

Although the lesions did not show a vascular distribution, they did seem to be related to the level of the arterial catheter, which was above the diaphragm in both infants.

The disinfectant solution used by us was 0.5% chlorhexidine in 70% spirit. This same preparation is used in our neonatal unit for all procedures—such as lumbar punctures, insertion of silastic catheters in preterm and term babies but in none has there been evidence of skin necrosis. A high blood alcohol level as found by Harpin and Rutter¹ is not an adequate sole explanation for these lesions. It, therefore, seems likely that both vascular² as well as local factors³ may be involved.

References

- 1 Harpin V, Rutter N. Percutaneous alcohol absorption and skin necrosis in a preterm infant. *Arch Dis Child* 1982; **57**: 477–9.
- 2 Mann NP. Gluteal skin necrosis after umbilical artery catheterisation. *Arch Dis Child* 1980; **55**: 815–7.
- 3 Wilkinson AR, Baum JD, Keeling JW. Letter: Superficial skin necrosis in babies prepared for umbilical arterial catheterisation. *Arch Dis Child* 1981; **56**: 237–8.

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Dr Rutter comments:

I am sure that skin necrosis associated with umbilical artery catheterisation is unrelated to the catheter itself; indeed, it has been described with attempted but failed catheterisation.³ Dr Al-Jawad's 0.5% chlorhexidine in 70% spirit is the culprit. It evaporates from exposed skin during most practical procedures but during umbilical artery catheterisation it tracks down the baby's abdomen and soaks into the underlying sheet. The back and buttocks are thus in contact with alcohol which cannot evaporate and therefore damages the immature skin. By contrast, the umbilical region where the solution was originally applied is unaffected because the alcohol evaporates quickly.

If Dr Al-Jawad and his staff abandon the use of chlorhexidine in spirit and change to the equally effective aqueous solution, I think they will see no further cases of haemorrhagic skin necrosis in the extremely preterm

infant. This has been our experience and, anecdotally, that of several other neonatal units in the UK.

Rickets in low birthweight infants

Sir,

The article by McIntosh *et al.*¹ described a high incidence of rickets in very low birthweight infants despite high dose ergocalciferol supplementation. Reference was made to our recommendation that plasma alkaline phosphatase activity could be used as a screening test for rickets in preterm infants.² It would be incorrect to suggest that the PAP activity in their patients was lower than that reported by us, or that our recommended levels for screening were too high. Plasma enzyme activities can be interpreted only in relation to the corresponding reference range for the method. In our paper we stated that a value of up to 5 times the upper limit of the adult reference range may be normal, while that at 6 times this value an x-ray film should be performed to exclude rickets. Although McIntosh *et al.* do not quote a range, the upper adult limit for their method approximates 110 U/l at 37°C and therefore all the patients with rickets in their report had values greater than 5 times this limit.

We suggest that plasma alkaline phosphatase activity should be expressed as a multiple, or ratio, of an easily established and verified value such as the upper limit of the adult reference range for the laboratory concerned; this would then allow for methodological and between-laboratory variation and enable the experiences of different centres to be compared.

References

- 1 McIntosh N, Livesey A, Brooke OG. Plasma 25-hydroxy-vitamin D and rickets in infants of extremely low birthweight. *Arch Dis Child* 1982; **57**: 848–50.
- 2 Kovar I, Mayne P, Barltrop D. Plasma alkaline phosphatase activity: a screening test for rickets in preterm neonates. *Lancet* 1982; **i**: 308–10.

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