

Infant feeding

Dummies

F A Bu'Lock

A personal perspective (see page 1121)

I have two sets of personal views on dummies (pacifiers, soothers)—from the perspective of a mother and also as a “hands on” paediatric cardiologist. Thus my reactions to the paper by Viggiano and colleagues,¹ in this issue, are a synthesis of both. I am actually rather “pro-dummy”; albeit in the right place, at the right time, and for limited applications.

I breast fed both my children, for around 10 months. They were born hungry and sometimes it was a struggle. They are both strong willed young men who like a cuddle. I may be a sort of paediatrician but I am not an “earth mother”, and the logistic and practical details of spending the first six months of each of their lives acceding to their needs to be permanently clamped to my chest were really rather more than I could manage. Also I went back to work when they were 4 months old so they also needed able to take at least some sustenance by other means. My first ever research project was using ultrasound to examine the intra-oral processes of sucking and swallowing,² so I was well aware that nutritive and non-nutritive sucking are not the same, and that breast and bottle feeding also use different tongue actions. I have to say I didn't really care very much. It may be heresy, but in those first few days post-

partum, exhausted and milk free, bottles and dummies were a lifesaver allowing some sleep between breast “feeds” until the milk came in. I am used to being short on sleep; it's an occupational hazard, but as a new mum, some sleep (and time) seemed to be required for the metabolic processes involved in milk production. Later on, dummies seemed essential to spacing the feeds out enough to allow a semblance of normality in my life, and hence a little sanity. I'm not decrying those who do have the capacity to nurse continuously, but it's not for everyone. My children did suck their fingers from time to time but seemed to lose them at the most crucial moments. This also happened with some dummies; it was in fact only the dummies that I initially acquired at work (with orthodontic teats) that eventually stayed in long enough to generate reliable sleep patterns. As my children got bigger, the dummy stayed more and more in the cot (or the parental pocket), and generally became part of the sleeping ritual rather than a habitual oral presence. However, they were also invaluable for major crises (hence the pockets!). Around the age of 2 years they were gradually withdrawn without difficulty. I could not have coped without them and neither boy has obvious malocclusion of their deciduous teeth. I'm also not sure how worried about it I would be if they had, either. I'd be more worried if

it was their permanent teeth, or if the paper by Viggiano *et al* suggested that they would have been hideously deformed and considerably socially handicapped by their appearance, but I don't think it does. Don't get me wrong; I am not in the “dummies to keep them quiet” brigade—both my children are frighteningly articulate and talked far too much, too soon, but I suspect that is genetic rather than dummy related!

As a cardiologist I am also fairly pro the generic use of dummies. They are a major aid to accurate echocardiography, and, at least in my view, infinitely preferable to chloral, midazolam, or worse. I get seriously irritated by the well meaning healthcare professional who attempts to deny the benefits of non-nutritive sucking to a freshly transferred neonate needing a detailed echocardiographic diagnosis to save their life. I don't really mind whether it's a dummy or a clean and expertly inserted finger, but I am sure that most highly trained professionals generally have better things to do than stand with a finger in a new arrival's mouth for half an hour or so! I also don't believe it's going to adversely affect their ability to breast feed in later life half as much as either getting the diagnosis wrong, or the treatment itself, will.

The other aspect of dummy use which has not been addressed is the role of dummy sucking during tube feeds in ventilated infants and whether this is beneficial to eventual re-establishment or indeed establishment, of oral feeding post-extubation other than in preterm infants.³ My gut feeling is that it is, but I am happy to be guided!

So, fascinating as Viggiano and colleagues' paper is, forgive me if I remain a little sceptical of its practical validity, I would not like to see dummies go the way of MMR!



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Infant health

Infants bed-sharing with mothers

M Wailoo, H Ball, P Fleming, M W Platt

Helpful, harmful, or don't we know? (see pages 1106 and 1111)

The publication in the *Lancet* of the European Concerted Action on sudden infant death syndrome (SIDS) (ECAS study)¹ resulted in front page headlines such as “Don't sleep with your baby” (*Daily Telegraph*: D Derbyshire, Science Correspondent, 16 January 2004). Yet the ECAS study said nothing new about bed-sharing and cot death: both the CESDI study (Confidential Enquiry into Stillbirth and Death in Infancy),² data from New Zealand,³ and work from Ireland⁴ have superficially come to similar conclusions. Is the quality of evidence such that paediatricians, midwives, and health visitors should reasonably dissuade mothers from bed-sharing or co-sleeping, or is there more to it than that?

