Archives of Disease in Childhood, 1989, **64**, 496–502

Personal practice

Follow up studies during the first five years of life: a pervasive assessment of neurological function

C AMIEL-TISON* AND A STEWART†

**Clinique Universitaire Baudelocque, Groupe Hospitalier Cochin, Paris, and †Departments of Paediatrics and Obstetrics, University College and Middlesex School of Medicine, (University College), London*

> . . . indeed, it is not easy to be accurate in an account of anything, however simple. Zoologists often disagree in their descriptions of the curve of a shell, or the plumage of a bird, though they may lay their specimen on the table, and examine it at their leisure; how much greater becomes the likelihood of an error in the description of things which must be in many parts observed from a distance, or under unfavourable circumstances of light and shade; and of which many of the distinctive features have been worn away by time. I believe few people have an idea of the cost of truth in these things; of the expenditure of time necessary to make sure of the simplest facts, and of the strange way in which separate observations will sometimes falsify each other, incapable of reconcilement, owing to some imperceptible inadvertency.[1]

These words were written by Ruskin in 1851, but they express precisely the problem that research workers face today when defining measures of outcome for use in follow up studies. Ruskin's comments have particular relevance to the collection and recording of data concerning the constellation of disorders generally referred to as 'cerebral palsy'.[1]

In the past, cerebral palsy was the commonest neurodevelopmental impairment reported in follow up studies of low birthweight or very preterm infants.[2,3] In particular, spastic diplegia was generally regarded as a typical condition among infants whose neonatal courses were complicated by apnoea and bradycardia. These impairments were believed to be so typical in infants who survived before the introduction of modern neonatal intensive care that it was proposed that their prevalence could be used as a measure of the quality of survival for audit, and comparative and epidemiological studies.[4] As interest widened and large epidemiological studies were planned, the need for a universally agreed, simple definition of cerebral palsy was recognised; agreement proved difficult. Various suggestions were made ranging from a simple description based on Little's original proposal,[5,6] to a complex standardised physiopathological classification of syndromes of cerebral palsy that was put forward by paediatric neurologists.[7] Interobserver agreement was studied and found to be poor according to either type of definition.[8] Longitudinal studies also indicated that the diagnosis of cerebral palsy was not consistent over time in individual children, at least not until they were older, and aged 5 years or more.[9] As a result of these observations warnings were expressed that perhaps cerebral palsy was unsuitable to use as an outcome measure, particularly among young children.[9,10]

While perceptions of the nature of cerebral palsy and its use as a measure of outcome were changing, so also were the circumstances in which the condition occurred. For example, as methods of neonatal care were progressively refined, so the pattern of insults to the newborn brain changed in character and extent. Many potentially damaging events were avoided, or at least ameliorated, although homoeostasis was not guaranteed. Indeed, infants began to survive after insults that had previously been thought to be fatal. As a consequence there was a change, both in the type of infants surviving and the brain lesions that they suffered.[11] For example, in a recent study even using the most liberal definition, cerebral palsy seemed to affect a minority of the neurologically impaired and disabled children.[12] Thus to describe the outcome of a group of infants solely according to the prevalence of cerebral palsy is both inadequate and misleading.

Proposals

We believe the problem could be overcome by

describing the neurological condition of each infant objectively according to clearly defined standards. This approach would eliminate the need to interpret findings to fit them into specific diagnostic categories, and it is likely that it would also overcome the problem of the apparent variation of diagnosis with age in individual children. There is already evidence indicating that it is the manifestations of brain damage that vary according to age, and not the underlying lesions.[13–17] For example, infants with neurological impairments in the first year of life have been reported to have significantly poorer scores in tests of cognitive function than their unaffected peers,[17 18] and they are believed by some to be particularly liable to present with learning difficulties as they grow older.[13 15 16 18 19] Thus these children represent an aspect of perinatal brain damage that is important as an index of outcome, but which may not be recognised as such because the early neurological signs seem transient and the learning difficulties are not identified until the children are much older and the early signs have been forgotten. In fact the transience of the early neurological signs is probably apparent rather than real. It is more likely that the signs are present but are often not detected because of the power of the toddler to defy neurological assessment between the ages of about 2 and 4 years.

