

The authors make it clear that late onset periventricular leucomalacia is three times more common than early onset cases. It is worth pointing out that this was not the situation that was apparent to perinatal pathologists in the days before ultrasonography existed. Only recently have significant numbers of small babies been kept alive long enough for periventricular cavitation to become manifest either by ultrasound or by direct observation postmortem. For these babies postnatal management is more likely to be relevant to their handicap than antepartum or intrapartum events. In historical terms antepartum and intrapartum brain damage has always afflicted the human race and is probably diminishing in western society. Late onset cerebral infarction in very prematurely born infants is a recent, iatrogenic problem whose increasing frequency lends greater urgency to its solution.

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- 1 Sinha SK, D'Souza SW, Rivlin E, Chiswick ML. Ischaemic brain lesions diagnosed at birth in preterm infants: clinical events and developmental outcome. *Arch Dis Child* 1990;65: 1017-20.

Dr Sinha comments:

Dr Barson's comments on terminology seem valid, but we fail to see any such confusion arising out of our own paper. We certainly did not use the term echogenicity, ischaemia, and periventricular leucomalacia as though the terms were synonymous. Each one has a precise meaning. Echogenicity simply means increased echodensity with no visible cyst formation, whereas ischaemia describes a pathophysiological process which cannot be diagnosed by ultrasound imaging. The only expressions we used synonymously were periventricular leucomalacia and multiple periventricular cysts. The ultrasound appearances of periventricular leucomalacia have been shown to correspond exactly to the sequence of changes observed in necropsy specimens and confirmed by published studies as well as our own observation (shared by Dr Barson himself). This is why we frequently use the expression 'consistent with ischaemic brain lesions'.

We state quite clearly that the 39 babies with 'ischaemic brain lesions' had areas of increased periventricular echogenicity that were associated with or progressed to multiple cyst formation. Among nine babies, in whom periventricular echogenicity was noted soon after birth, cysts were already present in five on the initial scan, carried out between 30 minutes to two hours after birth. In three others, cysts became visible on day 3 and 5. One baby with 'early' periventricular echogenicity died on day 3 and was noted to have necropsy changes diagnostic of early periventricular leucomalacia. It is possible that in three of these nine babies, cysts might have been present at birth but were too small to be seen on ultrasound scans. In the absence of continuous scanning we cannot be precise about the timing of these ultrasound changes,

but as the progression of initial echogenicity to cyst formation takes sometime between seven to 10 days, it seemed quite reasonable to deduce that these changes might have started before or during birth.

The ability to make serial observations of the brain non-invasively by ultrasound has led to a better understanding of the pathogenesis and natural history of ischaemic brain lesions. We agree that these lesions tend to occur mostly in relation to hypoxic-ischaemic injury either at birth or during the neonatal period but similar insults can occur antenatally. This notion is supported by a number of intrapartum Doppler investigations, and there is evidence both from human and animal studies showing damage to fetal periventricular white matter in response to various forms of hypoxic-ischaemic insults. The fact that in a third of the babies in our study, who later developed disabilities, the brain damage may have already taken place by the time of birth has important implications and should not be ignored. Indeed, at all gestational ages, perinatal problems represent a continuum including prenatal, intrapartum, and neonatal events.

Neonatal infections with coagulase negative staphylococci

SIR,—In their recent leading article Millar *et al* state that resistance of coagulase negative staphylococci to penicillin, aminoglycosides, and other antibiotics is common.¹

This is not our experience at Liverpool Maternity Hospital where coagulase negative staphylococci are responsible for most proved neonatal infections.² When any infection is suspected, our practice is to treat initially with ampicillin and gentamicin until the results of cultures are available after which treatment may be adjusted according to antibiotic sensitivities. Occasionally the infant has a coagulase negative staphylococci infection and has clinically improved despite *in vitro* resistance to ampicillin and gentamicin. If this is the case these antibiotics are often continued with good effect confirming a discrepancy between *in vivo* and *in vitro* sensitivities.

Since 1986 we have used amoxicillin/clavulanic acid (Augmentin, Beecham) for coagulase negative staphylococci infections caused by penicillin, methicillin, and gentamicin resistant organisms when the initial ampicillin and gentamicin combination has failed. In a series of 70 episodes of coagulase negative staphylococci septicaemia occurring between January and July 1987 only one strain was found to be resistant to amoxicillin/clavulanic acid. The majority of strains were β -lactamase producers.

Coagulase negative staphylococci infection cannot usually be differentiated clinically from other neonatal infection; using vancomycin to treat suspected coagulase negative staphylococci infection as suggested by Millar *et al* means using it in virtually all episodes of suspected neonatal infection. From our clinical and laboratory experience we do not agree that vancomycin, which is ototoxic, nephrotoxic, has to be given by infusion and is expensive, should be the drug of first choice

for the treatment of suspected coagulase negative staphylococci infection in the neonate.

