Letters to the editor

‘Head growth and cranial assessment at neurological examination in infancy’
SIR–Amiel-Tison and colleagues remind us of the importance of measuring head circumference in infancy and its continuing use as a predictor of neurodevelopmental impairment.1 This is especially true in resource-limited settings where other modalities of assessment, such as neuroimaging, are not available and prolonged follow-up schedules are impractical. They note that when severe cerebral atrophy follows hypoxic–ischaemic encephalopathy, head circumference may gradually decrease by 2 standard deviations (SD) over a two or three month period. In the severe cases too frequently encountered in developing countries, this process may actually occur over as short a time as 6 weeks: this is clinically useful.

As part of a study of neonatal encephalopathy (NE) in Kathmandu, Nepal we have previously described a cohort of 57 survivors of NE.2 Occipitofrontal head circumference (OFC) was measured using a Child Growth Foundation Lasso tape at birth, six weeks, and one year. All growth data were converted into SD scores (in the absence of local growth standards) using British 1990 growth reference data. Neurodevelopmental assessment at one year of age showed 18 infants to have major disability (mental developmental index <55 or psychomotor developmental index <55 on Bayley testing). Seventeen (95%) impaired survivors and two of the 39 (5%) unimpaired survivors of NE developed microcephaly (defined as a decline of at least 2SD scores) by 6 weeks of age.3 Both of the unimpaired survivors with microcephaly at six weeks showed symmetrical growth impairment at birth. By 1 year of age, 38 of the 57 survivors, including 21 (54%) of the apparently unimpaired group, had developed microcephaly. Interestingly, a group of healthy infants from the same Nepali population (n = 129) showed a significantly lower rate of head growth (averaging one SD) in the first year of life, compared with the British reference population.

In this setting, acquired microcephaly as early as 6 weeks of age is a highly specific (95%) indicator of impairment at one year, with a higher positive predictive value (78%) than the grade of NE (55%). Acquired microcephaly in an infant with moderate to severe encephalopathy increased the probability of developing major impairment from 55% to 68%.

The simple procedure of comparing head circumference measurements at birth and at 6 weeks of age when combined with systematic neurological evaluation in the newborn period, will allow the clinician working in even the most under-resourced setting to identify correctly more than two thirds of the infants who will go on to develop significant functional disability in early childhood following NE.

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‘Feeding difficulties in children with visual impairment with no other impairments’
SIR–Independent feeding is an important self-care skill that is occasionally delayed in children with severe visual impairment. Decades ago in British Columbia, Canada we began to see children with severe congenital vision loss and feeding difficulties, who otherwise had no other impairments. They drank fluids with ease, did not drool saliva, and their speech was normal. When introduced to solids, they readily took smooth pureed foods, but upon transition to mixed or lumpy consistencies they had difficulties. With solids they gagged and vomited, as a result of an apparent oral hypersensitivity. This pattern became increasingly problematic and difficult to correct. In some cases these children could not make the transition out of the smooth purees, causing nutritional and social difficulties.

There have been many plausible reasons for these difficulties. Severely visually impaired infants tend to demonstrate more tactile defensiveness than sighted children, so when textured foods are introduced, they may select and push lumps from their mouths with their tongues. Mature patterns of mastication develop in infants between the ages of 6 to 8 months.1 When the parents do not persist with new solids at this stage, the infant’s nervous system does not acquire new oral motor skills; they develop oral hypersensitivity and this specific type of eating disorder develops. Therefore, a potential cause of feeding difficulties may be the lack of appropriate feeding experiences during critical periods.1

Difficulties in perception of the process of feeding may also contribute to this difficulty.2 Children with severe impairments may not be able to visualize the feeding methods. As food is brought towards the mouth, the child may not be adequately prepared to accept it. The child may develop some defensiveness, which may lead to caregivers’ frustrations. This in turn could lead to feeding difficulties.

With early intervention services for children with visual impairment, this feeding disorder essentially disappeared in British Columbia and is now only seen when caregivers are

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References
markedly overprotective. Because this condition is so uncommon in children who are visually impaired, feeding specialists are generally unaware of its pathophysiology. When left untreated, years later oral sensory motor management becomes time consuming and difficult. Yet, advising the parents of children with visual impairment, at the appropriate time, could so readily prevent the development of this problem. Some professionals working with children who are visually impaired are aware of this eating disorder, but its descriptions have not appeared in the medical literature.

The prevalence of severe feeding disorders is well recognized in children with neurological causes of visual impairment. It is important that professionals also recognize that feeding difficulties can occur in children who are visually impaired without additional disabilities on the basis of abnormal early feeding experience. With recognition, proper attention to feeding skills for this population can occur and thereby reduce problematic difficulties in the future.

References