

astrocytoma. He presented at the age of 11 years with a 2 month history of ataxia and evidence of raised intracranial pressure. A posterior fossa tumour was partially removed and a ventriculo-atrial shunt inserted. Histology showed this to be an astrocytoma. His initial CAT scan showed no calcification. He received a 3 week course of irradiation as follows:

Dose(Gy)	Fractions	Duration(days)	Fields	Source
45	15	21	$\frac{R}{L}$ Lateral 6 MeV	

Five years later computerised axial tomography showed right occipital lobe calcification (Figure). Since operation and radiotherapy there has been a gradual deterioration in mental function and he is now at a school for the educationally subnormal.

This shows that intracranial calcification may occur in children who receive intracranial irradiation at a later age than those described by Pearson¹ and that it is not confined to children who are treated for medulloblastoma. Our patient and the 3 patients of Pearson¹ who developed intracranial calcification have educational problems. It seems more likely that calcification reflects the extent of the radiation damage than that calcification per se is harmful. The practical point is that children who have cranial irradiation need their educational progress monitored in view of the progressive intellectual deterioration that may occur.

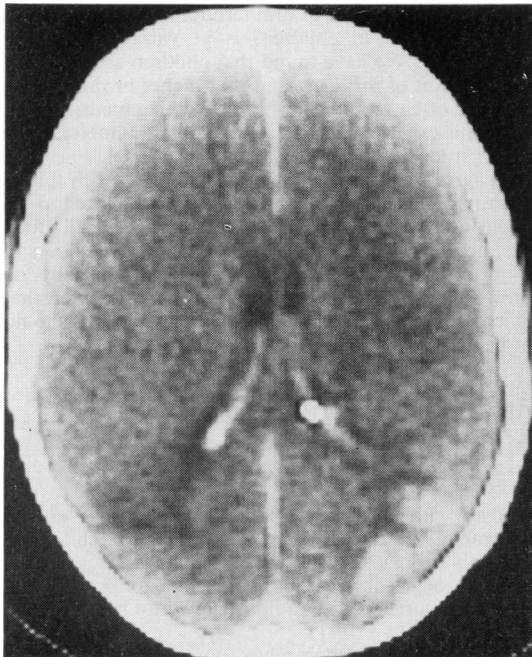


Figure Computed tomography scan showing calcification in right occipital lobe.

Reference

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Reference ranges for plasma creatinine

Sir,

In presenting the reference ranges for plasma creatinine in the newborn, Dr Rudd and his colleagues have made certain assumptions that could be misleading in interpreting the data presented.¹ They have excluded from their study infants who were oliguric, and were therefore considered to be in acute renal failure as a consequence of birth asphyxia. If this was the sole criterion for the diagnosis of acute renal failure they may well have included in their analysis infants with degrees of renal insufficiency who had never become oliguric, and excluded oliguric infants who were not in acute renal failure.²

The authors state that there was an appreciable increase in plasma creatinine values in babies who had required mechanical ventilation for respiratory distress syndrome. This finding agrees with the clearance studies of Guignard *et al.*, who showed a reduced glomerular filtration rate in infants with respiratory distress syndrome.³ Having made this assumption the authors then offer a reference range for plasma creatinine in which they have combined mean values for babies breathing spontaneously and babies requiring mechanical ventilation for respiratory distress syndrome from 7 days of postnatal age without having shown that infants previously ventilated subsequently achieve a normal glomerular filtration rate.

In normal term infants plasma creatinine is known to reach stable values within the first week of life and those for preterm infants towards the end of the first month.^{4,5} From the present study the findings would suggest that stable values for plasma creatinine in term infants are not reached until at least 14 days of age and that values obtained before this are not a reliable index of renal function.

The authors have produced a complicated formula from which a value for plasma creatinine may be calculated given the gestational and postnatal age. The value of the formula is questionable as the authors themselves clearly state that the range of calculated 'normal' values based upon it is wider than that derived from their empirical data.

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³ Guignard JP, Torrado A, Mazouni SM, Gautier E. Renal function in respiratory distress syndrome. *J Pediatr* 1976; **88**: 845-50.

