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 Mann¹), 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.

ANDREW R WILKINSON, J DAVID BAUM,
AND JEAN W KEELING
Department of Paediatrics and
Department of Paediatric Pathology,
John Radcliffe Maternity Hospital,
Headington, Oxford OX3 9DU

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 temporally² 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 $P > 0.1$ for either route). Only a randomised controlled trial could finally answer this question.

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.

ROSAMOND A K JONES
Norfolk and Norwich Hospital,
St Stephen's Road,
Norwich, Norfolk

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 Barr¹ 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.

M CASTRO-GAGO, P PAVÓN,
R RODRIGO, AND A DIAZ
Departamento de Pediatría,
Hospital General de Galicia,
Santiago de Compostela,
Spain

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