(Wagenvoort and Planten, 1954; Staubesand, 1955) produced morphological evidence that they were artefacts and that they resulted from invaginations of the arterial wall when tissues are stretched and then cut. This may happen during surgery as well as at necropsy. Moffat (1956) produced these changes, which are identical with those described by Salisbury and Keeling, experimentally.

As for the pulmonary vasculature, in our experience they are found particularly often in the thick-walled pulmonary arteries of newborn infants and of patients with pulmonary hypertension of whatever cause (Wagenvoort *et al.*, 1964), while in normal adult individuals they are usually limited to the elastic pulmonary arteries and do not occur in the thin-walled muscular arteries in which the tendency to invaginate is apparently much less.

References

Bucciante, L. (1945). Sula struttura dei vasi prostatici dell'uomo. *Atti della Società medico-chirurgica di Padova*, **23**, 5–24,

Moffat, D. B. (1956). On the so-called 'polypoid' cushions in arteries. *Acta anatomica*, **26**, 110–120.

Staubesand, J. (1955). Zur Problematik der sogenannten Polsterarterien in der Magenwand. *Bruns' Beiträge zur klinischen Chirurgie*, **190**, 367–374.

Wagenvoort, C. A., and Planten, J. T. (1954). Zijn de 'pedunculated nodules' physiologische vormsels? (abstract). *Nederlandsch tijdschrift voor geneeskunde*, **98**, 2867–2868.

Wagenvoort, C. A., Heath, D., and Edwards, J. E. (1964). *The Pathology of the Pulmonary Vasculature*, pp. 32–33. Thomas: Springfield.

C. A. WAGENVOORT
Department of Pathology,
Wilhelmina Gasthuis,
Amsterdam,
The Netherlands

Dr Keeling comments:

We are aware that vascular 'abnormalities' may be produced by the casual treatment of tissues. The lungs in this case were fixed before blocks were taken for histological examination and we note that Moffat (1956) was unable to produce vascular 'lesions' when tissues were treated in this way; nor were we, on re-examination of semiserial sections of the lungs, able to find evidence of local vessel damage, thus excluding reported causes of artefact.

Reference

Moffat, D. B. (1956). On the so-called 'polypoid' cushions in arteries. *Acta anatomica*, **26**, 110–120.

J. W. KEELING
Department of Pathology,
John Radcliffe Hospital,
Headington,
Oxford OX3 9DU

Weaning very low birthweight infants from mechanical ventilation using intermittent mandatory ventilation and theophylline

Sir,

We would like to support the observations of Dr Barr (*Archives*, 1978, **53**, 598) who found that theophylline therapy facilitated the weaning of infants from mechanical ventilation when apnoea and bradycardia occurred at low IMV rates. We have used this combination therapy in 2 infants with hyaline membrane disease (1620 g, 32 weeks' gestation; 2000 g, 34 weeks' gestation). Theophylline serum concentrations were closely monitored, and all were in the therapeutic range (6 to 11 mg/l). Both infants were severely ill. Each had bilateral pneumothoraces requiring chest tube drainage. Both developed significant apnoea and bradycardia at IMV rates less than 5, necessitating increased ventilatory support primarily in the form of repeated bagging during apnoeic episodes. The infants had dramatic responses to treatment with theophylline, and we were able to extubate both infants within 48 hours.

We too believe that theophylline can aid in weaning infants from ventilators when apnoea and bradycardia occur at low IMV rates. Theophylline can be beneficial not only for low birthweight infants but also for those in whom rapid extubation or a reduction in mean intrathoracic pressure is desirable. Our infants with pneumothoraces are good examples, and others (such as those with pneumopericardium) come readily to mind.

N. VILDAN ERKAN, HELEN ROBINSON,
CRAIG ROOP, AND PETER GAL
Department of Neonatology,
Georgia Baptist Medical Center,
and Department of Clinical Pharmacy,
Mercer University Southern School of Pharmacy,
Atlanta, Georgia 30312, USA

Detection of phenylketonuria

Sir,

Guthrie testing is performed at this hospital on blood samples received from the whole of Scotland, from children aged 6 to 14 days (Stevenson and Kennedy, 1974). Samples are assayed for phenylalanine, tyrosine, methionine, leucine, and galactose, blood samples being collected by hospitals or health visitors as appropriate, and a check on samples received compared with the number of registered births by the district medical officer.

Theoretically, therefore, all children should be included in this national scheme; although in practice the percentage of children sampled varies from 93–99% (Clayton, 1976).

In a recent search for the PKU cards of children born in Glasgow of known birth dates for another purpose, a surprising number of samples had either never been