Intra-alveolar haemorrhage in sudden infant death syndrome: a cause for concern?

N Yukawa, N Carter, G Rutty, M A Green

Abstract

Background—The “Back to Sleep” campaign in 1991 resulted in a dramatic decrease in the incidence of sudden infant death syndrome (SIDS). The proportion of presumed SIDS deaths being actually suspicious deaths from airway obstruction is likely to have become relatively greater. There is usually little pathological evidence to suggest smothering, but intra-alveolar haemorrhage appears to be more prominent in cases where interference with the airway is suspected.

Aim—To attempt to quantify intra-alveolar haemorrhage to see whether it could be used as a marker to distinguish between smothering/overlaying and SIDS. **Methods**—Intra-alveolar haemorrhage was quantified using digital image analysis on haematoxylin/eosin stained sections taken from the lungs of 62 consecutive infants who had died suddenly and unexpectedly. Cases were initially classified according to the original cause of death. After quantitation, the case histories were critically reviewed. Three pathologists independently made microscopic assessments of the degree of intra-alveolar haemorrhage in the first 24 cases to see whether these accurately reflected the quantitative results.

Results—73% of those infants with a history suggesting possible involuntary overlaying and 45% of those with a history suspicious of smothering had significant intra-alveolar haemorrhage (> 5% of total lung surface area assessed). From the history, the cause of death in 11 cases initially classified as SIDS would better have been given as “Unascertained.” Simple microscopic assessments underestimated the true extent of the haemorrhage in 33% (8/24).

Conclusions—If a moderate degree (at least 5%) of pulmonary parenchymal haemorrhage is observed, this may be an indicator of airway obstruction for a significant period, either from overlaying or possibly smothering. The diagnosis of SIDS may be being used inappropriately in such cases.

(J Clin Pathol 1999;52:581-587)

Keywords: sudden infant death; smothering; overlaying; intra-alveolar haemorrhage

ing event (ALTE). Since smothering is so difficult to distinguish from sudden infant death syndrome (SIDS) on a pathological basis,² it is likely that some of these infant deaths were ascribed to SIDS. Southall's group, with their use of covert video surveillance, has graphically highlighted the problem.³ Meadow has long recognised the problem of smothering as a component of Munchausen's syndrome by proxy (MSBP),⁴ although he has concentrated upon critically reviewing the histories of such deaths. His observations have been supported by those of Byard *et al.*⁵

Reports on the pathology of SIDS⁶⁻¹¹ observe that some degree of intra-alveolar haemorrhage is common in these cases. Grellner and Madea¹² noted that intra-alveolar haemorrhage was common in asphyxial deaths. However, we have found little published guidance as to how much intra-alveolar haemorrhage is “acceptable” to be compatible with a satisfactory diagnosis of SIDS.

In our own practice, we have gained the impression that intra-alveolar haemorrhage is more prominent and extensive in cases in which there were worrying circumstantial features suggesting that the child might have been smothered or overlaid. In order to determine whether this haemorrhage is a significant marker of a suspicious death, we reviewed a series of infant deaths and quantified the intra-alveolar haemorrhage using digital image analysis (DIA).

Methods

From the archives of the department of forensic pathology, we identified 62 consecutive cases of sudden unexpected infant death. Age and sex were noted and the cause of death given in the necropsy report was used to place each case into one of five categories: SIDS, unascertained, asphyxia, infection, and injury/trauma. The term “asphyxia” was used in its broadest sense for initial grouping of the cases.

Of the five haematoxylin and eosin slides of lung available in each case, three were randomly selected for each of the 62 cases. To assess the degree of intra-alveolar haemorrhage, each slide was viewed through a 4 mm square grid using a ×20 objective lens on a Nikon microscope. At 4 mm intervals, an alveolus was randomly selected and the area of the alveolus was measured by tracing around its on-screen image with a mouse linked to an Acorn Archimedes computer equipped with Digit Image Analysis software. The area of haemorrhage within the same alveolus was then recorded in a similar manner by tracing around the area of intra-alveolar red cells. The

Department of
Forensic Pathology,
University of Sheffield,
Medico-Legal Centre,
Watery Street,
Sheffield S3 7ES, UK
N Yukawa
N Carter
G Rutty
M A Green

Correspondence to:
Dr Carter.

