Archives of Disease in Childhood, 1985, **60**, 1113–1121

Original articles

Increase in cerebral palsy in normal birthweight babies

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SUMMARY A register has been compiled of the 421 children with congenital cerebral palsy born between 1960 and 1975 from a defined geographical area of North East England (population 770 000). There was a fall in the rate of cerebral palsy among very low birthweight babies between 1964 and 1975 and also in the small group with dyskinetic cerebral palsy. The rate rose, however, among babies weighing more than 2·5 kg at birth in the second half of the study, in parallel with changes in perinatal mortality. The net effect is that the overall congenital cerebral palsy rate (mean 1·64 per 1000 livebirths) showed a gradual rise between 1968 and 1975. This conclusion is reinforced by evidence of a rise in incidence among the subgroup of patients with severe cerebral palsy (as defined by an interval measurement of handicap) during the same period.

As perinatal mortality decreases there is an increasing need for an accurate measure of obstetric and neonatal morbidity.[1 2] The cumulative incidence of cerebral palsy has frequently been used for this purpose in the follow up of very low birthweight babies[3] but although well established in Western Australia, Sweden, Denmark, and Ireland, few community studies of cerebral palsy have been undertaken in this country in the past 25 years.[4] Until such studies are undertaken it is difficult to be certain that the results of the hospital based follow up studies of low birthweight babies are not attributable to selective referral, or that modern obstetric and neonatal paediatric practices do not have adverse effects on heavier babies. The results from such population based studies abroad are inconclusive[5] but the most recent work from the long running Swedish study suggests a rise in the incidence of cerebral palsy among babies of all weights since the late 1960s.[6]

A potentially serious source of error in all these studies is the difficulty of ensuring complete ascertainment. A substantial number of children are not diagnosed as having cerebral palsy until they are aged 3 to 4 years,[7] some milder cases resolve,[8] and some 30% may not come to the notice of conventional clinical surveys at all.[1 9] These problems might be minimised if an agreed way were found to identify patients with mild cerebral palsy who are most subject to incomplete ascertainment. Such a measure of severity might also show that seeming falls in prevalence could conceal real rises in the proportion of patients with severe cerebral palsy.

We describe an attempt to create a register of all cases of cerebral palsy among children in part of the North East of England. Those still alive and of school age were graded by severity using a measure of handicap.

Subjects and methods

An attempt was made to identify all cases of cerebral palsy, diagnosed at any time, among the children born between 1960 and 1975 to mothers resident in the health districts of Northumberland, Newcastle, and North Tyneside (total population approximately 770 000). These three districts form a well defined geographical area bounded by the Scottish border, the Pennine Hills, the North Sea, and the River Tyne. There are two university teaching hospitals and three other district general hospitals serving a range of inner city, urban, and rural communities. During the years covered by this survey there were, in addition, between five and

nine other small maternity homes in the area staffed by midwives without resident medical cover.

Information was also collected on cases born within this area to non-resident mothers ('imports'), or who lived in the catchment area at the time of the case reviews (see below). Multiple clinical sources of ascertainment were used (for example hospital activity analysis, paediatricians, the child development centre, community child health services, special schools, etc) as well as reviews of all child residents in local long stay hospitals for the mentally handicapped and searches of childhood death registers. This retrospective register is presently being brought up to date and turned into a continuing survey.

The definition of cerebral palsy used was that of the Little Club,[10 11] and the present paper is concerned only with 'congenital' cases (that is, cases that seemed to be due to a recognised illness after the first 28 days of life are excluded). Children with a primary diagnosis of meningomyelocele were not included, but children with other identifiable congenital syndromes (for example microcephaly, encephalocele, hydrocephalus) were included if they also had a motor disability that otherwise fulfilled the Little Club definition. The classification system used is that of the local developmental paediatrician, Dr E Ellis (Table 1). This is similar to that used in Oxford,[12] and was extended where necessary to that of Mitchell.[13] In common with these authors and others,[11 14] the term 'diplegia' is avoided, but we believe that a direct equivalent of the term as used by Hagberg[15] and Ingram[16] may be arrived at by using our categories 'quadriplegia' and 'paraplegia' in combination. On the other hand, it is clear that this grouping is not equivalent to 'diplegia' as described by Stanley[17] or Nelson and Ellenberg.[18] Moreover, we feel it is not possible to make their suggested distinction between so called 'diplegia' and 'quadriplegia' on the basis of a (subjective) assessment that the 'legs are affected the same as or less than the arms' in a consistent and reliable way.

