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Cerebral palsy: effects of twinning, birthweight, and gestational age

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Abstract

***Aims*—To determine the effects of birthweight and gestational age on the risk of cerebral palsy for multiple and singleton births.**

***Methods*—Children on the North East Thames Regional Health Authority Interactive Child Health System, born between 1 January 1980 and 31 December 1986, and notified as having cerebral palsy, were included. Cases of postneonatal onset, of known progressive, or non-cerebral pathology and with only mild signs were excluded. Rates and relative risks were calculated using the most complete data, which related to 1985-86, and comprised 102 059 singletons and 2367 twins. Logistic regression was used to examine the associations between being a twin, gestational age, and birthweight.**

***Results*—The crude rate per 1000 survivors at 1 year of age was 1.0 in singletons and 7.4 in twins. The relative risk was greatest in twins weighing more than 2499 g (4.5). However, after adjusting for reduced birthweight of twins it was the relative risk of twins weighing less than 1400 g that was significantly increased. Logistic regression confirmed that lower fetal growth, lower gestational age, and being a twin are all independent risk factors for cerebral palsy.**

***Conclusion*—The increased risk to twins of cerebral palsy is not entirely explained by their increased risk of prematurity and low birthweight.**

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Keywords: cerebral palsy, twins, singletons, birthweight, gestational age.

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The increased risk of both mortality and cerebral palsy in multiple births [1–3] has been known for many years. However, good population based estimates of the risks of cerebral palsy relative to singletons have been available only recently.[4–5] These associations are of interest firstly because rates of multiple births are increasing, in part due to the introduction of treatments for infertility.[6–7] Secondly, differences between the pattern of cerebral palsy in twins and singletons generate aetiological hypotheses.

A register of children notified as having cerebral palsy was set up in the North East Thames Regional Health Authority. Linkage of these data with the records of those children on the Regional Child Health Computer System (RICHS) permitted the calculation of population based risks for twins and singletons in this region.[8] This system includes data on gestational age that are not available from birth registrations. It made possible comparisons not previously reported: estimation of relative risks for gestational age, weight for gestational age, and birth weight categories adjusted for the different "normal" distribution of twins and singletons; and the use of the method of logistic regression to adjust for associations between these variables.

Method

The register was set up in 1985 with the aim of ascertaining affected children born since 1980. Multiple sources of ascertainment were used, including general practitioners, paediatricians, orthopaedic surgeons, and SCOPE (formerly the Spastics Society). The Office of Population Censuses and Surveys (OPCS) were asked for names of eligible children who were certified as having died with cerebral palsy. As a deliberate policy, no definition of cerebral palsy was given to notifiers and no inclusion or exclusion criteria were specified.

The paediatrician responsible for the management of each notified case was asked for a detailed assessment, and to obtain parental consent for release of medical information to the register at the child's next visit. Over 30 paediatricians and their staff contributed information to the study. Further information about each child was sought when they were between 3.5 and 4 years and again between 7 and 8 years, although this was not always forthcoming. Clinical information about the distribution, type, and severity of cerebral palsy was available for 85% of cases born in 1985 and 1986.

DENOMINATOR DATA AND MATCHING

In 1985 and 1986 RICHS data quality was validated by comparison with regional birth registration data, after excluding obvious duplicates (children born within the region but outside their district of residence or those who had moved within the region). For children about whom information on birthweight, number of children in the pregnancy, and outcome of birth was available, the two data sets were very similar, including the distribution of children of different birthweight groups for both twin and singleton births (table 1).

Matching children on the cerebral palsy register with their RICHS records permitted the validation of birth and residence data reported for the cerebral palsy cases. Matching was attempted for all cases notified to the register, but its success was limited for 1980 through

Table 1 Number of live births and survivors at one year in NETRHA in 1985 and 1986 from RICHS and OPCS data

		Birthweight					Total
		< 1000 g	1000-1499 g	1500-2499 g	≥ 2500 g	Not known	
Singleton:							
Live births	RICHS	252	531	5286	94245	1745	102059
	OPCS	256	545	5494	96322	124	102741
Survivors	RICHS	119	458	5127	93832	1735	101271
	OPCS	119	470	5323	95863	105	101880
Twins:							
Live births	RICHS	63	116	999	1103	86	2367
	OPCS	58	107	964	1070	7	2206
Survivors*	RICHS	29	98	983	1095	84	2289
	OPCS	26	91	942	1060	6	2125

* Survivors at one year.