Accepted for publication
3 April 1998

In recent years, there has been increased awareness that some women deliberately smother their children.¹ A proportion of these infants may present with an acute life threaten-

process was then repeated for other alveoli until the section had been mapped. The software calculated the extent of the intra-alveolar

haemorrhage, expressed as a percentage of the total alveolar area, using the following formula:

$$[\text{sum of areas of red cells in alveoli} \times 100] / \text{sum of area of alveoli}$$

For each case, the mean of the three results available for each slide was taken. The results were expressed graphically.

After quantifying the haemorrhage, the case histories were critically reviewed (blind to the DIA results) to discover any suspicious features in the history that might have indicated an unnatural death—for example, an atypical age for SIDS, multiple cot deaths in a family, sharing a bed with one or more adults, or evidence of non-accidental injury. Key historical features in each case were tabulated, together with the DIA results.

From the first 24 cases recovered from the archives, three pathologists (NY, NC, GR) were asked to review the same three slides on each case that were subsequently used for DIA. Each pathologist was asked to grade independently any haemorrhage they noted as mild, moderate, or severe (examples are shown in fig 1). No elaboration was made with regard to the actual area of haemorrhage that should be regarded as mild, moderate, or severe. The results were compared to assess concordance between the subjective analyses. The highest graded assessment for each case was compared with the DIA result.

Statistical analyses were undertaken using the χ^2 test with Yates correction.

Results

There were 40 male and 22 female infants, ranging in age from two weeks to 14 months.

SIDS CASES

There were 29 cases initially classified as SIDS (table 1). As the histories were reviewed in the light of the DIA findings, a pattern of similar history types began to emerge. Accordingly, the “SIDS” cases were grouped into subcategories of “typical SIDS,” atypical history, cardiac problems, infection, ?overlaid, and ?non-accidental injury (NAI). These groupings are shown in table 1, together with key features from the history. For statistical purposes, cases 1–18 were regarded as probable SIDS and χ^2 tests were applied using these results as the control group.

The quantitative DIA results are shown in table 2 and fig 2. There was a notable distinction between cases which showed intra-alveolar haemorrhage occupying less than 5% of the total lung area assessed and those showing more. These quantitative results accorded well with the subjective assessments of the extent of haemorrhage made by the three pathologists (table 3). Overall, a subjective result of “mild” corresponded to less than 5% haemorrhage as measured by DIA. As a result, we took 5% as the cut off point in deciding what degree of haemorrhage we would regard as being significant.

Following quantitation, we examined the histories of all the SIDS cases. Six of them had

Figure 1 Examples of degrees of haemorrhage in subjective assessments: (A) mild; (B) moderate; (C) severe.

Table 1 Cases of sudden infant death syndrome (SIDS), with subgroups according to type of history

