LETTERS TO
THE EDITOR

Epidemiology of cerebral palsy

EDITOR,—The recent collaborative publication on the epidemiology of cerebral palsy, incorporating data from three cerebral palsy registers, categorises levels of functional disability to reflect difficulties with ambulation, manual dexterity, and learning.

"We agree that "...severity of disability needs to be recorded..." to facilitate comprehensive monitoring and rational planning, but a measure of how such disability affects the life of a child would further improve our understanding..."

The North of England Cerebral Palsy Register contains data on children born between 1960 and 1990 to mothers resident in Newcastle, Northumberland, and North Tyneside. This Register includes a unitary measure of impact of disability, comprising six contributing dimensions: 1 The functional disabilities described by Pharaoh and colleagues would be captured within the dimensions of mobility, physical independence, and schooling. We reviewed data on children born 1984-9, and identified interesting similarities and differences to the findings of Pharaoh et al.

"During 1984-9, there were 57 605 live births in Newcastle, Northumberland, and North Tyneside. Among these live births were 316 neonatal deaths and 125 cases of cerebral palsy, giving a neonatal mortality rate of 5.5 per 1000 live births and a cerebral palsy rate of 2.2 per 1000 neonatal survivors. Birthweight and clinical type of cerebral palsy were known for all children. Data on the impact of disability are available for 100/125."

Neonatal mortality and cerebral palsy prevalences are almost identical with those found by Pharaoh et al, as is the observation that the type of cerebral palsy differs among different birthweight groups, with the lightest and heaviest groups showing fewer cases of diplegia and more cases of hemiplegia (table 1).

Table 2 shows the severity of impact of disability for mobility, physical independence, and schooling. After excluding those of unknown severity, like Pharaoh et al we found that over 40% of children, irrespective of birthweight, have severe mobility problems. Unlike Pharaoh and his colleagues, however, we found no differences in physical independence among birthweight groups (14/51 children born >2500 g vs 15/49 of those born <2500 g had severe problems, and in our cohort, the proportion of children with severe educational problems increased with decreasing birthweight, corresponding to some of the findings of follow up studies of children with low birthweight..."

"We conclude that the measure of the impact of disability on the lives of children permits greater precision in describing the epidemiology of cerebral palsy..." to the study group, of whom there were 101 survivors. At 2 years of age, four in the study group were lost to follow up as were two in the control group. Using the recalculation suggested by Tin et al, there is no longer a significant difference between the estimated incidence of cerebral palsy in the two groups.

Spinillo et al reported cerebral palsy in 2/92 (2.1%) cases and 4/184 (2.1%) controls. However, their two year follow up was 92 of 97 cases. Ascertainment of the outcomes of the remainder of the group, and applying Tin et al's "correction" would not have changed their conclusion of no difference in the incidence of cerebral palsy between infants born to hypertensive mothers and controls.

The case control study by Szymonowicz and Yu reported 27 cases and 26 controls and came to exactly the same conclusion as Gray et al. All had a two year follow up, but it is not stated in their paper whether complete follow up was achieved or whether the sample was selected retrospectively on the basis of the availability of two year follow up data. A prospective study designed to have 80% power to show a protective effect of pre-eclampsia of 25% against cerebral palsy, assuming a 5% cerebral palsy rate in the control group, would require more than 4000 babies in each study arm. Even to detect a 50% reduction from a 10% rate of cerebral palsy in the controls would require about 500 babies in each arm. The lowest number of cases of cerebral palsy in hypertensive mothers in the three case control studies is 216. In view of Tin et al's timely demonstration of the importance of the effects of incomplete follow up and the above considerations, it is prudent to await further prospective studies before accepting Gray et al's conclusion that "maternal hypertension has a protective effect against cerebral palsy in very preterm infants..."

Maternal hypertension and its association with cerebral palsy in very preterm infants

EDITOR,—Gray et al recently reported an apparently new observation that "maternal hypertension has a protective effect against cerebral palsy in very preterm infants." Do their data support this conclusion, which is different from the conclusion reached by two other case control studies 1 on this topic?

"In the same issue, Tin et al remind us that those infants who are difficult to follow up have different (worse) outcomes than those infants who could be followed up without great difficulty. They provide a key message that "...studies where it is not possible to see some children for assessment might usefully include a calculation of what the total prevalence would be if there was a fivefold difference in the proportion with the condition in question among the children who were not seen.""

Gray et al's study comprised 107 in the study group, of whom there were 101 survivors. At 2 years of age, four in the study group were lost to follow up as were two in the control group. Using the recalculation suggested by Tin et al, there is no longer a significant difference between the estimated incidence of cerebral palsy in the two groups.