1984 because RICHS data were not available for the whole region.

The matching process was in three stages. First, the birth date, the first four letters of first names and gender were matched with the RICHS system data, which did not include surnames. If cases from the register matched for these parameters they were then matched for birthweight (stage 2). If birthweights did not match or were absent, further matching was undertaken (stage 3) by seeking written information about the first four letters of surnames from district RICHS managers. For a registered case to be regarded as matched with RICHS data, the date of birth, first four letters of first name and gender, and either the birthweight, to within 30 g, or the first four letters of their surname, had to be the same.

Children were excluded if the cause of their motor signs was known to be progressive or not cerebral in origin, the cause of their cerebral palsy was a postneonatal event, or no limb had an involvement greater than 1 on the severity coding system.[9] Children who were not matched on the RICHS data, or where matched RICHS data did not include outcome of birth or number of children born, were also excluded. Cases were allocated to twin or singleton categories on the basis of RICHS data, regardless of information given to the register. The register included no triplet or higher order multiple births born in 1985-86.

ANALYSIS

Analysis was largely restricted to 1985 and 1986, the two years over which both data sets were of good quality. Crude birthweight specific rates of cerebral palsy were determined for survivors at 1 year and crude gestational age specific rates for maternities bearing at least one live child still surviving at 1 year. To allow for the effect of infant death when comparing twin and singleton births, a "death or cerebral palsy" rate has also been calculated.

Birthweight analysis was done in two ways. Firstly, traditional categories were used to allow comparison with other studies. Secondly, adjusted birthweight categories were created to allow "equivalent" comparison of twins and singletons. This was done by subtracting, from traditional birthweight category boundaries, the difference in mean birthweight between twins and singletons of the same gestational age, using standards reported from Scotland.[10] Thus where the mean birthweight of singletons was close to 2500 g (at 35 weeks of gestation), the mean birthweight of twins of the same gestational age was about 300 g less, so that the corresponding boundary for twins became 2200 g. Similarly, where the mean weight of singletons was close to 1500 g (30 weeks), the mean of twins was about 100 g less, making the boundary 1400 g. The difference of 100 g was also found where the mean weight of singletons was about 1000 g (26 weeks), making the corresponding boundary for twins 900 g.

Multiple logistic regression analysis was used to explore the relative importance of risk factors for cerebral palsy; twin *vs* singleton birth, gestational age and *fetal growth* (the difference in birthweight from mean birthweight for a given gestation, specific for twins and singletons). *Low fetal growth* was also regarded as a dichotomous variable and defined as all cases with fetal growth of less than 1.5 standard deviations below the mean fetal growth (about 675 g below the mean and 5.6% of babies). For this analysis only survivors at 1 year born between 24 and 42 weeks, with both gestational age and birthweight available, were included. A measure of fetal growth was used rather than actual birthweight because birthweight and gestational age are not independent. There was simple analysis of the non-linearity of effects of fetal growth and gestation. All adjustments to weight data were made using Scottish data,[10] as before. Although birthweights in Scotland are slightly higher than those in England,[11] it is likely that this difference is true of twins and singletons.

Rates of cerebral palsy and population attributable risk per cent (PAR%) have been calculated for 1980-86 to allow comparison with previously published work.[4-5]

Data were stored on SPSS/PC. Analysis of rates with exact binominal confidence intervals (CI) and relative risk with Taylor series CI was performed on EPIINFO6[12] and logistic regression analysis was performed using Stata (release 3.1, version 6).

Results

Twenty nine twins with cerebral palsy born between 1980 and 1986 were linked with regional data. Rates for cerebral palsy for twin cases for these years per 1000 survivors at 1 year were 6.3 (95% CI:4.2, 9.0) and 1.2 (95% CI:1.0, 1.3) for singletons, giving an increased

