommendations are made for the future care of the child. The key worker has the responsibility of ensuring that all the recommendations made at case conference are carried out. No child can be deregistered without the full consent of all members of the case conference. Once a child has been deregistered his name is removed from the Child Protection Register and all previous record of his registration is destroyed.

At the time of study there were six categories of registration[257]:

1. The child has received physical injury.
2. Neglect.
3. A family with a child under two years at risk of physical injury or neglect.
4. A child in a household with a known abuser.
5. Sexual abuse.
6. Any other.

The Area Child Protection Committee (ACPC) was chaired by the Director of Social Services and members are required to review all cases of child abuse to ascertain that the correct child protection procedures have been followed for each case.

There was an average of 1.5 siblings and half siblings per child on the Child Protection Register. These included all five babies who had died during the study period, but had excluded those who had died prior to the start of the study. In the local population the average SIDS rate during the study was 2.7 per 1,000 live births (39 SIDS), we would thus have expected 0.5 deaths to have occurred in the 127 children on the Child Protection Register. The finding was three SIDS which calculates at a rate of 23.6 per 1,000 children 0-2 years registered on the Child Protection Register.

An outline of the five deaths related to children on the Child Protection Register occurring during the period of study is as follows:-

Post perinatal deaths in siblings of children on the Child Protection Register 1987/88.

1. SIDS 1987. This child had four siblings on the Child Protection Register category 3, the reason being that the mother was a drug abuser. A new baby born October 1988 was registered category 3 because of his mother's drug habit.

2. SIDS 1988. This was the second child of a mother whose first baby had died as a SIDS in 1986. The baby was registered, at the age of four months, category 3 as the mother was in a mother and baby home under the care of the Social Services because of her multiple psychological problems and her apparent inability to care for her child.

3. SIDS 1987. This was the fourth child in a family of five children. The family were known to the Police department.

4. A baby and toddler had been left unattended for eight hours. The returning parent found the baby dead in his cot. The toddler was subsequently registered category 6 for three months and within a few weeks of deregistration was accidentally overlaid in 1988 by his father who was under the influence of alcohol.

5. A child was killed October 1987 by his father who was on treatment for depression at the time. A half sibling was registered category 3 at birth, November 1988, because of the mother's ineptitude as a parent. The father was imprisoned for the murder of his child. The half sibling was the child of the mother and her new cohabitee.

Regarding registered families who had had sudden infant deaths prior to the period of study, the findings were as follows:

1. A twin was found suddenly and unexpectedly dead in 1981, the surviving twin is still alive and is one of four siblings. All four children were registered on the Child Protection Register; three in category 3 and one in category 1. The children were registered in 1987.

2. A SIDS in 1984. There were two siblings who were registered in 1987, one in category 1, the other in category 4. These children were deregistered in September 1988.

3. A SIDS in 1984. There were three siblings, only one of whom was registered at birth in 1988 in category 3.

4. A SIDS in 1985. The mother said that she had smothered the child and had been charged with filicide at the time but there was insufficient evidence 294-296. A subsequent sibling was registered category 3 at birth in August 1987.

5. A SIDS in 1981. There were four siblings who had been registered prior to this death in category 3. A new baby was born in March 1987 and was registered at birth in category 3 and deregistered in January 1989.

6. A SIDS in 1979. There were five siblings, three of whom had been previously registered category 4. These children are still in care. A new baby was born in September 1988 but was not registered.

7. A SIDS in 1986. The mother of this child had had severe social and psychological problems. A new baby was born in 1988 and registered at birth in category 3. This child died registered as SIDS while still on the Child Protection Registration.

The histories of the families where there had been a sudden infant death previous to 1987 show that SIDS was not a reason per se for registering a child on the Child Protection Register. In most cases the siblings were already registered for other reasons, but SIDS may have been a contributory factor for registering the baby in case 3 where there was suspicion about the level of care for the new baby. In case 4 there had been formal charges against the mother but filicide was not proven. The baby born

in 1987 was thus judged to be at risk of abuse and was registered in category 3 soon after birth. This baby remains on the Child Protection Register.

SIDS and child abuse are found in all social strata of the population[279,280], thus the possibility that children on the Child Protection Register are of a different social or ethnic group from those who died as SIDS is unlikely as the social profiles of the two groups was similar. These families may well have similar adverse factors mitigating against them[281].

It was difficult to find all the siblings in the abusing families due to frequent changes of names and addresses. This was only done by checking Child Protection Register at regular intervals. No difficulties were encountered in following up the children who had died as the Post Perinatal Death Register was updated on a regular basis within the Department of Child Health.

Conclusions

There was a definite association between child abuse and cot death in Southern Derbyshire during the two year study.

A point prevalence study of the Child Protection and Post perinatal Death Registers was undertaken to substantiate the findings. This identified 1:60 chance of a sibling of a child on the Child Protection Register presenting and registered as SIDS[258]. These findings were published in "Child Abuse & Neglect" 1991. A copy of this paper is included.

Addendum

The Child Protection Group of the British Paediatric Association has now set up a ten district prospective study based on the method used in Southern Derbyshire described in this section. This study commenced January 1993 for an initial period of two years.

ANALYSIS OF 14334 BIRTHS 1987-88 BY CATEGORY OF ABUSE

AND DEATH REGISTRATION CAUSE

Category of abuse	Total no of children registered <2yrs (Rate/1000 0-2yrs)	Number of siblings of registered children	Number of previous SIDS in abusing families	Numbers of deaths in siblings of registered children 0-2 yrs
1. Child has received physical injury	14 (1)	14	2	0
2. Neglect	9)0.6)	15	0	0
3. Family with child <2yrs at risk of abuse	46 (3.2)	58	5	3
4. Child in household with known abuser	41 (2.9)	79	1	0
5. Sexual abuse	0	0	0	0
6. Other causes	17 (1.2)	25	0	2
Totals	**127 (8.9)**	**191**	**7**	**5**

Fig 14.1

ASSOCIATION BETWEEN CHILDREN IN THE CHILD PROTECTION REGISTER

AND SUDDEN INFANT DEATH SYNDROME (SIDS)

	SIDS	OTHER CHILDREN	TOTAL NUMBER OF CHILDREN 0-2 YRS
Children on the Child Protection Register 0-2 yrs	3	124	127
Children not on Child Protection Register 0-2 yrs	36	14298	14334
Total	**39**	**14422**	**14461**

Relative Risk 9.41 (C.I. 2.93, 30.15). Fishers Exact Test 2 tailed p=0.0005

Fig 14.2

Section 15

GENERAL DISCUSSION

The investigations described in this thesis demonstrate the steps taken to find the reasons for high post perinatal mortality rates in Southern Derbyshire and to identify adverse socio-economic, psychosocial and service factors which may have contributed to the child's death. Their progress culminated in the belief that a confidential study into each post perinatal death is an essential prerequisite of any investigation of this nature (refer to Section 11, p93). The relationship between North and Southern Derbyshire (refer to Section 12, p117) was of particular interest as the Districts differ both geographically and demographically and these differences were reflected in the post perinatal mortality rates (Figure 6.4, p45).

The questions addressed were:-

Why in Southern Derbyshire was there a higher infant mortality rate than in North Derbyshire and England and Wales?

What detail of enquiry was required to reveal the reasons for these differences?

Which of the adverse events in the child's life, preceding death, were amenable to action?

The report of the Area Working Party of 1978 (refer to Section 5, p34) was grossly inadequate, revealing the difficulties of study by retrospective case record review. However, a multidisciplinary approach to the problem was introduced in the District.

The Trent Multi District Confidential Enquiry into Perinatal and Infant Mortality 1983/84, described in Section 6, revealed that many post perinatal deaths had possibly preventable factors. This study also revealed its own deficiency of lack of depth of enquiry and posed more questions than it answered. The detail of the case studies was insufficient, and although possibly preventable factors were identified, more general information was required to explain the high post perinatal death rates in the District. Southern Derbyshire appears to be a microcosm of England and Wales (Figure 3.1, p24) and as such, should have average post perinatal mortality rates compared to the nation.

The data collected on all deaths in the District over an seven year period showed that the well known epidemiological factors associated with post perinatal deaths applied equally in Southern Derbyshire as elsewhere in the country (refer to Section 7, p46), but this did not supply the answers to the question as to what action should take place locally.

On analysing all data relating to deaths in the District, the critical question was that of possible preventability (refer to Section 10, p87) It was recognised that concepts of preventability are relative. Conditions where there are variable rates between places and different ethnic groups were considered, as well as those where there are known treatments. All children registered as Sudden Unexpected Death in Infancy were included in the possibly preventable grouping in the knowledge that this would include approximately 15% of cot deaths, which under the Beckwith[12] rubric are unpreventable. This group is not identifiable from registration data. Contrary to the belief that the diagnosis of Sudden Infant Death Syndrome increasingly replaced the diagnosis of infection after 1979, in Southern Derbyshire this applied to only 25% of the deaths in the District. However, the Sudden Infant Death Syndrome diagnosis had replaced the diagnosis of all children whose deaths had previously been ascribed to trauma (Figures 7.23 p69 and 7.25 p70). The concept of Sudden Infant Death Syndrome had been introduced by the Bergman[12] group in Seattle to replace suffocation but not trauma.

Over an 18 year period the local post perinatal mortality rate showed a slow decline, and within this finding the rate of post perinatal deaths which were ascribed to probably not preventable causes, showed a progressive and slow decline. From the clinical point of view the importance of the findings was the rate of deaths ascribed to possibly preventable causes showed little or no decrease (Figure 7.13, p63). This was also reflected in the relative rise in the proportion of deaths diagnosed as Sudden Infant Death Syndrome (Figure 7.24, p69). This held important implications for the primary care services.

The study of investigation of post perinatal deaths by confidential reviews used a more comprehensive method of assessing all the events surrounding the child's life and death, and the interplay of the family dynamics. This was the first local study where parents were visited at home and asked to offer information about the circumstances surrounding their child's life and death. It was also the first time that General Practitioners in the District had actively participated in enquiries and at a venue of their own choice. At these confidential reviews much previously unknown information

emerged which, had it been known, could in some instances, have prevented a child's death. The new information, often revealed at case discussion, was mostly related to standards of care within the primary care teams and was identified by the teams themselves, enabling them to agree and implement improved standards of care. Information made available from the confidential reviews was recognised as being the basis of a good method of improving quality standards within the child care services. A Senior Clinical Medical Officer is now allocated for one session per week, on a permanent basis, for the task of co-ordinating the process of case review and to act as chairman for all case discussions in the District.

When this system of confidential reviews was applied in the two year study of post perinatal mortality in both North and Southern Derbyshire (refer to Section 12, p117) the differences in the causal pattern of post perinatal deaths between the two Districts was exposed. The classification of post perinatal deaths into probably not preventable and possibly preventable after birth groups that the study facilitated, showed that the probably not preventable death rates were the same in both Districts, as were the completely unexplained (idiopathic) cot deaths (Figure 12.1, p127). The differences lay in the partially explained and possibly preventable cot deaths.

When individual clinico-pathological groups of death were studied, the background adverse factors were the same in both Districts. The differences lay in the number of babies dying associated with these adverse factors, in the possibly preventable after birth group of deaths. These adverse factors were equally preventable in the North and the South but there were more children in the South with these factors.

When the deaths were analysed into their different clinico-pathological groupings and by the presence of actionable social and medical factors, it was found that the categories of possibly preventable deaths had more actionable factors per case than the probably not preventable group of deaths. The greater proportion of avoidable factors per case was associated with the partially explained cot deaths and the possible filicides (Figures 12.3, 12.4 and 12.5, p129-130).

The correlation between generalised and individual measures of adverse factors has not been tested, namely the adverse factors exposed during confidential reviews and apparent general factors of deprivation as identified by Jarman. In the study in Southern Derbyshire (refer to Section 13, p132) post perinatal mortality rates were directly related to electoral wards with high Jarman scores of deprivation (Figures 13.1 and 13.2,

p134). These findings fit with the observations of the differences in prevalence of electoral wards with Jarman scores greater than 20 in Southern Derbyshire compared with the North District. However, by using the specific information gained from the confidential reviews, the adverse factors mitigating against individual families were identified. New standards of care were implemented by the primary care teams and service priorities were set for children in need. These needs should now be met under the guidelines of the 1989 Children Act[282] and the 1990 Community Care Act[283].

