Dexamethasone treatment and cerebral palsy

EDITOR,—Referring to the paper entitled Early postnatal dexamethasone treatment and increased incidence of cerebral palsy by Shinwell et al1, both its title and the first paragraph of the Discussion imply that the use of postnatal dexamethasone may lead to cerebral palsy. However, it is the misuse of the term “incidence” that gives rise to this interpretation. The authors did not, neither could they, provide incidence data. What they presented was cerebral palsy prevalence data. If it is accepted that prevalence and not incidence data are provided, then a very different interpretation of the findings can be made. Supposing the cerebral impairment of cerebral palsy occurred prepartum, then the use of dexamethasone may have allowed these children to survive, whereas, had they received the placebo, they would have died before a diagnosis of cerebral palsy could be made. This would account for the higher prevalence of cerebral palsy in the dexamethasone compared with the placebo group. Support for this interpretation comes from the authors’ statement “Eleven (22%) of the 51 children with cerebral palsy had normal neonatal ultrasound scans. All 11 of these infants were treated with dexamethasone”. This suggests that the cerebral impairment was prenatal in timing. Further support comes from the observation that there were fewer cases of IVH in the dexamethasone group, although the difference did not quite attain the conventional level of statistical significance.

It needs to be appreciated that prevalence = incidence × duration of disease. The duration of the disease is affected by how long the child (or fetus) survives. Unless it is known what happens to the fetus from the time of conception, it is not possible to determine incidence in those diseases that have their origin during uterine development.

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Confirmed group B streptococcus infection: the tip of the iceberg

EDITOR,—Beardsall and colleagues are to be congratulated on presenting further evidence that group B streptococcus gives rise to a significant burden of disease in some areas of the United Kingdom.1 Retrospective data collected at St George’s Hospital is in agreement with the authors’ suggestion that culture proven sepsis under represents the true burden of disease. Firstly, we conducted a retrospective search for cases of culture positive group B streptococcus (either blood or cerebrospinal fluid) amongst a cohort of consecutive births at the hospital between 1 January 1994 and 31 October 1998, and comprising 16 910 births.

Secondly, we conducted a retrospective analysis of all babies colonised with group B streptococcus (deep ear swabs taken in the first six hours of life which were positive for group B streptococcus), and who were screened and treated for suspected early onset infection in the first 72 hours of life, between 1 April 1997 and 31 March 1998. Probable early onset group B streptococcus infection was defined as: a positive deep ear swab in a baby with clinical pneumonia or sepsis (either fever >38°C on one occasion or >37.5°C on two occasions separated by at least one hour, or two or more of: poor perfusion, respiratory distress, thrombocytopenia, leucopenia <5 × 10⁹/L, persistent glucose imbalance or abdominal distension, bilious aspirates, or blood in stool in a baby <72 hours of age).

Twelve of 16 910 babies had blood cultures positive for group B streptococcus and group B streptococcus was cultured from the cerebrospinal fluid of one baby whose blood culture was negative. This gives an infection rate of 0.77/1000.

Of 3438 deliveries from 1 April 1997 to 31 March 1998, there were nine babies with probable group B streptococcus infection, giving an incidence of 2.6/1000.

Because of the usual problems related to retrospective data analysis this figure may still under represent the true burden of disease.

We are currently prospectively evaluating the incidence of probable as well as proven group B streptococcus infection in order to estimate the true burden of disease in our local population. It is only in the light of such data that we will be able to develop evidence based guidelines for the prevention and management of this disease.

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BOOK REVIEW


There are a number of books available on neonatal infections. What does this book offer that is new, apart from renaming neonatologists? The authors preface the book by saying it is not intended as a reference book, but as a concise up to date review.

It is really a series of reviews, written by a range of experts from Europe, South Africa, North and South America. There are strong chapters on maternal immunity and infection and, although there is some overlap of content, these are illuminating. The chapters on specific infections are certainly up to date, and include 1999 references.

HIV-1 infection and group B streptococcal infections are covered well, but the chapters on papillomavirus and hepatitis viruses are longer, whereas other bacteria such as staphylococci and Gram negative bacilli are not covered.

One of the book’s strengths is that developing countries are not ignored, although a chapter on the particular problems in these countries would have been even more valuable. The weaknesses include the cursory attention to neonatal bacterial infection and to the management of a baby with possible or proven sepsis, and the paucity of figures.

This book will sit on my shelf until I am asked a thorny question about HIV or toxoplasmosis, when I will use it as an up to date source of information. It is unlikely to gather much dust.

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Arch Dis Child Fetal Neonatal Ed 2001 84: F140
doi: 10.1136/fn.84.2.F140a

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