Avoidable factors in child death*

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SUMMARY   There were 131 deaths among children aged between birth and 14 years during a period of 18 months in one inner London area health authority. One hundred children died in hospital, 23 at home, and 8 elsewhere. Thirty three died of congenital, 46 of perinatal, and 34 of other medical causes. There were 18 violent deaths—16 among children over 1 year of age. Medical and social information, collected from analysis of records and interview with those involved, was considered by a review panel and the following conclusions were drawn: there was scope for prevention of congenital disease or malformation in only one of 33 cases; with routine pathological investigation the cause of death in postperinatal infancy was usually unclear—more detailed pathological investigation should be routinely available for the investigation of deaths in the first year of life; there were possible management failures in four of 8 non-malignant medical deaths in older children; malignant disease was promptly diagnosed; fatal accidents were almost entirely restricted to the children of families under marked psychosocial stress; and there was some evidence of faulty communication between health agencies and of the inappropriate routing of emergency admissions.

Some analyses of child deaths[1-5] have been concerned with the effect of adverse social factors; others have specifically considered perinatal,[6 7] post perinatal[8] or infant[9 10] mortality, malignant disease,[11-13] or accidents.[14-16] We report a study in which the emphasis was on detailed retrospective investigation of the factors leading up to each death of a child resident in a defined area of London during one 18 month period.

In 1978 the mortality rates among infants and children aged 1–14 years in this area (child population under 14 years 58 000) were higher by 48 and 72% respectively than regional and national figures.[17] We did not aim to examine these differences per se, but rather to investigate whether there were any potentially avoidable factors in the chain of events leading up to the deaths.

Subjects and methods

With the following exceptions—9 sudden infant deaths (SIDS) investigated as part of another study;[18] all babies dying within 24 hours of delivery; and low birthweight (less than 2500 g) infants dying before discharge from hospital—all children under

the age of 14 years resident in one London area health authority who died during an 18 month period were included. Each death was notified by the registrar of births and deaths, and a questionnaire, covering medical and social information and a detailed account of events leading to the child's death, was completed for each child. Data was collected from the records of any hospital where the child had been seen, the general practitioner, the community paediatric services and school, and the coroner, and this was supplemented whenever possible by interviews with involved doctors, health visitors, social workers, and the bereaved parents. Parental interviews were conducted by YS at the child's home. A parental interview took place only if there was prior agreement by the child's general practitioner and by the parents. It included taking a detailed history of the child's life and of events leading up to the death. All informants were asked whether they felt there were any avoidable factors in the chain of events leading to the death.

A committee consisting of two general practitioners, two consultant paediatricians, an area specialist in community medicine (child health), an area nurse (child health), a professor of clinical epidemiology,

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Table 1 *Classification of death by cause and age*

Age	Group I medical		Group II perinatal		Group III congenital	Group IV accident and violence	Total
	General	Malignancy	LBW	Term			
0–1 year	18	1	41	4	19	2	85
1–14 years	8	7	—	1	14	16	46
Total	26	8	41	5	33	18	131

LBW = Low birthweight.

an assistant director of social services, a coroner, and the medical officer to the study met regularly to review completed records and was asked to decide in each case whether, with the benefit of hindsight, the child's management seemed to have been less than optimal.

Results

During the 18 month period of the enquiry the total number of child deaths was 131 (see Table 1) and 65% (85 of 131) occurred in infants (ie children less than 1 year of age). The deaths were classified by main cause into four groups and then sub-divided into infants and children 1–14 years. Group I, 'medical', was subdivided into 'malignancy' and 'general' causes which included infections, conditions such as asthma, epilepsy, and SIDS. Group II comprised 'perinatal' causes and although low birthweight infants constituted most of this group, five term infants were also included. Group III encompassed deaths classified as 'congenital' which were, in the main, malformations and inborn errors of metabolism. 'Accidents and violence', group IV, were the commonest cause of death in the older children. Seventy six per cent (100 of 131) of the children died in hospital, 23 at home, and 8 at the site of an accident.

Group 1—medical (34 cases).

General (26 cases). In this group there were 8 children aged 1–14 years. Their diagnoses were status epilepticus, diabetes, avian intracellulare TB, epiglottitis, asthma, pneumocystis pneumonia, peritonitis from a ruptured appendix, and encephalitis possibly from Reye's syndrome.

A firm diagnosis was made in all these children either during life (first five) and confirmed in three cases at necropsy, or only at necropsy (last three). The history of the diabetic showed some features suggestive of antemortem hypoglycaemia, perhaps contributing to an unexpected necropsy finding of 'haemorrhagic pneumonia'. No necropsy was performed on the child who died of acute epiglottitis.

