Fantoms

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Remifentanil for Intubation: how much to give?

The paper by Choong *et al* is the second RCT of remifentanil that we have published (the first was by Pereira e Silva *et al* in 2007[1]). What is the bottom line? Well, it does what it says on the tin: remifentanil/atropine provides good quality intubation conditions, comparable to those achieved with fentanyl/succinylcholine/atropine. Is there a downside to remifentanil? Yes: muscle rigidity may be a problem if one uses too much, so they suggest keeping the remifentanil dose under 3 µg/kg. Pereira e Silva *et al* used remifentanil 1 µg/kg with midazolam 200 µg/kg so there seems no reason not to use a dose at the lower end of the range. The authors also highlight the problem of the very small doses needed for tiny babies—a microgram or even less. To achieve this requires massive dilution of the standard adult vials, with the attendant risk of getting the administered dose wrong. If neonatologists are to start using remifentanil routinely, we need a formulation that is safe for the neonatal population. Interestingly, remifentanil did not feature at all in the survey of pre-intubation medication published by Kelleher et al last year[2]. It will be interesting to see whether there is a shift towards remifentanil as the practice of intubation-surfactant-extubation becomes more widespread. ***See page 80***

Sildenafil for pulmonary hypertension: how much to give?

The first mention of sildenafil in *Archives* that I can find which related to treating pulmonary hypertension was in 2003. It is now quite commonly used in neonatal medicine and by paediatric cardiologists. It has an entry in the BNF for children stating unequivocally that the maximum dose is 2 mg/kg 4 hourly, a dose extrapolated from adult use and only validated in the literature by an anecdotal 'what works' approach rather than either dose ranging studies or pharmacokinetic data. So it is good, if a bit overdue, to be publishing the paper by Ahsman *et al* on the pharmacokinetics of sildenafil. The subjects were big babies who had received extracorporeal membrane oxygenation, so the results may or may not apply to small ex-preterm babies with chronic lung disease, but for me the take home message is that while a dose of 2 mg/kg will be plenty for some babies, for others it will be inadequate. So babies with pulmonary hypertension who do not seem to be responding to sildenafil may not be 'non-responders' at all—they may simply not be getting enough sildenafil. ***See page 109***

Outcomes: how are we doing?

One of the great advantages of maintaining population based surveys over the long term is that audits of changing outcomes over extended periods of time become possible. In this case, Roberts *et al* present the data from the long-running neonatal outcome survey in Victoria, Australia, between the beginning and the end of the 1990s. Improved survival seemed to be offset by increased rates of mild disability, but as they point out, this is at least as likely to be due to the change in psychometric instrument as to any change in the children, so the authors may be a bit harsh on themselves. And while it is important to have controls, as they demonstrate, we must not forget that the cases and controls will differ by factors other than prematurity alone – perhaps they will analyse this next? ***See page 90***

… and outcomes on steroids

With so many randomized controlled trials and meta-analyses demonstrating the value of antenatal steroids (it's 38 years since Liggins and Howie's paper[3]), why undertake a cohort study? Well, partly because RCTs can't answer all the important questions; and partly because the real, messy world is very different to even the most pragmatic of RCTs. In particular, the RCTs had difficulty recruiting substantial numbers of subjects at the lowest extreme of gestational age, so the true effectiveness of steroids at 23 to 25 weeks has always been controversial. The big cohort study by Manktelow *et al* has measured the effectiveness of antenatal steroids with some precision at these gestations, although at 23 weeks any true effect may still have been masked by small numbers. The main problem in studies of this kind is that like cannot be compared with like: the no-steroid group will probably have disproportionally contained the highest risk babies, such as those born outside delivery suites, or needing to be delivered so suddenly that there was no time for steroid administration. ***See page 95***

Early onset sepsis

About 14% (nearly 1 in 6) of of the babies ≤ 34 weeks studied by Dutta *et al* had proven early onset bacterial infection. Three quarters of these infections were *E. coli*, but the rest were all staphylococcal—there was not one group B streptococcal infection. What a contrast with the results of similar studies from Europe or the USA. Not only that, but either through the use of intrapartum antibiotics, or simply because of different bacterial ecology, the risk factors for early onset septicaemia were rather different to those that one would have predicted from a knowledge of the existing published data. This paper does not rewrite the story of early onset infections and their risk factors, but it does serve as a timely reminder that both microbiology and medicine can be very different in other settings. ***See page 99***

REFERENCES

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