The main services available to the community are those provided by General Practitioners and Health Visitors. The study of workload and performance (refer to Section 9, p77) showed that Health Visitor caseloads were increasing and were considerably higher than those in Trent Region and nationally, while General Practitioner caseloads of 0-4 year olds remained static (Figure 9.2, p82). There was no evidence that immunisation uptake rates were related to areas of deprivation, the rates were related to individual General Practitioners and varied from 0-100% Uptake (Figures 9.6 and 9.7, p85). This means that with increased efficiency an equal uptake of immunisation is possible across the whole District.

Dual epidemics of measles and pertussis coincided with peaks in post perinatal mortality. When immunisation rates increased and epidemics became smaller in size, there was no corresponding peak in mortality in epidemic years. These findings support the view that notifiable childhood disease is associated with post perinatal mortality. This study further raised awareness to the need for detailed paediatric pathology, and forwarded the quest for a paediatric pathologist in the District.

In the study of death registration trends (refer to Section 7, p46) there was a transference of diagnosis of trauma into the registration cause of Sudden Infant Death Syndrome during the years 1980-1988 (Figure 7.25, p70). The subsequent finding of the link between names on the Child Protection Register and those who had died as cot deaths led to the realisation that some cot deaths were related to child abuse. One of the most striking features over the past decade in Southern Derbyshire was the marked increase in children registered on the Child Protection Register (Figures 9.8 and 9.9, p86). This was almost certainly due to increased recognition of child abuse rather than a true increase in incidence, but had implications for health visiting work loads as more time was spent attending multidisciplinary child protection case conferences. The study investigating the relationship between child abuse and cot death showed a definite association between abusing families and those where a cot death had occurred. There

were pointers that physical abuse may be the linking factor between some cot deaths and child abuse. The prospective study of the Child Protection Register over two years (refer to Section 14, p135) confirmed the association between cot death and siblings on the Child Protection Register (Figure 14.2, p140). This observation required repeated investigation, extending into different communities. A group has now been set up by the child abuse section of the British Paediatric Association to replicate this study in ten health districts in England.

The evidence of the actionable adverse factors found at confidential review in cases of possible filicide (F deaths) was similar to those factors known to be associated with abusing families and to the most preventable group of cot deaths (B deaths). This finding suggests that the investigation of these two groups of deaths and the characteristics of deaths in child abusing families are the most important group for immediate study, as in theory preventive measures could be applied. There was no evidence that there was any association between child abuse and idiopathic cot death, but these deaths constitute a maximum of 15% of children who are registered as cot deaths. The correlation between cot death and child abuse, has encouraged the Community Services Unit to allocate Health Visitor resources in this direction. However, it must not be inferred from this that there is an element of child abuse in most cot deaths.

These studies have demonstrated the use of categorising post perinatal infant deaths into probably not preventable and possibly preventable after birth groups. It would have been useful if these rates had been available over the years as the rates in these two groups have proved to be of more value locally than the total post perinatal mortality rates. This was particularly the case when investigating the differences between North and Southern Derbyshire. If Districts produced their possibly preventable death rates, it would be a helpful quality indicator of the standard of child health services.

Details of individual cases obtained at confidential review showed that the adverse social factors acting on specific cases were the same in both North and Southern Derbyshire when applied to the clinico-pathological categories of death. Thus the alteration and improvement in services to individual families needs to be the same in both Districts. The differences in the total post perinatal mortality rates between the two Districts appeared to be related to the greater proportion of numbers of families living at disadvantage in Southern Derbyshire.

This series of studies identified a wealth of new information. The additional information made available at the case discussion was the most valuable source for identifying intervention points in the group of deaths which were possibly preventable. The outcomes from case review directly affect the care of the next child in the family and the care of other families with similar problems. The evidence from this study points to the importance of the role of the case discussion in the confidential review in the partially explained cot deaths.

This study is an exercise in descriptive epidemiology. The numbers of cases involved in a single district are such that conventional statistical methods cannot apply. It is theoretically possible that individual defects could be assessed locally, provided that a sufficient number of control children are studied. This would involve holding case discussions on thriving children. From the evidence available (refer to p99) this seems unlikely to be possible as both parents and general practitioners were loathe to spend time discussing many of the children who had died from inevitable causes.

By doing this type of case review, the information obtained explained the differences in mortality between North and Southern Derbyshire and produced information which was not obtainable by any other means and which has enabled me to develop local individual preventive measures. The hypothesis on the value of in depth individual case review would appear to be substantiated.

Section 16

CONCLUSIONS

1. A profile of post perinatal deaths in the District confirmed that the death rate was high and that the pattern of death by attributed cause, season, age and place of death was similar to that found elsewhere in the country. The examination of tr ends in death registration confirmed that the local infant mortality rate was higher than the national average on many occasions over an 18 year period. There had been a relatively greater fall in the rate of probably not preventable group than in the possibly preventable after birth grouping. The rates of possibly preventable deaths did not decrease against a falling post perinatal mortality rate. All the deaths ascribed to trauma transferred to the diagnosis of SIDS ICD 9 (798) in 1979. Only 25% of deaths ascribed to infection transferred to the SIDS diagnosis. There was no evidence of a shift in either age or low birth weight deaths.

2. The Study of workload and performance of General Practitioners and Health Visitors were quantitative and gave no indication of quality of care.

3. The system of confidential review identified new sources of information of remediable adverse factors which applied to all clinico-pathological categories of death.

4. The parallel study undertaken in North and Southern Derbyshire confirmed the differences in mortality rates between the two districts. The reasons for the differences were identified by relating the adverse factors to the clinico pathological groups of deaths.

5. There is a higher proportion of families with preventable adverse social factors and high Jarman scores of deprivation in Southern Derbyshire than in North Derbyshire. High Jarman scores of deprivation are related to the "possibly preventable" death grouping in Southern Derbyshire.

6. The comparative study of the Child Protection and Post Perinatal Mortality Registers demonstrated that a proportion of children dying as cot deaths were related to families with children on the Child Protection Register.

7. The application of confidential review into each post perinatal death revealed factors which would otherwise have remained undisclosed and, if acted upon, could have influenced the outcome.

8. Detailed investigation by case review disclosed failure in standards of care which were contributory to the high death rates.

9. Although larger studies may provide a means of statistical validation, it is recommended that similar studies by confidential review should be undertaken by every health district as a means of improving the quality of local child care.

10. The hypothesis is upheld - the information obtained at case discussion constitutes the major data base for diminishing post perinatal mortality.

REFERENCES

1. OPCS. Titchfield (Series VS1 1974-91) SD25.
2. Black D, Morris JN, Smith C, Townsend P. Inequalities in Health. The Black Report. Townsend P, Davidson N (eds). London: Penguin, 1982:15-6,42-3,51-64,206-16.
3. Report of the Committee on child health services. Fit for the Future (Court Report), Comnd 6684. London: HMSO, 197:3-14,39-41,85-91,114-28.
4. Johnson AW (ed). Social policy indicators. An analysis of the 1981 census for Derbyshire. Derbyshire County Council, 1986.
5. Registrar General's Census of population 1971. Population statistics, household statistics, economic characteristics. London: HMSO, 1982.
6. Registrar General's Census of population 1981. Population statistics, household statistics, economic characteristics. London: HMSO, 1982.
7. Research and Intelligence Unit, Derbyshire County Council. Unemployment figures for District Council Derbyshire and Great Britain 1983-1988. Derbyshire County Council, 1989.
8. OPCS General Household Survey, 1981. London: HMSO, 1983 (Series GHS;No 13).
9. Nicoll JP, O'Cathain A. Epidemiology of babies dying at different ages from SIDS. J Epidemiol Comm Health 1989;43:133-9.
10. Norvenius SG. Sudden Infant Death Syndrome in Sweden in 1973-1977 and 1979. Acta Paediatr Scand 1987 (Suppl 333):10,17-21,61-73,82-8,89-106.
11. Barrett AM. Sudden Death in Infancy In: Recent advances in paediatrics. Gairdner D, ed. London: Churchill, 1954:301-20.
12. Bergman AB, Beckwith JB, Ray CG, eds. Sudden Infant Death Syndrome. Proceedings of the second international conference on cases of sudden infant death in infants, September 1969, Seattle. University of Washington Press, 1970:47-54,83.
13. Home Office. Report of the Committee on Death Certification and Coroners Cmnd 4810. London: HMSO, 1971.
14. Willinger M, Stanley JL, Catz C. Defining the Sudden Infant Death Syndrome: deliberations of an expert panel convened by the National Institute of Child and Human Development. Pediatr Pathol 1991;11(5):677-84.
15. Report of the working party on the prevention of early neonatal mortality and morbidity (Oppé report). London: DHSS, 1976(HC(74)40).

16. Trent Multicentre study on infant and perinatal mortality 1983-85. Trent Regional Press, 1987.
17. Cowley H. Derbyshire Structure Plan, Derbyshire County Council, 1989.
18. Cowley H. Derbyshire Local plan scheme, Derbyshire County Council, 1990.
19. Jarman B. Identification of underprivileged areas. BMJ 1983;286:1705-9.
20. Jarman B. Underprivileged areas: validation and distribution of scores. BMJ 1984;289:1587-92.
21. Smith GD. Second thoughts on the Jarman Index. BMJ 1991;302:359-61.
22. Institute of Public Health, University of Surrey. Public Health Common Data Set, England: Southern Derbyshire District Health Authority, 1988. London: Department of Health, 1989.
23. Institute of Public Health, University of Surrey. Public Health Common Data Set, England: Southern Derbyshire District Health Authority, 1989. London: Department of Health, 1990.
24. Newman G. Annual Report of the Chief Medical Officer, Ministry of Health for 1933. London: HMSO, 1934:9-10,60-4.
25. Laslett P. Births, Marriages and Deaths. After the transformation in: The world we have lost - further explored. 3rd ed. London: Methuen, 1983:106-121,160-61,314-16,246-73.
26. Registrar General's Annual Report. London: 1891-1973.
27. Reports of Medical Officers of Health for Derbyshire. Derby: Bemrose, 1899-1973.
28. Reeves MP Round about a pound a week (Fabian Women's Group Study 1909-1913). London: Virago, 1979:176-94.
29. Registrar General's Annual Report for England and Wales 1905-1925 and 1911-1925. London: 1926.
30. Winter JM. Civilian Health in Wartime Britain. The Decline in Infant Mortality in Wartime Britain in: The Great War and the British people. Macmillan, 1987:141-53.
31. Dwork D. War is good for babies and other young children: a history of the infant and child welfare movement in England 1898-1918. London: Tavistock, 1987:3-51,124-66,208-20.
32. Williamson H. Return to Hell in: The Wet Flanders Plain. London: Faber & Faber, 1987:154.
33. Longmate N. The Green Ration Book in: The war years - how we lived then: a history of everyday life in the Second World War. London: Hutchinson, 1971:167-79.

34. DHSS. NHS Reorganisation England (Comnd 5055). London: HMSO, 1972.

35. DHSS. Management arrangements in the reorganised NHS (The Grey Book). London: HMSO, 1972.

36. Report of the Committee on Local Authority and Allied Personal Social Services (Seebolm report), Comnd 3703. London: HMSO, 1968:181-200.

37. Wynn M. A policy for prevention - health care for every child. Concern 1976;21:9-12.

38. Morris J N. Social inequalities undiminished. Lancet 1979;i:87-90.

39. Health Services Act 1980. Restructuring of the service. London: HMSO, 1982. 1982.

40. DHSS Health Services Management: Implementation of the NHS Management Inquiry report . London: DHSS, 1984 (HC(84)13).

41. A Joint Working Party on Child Health Surveillance. Knowledge and sills required for surveillance in: Hall report, Health for all children. Programme for Child Health Surveillance. Oxford: University Press, 1989:94-8.

42. World Health Organisation. Alma Ata declaration 1978: primary health care. Geneva: WHO, 1978.

43. World Health Organisation. Global strategy for health for all by the year 2000. Geneva: WHO, 1981.

44. Barwise S. Annual Report on the Health of Derbyshire for the year 1908. Derby: Bemrose & Sons, 1909:15.

45. DHSS. Confidential enquiry into post neonatal deaths. London: HMSO, 1970 (Reports on Public Health and Medical Subjects, No 125).

