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Interventional parenting programmes

A good idea that doesn't work: the Parent Baby Interaction Programme

Martin P Ward Platt

Perspective on the paper by Glazebrook *et al* (see page 438)

It is impossible to work with parents and children for any length of time without coming across situations where mothers, fathers, or both seem to need help with parenting. It has long been known that there are associations between the quality of parenting and children's outcomes. Additional difficulties with establishing appropriate parenting styles are imposed on families as a result of their baby needing intensive care. It is therefore important to find out which interventions, provided in the setting of a neonatal intensive care unit (NICU), might "improve" parenting; and whether this in turn could mediate better outcomes for babies, and their parents, in families to which such interventions are given.

Over the past 20 years a great deal of work has evaluated interventions to improve parenting, and it is fortunate that many of the published studies have been

randomised controlled trials. This in turn has allowed for two related Cochrane reviews, one in 2003¹ on child outcomes, and one in 2004² for maternal outcomes. For children, such programmes seem to lead to improvements in attachment and behaviour; and for mothers, indices of mental health, such as depression, show improvements. These effects seem to be mediated by increased maternal sensitivity towards the baby that in turn allows more secure attachment for the child, and perhaps a more rewarding relationship for the mother. So there seems to be reasonable evidence that, in general, some of these interventions work: but which, and for whom? And what is the best way to evaluate the efficacy of such interventions?

The paper by Glazebrook *et al*, evaluating the Parent Baby Interaction Programme (PBIP) in a NICU setting, is another randomised controlled trial that

further contributes to the literature on interventional parenting programmes. It is important for two reasons: its demonstration that this particular intervention programme was ineffective, and the rigour of the methodology with which that finding was demonstrated. The authors discuss the possible reasons for the ineffectiveness of the PBIP, and I will not repeat them here. But is worth emphasising just how similar the measured outcomes were in the subjects and controls, given that the study was strongly powered to detect clinically relevant group differences. This demonstration of complete ineffectiveness undermines the suggestion that more intervention would have thrown up an appreciable advantage for the index babies over the controls, and strongly suggests that PBIP simply does not work.

Finding that things do not work is good for healthcare because it prevents the enthusiastic adoption, on the basis of theoretical plausibility or extrapolation from other work, of programmes that have no benefit. Indeed a programme that is plausibly beneficial may even turn out to be harmful when properly studied. New programmes will either carry a new direct cost (for instance, the establishment of a new post to deliver or coordinate the programme) or impose opportunity costs (for instance, if nurses deliver a new kind of care they may have

to stop doing something else); so if they do not work, we should not waste resources on them. But it can be an uphill struggle to do rigorous evaluations of interventions that relate to parenting because they carry a veneer of being "obviously" good. We all want to be better parents, and we all want parenting to be better—for paediatricians, it is almost an article of faith. However, even though there is good evidence that some parenting programmes seem to work does not mean that every parenting programme will be effective. Each has to be evaluated on its merits.

So to the methodology. Glazebrook *et al* used a cluster-randomisation technique, a crossover design, pairing of units to account for socioeconomic variation, and two demographically different regions of the UK (Trent and the South West). This allowed them to maximise power,

minimise cross-contamination between groups, allow for deprivation and generalise the findings. Trials of this kind require a huge investment of time and money, but the pay-off for the National Health Service is even bigger, given that no unit could now plausibly make a bid to introduce PBIP in its hospital. Indeed it should also have the effect of increasing the care with which any other proposed parenting programme should be scrutinised before introducing it. We have to wonder whether the other trials in the Cochrane review were as well designed as this. If they had been, would the meta-analyses have come up with such unequivocal positive findings?

When a study that is as well designed and powerful as this one fails to show any effect of the intervention, it is important to look critically again at other studies that purport to show benefits of

analogous interventions. Above all, what this study adds to the literature is that every parenting intervention programme should be evaluated with the same rigour. Glazebrook *et al* have set the standard to which others must aspire.

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Correspondence to: Martin P Ward Platt, Royal Victoria Infirmary, Newcastle upon Tyne NE1 4LP, UK; m.p.ward-platt@ncl.ac.uk

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SGA and SIDS: causality or association

Small for gestational age infants and sudden infant death syndrome: a confluence of complex conditions

Carl E Hunt

Perspective on the paper by Malloy (see page 473)

"All illnesses have some hereditary contribution. Genetics loads the gun and environment pulls the trigger" (Francis Collins, Director, National Human Genome Research Institute, National Institutes of Health, 2006)

New data by Malloy¹ further substantiate fetal growth restriction or being small for gestational age (SGA) as a risk factor for sudden infant death syndrome (SIDS). The similar odds ratios for sudden unexpected death in infancy (SUDI) excluding SIDS and for SIDS may reflect at least in part diagnostic shifts based on custom and preference of the medical examiner in classifying infant deaths consistent with SIDS not as SIDS but rather as, for example, "accidental suffocation" or "other ill-defined and unspecified causes". These new confirmatory data are important, but they provide no new insights regarding potential causal mechanisms for this association between SGA and SIDS. Does being SGA directly increase the risk for SIDS, or is this an

indirect association because of an underlying causal biological or environmental risk factor common to both conditions?

Low birth weight is defined as a birth weight <2500 g and can be due to prematurity (<38 weeks' postmenstrual age) and/or to fetal growth restriction (SGA). SGA, typically defined as a birth weight <10th percentile for gestational age, can be due to a variety of pathophysiological conditions or diseases.² The growth restriction in SGA infants can be either symmetrical (weight, length and brain size affected) or asymmetrical (brain growth preserved), the former often associated with factors intrinsic to the fetus and the latter often associated with external factors. In addition to chromosomal anomalies and other syndromic conditions, symmetrical SGA can also be caused by intrauterine infection (toxoplasmosis, rubella, cytomegalovirus and herpes infections), inborn errors of metabolism and fetal drug (or toxin) exposure, such as to alcohol, cigarette smoke and drugs of misuse (eg, cocaine or opiates). Of note, all of these

exposures causally linked to SGA are also risk factors for SIDS whether SGA or not³ (table 1). Asymmetrical SGA typically has its onset in the second and third trimesters, when fetal nutrition increasingly contributes to energy storage. SGA infants are more likely to have problems with perinatal asphyxia or other perinatal disturbances, and are at increased risk for long-term deficits in growth, neurodevelopmental handicaps, and higher rates of early infant mortality in addition to SIDS.^{2–3} Immunological function in SGA infants may be depressed at birth and can persist at least into childhood.

Table 1 Shared maternal characteristics and fetal exposures, and putative genetic causal mechanisms shared by sudden infant death syndrome (SIDS) and low birthweight infants*

	SGA Preterm	
Maternal characteristics		
Including inadequate prenatal care, low socioeconomic status, race/ethnicity, young age, unmarried, increased parity	+	+
Fetal exposures		
Alcohol and cigarette smoke	+	+
Drugs of misuse, including cocaine and opiates	+	+
Genetic polymorphisms		
Infection and inflammation		
Interleukin-6	+	+
Interleukin-10	+	+
Glucokinase	+	

SGA, small for gestational age.
+ Characteristic shared with SIDS.
*See text for references.