Although most of these children had potentially treatable illnesses, few problems of medical management were identified which might, if better handled, have altered the outcome. Management was less than optimal in a 12 year old girl who had had undiagnosed asthma for many years.[19] The treatment of the final episode probably failed because of inadequate counselling by an inexperienced trainee GP, insufficient domestic supervision, delayed admission to hospital, and difficulty with attempts at resuscitation in casualty by junior staff. There was apparent failure to recognise the severity of the condition of a 10 year old girl, a known epileptic on anticonvulsant treatment, who was admitted to hospital in status epilepticus and who continued to convulse intermittently with cyanosis and rising pyrexia until she died 18 hours after admission. An 18 month old boy in hospital with oxygen dependent pneumonia for three weeks was incompletely investigated for immunodeficiency: at necropsy interstitial pneumonia due to *Pneumocystis carinii* was found. There were no definite weaknesses in the care of the remaining children in this age group.

There were 18 general medical infant deaths. Ten diagnosed as SIDS on their death certificates were, with one exception, covered by another investigation limited to sudden infant deaths[18] and are not considered further in this report. One SIDS occurred after that date in the period of this study. The child's parents resided in accommodation for the homeless, were known gypsies, and efforts to trace them after the death were unsuccessful.

Five other infants were admitted already dead or in extremis. Two of them died unexpectedly at home and were initially reported as cot deaths, but the cause of death was ascribed at necropsy to respiratory tract infection. Three were regarded as dead or almost dead by their parents and resuscitative measures were discontinued after one hour in two and five hours in one. The preceding symptoms of these five infants were seen as trivial by the parents, and only one had been seen by the family practitioner; the pattern of illness seen in these babies was very similar to that described in SIDS. In four the final diagnosis of respiratory tract infection was

made without histological examination or positive cultures. In one certified as 'septicaemia' there were negative cultures.

A further three infants also arrived at hospital gravely ill but lived rather longer. Encephalopathy was the recorded diagnosis in a three month old girl, admitted severely dehydrated after a day of diarrhoea and vomiting under the care of an unregistered baby minder. She died after four days intensive care: necropsy permission was refused but a liver biopsy was normal. One case was considered to be Reye's syndrome after chicken pox. A third infant, five weeks old, with a persistent umbilical discharge was seen by a clinical medical officer, a general practitioner, and a paediatrician and was finally admitted with pyaemia. A large paraumbilical abscess was found at necropsy. Gram negative rods, 'presumed Pseudomonas', were cultured from the blood, cerebrospinal fluid, and umbilical discharge. When all the data had been reviewed, it seemed, with the exception of the last case, that the application of a particular diagnostic label was often unsatisfactory.

Malignant disease (8 cases)
There were two neuroblastomas, two lymphoblastic malignancies, and one each of ependymoma, hepatoblastoma, rhabdomyosarcoma, and Letterer-Siwe disease. On admission all 8 children had signs of advanced disease: two were very anaemic, two had proptosis, two had acute abdominal pain, one was lethargic with an abdominal mass, and one presented with ataxia.

In 6 children the symptoms had been present for less than three weeks before referral. In three of these there had also been more long standing symptoms which were of uncertain relevance: constipation in a child with lymphoma, who was also multiply handicapped; excessive crying in the hepatoblastoma (the infant of an anxious mother with an appalling obstetric history); and a functional systolic murmur in a boy with acute lymphoblastic leukaemia.

The diagnosis was established within one week of hospital admission in 6 of the 8 children. In the child with Letterer-Siwe disease an early clinical diagnosis was only confirmed by skin biopsy after some weeks, and exploratory laparotomy was deferred in the multiply handicapped child thought to have acute or chronic constipation. No serious errors in management were apparent in the malignant group..

Group II—perinatal (46 cases). Forty one deaths were associated with low birthweight but five term infants were also included in this group—two who died of unexplained intracranial haemorrhage on day 3 of life and three who died of the later effects of asphyxial delivery.

Group III—congenital (33 cases). Fifteen (45%) deaths were caused by congenital heart disease (including one Down's syndrome with endocardial cushion defect); two had been lost to follow up (at 3 and 13 years after Mustard's operation for transposition of the great arteries and total repair of a truncus arteriosus, respectively) and two, who had been passed as normal on routine postnatal examination, died unexpectedly at home at four days and at 6 months of age (findings at necropsy were 'cor triloculare' and 'multiple atrioseptal defects').

The following abnormalities were present in the remaining 18 children: central nervous system abnormalities (CNS) (five cases)—meningomyelocele (three), hydrocephalus (one), cerebral malformation (one); other malformations (four cases, one each)—Crouzon's syndrome, first arch syndrome, unclassified multiple defects with normal chromosomes, osteogenesis imperfecta; inborn errors of metabolism (IE) (9 cases)—cystic fibrosis (two), neurolipidosis (two in one family), Tay-Sach's, Nieman-Picks, Leigh's, Schwachman syndrome, unclassified neurodegeneration (one each).

