Cranial ultrasound abnormalities in full term infants in a postnatal ward: outcome at 12 and 18 months

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Abstract

Objective—To investigate whether cranial ultrasound abnormalities found in low risk full term infants had any influence on neurodevelopmental outcome.

Methods-For 103 infants who had a neurological assessment, a cranial ultrasound examination, and for whom antenatal and perinatal data were collected within 48 hours of delivery, neurodevelopmental status was evaluated at 12 and 18 months. The results of a scored neurological examination and the Griffiths mental developmental scale were correlated with the presence and type of ultrasound abnormality found in the neonatal period. Results-None of the infants with ultrasound abnormalities showed any signs of cerebral palsy or severe developmental delay. There was also no significant difference between the overall neurological and neurodevelopmental scores of the infants with normal and abnormal ultrasound findings. However, when the individual subscales of the Griffiths test were analysed, all infants with bulky choroid or intraventricular haemorrhage had normal scores in all subscales, four of eight with periventricular white matter lesions had low scores on the locomotor subscale, and three of five with asymmetrical ventricles had low scores on the performance subscale. The presence of adverse antenatal and perinatal factors did not affect the outcome in this group.

Conclusion—Incidental ultrasound abnormality in full term neonates, in particular intraventricular haemorrhage, although common, appear to have a good prognosis. Longer follow up studies are needed to see whether some of these infants, in particular those with white matter lesions, develop dyspraxia or other minor neurological impairments at school age.

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Keywords: ultrasound; neurological assessment; intraventricular haemorrhage; brain; white matter; development

The advent of cranial ultrasound as a routine tool in neonatology has greatly improved our knowledge of the presence and incidence of brain lesions in the newborn infant. Cranial ultrasound has been used routinely for infants at risk of neurological impairment, such as those born prematurely¹⁻⁵ or who have suffered

from birth asphyxia,^{6 7} but less has been reported about the range of the cranial ultrasound findings and their long term significance in low risk infants.⁸⁻¹²

In 1996 we performed cranial ultrasound and neurological assessments in a cohort of 177 infants regarded as normal at birth. Thirty five of the 177 infants (20%) showed some ultrasound changes. As in previous studies, we observed a significant incidence of haemorrhages (6%) and asymmetrical ventricles (6%). We also observed that an additional 8% showed periventricular white matter echogenicity, which until then had not been described in normal full term infants. Some 8% of the infants had an unusually full choroid. The presence of abnormal features on ultrasound scans was associated with deviant patterns on the neurological examination (p < 0.0001).¹³

The aim of this study was to evaluate the neurodevelopmental outcome in this population to determine whether infants with cranial ultrasound abnormalities differ from those with normal scans.

Subjects and methods

The infants described in this study are part of a project investigating neurodevelopmental outcome in a cohort of infants admitted soon after birth to the postnatal ward at Queen Charlotte and Chelsea Hospital and regarded as normal by the obstetric and paediatric staff.¹⁴ The project was approved by the research ethics committee of the Royal Postgraduate Medical School.

SUBJECTS

While they were inpatients, mothers were asked if a neurological examination and cranial ultrasound scan could be performed on their infant within the first 48 hours of birth; 177 agreed to both.¹³

When the cohort of 177 infants approached the age of 12 or 18 months, their parents were contacted again to see if they were prepared to let their child have a neurological and developmental examination. Thirty seven families could not be traced because the address and the contact numbers available in the notes were no longer appropriate; three families had moved abroad, 24 had moved outside the area, and 10 were not interested in participating in the study. This resulted in a final cohort of 103 infants. Seventy six infants (46 boys and 30 girls) were assessed at the age of 12 months (range 11.7-13.3, mean 12.3), and 27 (12 boys and 15 girls) were assessed at the age of 18 months (range 17-20.3, mean 18.3).

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Table 1 Individual antenatal and perinatal factors

Antenatal factors	Perinatal factors
Amniocentesis	Abnormal cardiotocography
Intrauterine growth retardation	Abnormal Apgar scores
Reduced amniotic fluid	Low cord pH
Reduced fetal movements	Presence of meconium (grade I–III)
Infection)
Bleeding	
High blood pressure	
Unexplained abdominal pain	
Trauma	
In vitro fertilisation	
Breech position	

Details of antenatal and perinatal data were available for all the children.