Table 1 *Cases of congenital cerebral palsy among births to resident mothers 1960–75 (number of deaths shown in brackets)*

Spastic: Quadriplegia	118 (20)
Paraplegia	52 (1)
Hemiplegia	100 (1)
Monoplegia	4
Ataxia	17
Dyskinesia: Athetoid	11
Choreoathetoid	1
Torsion spasms	0
Spastic with dyskinesia	5
Spastic with ataxia	3
Other cerebral palsy	14 (4)
Total	325 (26)

Denominator data, including information on the birthweight of babies weighing less than 2·5 kg at birth, were obtained from annual health authority returns (LHS27/1, SD52) and from SH3 and local hospital statistics for analyses by place of birth. Live births to mothers resident in the catchment area fell from over 14 000 to less than 9000 per annum during the years covered by the survey while the percentage of these with a birthweight of 2·5 kg or less varied in each four year period as follows: 6·6, 6·3, 7·0, and 7·0%. Unfortunately accurate denominator data regarding birthweight are not available for babies of greater than 2·5 kg but as this seemed an important issue an estimate was derived from the birthweight distribution for England and Wales in 1981[19] (data for Newcastle upon Tyne for 1960–9 are virtually identical). For every case in the survey who was born in the catchment area, birthweight and address at birth were checked from contemporary or near contemporary medical records.

A sample of those live and resident cases born between 1964 and 1975 (that is, of school age in 1981) were reviewed to confirm the diagnosis and to assess severity using a measure of handicap. Those called for reviews were firstly a 50% random sample of cases already seen and diagnosed by the local consultant in developmental paediatrics (65%) and secondly all the children not previously assessed by this paediatrician. Diagnostic consistency was therefore attempted by having every child seen by at least one of three doctors from the same assessment unit. An aggregated handicap score was derived from the results of a detailed parental questionnaire and from observations made at interview.[20] During analysis, the handicap scores of the children not previously assessed by the developmental paediatrician were weighted by a factor of 0·5 to allow for the differential sampling. In Figs. 1 and 3 'n' refers to the actual cases for whom results were obtained (from a total sample of 142) while the plots are weighted.

Analyses were performed using the Statistical Package for the Social Sciences. The principal statistical tests used for the comparison of rates were χ^2 and χ^2 for trend.[21]

Results

Case finding. A total of 421 children with cerebral palsy born between 1960 and 1975 were included in the register. Thirty seven were excluded because they were 'imported' births (that is, born within the survey area from addresses outside) and 59 because, though now resident in the area, they had been born

to mothers who were not living in the area when they delivered. This left 325 children with congenital cerebral palsy born to resident mothers in the main series.

An analysis by source of ascertainment showed that over 70% were known to the local child development centre (the single most productive source), and inclusion of information from community health services increased this to 89%. To find the remaining 11% of cases, however, required an extensive search, but all except 3·5% of the total eventually found could have been identified using the three most productive of the subsidiary sources, that is hospital activity analysis computer files, a review of long stay hospital residents, and an analysis of the death registers held by the Office of Population Censuses and Surveys.

Age at diagnosis

Some 98% of the parents of reviewed cases claimed that the diagnosis had been made before the age of 5 years, and as the review was limited to surviving children this is likely to be an underestimate. On the other hand a study of age at first referral to the Child Development Centre among the 23% of children for whom this was the only source of ascertainment showed that more than a quarter of these were 'unavailable for ascertainment' until age 5 years or more (see Fig. 1). In view of this finding it was estimated that 10% of the cases born in 1975 and 5% of those born in 1974 would have been missed by the original search for cases in 1980/81. A repeated sweep of the sources used in 1980/81 in 1985 did indeed show for the first time, as predicted, two of the 20 cases born in 1975 and one of the 25 born in 1974.