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- 1 Millar MR, Todd N, Mackay P. Neonatal infections with coagulase negative staphylococci. *Arch Dis Child* 1990;65:1015-6.
2 Hensley OJ, Hart CA, Cooke RWI. Serious infections in a neonatal care unit: a two year survey. *J Hyg (Lond)* 1985;95:289-97.

Dr Millar comments:

Vancomycin is active *in vivo* and *in vitro* against 'methicillin resistant' coagulase negative staphylococci and side effects are well documented. Coagulase negative staphylococci infection is uncommon in the first week of life, in the absence of an intravascular catheter, and in the mature neonate, so it is certainly not necessary to use vancomycin for the treatment of all episodes of suspected neonatal infection. The use of amoxicillin/clavulanic acid (Augmentin) for the treatment of neonatal infections as advocated by Shaw *et al* is indeed unusual. Neither of the two components are active *in vivo* or *in vitro* against 'methicillin resistant' staphylococci when used as single agents. The implication of Shaw *et al* (although not stated) is that resistance of 'methicillin resistant' coagulase negative staphylococci to amoxicillin is determined by β -lactamase synthesis and that the altered affinity of penicillin binding proteins for β -lactamase antibiotics does not contribute to amoxicillin resistance. If the hypothesis that amoxicillin/clavulanic acid is effective against 'methicillin resistant' coagulase negative staphylococci *in vivo* or *in vitro* can be substantiated then those data should be published.

Endotoxin induced cochlear damage

SIR,—We read with interest the paper by Tarlow *et al* describing the cochlear damage in guinea pigs produced by endotoxin perfusion of the cochlea or injection into the cerebrospinal fluid. Two points are worth making.

Firstly, the concentration of endotoxin in the cerebrospinal fluid or the cochlea is not estimated, but may be several orders of magnitude greater than that in human meningitis. For example, concentrations in cerebrospinal fluid compatible with survival in meningococcal meningitis were 85-250 pg/ml in one study.² It would be valuable to know whether cochlear toxicity in the guinea pig occurs at endotoxin concentrations in the cerebrospinal fluid comparable with those found during human meningitis.

Secondly, endotoxin mediation of cochlear damage might have important implications for children with Gram negative septicaemias as well as those with meningitis. We have seen a child who first complained of unilateral tinnitus and deafness 12 hours after presenting with meningococcal septicaemia. He had no clinical or laboratory evidence of meningitis. Subsequent audiology confirmed complete left sided hearing loss. This damage could have been mediated by endotoxin circulating in plasma.

To test the hypothesis clinically that

endotoxin damages the cochlea, children with meningococcal disease would be a suitable population to study. Children present predominantly with septicaemia, meningitis, or a mixed picture of septicaemia and meningitis.³ Deafness in survivors of fulminant septicaemia, meningococcaemia without meningitis, and meningitis alone might be commoner in all groups than in a control population. A case-control follow up study of a cohort of children with meningococcal disease would answer this question.

The practical conclusion of such a study might be that clinicians should be ordering routine audiological follow up for patients with meningococcal septicaemia, as well as for those with meningitis.

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- 1 Tarlow MJ, Comis SD, Osborne MP. Endotoxin induced damage to the cochlea in guinea pigs. *Arch Dis Child* 1991;66:181-4.
- 2 Brandtzaeg P, Kierulf P, Gaustad P, Dobloug J, Mollnes TE, Sirnes K. Systemic meningococcal disease: a model infection to study acute endotoxaemia in man. *Prog Clin Biol Res* 1988;272:263-71.
- 3 Thomson APJ, Sills J, Hart CA. Meningococcal disease in Liverpool 1977-1987: mode of presentation. *Pediatric Reviews and Communications* 1990;5:109-16.

Head injury—how community paediatricians can help

SIR,—Head injury must be the major health issue among children in the United Kingdom, accounting for 15% of deaths in children aged 1-15 years.¹ However, we question the admission rate of 45 per 1000 children per year after head injury quoted by Dr Crouchman in her recent article.² We have recently conducted a retrospective review of all children aged less than 16 years who died or were admitted to hospital after head injury in the Northern region during the eight year period 1979-86.^{1,3,4} During this period head injury was the single major cause of admission to hospital, however, the annual admission rate was only a tenth of that quoted by Dr Crouchman, being 4.9 per 1000 children. In view of the importance of accurate epidemiological data in planning services for head injured children, we believe that it is imperative that this issue is clarified.

We would also like to suggest that community paediatricians should have a leading role in the prevention of head injury and not confine their activities simply to the provision of rehabilitation services. The recent evidence that serious head injury occurs more frequently at particular times of day and in areas of high material deprivation⁵ should surely stimulate community paediatricians to take the lead in initiating appropriately targeted preventive measures.