CRANIAL ULTRASONOGRAPHY

Cranial ultrasound scans were performed with an Advanced Technology Laboratory (ATL) mark IV sector scanner, using 5 and 7.5 MHz probes. The scans were reviewed for normal anatomy, ventricular size, and evidence of focal or diffuse increased echogenicity in the cerebral hemispheres and basal ganglia in the neonatal period.¹³

ANTENATAL AND PERINATAL FACTORS

All the maternal notes were searched for the presence of adverse antenatal and perinatal factors to determine their influence on outcome in children with normal and abnormal ultrasound findings. Table 1 shows a list of the individual antenatal and perinatal factors found.

FOLLOW UP CLINICAL EXAMINATION Neurological examination

The proforma used to record the neurological findings at 12 and 18 months consists of 37 items divided into three sections.¹⁴ The first section includes 26 items assessing cranial nerve function, posture, movements, tone, and reflexes. The second section includes eight items documenting developmental progress, and the third section includes three items evaluating state of behaviour. This examination has been standardised in a normal population at 12 and 18 months of age. Scores between 73 and 78 are considered normal.

Developmental assessment

The neurodevelopmental outcome was evaluated by the Griffiths mental developmental scale.¹⁵ The developmental quotients (DQs) were calculated for global development as well as for the individual subscales (locomotor, personal-social scale, hearing and language, eye and hand coordination, and performance). Results were classified as normal when the DQ was 80 or above, and abnormal when it was below 80.¹⁶

The examiner (LH) who performed the follow up assessment was not involved with the neonatal examination and was unaware of the early findings.

STATISTICAL ANALYSIS

The results of the outcome measures were analysed in the groups with normal and abnormal ultrasound findings (Student's t test, level of

Table 2 Details of antenatal and perinatal factors, global neuroscore, global developmental quotients, and subscale developmental quotients in infants with abnormal ultrasound

Cranial ultrasound finding	Antenatal factors	Perinatal factors	Global neuro score	Global DQ	Locomotor	Personal– social	Hearing– speech	Eye–hand coordination	Performance
Choroid cyst	IVF, infection		69	82	96	72	100	69	69
Arachnoid cyst			76	111	107	107	110	113	114
Asymmetrical ventricles	Breech		73	98	108	99	110	95	76
Asymmetrical ventricles	Amniocentesis, breech		76	91	91	90	93	89	92
Asymmetrical ventricles	Infection		77	97	107	85	102	122	73
Asymmetrical ventricles			77	98	91	123	106	103	64
Asymmetrical ventricles	Amniocentesis		74	114	128	110	118	122	93
IVH grade I*			77	94	112	80	103	95	80
IVH grade I			75	98	112	104	86	89	100
IVH grade IIa	IUGR, bleed		76	97	95	104	93	106	92
IVH grade IIa	IUGR		77	93	108	95	89	89	84
IVH grade IIa	Amniocentesis, breech		75	122	100	137	119	136	114
IVH grade IIb	Bleed	Grade II meconium	76	103	112	90	96	100	115
PV WM lesion	High blood pressure		78	78	70	85	86	78	76
PV WM lesion	IUGR		73	79	78	85	82	66	84
PV WM lesion			77	102	78	113	93	95	131
PV WM lesion			78	91	91	104	82	114	80
PV WM lesion	Reduced fetal movement		73	109	99	113	107	123	104
PV WM lesion	IUGR	Grade II meconium	75	121	103	149	132	118	100
PV WM lesion		Grade II meconium	75	107	79	124	115	109	98
PV WM lesion			75	104	107	101	101	108	106
Thalamic density	IUGR, amniocentesis		76	103	95	108	114	100	96
Thalamic density			75	107	123	94	93	112	126
Full choroid			74	104	99	99	103	112	108
Full choroid	Reduced fetal movement	Grade III meconium	78	120	120	95	125	112	139
Full choroid			77	98	113	93	100	95	89
Full choroid			77	121	124	122	132	118	104
Full choroid			77	118	106	132	117	122	110
Full choroid	High blood pressure		78	108	83	120	100	122	118
Full choroid	6 - F		78	103	130	89	90	113	110
Full choroid			74	110	129	114	100	111	97

*According to de Vries et al.16

IVH, intraventricular haemorrhage; IVF, in vitro fertilisation; IUGR, intrauterine growth retardation; PV, periventricular; WM, white matter; DQ, developmental quotient.