Case	Age	Sex	History	Resusc	Type of history	Path assess	DIA assess (%)
1	8 m	M	Found dead in cot	N	"True SIDS"	–	1.2
2	8 w	M	Premature at 32 w. BW 1.43 kg	N	"True SIDS"	1	0.7
3	12 m	M	Found dead in cot	N	"True SIDS"	–	1.9
4	3 m	M	Found "collapsed" in cot. ICU for hypoxic brain damage	Y	Atypical	–	2.3
5	3 m	M	Placed on bed. Found not breathing a few minutes later	Y	Atypical	1	2.0
6	7 w	F	Found dead 1 h after feeding. One of twins. Bloodstained fluid in peritoneal space	Y (prolonged)	Atypical (s)	–	6.7
7	4.5 m	F	Fed and put in cot. Bloodstained fluid around nose	Y	Atypical	–	4.7
8	2 w	F	Incidental probe-patent PDA. "Appearances consistent with SIDS but atypical age"	Y	Atypical (s)	3	15.2
9	11 w	M	Soft heart murmur	Unknown	Cardiac	–	1.5
10	2 m	F	Heart murmur. Found face down in cot	Unknown	Cardiac	–	2.2
11	13 m	F	"Cold" for 1 w. Face down in cot	Unknown	Infection	–	0.3
12	4 m	M	"Feverish" for a few days before death	N	Infection	–	0.5
13	7 m	F	"Snuffly" for several days. Found dead face down in cot	N	Infection	–	1.3
14	3 w	F	Unwell with viral gastroenteritis	Y	Infection	–	1.1
15	3 m	F	"Occasional coughs and colds"	Y	Infection	–	1.8
16	7 m	F	Recent gastroenteritis	N	Infection	–	1.7
17	5 m	M	"Unwell" for several days. Found collapsed with face between mattress and cot. ICU for hypoxic brain damage	Y	Infection	–	2.9
18	10 m	M	Live baby of twin pregnancy. "Cold" before collapse	Y	Infection	–	3.5
19	6 m	M	Found dead beside mother in bed	Y	?Overlain	1	2.0
20	7 w	M	Found dead in bed with mother	Y	?Overlain	2	9.7
21	10.5 m	M	Found dead between parents in bed	N	?Overlain	2	12.1
22	2 w	M	Found dead beside mother in bed	N	?Overlain	2	20.3
23	4 w	F	Contracted infection at birth. Found dead between parents in bed	Y	?Overlain	–	34.3
24	4 w	F	Wheezy for 2 days. Found dead in bed next to aunt	Y	?Overlain	3	39.7
25	10 m	M	Minor congenital abnormalities and VSD. Numerous fresh bruises	Y	?NAI (s)	2	6.2
26	11 m	M	Undernourished. Numerous healing scratch abrasions	N	?NAI	–	3.6
27	9 m	F	Bruises on face consistent with slap	N	?NAI (s)	–	18.3
28	5 w	M	Mother had psychiatric history. Found dead with pillow over face	Y	?NAI (s)	1	3.5
29	12 w	F	Sibling died of "SIDS" at 6 months	N	?NAI	–	3.5

BW, birth weight; DIA assess, quantitative digital image analysis result (mean); ICU, intensive care unit; m, months; N, no; NAI, non-accidental injury; Path assess, subjective analysis, highest result; PDA, persistent ductus arteriosus; Resusc, resuscitation attempted after found; (s), smothering suspected; VSD, ventricular septal defect; w, weeks; Y, yes.

historical features suspicious of overlaying; five showed more than 5% haemorrhage. Five of the SIDS cases had atypical histories, and two of these showed more than 5% haemorrhage. Five had features highly suggestive of NAI (albeit not fatal): two of these (cases 25 and 27) had numerous bruises and showed more than

5% pulmonary haemorrhage; one with a history suggestive of smothering (case 28) did not show marked haemorrhage. Eight had a history consistent with some form of infection; none of these showed more than 5% haemorrhage. There was significantly more haemorrhage in the lungs of cases 19–24 than in cases 1–18 ($\chi^2 = 8.1$, $0.01 > p > 0.001$, degrees of freedom (df) = 1).

Ten of the SIDS cases were in the group randomly selected for subjective assessment. There was good interobserver concordance (table 3); in only one was there marked discordance with the quantitative results (table 1).

NON-SIDS CASES

Results for this group are given in tables 3, 4, and 5 and in fig 3.

Unascertained

This category contained 10 cases. Eight of these had histories in keeping with overlaying or smothering but there had been insufficient evidence for a criminal investigation to proceed further. Of these eight cases, three had historical features suspicious of overlaying and two showed more than 5% haemorrhage (in the third only one slide was available for DIA so the result may not have been reliable); the remaining five infants had historical features suggestive of smothering, and two of these showed significant haemorrhage. One infant had a small recent subdural haematoma, insufficient to account for death. Another presented with convulsions; the original pathologist expressed the view that the child had probably been shaken but again there was insufficient pathological evidence for the case to be taken further.

