and 1/107. It is regrettable that the authors' considerable effort in searching for metabolic cause of SIDS was let down by some basic mathematics.

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3 Matsubara Y, Narisawa K, Tada K, *et al.* Prevalence of K329E mutation in medium-chain acyl-CoA dehydrogenase gene determined from Guthrie cards. *Lancet* 1991;**338**:552–3.

Dr Holton and colleagues comment:

Dr Smith is quite correct in pointing out that our negative results in testing for MCAD deficiency in cultured skin fibroblasts from 70 cases of SIDS were not incompatible with claims of a true prevalence of 3%. However, our paper made it clear that our claims that the incidence was less than 3% were based on our findings and those of others, the report of the Lyon group being cited in particular.[1] In an almost identical study to our own, the French workers found no positive findings in 107 SIDS cases. If our results are combined, the binomial probability theorem indicates that the incidence of MCAD is less than 1·7% with 95% confidence, or 2·6% with 99% confidence. On this basis, our claim was not unreasonable.

Perhaps it would be useful to summarise further work relating to the prevalence of MCAD deficiency in SIDS. Two other studies similar to those referred to above have been completed. In Sheffield, 160 SIDS cases (E Worthy, personal communication) and in Edinburgh 120 cases (G T N Besley, personal communication) were tested for MCAD deficiency, all with negative results. If all our results are pooled (457 cases) the prevalence of MCAD deficiency is calculated to be less than 0·65 or 1·00%, with 95 or 99% confidence respectively.

Dr Smith concludes that recent reports of population screening for the common MCAD deficiency mutation found carrier frequencies which supported our claim. In addition, the K329E mutation has been sought in DNA extracted from the liver of more than a 100 SIDS cases without finding any homozygotes for the defect.[2] Although it is important to appreciate that MCAD deficiency is a cause of sudden, unexplained death, the presentation is not typical of SIDS and it is a rare occurrence.

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Reducing the risk of cot death

SIR,—The nationwide campaign urging mothers to lay their babies on their backs to sleep is open to question. It would be unfortunate if the leaflets from the Foundation for the Study of Infant Deaths (FSID)[1] and its counterpart from the Department of Health with the unambiguous slogan 'Back To Sleep'[2] are taken to represent the views of paediatricians generally.

The assertion that 'there is no evidence that babies are likely to choke when lying on their backs' belies the considerable research into gastro-oesophageal reflux and laryngeal spasm, which is one of the major aetiological hypotheses. Altogether 70% of normal babies have been shown to have reflux during active sleep with 24 hour pH probes.[3] A high incidence of reflux has also been demonstrated in 'near miss' cases using barium swallows, pH probes, and isotope milk scans.[4 5]

Because the prone position is unsafe it does not follow that the supine position is safe. This latest U turn merely replaces one bad position with another. All horizontal positions encourage reflux with the risk of laryngeal spasm. What really matters is to raise the head of the cot. All studies of the supine-versus-prone position have neglected the important effect of gravity on reflux. The ideal sleeping position is with the head raised[5] but if babies must lie flat, the side is probably safer than the front or back. Better still, babies' cot mattresses should be wedge shaped.

Many parents are very worried by the risk of a cot death even if they do not voice their fears. The recommendations I have used for many years are:

- Lay your baby to sleep on one or other side, never the front or back
- Prop up the head of the cot by 10–12 cm (4–5")
- Keep the cot beside your bed in the first six months
- Learn to give the kiss of life
- Get medical advice if your baby is unwell

A nationwide campaign to reduce cot deaths is undoubtedly long overdue. However if it is to succeed, it is important that the recommendations are simple, sensible, and sound. I am seriously concerned about the widely publicised FSID and Department of Health guidelines.

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1 Foundation for the Study of Infant Deaths. *Reduce the risks of cot death.* London: FSID, 1992.
2 Department of Health. *'Back to sleep', Reducing the risk of cot death.* London: HMSO, 1992. (BTSI/E.)
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6 Barrie H. Sleeping position and SIDS. *BMJ* 1989;**298**:959.