Cranial ultrasound	Antenatal factors	Perinatal factors	Global neuroscore	Global DQ	Loco motor	Personal– social	Hearing– speech	Eye–hand coordination	Performance
Normal			78	108	129	110	107	106	84
Normal			71	93	87	104	86	100	92
Normal		Low cord pH	74	93	87	104	93	106	80
Normal			77	120	120	127	110	100	135
Normal			73	100	108	94	92	104	100
Normal			74	109	95	108	103	106	127
Normal		Abnormal CTG, abnormal Apgar	78	101	120	99	96	95	92
Normal	Amniocentesis, reduced fetal movement		75	96	116	81	107	95	80
Normal	Infection, bleed, reduced fetal movement		71	111	95	99	128	112	115
Normal		Grade II meconium	73	115	103	113	121	112	119
Normal	Reduced fetal movement		77	105	103	104	114	106	96
Normal			77	103	99	90	118	100	104
Normal			77	114	116	108	96	146	111
Normal	Bleed		77	114	116	104	125	118	104
Normal			78	111	101	118	122	117	89
Normal			75	118	99	113	121	112	135
Normal	Reduced amniotic fluid		76	107	108	113	118	106	88
Normal			78	104	95	113	96	112	108
Normal	Bleed		75	102	99	108	100	112	96
Normal Normal	Infection Infection, amniocentesis	Abnormal CTG Abnormal CTG, grade II	77	94	96	100	94	86	94
		meconium	78	101	103	113	93	100	96
Normal Normal	Amniocentesis, reduced	Abnormal CTG	77	102	99	104	118	100	88
11011111	amniotic fluid		78	119	129	127	114	118	108
Normal	Bleed	Abnormal CTG	77	110	116	108	125	106	92
Normal	Infection, amniocentesis.		77	99	116	108	89	100	84
Normal	inteetion, anno concento.		78	102	120	117	100	95	80
Normal	High blood pressure		78	111	87	127	107	112	123
Normal	Abdominal pain		78	108	120	108	100	118	96
Normal	Bleed		75	100	91	99	114	112	88
Normal	Bleed		78	129	117	122	132	112	148
Normal	Amniocentesis, reduced amniotic fluid		76	117	125	122	107	122	110
Manual	Amniocentesis			117	125		107	135	96
Normal	Ammocentesis		76 78	105		113 104	93	123	123
Normal Normal	Deduced freed an ensurement		78 78	105	87 103	104 117	107	123	123
Normal	Reduced fetal movement Amniocentesis		72	102	91	108	107	129	125
Normal	Bleed		73	118	109	122	118		114
Normal	Bieed	Grade III meconium	75	104	109	104	118	128 112	84
Normal		Low cord pH	78	116	112	104	127	100	115
Normal	High blood pressure	Low cold pH	75	92	87	85	94	92	102
Normal	riigii biobu pressure		76	105	112	99	110	106	96
Normal			78	105	125	97	103	100	93
Normal			78	128	123	122	118	129	143
Normal			75	109	1124	99	100	118	115
Normal			73	119	133	122	100	128	110
Normal	Amniocentesis		77	104	112	99	96	95	115
Normal	Reduced fetal movement		75	122	125	129	114	134	109
Normal	requeed retar hittyement		75	108	133	129	92	117	89
Normal	Trauma		75	108	129	101	92 96	106	106
Normal	11auilla		78 78	108	129	101 127	126	117	100
Normal			76	122	1134	132	120	117	102
Normal			78	120	100	132	103	126	86
Normal			78	110	100	120	103	120	102
Normal			78	117	118	124	119	127	102
Normal			77	109	90	107	102	122	123
Normal	Infection, high blood pressure		77	113	106	120	112	117	106
Normal	meetion, men biobu pressure		77	117	106	137	112	108	118
Normal			77	129	118	137	112	136	130
Normal		Abnormal CTG, grade II							
		meconium	78	112	102	111	115	109	119
Normal			78	111	96	128	115	109	98
Normal			76	109	99	104	121	106	108

Table 3 Details of antenatal and perinatal factors, global neuroscore, global developmental quotients and subscale developmental quotients in infants with normal ultrasound and normal outcome (n = 72)

DQ, developmental quotient; CTG, cardiotocography.

significance p < 0.05). The eight children who showed an unusually full choroid were classified separately, as we found it difficult to decide whether this finding was normal or represented a choroidal haemorrhage.¹³

Results

Tables 2–5 show details of the results. The incidence of abnormal ultrasound and antenatal and perinatal factors in the cohort of 103 children who participated in the follow up study was not significantly different from the incidence of the same factors in the original cohort of 177 who had a neonatal cranial ultrasound examination (p > 0.05).