Table 2 Data from NETRHA computed child health records 1985-1986 births

Category	Singletons	Twins
Numbers of total live births	102059	2367
Total maternities with at least one live birth	102059	1258
Total maternities with at least one survivor at 1 year	101271	1236
Infant deaths	788	78
Maternities affected by infant death	788	60
Cerebral palsy cases	104	17
Infant deaths per 1000 live births (95% CI)	7.7 (7.2 to 8.3)	32.9 (26.1 to 40.9)
Infant deaths per 1000 maternities* (95% CI)	7.7 (7.2 to 8.3)	47.7 (36.6 to 61.0)
Cerebral palsy per 1000 1 year survivors (95% CI)	1.0 (0.8 to 1.2)	7.4 (4.3 to 11.9)
Cerebral palsy per 1000 maternities** (95% CI)	1.0 (0.8 to 1.2)	11.3 (6.2 to 18.9)†
Cerebral palsy or infant death per 1000 live births (95% CI)	8.7 (8.2 to 9.3)	40.1 (32.6 to 48.8)
Cerebral palsy or infant death per 1000 maternities* (95% CI)	8.7 (8.2 to 9.3)	57.2 (45.0 to 71.5)‡

* Maternities with at least one live birth.
** Maternities with at least one survivor at 1 year.
† Numerator adjusted for three concordant twins.
‡ Numerator adjusted for three pairs of concordant twins and twin maternities affected by both cerebral palsy and cotwin death.

relative risk for twins for this period of 5.3 (95% CI 3.6, 7.7). The PAR% of cerebral palsy for twin survivors at 1 year in NETRHA for 1980-86 was 8.5%.

Births in 1985 and 1986, where the regional denominator data are complete and validated, included 104 registered singletons and 17 twins matched with their RICHS birth records. These were derived from 102 059 singleton, 2367 twins, and 85 higher order live births. Three sets were concordant for cerebral palsy. The co-twins of another three pairs had either been stillborn (one case) or had died in infancy.

Rates of death in infancy, cerebral palsy for survivors to 1 year, and "cerebral palsy or death in infancy," for 1985 and 1986, are shown in table 2 for singleton and twin births and maternities. Cerebral palsy rates from these years are statistically similar to 1980-86 rates.

The denominator data included 16 twin pairs known to have one surviving twin at 1 year where the other had been stillborn. The rate for cerebral palsy per 1000 of these maternities was 62.5 (95% CI 15.8, 302.3). In 38 denominator pairs one twin had been an infant death and one survived to 1 year. The rate for cerebral palsy per 1000 of these maternities was 52.6 (95% CI 6.4, 177.5). This compares with a rate per 1000 maternities of 11.8 (95% CI 6.5, 19.8), if both twins survived to 1 year.

Table 3 shows rates of infant death and cerebral palsy for conventional birthweight categories for both singletons and twins, and for birthweight categories for twins adjusted for the different distribution of "normal" twin weights. In table 4 rates for different gestational age groups have been calculated for maternities.

In the conventional birthweight groups the relative risk of cerebral palsy for twins compared with singletons was significantly increased for twins weighing 2500 g or more (relative risk 4.5; 95% CI 1.4, 14.4). The relative risks in the lower conventional groups were raised, but not significantly. The relative risk was also raised where the gestational age was greater than or equal to 37 weeks (relative risk 6.3; 95% CI 2.0, 20.1).

In the adjusted birthweight groups twins weighing less than 900 g had a relative risk of cerebral palsy of 4.8 (95% CI 1.4, 16.2) and between 900 and 1399 g, one of 3.1 (95%CI 1.1, 9.1). The increased rate of cerebral palsy for higher birthweight twins compared with singletons is no longer significant when adjusted birthweight categories are used.

Logistic regression analysis showed that lower fetal growth, lower gestational age, and

Table 3 Distribution and rates of cerebral palsy (CP) and infant death (ID) for twin and singleton births in 1985 and 1986, for birthweight categories

