PHH) but it would be an oversimplification to assign the differences in outcome to these facts.

Although treating hydrocephalus is all that can be done, it is certainly not all that is wrong in infants with PHH. Lipscomb et al chose to be optimistic about "early" intervention on the basis of good developmental quotients. However, hydrocephalus as an isolated problem is unlikely to produce problems with intelligence.[4] Animal studies also support the notion that the cortical layer is spared in hydrocephalus. Thus there is no theoretical reason to suppose "early" treatment of hydrocephalus is the reason for the difference between previous studies of PHH and that of Lipscomb et al. It is more likely that their series did not include infants with significant hypoxic-ischaemic insults. Our series did: two infants with PHH are microcephalic following shunting[5]—both had intraparenchymal haemorrhage affecting the white matter of one hemisphere, though we had no suspicion of cortical insults until follow-up.

How the extent of hypoxic-ischaemic insult should be assessed in premature babies with PVH remains unanswered. As Volpe et al[5] have shown by positron emission tomography, hypoxic-ischaemic damage may be more extensive than the size of the haemorrhage suggests. This fact must be considered in assessing outcome data in infants with PVH.

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DEXAMETHASONE FOR BRONCHOPULMONARY DYSPLASIA

SIR,—Like Dr Mammel and colleagues (June 18, p 1356) we have used dexamethasone to treat apparently end-stage bronchopulmonary dysplasia, though in an uncontrolled manner, in six infants over the past two years. Our criteria for treatment were almost identical to those of Mammel et al and also included hypercapnia ($PaCO_2$ >60 mm Hg) despite peak inspiratory pressures greater than 30 cm water and intermittent mandatory ventilation greater than 30 breaths/minute. We used a higher dose (2·0 mg/kg daily) but a shorter duration of therapy (four doses, given every 12 h over 2 days). All our patients were on parenteral antibiotics at the time of steroid administration.

Our results confirm the experience of Mammel et al with respect to acute improvement in respiratory status. Five of the six infants had prompt increases in PaO_2, and reduction in $PaCO_2$ and ventilator support requirements (peak inspiratory pressure and rate). Improvements were generally noted about 12 h after the first dose.

There was a concomitant occurrence of brisk diuresis in the five patients who responded to the dexamethasone. These infants had also been treated with diuretics and bronchodilators before the steroid, but these agents had not produced a similar effect. As Mammel et al suggest, two mechanisms of action may be reduction of pulmonary oedema and stabilisation of cell membranes.

The absence of subsequent sepsis in our six patients may be a reflection of antimicrobial therapy, shorter duration of treatment, or other factors.

The encouraging short-term benefits should lead to a more extensive controlled trial to evaluate the safety and efficacy of the drug in these patients, who are at high risk for morbidity and death.

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CAN NIFEDIPINE PROVOKE MENORRHAGIA?

SIR,—A 46-year-old woman presented with atypical chest pain. Investigations, including coronary angiography, were negative. Nifedipine 10 mg three times daily was prescribed and previous drugs (thyroxine 0·2 mg daily, bendrofluazide 5 mg daily) were continued. The nifedipine was withdrawn after 3 weeks because it produced tremor and generalised flushing. The patient's menstrual cycle had previously been regular (5/28 days), but within a week of starting nifedipine she had heavy vaginal bleeding which lasted for 5 weeks. Thereafter her menstrual cycle returned to normal and remained normal for 4 months. Nifedipine 10 mg three times daily was then reintroduced; it was withdrawn after 1 week because it again produced tremors and flushing and because, within a few days, the patient had a recurrence of heavy vaginal bleeding. The bleeding lasted for 4 weeks. Her menstrual cycle has since returned to normal.

A woman aged 44 presented with atypical chest pain. Results of exercise tests were equivocal. She has been treated with nifedipine 10 mg three times daily. Her menstrual cycle was previously regular (3/28 days), but since she started nifedipine 3 months ago her periods have been frequent and heavy (5/20 days). Blood count and coagulation screen are normal. Gynaecological assessment is planned.

While we have no proof, it seems likely that nifedipine provoked the menorrhagia in these patients. The underlying mechanism may have been local vasodilatation rather than disordered coagulation.

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MONOCYTE PROCOAGULANT ACTIVITY IN HYPEREOSINOPHILIC SYNDROME

SIR,—As stated in your June 25 editorial (p 1417) patients with the hypereosinophilic syndrome (HES) are at increased risk of thromboembolic complications and experience the consequences of fibrin deposition, particularly in the myocardial cavity and in large vessels. The pathogenesis of these complications, and of HES itself, remains unknown.

Recent evidence suggests that white blood cells might be involved in intravascular and/or extravascular fibrin deposition by producing procoagulant activities capable of triggering blood coagulation.[1,2]

We have studied the procoagulant activity (PCA) of white blood cells in a 54-year-old man with HES, with heart, lung, skin, and gastrointestinal involvement demonstrable both clinically and by investigation. Polymorphonuclear cells and mononuclear cells were isolated[3,4] and PCA was measured before and after incubation with a standardised stimulus (*Salmonella enteritidis* endotoxin, 1 μg/ml, 4 h at 37°C). The patient's polymorphonuclear cells (containing more than 75% eosinophils) lacked PCA (<0·5 thromboplastin units/10^4 cells) and did not develop any activity after incubation with endotoxin. Among normal white cells, monocytes are the source of PCA,[1] but our patient's monocytes, studied on three separate occasions over 1 month, generated, upon endotoxin stimulation, much more PCA than did monocytes from controls (150–240 versus 35–62 units/10^4 monocytes).

During therapy with vincristine and prednisone monocyte PCA fell; the ratio between the patient's PCA and the PCA of a control tested simultaneously dropped from 4·0 to 1·5. This paralleled a

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