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Dr Moncrieff comments:

I accept that it is possible that the 2 babies with mixed growths might have had infection, and am glad that Dr Guignard agrees that infection must always be confirmed by a suprapubic aspiration. We both think that looking for asymptomatic bacteriuria in healthy preterm babies, even high-risk ones, is not a profitable occupation.

Neutrophil function in infection-prone children

Sir,

The problem of obtaining reasonable controls when working with children is a perennial one. When performing esoteric immunological investigations for which the normal values are unknown, and which are almost certainly age-related, controls are of paramount importance. Recently you published an article describing neutrophil function tests on 24 children with recurrent bacterial infections, mainly those of the upper respiratory tract.[1] The average age of these children was not given, but the mean value of the ages listed was 4·5 years.

The control group consisted of 20 children, mean age 9 years, 8 with allergy to birch-pollen (asymptomatic at the time of sampling) and 12 admitted for operation for non-infectious causes.

It is a pity to nullify what may well be perfectly valid results by the use of so few controls so poorly matched for age.

Reference

[1] Håkansson L, Foucard T, Hällgren R, Venge P. Neutrophil function in infection-prone children. *Arch Dis Child* 1980; **55**: 776–81.

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Dr Håkansson and co-workers comment:

We agree that the number of controls was small and not completely matched for age. The problem associated with the collection of a control group of children is obvious but should be overcome if necessary. However, because we had no indications of any age-dependent variations in any of the variables presented, and because our control groups of children were indistinguishable from adult controls, we did not consider this to be of paramount importance for the conclusions in our paper. Subsequent studies have given further support to our conclusions and, if anything, such studies have shown a tendency towards somewhat higher values in children than in adults for one variable (the phagocytic rate of IgG-coated particle). We are therefore convinced that the results presented in our paper are valid.

Superficial skin necrosis in babies prepared for umbilical arterial catheterisation

Sir,

Mann's report[1] of gluteal skin necrosis after umbilical arterial catheterisation incriminates the catheter, but we have some evidence that the catheter may not be the culprit. Between July 1976 and May 1977 we saw 8 cases in whom findings were almost identical with the 3 he described. All the babies were of very low birthweights (mean 1010 g ± 205 SD) and gestation (mean 28 weeks ± 1·7 SD). Discoloration of the buttocks and lower back was noticed within the first 4 hours and the catheter was removed immediately from 6 babies. In the case of a baby who died, necropsy showed that the lesion was confined to the skin with haemorrhage in the dermis and superficial subcutaneous tissue. The vessels showed no evidence of thromboembolism and the underlying muscle and deep fat appeared normal. It was decided that the catheter was essential for clinical management of 2 babies and the damaged skin subsequently healed. After this 'epidemic' we carried out a postal survey of 16 neonatal intensive care nurseries in the UK. Nine centres reported a total of 14 similar cases in babies of low birthweights. Catheterisation of the umbilical artery failed in a baby who weighed 800 g, but skin necrosis over the buttocks and lower back had still occurred.

To study the sequence of events we introduced a policy of inspecting the back and buttocks *before* catheterisation to identify skin damage from other causes—such as breech delivery. The area was re-examined immediately after the procedure and it soon became apparent that many babies had been lying in a pool of chlorhexidine (0·5% in spirit) and povidone-iodine used to clean the umbilicus. We suspected that these solutions might be causing skin damage and so we used them sparingly and changed the sheet immediately after catheterisation.

Since January 1978 280 babies have been prepared for umbilical arterial catheterisation. In 14 (5%) some degree of skin damage was present before the procedure. Catheterisation was successful in 249 (89%) babies. We have used end-hole Argyle (ALOE Medical Co. St Louis,

MO, USA) or Oximetrix (Mountain View, CA, USA) catheters which are perfused with heparinised dextrose (1 IU per ml/min) and arterial blood pressure is monitored continuously. As we have seen no further cases of buttock skin damage in our nursery we feel confident in attributing this problem to the excessive use of disinfecting fluids which can be toxic to the thin skin of the very preterm infant. The sparing of the skin of the lumbar area, which is also supplied by the superior gluteal artery (so clearly present in the figure shown by Mann[1]), suggests the same cause in Nottingham.