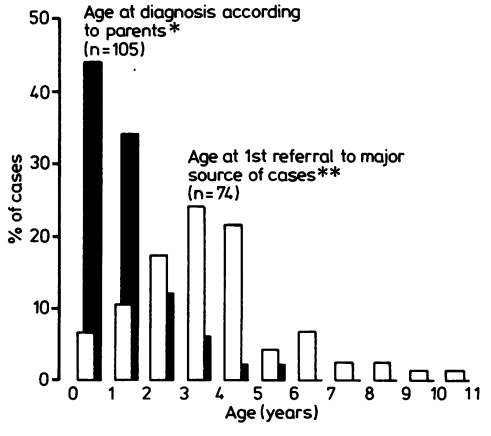

Fig. 1 *Distributions of age at diagnosis and age at referral in children with cerebral palsy.*

*From a review of resident survivors born between 1964 and 1975.
**All cases from main series where the only source of ascertainment was the child development centre.

Sources of error.

Migration

Children aged 0 to 5 years migrate out of the survey catchment area at the rate of about 3% per year (personal communication, Northern Regional Health Authority) and, assuming that there is not selective migration of children before the diagnosis of cerebral palsy, this will have led to an underestimate in all cohorts due to migration before diagnosis of the order of 5 to 12% (depending on which age distribution in Fig. 1 most accurately represents availability for ascertainment). Even though the Child Development Centre serves an area beyond the survey catchment area, only four cases (1·2% of the total) were traced where the diagnosis had in fact been made after migration.

A further potential source of error associated with inward migration was avoided by documenting mothers' addresses at the time of delivery from contemporary records. This led to the reclassification of 37 of the 362 cases originally thought to belong to the main series on the basis of place of birth and current address.

Deaths

The searches of the national death registers are likely to have missed some cases either because cerebral palsy was not coded as the underlying cause of death—only 42% of death certificates mentioning cerebral palsy in England and Wales were so coded in 1976 (personal communication from A Macfarlane) —or because the death was registered outside the catchment area. It would have entailed a very expensive computer search to find these latter cases. In addition, from the information available on the 26 children in the main series who were known to have died, it was estimated that the mortality rate among cases of cerebral palsy by age 7 years was 6% (compared with 11% from the National Child Development Study—1958 cohort).[22]

Unreviewed cases

During review of the children born between 1964 and 1975 who were resident in the area in 1981, 23 of the 165 children seen were reclassified as having no evidence of cerebral palsy or as having cerebral palsy of postnatal origin. This review included all the children whose diagnosis was uncertain, and it is notable that more than three quarters of the misdiagnoses arose among the 12% of potential

cases where the community health service was the only source of ascertainment. As older cases tended to be known to several sources, the resulting overestimate (that is, incorrectly diagnosed cases) remaining in the numerator is not likely to exceed 2% in the unreviewed cohorts (the children born before 1964).

Associated diagnoses

There is dispute in the published reports over the admissibility of children with identifiable congenital syndromes so these cases are listed in Table 2. Exclusion lowers the estimated incidence by a uniform 3 to 4% but does not otherwise alter the conclusions.

Overall error

The net effect of the first three of these potential errors (migration, deaths, and unreviewed cases) is a general undercount of about 8%, principally due to migration before diagnosis. There is no evidence to suggest that this shortfall is selective except that milder cases are known to be diagnosed later.[20] No adjustment is made for this possible error in the following analyses but its likely influence is discussed later.

Trends in rates.