Birthweight categories	CP/1000 survivors at 1 year (95% CI)	ID/1000 live births (95% CI)	CP or ID/1000 live births (95% CI)	CP	Infant deaths	Live births	Survivors at 1 year of age
Extremely low birthweight:							
Twins < 900 g	200 (57 to 437)	574 (422 to 717)	660 (507 to 791)	4	27	47	20
Singletons < 1000 g	42 (14 to 95)	528 (464 to 591)	548 (484 to 610)	5	133	252	119
Twins < 1000 g*	138 (39 to 317)	540 (409 to 666)	603 (472 to 724)	4	34	63	29
Very low birthweight:							
Twins 900 to 1399 g	62 (20 to 138)	198 (125 to 289)	248 (167 to 343)	5	20	101	81
Singletons 1000 to 1499 g	20 (9 to 37)	137 (109 to 170)	154 (125 to 188)	9	73	531	458
Twins 1000 to 1499 g*	51 (17 to 115)	155 (95 to 234)	198 (130 to 283)	5	18	116	98
Low birthweight:							
Twins 1400 to 2199 g	9 (3 to 22)	24 (13 to 40)	33 (20 to 52)	5	13	547	534
Singletons 1500 to 2499 g	6 (4 to 9)	30 (26 to 35)	36 (31 to 42)	33	159	5286	5127
Twins 1500 to 2499 g*	5 (2 to 12)	16 (9 to 26)	21 (13 to 32)	5	16	999	983
Normal birthweight:							
Twins ≥ 2200 g	1.9 (0.4 to 5.6)	10 (6 to 16)	12 (7 to 19)	3	16	1586	1570
Singletons ≥ 2500 g	0.6 (0.5 to 0.8)	4.4 (4.0 to 4.8)	5.0 (4.5 to 5.5)	57	413	94245	93832
Twins ≥ 2500 g*	3 (1 to 8)	7 (3 to 14)	10 (5 to 18)	3	8	1103	1095
Birthweight unknown:							
Twins	0 (0 to 43)	23 (3 to 81)	23 (3 to 81)	0	2	86	84
Singletons	0 (0 to 2)	6 (3 to 11)	6 (3 to 11)	0	10	1745	1735
Total:							
Twins	7 (4 to 12)	33 (26 to 41)	40 (33 to 49)	17	78	2367	2289
Singletons	1.0 (0.8 to 1.2)	7.7 (7.2 to 8.3)	8.7 (8.2 to 9.3)	104	788	102059	101271

* Rates using the same birthweight categories as singletons.

Table 4 Distribution and rates of cerebral palsy (CP) and infant death (ID) for twin and singleton maternities in 1985 and 1986, for gestational age

Gestational age categories	CP/1000** (95% CI)	ID/1000* (95% CI)	†CP or ID/1000* (95% CI)	‡CP	Infant deaths††	Live births*	Survivors age 1 year**
≤ 28 weeks							
Twins	63 (8 to 208)	549 (403 to 689)	588 (442 to 724)	2	28	32	51
Singletons	32 (13 to 64)	410 (359 to 461)	428 (378 to 480)	7	154	222	376
29 to 32 weeks							
Twins	51 (17 to 114)	89 (42 to 162)	129 (70 to 210)	5	9	99	101
Singletons	28 (18 to 42)	88 (70 to 109)	113 (93 to 136)	22	76	790	866
33 to 36 weeks							
Twins	9 (2 to 27)	25 (11 to 48)	34 (17 to 60)	3	8	326	326
Singletons	3 (2 to 5)	25 (21 to 31)	28 (23 to 34)	12	111	4252	4363
≥ 37 weeks							
Twins	4 (0.9 to 12)	20 (11 to 33)	24 (14 to 38)	3	14	704	704
Singletons	0.7 (0.5 to 0.9)	4.7 (4.3 to 5.1)	5.4 (4.9 to 5.8)	63	441	93636	94077
Unknown							
Twins	13 (0.3 to 72)	13 (0.3 to 71)	13 (0.3 to 71)	1	1	75	76
Singletons	0 (0 to 1.6)	2.5 (0.9 to 5.5)	2.5 (0.9 to 5.5)	0	6	2371	2377
Total							
Twins	11 (6 to 19)	48 (37 to 61)	57 (45 to 72)	14	60	1236	1258
Singletons	1.0 (0.8 to 1.2)	7.7 (7.2 to 8.3)	8.7 (8.2 to 9.3)	104	788	101271	102059

* Maternities with at least one live birth.
** Maternities with at least one survivor.
† Numerator adjusted for twin maternities affected by both cerebral palsy and cotwin death.
†† Maternities affected by infant deaths.
‡ Maternities affected by cerebral palsy.

being a twin all independently increased the risk of cerebral palsy. The correlations between cerebral palsy and gestational age and fetal growth were not, however, best described as linear.

Gestational age had the strongest influence on outcome, with each additional week in gestation decreasing the risk of cerebral palsy by a factor of 0.76 (95% CI 0.69, 0.82). The risk of cerebral palsy for children born before 33 weeks of gestation was additionally increased by a factor of 2.8 (95% CI 1.1, 6.9). However, small numbers preclude establishing whether the effect of gestational age on risk of cerebral palsy is due to steepening of a linear relation at very low gestational age or is indeed a step effect.