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Neurological adverse effects of early postnatal dexamethasone in very preterm infants

Environ.—Administration of dexamethasone very early after birth has been explored as a prophylactic treatment of bronchopulmonary dysplasia (BPD). The results have been conflicting. Saunders et al and the recent meta-analysis by Tushar Bhuta and Arne Olsson1 concluded a non-significant reduction in the odds of BPD. However, Tapia et al recently failed to show a reduction in its incidence. None of these studies has considered the impact of the treatment on the rate of periventricular leucomalacia (PVL). We used a protocol similar to Saund’s study design (0.25 mg/kg intravenously twice before 24 hours of life) in neonates born diffuse 20-week gestation with respiratory distress syndrome. This open study conducted over 20 months was interrupted after an important increase in cystic PVL rate had been observed. Indeed, the incidence of PVL (15%) was significantly higher than that observed during the preceding 20 months (7%) and the following 20 months (7%) (p < 0.02). Moreover, during the same period (73 treated and 99 untreated infants), PVL rate was higher in group treated with dexamethasone (25 vs 9%; p < 0.05, χ2 test after controlling for gestational age). Neonatal morbidity in the treated group was greater, but did not influence the high cystic PVL rate observed during the treatment period. Indeed, early neonatal mortality (< 7 days), the rates of respiratory distress syndrome, haemodynamic failure and congenital abnormality, as well as the circumstances of delivery known to be associated with high rate of PVL (premature rupture of the membranes, intrauterine infection, venal bleeding) were not statistically different before, during, and after the treatment period. Adverse effects on neuromotor functions have already been reported by Yeh et al in an early prolonged trial of dexamethasone. As far as the controlled trials on its benefits are concerned, early postnatal administration of dexamethasone should be considered with caution, as long as follow up results of other randomised studies are not yet available.

to be struck between the benefit of the treatment and any downstream adverse effects. Their question is important and needs careful study.


Impaired phagocytosis and opsonisation towards group B streptococci in preterm infants

EDITOR.—We were interested to see the article by Kallman et al, who have elegantly demonstrated that respiratory burst activity is reduced in preterm infants compared with term infants and adults, but can be increased (“primed”) by prior exposure to cytokines (rhGM-CSF) and interleukin 6. An alternative approach in treatment is to enhance defective PMNL function with the administration of rhGM-CSF in whole blood. Pediatr Res 1994;36:623-7.

BOOK REVIEWS


This is the third edition of a well recognised neonatal handbook. The authors declare their intention to provide a book of a practical nature as a ward guide for medical and nursing staff caring for newborn infants. Much revision has occurred since the previous edition and the depth and breadth of contributions is reflected in the lengthy list of editors and contributors, all of whom are recognised as experienced practitioners. Have the editors succeeded in their aim? Essentially, yes. The book is of a size and price to be readily accessible. It is very clear in its layout and provides essential neonatal information a shopfloor neonatologist needs. Each section has a useful bibliography as a source for more extensive information.

My main criticism is questionable information. A brief introductory sentence pointing out that local practices may be at variance with those detailed, and should be respected, would be helpful to junior and nursing staff. Some of the information is set out didactically with no hint that there is a wide body of practice. Instances would be the roles of ultrasound and serum screening in antenatal practice, the use of Ribavarin for RSV bronchiolitis, and the choice of anticonvulsants for seizures. There is little evidence that outcome in perinatal asphyxia is helped by the use of therapies to reduce raised intracranial pressure, so mannitol is not used in many centres. The value of cerebral ultrasound in determining cerebral oedema is very questionable and it is now recognised that antenatally diagnosed cystic adenomatous malformations of the lung can regress, obviating the need for lobectomy. The infusion dose of tolazoline is given as 0.1 mg/kg/hour; do they mean this or is this a printing error? These are minor quibbles as most of the management decisions relating to these points will be conducted at a senior level, but for juniors who use this book it would be helpful to know that not all questions have only one answer.

The one area in which it is important to state the opinion given is contentious, is the use of 40%, rather than 100% oxygen for resuscitation. It is important that all personnel called on to resuscitate newborns are aware of the local policy. The sections on caring for the family and the dying baby are extremely well written, particularly when considering the size and scope of the book. I recommend anyone working in this area to read them. The ethical and personal issues which have to be faced are clearly, succinctly and compassionately addressed.

The editorial team should be congratulated on producing such a compact yet useful book which, I am sure, will find its way onto many neonatal units.

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“Much study is a weariness of the flesh” stated the writer from Ecclesiastes. The acquisition of medical knowledge requires the consumption of much large, indigestible tomes. This compact textbook on critical care of the surgical newborn provides a suitable antidote. It is small, concise, light in weight, but packs a quality punch. It is a good balance of relevant embryology, antenatal diagnosis, appropriate investigations, and operative management. From a surgical perspective I found the chapters on prenatal diagnosis and ultrasound particularly useful, teaching me many things I probably ought to have known already.

It covers the usual range of child topics on neonatal care well and succinctly and in a sufficiently different way to be interesting. It also discusses ethics and anaesthetics and algaezia. The chapter on anaesthetics finally answered a question I have asked every anaesthetist but to which I have never received a satisfactory reply—namely, what is the intraoperative/anaesthetic death rate in babies? The intraoperative death rate quoted of 8.3/1000 for infants under one month seemed more realistic than the optimistic replies from colleagues. The intraoperative death rate of children between one to 12 months of 0.8/1000 seemed higher than I imagined, but will influence what I say to parents when seeking informed consent in the future.

Unusually for a textbook, it held my interest and attention and was readable and will influence my clinical practice in the future.

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