46. Morris JN, Heady JA. Social and Biological Factors in Infant Mortality. Lancet, 1955;i:343-9.

47. Patten JL. Infant death by nationality of the mother in the Borough of Haringey 1969-72 & 1973-75. Public Health Handbook 1978;92:5-8.

48. Richards IDG, McIntosh HT. Confidential inquiry into 226 consecutive infant deaths. Arch Dis Child 1972;47:697.

49. Carpenter RG, Emery JL. Identification and follow up of infants at risk of sudden death in infancy. Nature 1974;250:729.

50. Sjolin S. Infant mortality in Sweden in: Wallace Helen M (ed) Health care of mothers and children in National Health Services. Cambridge, Mass: Ballinger, 1975.

51. Wynn M, Wynn A. Nutrition and counselling in the prevention of low birth weight: some conclusions after ante natal care following a visit to Canada. London: Foundation for Education and Research in Child bearing, 1975.

52. Salkever DS. Economic class and different access to care: Comparisons among health care systems. Int J Services 1975;3:373.

53. Brotherston J. Inequality: Is it inevitable? In: Carter CO, Peel J, eds. Equalities and Inequalities in Health. London: Academic Press, 1976.

54. Davies IM. Perinatal and infant deaths: social and biological factors. OPCS Medical statistics division, 1975-77. London: HMSO, 1980 (Studies in medical and population subjects: No 41).

55. Working Party for Early Childhood Deaths in Newcastle. Newcastle survey of deaths in early childhood 1974/76 with special reference to sudden unexpected deaths. Arch Dis Child 1977;52:828-35.

56. Steiner H. An evaluation of child health clinic services in Newcastle upon Tyne 1972-74. Br J Prev Soc Med 1977;31:1-5,974-6.

57. Carpenter RG, Gardner A, McWeeny PM, Emery JL. Multistage scoring system for identifying infants at risk of unexpected death. Arch Dis Child 1977;52: 606-12.

58. DHSS Report of the Children's Committee to advise the Secretary of State on the means of improving the health and care of children and their families (Brimblecombe Report) London: DHSS, 1978.

59. Underhill R, Atkins N. Schoolgirl pregnancies. J Maternal Child Health; 1978; 3:404-9.

60. Bradshaw J, Edwards H, Sladen F, Weale J. Area variations in infant mortality 1975/77. J Epidemiol Community Med 1982;36(1):11-6.

61. Oakley JR, Tavare CJ, Stanton AN. Evaluation of the Sheffield system for identifying children at risk from unexpected death in infancy. DHSS multicentre post neonatal study. Arch Dis Child 1978;53:649-52.

62. Madeley RJ. Relating child health services to needs by the use of simple epidemiology. Public Health Handbook, London, 1978;92:224-30.

63. Emery JL. Planning improved urban child health in: Signposts in community medicine. Proceedings of the Annual Scientific Meeting of the Faculty of Community Medicine, London: Oxford University Press, 1978.

64. Pharoah POD, Morris JN. Post neonatal mortality. In: Sartwell PE, ed. Epidemiological Reviews I, 1979:170-83.

65. Lewis E. Mourning by the family after a stillbirth or neonatal death. Arch Dis Child 1979;54:303-6.

66. DHSS Report of Committee on Health Service development. Perinatal and neonatal mortality (Short Report). London: DHSS, 1980 (HC(80)13).

67. Report of the Multicentre Study of postneonatal mortality 1976-1978 (Knoweldon Report), Medical Research Unit, Sheffield. London: HMSO, 1985.

68. OPCS Infant and perinatal mortality 1986. District Health Authorities (Series DH3 87/4).

69. OPCS Sudden Infant death syndrome 1988-89. London: HMSO (Series DH3 91/1).

70. Rodrigues L, Botting B. Recent trends in post-neonatal mortality in England. Population Trends 1989;55:7-15.

71. Arneil GC, Brooke H, Gibson AAM, Harvie A, McIntosh H, Patrick WSA. National post perinatal infant mortality and cot death study, Scotland 1981-82. Lancet 1985;i:740-3.

72. Pharoah POD, Alberman ED. Annual statistical review. Arch Dis Child 1988;63:1511-5.

73. Pharoah POD, Alberman ED. Annual statistical review. Arch Dis Child 1990;65:147-51.

74. Centers for Disease Control. Seasonality in Sudden Infant Death Syndrome - United States, 1980-87. MMWR 1990;39:891-5.

75. Carpenter RG, Gardner A. Variations in unexpected infant death rates related to age, sex and season. OPCS London School of Hygiene and Tropical Medicine. Studies in SIDS. London: HMSO, 1982:23-31 (Studies on medical and population subjects no 45).

76. Osmond C, Murphy M. Seasonality and the sudden infant death syndrome. Paediatr Perinat Epidemiol 1988;2:237-45.

77. Peterson DR, Sabotta EE, Strickland D. Sudden infant death syndrome and epidemiological perspective: etiologic implications of variation with season of the year. Ann NY Acad Sci 1988;533:6-12.

78. OPCS Mortality statistics perinatal and infant: social and biological factors 1986. London: HMSO, 1988 (Series DH3 No 20).

79. Kinmonth A. Review of the epidemiology of SIDS and its relationship to temperature regulation. Br J Gen Pract 1990;40:161-3.

80. Macarthur BA. A study of SIDS by age. Child Care Health Dev 1987;13:197-206.

81. Sunderland R, Gardner A, Gordon RR. Why did post perinatal mortality rates fall in the 1970s? J Epidemiol Community Health 1986;40:228-31.

82. Golding J. Is the sudden infant death syndrome preventable? J Roy Soc Med 1988;81:65-6.

83. Golding J, Limerick S, MacFarlane A. Sudden infant death patterns, puzzles and problems. Shepton Mallet Open Books 1985:22-42,94-102.

84. Couriel JM, Davies P. Costs and benefits of a community special care baby service. BMJ 1988;296:1043-6.

85. Mitchell RG. Objectives and outcome of perinatal care. Lancet 1985;ii:931-4.

86. Protestos CD, Carpenter RG, McWeeny PM, Emery JL. Obstetric and perinatal histories of children who die unexpectedly (cot death). Arch Dis Child 1973;48:835-41.

87. Baird D. The epidemiology of low birth weight: Changes in incidence in Aberdeen 1948-1972. J Bio Soc Sci 1974;6:323-41.

88. DHSS London: DHSS, 1987 (Series LHS 27/1). Low birth weight births and mortality, 1986.

89. Fedrick J. Sudden unexpected death in infants in the Oxford record linkage area: an analysis with respect to time and place. Br J Prev Soc Med 1973;27:217-24.

90. Kyle D, Sunderland R, Stonehouse M, Cummins C, Ross O. Ethnic differences in incidence of SIDS in Birmingham. Arch Dis Child 1990;65:830-3.

91. Irgens LM, Skjaerven R. SIDS and post perinatal mortality in Norwegian birth cohorts 1967-1980. Acta Paediatr Scand 1986;75:523-9.

92. Allen DM, Buehler JW, Hogue CT, Strauss CT, Smith JC. Regional differences in birthweight specific infant mortality. MMWR 1987;102:138-45.

93. Taylor EM, Emery JL. Categories of preventable unexpected deaths. Arch Dis Child 1990;65:535-9.

94. Sunderland R, Sunderland EP. Invalid certification of young deaths. Arch Dis Child 1983, 58:867-74.

95. Sonnabend OAR, Sonnabend WFF, Krech H, Molz G, Sigrist J. Continuous microbiological and pathological study of 70 sudden and unexpected infant deaths. Toxogenic intestinal clostridium botulinum infection in 9 cases of sudden infant death syndrome. Lancet 1985;i:237-41.

96. Williams FLR, Lloyd OLL. Mortality at early ages in Scottish communities 1959-83. Geographical distribution and associations with selected socio-economic indices. Public Health 1990;104:227-37.

97. Goldblatt P. Changes in Social Class between 1971 and 1981: Could these affect mortality differences among men of working ages? Population Trends, 1988;51:9-17.

98. OPCS. Mortality statistics Childhood 1984 Deaths by cause. England and Wales. London: HMSO, 1985 (Series DH3 No 16).

99. OPCS Mortality statistics perinatal and infant: social and biological factors for 1984. London: HMSO, 1986 (Series DH3 No 17).

100. OPCS Infant and perinatal mortality 1985. RHAs. London: HMSO, 1987 (Series DH3/87/3).

101. OPCS Infant and perinatal mortality 1985. DHAs. London: HMSO, 1987 (Series DH3 87/2).

102. Gillies DRN, Lealman GT, Lamb KM, Congdon P. Analysis of ethnic influence on stillbirths and infant mortality in Bradford 1975-81. J Epidemiol Community Health 1984;38:214-7.

103. Balarajan R, Raleigh V, Soni Botting B. SIDS and post-neonatal mortality in immigrants in England and Wales. BMJ 1989;298:716-20.

104. Gordon RR, Sunderland R. Maternal age illegitimacy and post-neonatal mortality. BMJ 1988;297:774.

105. OPCS Mortality statistics childhood 1985. London: HMSO, 1986 (Series DH3 No 19).

106. OPCS SIDS 1983-84. London: HMSO, 1985 (Series DH3 85/4).

107. Pharoah POD, Alberman ED. Annual statistical review. Arch Dis Child 1987;62:745-51.

108. Macfarlane A. The downs and ups of infant mortality. BMJ 1988;296:230-1.

109. Emery JL, McWeeny PM. Unexpected post-neonatal deaths (cot deaths) due to recognisable disease. Arch Dis Child 1975;50:191-6.

110. Department of Health On the state of the Public Health: The annual report of the Chief Medical Officer of The Department of Health for the year 1988. London: HMSO, 1989.

111. Institute of Public Health, University of Surrey. Public Health Common Data Set. England and Wales 1990. London: Department of Health, 1991.

112. Froggatt P, Lynas MA, MacKenzie G. Epidemiology of sudden unexpected death in infants (cot death) in Northern Ireland. Brit J Prev Soc Med 1971;25:119-34.

113. Naeye RL, Ladis B, Draye JS. Sudden infant death syndrome: a prospective study. Am J Dis Child 1976;130:1207-10.

114. Milner AD. Recent theories on the cause of cot death. BMJ 1987;295:1366-8.

115. Watson E, Gardner A, Carpenter RG. An epidemiological study of unexpected death in infancy in nine areas of Southern England. Med Sci Law 1981;21:89-98.

116. Taylor EM, Emery JL. Trends in unexpected infant deaths in Sheffield. Lancet 1988;ii:1121-3.

117. Tonkin S. Sudden infant death: a hypothesis of causation. Pediatrics 1975;55:650-61.

118. Emery JL. Is sudden infant death syndrome a diagnosis or is it just a diagnostic dustbin? BMJ 1989;299:1240.

119. Valman B. Preventing infant deaths. BMJ 1985;290:339-40.

120. Fried A, Rhodes PH, Guyer B, Binks NJ, Harrison MT, Hague CJR. The postponement of neonatal deaths in the post-neonatal period: evidence from Massachussetts. Am J Epidemiol 1987;127(1):161-70.

121. OPCS Congenital malformations 1985. London: HMSO, 1986 (Series MB3 86/2).

122. Carpenter RG, Gardner A, Jepson M. et al Prevention of unexpected infant death. Evaluation of the first seven years of the Sheffield programme. Lancet 1983;i:723-7.

123. OPCS Mortality statistics. Perinatal and infant social and biological factors from 1975-1988. London: HMSO, 1989 (Series DH3).

124. Nicoll A, Gardner A. Whooping cough and unrecognised post perinatal mortality. Arch Dis Child 1988;63:41-7.

125. Gilbert RE, Fleming PJ, Azaz Y, Rudd PT. Signs of illness preceding sudden unexpected death in infants. BMJ 1990;300:1237-9.

126. Stanton AN. Overheating and cot death. Lancet 1984;ii:1199-1201.

127. Sunderland R, Emery JL. Febrile convulsions and cot death. Lancet 1981;ii:176-80.

128. Uren EC, Williams AL, Jack I, Rees JW. Association of respiratory virus infections with SIDS. Med J Aust 1980;1:417-9.