Table 2 Digital image analysis results: mean per cent haemorrhage in cases diagnosed as sudden infant death syndrome (SIDS)

Case numbers	Mean % haemorrhage	Range
1–3 ("typical")	2.53	0.7 to 1.9
4–8 ("atypical")	6.18	2.0 to 15.2
9–10 (cardiac)	2.60	1.5 to 2.2
11–18 (infection)	1.64	0.3 to 3.5
19–24 (?overlaying)	19.68	2.0 to 39.7
25–29 (?NAI)	7.02	3.2 to 18.3

NAI, non-accidental injury.

Figure 2 Digital image analysis of intra-alveolar haemorrhage in cases classified as sudden infant death syndrome.

Table 3 Subjective assessments by three pathologists (A, B, C) of extent of haemorrhage compared with digital image analysis (DIA) results

Case	Degree of haemorrhage (1–3)			DIA (%)
	A	B	C	
2	1	1	1	<5%
5	1	1	1	<5%
8	2	3	2	>8%
18	1	1	1	<5%
19	1	1	1	<5%
20	2	2	2	>8%
21	2	2	2	>8%
22	2	1	1	>8%*
24	3	3	3	>8%
25	2	2	2	>5%
28	1	1	1	<5%
36	3	3	3	>8%
37	1	1	1	>5%*
39	1	1	1	<5%
41	1	1	1	<5%
43	1	1	1	>5%*
50	1	1	1	>5%*
51	1	1	1	>8%*
52	1	1	1	>8%*
54	2	3	3	>8%
55	1	2	1	<5%*
56	1	1	1	<5%
57	1	1	1	>5%*
62	1	1	1	>8%*

1, mild; 2, moderate; 3, severe.

*Mode result disagrees with DIA.

Three of these cases were used in the subjective assessment exercise. There was little overall disagreement between pathologists, but in one case there was a marked underestimate of the extent of haemorrhage compared with the DIA result.

Asphyxia

There were 15 deaths from asphyxia. Four were from accidental hanging and one was from plastic bag asphyxia. One death followed a

convulsion witnessed only by the carer; there was no history of fits and none occurred after the presenting episode. Eight deaths resulted from smothering or overlaying, these causes of death being supported by consistent admissions by the carer. The DIA results showed that in these eight cases (two overlaid, six smothered), one showed more than 5% haemorrhage and four showed more than 8% haemorrhage. Three of the four hanging cases showed less than 5% haemorrhage. One case of admitted compression of the neck showed less than 5% haemorrhage.

Six of the 15 asphyxia cases were subjectively assessed. Again, there was good interobserver concordance, but in four cases the extent of the haemorrhage had been underestimated when compared with the DIA results.

Infection

There was one case of congenital cytomegalovirus infection that showed more than 5% haemorrhage. Three of this subclass were assessed subjectively. In one case the result underestimated the amount of haemorrhage measured by DIA.

Injury/trauma

More than 5% haemorrhage was present in two of the four cases. Subjective assessment of one of these had underestimated the DIA result.

REVIEW OF ALL CASES

All 62 case histories were reviewed in the light of the DIA results. Comparison of the “typical SIDS” group with the “unascertained” category showed that the latter had a marginally significant increase in intra-alveolar haemorrhage.

Table 4 Cases of sudden infant death not classified as SIDS (sudden infant death syndrome), grouped according to cause of death

