Highlights from this issue

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MORE ADEPT INFORMATION

The original ADEPT study, in which early versus late introduction of enteral feeds was compared in babies identified *in-utero* as having absent end-diastolic flow, showed that the earlier starters achieved full enteral feeds considerably sooner than the late starters, with no difference in rates of necrotising enterocolitis. Kempley and colleagues now report a more detailed analysis of the actual achieved rates of feed advancement in babies <29 weeks in ADEPT, from which it can be seen that these babies only tolerated very much slower feed increases than that suggested in the protocol. To put the achieved rate of advancement in perspective, in the first postnatal week it was only about a fifth of the rate specified in the 'slow' arm (18 mL/kg/day) of the current SIFT trial (HTA 11/01/25: Speed of Increasing milk Feeds Trial of rates of enteral feeding). ***See page F6***

ANTENATAL STEROIDS

One of the problems with randomised controlled trials is that just because a few extreme subjects are included, it does not follow that the overall benefits or harms demonstrated in the trial necessarily apply to such subjects. So it has been for trials of antenatal steroids in which the numbers of subjects at the extreme of viability—23/24 weeks—have commonly been too small to be confident that the overall benefits of therapy necessarily apply to these babies. To demonstrate benefit or harm more broadly, and to analyse dose response, it is more helpful to conduct cohort studies with good control for confounding, such as this one (Wong and colleagues) from the NICUS network in Australia. It has the triple strengths of being population based, very large, and focused on only the highest risk babies <29 weeks. Clear and significant benefits were seen for babies from 24 to 27 weeks even with incomplete steroid courses But even in this large study, there were too few infants at 23 weeks, and too few untoward outcomes at 28 weeks, to achieve statistical significance for the observed improvements in outcome for these babies. ***See page F12***

GROWING TINY BABIES

It is instructive to read Cole's paper on birth weight and longitudinal growth in the context of the paper by Kempley *et al* that I highlight above. Cole has taken the weights of all comers at the various gestations, making the implicit assumption that babies are homogeneous with respect to growth at any particular gestation. Kempley *et al* exemplify why this assumption is false: one of the key reasons for the preterm delivery of babies is the observation of poor fetal growth, or the presence of a condition in the mother (such as pre-eclampsia) that can compromise fetal growth. Not only do these babies have lower birth weight z-scores compared with spontaneous deliveries, but their reduced tolerance of enteral feeds will probably impact on their early postnatal growth. It would be interesting to repeat Cole's study separating out those babies delivered after spontaneous labour from those delivered for obstetric reasons: would the same longitudinal growth charts work for both groups? Nevertheless, the development of standards for longitudinal 'real' growth is an advance on our current use of charts based on cross-sectional birth weights. ***See page F34***

GROUP B STREPTOCOCCI IN BREAST MILK

Little by little, we are gaining more knowledge about the conundrum of group B streptococcal carriage in mothers, and its contribution to early onset infection in babies. Late onset infection has different complexities since babies' relationship with pathogens depends on age, mode of feeding, and other aspects of their ecology. Filleron and colleagues have taken an unconventional approach in assembling data from the literature on group B streptococci in breast milk, and its possible relationship to late onset infection, because the published data are so sparse. Perhaps they have defined the questions more than they have provided answers, but either way the paper makes for stimulating reading. ***See page F41***

CRP: NOT NICE

Not everyone is as convinced about the value of c-reactive protein measurements as the National Institute of Health and Care Excellence (NICE) guideline development group for 'Antibiotics for early onset neonatal infection' evidently was. Lacaze-Masmonteil and colleagues have therefore provided some welcome new data on the possible value of measuring CRP just once, at 18 h of age, with a point-of-care instrument, so that the answer does not have to wait for laboratory processing time. Their interesting result is that this test appears to have a very high negative predictive value in babies who have been started on antibiotics on the basis of risk factors for infection, as long as other aspects of the baby's clinical state, including gestational age, are considered as well. Babies, and their mothers, stand to gain a lot from this: rather than wait for 36 or 48 h, or even longer, for negative blood cultures, it might be possible to discharge them home (and reduce their antibiotic exposure) by 24 h. ***See page F76***

TARGETING THE EYE

Retinal examination of retinopathy or prematurity is unpleasant for babies. The idea that it might be possible to reduce the number of examinations for some babies, while perhaps targeting more frequent examinations to the babies at highest risk, is intuitively attractive. WINROP provides a clinical algorithm by which this might be achievable, and Piyasena and colleagues have successfully evaluated this approach in Scotland. Key to its effectiveness is accurate serial measurement of weight. It remains to be seen whether WINROP will win against the alternative strategy of measuring urinary NTproBNP, which is also currently under investigation (ClinicalTrials.gov Identifier: NCT01861470). ***See page F29***