Archives of Disease in Childhood, 1987, **62**, 869–871

Correspondence

Vasoactivity of the major intracranial arteries in the newborn child

Sir,

I read with interest the paper of Drayton and Skidmore, showing vasoactivity of the cerebral circulation in preterm infants.[1] I note in their study population four infants had developed intraventricular haemorrhage and five infants had signs of periventricular leucomalacia. Volpe has shown with positron emission tomography that intracranial haemorrhage has a dramatic effect on regional cerebral blood flow,[2] and it is well recognised that intracranial haemorrhage can cause arterial spasm. Previous studies have shown that intraventricular haemorrhage occurs soon after birth in many infants.[3 4] It is possible that the changes in cerebral vasoactivity that Drayton and Skidmore describe in their paper are secondary to intraventricular haemorrhage. If the results of the nine infants with either periventricular haemorrhage or leucomalacia are removed from the preterm study population, do the results reported still pertain? If so this makes a much more interesting paper.

References

1 Drayton MR, Skidmore R. Vasoactivity of the major intracranial arteries in newborn infants. *Arch Dis Child* 1987;**62**:236–40.

2 Volpe JJ, Herscovitch P, Perlman JM, Raichle ME. Positron emission tomography in the newborn: Extensive impairment of regional cerebral blood flow with intraventricular haemorrhage and haemorrhagic intracerebral involvement. *Paediatrics* 1983;**72**:589–601.

3 Ch de Crespigny L, Mackay R, Murton LJ, Roy RND, Robinson PH. Timing of neonatal cerebroventricular haemorrhage with ultrasound. *Arch Dis Child* 1982;**57**:231–3.

4 Beverley DW, Chance GW, Coates CF. Intraventricular haemorrhage—timing of occurrence and relationship to perinatal events. *Br J Obstet Gynaecol* 1984;**91**:1007–13.

D Beverley
Clarendon Wing,
The General Infirmary at
Belmont Grove,
Leeds LS2 9NS

Dr Drayton comments:

Dr Beverley enquires whether the changes in cerebral artery calibre reported in March 1987 could be secondary to intracranial pathology. The nine infants with abnormal real-time ultrasound scans were heterogenous for both type and timing of abnormal appearance. When these infants are removed from the analysis the same results pertain. In the remaining 21 infants, mean ACA velocity increased from 2·5 to 5·5 cm/second; mean MCA velocity increased from 4·8 to 8·8 cm/second. There was no significant change in head and neck perfusion and the mean standardised flow:velocity ratio decreased from 19·5 to 8·5.

These changes are clearly not the result of intracranial haemorrhage, but the implied early vasodilation is more likely to be a factor in its genesis.

M R Drayton
Department of Neonatal Medicine,
Southmead General Hospital,
Westbury-On-Trym,
Bristol BS10 5NB

Cardiovascular effects of tolazoline and ranitidine

Sir,

Tolazoline, used in standard doses, reliably produces gastric erosions and bleeding. The article by Bush *et al*[1] suggests that ranitidine should not be used to counteract this undesirable side effect. For the past seven years we have used 2·8% sodium bicarbonate stomach washouts to reduce bleeding from erosions caused by tolazoline. In full term infants we use 10 ml of this solution to wash out the stomach every two hours until the bleeding stops, and subsequently, at longer intervals. Usually 1–2 ml remains in the stomach each time. We have had no problems with hypernatraemia to date, although most infants develop a mild alkalosis. Bleeding usually stops or is greatly diminished within 24 hours.

Reference

1 Bush A, Busst CM, Knight WB, Shinebourne EA. Cardiovascular effects of tolazoline and ranitidine. *Arch Dis Child* 1987;**62**:241–6.

E M Kiely
Hospital for Sick Children,
Great Ormond Street,
London WC1N 3JH

Management of asthma in schools

Sir,

Hill, Britten, and Tattersfield's views on the management of asthma in schools and Dr Couriel's letter, suggesting that the school medical service could help to identify the underdiagnosed and undertreated,[1 2] spur me to enlighten them on the school doctor's role. Every mother seen in the small group of schools where I work is asked if their child

has any relevant symptoms. If asthma is diagnosed this is fully discussed with parent and child and types of treatment outlined. If the diagnosis is new or if treatment administered so far is ineffective such information is passed on (*most politely*) to the family doctor. There is rarely a response. When the child is next seen at school, one is told that: (i) the cough/wheezing is better or; (ii) that it persists with or without treatment, or; (iii) 'my doctor says this isn't asthma'. Thus identification of asthma in school is easy; improving treatment is more difficult.