Case	Age	Sex	History	Category	Resusc	Path assess	DIA assess (%)
30	4 m	M	Overlain; found dead between parents in bed	U (o)	Y (prolonged)	–	0.8
31	6 m	F	Mild head injury. History suggestive of suffocation	U (s)	N	–	1.0
32	3.5 m	M	Asphyxia. Sibling died of SIDS	U (s)	Y	–	4.4
33	9 m	M	Asphyxia from inhaling stomach contents. Small subdural haematoma	U	Y	–	5.5
34	6 m	F	Generalised fitting; probably shaken	U	Y (prolonged)	–	17.2
35	8 m	M	Probable suffocation with pillow	U (s)	Y	–	19.3
36	12 d	F	Probable shaking and mechanical asphyxiation	U (s)	Y	3	53.1
37	4 m	M	In bed with drunk mother. Found face down	U (o)	N	1	6.0
38	6 w	M	In bed with adult. Found dead. Probable overlaying	U (o)	Unknown	–	11.8
39	7 m	M	Allegation of smothering, later withdrawn	U (s)	Y	1	3.6
40	9 m	M	Accidental hanging, coat trapped on pole	A	Y	–	0.3
41	10 m	M	Accidental hanging, suspended from cot side	A	N	1	0.5
42	13 m	M	Accidental hanging, head wedged in cot side	A	Unknown	–	0.8
43	6 m	M	Convulsion of unknown cause. Hypoxic brain damage	A	Y (ITU)	1	1.0
44	8 m	M	Compression of neck, bruises on neck	A	Y	–	0.1
45	13 m	F	Accidental plastic bag asphyxia	A	N	–	4.6
46	6 m	M	Accidental hanging from bars of cot	A	N	–	6.3
47	10 m	M	Mechanical asphyxia with pillow and hands; confession by mother	A (s)	Y (ITU)	–	0.5
48	2.5 y	M	Mechanical asphyxia with pillow	A (s)	N	–	0.5
49	3 w	M	Overlain by parents on sofa	A (o)	Unknown	–	2.2
50	14 m	F	Mechanical asphyxia; mother pressed on face	A (s)	N	1	4.3
51	6 m	F	Failure to thrive. Inhalation of vomit due to deliberate airway occlusion	A (s)	Y	1	10.4
52	5 w	M	Overlaying. Found trapped beneath drunken father	A (o)	N	1	16.0
53	6 m	M	Inhalation of gastric contents due to airway obstruction. Shunt for brain tumour	A (s)	Unknown	–	17.3
54	19 m	M	Smothering admitted by parent	A (s)	Y	3	28.6
55	9 m	F	Viral myocarditis	Inf	N	1	0.3
56	10 w	M	Bronchopneumonia and pyelonephritis	Inf	N	1	0.7
57	13 w	M	Congenital cytomegalovirus infection	Inf	N	1	6.1
58	7 m	M	Acute bronchiolitis	Inf	N	–	3.5
59	3.5 m	M	Head injury—NAI	T	Y (ITU)	–	0.1
60	6 w	F	Subdural haematoma due to shaking	T	Y (ITU)	–	0.6
61	11.5 m	F	Head injury due to shaking and blows on the head	T	Y (ITU)	–	5.5
62	18 m	M	Head injury—NAI	T	Y (ITU)	1	11.3

A, asphyxia; BW, birth weight; d, days; DIA assess, quantitative digital image analysis result (mean); ICU, intensive care unit; Inf, infection; m, months; N, no; NAI, non-accidental injury; (o), overlaying admitted or suspected; Path assess, subjective analysis, highest result; PDA, persistent ductus arteriosus; Resusc, resuscitation attempted after found; (s), smothering suspected; T, trauma/injury; U, unascertained; VSD, ventricular septal defect; w, weeks; Y, yes; y, years.

Table 5 Digital image analysis results: mean per cent haemorrhage in cases of sudden infant death not classified as SIDS (sudden infant death syndrome)

Case numbers	Mean % haemorrhage	Range
30–39 (unascertained)	12.27	0.8 to 53.1
40–54 (asphyxia)	6.23	0.1 to 28.6
55–58 (infection)	2.65	0.3 to 6.1
59–62 (trauma)	4.38	0.1 to 11.3

rhage ($\chi^2 = 8.9$, $0.01 > p > 0.001$, $df = 1$). In 11 cases where overlaying was either admitted consistently, or was a strong possibility based on the history, eight (73%) showed more than 5% haemorrhage: six of these had originally been classified as SIDS. These cases showed significantly more haemorrhage than the “typical SIDS” cases (1–18) ($\chi^2 = 8.9$, $0.01 > p > 0.001$, $df = 1$). In 11 cases where smothering was either admitted or was a possibility, five (45%) showed more than 5% haemorrhage. This was not significant in comparison to the “typical SIDS” group ($\chi^2 = 2.72$, $0.1 > p > 0.05$, $df = 1$).