⁴ Feldman H, Guignard JP. Plasma creatinine in the first month of life. *Arch Dis Child* 1982; **57**: 123-6.

⁵ Trompeter RS, Al-Dahaan J, Haycock GB, Chik G, Chantler C. Normal values for plasma creatinine concentrations related to maturity in normal term and preterm infants. *Int J Pediatr Nephrol* 1983 (in press).

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Lung function after acute bronchiolitis

Sir,

It is certainly important to know that after acute bronchiolitis 60% of infants may continue to wheeze. We would, however, like to comment on the remarkable lung function results which are claimed by Henry *et al.*¹ to justify the assertion that an appreciable number of these children remain grossly hyperinflated with increased airways resistance for up to a year.

The plethysmographic measurement of thoracic gas volume (TGV) has been shown to be inaccurate in the presence of airways obstruction, leading to an overestimation of TGV.²⁻⁴ The errors will be greatest when measurements of TGV are made from airway occlusions close to end expiration and when the upper airways are unsupported.^{2,5,6} Moreover, the use of a storage oscilloscope with angle measurement will also lead to overestimation of TGV if looping of the plethysmograph/mouth pressure relation is present—almost always the case in severe airways obstruction⁷—or if it is impossible to correct for any tidal volume included (for example, if the airways occlusion did not precisely coincide with end expiration). Henry *et al.* in a group of infants with probable airways obstruction, report a technique which relied on presumed end expiratory occlusions without upper respiratory tract support, using an oscilloscope recording technique and without correcting for tidal volume included. This may explain their values of TGV of up to 3 times the expected TGV.

We would like to illustrate the scale of the errors in TGV caused by some of these artefacts (Figure). A 6 month old recurrently wheezy 6.7 kg infant was studied while sedated with 600 mg chloral and lying supine in a whole body plethysmograph. Measurements of TGV were made at several lung volumes within the tidal range, the analysis being performed from chart recordings.⁸ The modest and consistent hyperinflation found with end inspiratory occlusions contrasts remarkably with the erroneous values near end expiration. The latter are close to the values of Henry *et al.*

Finally we would like to comment on the minimally raised values of airways resistance (R_{aw}) which were found by Henry *et al.* in their patients shortly after recovery from bronchiolitis, and the normal values found 12 months later.⁸ It is difficult to reconcile this with the assertion that airways disease persisted. Since many of

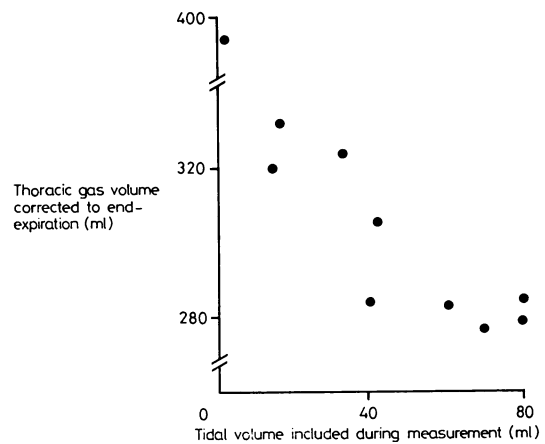


Figure Measurements of thoracic gas volume in a wheezy infant.

the infants were still symptomatic the problem may be technical. The ratio of plethysmograph/mask pressure change during TGV measurement is required to calculate airways resistance. An error in the ratio which leads to an overestimation of TGV will lead to an underestimation of airways resistance. (It is incorrect to claim that the reference values for R_{aw} ⁸ are inappropriate; in the normal reference population, flow at two thirds maximum inspiratory flow rate is laminar). In the patient illustrated here (Figure) the airways resistance for an end expiratory measurement of TGV would have been 34 $\text{cmH}_2\text{O/l/s}$ (only double the expected value for the infant's size, a change) whereas the accurate value derived from an end inspiratory occlusion was 54 $\text{cmH}_2\text{O/l/s}$.

While recognising the vast effort which was required in following up 93 children and measuring their lung function we would like to point out the inaccuracies of the lung function results. Great care is required in the measurement and interpretation of lung function measurements in infants with lung disease before the results may be used to determine prognosis.

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