With regard to the three questions asked by Drs Hill, Britten, and Tattersfield:

1 How well are schools informed about children with asthma? This varies greatly. The question then is who should tell whom? School medical officers do not see children in this area until the term after school admission, furthermore, they are well advised on confidentiality of medical information. It would be confusing and unhelpful if the school doctor were to tell the school that a child has asthma, especially if parent or family doctor is unwilling to accept this diagnosis. Such a problem is not insoluble but takes time and tact.

2 How much access should children have to inhalers? The answer should surely be an individual one depending on many factors such as age, reliability, capability etc. Unfortunately, there have been schools where head-teachers disallow any treatment in school or insist on mothers coming to school to administer it. It is to be hoped there is now a more considerate view.

3 Should teachers be given instructions in managing childhood illness? For asthma this would be generally useful but such advice appears to attract interest mainly from those personally involved with asthma and those teaching the very young.

In conclusion, there is undoubtedly a need for much more widespread education on asthma and your Journal is to be congratulated on having four articles on this subject in the April issue.

Meanwhile, one continues to try to spread the word as 'that school doctor.'

References

[1] Hill RA, Britton JR, Tattersfield AE. Management of asthma in schools. *Arch Dis Child* 1987;**62**:414–5.
[2] Couriel JM. The school entry examination. *Arch Dis Child* 1987;**62**:432.

HELENOR PRATT
Wycombe General Hospital,
High Wycombe,
Buckinghamshire
HP11 2TT

Autosomal dominant polycystic kidney disease in children

Sir,

Taitz *et al* refer to autosomal dominant polycystic kidney disease (ADPKD) in children and the value of the family history in its diagnosis.[1] Their observation is in accordance with our experience in the cooperative study on cystic kidneys of the *Arbeitsgemeinschaft für Pädiatrische Nephrologie*. We know about 49 children with ADPKD, including cases with 'Potter sequence', as well as children with only slight ultrasonographic changes without any renal impairment. The authors' conclusion, drawn from the bimodal distribution of the age at detection of previously published cases, which postulates two distinctive groups is not without problems. As the authors admit themselves, childhood cases may be underreported. Complete ascertainment of a great number of children at risk is necessary; and rare cases with enlarged kidneys should also be detected. There are at least six possible explanations for early onset, of which Taitz *et al* mention only two (1 and 2):

1 Homozygosity for ADPKD is unlikely because, as the authors mention, there are no reports of children with early signs both of whose parents have ADPKD.
2 Cases of early manifestation might be due to a specific genotype. The authors favour the hypothesis that children with grossly enlarged kidneys are either compounds for the gene of the autosomal recessive (ARPKD) as well as of the autosomal dominant variety, or that they are heterozygous for both mutations at different loci.
3 Early clinical signs could be part of a spectrum of severity, ranging from cases with grossly enlarged kidneys in newborn infants to children who don't show any symptoms until adulthood. Age at onset might then show a unimodal distribution due to a polygenic background, as is known from Huntington's chorea.[2]
4 One possibility could be the existence of 'modifying alleles' at the same gene locus[3] to explain rare pedigrees of spinal muscular atrophy.[4] The critical study would be to screen systematically a large number of children at risk for ADPKD, starting at birth, or even prenatally. A bimodal distribution of age at onset would strongly point to the existence of specific genotypes, either due to compounds or double heterozygosity, where the frequency of the second mode would even permit an estimate of the prevalence of the responsible gene(s).
5 Although unlikely because of transmission through both affected fathers as well as affected mothers, sex related modifying influences should also be considered as occurs in early onset cases of myotonic dystrophy.
6 Rare exogeneous factors (infections, nutrition?) should also be considered.

Despite the so far unknown nature of early manifestation of ADPKD in children the observation of familial cases should be taken into account in genetic counselling.

References

[1] Taitz LS, Brown CB, Blank CE, Steiner GM. Screening for polycystic kidney disease: importance of clinical presentation in the new-born. *Arch Dis Child* 1987;**62**:45–9.
[2] Wendt GG, Drohm D. Die Huntingtonsche Chorea. Eine populations-genetische Studie. In: Becker PE, Lenz W, Vogel F, Wendt GG, eds. *Fortschritte der allgemeinen und klinischen Humangenetik*. Stuttgart: Georg Thieme, 1972.
[3] Zerres K, Hansmann M, Knöpfle G, Stephan M. Prenatal diagnosis of genetically determined early manifestation of autosomal dominant polycystic kidney disease? *Hum Genet* 1985;**71**:368–9.