129. Bonser RSA, Knight BH, West RR. Sudden infant death in Cardiff. Association with epidemic influenza and with temperature 1955-79. Int J Epid 1978;7:335-9.

130. Southall DP, Thomas MG, Lambert HP. Prolonged expiratory apnoea and severe hypoxaemia in infants with pertussis. Arch Dis Child 1988;63:598-605.

131. Department of Health. Immunisation against infectious diseases. London: HMSO, 1990.

132. Royal College of General Practitioners, Swansea Research Unit. Effect of low pertussis vaccination on a large community. BMJ 1981;282:23-6.

133. Miller D, Wordsworth J, Diamond J, Ross E. Pertussis vaccine and whooping cough as risk factors in acute neurological illness and death in young children. Dev Biol Stand 1985;61:389-94.

134. Peterson DR. Evolution of the epidemiology of SIDS. Epidemiol Rev 1980;2:97-112.

135. Kroll JS. Bacterial virulence in an environmental response. Arch Dis Child 1990;65:361-3.

136. Carpenter RG, Gardner A. Environmental findings and sudden infant death syndrome. Lung 1990 suppl: 358-67.

137. Telford DR, Morris JA, Hughes P, Conway A et al. The nasopharyngeal bacterial flora in the sudden infant death syndrome. J Infect 1989;18:125-30.

138. Forsyth KD, Weeks SC, Koh L, Skinner J, Bradley J. Lung immunoglobulins in the sudden infant death syndrome. BMJ 1989;298:23-6.

139. Patrick WJA, Carrington D, Armstrong AA, Gibson AM, Urquhart GED. Eight year study of viral isolates from cot deaths in Glasgow. Scot Med J 1989;34:462-4.

140. Zink P, Drescher J, Verhagen W, Flik J, Milbradt H. Serological evidence of recent Influenzae virus A(H3 N2). Infections in forensic cases of SIDS. Arch Virol 1987;93:223-32.

141. Stalleybrass CO. The principle of epidemiology. London: Routledge, Keegan and Paul, 1931:280.

142. Williams AL, Uren EC, Brotherton L. Respiratory viruses and sudden infant death. BMJ 1984;288:1491-3.

143. Ferguson DM, Horwood CJ, Sharman FT. Parental smoking and respiratory illness in infancy. Arch Dis Child 1980;55:358-61.

144. Chen Y, Li W, Yu S. Influence of passive smoking on admissions for respiratory illness in early childhood. BMJ 1986;293:303-6.

145. DHSS Comparing Health Authorities: Health Service indicators 1983/86. London: DHSS, 1988.

146. Newlands M, Davies LM. The use of performance indicators for immunisation rates in general practice. Public Health 1988;102:269-73.

147. Whitehead M. The Health Divide In: Inequalities in health. London: Penguin, 1988:286-305.

148. Smith GD, Bartley M, Blane D. The Black report on socio-economic inequalities in health: 10 years on. BMJ 1990;301:373-7.

149. Royal College of General Practitioners, working party on prevention. Report from General Practice 22. Healthier children - thinking prevention. London: Royal College of General Practitioners, 1982.

150. Taylor EM, Emery JL. Family and community factors associated with infant deaths that might be preventable. BMJ 1983;287:871-4.

151. Taylor EM, Emery JL. Two year study of the causes of post perinatal deaths classified in terms of preventability. Arch Dis Child 1982;57:668-73.

152. Ashton J. Public health and primary care: Towards a common agenda. Public Health 1990;104:387-98.

153. Cooper NA, Lynch MA. Lost to follow up. A study of non attendance at a general paediatric out patient clinic. Arch Dis Child 1979;55:765-9.

154. Madeley RJ, Hull D, Holland T. Prevention of post neonatal mortality. Arch Dis Child 1986;61:459-63.

155. Madeley R. Preventing SIDS: Future strategies. Health Visitor 1988;61:241-3.

156. Carpenter RG. Preventing unexpected infant deaths by giving extra care to high risk infants. Health Visitor 1988;61:238-40.

157. Gordon RR, Sunderland R. Equality in death. BMJ 1987;259:529.

158. Report of the Joint Group on Performance Indicators to the Secretary of State for Social Services (The Graham Report). London: DHSS, 1985:27-30.

159. The NHS Training Authority and DHSS. Statistics about the clinical activity in services provided to and in the community: comprising the recommendations contained in the 5th report of the Health Services Information Steering Group to the Secretary of State. London: DHSS, 1987:11-3,17-9,21-9,32-3.

160. Watkins CJ. Can GPs prevent the Sudden infant death syndrome? BMJ 1989;298:1333-4.

161. Beswick K, Lynch MA, Roberts J. Child abuse and general practice. BMJ 1976;2:800-12.

162. Warden J. Action on infant mortality. BMJ 1989;299:296.

163. Warden J. Infant death. BMJ 1989;298:8.

164. Warden J. From budgets to babies. (Letter from Westminster.) BMJ 1988;296:943.

165. Rethans JJ, Sturmans Ferd, Drop Riet, Van der Vleuten C. A method for introducing standardised (simulated) patients into GP consultations. Br J Gen Pract 1991;41:94-6.

166. Assessment of the performance of GPs by the use of standardised (simulated) patients. Br J Gen Pract 1991;41:97-9.

167. Stone GH. Suggested guidelines for measuring health indices in the community. Comm Med 1985;7:295-8.

168. Jarman B, Bosanquet N, Rice P, Dollimore M, Leese B. Uptake of immunisation in district health authorities in England. BMJ 1988;296:1775-8.

169. Walker AM, Jick H, Perera DR, Thompson RS, Knauss TA. Diphtheria tetanus pertussis immunisation and Sudden Infant Death Syndrome. Am J Pub Health 1987;77:945-51.

170. Bernier RH, Frank JA, Donders TJ. Diphtheria, tetanus toxoids pertussis vaccination and SIDS in Tennessee. Clinical and Laboratory observations 1979;101(3).

171. Taylor EM, Emery JL. Immunisation and cot death. Lancet (letter) 1982;ii:721.

172. WONCA 1986. Towards 2000: abstracts from the plenary sessions of the 11th Conference of The World Organisation of National Colleges, Academic Institutions of General Practitioners/Family Physicians (WONCA 1986). J R Coll Gen Pract 1986, June: 253-4.

173. Appleyard WS. Children's services in the new NHS - a struggle for survival. Arch Dis Child 1990;65:635-7.

174. National Children's Bureau Policy and Practice Review Group. Investing in the future. Child Health ten years after the Court report. London: National Childrens Bureau, 1987.

175. Elfer P. Community health care for families with young children. Highlight (Bulletin of the National Childrens Bureau) No 87, 1989.

176. Gregory J. Observations on the duties and offices of the physician and on the method of presenting enquiries in philosophy. W. Strachan & J. Cadell, London, 1770.

177. Barker W. Cot Deaths: no longer a mystery. Empowerment, University of Bristol, 1989;1-12.

178. World Health Organisation. International classification of impairments, abnormalities and handicaps. A manual of classification relating to the consequences of disease. Geneva: WHO, 1981.

179. Paediatric Research Unit, Royal Devon and Exeter hospital. A suggested model for inquiries into perinatal and early childhood deaths in a health care district. Exeter: Royal Devon and Exeter Hospital, 1983.

180. Davie R, Butler N., Goldstein H. From birth to seven. Second report of the National Child Development Study (1958 cohort). London: Longman, 1972.

181. Reading R, Jarvis S, Openshaw S. Measurement of social inequalities in health and use of health services among children in Northumberland. Arch.Dis Child 1993;68:626-31.

182. Reading R. Equity and community child health. Arch Dis Child 1993;68:686-90.

183. Watt IS, Franks AJ, Sheldon TA. Rural health and health care. BMJ 1993;306:1358-9.

184. Joint Report of the Working Party towards an integrated child health service (Brotherston Report), London: HMSO, 1973.

185. Harrison M, Hart L. Homestart. In: Macfarlane JA, ed. Progress in Child Health. 3rd ed. Edinburgh: Churchill Livingstone, 1987.

186. Townsend P, Phillimore P, Beattie A. Inequalities in health in the Northern region: An interim report Bristol Northern Regional Health Authority 1986.

187. Townsend P, Phillimore P, Beattie A. Health and deprivation: inequality and the north. London: Croom Helm, 1988.

188. Bain AD, Bartholomew SEM. A review of the Sudden Infant Death Syndrome in S.E. Scotland. Health Bull 1985:51-9.

189. Hogue JR, Strauss LT, Buchler JW, Smith JC. Overview of the Mortality surveillance , National Infant Mortality Surveillance (NIMS) 1980 project. MMWR 1989;38:1-46.

190. Fox AJ, Jones DR, Goldblatt PO. Approaches to studying the effect of socio-economic circumstances on geographic differences in mortality in England and Wales. J Med Bull 1984;40(4):309-14.

191. Brennan ME, Lancashire R. Association of childhood mortality with housing status and unemployment. J Epidemiol Community Health 1987;32:28-33.

192. Skuse D. Extreme deprivation in early childhood. II Theoretical issues and comparative review. J Child Psychol and Psychiatry 1984;25:543-72.

193. Lee NNY, Chan YF, Davies DP, Lau E, Yip DCP. SIDs in Hong Kong. Confirmation of low incidence. BMJ 1989;298:721.

194. Nicoll A, Mann N, Mann S, Vyas H. The child health clinic: results of a new strategy of community care in a deprived area. Lancet 1986;i:606-8.

195. Hassell IB. The cot death enigma. Med J Aust 1987;147:214-6.

196. Southall DP, Samuels MP, Talbert DG. Recurrent cyanotic episodes with severe arterial hypoxaemia and intrapulmonary shunting: a mechanism for sudden death. Arch Dis Child 1990;65:953-61.

197. Valdes-Dapena M. The pathologist and the sudden infant death syndrome. Am J Pathol 1982;106:118-31.

198. Southall DP, Alexander JR, Stebbens VA, Taylor VC, Janczynski RE. Cardiorespiratory patterns in siblings of babies with Sudden infant death syndrome. Arch Dis Child 1987;62:721-6.

199. Paton JY, MacFadyen UM, Simpson H. Sleep phase and gastro-oesophageal reflux in infants at possible risk of SIDS. Arch Dis Child 1989;64:264-9.

200. Stanton AN, Downham MAPS, Oakley JR. Controlled study of terminal symptoms and the action taken for them in the first 100 sudden unexpected home deaths in the DHSS multi-centre study of post-neonatal deaths. Arch Dis Child 1978;53:834-5.

201. Smithells RW, Shepherd S, Schorar CJ et al. Apparent prevention of neural tube defects by periconceptual vitamin supplementation. Arch Dis Child 1981;56:911-8.

202. Sinclair Smith C, Dinsdale F, Emery JL. Evidence of duration and type of illness in children found unexpectedly dead. Arch Dis Child 1976;51:424-8.

203. Mitchell EA, Scragg R, Stewart AW et al. Results from the first year of the New Zealand cot death study. NZ Med J 1991;104:71-6.

204. Taylor EM, Emery JL. Enquiries into perinatal and early childhood deaths in a health care district. Arch Dis Child 1984;59:1109-10.

205. Griffiths R, White M, Stonehouse M. Ethnic differences in birth statistics from central Birmingham. BMJ 1989;298:94-5.

206. Spencer NJ. Parents recognition of the ill child. Progress in child health. Macfarlane JA, ed. Vol 1. Edinburgh: Churchill Livingstone, 1984.

207. British Paediatric Association outcome measurements for Child Health. London: British Paediatric Association 1992;5-9.

208. Powell J, Machin D, Kershaw CR. Unexpected SID in Gosport. Some comparisons between service and civilian families. Journal of the Royal Naval Medical Services 1983;69:141-50.

209. Pugh EJ, Statham R, Jarvis J. Cot deaths, still births and the probation service: a potentially recognisable at risk group. Arch Dis Child 1987;62:146-7.

210. Carstairs V. Multiple deprivation and health state. Comm Med 1981;3:4-13.

211. Carstairs V, Russell M. Deprivation: explaining differences in mortality between Scotland and England. BMJ 1989;299:886-9.