CRANIAL ULTRASOUND FINDINGS

A completely normal scan at birth was found in 72 of the 103 infants examined at follow up; the remaining 31 had some changes. Periventricular white matter echogenicities were present in eight of the 31, intraventricular haemorrhage in six, asymmetrical ventricles in five, unilateral thalamic densities in two, a choroid cyst in one, and a large infracerebellar space (possible

Table 4 Details of antenatal and perinatal factors, global neuroscore, global developmental quotients and subscale developmental quotients in infants with normal ultrasound and abnormal outcome

Cranial ultrasound	Antenatal factors	Perinatal factors	Global neuroscore	Global DQ	Locomotor	Personal–social	Hearing–speech	Eye–hand coordination	Performance
Normal		Abnormal CTG	72	86	87	76	96	95	80
Normal	IUGR		72	90	74	85	110	95	84
Normal			78	101	78	122	114	106	88
Normal			75	79	91	53	82	103	84
Normal			77	93	108	95	93	78	92
Normal	Amniocentesis, bleed		73	83	61	90	86	95	88
Normal	Breech		73	99	99	104	93	95	76
Normal	Trauma		78	90	103	81	93	100	76
Normal			63	90	69	97	92	106	93
Normal			74	89	95	98	76	103	90
Normal			75	90	79	107	92	91	82
Normal			77	87	96	98	72	96	90

DQ, developmental quotient; CTG, cardiotocography; IUGR, intrauterine growth retardation.

arachnoid cyst) in one. An unusually full choroid was found in eight infants.

ADVERSE ANTENATAL AND PERINATAL FACTORS Adverse antenatal factors were found in 29 of 72 infants (40%) with a normal ultrasound and in 16 of 23 infants (61%) with abnormal ultrasound. There was no significant difference in the relative incidence of the individual factors, but intrauterine growth retardation was more common in the infants with abnormal ultrasound (5/23 (22%) v 1/72 (1%)).

Adverse perinatal factors were recorded in 11 of 72 infants (15%) with normal ultrasound and in three of 23 (13%) with abnormal ultrasound findings. There was no significant difference in the total incidence of adverse perinatal factors in the two groups, but cardiotocography abnormalities only occurred in the group with normal ultrasound. Tables 2–4 show details of the type and incidence of the adverse antenatal and perinatal factors.

NEUROLOGICAL EVALUATION

Neurological examination was normal in 96 and abnormal in seven of the 103 infants studied.

Of the 72 infants who had a normal cranial ultrasound scan, 66 had a normal and six an abnormal neurological examination. Of the 23 infants with abnormal ultrasound findings, 22 had a normal and one an abnormal neurological examination. All eight children with full choroid had a normal neurological examination.

When the scores on the neurological examination were analysed, the difference between the three groups was not significant: 75.93 (2.52), 76.63 (1.69), and 75.35 (2.01) for the group with normal cranial ultrasound results, the group with full choroid, and the group with abnormal ultrasound results respectively (mean (SD)). Tables 2–4 give the individual scores of the neurological examinations in the cohort in relation to ultrasound findings.

DEVELOPMENTAL SCALES

Of the 103 infants examined, 100 had normal and three had abnormal global DQ.

Eighty three of the 103 infants assessed had normal scores on all the subscales. The other 20 had mild delay (DQ between 61 and 79) on at least one subscale. None of the infants in our cohort had severe delay.

Sixty of the 72 infants with normal ultrasound scans had a normal score on all the subscales and 12 had at least one abnormal score.

Fifteen of the 23 infants with abnormal ultrasound scans had normal and eight abnormal scores on at least one of the subscales.

Table 5 The incidence of normal and abnormal neurological examination and Griffiths scores in the children with different ultrasound findings

	Neurological examination	Full scale	Locomotor	Personal– social	Hearing– speech	Eye–hand coordination	Performance
Normal cranial ultra	asound (n = 72)						
Normal	66	71	68	70	70	71	70
Abnormal	6	1	4	2	2	1	2
Full choroid $(n = 8)$)						
Normal	8	8	8	8	8	8	8
Abnormal	0	0	0	0	0	0	0
Haemorrhage (n =	6)						
Normal	6	6	6	6	6	6	6
Abnormal	0	0	0	0	0	0	0
Asymmetric ventric	les (n = 5)						
Normal	5	5	5	5	5	5	2
Abnormal	0	0	0	0	0	0	3
Periventricular whit	e matter densities (1	n = 8)					
Normal	8	6	4	8	8	6	7
Abnormal	0	2	4	0	0	2	1
Thalamic densities	(n = 2)						
Normal	2	2	2	2	2	2	2
Abnormal	0	0	0	0	0	0	0
Arachnoid cyst (n=	1)						
Normal	1	1	1	1	1	1	1
Abnormal	0	0	0	0	0	0	0
Choroid cyst (n=1)							
Normal	0	1	1	0	1	0	0
Abnormal	1	0	0	1	0	1	1

All eight infants with full choroid had normal scores on all the subscales.

