advancing the onset of the pubertal growth spurt and had no adverse effect on adult height prediction.

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Ceftazidime in neonatal infections

Sir,

Ceftazidime has been shown to be effective against both Gram negative and Gram positive organisms commonly causing neonatal infections.¹ It is now being used as a first line antibiotic for the treatment of neonatal infections for the reasons given by Low *et al.*²

Their analysis suggested that ceftazidime may not be effective against Gram positive organisms. I was surprised that case 12 was included. Would it not be too optimistic to expect sterilisation of the cerebrospinal fluid 6-75 hours after a single dose when using any antibiotic? Why (case 11) was penicillin only given 15 hours before death (day 11) when ceftazidime had been begun at 16 hours, the infant's condition was deteriorating, and the organism was not sensitive? The persistence of *Staphylococcus aureus* (case 9), despite an adequate serum concentration, suggests that the child may have had an infected venous catheter or a collection of pus.

On the basis of this report I feel it is premature to abandon the use of ceftazidime as a first line drug for the treatment of neonatal infections.

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Drs Low and Bissenden comment:

Dr Blumenthal's comments arise partly because our study was based on 14 cases of proved sepsis, each of which would have merited further discussion, and the need to restrict clinical data to the absolute minimum within the constraints of the Table. Nevertheless, there is obvious confusion over case 12. This was group B streptococcal meningitis, diagnosed early, treated promptly, and where sterilisation of the cerebrospinal fluid was achieved after 54 hours of treatment (not 6-75 hours—that was when the cerebrospinal fluid ceftazidime concentration was measured). It seemed a reasonable case to report, and we

see no reason to exclude it. What is difficult to explain in that particular case, and in the case of staphylococcal septicaemia (case 9), is that despite these organisms, on the basis of their minimum inhibitory concentrations being sensitive to ceftazidime, and bacteriocidal concentrations of ceftazidime achieved in cerebrospinal fluid and blood, organisms could still be cultured after 54 and 36 hours respectively. Maybe there was a focus of infection seeding the blood in case 9, but flucloxacillin addition coincided with bacteriological eradication and clinical improvement. Finally, case 11 (also group B streptococcal septicaemia) was diagnosed late on the second day of life, as her symptoms had been ascribed to hyaline membrane disease. She continued to deteriorate throughout the next 15 hours of ceftazidime treatment (two doses), and penicillin was then added, which resulted/coincided in eventual bacterial eradication. Bilateral intraventricular haemorrhages had already occurred, however, and intensive care was stopped on day 11.

de Louvois's article,¹ quoted by Dr Blumenthal, no more shows ceftazidime to be effective against Gram positive organisms than our paper shows it to be ineffective; it merely reviewed theoretical advantages of ceftazidime, with which we would not argue. By no means should ceftazidime be abandoned for use in neonates on the basis of three cases of Gram positive sepsis which fared badly. Much more experience is necessary, but we can only say from our data that it was inappropriate as a *single* first line antibiotic.

References

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Campylobacter enteritis and bloody stools in the neonate

Sir,

Surely a throw away remark as in the Discussion of the paper by Youngs *et al*¹ should be either refereed or edited out. I refer to 'in our experience anal fissures are not infrequently associated with necrotising enterocolitis and other forms of colitis in the newborn': this in a paper on campylobacter not necrotising enterocolitis or anal fissures. If mentioned it must surely be substantiated—what is their evidence?

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Dr Youngs and co-workers comment:

The statement quoted by Dr McIntosh is based upon observations during an epidemic of necrotising enterocolitis in 1981 and 1982. The presence of inflammation of the distal large bowel in necrotising enterocolitis was shown by