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- 1 Sharples PM, Storey A, Aynsley-Green A, Eyre JA. Avoidable factors contributing to death of children with head injury. *BMJ* 1990;300:87-91.
- 2 Crouchman M. Head injury—how community paediatricians can help. *Arch Dis Child* 1990;65:1286-7.
- 3 Sharples PM, Storey A, Aynsley-Green A, Eyre JA. Causes of fatal childhood accidents involving head injury in the Northern region, 1979-86. *BMJ* 1990;301:1193-7.
- 4 Sharples PM, Aynsley-Green A, Eyre JA. The management of children with head injuries in district general hospitals. *Arch Dis Child* 1988;63:570.
- 5 Kraus JF, Rock A, Hemyari P. Brain injuries among infants, children, adolescents and young adults. *Am J Dis Child* 1990;144:684-91.

Dr Crouchman comments:

I must thank Drs Sharples and Eyre for pointing out the typing error in the first sentence of my paper. It should, of course, have read 45 per 10 000 children per year.

I agree that there is an important role for the consultant community paediatrician in the prevention of childhood accidents. My own view is that health education has only a limited place, and that every district needs a multi-agency group with the teeth to influence local housing, street planning, and the provision of safe play space.¹ It would seem very appropriate that consultant community paediatricians should contribute to this group as part of their essential public health function.²

- 1 Crouchman M. Children with head injuries. *BMJ* 1990;301:1289-90.
- 2 Discussion Document from Joint Working Party of the Faculty of Public Health Medicine and the British Paediatric Association. *Working together for tomorrow's children*. London: BPA, 1990:9-11.

Doppler assessment of pulmonary artery pressure in acute phase of hyaline membrane disease

SIR,—I read with interest the paper by Evans and Archer.¹ The study is well described with clear presentation of results.

I would, however, take issue with the definition of right ventricular ejection time (RVET). The authors define RVET as the time interval between the systolic waveform leaving its peak velocity and returning to the baseline. The total RVET is more appropriately defined as the time from the onset of ejection to that of zero flow. The latter definition is that used by Kitabatake *et al* and this is the main reference article by the authors.²

Any error in the measurement of total RVET will be reflected in the ratio time to peak velocity: right ventricular ejection time (TPV: RVET). The ratio as defined by Kitabatake *et al* was shown to have a linear inverse relationship to log₁₀ of the mean pulmonary artery pressure.² Any relationship to pulmonary artery pressure by the ratio TPV: RVET as described by the authors has not been verified.

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- 1 Evans NJ, Archer LNJ. Doppler assessment of pulmonary artery pressure and extrapulmonary

shunting in the acute phase of hyaline membrane disease. *Arch Dis Child* 1991;66:6-11.

- 2 Kitabatake A, Inoue M, Asao M, *et al*. Non-invasive evaluation of pulmonary hypertension by a pulsed Doppler technique. *Circulation* 1983;68:302-9.

Drs Evans and Archer comment:

We are grateful to Dr Craig for drawing our attention to an error in the text which we failed to notice at proof reading. The correct definition of right ventricular ejection time (RVET) and the one which we did indeed use is shown in the following phrase which is a corrected version of the text: 'RVET was the time interval between the systolic wave form leaving and returning to the baseline'. This is consistent with Dr Craig's definition.

Photic sneezing

SIR,—Photic sneezing is well documented in the ophthalmological¹ and neurological² literature. Light induced sneezing appears to be more common in male than female, white than black, and in those with a positive family history. Photic sneezing, an uncontrollable paroxysm of sneezing provoked by sudden exposure to intense bright light, is an occupational hazard for the ophthalmologist.

An enhanced photic sneeze reflex has recently been reported in patients with cystinosis.^{3,4} The mechanism of the reflex is obviously complex involving optic, oculomotor, and trigeminal nerves, autonomic pathways, and central brainstem structures. I am a 'sufferer' from photic sneezing, which is most noticeably inconvenient when driving into bright sunlight, and I have two boys who also demonstrate the reflex. I note, with interest, how frequently I have observed the reflex in babies.

I wonder about the evolutionary process behind this bizarre reflex? The reflex appears to be less marked as the child matures. Is this because it serves no useful purpose and is a 'vestigial' reflex?

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- 1 Lewkonia I. An infrequent response to slit lamp examination. *Br J Ophthalmol* 1969;53:493-5.
- 2 Everett HC. Sneezing in response to light. *Neurology* 1964;14:483-90.
- 3 Smith R. Editorial. *Br J Ophthalmol* 1990;74:705.
- 4 Katz B, Melles RB, Wenson MRS, Schneider JA. Photic sneeze reflex in nephropathic cystinosis. *Br J Ophthalmol* 1990;74:706-8.

Duplicate publication

SIR,—I read your recent editorial on duplicate publication with interest and some wry amusement.¹ This is not a new problem and has exercised the minds of editors of general as well as specialist journals.² It is surprising that the editors, all distinguished clinicians, should after an accurate diagnosis spend their efforts discussing control of symptoms with no reference to treating the underlying cause.

Clinicians will continue to submit multiple papers so long as appointment boards contain individuals who believe that a doctor who has published 10 papers must be twice as good as one who has published five. Why a good clinician, who wishes to practice as such,