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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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 amoxycillin/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 amoxycillin/clavulanic acid. The majority of strains were \( \beta \)-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 infection. From 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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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 intravenous 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 amoxycillin/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 amoxycillin is determined by \( \beta \)-lactamase and that the altered affinity of penicillin binding proteins for \( \beta \)-lactamase antibiotics does not contribute to amoxycillin resistance. If the hypothesis that amoxycillin/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.3 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 fulminating 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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Dr Crouchman comments

I must thank Dr Sharple and Eyre for pointing out the typing error in the first sentence of my paper. It should, of course, have read 45 per 1000 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 multiagency 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.


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. 3 Any relationship to pulmonary artery pressure by the ratio TPV: RVET as described by the authors has not been verified.


Photoc sneezing

SIR.—Photoc sneezing is well documented in the ophthalmological 1 and neurological 2 literature. Light induced sneezing appears to be more common in male than female, white than black, and in those with a positive family history. 3 Photoc 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 cistinosis. 4 The mechanism of the reflex is obviously complex involving optic, oculomotor, and trigeminal nerves, autonomic pathways, and central brainstem structures. 5 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 because it serves no useful purpose and is a 'vestigial' reflex?

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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,
Endotoxin induced cochlear damage.

A Thomson and O Marzouk

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Updated information and services can be found at:
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