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Spinal disease presenting as acute abdominal pain

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that from contentions intraventricular hemorrhage babies Laminectomy Bristol Frenchay Hospital, myelitis.


We sent a copy of this letter to the authors, who reply below.—Ed, BMJ.

SIR,—Mr Flood and others are correct in what they write, but their discussion of spinal infection covers only part of the clinical problem. They have assumed that "spinal infection" means osteomyelitis of the spine, whereas the commonest infective cause of paraplegia is extradural spinal abscess, as in the presentation of such patients there are usually no changes on the plain radiographs, no osteomyelitis, and the abscess is diffusely distributed through the epidural space. Laminectomy and drainage are the correct procedure in such cases, except, rarely, when it is secondary to osteomyelitis.

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Protective effect of vitamin E against intraventricular hemorrhage in premature babies

SIR,—Dr Malcolm Chiswick and others (9 July, p 81) have added another substance to the rapidly growing list of agents reported to reduce the incidence of periventricular hemorrhage.1-3 The data they present may be challenged, as must the relevance of their findings. The hypothesis they put forward hinges on vitamin E reducing the risk of extension of subependymal hemorrhage into the ventricles, and they claim to have shown that it does so. The assumption is made that ultrasonically distinguished pure subependymal hemorrhage from subependymal hemorrhage that has ruptured into the lateral ventricles, a contention I believe to be unjustified. Haemorrhage within the subependymal layer and thrombus within the ventricles are certainly echogenic, but there is no reliable evidence that liquid (unclotted) blood within the ventricles produces echoes; consequently these two types of haemorrhage cannot be separated reliably. A clue to liquid blood in the ventricles during the acute phase of peri-

SIR.—Dr Malcolm Chiswick and others seem surprised that the dosage of vitamin E may be associated with impaired primary haemostasis for two reasons. Firstly, it has been shown to reduce the ability of normal adult platelets to aggregate.4 It was estimated that at plasma concentrations of about 70 μmol/l (3 mg/100 ml) in practice the highest concentration achievable by oral supplementation and yet lower than the peak concentrations found in some of the parenterally treated babies, collagen induced aggregation would be reduced by up to half. Secondly, it has been observed that neonatal plasma has a reduced capacity to generate prostacyclin (PGI₂) in vitro, and that exogenous vitamin E, albeit at a concentration five times that found in the plasma of the Manchester babies, would restore PGI₂ generation to normal adult concentrations.5 In a healthy adult volunteer we have recently shown that after three days of ingesting dl-α-tocopherol acetate, 20 mg/kg body weight per day, the urinary excretion of 6-keto PGI₂, a stable degradation product of PGI₂, was increased from a mean of 962 (SE 4.42) fmol/mmol creatinine to 1695 (SE 26.3) fmol/mmol creatinine (p <0.02). Although confirmation is required in other subjects, this result suggests that vitamin E at these pharmacological dosages may promote PGI₂ production in vivo.

In the light of this it would be premature to assume that the very wide safety margin of vitamin E administration in other subjects may justify its use in the premature babies. Therefore, important to document the occurrence of theoretical complications such as patent ductus arteriosus or pathological bleeding in babies thus treated. We would be interested to know if such problems were encountered in the Manchester study.

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* We sent a copy of these letters to the authors, who reply below.—Ed, BMJ.

SIR.—We agree with Dr Levene that the absence of echoes from within the ventricles does not exclude the presence therein of liquid blood. Although ventricular asymmetry may be observed in infants undergoing intraventricular haemorrhage we do not feel it is specific for that condition. Our criteria for the diagnosis of subependymal and intraventricular haemorrhage are similar to those of other investigators who use the designation of "grade 1" to denote subependymal hemorrhage and grades 2-4 for intraventricular hemorrhage manifest as echogenicity within the ventricles. We chose for simplicity to lump together grades 2, 3, and 4. In the control group four of the nine babies who suffered intraventricular hemorrhage had non-progressive ventricular dilatation (grade 3; n = 2) or extension of the hemorrhage from the ventricles into the brain parenchyma (grade 4; n = 2; both died). In the babies receiving vitamin E supplements one of the three with intraventricular hemorrhage had non-progressive ventricular dilatation (grade 3).

The preliminary results of our study must not be dismissed on the basis of comparisons with other investigations where vitamin E was given to preterm babies to prevent retro-

ventricular fibrosis and where the mortality or the prevalence of intraventricular haemorrhage was reported. Important variables include the

The authors have not only added another substance to the rapidly growing list of agents reported to reduce the incidence of periventricular hemorrhage but have shown that vitamin E is a very strong antioxidant that may be able to protect the brain from the damaging effects of free radicals.