Imposed upper airway obstruction in small children

SIR,—Surveilance of one of the 14 cases described by Samuels and his colleagues[1] was undertaken in the department of child health in this hospital with their advice and support. In addition to the videos they describe, we recorded sound as well and found this to be of considerable importance. Although perpetrators do not know that they are being watched, they are certainly aware of the possibility of being interrupted by someone entering the room. They may go to considerable lengths to disguise what they are about and this was certainly true in our case. As a result, it may not be easy to demonstrate what is happening on video alone. Some of the most compelling evidence which led to a successful outcome of the case arose from the ability to compare what we could see being done to the child with what the perpetrator was saying at the time. In addition, the audible change in a child's cry as the airway is obstructed is unmistakable even if the way in which that obstruction is being achieved is subtle.

Samuels and his colleagues describe the very careful preparation required for covert video surveillance. I would also emphasise the importance of continuing support for all the professionals involved, be they doctors, nurses, or police officers. Surveillance may be necessary for many days—all involved are only too well aware of the possible consequences of a few moments inattention, of any failure of communication, or indeed, of inadvertently betraying to the perpetrator that surveillance is being undertaken.

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1 Samuels MP, McClaughlin W, Jacobson RR, *et al.* Fourteen cases of imposed upper airway obstruction. *Arch Dis Child* 1992;**67**:162–70.

ABC of child abuse

SIR,—Torn frenula in children have been said to be 'virtually diagnostic' of non-accidental injury (NAI).[1] However, two recent cases indicate that this is not always so.

The first case was a 1 year old boy, whose sister was attending our casualty department for an unrelated reason. The boy was walking around the waiting room and fell flat onto his face. Examination of the crying child revealed a torn frenulum of the upper lip. The whole incident was witnessed by professional nursing staff and so the innocence of the incident cannot be doubted.

The second case involves the 14 month daughter of the author. After attempting to climb a vegetable rack, my daughter fell backwards, pulling the vegetable rack onto herself. Rapid investigation of the source of the subsequent bleeding confirmed my worst fears—she had a torn frenulum of the upper lip, presumably where it had been caught on the wire basket. I'm afraid that readers will have to take my word as to the innocence of this injury (what self respecting paediatrician would ever dare take such an injury to their local casualty department!). A torn frenulum is classically said to occur when a bottle or spoon is forced into the mouth of a child.[2] This association is probably strong enough to warrant the usual inquiries by the child protection agencies to see if NAI has occurred. However, before guilt of the child carers is assumed, it should be borne in mind that a torn frenulum is no more pathognomonic of

NAI than reflex anal dilatation is of child sexual abuse.[3]

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1 Speight N. ABC of child abuse: non-accidental injury. *BMJ* 1989;**298**:879–81.
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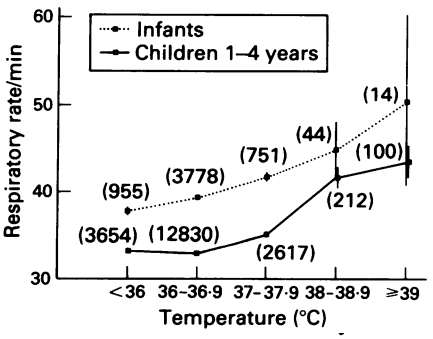
Effects of body temperature on respiratory rate in young children

SIR,—Dr Simoes and colleagues have reported studies of variability in measurements of respiratory rate in young American children, but did not consider possible effects of body temperature on these measurements.[1] Previous work suggested a weak association between body temperature and respiratory rate in young infants studied in Australia and Britain.[2] Current World Health Organisation guidelines for the management of acute respiratory infections in children recommend that young children with cough or difficult breathing and raised respiratory rate should be treated for pneumonia irrespective of temperature.

In a community study of acute respiratory infections undertaken at the MRC Laboratories in the Gambia, weekly measurements of temperature and respiratory rate were made on a population including approximately 500 children under the age of 5 years, over a one year period. This study is described in detail elsewhere.[3] Although these repeated observations are technically not independent, we consider that measurements of respiratory rate and temperature carried out not more than once weekly on a young child may reasonably be assumed to be independent. A total of 25 025 observations on 685 young children were made. In 70 instances abnormalities on chest radiography were found and these observations have been excluded from the following analysis. The relationships between temperature and respiratory rate for infants (5542 observations), and for children aged 1 to 4 years (19 413 observations), are shown in the figure. In both groups, mean respiratory rate shows a steady increase with increasing temperature of appoximately 2·5 min °C over the temperature range shown. A similar analysis restricted to children with cough (2537 observations) showed a similar relationship (data not shown).



