

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 through 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 lipid-treated infants 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 they now feel should be conducted. 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 for their 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 early use 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

SIR,—In his excellent article about the aetiology of malocclusion of the teeth,¹ 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 bottle 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 above, or to a much lesser extent, thumbsucking. We, therefore, encourage our housestaff very strongly to promote discontinuation of bottle feeding or pacifier use after 1 year of age. The American Association of Pediatric Orthodontists is in full agreement with this.

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1 Leighton BC. Aetiology of malocclusion of the teeth. *Arch Dis Child* 1991;66:1011-2.

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.^{1 2} Crossbites 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 use of a comforter or prolonged use 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.

1 Leighton BC. Symposium on aspects of the dental development of the child. 2. The early development of cross-bites. *The Dental Practitioner* 1966;17:145-52.

2 Leighton BC. Early recognition of normal occlusion. In: McNamara, J. ed. *The biology of occlusal development*. Ann Arbor: The Center for Human Growth and Development, University of Michigan, 1977:147-67.

Vocal cord paralysis as a presenting sign of acute spinal muscular atrophy (SMA type 1)

SIR,—Acute diaphragmatic dysfunction can be the first sign of spinal muscular atrophy (SMA) type 1 (Werdnig-Hoffmann syndrome),¹ but early respiratory distress due to vocal cord paralysis has to our knowledge not been reported.

Case report

A girl aged 5 weeks was admitted for a progressive inspiratory stridor of recent onset. Pregnancy, birth, and the first month of life were uneventful. Tendon reflexes were brisk. Moderate axial hypotonia without limb weakness was attributed to her respiratory problem. Direct laryngoscopy showed a complete paralysis of both vocal cords in paramedian position. Lungs and diaphragm were normal on the chest radiograph. Tracheostomy was required at 6 weeks. Extensive work-up to look for a known cause of vocal cord paralysis in infancy remained negative.²

At 4 months, swallowing difficulties were reported. Three weeks later a massive hypotonia with severe weakness of all extremities, areflexia, and fasciculations of the tongue were found. Facial and ocular movements were spared.

Electromyography showed denervation, with fibrillation potentials in proximal and distal muscles of the left leg and pectoralis major. Sensory and motor conduction velocities were normal, with a reduced compound muscular activity amplitude. A biopsy specimen of the quadriceps showed group atrophy with increased connective and adipous tissue. No inflammatory reaction, abnormal material, or significant ultrastructural changes were found. Serum creatine kinase was normal.

The child died at 5 months. A necropsy was refused.

Clinical evolution and results of the electromyography and muscle biopsy were typical of SMA type 1, but vocal cord paralysis was a

very puzzling presentation. Tracheostomy was required before the occurrence of peripheral signs of the disease. Classically, bulbar dysfunction appears late in the course of SMA type 1, respiratory failure being a consequence of progressive involvement of intercostal muscles.

Isolated vocal cord dysfunction has been described in SMA, but in a variant of the juvenile form (type 3) with distal weakness.³ The diagnosis of SMA type 1 should be considered when the clinician is confronted by unexplained vocal cord paralysis in infants.

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1 McWilliam RC, Gardner-Medwin D, Doyle D, Stephenson JBP. Diaphragmatic paralysis due to spinal muscular atrophy. An unrecognized cause of respiratory failure in infancy. *Arch Dis Child* 1985;60:145-9.

2 Cohen SR, Birns JW, Geller KA, Thompson JW. Laryngeal paralysis in children, a long term retrospective study. *Ann Otol Rhinol Laryngol* 1982;91:417-24.

3 Young ID, Harper PS. Hereditary distal spinal muscular atrophy with vocal cord paralysis. *J Neurol Neurosurg Psychiatry* 1980;43:413-8.

Trends in preterm survival and cerebral haemorrhage

SIR,—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.¹ 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.

Alas the author cannot resolve this matter simply by re-examining the ultrasound reports and apportioning them to deaths or survivors. 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 the 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 knowing 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

weight in reverse. The paediatrician who knows only the overall incidence of cerebral haemorrhage in his patients is in no position to account for why some die.

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1 Cooke RWI. Trends in preterm survival and incidence of cerebral haemorrhage 1980-9. *Arch Dis Child* 1991;66:403-7.

Professor Cooke comments:

While I agree that 'it cannot be assumed that the overall incidence of cerebral haemorrhage as shown by ultrasound during life is identical to its incidence at death', I doubt that it is very different. Nowadays, most preterm infants dying in intensive care units do so after several days, when most cerebral haemorrhage has already occurred. Postmortem examinations were permitted in just over half those infants dying, and correlation with antemortem ultrasound diagnosis was very good. The timing of cerebral haemorrhage by daily ultrasound scans is likely to be considerably more accurate than necropsy.

The intention of my paper was to show that improved survival during the last decade has not been matched by a reduction in cerebral haemorrhage (in both survivors and those dying), implying that the causes of mortality and cerebral morbidity were not necessarily the same. I was not trying to suggest that haemorrhage either caused death or promoted survival.

If annual haemorrhage rates are examined according to whether the infant lived or died, no significant changes are seen over the decade. The chances of surviving with cerebral haemorrhage of any grade increased significantly from 50 to 70% over the decade because of increased survival overall. The survival of infants with major haemorrhage remains unchanged at 40%.

While appreciating the value of a good perinatal pathology service, which Dr Barson naturally seeks to emphasise, I still believe that a more complete cohort for studying survival and cerebral haemorrhage rates is likely to be achieved in vivo rather than by postmortem studies.