Of the five SIDS cases with atypical histories, two had more than 5% haemorrhage. Of the 12 SIDS (infection) and non-SIDS infection cases, only one (8%) had more than 5% haemorrhage.¹⁰

Overall, there was good interobserver concordance between the three subjective assessments of the extent of haemorrhage. Using the mode result, eight of the 24 assessments (33%) underestimated and one (4%) overestimated the true extent of haemorrhage as shown by DIA. There was no association between the extent of the haemorrhage and the presence or absence of attempts at resuscitation.

Discussion

SIDS was formally recognised as a cause of death in the early 1970s and gave a useful label for parents to cling to when their infant had died without any adequate pathological explanation. A convenient label is perhaps a disincentive for pathologists to look too closely into the circum-

stances of the death, especially as the histories available to them are often inadequate. Despite exhaustive research into SIDS, no single aetiology has been identified. Suggestions about sleeping position^{13–15} and investigations into cardiorespiratory,^{16–19} thermoregulatory,²⁰ and immunological^{21–22} causes have proved almost completely fruitless in explaining the deaths. One area of success has been the identification of disorders of fatty acid metabolism.²³ However, the number of children suffering from such rare disorders is very small, even though such metabolic defects are one of the very few satisfactory explanations for recurrent cot deaths.

Southall's group³ has proved beyond all doubt that infants are sometimes deliberately smothered. There are few, if any, external signs of smothering/overlaying to be found in an infant: petechiae are unusual² and the distribution of hypostasis may be misleading, particularly if any length of time has elapsed between death and necropsy. From our personal experience, we have observed that in cases where we suspect that smothering or overlaying may have occurred it is common to see abundant blood-stained fluid issuing from the mouth and nose; this is often a reflection of the haemorrhagic appearances of the lungs. Our DIA results show that in 73% of all the cases where overlaying was either admitted or suspected, there was an increase in the amount of intra-alveolar haemorrhage amounting to more than 5% of total lung surface. Less than half of those infants suspected of being smothered showed more than 5% haemorrhage, but the sample size was small.

Some cases where there was no previous suspicion of airway obstruction showed an increase in intra-alveolar haemorrhage. However, only one of these cases was in the “infectious” category, and no such increase was seen in either of the cardiac cases or in the cases where all the features were absolutely typical of SIDS. Two “SIDS” cases with atypical histories (very young age and unexplained blood-stained fluid in the abdominal cavity, respectively) did have significant haemorrhage, as did two cases of suspected NAI: we cannot exclude the possibility that smothering, or at least compression of the trunk, may have occurred in these cases. As haemorrhage seems common in overlaying and is sometimes seen in smothering, it may be that intermittent obstruction of the airways precipitates the haemorrhage, either as a result of raised intrathoracic pressure or possibly through inhalation of gastric contents.

We are always suspicious when presented with infants with evidence of inhaled material deep in the peripheral airways. At a macroscopic level, active inhalation is usually easily distinguishable from artefactual deposition of gastric contents in large airways postmortem. Aspiration may account for the haemorrhage occasionally seen in hanging: incomplete suspension can cause intermittent hypoxia and there can be elements of self strangulation; in most complete hangings, death is thought to occur within seconds. Pulmonary oedema and

Figure 3 Digital image analysis of intra-alveolar haemorrhage in cases of infant death not classified as sudden infant death syndrome.

haemorrhage have been noted in cases of fatal strangulation,^{12–24} where intermittent hypoxia and aspiration might be expected, although these lung changes were seen in causes of death other than strangulation.