By birthweight

The distribution of cerebral palsy by birthweight is illustrated in Fig. 2. This shows not only the familiar exponential rise in the risk of cerebral palsy as birthweight decreases below 2·5 kg but also that there is a U shaped distribution of cerebral palsy rates among births of 'normal' birthweight (that is, 2·5 kg or more). These increases in rates at both ends of the 'normal' birthweight range are significant, not only for all cerebral palsy ($\chi^2=8·8$) but also

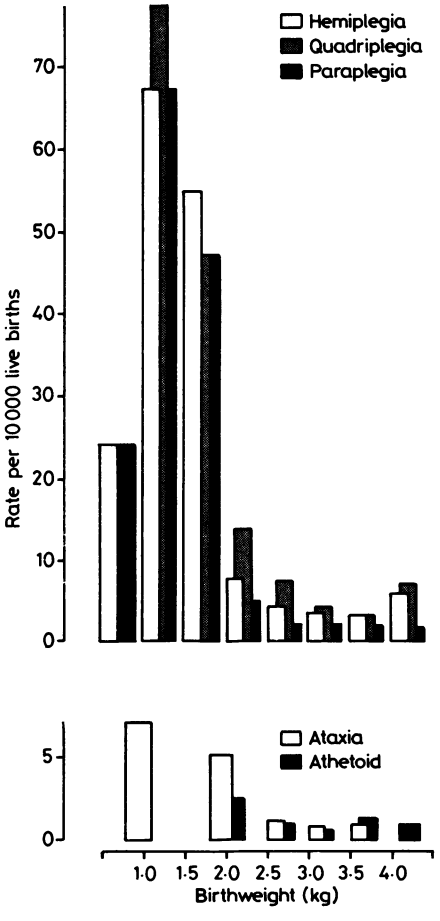

Fig. 2 *Distribution of cerebral palsy by birthweight (1960–75).*

Table 2 *Cases of cerebral palsy with other associated diagnoses*

Year of birth	*Sex*	**Place of birth*	*Birthweight (g)*	*Diagnosis*
1963	F	A	2920	Spastic quadriplegia, hydrocephalus
1964	F	B	3090	L hemiplegia, encephalocele
1965	M	A	4167	R hemiplegia, hydrocephalus, encephalocele
1966	F	B	3203	L hemiplegia, encephalocele
1966	M	B	4026	L hemiplegia, hydrocephalus
1969	M	A	2906	Spastic quadriplegia, hydrocephalus
1971	F	A	3500	Spastic quadriplegia, encephalocele
1971	M	A	3210	Spastic quadriplegia, encephalocele, hydrocephalus
1973	M	B	1913	R hemiplegia, hydrocephalus
1973	F	B	3189	L hemiplegia, hydrocephalus
1974	M	A	1825	Spastic quadriplegia, microcephaly
1974	F	B	2948	L hemiplegia, hydrocephalus

*A=university consultant units; B=other consultant obstetric units.

for quadriplegia (χ^2=10·16—both with 3DF and P<0·05) and make a major absolute contribution to the number of children with cerebral palsy.

By date of birth (Table 3)
Although when analysed in four year sets the increase in the overall incidence rate of congenital cerebral palsy in 1972–5 does not achieve statistical significance, the annual data (Fig. 4) show a rising trend during the second half of the period studied (χ^2=2·96, 0·05<P<0·1). Furthermore, when analysed by birthweight this overall picture disguises sharply contrasting patterns in the low and normal birthweight groups as multivariate analysis would show. In particular, the rate for babies weighing less than 1·5 kg at birth shows a significant reduction since 1964–7, a trend that persists when babies weighing less than 1 kg are excluded and regardless of whether live births or early neonatal survivors are regarded as the denominator. The results of this restricted analysis, however, need to be treated with care as this comparison was not envisaged before data collection was complete. The fall, which is fairly small in numerical terms, is outweighed in the total rates by an increase in congenital cerebral palsy in heavier babies. Here the annual rates, this time for babies weighing more than 2·5 kg at birth, show a significant rising trend between 1968 and 1975 (χ^2=4·56 P<0·05). Lastly the analysis by birthweight within this 'normal' birthweight group (using extrapolated denominators—see Methods) suggests, not only that this rise in rates is principally confined to those babies weighing between 2·5 and 4 kg at birth, but also that there has been a significant fall in rates at the higher extreme of birthweight (that is in babies of more than 4 kg at birth).