212. Adelston AM, MacDonald Davies IM, Weatherall JAC, White GC. Sudden Infant Death: Social, biological and regional factors. Studies in SIDS. HMSO, 1982 (Studies in Method and Population Subjects No. 45).

213. Report of the Working Group in the confidential enquiry into stillbirths and deaths in Infancy. London: DH 1990. CESDI.

214. Kimmance KJ, Waters WE. Infant mortality and army families: a case-control study. BMJ 1992;305:1197.

215. Berlin A, Spencer JA, Bhopal RS, van Zwanenberg TD. Audit of deaths in general practice: pilot study of the critical incident technique. Quality in Health Care 1992;1;231-35.

216. British Paediatric Association. Paediatric Medical Audit, London: 1993:6-10.

217. Russell IT, Wilson BJ. Audit: the third clinical science? Quality in Health Care 1992;1;51-5.

218. Ellis BW, Sensky T. Audit in Person. A clinicians guide to setting up audit. BMJ 1991;302:704-7.

219. Bowden D Walshe K. Audit in Person. When medical audit starts to count. BMJ 1991;303:101-3.

220. Bhopal RS Thomson R. Audit in Person. A form to help learn and teach about assessing medical audit papers. BMJ 1991;303:1520-2.

221. Joint Commission on Accreditation of Health Care Organisations. Primer on Indicator Development and Application: Measuring quality in health care. Illinois: Joint Commission on Accreditation of Health Care Organisations, 1990:3-5,8-21,37-90,93-4.

222. Flanagan JC. The critical incident technique. Psychological Bulletin 1954;51:327-58

223. Waterston T. A critical incident study in child health. Medical Education 1988;22:27-31.

224. Grol R. Development of guidelines for general practice care. Br. J. Gen Pract 1993;43:146-51.

225. Appleby L, Dickens C. Mothering skills of women with mental illness. BMJ 1993;306:348-9.

226. St. James-Roberts I. Persistent infant crying. Arch Dis Child 1991;66:653-5.

227. Asch SS. Post partum reactions. Some unrecognised variations. Am J Psychiatry 1974;131:870-4.

228. Rao M, Hoinville E. Review of post perinatal mortality in a health district with a garrison town. BMJ 1988;297:662.

229. Graham H. Women's smoking and family health. Soc Sci Med 1987;25:47-56.

230. Lowry S. Housing and health: indoor air quality. BMJ 1989;299:1388-90.

231. Cox AD, Pound A, Mills M, Pickering C, Owen AL. Evaluation of home visiting and befriending scheme for young mothers: Newpin. J Roy Soc Med 1991;84:217-20.

232. Hill AM. Trends in paediatric medical admissions. BMJ 1989;298:1479-83.

233. Durojaiye LIA, Hutchinson T, Madeley RJ. Improved primary care does not prevent the admission of children to hospital. Public Health 1989;103:181-88.

234. Asch SS. Crib deaths. Their possible relationship to post partum depression and infanticide. Journal of the Mount Sinai Hospital 1968;XXXV(3):214-20.

235. Emery JL. Families in which two or more cot deaths have occurred. Lancet 1986;i:313-5.

236. Karmali J, Madeley RJ. Mothers' attitudes to a child health clinic in a deprived area of Nottingham. Public Health 1986;100:156-65.

237. Olds DL, Henderson CR, Tatelbaum R, Chamberlin R. Improving the delivery of prenatal care and outcomes of pregnancy. A randomised trial of nurse home visitation. Pediatrics 1986;77(1):16-28.

238. Waine C. Feeding problems. Maternity & Child Health 1985;10:203-7.

239. Graham P. Social class, social disadvantage and child health. Children and Society 1978;2(1):11-9.

240. Brimblecombe FSW In: Bridging in health: reports of studies on health services for children. MacLachlan G (ed) London: Oxford University Press, 1975.

241. Morley CJ, Thornton AJ, Cole TJ, Hewson PH, Fowler MA. Baby check: a scoring system to grade the severity of acute systematic illness in babies under 6 months old. Arch Dis Child 1991;66:100-5.

242. Denny E. The future of health visiting. Health Visitor 1989;62:250-51.

243. Dunst CJ, Leet HE. Measuring the adequacy of resources in households with young children. Child: Care, Health and Development 1987;13:111-25.

244. Lowry S. Housing and health: an introduction to housing and health. BMJ 1989;299:1261-2.

245. Lowry S. Housing and health: temperature and humidity. BMJ 1989;299:1326-8.

246. Lowry S. Housing and health: sanitation. BMJ 1990;300:177-8.

247. Walker RD. Study of out of hours visits to children. J R Coll Gen Pract 1985;35:427-28.

248. DHSS. Report on Confidential Enquiries into maternal deaths in England and Wales 1979-1981. London: HMSO, 1986.

249. Campling EA, Devlin HB, Hoile RW, Lunn JN. The report of the National Confidential Enquiry into Perioperative Deaths 1990. London: National Confidential Enquiry into Perioperative Deaths, 1992.

250. Buntinx F, Knottnerus JA, Crebolder HFJM, Seegers T, Essed GGM, Schouten H. Does feedback improve the quality of cervical smears? A randomized control trial. Br J Gen Pract, 1993;43:194-98.

251. Mugford M, Banfield P, O'Hanlon M. Effects of feedback on information on clinical practice:a review. BMJ 1991;303:398-402.

252. Mitchell MW, Fowkes FGR. Audit Reviewed: Does feedback on Performance change Clinical Behaviour? J R Coll Physicians London 1985;19:251-54.

253. Newlands M, Adamson EA, Ghulam S, Saleh M, Emery JL. Differences in post perinatal infant deaths between North and Southern Derbyshire. Arch Dis Child 1991;66:1421-25.

254. Talbot RJ. Underprivileged areas and health care planning: implications of use of Jarman indicators of urban deprivation. BMJ 1991;302:383-6.

255. Pearson M. Ethnic differences in infant health. Arch Dis Child 1991;66:88-90.

256. Davidson Ward SL, Bautista D., Chan L et al. Sudden infant death syndrome in infants of substance abusing mothers. J Pediatr 1990;117:876-81.

257. Derbyshire Area Review Committee. Child Abuse. Handbook and procedures practice: guide notes. Matlock: Derbyshire County Council, 1986.

258. Newlands M, Emery JL. Child abuse and cot death. Child Abuse and Neglect 1991;15:275-8.

259. Oliver JE. Dead children from problem families in N.E. Wiltshire. BMJ 1983;286:115-7.

260. Olds DL, Henderson CR, Chamberlain R, Tatelbaum R. Preventing child abuse and neglect: A randomised trial of nurse home visitation. Pediatrics 1986;78:65-78.

261. Lynch MA. Child abuse. The critical path. J Maternal Child Health 1976;1:25-9.

262. Roberts J, Golding J, Keeling J, Smith B, Lynch MA. Is there a link between cot death and child abuse? BMJ 1984;289:789-91.

263. Roberts J, Lynch MA, Golding J. Postnatal mortality in children from abusing families. BMJ 1980;281:102-4.

264. Department of Health. Perinatal and neonatal and infant mortality: Government reply to the first report from the Social Services Committee session 1988-89. London: HMSO, 1988.

265. World Health Organisation. Manual of the international statistical classification of diseases, injuries and causes of death. IXth revision (1) Geneva: WHO, 1977.

266. Jarman B. General practice, the NHS review and social deprivation. Br J Gen Pract. 1991;41:76-9.

267. Chase HD, Davies PRT. Calculation of the underprivileged area score for a practice in inner London. Br J Gen Pract 1991;41:63-6.

268. Carr-Hill RA, Sheldon T. Designing a deprivation payment for GPs: The UPA(8) wonderland. BMJ 1991;302:393-6.

269. Hutchinson A, Fry C, Sandhu B. Comparison of two scores for allocating resources to doctors in deprived areas. BMJ 1989;299:1142-4.

270. Department of the Environment. Urban deprivation. London: DOE, 1983 (Information Note No. 2 from the Inner Cities Directorate).

271. Moore A. Preventable deaths in Wolverhampton. BMJ 1986;293:656-8.

272. Nam CB, Eberstein IW, Deeb LC. SIDS as a socially determined cause of death. Social Biology 1989;36(1-2):1-8.

273. Curtis SE. Use of survey data and small area statistics to assess the link between individual morbidity and neighbourhood deprivation. J Epidemiol Community Health 1990;44:62-8.

274. Meadow R. Suffocation. BMJ 1989;298:1572-3.

275. Emery JL. Infanticide, filicide and cot death. Arch Dis Child 1985;60:505-7.

276. Southall DP, Stebbens VA, Rees SV, Lang MH, Warner JO, Shinebourne EA. Apnoeic episodes induced by smothering in 2 cases identified by covert video surveillance. BMJ 1987;294:1637-41.

277. Chapman MGT, Woodmansey AG. Point of view: Case conferences for child abuse. Arch Dis Child 1986;61:1141.

278. Lea-Cox C, Hall A. Attendance of General Practitioners at Child Protection case conferences. BMJ 1991;302:1378-9.

279. Smith SM, Hanson R, Noble S. Parents and battered babies: A controlled study. BMJ 1973;4:388-91.

280. Steinberg CD, Catalano R, Dooley D. Economic antecedents of child abuse and neglect. Child Development 1981;52:975-85.

281. Helfer RE. The perinatal period. A window of opportunity for enhancing parent-infant communication. An approach to prevention. Child Abuse & Neglect 1987;11:565-97.

282. The Children Act 1989. London: HMSO, 1989.

283. The National Health Service and Community Care Act. London: HMSO, 1990.

APPENDIX 1

CASE 3/87

Home interview: 24th April 1987
Case conference: 29th July 1987

I was notified on 10th February 1987 by the Coroner of this child who was found dead at home in his cot aged 3½ weeks. The registered cause of death was sudden unexpected death in infancy. Case enquiry disclosed many previously undiscovered facts surrounding the child's life and death, whereas only the police history was available to the Pathologist.

Background circumstances

This was a first born male child of a 28 year old primigravida. The mother was employed as a bank clerk and her husband, the father of her child, was a joiner. They had been married for four years, yet this was an unplanned pregnancy due to "failed contraception".

The couple had lived in the same house since their marriage. The house was a comfortable, well maintained three bedroomed house with central heating, three quarters of a mile from the surgery and the clinic and four miles from the nearest hospital. They possessed both a car and a telephone.

This family was described as a "healthy middle class family" by the Health Visitor and General Practitioner. The medical and social family histories of both the mother and father were unremarkable and the family had not been subjected to any periods of acute stress. There were no financial worries nor family friction. Neither the couple nor their parents were known to the Social Services Department. The mother was described as a rather anxious, intelligent girl who was well supported by her husband.

Ante-natal and Obstetric care

The first ante-natal visit was at nine weeks gestation. Ante-natal care was shared between the General Practitioner and the hospital. All ante-natal appointments were kept, eight visits to the General Practitioner and five to the hospital. During the 12th week of pregnancy Mrs S developed glycosuria. A subsequent glucose tolerance test was normal. At 18 weeks and again at 20 weeks .

Mrs S had small antepartum haemorrhages and was admitted to the Derbyshire Women's Hospital for rest. Her general health was good during pregnancy and Mrs S was not a smoker and did not drink alcohol.

A foetoscan at 31 weeks was normal. At 34 weeks there was spontaneous rupture of membranes and she delivered a male child by spontaneous vertex delivery, the baby being monitored by scalp electrodes throughout labour. There were type 1 dips in the second stage of labour which had lasted for 68 minutes. The Apgar scores were 10 at 1 minute and 10 at 5 minutes.

The Baby

A male child. Date of birth: 14th January 1987.
Date of death: 8th February 1987 aged 3½ weeks. Gestation: 34 weeks, not small for dates.
Birth weight: 2.32 kilograms.
Head circumference: 32 centimetres.
Length: 46 centimetres.

He did not require resuscitation at birth but was admitted to the Special Care Baby Unit for 20 days during which time he was tube fed for 13 days. Physiological jaundice was apparent on day two, peaking on day four. This was treated with phototherapy for one day.

His haemoglobin was 17 grams. The white cell count 13.6. Urine analysis showed less than 5 leucocytes. He was discharged home on the 20th day weighing 2.35 kilograms. He was prescribed Abidec 0.6 millilitres in the morning, Niferex one drop per pound three times daily and Folic acid 100 micrograms in the morning. A follow up appointment was offered six weeks after discharge.

