

LETTERS TO THE EDITOR

Practical management of hyperinsulinism in infancy

EDITOR,—We enjoyed the article on practical management of hyperinsulinism by Aynsley-Green *et al.*¹ It re-emphasises the importance of accurate measurement of blood glucose and insists on an accurate laboratory method and not a bedside screening test for diagnosing hypoglycaemia. However, in certain situations, the use of a bedside test is unavoidable—for example, if there will be a long delay before a laboratory result can be obtained, in general practice, home visits, or during transport. In many hospitals, bedside tests are used to identify high risk babies with suspiciously low values who need accurate laboratory measurements of blood glucose.

Most rapid bedside blood glucose measuring devices have been validated in the range above 2.6 mmol/l, using adult blood. We conducted a study to test the accuracy of two commonly used bedside methods of glucose estimation in the clinically important range of 0.5–4 mmol/l, using neonatal cord blood with a packed cell volume over 0.5.

Cord blood samples were allowed to stand for various periods of time to allow the glucose levels to fall in the range of 0.5–4 mmol/l. A total of 103 samples were analysed simultaneously in duplicate by (a) the Cobas hexokinase method in the laboratory, (b) Hemacue, and (c) Precision QID.

With the laboratory hexokinase method as the standard, the sensitivity and specificity of Precision QID for detecting hypoglycaemia (blood glucose less than 2.6 mmol/l) were 86% and 89% respectively; for Hemacue they were 83% and 100%. On average, blood glucose measured by Precision QID was 0.21 (0.32) mmol/l (mean (SD)) higher than when measured by the hexokinase method on paired samples in this low range. Blood glucose measured by Hemacue was on average 0.34 (0.23) mmol/l higher than when measured by the hexokinase method.

Both these bedside methods tend to overestimate the blood glucose slightly in relation to the standard laboratory method. Our study suggests that in situations in which bedside glucose is the only available estimate of blood glucose, a value of over 3.0 mmol/l would be needed for hypoglycaemia to be confidently excluded.

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Rationing of antibiotic use in neonatal units

EDITOR,—We read with interest the article by Isaacs on the rationing of antibiotic use in neonatal units.¹ This encourages the use, where possible, of flucloxacillin and an aminoglycoside as empiric treatment of late onset sepsis.

While this represents a valid approach to the empiric treatment of late onset infection, the epidemiology of bacterial sepsis will vary from unit to unit. In our unit we use a combination of vancomycin and cefotaxime. In 1999, of 159 positive blood cultures, coagulase negative staphylococci were isolated from 124 (78%). All were sensitive to vancomycin, but only 63% were resistant to netilmicin, 89% to cefotaxime, and 91% to flucloxacillin. In most cases, there was a sudden rather than insidious deterioration in the baby with raised C reactive protein, suggesting true infection rather than contamination. This is further supported by the fact that in 94% of cases a coagulase negative staphylococcus was the sole isolate and the patients responded to appropriate treatment.

Although we consider the use of vancomycin to be essential for empiric treatment of late onset sepsis, we are aware of the problems associated with its overuse. The emergence of vancomycin resistant organisms including vancomycin resistant enterococci and vancomycin insensitive *Staphylococcus aureus* is, of course, a concern, but, in spite of continuing surveillance, this has not been observed in our unit. We agree that, to prevent the emergence of resistant gram positive organisms, it is vitally important to stop vancomycin treatment if cultures are negative after 48 hours. Some 96% of blood cultures that grow an organism do so within 48 hours,² and discontinuation after this time is not associated with increased morbidity.³

In conclusion, we would suggest that antibiotic policies remain unit specific, based on the prevalent microorganisms and their known sensitivities.

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Prophylaxis of neonatal vitamin K deficiency bleeding in premature infants

EDITOR,—In March 1998, the Department of Health issued new guidelines for prophylaxis of neonatal vitamin K deficiency bleeding.¹ The recommended oral preparation for term infants was Konakion Mixed Micellular Paediatric (Konakion MM Paed). The recommendations did not, however, include specific guidelines for premature infants.

We undertook a survey of Welsh neonatal units one year after publication of these guidelines. We were interested in the formulation of vitamin K being given to term infants and the regimen being used for premature infants. Just over three quarters of the units (11/14) replied to postal or telephone questionnaires (including the regional referral unit and all five sub-regional units). Of the 10 units that offered oral doses to term infants, only four provided the recommended Konakion MM Paed. Others used injectable Konakion orally (or "Orakay"). Case reports from Germany and Australia show late onset bleeding after three oral doses of injectable vitamin K, which suggests that this preparation does not give adequate protection when given by mouth.²

Ten units responded with information on well preterm infants; nine of these used the intramuscular route (eight Konakion, one Konakion MM Paed). For unwell preterm infants, all 10 respondents used the intramuscular route (nine Konakion, one Konakion MM Paed). The dose of Konakion given to preterm infants varied widely between units (table 1).

Table 1 Intramuscular Konakion administration for premature infants in Welsh neonatal units

Dose	Number of units	
	Well preterm	Unwell preterm
0.2 mg/kg	1	1
0.4 mg/kg*	1	1
0.25–0.5 mg†	1	1
0.5 mg	1	2
1.0 mg	4	4

*Infants less than 2.5 kg.

†Depending on weight.

For extremely premature infants, some units used intravenous vitamin K. However, this route is not effective for long term prophylaxis and the safety of a potentially high peak of serum vitamin K following an intravenous dose has not been assessed.³

Perhaps the reluctance to use the new licensed preparation stems from the lack of long term safety data. There is also a financial disincentive to use Konakion MM Paed rather than older preparations. In order to provide prophylaxis for 1000 term infants (estimating that 20% are exclusively breast fed), at current prices⁴ the comparison is as follows: oral Konakion MM Paed, £3410; Konakion given orally, £322; Konakion given intramuscularly, £230.

Research is urgently needed to ascertain the most appropriate route, preparation, and dosage schedule for premature infants, who are at high risk of vitamin K deficiency bleeding and resultant intracranial haemorrhage.

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