Methods

The scheme we propose is designed to record the consequences of brain damage in a purely objective manner. Objective neurological information is obtained from a structured neurological examination using standardised procedures to elicit the signs and to judge the findings. Throughout the first year of life the scheme and standards for neurological examination already described by one of us may be used.[20] The scheme includes detailed instructions for eliciting the signs. The same principle and methods may be applied from the second year of life using age-appropriate standards to judge the findings. Experience has shown that by the end of the first year of life all major impairments can be detected, and any persisting minor impairments must be regarded as significant. Thus categorisation of neurodevelopmental condition at this age may have both meaning and predictive value.[17] We therefore give here standards starting from the age of 9 months (table 1) although standards for use throughout the first year are available elsewhere.[20] A record form for the neurological data that are obtained at each examination is provided (table 2). We recommend that each child should have an individual record form. Findings should be recorded on this form either according to the presence or absence of a sign or as absolute measurements of angles in degrees. The findings may then be judged according to the standards appropriate for age given in table 1 and added to the results of assessments of hearing, vision, and behaviour, measurements of head growth, and of psychometric testing, to give a comprehensive assessment of a child at a particular age. The choice of methods for tests of hearing and vision, and of psychometric assessments, depends on the age of the child and the availability of the tests.

Findings judged to be normal because—for example, the size of an angle or a test score falls within the range appropriate for age—may be coded 0. Findings judged to be mildly deviant on the basis of angle measurements or test scores may be coded 1, and severely deviant findings may be coded 2. This coded information may be recorded on the chart given in table 3 to provide a summary of all the findings in an individual child at a particular age. When making this summary a single mildly deviant item (coded 1) may be ignored. Two or more mildly deviant items (giving a total of 2 or more) indicate abnormality, and severely deviant items (coded 2) always indicate abnormality, even if there is only one item in this category. Finally, on the basis of this coded information, neurodevelopmental condition can be described as 'unimpaired', 'impaired, but without disability' (mildly deviant), or 'impaired with disability' (severely deviant).

Because the type of records that we propose may form the basis of a minimum set of data for an individual child that may be used to monitor progress or to provide information for research, provision has been made on the summary form (table 3) for recording some additional facts, under the heading 'other information'. These include conditions which, although not necessarily developmental in origin, may influence a child's progress; they can also be used to provide a wider definition of abnormality if required. Examples include refractive errors, chronic lung or gastrointestinal disease, and growth.

THEORETICAL BASIS OF THE SCHEME FOR NEUROLOGICAL EXAMINATION

In an earlier report Amiel-Tison described a waxing and waning pattern of neuromotor development from 28 weeks' gestation to the end of the first year of life.[21] This description was based on the clinical evaluation both of posture and passive tone at rest, and of motor activity and postural reactions; it conformed with proposals originally made by both Thomas and Saint-Anne Dargassies,[22] and by Peiper.[23] For example, from 28 to 40 weeks' gestation the acquisition of muscle tone and motor

