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Correspondence

Revised Gairdner-Pearson growth charts

Sir,

Since 1971 when these charts were published,[1] ultrasonography has enabled gestational age to be assessed more reliably. Recent observations by Keen[2] in Sheffield give birthweight for the period 14 to 42 weeks' gestation. Between 32 and 36 weeks these values are significantly lower than those shown in our graphs, which were derived from data collected in Aberdeen between 1948 and 1964.

Other recent observations on birthweight of preterm infants are those of Kitchen from Melbourne[3] and Brooke from London.[4] Both of these cover the range 28 to 30 weeks (but not later): for this two week period their mean values accord well with those of Keen and also with the values shown in our 1971 charts.

Data from length (crown–heel) and head circumference are given by Kitchen for the period 28 to 30 weeks.[3] Both sets of mean values are slightly (about 4%) lower than those shown in our 1971 charts.

The charts have been revised (Castlemead Publications, Swains Mill, 4A Crane Mead, Ware, Herts 3G12 9PY) in the light of the recent studies quoted. Centiles 3, 10, 90, and 97 were derived from SDs when necessary. The most considerable change in the charts affects birthweights of preterm infants between 32 and 36 weeks' gestation.

References

1 Gairdner D, Pearson J. A growth chart for premature and other infants. *Arch Dis Child* 1971;**46**:783–7.

2 Keen DV, Pearse RG. Birthweight between 14 and 42 weeks' gestation. *Arch Dis Child* 1985;**60**:440–6.

3 Kitchen WH, Bajuk B, Lissenden JV, Yu VYH. Intrauterine growth charts from 24 to 29 weeks' gestation. *Aust Paediatr J* 1981;**17**:269–72.

4 Brooke OG, McIntosh N. Birthweights of infants born before 30 weeks' gestation. *Arch Dis Child* 1984;**59**:1189–90.

DOUGLAS GAIRDNER AND JULIE PEARSON
17 Rutherford Road,
Cambridge CB2 2H

Lesions in the neonatal brain

Sir,

Rushton and colleagues are to be congratulated on their correlations between ultrasound scans and macroscopic postmortem pathology.[1] Their suggestion that parenchymal lesions associated with subependymal/intraventricular haemorrhage are of secondary ischaemic nature is in accord with previous studies.[2–4] Rushton *et al* have, however, failed to provide the histological evidence that would be needed to support their contention that the bleeding had occurred in all cases into an area of periventricular leucomalacia.

From our own studies into these lesions, Dr Pape and I suggested that the primary matrix bleed might well obstruct the vein branches traversing the subependymal matrix with consequent venous infarction. Such a sequence would be favoured by an episode of hypotension after the initial bleed. Undoubtedly, some lesions occur, as described by Rushton *et al*, by a process of severe ischaemia due to hypotension followed by reflow and haemorrhage into the ischaemic tissue. This sequence could account for separate but coincident bleeds into subependymal matrix and periventricular tissue.

To claim, as Rushton *et al* do, that all apparent parenchymal extensions of subependymal haemorrhage are due to periventricular leucomalacia seems as simplistic as the claim they incorrectly attribute to Pape and Wigglesworth that all subependymal/intraventricular haemorrhage is due to cerebral hyperperfusion.

The arrows loosed by Rushton and colleagues at current views on pathophysiology of neonatal cerebral lesions might perhaps fly straighter if the missiles themselves were correctly spelt (sagittal or parasagittal from 'sagitta', an arrow: *not* saggital!).

J S WIGGLESWORTH
Royal Postgraduate Medical School,
London W12 0HS

Dr Rushton and co-workers comment:

The omission of histological data was predicated in part by the authors' belief that microscopic examination of these lesions does not provide incontrovertible evidence as to the aetiology of the haemorrhagic lesions in the white matter and in part by limitations of space. Such arguments are perhaps more appropirate in a less clinical context. We do not claim, as Wigglesworth suggests, that all parenchymal extensions of subependymal/intraventricular haemorrhage lesions are due to periventricular leucomalacia but that the apparent ultrasonic 'extension of an intraventricular haemorrhage is more probably the result of haemorrhage into ischaemic periventricular tissue'. Such speculation may well extend beyond currently accepted data, as Levene emphasises in his commentary, but currently accepted data does not in our or your commentator's opinion completely resolve the interrelationship between subependymal/intraventricular haemorrhage and periventricular leucomalacia.

We are fully aware of the pioneering work of Drs Wigglesworth and Pape in the study of hypoxic and ischaemic lesions of the brain. As we were unable to discuss all the factors related to subependymal/intraventricular haemorrhage our apparently over simplistic approach was based on one of the two models presented by Pape and Wigglesworth,[3] one of which 'involves the effects