By type of cerebral palsy (Table 4)
The only type of cerebral palsy to show a significant change in incidence over the study period is the relatively rare dyskinetic form. Table 5, however, shows that the changes in the birthweight specific incidence of cerebral palsy seen in Table 3 are largely due to changes in the corresponding rates for 'diplegia'. Furthermore the rate of diplegia for children of normal birthweight shows a significant upward trend between 1964 and 1975.

By place of birth (Table 6)
Cases were divided into four groups—babies born in university (A) and non-university (B) hospitals with resident obstetric staff, domiciliary births (C), and births in hospitals without resident obstetric staff (D). Births to non-resident mothers ('imports') were included in these analyses because denominator statistics (SH3 returns) relate to place of birth rather

Table 3 *Incidence of cerebral palsy by birthweight (rate per 10 000 livebirths (no))*

Birthweight		*1960–63*	*1964–67*	*1968–71*	*1972–75*	χ^2 *(trend) 1964–75*
<1·5 kg		111 (5)	317 (12)	168 (7)	35 (1)	↓ **
<1·5 kg†		338	839	288	64	↓ ***
1·5–2·5 kg		62·8 (21)	50·4 (15)	77·1 (22)	73·5 (18)	ns
2·5–4·0 kg		11·5 (57)	10·7 (49)	10·3 (41)	15·3 (51)	ns
>4·0 kg		25·1 (11)	19·7 (8)	8·5 (3)	6·8 (2)	↓ *‡
All births		16·3 (94)	16·1 (86)	15·6 (73)	18·4 (72)	ns
Scpr§	(SEM)	100 (10)	100 (11)	94 (11)	111 (12)	

Statistical significance *P< 0·05, **P<0·01, ***P<0·001.
†Using early neonatal survivors as the denominator.
‡χ^2 for 1960–67 *v* 1968–75.
§Scpr=birthweight standardised cerebral palsy rate (indirect)[20] 100=1969–75 resident births.
The denominators may be obtained from the corresponding author.

Table 4 *Incidence by type of cerebral palsy† (rate per 10 000 livebirths (no))*

Diagnosis	*1960–63*	*1964–67*	*1968–71*	*1972–75*	χ^2
Quadriplegia	5·0 (29)	5·4 (29)	6·8 (32)	7·3 (28)	ns
Paraplegia	3·5 (20)	2·1 (11)	1·7 (8)	3·3 (13)	ns
Right hemiplegia	2·9 (17)	2·8 (15)	3·2 (15)	3·3 (13)	ns
Left hemiplegia	1·7 (10)	2·4 (13)	1·5 (7)	2·0 (8)	ns
Ataxia	1·2 (7)	1·3 (7)	0·4 (2)	1·0 (4)	ns
Dyskinesia‡	0·9 (5)	1·9 (10)	0·2 (1)	0·3 (1)	↓ *§

*Statistical significance P<0·05.
†Hemiplegias NOS (2), monoplegias (4), and other CP(14) excluded—mixed forms included with ataxia or dyskinesia (see Table 1).
‡These 17 cases include 5 with mixed spasticity and athetosis as well 1 child with choreoathetosis.
§χ^2 for 1960–67 *v* 1968–75.

Table 5 *Incidence of diplegia† by birthweight (rate per 10 000 livebirths (no))*

Birthweight	*1960–63*	*1964–67*	*1968–71*	*1972–75*	χ^2 *(trend) 1964–75*
All weights	8·5 (49)	7·9 (42)	8·8 (41)	10·5 (41)	ns
<1·5 kg	88·9 (4)	185 (7)	144 (6)	0 (0)	↓ **
<1·5 kg‡	270	490	247	0	↓ **
1·5–2·5 kg	41·9 (14)	36·9 (11)	31·5 (9)	40·8 (10)	ns
2·5–4·0 kg	5·5 (27)	4·1 (19)	6·0 (24)	9·0 (30)	↑ **
>4·0 kg	9·1 (4)	12·3 (5)	5·6 (2)	3·4 (1)	ns

Statistical significance **P< 0·01.
†Paraplegia+quadriplegia+spastic ataxia.
‡Using early neonatal survivors as the denominator.