Table 1 *Standards for coding neurological data*

	9–17 months	*18–60 months*
1 (a) **Neuromotor: hypertonia**		
Arms:		
Scarf		
Anterior axillary line to midline	1	1
Less than anterior axillary line	2	2
Candlestick posture*: present/fixed	2	2
Hand flapping:		
tightening	1	1
nearly absent	2	2
Legs:		
Adductor angle 110°–40°	1	1
<40°	2	2
Heel-ear angle 110°–100°	1	—
100°–90°	—	1
<100°	2	—
<90°	—	2
Popliteal angle 110°–100°	1	1
<100°	2	2
Ankle angle 80°–90°	1	1
>90°	2	2
Ankle stretch response—		
phasic	1	1
tonic	2	2
clonus	2	2
Axis:		
Imbalance of tone		
Excessive extension *v* flexion	1	1
Permanent opisthotonus	2	2
Falls back from sitting— present	2	2
'Scissoring', held up— present	2	2
1 (a) **Neuromotor: hypotonia**		
Arms:		
Scarf:		
Minimal resistance	—	1
Flaccid (no resistance)	2	2
Hand-flapping—flaccid (no resistance)	2	2
Legs:		
Adductor angle:	Impossible to define	Impossible to define
Heel-ear angle:	Impossible to define	—
>150°	—	1
Popliteal angle:	Impossible to define	—
>160°	—	1
Ankle angle:	Impossible to define	Impossible to define
Axis:		
Global hypotonia (excessive flexion and extension):		
'Rag doll'†	2	2
Falls forward, sitting— present	2	2
Supporting/standing response—feeble/nil	2	2
1 (a) **Reflexes**		
Limbs:		
Tendon—very brisk	1	1
clonus	2	2
Primitive—asymmetrical tonic neck reflex present	2	2
1 (a) **Reflexes** (cont.)		
Postural—lateral propping/ parachute		
Nil at 10 months	1	—
Nil at 12 months	2	—
Hesitant	—	1
Absent	—	2
Hands—fisting/cortical thumbs, tendency	1	1
fixed	2	2
1 (a) **Asymmetry**		
Asymmetrical findings (difference of ⩾1 grades or ⩾10° passive tone or both)	1	1
1 (a) **Cranial nerves**		
Abnormal	1	1
1 (b) **Neuromotor**—consequences at examination—record abnormality as 'yes'		
Head control: imperfect or absent		
Sitting: unstable—falls forward or back — or absent		
Standing: unsteady or absent		
Walking: few steps or absent after 18 months		
Gait: 'inelegant' or abnormal		
Use of right upper limb impaired		
Use of left upper limb impaired		
Use of right lower limb impaired		
Use of left lower limb impaired		
2 **Neurosensory**		
Hearing:		
Sensory loss, high tone, present	1	1
Sensory loss speech frequencies, present	2	2
Eyes/vision:		
'Fix-track' behaviour: Poor, brief, one plane only, or absent	2	2
Nystagmus or roving eye movements: present	2	2
3 **Neurobehavioural**		
Seizures: with or without treatment, present	2	2
Hyperexcitability: one or all present	1	1
Lethargy: one or all present	1	1
Involuntary movements: present	2	2
Sucking or swallowing: absent	2	2
Drooling: present	1	1
4 **Head growth**		
Head circumference:		
Two standard deviations above or below the mean	2	2
Growth velocity: <10 or >90 centile	2	2
Ventriculoperitoneal shunt: present	1	1
5 **Psychometric tests—define abnormality according to test instructions—for example:**		
McCarthy scale of children's abilities:		
General Cognitive Index—70–79: Impaired		
General Cognitive Index—<70: Impaired with disability		

*Upper arm in line with shoulder with maximum supination and flexion to 90° at elbow; †very floppy with unlimited extensibility.