First, we must question the validity of extrapolating health messages from case controlled data sets. Bradford-Hill suggested robust criteria (temporal relationship, specificity, biological plausibility, coherence; others would add dose response) for inferring causality from associative data when prospective randomised trials are impossible.⁵ It took some time before these criteria were satisfied to such an extent that the successful “back to sleep” campaign could be accepted as public policy. That success should not seduce us into accepting a lower standard of evidence of causality for some “new” hypothetical risk factor. Arguably, now that we have good reason to promote supine sleeping, an appropriate thermal environment, and the avoidance of cigarette smoke, the benefit of any further message on reducing the risk of SIDS is likely to be marginal at best.

Second, there is a general lack of understanding about the heterogeneity of bed-sharing in particular, and infant sleep environments in general, in the data collection and analyses of case control studies. These can seriously undermine results such as that from ECAS. The

definitions of infant sleep conditions used in the majority of these studies do not necessarily reflect the reality of infant sleep environments as experienced by the parents and infants. Not all studies have allowed for the use of alcohol or other drugs, nor have they all distinguished manifestly unsafe sleeping environments such as co-sleeping on sofas. It is important to separate sleeping with mother alone, with mother and father, with father alone, in a bed with another child (either with or without an adult), or an unrelated adult sleeping with the infant. It is also important that studies distinguish bed-sharing to facilitate breast feeding; and bed-sharing that is habitual as opposed to occasional; because these states have major physiological differences.

Third, in some of the studies (not the CESDI one) the definition of bed-sharing included babies who spent part of the night in the bed but were put back elsewhere before being found dead, and some who bed-shared for part of the sleep, but were found in the adult bed alone—either before the adult came to bed, or after the adult got up. In these circumstances the death cannot reasonably be attributed to the presence of an adult. Intermittent bed-sharing may only occur when infants are brought into the bed when “mardy” or “twisty”, and these infant behaviours may be a marker for an infection or other illness.⁶ When this illness is fatal on the only night of bed-sharing it creates a coincidence which in large case control studies marks the bed-sharing as a “risk factor”, if no differentiation is made between habitual and intermittent bed-sharing, or the reasons for the bed-sharing are not adequately ascertained. It also raises important questions about the vulnerability of individual infants which may make them succumb to an apparently minor infection from which other normal infants will emerge unscathed.

The only analysis to date that has attempted this level of sophistication was that derived from the CESDI/SUDI study,⁷ which found that for non-smokers the apparent association with bed-sharing was explained by other factors than the practice of bed-sharing itself. Unfortunately, its conclusions are being overshadowed by more recent studies with less robust data sets.

In contrast to the generally negative stance of these epidemiological investigations, all of which focus on infant death rather than infant health, we argue that there is much to be said for bringing a baby into the adult bed in certain circumstances. We suggest that bed-sharing has been a soft target for SIDS campaigners because it seems to involve a straightforward parental choice in that there is no apparent harm from the alternative arrangement of solitary sleeping. But no parent-child behaviour is free of cost and benefit, and unqualified advice against bed-sharing might well result in an increase in other, more hazardous behaviours. For instance, faced with official disapproval of bed-sharing, mothers might choose to feed at night on a sofa, and fall asleep there with their baby; yet this environment appears to be by far the most unsafe for co-sleeping. We therefore challenge on several grounds the assumption that solitary infant sleeping is somehow optimal, when in worldwide and evolutionary terms it has not been the norm.

First, non-human primate mothers generally maintain intimate contact with their infants in the immediate postpartum period and for the first few weeks, both waking and sleeping, and so do human mothers in many cultures today.⁸ Even in the developed world, mother-infant bed-sharing is a common strategy for night-time care giving in the early months of an infant's life, particularly for breast fed babies.^{6,9} It is common among new parents following discharge from hospital, and is more prevalent among neonates than older infants.¹⁰

Second, there is now an increasing body of evidence relating to the behaviour and physiology of bed-sharing that has been obtained both in sleep laboratories and the home environment.^{11,12} These studies have shown that bed-sharing is associated with longer and more restful maternal and infant sleep,¹³ and with successful breast feeding.¹⁴ Babies who sleep with their

mothers feed more frequently (thus stimulating the milk supply), and are more likely to breast feed for longer than those babies who breast feed without bed-sharing.⁹ Babies sleeping habitually with parents are more rousable and may be more easily recognised as being unwell because of their proximity to mother.¹² Likewise, breast feeding mothers bed-sharing with their babies tend to sleep more lightly and are more rousable in the presence of their infant than are mothers who rarely or never bed-share.¹⁵ This emphasises the importance of breast feeding in relation to bed-sharing, and highlights the difference between habitual and occasional bed-sharing, which holds whether or not there is breast feeding.

Third, we have observed that mothers instinctively take up a protective posture when sharing a bed with their infants, lying in a fetal position with their lower arm above the infant's head and the infant lying within around 20–30 cm from the mother's chest. The position of the mother's thighs prevents the baby from sliding down the bed. An extraordinary range of dyadic behaviours can be observed: the infant and mother start to synchronise their sleep states; move towards each other or away from each other as dictated by temperature (and babies demonstrably do not overheat in this situation); breast feeding can take place without either party being technically awake; and both parties touch each other, particularly the mother touching the baby. It is clear from the work so far that we are only just beginning to unravel the complexities of bed-sharing behaviour, and that without such an understanding, simplistic descriptions such as "safe" or "hazardous" are meaningless.