Wozniak). A few hours spent working through a collection of 'data interpretation' problems such as that compiled by Walter, Lenton, and Gabriel (Butterworths) was time well spent.

Although many of my contemporaries based their reading around the standard British paediatric text by Forfar and Arneil, I personally found the American equivalent, Nelson (Behrman and Vaughan) to be more user friendly, though the fact that it reflects American practice is a drawback. In addition, I enjoyed working from many smaller specialist texts. I liked the layout of *Heart Disease in Paediatrics* by Jordan and Scott (Butterworths) and found *Respiratory Illness in Children* (Phelan, Landau, and Olinsky; Blackwell) another text capable of keeping me interested and awake after a night on call.

As previous contributors to this section have noted, the mysteries of child development were never so well unravelled as in Illingworth's *The Development of the Infant and Young Child* (Churchill) and it was read from cover to cover. Other texts grubby with use are Robertson's *Manual of Neonatal Intensive Care* and the Department of Health's publication *Immunisation against Infectious Disease* (particularly recommended as it was free).

Time did not permit exhaustive coverage of many of the specialist texts available and so *Liver Disease in Childhood* (Mowat), *Paediatric Neurology* (Brett; Churchill), and *Practical Paediatric Gastroenterology* (Walker-Smith, Hamilton, and Walker; Butterworths) were delved into on a (sometimes highly) selective basis.

I have always tried to keep copies of good reviews of a particular topic and have found the *North American Clinics in Pediatrics* to be a prolific source of these. The latest in the series of *Recent Advances in Paediatrics* series (edited by David; Churchill) maintains the high standards set by previous authors and is useful preparation for the viva part of the clinicals. Further recommended preparation for the viva includes a review of the past year's annotations section in this journal. Streetwise candidates will also have searched recent leader articles in the *BMJ* and *Lancet*, reading those with a paediatric bent, and only those intent on resitting will have failed to research the literature on anything paediatric receiving recent media attention!

There are, of course, many more things to be read and in retrospect the task seems no less enormous. Good luck to all those attempting the leap.

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many children from collapsing under a strain, and the positive benefits of stress for personal growth. For those interested in research in this field, there are numerous hints about areas where all is speculation and guesswork, which would benefit from further investigation.

The author also draws threads together from biochemistry, physiology, and psychology to help to explain both how people react differently under similar conditions and the mechanisms by which this happens. Different techniques involved in psychological research are described.

The final section of the book is probably of the most practical use to the paediatrician or child psychiatrist. This grows out of previous chapters to suggest how to investigate and treat children whose current problems may stem from past experiences. Included is some advice regarding post-traumatic stress disorder, so much in the news recently, but with particular reference to children. The book concludes with a welcome section on how insights from psychological research should guide public policy related to children and families in the future.

There are extensive references and a useful index. As a paediatrician with an interest in aspects of child guidance I found this book informative, though requiring concentration as much of the jargon was unfamiliar. In this context, the few minor misprints were irritating and distracting. However I would recommend it as an interesting book for all who work with children to dip into and particularly as a review of the state of the art and 'where to next' for those interested in research in this field.

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The Special Education Handbook. Phillip Williams. (Pp 434; £55 hardback.) Open University Press, 1991. ISBN 0-335-09314-0.

Every profession has its own jargon and special education brings together teachers, therapists, doctors, psychologists, nurses, and social workers. Anyone new to the field knows what it is like to feel bamboozled by unfamiliar terminology. They, like me, will find this book invaluable.

The term 'handbook' is somewhat misleading, this is a comprehensive reference book with items as diverse as Apgar scores and Versabril explained. Topics are presented in alphabetical order and the whole spectrum of special educational need is covered. I was impressed by the medical entries: Down's syndrome, diabetes, and fragile X syndrome are among the many conditions that are clearly and thoughtfully discussed. The emphasis is on practical information that is relevant to education, but all the important facts are included. Medical contributions are not limited to specific conditions, there is mention here of items such as distraction testing, exclusion diets, and child abuse.

As a doctor I have found this book useful as a reference for educational terms such as DATAPAC, and for providing information on the various psychological tests in use. As a newcomer to Britain I have found the synopses of the Warnock, Fish, and Swann reports and the information on relevant legislation very helpful. Entries are readable, concise, and up to date and references for further reading are supplied throughout the text. The summary of the Children Act 1989 is one of the best I have seen so far.

SPRING BOOKS

Books I used while working for the MRCP

Knowledge, it has been said, peaks early in the career of a paediatrician, around the time that membership is undertaken, and is thereafter in steady decline, supplanted by the acquisition of clinical experience. How then to achieve a peak sufficiently high to ensure the clearing of the MRCP hurdle?

In preparing for the written exam I worked, more than once, through the collections of 'grey cases' by Field and Stroobant (relatively easy) and Joss and Rose (more taxing and probably more representative!) and devoured any and all of the colour atlases (Dyanski-Klein, Milner and Herber, Booth and

Life Experiences, Development and Childhood Psychopathology. Ian M Goodyer. (Pp 292; £39 hardback.) John Wiley and Sons, 1990. ISBN 0-471-91602-1.

My first thought on receiving this book was—this is what we need, something that tells us what proved effect life experiences have on children. The author attempts to tackle this large and difficult subject, and approaches it developmentally, taking into account the different effects of the same event or stress on children at various developmental stages. Deliberate effort has been made to sort out 'fact' proved by research, from assumption and gut feeling, and to tease out key factors leading to psychopathology from those which have only a secondary effect, acting via intermediary forces. Time is also taken to elucidate the protective factors which prevent