Letters to the editor

'Maternal antecedents to cerebral palsy in preterm infants'

SIR–Peter Gray and colleagues[1] recently reported a reduction in the incidence of cerebral palsy (CP) in infants born between 24 and 27 weeks' gestation after antenatal administration of corticosteroids, in comparison with control infants matched for gestational age (odds ratio=0.4, 95% CI 0.1 to 0.98).

We have recently completed a similar study comparing 148 infants with CP with 291 matched control infants for all gestational ages, in which we examined antenatal variables. We would like to report a very similar finding of a relative risk ratio (RRR) of 0.44 (CI 0.25 to 0.77, unadjusted for matching) for preterm babies of less than 37 weeks' gestation (25 of 78 infants with CP, 80 of 154 control infants).

If data pertaining to steroids given in labour are included, the RRR is 0.49 (CI 0.28 to 0.86, $p=0.013$; 237 observations included in the analysis; multivariate logistic regression was used with adjustments for sex, plurality, primiparous or not, and birthweight <2500 g or ≥ 2500 g).

RRR for less than 32 weeks' gestation at birth was 0.36 (CI 0.18 to 0.73, $p=0.005$; 155 observations included in the analysis). Further breakdown of this group gave RRR of 0.29 (CI 0.06 to 1.34, $p=0.113$; 37 observations included in the analysis) for <27 weeks' gestation and for 28 to 31 weeks the RRR was 0.36 (CI 0.16 to 0.82, $p=0.014$; 118 observations included in the analysis). For these further analyses, birthweight was not used as an adjustment variable.

Our data supports Dr Gray's assertion that the use of antenatal steroids significantly reduces the risk of CP in babies born before 32 weeks' gestation.

Janet Walstab[a]
Robin Bell[b]
Dinah Reddihough[c]
Shaun Brennecke[d]
Christine Bessell[e]
Norman Beischer[f]

[a]*Research Assistant, Murdoch Children's Research Institute;*
[b]*Perinatal Epidemiologist;*
[c]*Director, Child Development and Rehabilitation, Royal Children's Hospital, Parkville;*
[d]*Professor University of Melbourne Department of Obstetrics and Gynaecology, Royal Women's Hospital, Carlton;*
[e]*Medical Director, Labour Ward, Monash Medical Centre, 246 Clayton Road, Clayton;*
[f]*Director, Medical Research Foundation for Women and Babies, East Melbourne, Victoria, Australia.*

Correspondence to:
[a]*Cerebral Palsy Aetiology Research*
Department of Child Development and Rehabilitation
Royal Children's Hospital
Flemington Road
Parkville
Victoria 3052, Australia.
E-mail:cpreg@cryptic.rch.unimelb.edu.au

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'Sagittal sinus thrombosis in a teenager: homocystinuria associated with reversible antithrombin deficiency'

SIR–We report a 12-year-old male with superior sagittal sinus thrombosis diagnosed on MRI. He had minor Marfanoid features with long fingers, pectus excavatum, pes cavus, and long-standing cognitive and behavioural difficulties. A diagnosis of classical homocystinuria was made on the basis of a fasting plasma homocysteine level of 246 μmol/l (reference: 5–15 μmol/l) and a plasma methionine of 413 μmol/l (reference: <77 μmol/l). At the same time he was also found to have a reduced antithrombin (AT III) activity of 54% (reference: 78–124%). Antithrombin deficiency is usually autosomal dominant but both parents had normal levels.

Initial treatment of his sinus thrombosis with intravenous heparin was followed by subcutaneous low-molecular-weight heparin. Homocystinuria was treated with pyridoxine with the subsequent addition of betaine. Homocysteine and methionine levels fell to 55 and 43 μmol/l respectively. Concurrently the antithrombin activity increased to borderline low/normal levels and anticoagulation treatment was discontinued. There was a good clinical recovery from the sagittal sinus thrombosis. The association of homocystinuria and antithrombin deficiency has been described although the mechanism is unclear.[1,2] In this case, as in previously reported cases, antithrombin deficiency was corrected by treatment of the homocystinuria.

The phenotype of homocystinuria is variable and may be subtle. When antithrombin deficiency is identified in the investigation of a thrombotic event, underlying homocystinuria should be excluded. Where such an association exists, lifelong anticoagulation can be avoided as antithrombin activity is corrected by treatment of the homocystinuria.

Ewoud Vorstman,[a] *Specialist Registrar Paediatrics*
David Keeling,[a] *Consultant Haematologist*
James Leonard,[b] *Professor of Paediatric Metabolic Disease*
Michael Pike,[a] *Consultant Paediatric Neurologist*

[a]*Department of Paediatrics, Oxford Radcliffe Hospital NHS Trust, Oxford;*
[b]*Institute of Child Health, London, UK.*

Correspondence to:
Ewoud Vorstman
Department of Paediatrics
John Radcliffe Hospital
Headington
Oxford OX3 9DU, UK
E-mail: e.vorstman@virgin.net

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