The story of the role of health professionals in prone and supine sleeping was a classical contrast of hubris and nemesis: the well intentioned promotion of a behaviour based on extrapolation from the physiology of preterm babies, but an outcome that, with hindsight, caused unknown numbers of unnecessary infant deaths across the developed world. We cannot afford not to learn from our recent history. We must also be careful about using the "risk" of an intrinsically highly unlikely event, that of unexplained sudden infant death, as a lever for modifying maternal behaviour: in any case, just saying "don't do it" is ineffective in changing anyone's behaviour. Since the advantages of breast feeding have an evidence base that does not feature the risk of cot death at all, and our understanding of the interrelationship between bed-sharing and breast feeding is still quite primitive, we should be very reticent about taking a view on the safety or otherwise of bed-sharing until we understand a great deal more about it.

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IMAGES IN PAEDIATRICS

Perception of death

The drawings (figs 1 and 2) were made by Lisa, a 9 year old girl with terminal cystic fibrosis. The pictures were made during her hospital admissions, three months apart, with the last one (fig 2) a few days before her death. It is very interesting how this child perceived the deterioration of her condition through these drawings, although she was always cheerful, hardy ever complained, and had a remarkable courage during the course of the disease.

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Figure 1



Figure 2

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PostScript

LETTERS

First do no harm...

A 9 day old infant presented to hospital with an erythematous, diffusely swollen right foot, shown in figs 1 and 2.

The infant had been slow to establish breast feeding and was not discharged from hospital until day 7 of life. Her father had noticed the inflamed foot during a nappy change earlier that morning. Both parents felt the infection was the result of a tight hospital name band. The firm plastic band had caused small lacerations to the right ankle prior to its removal.

Hospital name bands are useful in the identification of patients prior to the administration of drugs and are a simple tool in the prevention of abduction from hospital.¹ It can be very difficult to fasten the bands tightly enough for them to remain attached but not cause superficial lacerations, particularly if the child has dry, peeling skin. These bands can frequently be found adorning the floor or discarded items of clothing on postnatal wards.

As paediatricians we are urged to place the child's best interests at the centre of all clinical considerations. We have a responsibility to safeguard the reputation of paediatricians through our personal clinical practice. This child had an iatrogenic injury following a non-essential intervention resulting in hospital readmission. She received a full course of antibiotics, exposing her to the well documented risks of allergic reaction, nephrotoxicity, and vestibular and auditory damage.

Perhaps it is time for us to reconsider techniques for the attachment of hospital name bands to newborn infants. Although name bands could be manufactured using



Figure 1



Figure 2

softer materials, this would increase the ease with which such bands could be removed or switched. A more practical suggestion would be to label cord clamps with an identifier. Cord clamps do not fall off and cannot easily be removed by non-medical personnel. This technique could be combined with security tags, footprinting, and the retention of cord blood samples at individual hospitals' discretion.

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Consent has been obtained for figures 1 and 2

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Reference

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Is fragmentation of schedules hampering the uptake of hepatitis B vaccine?

The rising number of recommended childhood vaccines can be challenging for parents for two reasons—up to eight clinic visits for immunisation alone in the first 18 months (including BCG and hepatitis B), and the concern regarding the number of injections given per visit.¹ This in turn may affect the uptake of newer, but nevertheless important vaccines such as hepatitis B (HB).

We reviewed the uptake of HB and other childhood immunisations of 23 at risk infants born to HbsAg positive mothers in a district general hospital over a four year period (January 1999 to January 2003) and studied the reasons for immunisation failure.

Table 1 Uptake of HB and other childhood immunisations of 23 at risk infants born to HbsAg positive mothers

Immunisation	%
1st dose HB (in 1st 48 h) uptake	91.3
2nd dose HB (1 mth) uptake	73.9
3rd dose HB (2 mth) uptake	65.2
4th dose HB (12 mth) uptake	47.8
3 doses of DPT/Haemophilus influenzae type b (Hib)/MenC uptake	78.3
MMR (12-15 mth)	65.2
BCG	84.0

Maternal case notes, the local community computer database, and GP records were retrospectively reviewed. Families were contacted whenever possible to determine the reasons behind the non-compliance. Table 1 shows the results.

Our audit confirms the well known pattern of high initial uptake followed by exponential decline as reported in previous audits.² This has been ascribed to poor parental understanding about the importance of completion of the full course.²

However, we found out that out of 11 cases who had the 1st dose but missed subsequent doses, three (27%) had moved out of the area, three (27%) did not receive appropriate notification (due to change of name or address), and five (46%) felt that there were too many attendances to complete the immunisation.

