Short reports

Deaths in cots are not always cot deaths

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SUMMARY Agonal histories were traced for 1182 of 1782 postneonatal deaths in Sheffield between 1947 and 1979. Twenty eight per cent of these deaths were histologically inexplicable, 19% were from infections and 15% from malformations. These sudden infection and malformation deaths were explicable, yet by some definitions they would be considered as cot deaths. Varying definitions may lead to confusion, hindering the search for aetiological mechanisms.

After the first conference on causes of sudden death in infants in 1963,¹ a rapid increase in the number of these deaths was recorded and numbers of deaths from gastrointestinal and respiratory infections declined sharply at about the same time.² This may have been an artefactual diagnostic 'fashion' which makes interpretation of epidemiological data difficult. It was hoped the final report of the Departments of Health and Social Security multicentre study into postneonatal mortality would clarify this because of the standardisation of necropsy and clinical data. A recent report, however, from that study about the pattern of illness before cot death stated that 'Babies who died unexpectedly were eligible for inclusion . . . if . . . their death was not explained by life threatening congenital defects, the complications of prematurity, neoplasia, or violence'.³ Because this definition may include many infectious diseases and some malformations a search was made through records of Sheffield child deaths since 1947 to determine the magnitude of the possible errors introduced by this.

Subjects and methods

All original death certificates, necropsy reports, and available coroner's records were examined for every Sheffield child aged more than 1 month and less than 1 year who died between 1947 and 1979. Details of Sheffield children who died outside the city were traced through records kept by the medical officer of health. Children were included in the

study if their given home address was in the official Index of Sheffield Streets current at the time of death. A detailed description of the methodology used to minimise observer variation (especially changes in diagnostic sensitivity) has been previously described.⁴

Details of each death and necropsy were reviewed and the cause of death classified into one of seven categories (infection, sudden infant death syndrome, malformation, tumour, trauma, epinatal, and other—which were almost all deaths in children with cerebral palsy). Deaths due to events at the time of birth but occurring after the perinatal period have been classified as epinatal. Where there was no clinical, historical, or pathological evidence of more than 24 hours' illness the death was classified as a sudden infant death. Additionally some sudden infant death victims had had a mild coryzal illness of more than 24 hours' duration and some were reported to have died in hospital (usually in the casualty department) but all deaths were clinically unexpected and there were no consequential pathological findings despite detailed histological study—that is, these were anatomically completely unexplained deaths.⁵ It is not clear what conditions are embraced by 'life threatening congenital defects'³ and it is equally difficult to determine, after death, that a malformation such as cleft palate or uncomplicated patent ductus arteriosus might not have been lethal in different circumstances. Lethal malformations were arbitrarily classified as those where a child so afflicted would not normally be expected to survive the postneonatal period.

Results

There were 1782 deaths in the postneonatal age group (1 to 11 months) during the 33 years studied but in 600 there was doubt about the child's clinical state before death. Of the remaining 1182 deaths, 837 (71%) were sudden (that is, less than 24 hours' illness) and nine of these had brief prodromal symptoms. Altogether 552 of the 741 deaths which occurred outside hospital were known to be sudden. Details of the 1782 deaths are given in Table 1 and

Table 1 Postneonatal deaths in Sheffield, 1947-79

Cause	Sudden	Expected	Not known	Sudden home	Total
SIDS					
No prodroma	452				
Prodroma	9	—	30	377	491
Malformation					
Lethal	52	109	95	22	256
Other	76	80	39	36	195
Infection	157	84	374	80	615
Epinatal	11	14	7	4	32
Trauma	31	1	1	14	33
Tumour	4	13	4	1	21
Other	45	44	44	18	139
	837	345	600	552	1782

SIDS=sudden unexpected infant death syndrome.

Table 2 Postneonatal malformation deaths by system, Sheffield 1947-79

	Total	Lethal	Home	Sudden	Sudden lethal
Central nervous system	105	91	27	9	9
Cardiovascular system	172	125	40	63	27
Respiratory system	16	9	3	6	4
Alimentary system	53	10	9	11	1
Urogenital system	20	4	9	12	4
Chromosomal	52	10	13	17	1
Other	33	21	9	10	6
	451	270	110	128	52

Table 3 Postneonatal infection deaths, Sheffield 1947-79

	Total	Sudden	Home
Gastroenteritis	243	65	53
Pneumonia	183	35	78
Pertussis	50	—	7
Meningococcaemia	29	21	5
Tuberculosis	11	—	—
Bronchiectasis/empyema	23	11	9
Measles	9	1	4
Epiglottitis	1	1	—
Meningitis	12	6	3
Encephalitis/polio	18	3	—
Other	36	14	14
	615	157	173

of the deaths attributed to malformation and infection in Tables 2 and 3.

