Table 2 Length of illness (days). Values, median (range)

	Control group (n=46)	Physiotherapy group (n=44)	Mann-Whitney U test
Before admission	3 (1-14)	3 (1-10)	ns
Hospital stay	4 (1-15)	4 (2-11)	ns
After discharge	4 (0-14)	4 (0-15)	ns
Total length of illness	14 (4-27)	13 (7-26)	ns

selected set of patients, in whom chest physiotherapy might be thought to be specifically indicated, there were no differences in daily scores or length of illness.

No child required immediate cessation of physiotherapy due to acute deterioration during a treatment session, although many children were noted to become more distressed during and immediately after treatment, albeit only temporarily.

Discussion

Objective evaluation of chest physiotherapy is difficult, which may largely explain why there is so little published information available. Mellins,⁵ in a review of pulmonary physiotherapy in children, describes data from an unpublished study by D C Shannon showing apparent improvement in ventilation and perfusion after physiotherapy in a 5 month old infant with bronchiolitis.

Using simple clinical criteria, however, we have been unable to show any positive benefit from chest physiotherapy on the natural course of acute viral

bronchiolitis. We did not attempt to make any assessment of changes in lung function that might have occurred during each treatment episode as we were more concerned to elicit any possible overall effect upon the child and his illness. Although no child was made so ill as to necessitate immediate cessation of physiotherapy during treatment, it is generally felt, and in our view correctly, that any form of handling during the acute illness upsets these babies, and that they seem to be least distressed if left undisturbed as far as is practicable. Chest physiotherapy requires considerable handling, and as we have been unable to show any benefit from its use, we recommend that physiotherapy should not be used routinely in the management of acute bronchiolitis.

We thank the children and their parents for their cooperation. We also thank Dr E J Hiller and Dr D A Curnock for their helpful advice and for allowing us to study patients under their care.

References

- ¹ Martin AJ, Gardner PS, McQuillin J. Epidemiology of respiratory viral infection among paediatric inpatients over a six-year period in north-east England. *Lancet* 1978;ii:1035-8.
- ² Reynolds EOR, Cook CD. The treatment of bronchiolitis. *J Pediatr* 1963;63:1205-7.
- ³ Milner AD, Hull D. *Hospital paediatrics*. Edinburgh: Churchill Livingstone, 1984:88.
- ⁴ Henry R, Milner AD, Stokes GM. Bronchiolitis (letter). *Am J Dis Child* 1983;137:805.
- ⁵ Mellins RB. Pulmonary physiotherapy in the pediatric age group. *Am Rev Respir Dis* 1974;110(Suppl):137-42.

Correspondence to Dr M S C Webb, Children's Respiratory Unit, Queen's Medical Centre, Nottingham NG7 2UH.

Received 22 July 1985

White matter attenuation and megalencephaly

R O ROBINSON

Newcomen Centre, Guy's Hospital, St Thomas Street, London

SUMMARY The computed tomogram of a 6 month old girl with familial megalencephaly showed widespread attenuation throughout the white matter. She continued to be developmentally and neurologically normal. Her scan at age 3 years was normal apart from the megalencephaly. A tentative explanation for this unusual series of events is offered.

Generalised low attenuation of the white matter is

usually accompanied by an abnormal clinical picture. A child with megalencephaly who was otherwise neurologically and developmentally normal, and in whom this picture gave rise to a period of diagnostic uncertainty is reported. A tentative explanation is suggested.

Case report

This girl, who is now aged 3½ years, was first seen at 6 months for evaluation of macrocephaly, her head

Fig. 1 Computed tomogram of the patient at age 6 months.

Fig. 2 Computed tomogram of the patient at age 3 years.

circumference having risen from the 25th centile at birth to 3 SDs above the mean. She was otherwise well. There was no evidence of raised intracranial pressure, and apart from a little motor delay in specifics related to the mechanical effect of her large head, she was developmentally and neurologically normal. It was noted that the head circumferences of both her parents lay on the 90th centile. Computed tomogram showed widespread attenuation throughout the white matter (Fig. 1). Enzyme assays excluded metachromatic and Krabbe's leukodystrophies, and Gm₁ and Gm₂ gangliosidoses. Her electroencephalogram was normal as were her visual evoked responses. A provisional diagnosis of Alexander's disease in the presymptomatic stage was made, but fortunately not communicated (with its implications) to her parents. Thereafter her head circumference grew normally at 3 SDs above the mean and her development continued at an age appropriate rate. At age 3 years she had a second computed tomogram which was normal (Fig. 2).

Discussion

Low attenuation of the white matter is a characteristic finding in the childhood leukodystrophies. The

cause of this appearance is conjectural. It may be due to the presence of myelin breakdown products or white matter oedema, as in the more rapidly progressive leukodystrophies. This latter suggestion seems particularly relevant in the spongiform leukodystrophies described by Alexander and van Bogaert and Bertrand in 1949.^{1 2} Absence of myelin alone will not suffice. The white matter appearances in Pelizaeus-Merzbacher's disease, the most chronic of these conditions, may be normal on computed tomogram. The appearance is also seen in acute leukoencephalitis, a presumed virus associated encephalopathy in which the brunt of the inflammatory process falls on the white matter.

Clearly these explanations do not apply here. There is no suggestion of a progressive disorder: she has experienced no acute neurological illnesses. Severe delay in central nervous system myelination, incompatible in any case with clinical normality, should not by the above reasoning be associated with pronounced changes on computed tomogram: in addition the normal visual evoked response latency argues against this supposition.

She would, however, seem to have familial megalencephaly. In this condition the rate of head growth is abnormally high in the first six months.

This is presumably due in part to acquisition of new myelin which is normally proceeding in the cerebral hemispheres at this time. It is tentatively suggested that in this case where the rate of head growth was extremely rapid, the processes associated with a high level of metabolic activity were sufficient to cause low attenuation on computed tomogram. An analogous situation seems to exist on resumption of a normal diet after a period of either intrauterine or postnatal malnutrition. Here rapid head growth in infancy may be accompanied by the transient occurrence of signs of raised intracranial pressure. In one personally observed case this has not, however, been accompanied by abnormal appearance of the white matter on computed tomogram. At no time had our patient clinical signs of raised intracranial pressure.

Impressive and alarming though the computed

tomograms were, because the child was clinically unaffected, diagnostic uncertainty was expressed and a guarded prognosis was not given to the parents. It is important to recognise that even after 10 years relatively widespread use we are still exploring the clinical implications of computed tomography technology.

References

- ¹ Alexander WS. Progressive fibrinoid degeneration of fibrillary astrocytes associated with mental retardation in hydrocephalic infant. *Brain* 1949;**72**:373-81.
- ² Van Bogaert L, Bertrand I. Sur une idiotie familiale avec dégénérescence spongieuse du névraxe. *Acta Neurol Belg* 1949;**49**:572-87.

Correspondence to Dr R O Robinson, Guy's Hospital, St Thomas Street, London SE1 9RT.

Received 12 August 1985