Table 6 *Incidence of cerebral palsy by place of birth (rate per 10 000 livebirths (no))*

Place of birth	*1960–63*	*1964–67*	*1968–71*	*1972–75*	*1960–75*	χ^2
A University consultant units	24·2 (38)	23·4 (40)	22·0 (48)	18·3 (40)	21·7 (166)*	ns
(A+B−'imports')	19·7 (52)	20·7 (57)	17·6 (59)	19·2 (63)	19·2 (231)	ns
B Other consultant units	20·3 (26)	18·2 (27)	12·3 (22)	15·7 (26)	16·3 (101)	ns
C Home deliveries	16·5 (26)	12·6 (11)	9·9 (2)	— (0)†	14·5 (39)*	ns
D Units without resident staff	10·4 (16)	10·1 (17)	10·8 (12)	13·7 (8)	10·8 (53)	ns

*Scpr 1960–75 (see Table 3 legend) group A=126 (9); group C=94 (16).
†Less than one case 'expected' (318 livebirths only).

than to home address. Within these groups there were no significant changes over time in the overall incidence of cerebral palsy even when this was standardised for variations in birthweight distribution (where this information was available), and even though the proportion delivered in hospitals with resident obstetric staff rose from 45% of all births in 1960 to more than 90% in 1975.

On the other hand it is noticeable that the rate of cerebral palsy is highest among births in the university teaching centres (A) and lowest among those in units without resident obstetric staff (D). These differences by place of birth show a highly significant trend (group A *v* B *v* C *v* D χ^2 for trend=21·79 P<0·001) which is not substantially changed by abstracting imported births (A+B−imports *v* C *v* D χ^2 for trend=15·69 P<0·001). Standardisation for birthweight distribution (where available) seems to moderate these differences but the group A rate still remains significantly raised.

Handicap. A total of 142 school age children with congenital cerebral palsy were reviewed as part of a study to determine the extent of their handicaps and disabilities. A further five who were eligible for review were ascertained too late for study. As explained in Methods section, 71 of these children were a 50% random sample and the following analyses are weighted to allow for this. The results are therefore considered to be representative of all the 220 live resident cases of congenital cerebral palsy born between 1964 and 1975. No attempt was

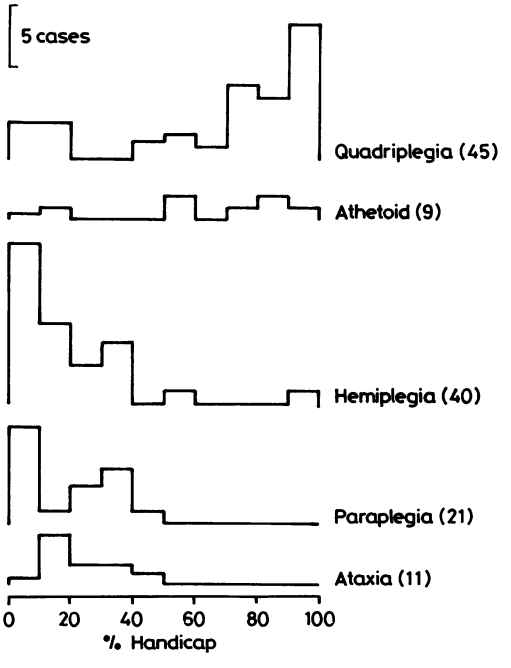

Fig. 3 *Patterns of handicap associated with different types of cerebral palsy 1964–75.*

A single measure of overall handicap[20] was assigned to each child by the weighted aggregation of 72 separate items scored at review (both by direct observation and by parental questionnaire).

made to trace and review children who were no longer in full time education.