Events following discharge from hospital
Date and time of death: Sunday 8th February 1987 before 8.30 am.

The baby was fed on SMA Gold Cap from birth. Mrs S reported that the baby had always been difficult to feed, being snuffly and short of breath while feeding. No regular pattern of feeding had been established. He developed a cough and cold three days before his death and Amoxil had been prescribed by the General Practitioner. The day before death the baby seemed better but had possetted after feeding.

On the day of death he was fed at 3.00 am but was restless. He settled in his cot at 5.00 am after being nursed. The parents awoke at 8.30 am and found the baby dead; they immediately dialled 999 and the baby was taken with his parents to the Accident and Emergency department. He was certified dead on arrival.

This child had only been home for five days before his death during which time a Health Visitor had visited once and had also advised the mother by telephone to seek the doctor's advice concerning the baby's cold and cough. The General Practitioner had visited the home twice for post-natal visits and had seen the baby at the surgery for his cold and cough when he prescribed Amoxil.

Pathology

This was a well nourished normally formed male infant. There were no marks of injury or neglect. The trachea contained pale, creamy mucous. The pleurae appeared normal. The lungs appeared congested. There were prominent lymph nodes in the mesentery. There was no histology or microbiological examination.

Diagnosis

Sudden Unexpected Death in Infancy.

Case conference - major points

1. This was an anxious, intelligent middle class mother who was well supported by her husband and parents.
2. It was an unplanned pregnancy, but both parents were interested and involved with the pregnancy and the baby. The mother had bonded well with the baby.
3. The child was on the Health Visitor "worry" file due to his prematurity.
4. The mother had used the services competently, had recognised the child's illness early and had sought advice. However, she had never developed a rapport with a particular doctor in the practice, consulting whichever doctor was available at the time. She did not contact the General Practitioner after the baby died and she declined to see the baby after he was certified dead.
5. The mother had stated at home interview that she felt the child would not have died if she had been able to take the baby back to the Special Care Unit for admission.
6. The General Practitioner had not received a discharge letter from the Special Care Unit before the baby died. The letter was dated two days after the baby's death. He had not received an explanation why the baby had been in the Special Care Unit

for so long. The mother had omitted to tell the General Practitioner that the baby had been tube fed.

7. The baby was discharged from the Special Care Unit only having gained 3 grams (weight 3.35 kilograms).
8. The discharge note was inadequate for the General Practitioner's needs for caring for the child in the community.
9. There had been sensitive handling of the baby's parents after the death of the child.
10. There had been good Social Work input and counselling input to the family after the baby's death.

Questions

1. Should this child have been discharged from the Special Care Unit at that time?
2. Should he have been readmitted with his respiratory tract infection?
3. Should the mother have had free access to the Special Care Unit, bypassing the General Practitioner?

Recommendations made at the end of the conference

1. There should be "open" admission facilities for all ex Special Care Unit babies.
2. Communications between the Paediatricians and General Practitioners should be speeded up.

Action

I discussed these recommendations with the paediatrician in charge of the neonatal unit.

1. All mothers whose children have been in the Special Care Unit are now informed that they can contact the Special Care Unit any time if they are worried, when they will be advised or seen.
2. Discharge notes now contain more information and a telephone call is made to the General Practitioner and/or Health Visitor when the baby is to be discharged.

Conclusions

This death was classified as A3 (prematurity) and was considered to be probably inevitable. It was a multifactorial death in a baby compromised at birth who had acquired a respiratory tract infection.

This was one of the first enquiries that we undertook in the District. The pathology was insufficient and there were no microbiology reports. The pathology of the brain and the

lungs would have enhanced the findings and in this case the history really gave the conclusions.

CASE 6/87

Home interview: 22nd April 1987
Case conference: 10th July 1987

I was notified of the death of this child on 28th January 1987 by the family Health Visitor. He died in the City Hospital, Nottingham. The registered cause of death was Haemophilus influenzae meningitis.

Background circumstances

This was the second liveborn child of a 26 year old gravida 2, a planned pregnancy. The mother had been a part-time petrol pump attendant and her husband, the father of the child, was a foreman at a local garage. They had been married for six years. Their first child, a boy, was born in July 1984 and had presented no problems, although the mother had suffered from anxiety at that time.

The family had lived in the same house since their marriage. Their home was a privately rented, well maintained terraced house with three bedrooms, situated in a socially deprived village in the coalfields of east Derbyshire, nearer to Nottingham than to Derby City. It was a half hour walk to the surgery and clinic and a three minute walk to the bus stop from home. The family had both telephone and a car.

The mother had a medical history of scarlet fever when she was six weeks old and a systolic murmur was heard at the age of 16 years. She had had a dilatation and curettage for irregular bleeding in 1981 and removal of a small benign lesion from the nipple in 1981. She suffered from migraine. The father suffered from asthma and bronchitis as a child and had had a squint rectified during childhood. As an adult he suffered from hayfever.

This was a well supported family living in a homely house. Both parents were competent and caring. Neither parent drank alcohol but the mother was a "moderate" smoker both during pregnancy and during the baby's life.

Ante-natal and Obstetric care

The mother made her first ante-natal visit at 12 weeks. Ante-natal care was shared by the General Practitioner and the City Hospital, Nottingham. Nine visits were paid to the General Practitioner and one appointment at the Hospital.

At 12½ weeks Mrs W had an antepartum haemorrhage and was admitted to hospital but settled spontaneously. At 25-26 weeks she developed pre-eclamptic toxaemia and was readmitted on 10th October 1986. Subsequently labour was induced with Syntocinon because of uncontrolled blood pressure. Foetoscans at 14 weeks and the day before delivery did not reveal any foetal abnormality. The baby was normal size for dates. Diazepam and Hydrallazine had been prescribed for her pregnancy induced hypertension.

Normal vaginal delivery ensued with the foetus being monitored by scalp electrodes. The baby was resuscitated by suction and oral oxygen and transferred to the Special Care Unit. The Apgar was 7 at 1 minute and 9 at 5 minutes.

The Baby

A male child. Date of birth: 15th October 1986.
Date of death: 28th January 1987 aged 15 weeks.
Gestation: 26 weeks, not small for dates.
Birth weight: 1.1 kilograms.
Head circumference: 26 centimetres.

Problems

1. Extreme prematurity.
2. Hyaline membrane disease.
   After an apnoeic attack at 6½ hours the baby was established on ventilation for six days initially.
3. Delayed closure of the ductus arteriosus. On day five the baby developed a murmur and was treated with Indomethacin and Frusemide. The murmur disappeared the next day and did not recur.
4. Recurrent apnoea. Following the weaning from the ventilator the baby had increasing episodes of apnoea associated with cyanosis and bradycardia requiring further ventilation on day 9 and day 11. Apnoea attacks remained a problem until he was five weeks old.
5. Hyperbilirubinaemia. On day two the baby became jaundiced peaking to 150 micromoles per litre. He was treated with phototherapy including Hercules and the problem gradually resolved.
6. Necrotising enterocolitis. On day 43 he passed large amounts of fresh blood in his stool. He was treated with antibiotics and intravenous feeding for one week.

7. Hypocalcaemia. On day two the serum calcium fell to 1.22 milimoles per litre. He was treated with intravenous supplement.
8. Hyponatraemia. On day 11 the serum sodium fell to 127 milimoles per litre, but responded to intravenous supplements.
9. Retinopathy of prematurity. The baby had stage two retinopathy of prematurity in both eyes.

The baby received four blood transfusions during his stay in hospital. Cranial ultrasound scans were normal. By the time of his discharge to his mother at home on 18th December 1986 the child was thriving well on Cow and Gate, three hourly.

His weight was 1.9 kilograms, haemoglobin 9.7 grams per decilitre. His only medication was DHSS vitamin drops, five per day, Sytron 10 drops bd.

On discharge from hospital the baby was included in the Nottingham's Passport Care Scheme. This scheme is a special developmental paediatric follow up of neonatal intensive care babies. The babies on this scheme have been of very low birth weight (less than 1501 grams). The objectives are to assess the quality of life of babies who have had intensive care, to provide opportunities for diagnosing problems early, and care and therapy for those who have handicapping conditions.

Information is fed back to the neonatal unit to effect new treatment methods. The scheme is a means of researching normal and abnormal development and for training staff in developmental paediatrics.

Events following discharge from hospital

At 11 weeks of age a routine medical and developmental assessment was made by the General Practitioner and the Health Visitor. The baby was lively and well. A bilateral inguinal hernia was diagnosed.

At 13 weeks the baby developed an upper respiratory tract infection which made feeding quite difficult. He was treated by the General Practitioner with Amoxil and Ephedrine nose drops. He was reviewed by the paediatrician at 14 weeks and given a follow up appointment in a further three months. He was well at this time but was referred to the Paediatric Surgeon for repair of his inguinal hernia, the appointment being made for the day on which the baby died.

During his period at home the baby gained weight consistently and appeared to make a complete recovery from his upper respiratory tract infection. He was seen by the Health Visitor one week before he died, and was reported well.

On 26th January 1987 the baby was fed at 11.00 pm and slept through the night for the first time. Mrs W had to waken the baby at 8.30 am on 27th January 1987 when she found the baby pale and limp and he would not take his feed. She recognised that he was ill, but knowing that she was to attend the Paediatric surgeon in the afternoon of that day she telephoned the Hospital to see what she should do. The receptionist recommended that Mrs W should keep the appointment at the hospital and Mrs W took no further action. On arrival at the Out Patient Department the baby was moribund and taken immediately for intensive care. He subsequently died on 28th January 1987 at 10.20 am. His age at death was 15 weeks.

Pathology

A detailed paediatric post-mortem examination was performed which showed all the features of severe overwhelming sepsis with Haemophilus Influenzae meningitis.

Major points from the case conference

1. This was an extremely premature baby from a good home. He received excellent care and had thrived well.
2. The parents were seen as competent, mature and caring by both hospital and community staff.
3. The mother had had post-natal depression treated by Imipramine and had, apparently, recovered well by three months after the baby's birth.
4. This child had been severely compromised at birth and thus had not been able to cope with the bacterial infection.
5. Many people were involved in the care of this child after his discharge from the hospital.
6. Mrs W reacted quickly and appropriately to the baby's first infection.
7. Mrs W reacted inappropriately to the terminal infection, due to the fact that she was due to be seen at the hospital that day.
8. The baby was thus six hours without treatment which may have been life saving.
9. Mrs W spoke only to the receptionist at the hospital. She may not have described the baby's illness very accurately, but did not consult the General Practitioner, being satisfied with a layman's advice when she knew her baby was ill.

10. Members of the conference felt very strongly that parents should not be "fobbed off" by receptionists' advice about illness and that a doctor should always speak to the parents under these circumstances.

Recommendations

It was recommended that I should write to the Secretary of the Local Medical Committee to request that General Practitioners should insist that their patients are dealt with by senior doctors at the hospital, either by telephone or at the clinic. This was done and General Practitioners regularly make verbal complaints to consultants concerning the above. Those who are now to become independent contractors have written this request into their contractual agreements.

Conclusions

The clinico-pathological classification was a B2 death. Symptoms were present but treatment was delayed. Adverse social factors were absent Although this was an overwhelming infection in a severely compromised baby, the six hour delay in treatment may have affected the outcome.

It was concluded that this was a possibly preventable death.

CASE 20/87

Home interview: 9th October 1987
Case Conference: 10th February 1988

I was notified of the death of this child on 6th July 1987 by the family Health Visitor. The Coroner informed me on 8th July 1987 that he had certified the cause of death as acute respiratory infection. This was a Home Office case and the post mortem examination was undertaken by the Home Office Pathologist.

The information available to the Pathologist was limited in content at the time of examination. This was a baby aged three months who had died on 4th July 1987 under suspicious circumstances. He had been left alone in the house with his two year old brother for five hours while his mother was at work and his father was out drinking.

Background circumstances

This was the fifth child of a 26 year old gravida 5. The mother worked as a part-time shop assistant and her husband, aged 32, father of all the children, was unemployed at the time. The family were Irish Roman Catholics, the father having come from a rigid Catholic family. He expected his wife to be at home to be a housewife to look after the family. The mother came from a less rigid background and could not come to terms with her husband's attitude concerning her job which was the cause of numerous family rows and was used as a reason for the father's periodic heavy drinking bouts. There was often violence between the parents, but never to the children.