Children with *low fetal growth* were at a fourfold increased risk of cerebral palsy (odds ratio 4.0; 95% CI 2.3, 6.8). For children who were not of low fetal growth, the risk of cerebral palsy was decreased by a factor of 0.82 for each increase in fetal growth of one standard deviation. This protective effect was not, however, significant (P= 0.10).

Being a twin nearly doubled the risk of cerebral palsy (odds ratio 1.9; 95% CI 1.1, 3.4). The risk (odds ratio) of cerebral palsy for twins compared with singletons was 5.6 (95% CI 1.2, 26.2) times greater in babies of low fetal growth, compared with 1.7 (95% CI 0.93, 3.1) times greater for babies who were not of low fetal growth. However, these two effects were not significantly different (P= 0.15) from the combined odds ratio of 1.9.

Table 5 shows cerebral palsy type and distribution for singletons and twins. Fifty three per cent (95% CI 27.8, 77.0) of twins and 29.8% (95% CI 21.2, 39.6) of singletons had asymmetric cerebral palsy, either hemiplegia or asymmetric quadriplegia. Despite an increased proportion of preterm twins a smaller per cent had diplegia. Three out of 17 twins (17.6%; 95% CI 3.8, 43.4) were reported to have porencephalic cysts, compared with four out of 104 singletons (3.8%; 95% CI 1.1, 9.6).

Neither sex distribution nor order of birth affected the risk of cerebral palsy.

Table 5 Numbers (%) of twins and singletons for different distributions and neurological type of cerebral palsy maternities in 1985 and 1986

	Singletons (%) (95% CI)	Twins (%) (95% CI)
Distribution		
Hemiplegia	18 (17.3) (10.6 to 26.0)	5 (29.4) (10.3 to 56.0)
Asymmetric quadriplegia	13 (12.5) (6.8 to 20.4)	4 (23.5) (6.8 to 49.9)
Diplegia	19 (18.3) (11.4 to 27.1)	2 (11.8) (1.5 to 36.4)
Quadriplegia	33 (31.7) (22.9 to 41.6)	5 (29.4) (10.3 to 56.0)
Monoplegia	4 (3.8) (1.1 to 9.6)	0 (0) (0.0 to 19.5)
Not available	17 (16.3) (9.8 to 24.9)	1 (5.9) (0.1 to 28.7)
Type		
Athetoid or ataxia or hypotonia or dystonia alone	8 (7.7) (3.4 to 14.6)	1 (5.9) (0.1 to 28.7)
Mixed but no spasticity	4 (3.8) (1.1 to 9.6)	1 (5.9) (0.1 to 28.7)
Mixed with spasticity	22 (21.2) (13.8 to 30.3)	3 (17.6) (3.8 to 43.4)
Spasticity alone	53 (51.0) (41.0 to 61.0)	11 (64.7) (38.3 to 85.8)
Not available	17 (16.3) (9.8 to 24.9)	1 (5.9) (0.1 to 28.7)

Discussion

The rate of cerebral palsy for NETRHA for all years and 1985 and 1986 is lower for singletons than reported in Western Australia,[4] but similar for both twins and singletons to that reported in California.[5] This paper has largely been confined to the years where both numerator and denominator are of good quality.

Findings in NETRHA confirm the significant increase in the risk of cerebral palsy in twins weighing 2500 g or more.[4-5] They differ from those previously reported by showing an increased risk of cerebral palsy in twins for two of the three low birthweight categories, albeit not at a level of significance.

The need to make adjustments in line with known birthweight distributions of different populations being compared has been suggested for evaluating perinatal mortality of white and black populations in the USA,[13] and in twins and singletons.[14] Analysis which compares rates of cerebral palsy for these adjusted birthweight groups with the equivalent conven-

tional categories for singletons shows an increased risk of cerebral palsy in all weight categories that is significant in two of the three low birthweight categories.

Few other reports have looked at risk of twins for cerebral palsy by their gestational age. When the risk of having one or more twins affected by cerebral palsy is compared with the risk in singleton maternities, an increased risk is shown in all gestational age groups. The relative risk is greatest, and significant, for twins born at or after 37 weeks of gestation, although their absolute rate is lower than that for more immature twins.

Multiple logistic regression analysis confirmed the importance of twinning as a risk factor for cerebral palsy, regardless of weight and gestational age. It has also confirmed that there are independent effects of gestational age and fetal growth. Statistically, however, these three risk factors still only account for a minority of all cases of cerebral palsy seen.