By type of cerebral palsy
The distribution of handicap scores is illustrated in Fig. 3. The patterns vary from the typically more 'severe' quadriplegic form of cerebral palsy through to the 'milder' paraplegic and ataxic varieties.

By date of birth
The distribution of handicap scores in the reviewed sample has been applied to the incidence rates of survivors from the 1964 to 1975 resident birth cohorts (Fig. 4). The extrapolation assumes that the reviewed (resident) cases do not differ from the original cohort survivors and as 85% of the two samples are in common, assumes in effect that 'migrants in' are similar to 'migrants out' with respect to handicap distribution. This analysis shows an almost threefold rise in the rates of severe cerebral palsy (that is, greater than 90% handicap) between the third and fourth four year periods which could account for virtually all the change in overall cerebral palsy rate during the same period (Table 7).

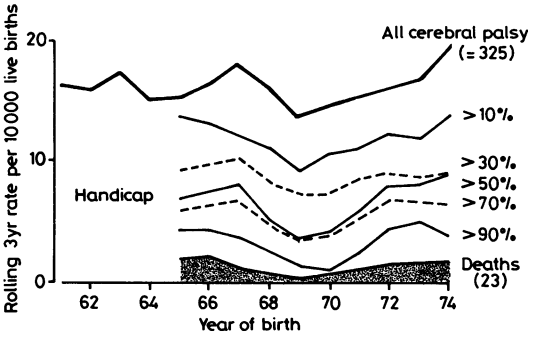

Fig. 4 *Incidence of cerebral palsy 1960–75 showing the effect on the cerebral palsy rate of using differing 'severity' thresholds.*

The distribution of handicap among the survivors is extrapolated from the reviews of a sample of resident children (see text). Children born before 1964 were not reviewed.

Discussion

Survey accuracy. Several potential sources of error have been explored, each of which could lead to serious inaccuracy in ascertainment in surveys of this type. Difficulties in case finding underline the need for multiple sources of ascertainment in clinical surveys. Surveys which rely on a single source must nearly always be considered unreliable.[26] Late diagnosis is known to be a problem[7] and even where the youngest children are aged 5 years, the distribution of age at which the child becomes 'available for ascertainment' may lead to an important shortfall. The combination of this effect with migration, however, is the more serious problem and could lead to a 5 to 12% undercount in all cohorts even in an area of relatively slow population movement. Stanley[17] paid considerable attention to this problem but the issue seems to have been ignored in most other surveys.

Unless cases referred to registers are reviewed then there is also a risk of diagnostic inaccuracy especially where high yield, low specificity agencies (for example community health services) are used. Despite this, some registers continue to operate without case reviews. At the same time it is worth noting the difficulty in relating cases clearly to the correct denominator unless the address of the mother at birth is firmly established. This requires a search of birth records for all cases. If place of birth or current address, or both are used, serious over ascertainment may result.

Apparent variations in the distribution of handicap over time may be artefactual (for example due to an age related effect), may be real (although there does not seem to be any a priori reason why this should occur, at least not within diagnostic type), or may reflect variations in the ascertainment threshold which can effectively disguise a true change in cerebral palsy rates.

A persistent characteristic of these potential

Table 7 *Incidence of cerebral palsy by severity of handicap† (rate per 10 000 livebirths (approximate no))*

Handicap	*1960–63*	*1964–67*	*1968–71*	*1972–75*	χ^2
All grades	16·3 (94)	16·1 (86)	15·6 (73)	18·4 (72)	ns
Handicapped > 10%	— ( )	13·1 (70)	10·1 (51)	13·2 (52)	ns
> 30%	— ( )	9·5 (51)	7·6 (35)	9·1 (35)	ns
> 50%	— ( )	6·8 (36)	5·1 (24)	8·7 (34)	ns
> 70%	— ( )	5·9 (31)	4·4 (21)	6·6 (26)	ns
> 90%	— ( )	3·9 (21)	1·7 (8)	4·6 (18)	*

