Table Proportion of infants abnormal at follow up

Degree of periventricular haemorrhage	Subependymal haemorrhage	Intraventricular haemorrhage (No VPS)	Parenchymal extension (No VPS)	All	Subependymal/intraventricular haemorrhage only
Ventricular enlargement	1/3	4/9 (1)	14/16 (7)	19/28*	5/12†
No ventricular enlargement	3/28	3/10	1/1	7/39*	6/38†

* $\chi^2 = 15$, $P < 0.001$; † $\chi^2 = 2.2$, $P > 0.1$. VPS = ventriculo-peritoneal shunt.

Abnormal outcome is strongly associated with ventricular enlargement ($P < 0.001$). However, ventricular enlargement is also strongly associated with the degree of haemorrhage, in particular the presence of parenchymal extension of a bleed. Twelve of 50 babies with subependymal haemorrhage or simple intraventricular haemorrhage, and 16 of 17 with parenchymal extension of intraventricular haemorrhage (PAR H) had ventricular enlargement. Only 2 babies with PAR H appeared normal at follow up. It seems that the association of ventricular enlargement with poor outcome in this study is largely due to the bias introduced by the poor outcome of babies with large haemorrhages. If infants with subependymal or intraventricular haemorrhage alone are considered the relation between ventricular enlargement and outcome is no longer statistically significant. A similar bias is likely to have occurred in the study of Palmer *et al.* in that 12 of 14 babies without ventricular enlargement had small bleeds, while only 1 of 10 babies in the ventricular enlargement group had small bleeds ($P < 0.005$). Until larger numbers are available it will not be possible to allow for the effect of bleed size in analysis of the effect of ventricular enlargement on subsequent outcome. It is also not possible to conclude that ventricular enlargement has a greater effect than bleed size on subsequent development. This has implications for the rationale of conducting a trial of early intervention by cerebrospinal fluid drainage in posthaemorrhagic ventricular enlargement.

Reference

- Palmer P, Dubowitz LMS, Levene MI, Dubowitz V. Development and neurological progress of preterm infants with intraventricular haemorrhage and ventricular dilation. *Arch Dis Child* 1982; **57**: 748–53.

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Plasma terbutaline levels in asthma

Sir,

The reported safety of oral terbutaline in a dose of 250 $\mu\text{g/kg}$ in children prompts me to comment.¹ As a regular prescriber of this drug I have found a marked individual variation in response. Doses as high as 140 $\mu\text{g/kg}$ have caused tremor severe enough to render writing impossible and recently a dose of 330 $\mu\text{g/kg}$ (7.5 mg) has been reported to cause seizures.^{2 3}

It should be noted by your readers that Dinwiddie *et al.*

studied only 5 patients.¹ In a larger and more comprehensive study it has been shown that doses in excess of 100 $\mu\text{g/kg}$ produce side effects with no improvement in pulmonary function.²

References

- Dinwiddie R, Gewitz M, Van Der Laag H, Frame MH. Plasma terbutaline levels in asthma. *Arch Dis Child* 1983; **58**: 223–4.
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- Friedman R, Zitelli B, Jardine D, Fireman P. Seizures in a patient receiving terbutaline (letter). *Am J Dis Child* 1982; **136**: 1091–2.

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Dr Dinwiddie comments:

Our clinical experience of oral terbutaline, often given in the higher dose in children, is at variance with Dr Blumenthal's. We have found that children are generally more tolerant of this drug and that higher proportionate doses are often required to produce a therapeutic effect. It was this experience that prompted us to undertake the present study.

We agree that there is individual variation with this as with any drug and the dose must always be tailored to the patient and his response to treatment. Our small study does, however, show that children may be given doses of up to 250 $\mu\text{g/kg}$ with good response and usually no untoward side effects. We should point out, however, that the maximum dose of 5 mg as stated in our paper should not be exceeded.

Intracranial calcification and childhood medulloblastoma

Sir,

We read with interest the paper by Pearson *et al.*¹ on intracranial calcification in survivors of childhood medulloblastoma. They observed intracranial calcification in 3 of 5 survivors of medulloblastoma who had undergone surgery and radiotherapy but had not had chemotherapy.

We would like to report a boy, now aged 16 years, who developed intracranial calcification following cranial irradiation and partial surgical removal of a cerebellar