The relatively high uptake of DPT/MenC/Hib and even MMR in comparison to HB suggest that this specific immunisation failure may be partly due to fragmentation as reported by 46% of the parents.

We feel that this low uptake of HB immunisation could be circumvented by giving second and third dose of hepatitis B along with the 1st and 2nd doses of DPT/MenC/Hib (at 8 and 12 weeks of age), either as a combination vaccine or as a separate vaccine.³ The fourth dose of HB can be combined with MMR. We feel that there is a need for a larger national audit to address this issue as it can have an important implication on the immunisation schedule.

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Table 1 Hib IgG GMC, with 95% CI, following primary immunisations, a 4th dose, and at time of study for subjects who did or did not receive a booster dose of Hib in infancy, and proportions achieving concentrations ≥ 0.15 and $1.0 \mu\text{g/ml}$ at time of study

	n	Hib IgG GMC, $\mu\text{g/ml}$ (95% CI)			≥ 0.15 n (%)	≥ 1.0 n (%)	p value
		After primary immunisation	After booster dose	At time of study			
Boosted infants: All	26	0.18 (0.13 to 0.26)	3.42 (1.65 to 7.10)	0.23 (0.14 to 0.39)	14 (26%)	2 (8%)	<0.0001*
Boosted infants: Post-primary <0.15	12	0.08 (assigned)	1.21 (0.42 to 3.51)	0.25 (0.09 to 0.71)	5 (42%)	2 (17%)	0.80†
Boosted infants: Post-primary ≥ 0.15	14	0.37 (0.28 to 0.50)	8.30 (3.00 to 19.14)	0.22 (0.14 to 0.37)	9 (75%)	0 (0%)	
Non-boosted infants	7	1.60 (1.18 to 2.18)	NA	0.25 (0.04 to 1.61)	2 (29%)	2 (29%)	

*† test comparing Hib IgG after booster dose and at time of study in subjects who had received a booster dose in infancy.

†† test comparing Hib IgG at time of study in subjects who had Hib IgG <0.15 or $\geq 0.15 \mu\text{g/ml}$ after primary immunisations.

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Hib IgG persistence following early booster dose

A diphtheria/tetanus/acellular pertussis–*Haemophilus influenzae* type b vaccine (DTaP–Hib), introduced to the UK in 1999, was associated with poor primary Hib responses¹ and a resurgence of Hib disease in the population.² Consequently, in 2003, the UK Department of Health undertook a campaign to immunise children aged 6 months–4 years with an additional dose of Hib. We have previously shown a significant rise in Hib titres following an additional Hib dose, given before one year, in infants with very low primary responses.³ Here we describe how that response persists.

In our previous studies preterm infants with Hib IgG <1.0 $\mu\text{g/ml}$ following primary immunisations with DTaP–Hib¹ received a 4th dose of Hib conjugate before 1 year of age.³ In this new study (LREC approved), 33 subjects from the previous studies were enrolled and blood was taken prior to the catch-up campaign. Mean gestational age at birth was 29.6 weeks (range 25–31.7 weeks). Twenty six had received a booster dose at <1 year of age (mean 0.62 years). Mean age at study was 2.89 years (range 2.25–3.41 years).

Hib IgG geometric mean concentrations (GMC) after primary immunisations, 4th dose, and at time of study, and proportions achieving concentrations of 0.15 and $1.0 \mu\text{g/ml}$ are shown in table 1.

Nineteen subjects had previously had concentrations high enough to allow determination of post-4th dose Hib IgG avidity. Of these, seven had an IgG concentration on re-bleeding in this study sufficient to allow determination of avidity. The GM avidity index post-4th dose was 76.94 (95% CI 52.16 to 113.50), increasing to 138.19 (95% CI 71.70 to 266.33) at time of study ($p = 0.10$).

Within three years of a 4th Hib dose, Hib IgG levels have fallen significantly and the proportion of infants with detectable Hib IgG is very low. There is evidence of avidity maturation over this time, but this should be interpreted cautiously given the small numbers.

If protection from Hib disease depends on a level of circulating Hib IgG and not simply on immunological memory, then our findings suggest that a single additional dose before 1 year may be insufficient in those with poor primary responses. Indeed, even children who had acceptable responses (>1.0 $\mu\text{g/ml}$) to primary immunisations had low levels of Hib IgG in this study. It remains imperative that Hib surveillance continues and that the potential need for further Hib doses be kept in mind. In some infants one additional dose may be insufficient.

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CORRECTION

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M Wailoo, H Ball, P Fleming, *et al*. Infants bed-sharing with mothers (*Arch Dis Child* 2004;**89**:1082–3). The last author of this paper was spelt incorrectly and should be M P Ward Platt. We apologise for the error.