Dr Williams suggests that length of ventilation could account for the association. When duration of ventilation during the first month of life is entered in the regression, it does not account for the correlation seen. This variable was not included in the final paper because, as explained, it both may cause and be caused by CLD. Vitamin A at 800 U per day was used throughout the period of the study either in oral or intravenous supplements. Dr Williams is somewhat selective in his quotations from the paper of Hammerman and Aramburo, in omitting to mention that 7/20唇裂婴儿 went home on oxygen compared with 0/22 of the controls.

I agree with Dr Wilson and his senior colleagues when they state that association does not necessarily imply causation, and that my cohort study can only generate a hypothesis to be tested. However, they robustly counter my proposition that the ‘theoretical gains made from early lipid infusion may be outweighed by an increase in CLD’ with their own small case control study showing an association between low energy intake and CLD. Surely what is sauce for the goose is good for the gander? Also the two sets of findings are not incompatible.

Dr Wilson and colleagues offer me advice on the size of randomised controlled trial which their formulae should be confined. Trial size calculations are based on prior assumptions about the size of the effect of the intervention and the incidence of the target condition in the population. Their calculations are correct in these assumptions, although they give no reason for choosing a reduction of CLD from 60% to 40% as the effect sought. The odds ratio for lipid use in the first 21 days was 8:1 in the study reported, suggesting that a considerably smaller study may be needed. I certainly support randomised trials and further studies are currently underway.

While there is good reason to believe that better nutrition both before and after birth may make an important contribution to improved recovery from lung disease in the preterm infant, there is also good reason to believe that any rise of lipid infusions may not be the best way of achieving this.

The firmness with which this was expressed in my concluding remarks is at least partly due to editorial changes, so perhaps they should share the reputation for draconian proscription!

Aetiology of malocclusion of the teeth

Str.—In his excellent article about the aetiology of malocclusion of the teeth, Professor Leighton does not mention the most common cause of malocclusion we see on this side of the Atlantic, at least in an inner city population of New York City: prolonged feeding and/or pacifier use.

We see many children over the age of 1 year who already show the typical V shape of the incisors in the maxilla, as compared with the U shape of the mandible, indicating the opposite, or to a much lesser extent, thumbsucking habits. Therefore, we encourage our housestaff very strongly to promote discontinuation of bottle feeding or pacifier use after 1 year of age. The American Academy of Pediatric Orthodontists is in full agreement with this.

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Professor Leighton comments:

Many surveys have shown a relationship between sucking habits and malocclusion of the teeth in the deciduous dentition, and even in the mixed dentition. However, it has been shown in a longitudinal study extending from birth to maturity (more than 17 years of age), that the effects of the sucking habit, an increased overjet and an open bite, are both resolved eventually when the habit ceases before the permanent dentition is established. Consequently, are usually corrected spontaneously when the deciduous canines are shed. There were a few cases where a large overjet remained, but these occurred just as frequently where no sucking habit had been indulged. The clinical value of a feeding bottle might be justifiably condemned for hygienic reasons, but not because of any effect it may have on the teeth, which is transitory.

Trends in preterm survival and cerebral haemorrhage

Str.—The observation by Cooke that the survival of preterm infants over a 10 year period improved while cerebral haemorrhage diagnosed by ultrasound showed no significant trend remarkably makes no comment on the incidence of this pathology among deaths as opposed to survivors. If the author wishes to demonstrate a relationship, or the absence of a relationship, between a potentially lethal condition and the numbers of deaths (or survivors as practitioners in child health tend to view the matter) then knowing the incidence of the condition among patients who die is invaluable.

It cannot be assumed that the overall incidence of cerebral haemorrhage as shown by ultrasound during life is identical to its incidence at death. If cerebral haemorrhage is more common in patients who die its lethal potential is to some degree demonstrated. If cerebral haemorrhage is more common in survivors, a beneficial effect or association has to be considered. If the incidence is the same then one has to regard cerebral bleeding as a phenomenon which is unrelated to death or survival and which is there by chance.

The author cannot resolve this matter simply by re-examining the ultrasound reports and asporting them to death certificates. This is because it would be open to the criticism, as indeed the paper as it stands is open to the criticism, that cerebral haemorrhage occurred after the last cerebral scan and was in fact the major cause of death. This criticism can only be satisfied by postmortem ultrasonography or what is far better, necropsy. The advantage of the latter is that the age and extent of haemorrhage and its relevance to the outcome is more accurately assessed.

As a pathologist I cannot help being amused by the irony of this situation. Pathologists are always open to the criticism that photographs the incidence of a disease in patients who die does not in itself indicate the incidence in those who are alive. The argument still carries

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