Table 2 *Individual record form for neurological data*

Section				*1*	*2*	*3*	*4*	*5*
1 **Passive tone**	Upper limb:	Scarf sign	Value					
		Candlestick*	No/yes					
		Hand flapping	OK/not					
	Lower limb:	Adductor angle	Value					
		Heel-ear angle	Value					
		Popliteal angle	Value					
		Ankle angle	Value					
		Ankle stretch response	Type					
	Axis:	Ventral flexion	OK/not					
		Dorsal extension	No/yes					
		Opisthotonic posture	No/yes					
1 **Active tone**	Head control:		Yes/no					
	Sitting:		Yes/no					
		Falls backwards	No/yes					
		Falls forward	No/yes					
	Standing:		Yes/no					
		'Scissoring'	No/yes					
	Walking:		Yes/no					
	Gait:		OK/not					
	Use of lower limbs:		Yes/no					
1 **Reflexes**	Primitive:	Moro response	No/yes					
		Asymmetrical tonic neck reflex	No/yes					
	Tendon:	Biceps	OK/not					
		Knee	OK/not					
		Ankle	OK/not					
	Clonus:		No/yes					
	Protective:	Propping	Yes/no					
		Parachute	Yes/no					
	Hands (right/left):	Fisting	No/yes					
		Cortical thumbs	No/yes					
	Use of upper limbs and hands:		OK/not					
1 **Asymmetry**	Arms/legs/axis:		No/yes					
1 **Cranial nerves**			OK/not					
2 **Ears**	Pure tone audiogram (SOM/HTHL/SNHL)†:		OK/not					
2 **Eyes**	'Fix'/'track' behaviour:		OK/not					
	Nystagmus/roving movements:		No/yes					
	Visual field:		OK/not					
	Visual acuity (right/left):		Value					
	Squint:		No/yes					
	Sunset:		No/yes					
3 **Seizures** (treated or not)			No/yes					
3 **Hyperexcitability**								
	Tremor/clonic movements:		No/yes					
	Sleep disorders:		No/yes					
	Excessive crying:		No/yes					
3 **Lethargy**	Reduced activity:		No/yes					
	Drowsy:		No/yes					
	Infrequent crying:		No/yes					
3 **Involuntary movements**								
	Upper limbs:		No/yes					
	Lower limbs:		No/yes					
3 **Sucking/swallowing**			OK/not					
3 **Drooling**			No/yes					
4 **Head growth**	Head circumference (cm):		Value					
	Velocity (centile):		Value					
4 **Ventriculoperitoneal shunt**			No/yes					

*Upper arm in line with shoulder with maximum supination and flexion to 90° at elbow; †secretory otitis media, high tone hearing loss, sensory neural hearing loss. Full details of procedures to elicit neurological signs have been previously published.[20]

Table 3 *Record form to summarise neurodevelopmental state at any age within the first five years of life*

Name ..... Date of birth ..... Date of test ..... Gestational age ..... Corrected age .....

1 Neuromotor (tone and reflexes)

(a) Findings:

Axis: Global hypotonia ☐
Imbalance (extensor>flexor) ☐

Limbs:	Right Arm	Right Leg	Left Arm	Left Leg
Hypertonia				
Hypotonia				
Asymmetry			/	/
Exaggerated tendon reflex				
Abnormal hands		/		/
Abnormal postural reflex				

(b) Consequences at examination:

	Yes	No
Head control		
Sitting		
Standing		
Walking		
Normal gait		
Use of right upper limb/hand		
Use of left upper limb/hand		
Use of right lower limb		
Use of left lower limb		

2 Neurosensory

Hearing loss ☐
Nystagmus ☐
Cortical blindness ☐

3 Neurobehavioural

Seizures (treated or not) ☐
Irritability ☐
Depression ☐
No sucking ☐
Drooling ☐
Abnormal cranial nerves ☐

4 Head growth
Occipitofrontal circumference:

<two SD ☐
>two SD ☐
Ventriculo-peritoneal shunt ☐

Velocity
Cm/year ☐
Centile ☐

5 Psychometric tests

Subscales	Value	Standard score
1		
2		
3		
4		
5		
Overall		
Achievement score		

Conclusions

1 Neuromotor	
(a) impairment	☐
(b) function affected	☐
2 Neurosensory	☐
3 Neurobehavioural	☐
4 Head growth	☐
5 Psychometric tests	☐

Normal ☐ **Impaired** ☐ **Impaired with disability** ☐

Other information

1 Severe refractive error or retinopathy, or both	☐
2 Squint	☐
3 Conductive hearing loss	☐
4 Chronic lung disease	☐
5 *Chronic gastrointestinal disease	☐
6 Growth problem	☐
7 Congenital malformation	☐
8 Socioeconomic deprivation	☐
9 Other	☐