Discussion

The concept of a sudden infant death syndrome arose when, after reductions in deaths from infectious disease, it was found that a sizeable proportion of infant deaths occurred in children who had seemed to be completely well and in whom no consequential pathological features could be found.⁶

These sudden deaths are dealt with by the coroner and forensic pathologists who are concerned primarily with the identification of unnatural deaths. Having excluded unnatural causes, the problem of labelling these deaths remained. Emery argues that sudden infant death syndrome, cot death, crib death, and other synonyms were invented for the convenience of considering these deaths as a single entity.⁷ The possibility of a multiple aetiology for sudden infant deaths has been known to forensic pathologists for many years.^{8 9}

The description and publicity of the sudden infant death syndrome in the early 1960s was followed by a rapid increase in the number of these deaths registered around the world.⁶ Some of this increase was probably correction of the registration of deaths which, because of the previous lack of a suitable diagnostic label, had been recorded as respiratory or gastrointestinal infections. Doubts have been expressed, however, about the quality of paediatric certification and the continuing increase in registrations of sudden infant death syndrome deaths.²

Seventy six of the 128 sudden deaths caused by malformation in this series were from conditions not usually considered as life threatening congenital defects (Table 2), but they could not be considered as inexplicable deaths: nor could the 157 sudden

deaths from infection (Table 3) be classified as inexplicable. Thus by the definition of Stanton and Oakley,³ among the 837 sudden postneonatal deaths in this series, 739 would be cot deaths of which only 62% were histologically unexplained.

Fear of cot death haunts many young parents. Inflated numbers of cot deaths do not ease their anxiety. Misdiagnosis also hinders attempts to understand aetiological mechanisms. Published reports on the sudden infant death syndrome are confused by variations in definition. Meningococcaemia and leukaemia may both present with purpura but this does not mean that they share a common aetiology, and considering them as the same can only confuse.

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Successful treatment of fetal atrial flutter and congestive heart failure

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SUMMARY Fetal supraventricular tachycardia may cause congestive heart failure, hydrops fetalis, and intrauterine death. Tachycardia in a fetus of 34 weeks' gestation was diagnosed as atrial flutter by echocardiography, and was successfully treated by giving the mother digoxin.

Since visualisation of the fetal heart by echocardiography has become available, prenatal diagnosis of congenital cardiac anomalies and fetal arrhythmia has been possible.¹⁻³ We have evaluated the structure of the fetal heart and the great vessels by two dimensional echocardiography, and diagnosed arrhythmia by simultaneously recording the motion of the atrial wall and the ventricular wall of the fetal heart by M mode echocardiography. Certain fetal arrhythmias such as supraventricular tachycardia or A-V block have clinical importance, since they may be the cause of congestive heart failure and evaluation and treatment of these conditions are, therefore, necessary.

Case report

The mother, a 28 year old gravida 1, para 0, had an uneventful pregnancy until week 34 of gestation when she complained of abdominal pain and was referred to Kurume University Hospital. Fetal echocardiography was carried out and showed an atrial rate of 460 beats/minute and a ventricular rate of 230 beats/minute, without any structural anomalies of the heart (Fig. 1). Atrial flutter was diagnosed. Digoxin (0.5 mg) was given to the mother and further doses of 0.5 mg and 0.25 mg were administered three and 12 hours later. The fetal heart rate was continuously monitored during this time and repeated electrocardiograms were made. Five hours after the third dose of digoxin, the fetal heart rate dropped from 230 to 135 beats/minute: at that time the digoxin concentration in maternal venous blood was 1.4 ng/ml. The mother was treated by a daily oral dose of 0.25 mg of digoxin and showed no signs of digoxin intoxication. Echocardiography, undertaken when the fetal heart rate had returned to normal showed massive pleural