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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Dr Holdom 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 study does show that our claims of an incidence of 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 in scope to one 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 K292E mutation has been seen 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 for MCAD deficiency is a cause of sudden, unexplained death, the presentation is not typical of SIDS and it is a rare occurrence.


Reducing the risk of cot death

Stir.—The nationwide campaign urging mothers to lay their babies on their backs to sleep is open to question. It would be unfortu-

nate if the leaflets from the Foundation for the Study of Infant Deaths (FSID) and its counterpart from the Royal College of Paediatrics and Child 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 hypothesi.

In a recent study of 120 cases of SIDS, two-thirds of normal babies have been known to reflux during active sleep with 24 hour pH probes. A high incidence of reflux has also been demonstrated in ‘near miss’ cases using barium swallows, pH probes, and isotopic milk scans.1

Since the prone position is unsafe it does not follow that the supine position is safe. This latest study has merely replaced 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 effects of gravity on reflux. The ideal sleeping position is with the head raised7 but if babies must lie flat, the side is probably safer than the front or back. Better still, babies’ cot mattresses should be wedged up.

Many parents are very worried by the risk of a cot death even if they do not voice their fears. The recommendations I have made 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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Imposed upper airway obstruction in small children


2 ABC of child abuse

Stir.—Torn frenula in children have been said to be ‘virtually diagnostic’ of non-accidental injury (NAI).1 However, 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 involved 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 torn a 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...
Reducing the risk of cot death.

H Barrie

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Updated information and services can be found at:
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