Normal: a total of 1 or less in sections 1–4 and scores in section 5 within the normal range for the test used. **Impaired without disability:** a total of 2 or more in section 1 (a) and no functional consequences recorded in 1 (b); and/or a total of 1 in sections 2, 3, and 4, and/or scores in section 5 within the 'doubtful' range. **Impaired with disability:** a total of 2 or more in section 1 (a) with functional consequences recorded in 1 (b), and/or 2 or more in sections 2, 3, and 4, and/or scores in the abnormal range for the test used in section 5, or a combination of these. **Other information** is not scored or used in the conclusions.
*=usually after necrotising enterocolitis.

function spreads from the lower extremities in the direction of the head. After full term the process is reversed so that relaxation and the development of motor control proceed downwards for the next 12 to 18 months. Thus the upper limbs and extremities begin to relax and acquire skills before the lower limbs, and in the axis head control is acquired first, followed by the ability to sit, to stand, and finally—to walk. The timing of this sequence is subject to wide individual variations in addition to familial and ethnic influences, but the order is constant in all normal individuals. Thus from a knowledge of the successive stages of maturation throughout the sequence it is possible to detect abnormal clusters of responses that may be used to identify deviant development. Using these observations a scheme for the evaluation of neurological development during the first year of life was designed.[20] In practice this scheme may be applied up to the age of 18 months. We believe that this is justified because, owing to wide individual variations, the sequence culminating in independent walking is often not completed by the end of the first year of life in infants who later prove to be normal. Independent walking, however, should be achieved by 18 months; failure to do so usually indicates abnormality.[24]

The orderly progression of neuromotor development continues after the first year though the changes are more subtle and result largely from improvements in motor organisation. The advances in motor control that result may be identified and assessed,[25] and passive tone at rest may still be measured. Thus the same type of assessment based on observation and manipulation of the child that is used to evaluate progress in the first year of life may be applied as the child grows older.

In order to derive standards from the second year of life we considered both the original data of Saint-Anne Dargassies[26] (that were obtained by examination of a group of normal infants followed up to the age of 2 years) and the data from our own follow up studies of infants born at full term[13 27] and those born very preterm (Stewart *et al*, unpublished observations).[12 17 28 29] Our own findings were in agreement with the standards for neuromotor function proposed by Saint-Anne Dargassies[26] up to the end of the second year of life. Thereafter we observed little change with age in posture and passive tone at rest, or in motor activity and postural reactions. We therefore consider that standards for these measures remain more or less constant from the end of the second year to the age of 4 or 5 years, and in practice can be applied from the age of 18 months when the early standards cease to apply.

When examining our own data we noted that the range of normal variation was as wide in the older children as had been observed during the first year of life in the earlier studies.[20] To take account of this observation the definitions of abnormality have to be fairly stringent to avoid including minor variations that may be of little consequence.

EXPERIENCE OF THE SCHEME IN USE

We have been using the scheme for neurological examination[20] and teaching it to paediatricians and physiotherapists for many years. Familiarity with examining and handling infants and young children is the only essential requirement for acquiring competence, but a basic knowledge of paediatric neurology helps with interpretation as it permits the examiner to extend the examination if there are abnormal or equivocal findings. We have found that successive generations of research fellows of variable experience have quickly become skilled with good interobserver reliability, a matter that is discussed in more detail elsewhere.[20]

The neurological examination takes about 10 minutes to carry out. Though it has been designed to be objective as far as possible, there are a few circumstances in which particular caution must be exercised when interpreting the findings.

Wide individual variation in passive tone makes the definition of mild symmetrical abnormalities difficult; as a consequence these conditions are probably underestimated. By contrast, asymmetry is usually accurately recognised though it does not necessarily indicate that one side is normal.