There were few missed antenatal diagnoses. Six of the 9 children with IE were the first affected in the family, the only exceptions being the two siblings with neurolipoidosis and the child with unclassified neurodegeneration, whose sibling had died before the study began. In both these cases there was only one year's difference in age between the older and younger sibling, and the diagnosis was made too late for genetic counselling to be useful.

Of the five children with structural CNS abnormalities, four had neural tube defects. The mother of one had had a raised serum α fetoprotein (AFP) at 20 weeks' gestation which was not confirmed on repeat estimation or at amniocentisis; two others had not been offered an AFP estimation, apparently through administrative oversight; and one patient had arrived from abroad when pregnancy was already too advanced for the test to be useful.

Group IV—accidents and violent deaths (18 cases). This group comprised 13% (18 of 131) of the total deaths, and 35% of those over the age of 1 year, the biggest single group at that age. Parental interviews were only obtained in 8 cases; in a further 6 information was available from agencies who had

Table 2 *Accidental or violent deaths*

Cause of death	No	Child known to social services	Previously in care or fostered	Before child's death		One parent in care during childhood
				Mother battered	Deserted by father	
Traffic accident						
Road	5	3	2	1	1	1
Railway	2	2	—	—	1	—
Deliberate violence:						
By family member	3	2	—	1	1	1
By stranger	1	1	1	—	1	1
Drowning	3	2	2	1	2	1
Fire	2 (siblings)	2	2	—	2	—
'Suffocation' (in mother's bed)	1	—	—	—	—	—
Accidental strangling	1	—	—	—	—	—

been concerned with the children before their death. Little information was available on one road traffic accident and one drowning where the parents declined interview, on one child thought to be the victim of deliberate violence, and on the suffocated child whose general practitioner strongly advised against contact being made with the parents.

It will be seen from Table 2 that 12 of 18 children were known to the social services department before death. Four were, or had previously been, on a 'non-accidental injury register'. Interestingly, one of these died in a railway accident; two drowned while unsupervised, one in a canal, the other in the swimming pool of his foster parents; and only one died from adult violence. In the last, numerous attempts to gain access to the family had been unsuccessful and, as there were no known legal grounds for intervention, the home was only entered after the child's death (from haemopericardium). Living conditions were then found to be so bad that both her siblings were taken into care. Another infant had been regarded by doctors, health visitors, and social workers as being at considerable risk, but was not on the register; she died of a fractured skull during a two to three week period when workers in close contact with the family happened to be away. Her two siblings had already, before her death, been received into care.

Seven of the children had previously been either taken into care or fostered, and the siblings of a further two had also been fostered. Three of the mothers had been assaulted by a cohabitee and a further mother had been admitted for psychiatric care before the child's accidental death and was being followed up by a hospital social worker. The family of another child (who ran into the path of an oncoming train) had been assisted by social services for many years with financial and housing problems.

Eight of the families had been deserted by their fathers before the death and one or other of the parents of four children had themselves been in care in early childhood.

Three of the children and four of their siblings had previously been admitted to hospital for accidents including burns (three); injuries, ie after a fall from a window (one) and car accident (one); and overdoses (two).

There was evidence suggesting a lack of parental supervision at the time of the fatal event in two boys who died in separate incidents while playing on railway tracks, in three children who drowned, and in the girl who was murdered. A three year old who drowned in a canal had on a previous occasion been found wandering on the side of a 6 lane highway at 10 pm. Specific environmental hazards were identified in a few cases, eg defects in the fence alongside a railway track and along a canal, and the lack of a barrier between road and play alley on a housing estate.

The death of one infant from 'suffocation' might perhaps have been more correctly classified as SIDS. A boy aged 13 years strangled himself, apparently inadvertently, while playing with a dog lead.

Discussion and conclusions

The outstanding feature of the deaths in group I was the high incidence of inconclusive diagnosis in infancy—a similar finding to studies in Glasgow, Newcastle, and Sheffield.[10][20][21] Eighty three percent (15 of 18) either died at home or were admitted moribund; 10 infants were certified as SIDS and an additional five deaths were sudden, unexpected, and, on the data available, not clearly distinguishable from that syndrome. Three were admitted already extremely ill but survived a few days. Necropsies

had been performed in 17 of the 18 infants and in only one child, a case of pyaemia, was a firm aetiological diagnosis made. This may have been due in part to a genuine lack of pathological evidence but, in more than half the cases, necropsy reports lacked detail and histological data. Only in the case of pyaemia did it seem clear that earlier diagnosis might have led to a different outcome.

