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NITRIC OXIDE AND THE BRAIN

Just as we have all been getting used to nitric oxide as a compound with desirable vaso-active properties when given by inhalation; and just as we have been considering promoting its production pharmacologically with new drugs such as sildenafil; along comes a new family of compounds that inhibit nitric oxide synthase. These may become important in neonatal care because the production of locally toxic nitric oxide appears to be a mediator of the damage caused by ischaemia and reperfusion injury in the brain, so inhibiting nitric oxide synthase may be a useful strategy following severe birth asphyxia. Nitrotyrosine is a compound formed when nitric oxide combines with oxygen, forms peroxynitrite, and reacts with tyrosine. Groenendaal *et al* have demonstrated that nitrotyrosine was widely distributed in the brains of babies who died following perinatal asphyxia, but not in the brain of a control infant who died with spinal muscular atrophy. Interestingly, the distribution of nitrotyrosine in the brains of the asphyxiated infants was wide and variable, with hippocampal damage as well as ''watershed'' and basal ganglia damage. Although the authors do not highlight this, it would be consistent with the suggestion that asphyxia might sometimes damage later cognitive function more than motor function in certain babies.

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ASPHYXIA AND COGNITIVE FUNCTION

It is timely, in the light of the above paper, that Gonzalez and Miller should review impaired cognitive function, without cerebral palsy, as a complication of perinatal asphyxia. Certainly the received wisdom is that disordered motor function is the dominant long-term complication, and that learning difficulties may or may not accompany serious (four limb) cerebral palsy, but it is plausible that in some infants the asphyxial insult may selectively affect parts of the brain predominantly related to cognitive rather than motor function. Readers may take issue with reading too much into the short-term outcomes of the recently published randomised controlled trials of hypothermia (using Bayley MDI scores at 18 months), but the published data using cognitive instruments at school entry are harder to dismiss, even though these data are mostly from case control or cohort studies.

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PERINATAL DATA FROM BEFORE THE DAWN OF PAEDIATRICS

What can data from up to 200 years ago tell us about perinatal care? Quite a lot, as Woods shows us in his thought-provoking paper. The lessons I drew were first, that the very fact of introducing systems for auditing stillbirths was a catalyst to drive public health change. Second, the introduction of quality assured midwifery, prioritising those areas with the highest stillbirth rates, was the single most important common factor in bringing down the highest rates in scandinavian countries in the 19th century. And third, one of the key participants with responsibility for recording the stillbirths was not medical: it was the clergy, who were presumably already used to recording adult marriage and death, and who in relation to recording stillbirth were perhaps most likely to be impartial in their work.

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MEASURING BEHAVIOUR IN EX-PREMS AT SCHOOL ENTRY...

Trying to get a handle on some of the ''difficult'' outcomes of survivors of premature birth is a particular challenge, because it is tempting to attribute to ''prematurity'' outcomes that may in fact have been predestined by other factors that are themselves associated with prematurity. Behaviour is one such ''difficult'' outcome, and the contribution of Reijneveld *et al* to the literature is to ascertain behavioural status at the age of 5 years (school entry), as this may subsequently track through the school years. They have the enormous advantage of a high quality control group consisting of contemporaneous national cohort data, so the findings should certainly be taken seriously. But one cannot help noticing that the mothers of the premature babies were of lower educational attainment than the controls, and one cannot help but wonder what other unmeasured factors about these children's family backgrounds might have been the ''real'' causes of their behaviour patterns.

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...AND MEASURING BRONCHO-PULMONARY DYSPLASIA

Just as we all know what we mean by behaviour, but it is quite hard to measure, so we all know broncho-pulmonary dysplasia (BPD) when we see it, but again it is quite hard to measure. Quine *et al* have just made it a lot easier, using apparatus available to us all rather than complex and temperamental technology. This may be a real step forward, since it is meaningless to ascertain rates of ''oxygen dependency at 36 weeks'', or ''in oxygen at discharge'', if we do not distinguish properly between the baby in a small trickle who will be off within 3 months, and the baby in half a litre who will have severe and prolonged respiratory disability. Furthermore, their method could be implemented routinely in any unit that wanted to introduce a higher level of sophistication into their audit of respiratory morbidity, as well as being potentially useful in randomised controlled trials with long-term oxygen dependency as an outcome.

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