The family had not been known to the Social Services Department prior to the baby's death, but the father was known to the police for seven previous convictions for breach of the peace while under the influence of alcohol.

The family had lived in their privately owned home for four years. It was a comfortable well maintained terraced house with three bedrooms. The parents had been married for eight years and had a large supportive extended family living in the Derby area. They had a telephone but did not have the use of a car.

The family comprised five boys. The last baby was unplanned as the mother had forgotten to take her oral contraceptives while taking antibiotics for an infection. The boys were born in 1980, 1981, 1982, 1985 and 1987.

The mother's medical history was unremarkable. The father's medical history was good but he had a brother who died in infancy. This child had been sick and had not died unexpectedly. Neither parents smoked and the mother only had an occasional drink. The father had periodic drinking binges which had increased in frequency since he had become unemployed two years before the baby's death.

This family was part of the intensive health visiting project which had been undertaken during 1986/1987. The family knew and related well to the Health Visitor who had no particular concerns about the family. The family was seen as caring and competent with plenty of support from relatives. No mention had been made of the father's drinking bouts but he had never been known to harm the children.

Antenatal and Obstetric care

The first ante-natal attendance was not until 22 weeks and Mrs O only attended the General Practitioner once. She then attended the hospital clinic on eight occasions.

At 34 weeks gestation Mrs O developed a proven urinary tract infection. She was admitted to hospital for one week for rest and assessment and was treated with Ampicillin. Serum alpha-foetoproteins were not done.

Foetoscans at 31, 34 and 39 weeks were normal but Mrs O's weight had started to tail off. Labour was induced at term + 3 because of the tail off in the weight of the mother. The baby was born by vertex delivery.

<u>The Baby</u>

A male child. Date of birth: 20th March 1987.
Date of death: 4th July 1987 aged 3 months.
Gestation: Term + 3.
Birth weight: 3.76 kilograms.
Length: 52 centimetres.
Head circumference: 36 centimetres.
Apgar at 1 minute was 9.

The child did not require resuscitation and was fed on Ostermilk Complete Formula from birth. He was discharged home with his mother on day two. He established a regular feeding pattern by four weeks when he began to sleep longer at night.

Home care

Once home the baby thrived well. The General Practitioner visited on 2 occasions for routine post-natal visits. Despite the fact that this child was on the increased health visiting project, great difficulty was encountered by the Health Visitor in visiting this family. Nine successful home visits were made, but there were also 10 no access visits. The mother and baby attended the clinic on one occasion for a routine medical inspection.

The Health Visitor had made numerous telephone calls to the family to make appointments both for routine visits and for the mother's post-natal examination. Each time the child was seen he was well and thriving with his weight on the 50th centile until the last visit when his weight had fallen off below the 50th centile. There was no concern from either the mother or the Health Visitor and weaning was discussed. The mother had recently changed from Ostermilk Complete Formula to SMA Gold Cap as she stated that she could not get the Ostermilk due to a recent Salmonella scare. At 10 weeks the baby began to posset after feeds. The child was examined at the clinic by the Clinical Medical Officer and the mother was reassured.

The baby had not received any routine immunisations but appointments were being made.

Events leading to the child's death

There had been no concern about the baby until the day before his death and this concern was only in retrospect. None of the siblings had been ill in the weeks prior to the baby's death. None of the children had ever been seen for any injury.

The days preceding death had been very hot. At the time of the home interview the mother recalled that the day before death the baby had been restless although she had not discussed this with the Health Visitor who had called that day. The symptom chart maintained by the mother and the Health Visitor had recorded a fall off in weight from the 50th centile, and the fact that weaning had been discussed.

Mr O had been drinking heavily during the week before the baby died. This had not been reported to the Health Visitor.

On the night before death the mother recalled that she had been in to see the baby who "looked funny", he was lying on his back with his mouth open. Mrs O had picked him

up and noted that he had a small sore on his left ear. He woke up and seemed to be normal.

On the day of death the baby took eight ounces of Ostermilk at 10.30 am and was laughing and gurgling. At noon Mrs O left for work with the three older children leaving Mr O to care for the two younger children. During that morning there had been a violent row between the parents concerning Mr O's recent drinking binge and Mrs O's job. Mrs O had left for work while they were both angry and upset. After his wife's departure Mr O fed the baby and settled him down, but the baby had vomited back his feed. Mr O left the house at approximately 1.00 pm but he had noted that the baby's eyes were half open/half closed and that the baby would not suck on his dummy. Mr O had not intended to leave the house for long but during his excursions of that day he had told his friends and relatives that the children were being cared for.

At 6.15 pm Mrs O returned from work to find the baby dead in his carrycot on the settee and the toddler roaming round the sitting room on his own. The emergency services and General Practitioner were summoned and the Police Surgeon and General Practitioner certified death.

Later that evening the police charged Mr O with neglect of two children contrary to Section 1 (1) of the Children and Young Persons Act 1933.

Pathology

The body was that of a well nourished male infant aged three months. There was no evidence of injury apart from a small fresh bruise in the left external ear. The bronchial tree contained fine, frothy blood stained and slightly purulent mucous. The lungs showed bilateral congestion with early broncho-pneumonic change at the bases. The stomach contained partly curdled milk. There was no vomit in the mouth. The mesenteric glands in the ileocaecal area showed minor soft enlargement. The right middle ear contained soft purulent fluid. Microbiological examination of material from the nose, trachea, bronchus and the middle ear showed the presence of Staphylococcus aureus.

Major points from the case conference

1. This was a staunch Irish Catholic family with caring parents of five healthy boys.
2. They lived in a comfortable home. The children were apparently happy at home and at school.

3. The father had been unemployed for two years. He thought of himself as the breadwinner and was unhappy about his wife's job. The father went on regular drinking binges when he was often violent towards his wife but never to the children.

4. This family was part of an intensive health visiting project when the child had been seen on a weekly basis. A symptom chart had been kept and the baby had been weighed weekly. The Health Visitor had often found difficulty in access to the home due to the mother working.
5. At the time of death no terminal illness had been recognised either by the Health Visitor or by the parents. The father was acutely stressed on the day of the baby's death due to the row with his wife.
6. No-one knew that the children had been left alone as the father had covered his tracks. We do not know whether the children had been left alone before.
7. The baby had vomited his final feed and would not suck on the dummy. We do not know whether the baby was dead or moribund at this time and the father had left in panic because he did not know what to do.
8. The General Practitioner had very little to do with this baby. Ante-natal care had been undertaken by the hospital and the General Practitioner had been unaware that the mother had a urinary tract infection during pregnancy. The routine post-natal visits had been undertaken and there had been no other contact with the family until after the death of the child.

Addendum

A multi-disciplinary case conference was convened by the Social Services under the Child Protection procedure on 20th July 1987.

I had strongly recommended that the toddler be registered on the Child Protection Register as I felt that he was in danger. The members of the case conference did not agree, but the child was subsequently registered after the case had been taken to the Area Review Committee at my request. The toddler remained on the Child Protection Register until 30th October 1987 and the family was under surveillance by the Social Services. The case was then closed by the Social Services.

In November 1987 Mr O started drinking again and marital disputes resumed. Mrs O again became pregnant, expected date of delivery being September 1988.

Second death

In February 1988 Mrs. O was admitted to hospital with a urinary tract infection and the children were in the shared care of the father and the grandparents.

On 26th February Mr O took the toddler home from his grandparents in the evening and stopped off at several pubs, arriving home at 12.30 am on 27th February. He fell asleep on the settee with the child. He awoke at 6.00 am, he thought the child was "normal". He went back to sleep and woke at 3.00 pm and found the child dead. The child was aged 2 years 8 months.

The cause of death was diagnosed as asphyxiation and Mr O was arrested and charged with manslaughter. He was subsequently acquitted, the verdict being accidental death.

This death was preventable and resulted in complete disruption of the whole family unit.

Recommendations

At the time of our case conference the mother was already pregnant again and the toddler was still alive. The case conference recommended that the toddler should be maintained on the Child Protection Register; that the family should receive intensive support from Social Services as and when required and that when the new baby was born Social Services should convene a case conference to discuss the care and support of this baby.

The breakdown in communication between the General Practitioner and the Obstetrician was to be pursued by the General Practitioner.

Action

I attended the Child Protection case conferences concerning this family, but failed to get Social Services surveillance on this family beyond October 1988. Events concerning the death of the toddler precipitated support from Social Services.

The remaining three boys were placed on the Child Protection Register. The wardship proceedings were instigated, with the children to remain in the mother's care providing she did not allow the children to reside in the same household as the father.

The Social Services Department, in addition to the child protection issues, were now needed to support the mother as a single parent.

Conclusions

The clinico-pathological classification of the first baby who died and who we had investigated was an F2 death. At the time of death no illness had been recognised either by Health Visitor or by parent. Adverse social factors were present in that the father was a heavy drinker and there were bouts of violence with his wife. On the day of death he had been upset and stressed due to a quarrel with his wife.

We do not know whether this baby was dead before the father went out or whether the baby died during his absence. We do not know what the toddler was doing during the time of the father's absence.

It was concluded that this was a preventable death.

CASE 28/87

Home interview: 5th May 1988.
Case conference: Not recorded.

This baby died at home on 22nd November 1987 and was notified to me on 24th November by one of the clerical staff in the Department of Child Health, via the family Health Visitor. The death was confirmed by the Coroner on the same day. The registered cause of death was acute congestion of the lungs (Sudden Unexpected Death in Infancy).

Background circumstances

This was a first born male child of a 22 year old primigravida. It was a planned pregnancy. The mother was a part-time shop assistant who had been married for four years to a bricklayer who was 10 years her senior. Her husband had been married before and there had been one child from his previous marriage.

The couple had lived in the same house since their marriage, the father having lived there during his previous marriage. The house was owner-occupied with three bedrooms. It was heated by electric and coal fires. The family did not possess a telephone or a car and lived five miles from the General Practitioner's surgery and one mile from the clinic. They had the use of the neighbour's telephone and a friend's car for emergencies as the bus service was poor.

The couple were described as happily married and stable with no financial or housing problems, but Mrs. P's parents who were managers of a local public house had lost their living just prior to the baby's death. The family were not known to the Social Services Department.

The mother was an asthmatic, having had two hospital admissions as a child for pneumonia, asthma and bronchitis. The father's past social and medical background were unremarkable. Both parents were smokers. The mother did not drink alcohol and the father did so only rarely.

Ante-natal and Obstetric care

The first ante-natal visit was at eight weeks and ante-natal care was shared between the General Practitioner and Derby City Hospital. There were 11 visits to the General Practitioner and four to the Hospital.

At 25 weeks gestation Mrs P was prescribed Amoxil for bronchitis and at 38 weeks developed puffy feet when she was advised to rest. She used a Salbutamol inhaler during pregnancy. Both parents attended parentcraft classes. There were no complications of pregnancy. The serum alpha-foetoprotein was normal as was the routine foetoscan.

Mrs P went into spontaneous labour at term. The baby was a normal vertex delivery monitored during labour by foetal scalp electrodes. The mother's recovery was uneventful.

The Baby

A male child. Date of birth: 19th October 1987.
Date of death: 22nd November 1987 aged 5 weeks.
Gestation: Term.
Birth weight: 2.53 kilograms.
Length: 48 centimetres.
Head circumference: 33 centimetres.
Apgar scores at 1 and 5 minutes: 8 and 10.

No resuscitation was required and the baby was discharged home with his mother on the second day. He was fed on SMA Gold Cap from birth.

Events following discharge from hospital

The baby was established on a three hourly feeding pattern from birth but was inclined to be "windy". He always woke in the night for his feeds. Mother and baby were visited on two occasions by the General Practitioner and no problems were reported. The Health Visitor visited on three occasions and both mother and baby were well.

Three days before the baby died Mrs P took the baby to the clinic for a routine weight check. The baby was thriving well and was achieving his developmental milestones. There were no problems with bonding.