Struggling with defiant, uncooperative children may affect the precision of the examination. This tends to be a problem when examining 2–3 year olds who resent procedures designed to elicit passive tone, such as the 'scarf' sign, and occasionally their behaviour prevents completion of the examination. When struggling does occur, results will always be biased in the direction of hypertonicity.

Postural deformities secondary to prolonged immobilisation during intensive care may affect some measurements. For example, shortening of upper limb girdle muscles or of the extensor group of neck muscles may occur during prolonged periods of mechanical ventilation. This may mimic increased passive tone and lead to difficulties in interpretation.[21] Likewise, 'scissoring' within the first year may have similar postural origins. In this circumstance the sign does not necessarily support a diagnosis of spastic diplegia.[30] In general when such findings are isolated ones they are more likely to be postural than central.

Conclusions and implications

In clinical practice, follow up teams may use this

scheme to help them offer continuous care to infants and their parents from the time the infants go home. If the findings are normal it will be possible to reassure many parents at an early stage,[29] and infants with impairments may be recognised so that treatment can be started and help provided for the parents as soon as possible. Infants with movement disorders or cerebral palsy will be included among those with neurological impairments and treated as soon as they are recognised, though they may not be labelled precisely until years later.

In research the scheme provides objective data that can be used in longitudinal studies and analysed in detail, item by item. In epidemiological studies these data permit categorisation of neurodevelopmental condition without the need to use any physiopathological nosology. Thus it should be possible to trace the influence of perinatal events right through infancy and the preschool period to the age when the diagnosis of learning difficulties and other school problems is possible.

Difficulties are inherent in the examination of the preschool child; they cannot be ignored or bypassed. From our own experience, however, we believe that an analytical approach to the assessment of neurological function is feasible, with interobserver reliability and consistency over time in an individual child. We also believe that this approach can help to overcome many of the problems of recording and reporting in follow up studies, so aptly described by Ruskin's elegant prose.[1]

Dr Amiel-Tison is supported by the Institut de la Sante et Recherche Medical and Dr Stewart by the Medical Research Council.

References

1 Ruskin J. *The stones of Venice.* Boston: Dana Estes, 1851: foreword.

2 Stanley F, Alberman E. Birthweight, gestational age and the cerebral palsies. In: Stanley F, Alberman E, eds. *The epidemiology of the cerebral palsy.* Clinics in developmental medicine No 87. London: Spastics International Medical Publications, 1984:57–68.

3 Ingram TTS. A historical review of the definition and classification of the Cerebral Palsies. In: Stanley F, Alberman E, eds. *The epidemiology of the cerebral palsy.* Clinics in Developmental Medicine No 87. London: Spastics International Medical Publications, 1984:1–11.

4 Hagberg B, Hagberg G, Olow L. The changing panorama of cerebral palsy in Sweden, 1954–1970. II. Analysis of various syndromes. *Acta Paediatr Scand* 1975;**64:**193–200.

5 Bax M. Terminology and classification of cerebral palsy. *Dev Med Child Neurol* 1964;**6:**295–7.

6 Little WJ. On the influence of abnormal parturition, difficult labours, premature birth, and asphyxia neonatorum, on the mental and physical condition of the child, especially in relation to deformities. *Transactions of the Obstetrical Society of London* 1862;**3:**293–344. (Reprinted in *Cerebral Palsy Bulletin* 1958;**1:** 5–36.)

7 Hagberg B, Hagberg G, Olow L. The changing panorama of cerebral palsy in Sweden 1954–1970. I. Analysis of the general changes. *Acta Pediatr Scand* 1975;**64:**187–92.

8 Blair E, Stanley FJ. Interobserver agreement in the classification of cerebral palsy. *Dev Med Child Neurol* 1985;**27:**615–22.

9 Nelson KB, Ellenberg JH. Children who 'outgrow' cerebral palsy. *Pediatrics* 1982;**69:**529–35.

10 Nelson KB, Ellenberg JH. Neonatal signs as predictors of cerebral palsy. *Pediatrics* 1979;**64:**225–32.

11 Larroche JC, Bethmann O, Couchard M. Brain damage in the premature infant: early lesions and new aspects of sequelae. *Ital J Neurol Sci* 1986;**5**(suppl):43–52.