We thank all the paediatricians who took part in our survey and would be interested to hear if they, or others, have had further experience with this complication.

Reference

[1] Mann N P. Gluteal skin necrosis after umbilical artery catheterisation. *Arch Dis Child* 1980; **55**: 815–7.

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Dr Mann comments:

If disinfecting fluids were the culprits, sites of maximum impact would be expected at pressure points and not the soft tissue of the buttock.

Xanthines and necrotising enterocolitis

Sir,

Robinson *et al.*[1] described 3 preterm infants who developed necrotising enterocolitis (NEC) after oral theophylline, and discussed the possible role of reduced gastrointestinal motility. They pointed out that the infants had been sick and were at high risk of developing NEC. The well recognised clustering of cases both geographically and temporally[2] makes it difficult to judge the significance of case reports. Unfortunately, Robinson *et al.*[1] gave no indication of how often xanthines were used in their unit nor did they mention the prevalence of NEC in their nursery at that time.

Theophylline was introduced for the treatment of apnoea at Hammersmith Hospital in August 1977. The discharge summaries of all infants of 32 weeks' gestation, or less, admitted to the neonatal intensive care unit between August 1977 and January 1980 have been reviewed and cases of NEC extracted. Each case showed intramural air on abdominal *x*-ray films or the typical appearance of the bowel at laparotomy or necropsy. The incidence of NEC in infants receiving (or not receiving) xanthines is shown in the Table. The only infant admitted after developing NEC is excluded. Obviously the groups were neither randomised nor matched but these figures do not suggest a strong link between treatment with xanthines (whether oral or intravenous) and NEC (Fisher's exact test $P>0\cdot1$ for either route). Only a randomised controlled trial could finally answer this question.

Table *Incidence of NEC and previous treatment with xanthines in 285 infants of 32 weeks' gestation or less admitted between August 1977 and January 1980*

	No NEC	*Proved NEC*
Oral theophylline (n=21)	18	3
Intravenous theophylline only (n=27)	26	1
No xanthines (n=237)	225	12

References

[1] Robinson M J, Clayden G S, Smith M F. Letter: Xanthines and necrotising enterocolitis. *Arch Dis Child* 1980; **55**: 494–5.

[2] Book L S, Overall J C, Jr, Herbst J J, Britt M R, Epstein B, Jung A L. Interruption of necrotising enterocolitis clustering by infection-control measures. *N Engl J Med* 1977; **297**: 984–6.

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Dr J A Kuzemko of Peterborough comments:

During the period 1970–80 236 neonates received theophylline or aminophylline for recurrent apnoeic attacks; 3 developed NEC and 1 died. Another 6 infants treated differently during that period also developed NEC and 2 died. An association between NEC and xanthines is unlikely.

Prostaglandin synthetase inhibitor in congenital chloride diarrhoea

Sir,

Minford and Barr[1] reported successful treatment of hyperaldosteronism in congenital chloride diarrhoea with a prostaglandin synthetase inhibitor.

We also have treated a 12-month-old girl with congenital chloride diarrhoea, during a diarrhoeal crisis, with the same prostaglandin synthetase inhibitor (ketoprofen). The initial dose was 10 increasing to 20 mg a day after 1 week. The treatment was given for 8 weeks. Before treatment she presented with metabolic alkalosis, hypochloraemia, hypokalaemia, hyperkaliuria, plasma renin activity of 352 ng/ml per hour, and urinary aldosterone of 1900 ng/ml per hour. With this treatment the serum potassium became normal and the kaliuria diminished. At the end of treatment the plasma renin activity had fallen to 1·9 ng/ml per hour, and the urinary aldosterone to 625 ng/ml per hour. The patient has remained free of crisis for 3 months.

We agree that prostaglandin synthetase inhibitor is a promising treatment for the hyperaldosteronism which occurs in congenital chloride diarrhoea.

Reference

[1] Minford A M B, Barr D G D. Prostaglandin synthetase inhibitor in an infant with congenital chloride diarrhoea. *Arch Dis Child* 1980; **55**: 70–2.

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