Table 5 shows the incidence of normal and abnormal neurological examination and Griffiths scores in the cohort subdivided according to ultrasound findings.

Tables 2-4 give details of the findings in all the infants. The results show that, whereas the presence of haemorrhages or full choroids was not associated with abnormal results on any of the subscales, white matter lesions were associated with locomotor delay in four of eight infants and asymmetrical ventricles with delay in performance in three of five infants (table 2). The presence of adverse antenatal or perinatal factors had no impact on outcome in infants with abnormal scans.

Discussion

The aim of this study was to evaluate whether the outcome of a cohort of infants regarded as normal in the neonatal period, but with abnormal features on neonatal cranial ultrasound examination, differed from those with normal scans. We were able to follow 60% of the original cohort with neonatal ultrasound and we feel there was no bias, as there was no selection other than not being able to find the infants. The refusal rate among those traced was low, and in fact the incidence of ultrasound abnormalities and adverse antenatal/perinatal factors was similar in the original and in the follow up cohort.

At follow up at 12 or 18 months, none of the children in our cohort showed any sign of cerebral palsy and there was no significant difference between the infants with and without abnormal ultrasound results on neurological examination.

Although the scores on the Griffiths developmental test in the group with ultrasound abnormalities were lower than in the group with normal cranial ultrasound, the difference was not significant The numbers were too small to allow a meaningful statistical analysis of the results in relation to the different types of ultrasound findings, but it is of interest that intraventricular haemorrhages and thalamic densities were always associated with normal scores on all the subscales. Other ultrasound abnormalities were associated with some degree of impairment. Four of the eight infants with periventricular white matter echogenicity had low gross locomotor scores, while those with asymmetrical ventricles had normal locomotor scores but three of five had lower scores in the performance scales. The infants who showed a full choroid, a finding that we were uncertain how to classify on the neonatal ultrasound, not only did not show any abnormal results but, as a group, had higher scores than the infants with normal ultrasound.

Our results are in basic agreement with previous studies which showed that unilateral thalamic densities were associated with a normal outcome.17 18 Also in agreement with previous studies,¹⁹⁻²¹ we found that intraventricular haemorrhage, even when large, if not complicated by severe ventricular dilatation or paren-

Key messages

- Incidental ultrasound abnormalities in full term neonates are common but the infants appear to have a good prognosis at 12 and 18 months
- Unilateral thalamic densities and intraventricular haemorrhages not complicated by parenchymal extensions were not associated with neurological sequelae

chymal extension was not associated with any neurological or developmental sequelae at 12 or 18 months of age.

As all the infants in our cohort only had one scan within 48 hours, our data on periventricular densities are not easily comparable with previous studies where outcome depends on the persistence and evolution of the lesions.²² ²³

As adverse antenatal factors such as infection and bleeding tendencies have been reported to be associated with increased risk for cerebral palsy,²⁴ we also tried to evaluate whether the variability in outcome observed in our infants could be related to the presence of adverse antenatal or perinatal factors. Although antenatal factors were more common in the infants with ultrasound abnormalities, we were unable to find any significant association between the presence or the type of any of these factors and the outcome.

Our results suggest that some abnormality on cranial ultrasound is relatively common in a population considered to be at low risk for neurological sequelae in the neonatal unit, but they are not associated with cerebral palsy or other major neurological or developmental abnormalities. As the follow up in our cohort was relatively short, we do not know whether any of these infants would develop dyspraxia or minor neurological impairment at school age, as found in preterm infants with similar lesions. It would be of interest to know whether any children who do develop minor neurological problems also had minor developmental abnormalities on the short term follow up.