Statistical significance *P<0·05.
†Distribution of handicap among survivors extrapolated from study of a sample of residents (see text)—actual deaths are added to the predicted number of survivors with more than 90% handicap.

errors (low specificity sources of ascertainment, late diagnosis, diagnostic inaccuracy, and variations in ascertainment threshold) is that they mostly affect milder cases, and it is for this reason that future sürveys should attempt to overcome the problem of grading severity. Earlier results from the present survey suggest that a combination of simple disability tests may suffice to categorise cases of cerebral palsy by severity.[20]

Evidence exists from other studies to support the notion of a major untapped pool of mild cerebral palsy in the community.[1 9] In the present study, the ordering of the cases by degree of handicap suggests that this under ascertainment will principally affect hemiplegia, paraplegia, and ataxia, and one might speculate that close examination of 'normal' schoolchildren might lead to a substantial increase in the ascertainment of these forms of cerebral palsy. On the other hand it is not considered likely that these errors will have influenced the results of analyses by birthweight or by place of birth except insofar as a generalised undercount tends to minimise statistical power.

Trends in cerebral palsy rates. The falling incidence of cerebral palsy in very low birthweight babies during the period of study is in line with previous hospital based studies.[3] It is notable, however, that for the same period the population based rates are about half those from these 'centres of excellence', confirming the selection bias that attends such studies. Hagberg *et al* have found similar trends in low birthweight specific cerebral palsy rates (though Swedish rates seem to have risen again in the early 1970s).[6 15] A further interesting finding is a seemingly significant decrease in cerebral palsy among very heavy babies which does not seem to have been reported previously.

Meanwhile, for babies of 2·5 to 4 kg there is an important rise in the congenital cerebral palsy rate during the period of the study, and, since these babies account for about two thirds of all cases, this trend dominates the overall rate. A further point concerning the birthweight specific rates for babies weighing over 2·5 kg is that there is a remarkable coincidence here with a rise between 1968 and 1975 in the local perinatal mortality rate for this birthweight group.

When the figures for the whole period are analysed by birthweight there is seen to be a marked U shaped distribution of rates above 2·5 kg and the overall impression is of an exponential rise in the risk of cerebral palsy as birthweight deviates from the population mean. There is no sign of the bimodal pattern seen in raw frequency distributions by birthweight;[23] it may be that the bimodal pattern is no more than an artefact due to the combination of an exponential rate change with a normally distributed denominator.

The seeming decline in the frequency of dyskinetic cerebral palsy is unlikely to be due to incomplete ascertainment as dyskinetic children are usually severely handicapped. This trend has been noted by others,[15 17] and is probably linked to the decline in the incidence of haemolytic disease.[24] The diplegias seem to make an important contribution both to the rise in overall rates for normal birthweight cases and also to the fall in rates for those weighing 1·5 kg or less at birth. Paraplegia, a typically mild form of cerebral palsy, accounts for about 30% of 'diplegia', however, and there may therefore be some doubt about the completeness with which these cases have been identified.

The results of analyses by place of birth seem to parallel those from similar analyses of perinatal mortality.[25] Babies born at home and in small maternity units without resident obstetric staff have lower cerebral palsy rates than babies born in consultant units (especially teaching hospitals) even when 'imported' births from outside the survey area are discounted and differences in birthweight distribution are allowed for.

The overall result is that there has been a rise in the incidence of congenital cerebral palsy in the latter half of the study period (that is, between 1968 and 1975). This conclusion is reinforced by the trend among the most severely handicapped children (that is, among those least likely to be affected by diagnostic and ascertainment errors) during the same period. This rise is taking place primarily among children of normal birthweight, outweighing the effect of the fall in the rate among very low birthweight babies and the virtual disappearance of dyskinetic cerebral palsy.

We are indebted to Dr E Ellis for his support and guidance and to the North East branch of the Spastics Society for funding the establishment of the Cerebral Palsy Register. Actual denominators may be obtained from the corresponding author.

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Received 5 August 1985