The equal proportion of first and second twins with cerebral palsy in North East Thames is insufficient to discount a real difference in birth order of twins with cerebral palsy if a larger group of twins were studied. If only twins not otherwise at increased risk of cerebral palsy, namely those who have a normal surviving co-twin aged 1 or more, are considered and published data from California[5] and Western Australia[4] are added to our findings, 27 out of 39 cases (69%; 95% CI 52, 83) with cerebral palsy were second born.

It has been suggested that twinning confers an increased risk of cerebral palsy because of increased vulnerability of blood flow in utero.[15] A recent study of hemiplegia in twins found that this condition was notable for the lack of concordance in twin pairs.[16] The excess of cases of asymmetric cerebral palsy in twins in this sample is of interest but numbers are too small to permit meaningful statistical comparisons.

The population attributable risk per cent quantifies the burden of cerebral palsy due to twinning in NETRHA. RICHS data for 1990 show an increased proportion of twin survivors compared with 1980-86 data. If the risk of cerebral palsy in twins and singletons is the same in 1990 as calculated for 1980-86, the increased proportion of twins would have increased the PAR% from 8.5% to 9.1%.

The implications of an increased risk of cerebral palsy or infant death for twins is undeniably important for counselling parents facing the choice of a multiple or singleton pregnancy. The implications for the population of a substantial, and potentially increasing, proportion of cerebral palsy cases from multiple births is important for prevention and service planning. Clear hypotheses about the likely aetiologies of the increased risk of cerebral palsy for twins compared with singletons are hard to disentangle. This could be because the aetiology is multifactorial and confounding factors and sample size are preventing the emergence of clearer patterns. A multicentre study, which has combined data from cerebral palsy registers in Britain, the USA, and Australia, is in progress and may clarify these issues.

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1 Alberman E. Cerebral palsy in twins. *Guys' Hospital Reports* 1964; **113**:285-95.
2 McCarthy BJ, Sachs BP, Layde PM, Burton A, Terry JS, Rochat R. The epidemiology of neonatal death in twins. *Am J Obstet Gynecol* 1981; **141**:252-61.
3 Ghai V, Vidyasagar D. Morbidity and mortality factors in twins. *Clin Perinatol* 1988; **15**:123-57.
4 Petterson B, Nelson KB, Watson L, Stanley F. Twins, triplets, and cerebral palsy in births in Western Australia in the 1980s. *BMJ* 1993; **307**:1239-43.
5 Grether JK, Nelson KB, Cummins SK. Twinning and cerebral palsy: experience in four northern California counties, births 1983 through 1985. *Pediatrics* 1993; **92**:854-8.
6 Levene MI, Wild J, Steer P. Higher multiple births and the modern management of infertility in Britain. *Br J Obstet Gynaecol* 1992; **99**:607-13.
7 OPCS. *The health of our children*. Decennial Supplement, Series DF No. 11, OPCS. London: HMSO, 1995: 71.
8 Mlika A, Alberman E, Evans P. Use of regional child health records to study risk factors for cerebral palsy in multiple births. *J Epidemiol Comm Health* 1993; **47**:420-1.
9 Evans PM, Alberman E. Recording motor defects in children with cerebral palsy. *Dev Med Child Neurol* 1985; **27**:404-6.
10 Information and Statistics Division, Scottish Health Service, Common Services Agency. *Scotland. Birthweight, head circumference and length for gestational age*. Edinburgh: ISD Publication.
11 Buckler JMH, Green M. Birth weight and head circumference standards for English twins. *Arch Dis Child* 1994; **71**:516-21.
12 Dean AG, Dean JA,Coulombier D, Brendal KA, Smith DC, Burton AH, *et al*. Epi Info, Version 6: *A word processing, database, and statistics program for epidemiology on microcomputers*. Atlanta, Georgia: Centers for Disease Control and Prevention, 1994.
13 Wilcox A, Russell I. Why small black infants have a lower mortality rate than small white infants: The case for population specific standards for birth weight. *J Pediatr* 1990; **116**:7-10.
14 Buekens P, Wilcox A. Why do small twins have a lower mortality than small singletons? *Am J Obstet Gynecol* 1993; **168**:937-41.
15 Scheller JM, Nelson KB. Twinning and neurologic morbidity. *Am J Dis Child 1992*;**146**:1110-13.
16 Goodman R, Alberman E. A twin study of congential hemiplegia. *Dev Med Child Neurol* 1996; **38**:3-12.