- 1 Pape KE, Blackwell RJ, Cusick G, et al. Ultrasound detection of brain damage in preterm infants. *Lancet* 1979;i:1261–74.
- Hope PJ, Gould SJ, Howard S, Hamilton PA, Costello AM de L, Reynolds EOR. Precision of ultrasound diagnosis of pathologically verified lesions in the brains of very preterm babies. Dev Med Child Neurol 1988;30:457-71.
- 3 Paneth N, Rudelli R, Kazam E, Monte W. Brain damage in the preterm infant. Clinics in developmental medicine. London: Mac Keith Press, 1994:131.
- 4 Rennie IM. Neonatal cerebral ultrasound. Cambridge: Cam-
- Freinre Jr. Iconata Freiser and Massana. Cambridge: Cambridge: Cambridge: Cambridge: Cambridge: Source and S
- 6 Eken P, Jansen GH, Groenendaal F, Rademaker KJ, de Vries L. Intracranial lesions in the full term with hypoxicischemic lesions. Neuropediatrics 1994;24:301-7
- 7 Levene MI. The asphyxiated newborn infant. In: Levene MI, Lilford RJ, Bennett MJ, Punt J, eds. Fetal and neonatal neurology and neurosurgery. Edinburgh: Harcourt Publish-ers Ltd, 1995:405–25.
- 8 Guekos-Thoni U, Bolthauser E, Willi UB. Intraventricular hemorrhage in full-term neonates. Dev Med Child Neurol 1982;24:704-5.
- 9 Scher NS, Wright FS, Lockman LA, et al. Intraventricular hemorrhage in the full-term neonate. Arch Neurol 1982;39:769-83.
- Hayden CK, Shattuck KE, Richardson CJ, Ahrendt DK, House R, Swiscuk LE. Subependymal germinal matrix hemorrhage in full-term neonates. *Pediatrics* 1985;75:714– 18

- 11 Shen EY, Huang FY. Subependymal cysts in normal neonates. Arch Dis Child 1985;60:1072-4.
- 12 Heibel M, Heber R, Bechinger D, Kornhuber HH. Early diagnosis of perinatal cerebral lesions in apparently normal full-term newborns by ultrasound of the brain. Neuroradiology 1993;35:85–91.
- 13 Mercuri E, Dubowitz L, Paterson-Brown S, Cowan F. Incidence of cranial ultrasound abnormalities in apparently well neonates on a postnatal ward: correlation with antenatal and perinatal factors and neurological status. Arch Dis Child Fetal Neonatal Ed 1998;79: F185-9.
- 14 Dubowitz L, Dubowitz V, Mercuri E. The neurological assessment of the preterm and full-term infant. *Clinics in* developmental medicine, 2nd ed. Mac Keith Press, London, 1999:148
- 15 Griffiths R. The abilities of babies: a study in mental measurement. Amersham: Association for Research in Infant and Child Development, 1986.
- 16 de Vries LS, Dubowitz LMS, Dubowitz V, et al. Predictive
- value of cranial ultrasound in the newborn baby: a reappraisal. *Lancet* 1985;ii:137–40.
 de Vries LS, Smet M, Goemans N, Wilms G, Devlieger H, Casaer P. Unilateral thalamic haemorrhage in the preterm and full-term newborn. *Neuropediatrics* 1992;23: 153-6.

- Garg BP, De Myer WE. Ischaemic thalamic infarction in children: clinical presentation, etiology and outcome. *Pedi-atr Neurol* 1995;13:46-9.
 Papile L-A, Munsick-Bruno G, Schaefer A. Relationship of
- cerebral intraventricular hemorrhage and early childhood neurologic handicap. J Pediatr 1983;103:273–7.
- 20 Stewart AL, Thornburn RJ, Hope PL, Goldsmith M, Lipscomb AP, Reynolds EOR. Ultrasound appearance of the brain in very preterm infants and neurodevelopmental outcome at 18 months of age. Arch Dis Child 1983;58:598-604
- Dubowitz LMS, Dubowitz V, Palmer PG, Miller G, Fawer C-L, Levene MI. Correlation of neurologic assessment in the preterm newborn infant with the outcome at one year.
- The precent rewoon main with the outcome at our year. J Pediat 1984;105:452–6.
 Vries LS, Regev R, Pennock JM, Wigglesworth JS, Dubowitz LMS. Ultrasound evolution and later outcome of infants with periventricular densities. *Early Hum Dev* 1988;16:225–33. 22 de
- 23 Guzzetta F, Shackelford GD, Volpe S, Perlman JM, Volpe JJ. Periventricular intraparenchymal echodensities in the pre-mature newborn: critical determinant of neurologic outcome. *Pediatrics* 1986;**78**:995–1006.
- 24 Nelson KB, Dambrosia JM, Grether JK, Phillips TM. Neonatal cytokines and coagulation factors in children with cerebral palsy. Ann Neurol 1998;44:665–75.