12 Costello AMdeL, Hamilton PA, Baudin J, *et al.* Prediction of neurodevelopmental impairment at 4 years from brain ultrasound appearance in very preterm infants. *Dev Med Child Neurol* 1988;**30:**711–22.

13 Amiel-Tison C, Dube R, Garel M, Jequier JC. Outcome at age 5 years of full term infants with transient neurologic abnormalities in the first year of life. In: Stern L, ed. *Intensive care IV.* New York: Masson, 1983:247–57.

14 Owen MJ, Lewis SW, Murray RM. Obstetric complications and schizophrenia: a computed tomographic study. *Psychol Med* 1988;**18:**331–9.

15 Hadders-Algra M, Huisjes HJ, Touwen BCL. Perinatal correlates of major and minor neurological dysfunction at school age: a multivariate analysis. *Dev Med Child Neurol* 1988;**30:**472–81.

16 Hadders-Algra M, Huisjes HJ, Touwen BCL. Perinatal risk factors and minor neurological dysfunction: significance for behaviour and school achievement at nine years. *Dev Med Child Neurol* 1988;**30:**482–91.

17 Stewart AL, Costello AMdeL, Hamilton PA, *et al.* Relation between neurodevelopmental status at one and four years in very preterm infants. *Dev Med Child Neurol* 1989 (in press.)

18 Prasse DP, Siewert JC, Ellison PH. McCarthy performance and neurological functioning in children born 'at risk'. *Journal of Psychoeducational Assessment* 1983;**1:**273–83.

19 Amiel-Tison C, Ellison P. Birth asphyxia in the fullterm newborn: early assessment and outcome. *Dev Med Child Neurol* 1986;**28:**671–82.

20 Amiel-Tison C, Grenier A. *Neurologic assessment within the first year of life.* New York: Oxford University Press, 1986.

21 Amiel-Tison C. Pediatric contribution to the present knowledge on the neurobehavioural status at birth. In: Mehler J, Fox R, eds. *Neonate cognition.* Hillsdale: Laurence Erlbaum, 1985: 265–280.

22 Thomas A, Saint-Anne Dargassies S. *Etudes neurolgiques sur le nouveau-né et le jeune nourrisson.* Paris: Masson, 1952.

23 Peiper A. *Cerebral function in infancy and childhood.* New York: Consultants Bureau Enterprises, 1963.

24 Chapelais JdeZ, McFarlane JA. A review of 404 'late walkers'. *Arch Dis Child* 1984;**59:**512–16.

25 Frostig M, Welty Lefever D, Whittlesey JRB. A developmental test of visual perception for evaluating normal and neurologically handicapped children. *Percept Mot Skills* 1961;**12:**383–94.

26 Saint-Anne Dargassises S. *The neuromotor and psychoaffective development of the infant.* Amsterdam: Elsevier, 1986.

27 Amiel-Tison C. Cerebral damage in full-term: aetiological factors, neonatal status and long term follow-up. *Biologia Neonatorum* 1969;**14:**234–50.

28 Stewart AL, Reynolds EOR, Hope PL, *et al.* Probability of neurodevelopmental disorders estimated from ultrasound appearance of brains of very preterm infants. *Dev Med Child Neurol* 1987;**29:**2–11.

29 Stewart AL, Hope PL, Hamilton PA, *et al.* Prediction in very preterm infants of satisfactory neurodevelopmental progress at 12 months. *Dev Med Child Neurol* 1988;**30:**53–63.

30 Grenier A. Prévention des déformations précoces de hanche chez les nouveau-nés à cerveau lésé. *Ann Pediatr* 1988;**35:**423–7.

Correspondence to Dr Ann Stewart, Department of Paediatrics, The Rayne Institute, University College, University Street, London WC1E 6JJ.