Relationships between respiratory rate and temperature in young Gambian children. Vertical bars represent 95% confidence intervals of the mean, and numbers of observations for each point are shown in parentheses.

The data presented, in accordance with experimental results on the effects of temperature on breathing,[4] suggest that raised respiratory rates may be partly attributable to increases in body temperature. We earlier reported that in children with cough or difficult breathing respiratory rate is a valid predictor of the presence of clinical or radiological pneumonia.[3] The findings presented here do not challenge this, but they suggest that this relationship between fever and respiratory rate may account for some of the false positive diagnoses of pneumonia in children with cough or difficult breathing, fever and raised respiratory rate. This issue may be of particular importance in areas in which malaria is prevalent as it has been shown that there may be a very substantial overlap of clinical presentation in children with malaria and pneumonia.[5] The possible effect of this phenomenon on the specificity of raised respiratory rate as an indicator for pneumonia needs further investigation.

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Consumer safety and child choking attacks

SIR,—From time to time you publish letters which do not have any direct relevance to immediate past publications and it would be helpful if such letters indicated their origin. One such letter recently published gives no explanation as to why Drs Matthes, Sibert, and Levene were concerned about possible inhalation of foreign bodies from toys.[1] Those paediatricians who help local authority consumer protection departments by assessing or commenting on the safety of toys will be aware there has been a recent increase in the vigilance of trading standards officers regarding choking hazards to children because of a number of deaths. Dr Levene chaired a working party under the auspices of the Child Accident Prevention Trust, which found little published evidence of any serious hazard from the inhalation or ingestion of hair plucked from toys.[2] This report is being used by manufacturers to defend their products against legal action even though safer alternative materials are available.

The recent letter refers to a survey of paediatricians and ear, nose, and throat surgeons throughout Wales seeking to identify their awareness of choking hazards to young children from hair or other small objects. It is gratifying that they knew of no such hazard but we suggest that the wrong people were asked the wrong questions. The children who died in Leeds (from obstructive inhalation after ingestion of hair from a toy donkey) and in Birmingham (after inhalation of a small piece of plastic from inside a novelty chocolate egg) were unknown to paediatricians or ear, nose, and throat surgeons because casualty doctors and pathologists dealt with them.

Life is full of hazards and it would be impossible to ever legislate them all away. Even if this could be done it would then so grossly distort normal childhood experience as to be unacceptable. There are, however, measures that can be taken to control unnecessary hazards and we are of the opinion that inappropriately long hair that is inadequately fixed to a fur fabric is not suitable for the exterior decoration of any toy. It is to be expected that young children will pluck or suck the hair and may then inhale or ingest with the risk of asphyxiation or bezoar formation. Small pieces of plastic that may occlude the airway are also inappropriate in toys intended for young children.

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1 Matthes J, Sibert J, Levene S. Children choking on foreign bodies from toys. *Arch Dis Child* 1991;**66**:1104.
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Coroners' records of accidental deaths

SIR,—Dr Levene has demonstrated the potential of using coroners' records as a source of data relevant to child accident prevention studies.[1] In a similar retrospective study in this district using the coroner's records we discovered 69 children aged under 15 years who had died as a result of an accident between the years 1980–9 inclusive. Road traffic accidents represented the commonest fatal accident with falls, drownings, and asphyxia accounting for the remainder. Head injury was the commonest reported cause of death. Most deaths occurred within 2 km of the child's home while children were playing without supervision. We encountered an association between social class and incidence of accidents with 10 times as many accidents occurring in classes IV and V than in I and II. There was, in addition, a clustering of cases in areas with high deprivation scores.

This information was of great use to us in planning local child accident prevention strategy as it enabled us to target limited resources to areas where they were needed most. However, as in Dr Levene's case, we were made aware of the limitations of using coroner's records alone for this purpose. We discovered that inquisitions relate to deaths occurring to children who died within the boundaries of our district only. During the period of our study we became aware that several local children had died while visiting other districts but this information would not