We suggest that marked intra-alveolar haemorrhage may be a marker of imposed upper airway obstruction, whether accidental or deliberate. Pulmonary oedema and alveolar haemorrhage are observed in SIDS,^{8–10} but we question whether all of the cases until now regarded as SIDS (and forming the basis of published reports) really are “true SIDS” cases, particularly in view of our findings.

There is always the problem of sampling error when undertaking any histological examination. While accepting that there are five lobes in the lungs, we would not have expected there to be a significant sampling error by assessing sections from only three of the five lobes—that is, more than 50% of the available surface area. DIA is a very time consuming process and therefore is obviously not suitable for routine use. It was for this reason that we undertook the subjective assessments of the 24 cases. The grading system was simple—pathologists only had to estimate what they thought was mild, moderate, or severe haemorrhage. Despite these minimal guidelines, there was good concordance between individual pathologists. However, in eight of the 24 cases, these assessments were underestimates of the extent of haemorrhage quantified by DIA. In no case was there subjective overestimation. We suggest that if a pathologist finds a moderate degree of haemorrhage, then the history should be carefully re-examined to see if it yields any suspicious features that might indicate that upper airway obstruction had taken place.

We do not suggest that intra-alveolar haemorrhage is a specific marker of upper airway obstruction—our results show that it is not—but its apparent frequency is striking in those cases where this mode of death is suspected. Some studies have suggested that the presence of hemosiderin in the lungs of infants may be a marker of previous episodes of smothering or acute life threatening events.^{25–26} If this were the case, one would expect that there was intra-alveolar haemorrhage in the first place, which resulted in the hemosiderin being deposited.

The history is of vital importance in raising suspicion of airway obstruction. When we examined the histories in the light of DIA, we were surprised that some of the cases had originally been so readily ascribed to SIDS. Over the years, we have become far more critical of the histories presented to us. We now nearly always request that much greater specific detail be supplied. We are very unwilling to give SIDS as a cause of death, particularly since we have encountered several cases of “multiple SIDS” within families, with no reason for the deaths. We accept that there is an inherent bias in our “referral” cases, but then the investigation of suspicious death is the province of the forensic pathologist. The “Back to Sleep” campaign²⁷ resulted in a dramatic decrease in the number of so called SIDS cases; our cases were from before, during, and

after this campaign. With the decrease in SIDS, we would expect the relative proportion of suspicious cases to increase. Therefore it does not seem surprising the many of our cases had atypical histories, and with hindsight it would have been better if 11 cases had not been labelled as SIDS.

Overlaying is a problem which has been recognised for centuries: part of the *Children and Young Persons Act 1933* was designed to reduce the incidence of such deaths. However, we are satisfied from our own subjective experience that they still occur, even when the carer sleeping with the child is not under the influence of alcohol. Regrettably, few data are available to support this experience, which we accept is subjective.

One of the most difficult pathological problems is distinguishing SIDS cases from those of suspected deliberate upper airway obstruction. Pulmonary oedema is unhelpful as a discriminator, but our results have shown that intra-alveolar haemorrhage may be helpful, though it should not be regarded as the major criterion for instigating a criminal inquiry. If such haemorrhage is seen in a sudden unexpected infant death, this should prompt a very critical examination of the circumstances of the death. If the pathologist has any doubts regarding the aetiology of the death, the cause of death should be given as “unascertained.”