On the night preceding his death, the baby, aged five weeks, took five ounces of his feed but Mrs P reported that he was "niggly". She gave him some gripe water and the baby settled in his cot about 12.30 am. The mother woke at 5.00 am realising that the baby had not cried for his 1.30 am feed. She saw the baby lying on his face in his cot with one arm hanging down from the cot. He was very white.

The father tried to resuscitate the baby while a neighbour called for the General Practitioner who undressed and examined the baby and confirmed death.

Subsequent events

The police arrived and asked the mother why she kept the baby undressed, which she found very distressing. The baby was taken away but the parents did not know where he was for three days. They were then told he was in the Chapel of Rest and they went to visit him.

The parents received counselling from the Consultant Paediatrician which they found helpful, but the mother would have liked to have discussed the baby further with her own doctor.

Pathology

This was a normally developed male infant with no external abnormalities. The mucosa of the pharynx and larynx was normal. There was a little mucous present in the trachea. The lungs were expanded and showed some congestion of the lower lobes and were slightly oedematous. There was focal intracellular oedema with underlying oedema in the larynx. There was a heavy growth of Haemolytic streptococci from the trachea and a light growth from the lungs.

Major points

1. A planned pregnancy of capable, caring parents.
2. The mother was on treatment for asthma.
3. There were no adverse social circumstances.
4. The child had been well and thriving up to the night before death when his symptoms were insufficient to alarm the parents.
5. There was presence of a terminal disease which had no recognised association with the baby's death.
6. The baby had been found dead face down in his cot aged five weeks.
7. The police had upset the mother unnecessarily.

8. The mother had not been able to discuss the baby's death freely with the General Practitioner.

Recommendations

The police need further training on how to handle families who have had a cot death.

Action

I discussed this problem with the Chief Detective Superintendent in charge of child abuse and cot death and as a result was asked to give talks on cot death as part of the police training programme.

Problems still exist with insensitive handling of parents which seems to depend very much on the personalities and personal prejudices of those officers concerned. However, a Senior Police Officer and a WPC always attend and the police have cut down the number of policemen who visit the home after a child's death.

Conclusions

The clinico-pathological classification of this death was in the C3 grouping. There were no adverse social factors which were contributory to death. We did not know whether this death could have been prevented.

Addendum

Mrs P has since given birth to a healthy male child on 20th January 1989. She was given an apnoea monitor for this baby. The baby was kept under review in the CONI project (Care of the Next Infant) which was instigated under the auspices of the Foundation for the Study of Infant Deaths.

CASE 21/88

Home interview: 22nd February 1989.
Case conference: 5th May 1989.

I was notified of the death of this child on 26th April 1988 by the Coroner. The baby had been found collapsed, ?dead in his cot by his mother at 11.30 pm on 20th April 1988. He was certified dead on arrival at the Accident and Emergency Department at 00.20 hours on 21st April 1988. The baby had been taken to the hospital by ambulance accompanied by his mother.

History to the pathologist

This baby was found dead on arrival at Derbyshire Childrens Hospital. He had died in his carrycot whilst in transit home from a shopping trip. He had appeared fit and well prior to undertaking the journey. There was no inquest.

Home circumstances

This was the second child of a 33 year old gravida 2. It was an unplanned pregnancy. The mother had previously been employed as a civil servant and the father was employed as a motor mechanic. They had been married 10½ years and had lived in the same house all their married life. Their first child was a female born in 1984. She had been of low birth weight and had feeding difficulties. Although she was prone to upper respiratory tract infections both she and the family had been well in the weeks preceding the baby's death. The family owned their two bedroomed centrally heated home which was rather cramped. They had their own car and telephone. The house was situated five miles from the General Practitioner's surgery and within easy reach of the bus stop. They had no financial difficulties and they were not known to the Social Services Department. They were not on any of the At Risk files. The family had been registered with the same General Practitioner for 10 years. The mother had a history of jaundice at age 17 years and had a benign lesion removed from her right breast. She had a history of frequent vaginal candida. She was obese and was a hay fever sufferer. She did not smoke and rarely drank. The father had had pneumonia after birth. He came from an atopic family and had his tonsils and adenoids removed as a child. In June 1987 he became acutely ill with Pneumococcal pneumonia and septicaemia. This caused him to stop smoking.

Ante-natal and Obstetric care

This was an unplanned pregnancy. The mother's first ante-natal attendance was at six weeks and ante-natal care was shared between General Practitioner and hospital. There were eight visits to the General Practitioner and three to the hospital. The only complication of pregnancy was swollen ankles and excess weight gain towards the end of the pregnancy. Alpha-foetoproteins were normal and foetal scans at 17 and 13 week were normal. The pregnancy went full term. There was no foetal stress in labour but an emergency Caesarian section for cephalo-pelvic disproportion was performed. The mother was discharged with wound infection and anaemia on the eighth day. She was prescribed iron and antibiotics.

The Baby

A male child. Date of birth: 9th February 1988.
Date of death: 21st April 1988 aged 10 weeks. Birth weight: 3.33 kilograms.
Length: 54 centimetres.
Head circumference: 36 centimetres.
Apgar scores at 1 minute and 5 minutes were 1 and 8 respectively.

He was resuscitated by the clearing of airways and oxygen by mask. He was rather snuffly after birth, but he did not require treatment in the Special Care Baby Unit. He was prescribed antibiotics while in hospital and he was discharged home with his mother on the eighth day. He was fed on SMA Gold Cap. He was a good feeder with a regular feeding pattern established by four weeks. At eight-nine weeks rusk and Milupa cereal were introduced. The baby thrived and grew quickly. He was seen at six weeks by the General Practitioner for his developmental paediatric examination.

Events leading to death

The baby had seemed well and had had his first diphtheria, tetanus and polio immunisation. The baby and his elder sibling spent the latter part of the day with their paternal grandparents while the parents went shopping. The family returned home by car in the late evening, approximately a half hour car journey. The baby was strapped in the back seat in his carrycot, being fully clothed and covered with two blankets and a cot cover. He was alive at 10.00 pm at the commencement of the journey. On arrival home he appeared to be sleeping and was put down as he was. The little girl was put to bed and the shopping was unpacked. At 11.30 pm the mother went to feed the baby and found him dead. The father tried to resuscitate the child while the mother

called an ambulance which arrived very quickly. The mother went in the ambulance with the baby and the father followed by car.

Pathology

This was a well nourished infant with a slight perioral dermatitis. There were no signs of injury. The larynx and trachea were mildly congested. There were gastric contents present in the trachea. The lungs were well aerated. There was no lymphadenopathy. There were no pathological findings to account for this baby's death, although very minor changes in the trachea and lungs were observed.

Case conference summary

This was a thriving baby who had not caused concern to either professionals or parents. There was good parental supervision. The family were well supported. There were no immediate financial problems. The family had all been well prior to the baby's death. The baby was perhaps over-clothed during the car journey. We did not know whether this baby had died on the journey home or after he had reached home. He was very wet when he was found. He had had DPT and polio immunisations on the day of death with no apparent reaction.

Conclusions

This was classified as a G death. There were no significant pathological findings, but no histology had been done. There was no history of illness. After full enquiry no member of the conference could suggest whether this death was preventable or not.

Addendum

The mother became pregnant again soon after the baby's death. She had a termination of pregnancy and was fitted with an intrauterine device as she could not face another pregnancy. Despite counselling and offer of extra support for her next child she has not had any further children.

CASE 53/88

Home visit: Mother refused.
Case conference: 22nd November 1989.

I was notified of this death on 3rd January 1989 by the Health Visitor and by the Coroner on 6th January. The baby had been found dead by the mother at 8.00 am on 26th December 1988. A full paediatric necropsy was ordered by the Coroner on behalf of the Home Office.

History available to the Pathologist

This was an infant of a 16 year old mother who had lived in a Social Services family centre. The infant sometimes slept in the mother's bed although she alleged that the baby had been found dead face down in his cot. The Police Surgeon had noticed that lividity did not fit the pattern of the story and she later confessed that the baby had been in the bed with her.

Home circumstances

This was the first child of a 16 year old gravida 1. The baby's father was a 16 year old who ended his relationship with the mother before the baby was born. It was an unplanned pregnancy and the mother was counselled concerning abortion versus keeping the baby. The mother was still at school when she became pregnant but had a history of truancy and disruptive behaviour at school and had been involved in glue sniffing. As her pregnancy progressed she attended a unit for pregnant schoolgirls organised by Derbyshire Education Authority. She was an emotionally deprived girl who had an unhappy childhood. She was of mixed ethnic parentage which she regarded as a big issue. As a small child she and her younger sister had been fostered on the death of her mother. This arrangement broke down in 1987 and she started stealing. The Social Services Department transferred her from her foster home to a family centre. She was further stressed by the death of her father that year and soon became pregnant. The father of the child had also had an unhappy childhood. He had been brought up by his father and had periods when he had been taken into care. Both mother and father had been known to the Social Services Department for most of their lives. The mother had been registered with the same General Practitioner for seven years but the General Practitioner knew nothing about her social history. He could only comment that her medical history was unremarkable and that after the birth of the child she had not kept appointments at the surgeries.

Ante-natal and Obstetric care

The mother first attended for ante-natal care at ten weeks. Ante-natal care was shared by the General Practitioner and the hospital. The link worker at the unit ensured that she attended for her ante-natal care. She was an uncommunicative girl with her carers, smoked and went on occasional drinking binges. Despite this she was well during the pregnancy. The alpha-foetoproteins were normal as was the routine foetoscan. The baby was born at 38 weeks gestation by spontaneous vertex delivery. There was no foetal distress. Mother and baby were discharged to the family centre under the care of the Community Midwife, General Practitioner and Social Services. She did not attend for her post-natal examination.

The Baby

A male child. Date of birth: 26th November 1988.
Date of death: 26th December 1988 aged 4 weeks.
Weight: 2.96 kilograms.
Length: 53 centimetres.
Head circumference 34.5 centimetres.
Apgar score at 1 minute was 5.

He was resuscitated with Narcan x two and admitted to the Special Care Baby Unit for a few hours for observation. He was bottle fed from birth on Cow and Gate Premium and was normal apart from a right undescended testicle.

Events leading to death

The Health Visitor visited three times and on her last visit, three days before the baby's death, the mother had been upset because the link worker from the school unit had chastised her for not feeding the baby regularly and having more interest in going out than caring for the baby. The mother had always been uncooperative with "officials" and resented being told what to do. The baby was well when seen at this visit. On the night before the baby died the mother took the child into bed with her, despite having been warned repeatedly of the dangers of this practice. On finding the baby dead she told the police that the baby had been in his cot and was lying face downwards dead. On further questioning she confessed to having the baby in bed with her all night, but the baby had been well prior to his death.

Pathology

The baby was normally developed and reasonably clean and well cared for. Post mortem examination did not reveal any signs of injury. Histology was within normal limits and no pathogenic organisms were isolated. The prominence of congestion and the numbers of internal petechiae found suggested asphyxiation, but this diagnosis could not be arrived at beyond reasonable doubt. The Pathologist certified the death as sudden infant death syndrome.

Summary

This case enquiry was incomplete as the mother would not participate herself, nor would the General Practitioner attend the case discussion. This was the child of an immature, uncommunicative mother who had no idea of good parenting. She received little in the way of family support and was in the care of the Social Services. Bonding was poor, she looked to her own needs, attending to the baby when she felt like it or was made to. This mother had not had any experience of good parenting herself. She was in a state of chronic stress with the additional crisis of her father's death and being abandoned by her boyfriend. She was dependant on professional services which she resented and did not trust. There had been a great deal of professional input from the Social Services Department, the staff of the school unit and the Health Visitor, but members of the conference felt that although individual help had been available, better communication between the different agencies would have been more beneficial to the mother.

Conclusions

By history and pathological findings this case was an E death. The conference thought that accidental suffocation had taken place and that this death was preventable. Lack of competence of the mother was the major contributing factor leading to death. More coordinated supervision could have helped. We had access to information from the school unit and Social Services which had not been available to the Pathologist or to the General Practitioner.

Addendum

The Health Visitor became increasingly involved with the mother after the death of the child, visiting for a further six months. Despite this continued input the mother's behaviour remained just the same. She did not attend for appointments and refused any form of contraception. When last heard of she had had a termination of pregnancy and had moved from the family centre to live with a new boyfriend.