In the deaths among older children, the diagnosis was clear in most, and possible errors of management were evident in a few. For example, the child with pneumocystis pneumonia was potentially treatable. It is striking, however, that subsequent to this study a further sibling of this child died at three months of age of severe combined immunodeficiency and infection. Avoidable elements may have contributed to the asthmatic, epileptic, and perhaps diabetic deaths. Malignant disease seemed to be promptly diagnosed.

Although full family studies were not always available, of the 33 deaths from congenital causes, 29 seemed to have been the first occurrence of the defect. This left little scope for detection during pregnancy except by population screening. In three mothers in whom termination of pregnancy might have been offered for NTD, antenatal diagnostic tests failed to establish the diagnosis in two, and were not offered in one.

Among the violent deaths, the level of social disorganisation was striking. A comparative study of children from a random population sample was not undertaken, but of 18 children in the present study dying from non-violent causes and matched as closely as possible for age and sex, only three were known to social services—two for medical appliances and one family for housing problems. None of the control children had been in care. This contrasts with the observation that 10 of 18 children who died of accidents or violence had at some time been in care, fostered, or been on the 'at risk' register. Often the lives of their parents had also been disturbed and many fathers had deserted the family. With the benefit of hindsight, it seemed that one child on the non-accidental injury register might have survived had she been in care, but against this must be set the child previously in care who drowned three months after being sent to an apparently excellent family for fostering.

It is difficult to avoid the conclusion that there is a strong link between fatal accidents in children and the circumstances of their families, though it may not be possible to establish a causal relationship. A similar association has been found relating morbidity from accidents to maternal depression, 'vulnerable families', and accident repeaters[22-25]; but not in all studies.[16] In our population several of the children had previously been admitted to hospital for accidents, as had their siblings. Furthermore, physical hazards, both domestic and outside the home,[26 27] seemed less clearly responsible than lack of adult supervision.

Three other general points were apparent. One was inappropriate routing of emergency admissions. Three critically ill children were initially transported by ambulance to hospitals lacking suitable facilities and then transferred to other nearby hospitals.[28] Although the delay seemed unlikely to have affected the outcome in the particular cases, it is easy to see how fatal delays may occur. Secondly, continuity of shared care between hospitals or other health agencies was sometimes faulty. This applied to two children who had received corrective surgery for congenital heart defects but were lost to follow up until sudden death in one and sudden deterioration before death in another: their life expectancy was limited but lack of supervision may have contributed to their early death. Finally, many of the interviewed bereaved parents wanted clearer information about the child's death and more frequent visits. Every parent seen expressed gratitude for the opportunity of an interview.

A regular system of enquiry into child deaths is a useful part of child health surveillance. In any further enquiry we would suggest prospective involvement of a paediatric pathologist and more detailed consideration of the antenatal period.

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Commentary

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Child mortality rates in this country are higher than in Scandinavia. When the causes of death are analysed we find that the differences occur in the groups of 'indefinite symptoms', 'acute infections', and 'cot deaths' while with congenital heart diseases and meningitis the rates are almost the same throughout Northern Europe. Hospital statistics suggest that the level of hospital paediatric care in this country is equal to that anywhere, but that we fall behind on the home front.

Identifying preventive factors in the community is difficult: it is not easy to get basic information. The above study by Sherman, Matthew, and Boyd illustrates two major problems—they were only able to interview the families of 8 of the 18 children whose deaths were 'accidental' and they were limited by inadequate information from necropsies. Such studies are asking new questions requiring new skills. It is no longer sufficient to know that a child died of or with pneumonia or pneumococcal meningitis. If a boy has died with meningitis, how long was he ill before he died? How seriously ill? Did he have symptoms? Were they recognised? What was done about them? Was the response adequate and by whom? We need measurements of the threshold of action for parents—the amount of understanding parents have of instructions. If a child died from 'an accident', we need an assessment of the level and consistency of child supervision and restraint. Is the need for some of these social measurements as great as for some new laboratory test?

Sherman, Matthew, and Boyd rightly say that they need the involvement of a paediatric pathologist. Paediatric pathologists are very 'thin on the ground' and already involved in diagnostic procedures needed for hospital patients. The pathologist contributing to a social work team needs to develop new tools to answer new questions. It helps the investigation only a little to know that a child died with a pneumonia. Why did this child die and not the 100 others who had the same infection? How sufficient was the pneumonia to explain death? What was the immune or biochemical state of this child that made this degree of pneumonia fatal? How long had the child been ill and how severely ill before death? These questions need to be estimated irrespective of the given history. This calls for a new paediatric pathology input just as for a new social paediatric input.

It is hoped that this London group will strengthen their team and continue their work. There is not the personnel to carry out similar studies everywhere and although a few other groups exist, more are needed.