- Emery JL. Infanticide, filicide and cot death. *Arch Dis Child* 1985;60:505–7.
- Berry PJ. Pathological findings in SIDS. *J Clin Pathol* 1992; 45S:11–6.
- Southall DP. Imposed upper airway obstruction. Apnoeic spells or suffocation? *Bull R Coll Pathol* 1995;89(January): vii.
- McClure RJ, Davis PM, Meadow SR, et al. Epidemiology of Munchausen syndrome by proxy, non-accidental poisoning and non-accidental suffocation. *Arch Dis Child* 1996;75: 57–61.
- Byard RW, Carmichael E, Beal S. How useful is post mortem examination in sudden infant death syndrome? *Pediatric Pathol* 1994;14:817–22.
- Tschernig T, Kleeman WJ, Pabst R. Bronchus-associated lymphoid tissue (BALT) in the lungs of children who had died from sudden infant death syndrome and other causes. *Thorax* 1995;50:658–60.
- Kleemann WJ, Hiller AS, Troger HD. Infections of the upper respiratory tract in cases of sudden infant death. *Int J Legal Med* 1995;108:85–9.
- Aoki Y. Histopathological findings of the lung and trachea in sudden infant death syndrome; review of 105 cases autopsied at Dade County Medical Examiner Department. *Jpn J Legal Med* 1994;48:141–9.
- Bunai Y, Oyha I, Brinkmann B. Pathological approaches to SIDS. Special emphasis on the histopathological differences between SIDS and asphyxia. *Jpn J Legal Med* 1992; 46:405–6.
- AFIP. *Histopathology atlas for the sudden infant death syndrome*. Washington DC: Armed Forces Institute of Pathology, 1993.
- Berry PJ, Keeling JW. The investigation of sudden unexpected death in infancy. In: Anthony PP, MacSween RNM, eds. *Recent advances in histopathology*, vol 14. Edinburgh: Churchill Livingstone, 1989:251–80.
- Grellner W, Madea B. Pulmonary micromorphology in fatal strangulations. *Forensic Sci Int* 1994;67:109–25.
- Kaada B. Why is there an increased risk for sudden infant death in prone sleeping? Fear paralysis and atrial stretch reflexes implicated? *Acta Paediatr* 1994;83:548–57.
- De Jonge GA, Engelberts AC. Cot deaths and sleeping position. *Lancet* 1989;ii:1149–50.
- Sato Y, Kagehara B, Mukai T, et al. Diagnosis of asphyxia on the sudden infant death—prone sleeping position and vomit aspiration. *Jpn J Legal Med* 1992;46:407–12.
- Martin RJ, DiFiore JM, Korenke CB, et al. Vulnerability of respiratory control in healthy preterm infants placed supine. *J Pediatr* 1995;127:609–14.
- Hunt CE. The cardiorespiratory control hypothesis for sudden infant death syndrome. *Clin Perinatol* 1992;19:757–71.

- 18 Gaultier C. Cardiorespiratory adaptation during sleep in normal infants and children. *Pediatr Pulmonol* 1995;**19**: 105–17.
- 19 Kendeel SR, Ferris JA. Fibrosis of the conducting tissue in infants. *J Pathol* 1975;**117**:123–30.
- 20 Fleming PJ, Azaz Y, Wigfield R. Development of thermoregulation in infancy: possible implications for SIDS. *J Clin Pathol* 1992;**45**(suppl):17–19.
- 21 Howat WJ, Moore IE, Judd M, *et al.* Pulmonary immunopathology of sudden infant death syndrome. *Lancet* 1994;**343**:1390–2.
- 22 Reid GM, Tervit H. Sudden infant death syndrome (SIDS): immunoglobulins and hypoxia. *Med Hypotheses* 1995;**44**: 202–6.
- 23 Howat AJ, Bennett, MJ, Variend S, *et al.* Defects of metabolism of fatty acids in the sudden infant death syndrome. *BMJ* 1985;**290**:1771–3.
- 24 Brinkmann B, Puschel K. Histomorphological alterations of lung after strangulation. *Z Rechtsmed* 1981;**86**:175–94.
- 25 Risse M, Weiler G. Haemosiderin findings in the liver, spleen and lung in newborn infants and infants. *Z Rechtsmed* 1987;**98**:181–90.
- 26 Stewart S, Fawcett J. Interstitial haemosiderin in the lungs of sudden infant death syndrome: a histological hallmark of “near-miss” episodes? *J Pathol* 1985;**145**:53–8.
- 27 Department of Health. *Reducing the risk of cot death*. Report of the Chief Medical Officer’s Group on the sleeping position of infants in cot death. London: HMSO, 1991.