

Cerebral palsy and socioeconomic status: a retrospective cohort study

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Aims: To study the relation between risk of cerebral palsy and socioeconomic status.

Methods: A total of 293 children with a diagnosis of cerebral palsy out of 105 760 live births between 1 January 1982 and 31 December 1997 were identified from the special conditions sub-file of the West Sussex Computerised Child Health System.

Results: There was a linear association between risk of cerebral palsy and socioeconomic status (SES) measured by the Registrar General's social class (RGSC) and enumeration district (ED) ranked into quintiles by the Townsend Deprivation Index derived from 1991 census data. Fifty one per cent and 30% of cases of cerebral palsy were statistically "attributable" to inequality in SES using the RGSC and ED quintile respectively. A linear association was also noted for singleton live births. The association between risk of cerebral palsy and ED quintile persisted in a logistic regression model that included birth weight and gestational age, although that between RGSC and cerebral palsy no longer reached conventional levels of statistical significance after adjustment.

Conclusions: A strong association was observed between socioeconomic status and the risk of cerebral palsy, which was only partly accounted for by the known social gradients in birth weight and gestational age.

The negative impact of socioeconomic deprivation has been shown for many aspects of child mortality and morbidity.^{1–3} The relation between socioeconomic status (SES) and the risk of cerebral palsy is not clear. An observed association between SES and cerebral palsy might suggest aetiological factors and pathways to prevention as well as being important in the planning of services and the allocation of resources.

Low birth weight and prematurity are the strongest risk factors for cerebral palsy.⁴ Given the observed association between these factors and SES,^{5–7} an increased prevalence of cerebral palsy with low SES is expected. However, studies have produced conflicting results. Table 1 summarises the results of studies conducted in developed countries. In developing countries, there is a higher proportion of children with postnatally acquired cerebral palsy with a more clearly established socioeconomic gradient.

West Sussex has a population of 750 000 with roughly 8000 births per year. Compared with national figures, fewer belong to ethnic minority groups and there is a lower rate of unemployment. However, there is a wide variation in SES with areas of deprivation.⁸

In the present study, population based, routinely collected data from West Sussex are used to:

- Examine the relation between the prevalence of cerebral palsy and SES and between SES and the type and severity of cerebral palsy
- Examine the degree to which any relation between SES and the prevalence of cerebral palsy is mediated by socioeconomic differences in birth weight and gestation.

METHODS

Case ascertainment and data collection

The study was undertaken using the computerised Child Health System in West Sussex. The study sample comprised all live births to mothers resident in West Sussex between

1 January 1982 and 31 December 1997. A Child Health Record was routinely generated using information recorded concurrently by midwives and in the birth registration. Birth weight, gestation, plurality, Registrar General's social class at birth (based on father's occupation), and current postcode were entered onto a study database. Current postcodes were converted to enumeration districts (100–150 households) using the Office of National Statistics 1991 Enumeration District/Postcode Directory⁹ and ranked in quintiles on the basis of Townsend Deprivation Indices (TDI) calculated using 1991 Census data.¹⁰

Children with cerebral palsy were identified from the Special Conditions sub-file.¹¹ Index children were those who were recorded as having a diagnosis of cerebral palsy confirmed at 2 years of age or older or who had died under the age of 2 and who were recorded as having a prior diagnosis of cerebral palsy, irrespective of whether the cerebral palsy was judged to have been acquired pre- or postnatally. All children diagnosed as having cerebral palsy had been seen by paediatricians in the Community Child Health Service who routinely recorded the type and severity of cerebral palsy. The Special Conditions sub-file is updated after each contact between the child and the family. The diagnosis of cerebral palsy and the recording of type (classified as hemiplegia, diplegia, quadriplegia, and other) and severity (mild, moderate, and severe, based on the degree of functional impairment at last visit) was made on clinical grounds and not on the basis of standardised criteria.

Analysis

The prevalence of cerebral palsy was calculated for all live births, separately for singleton and multiple births and for birth weight (<1500, 1500–2500, >2500 g) and gestational age (<37, 37–40, >40 weeks) groups according to RGSC and

Abbreviations: ED, enumeration district; RGSC, Registrar General's social class; SES, socioeconomic status; TDI, Townsend Deprivation Indices

Table 1 Cerebral palsy and socioeconomic status literature

Study	Type	Comparison	SES measure	Results
Dowding Ireland 1976–81 ¹³	Retrospective population based study	CP v live births	RGSC	Increased risk among disadvantaged
Stanley Australia 1966–75 ¹⁴	Retrospective population based study	CP v total population	Father's occupation	Increased risk among disadvantaged
Nelson USA 1959–66 ¹⁵	Prospective cohort study	CP v non CP	Score based on occupation and income	No association
Emond UK 1958 and 1970 ¹⁶	Prospective study of 2 cohorts— nested case-control	CP v matched controls	RGSC	No association
Lagergren Sweden 1969–70 ¹⁷	Retrospective population based study	CP v total Swedish population	Social group	Highest prevalence in highest social group
Dolk N. Ireland 1977–89 ¹⁸	Retrospective population based study	CP v births	Electoral ward quintiles using TDI	No association
Dolk UK 1984–90 ¹⁹	Retrospective population based study	CP v births CP at age 5 v 1–7 year olds	Electoral ward quintiles using Carstairs Index	Increased risk in disadvantaged

ED quintile using SPSS for Windows.¹² The prevalence of each type and grade of severity was also recorded. A χ^2 test for linear trend was used to examine the association between prevalence of cerebral palsy and SES. Odds ratios were determined for each RGSC and ED quintile using the least deprived social class and quintile as references. Separate logistic regression analyses were performed among singleton births including birth weight, gestational age, and either RGSC or ED quintile as independent variables.

The proportion of cerebral palsy statistically “attributable” to social inequality was estimated by comparing the overall population risk of cerebral palsy with the risk that would have been found if the risk in the least disadvantaged groups (for both RGSC and ED quintile) applied to the whole population.

Only children of families resident in West Sussex at the time of the child's birth were included in the study. Of these children, those born out of the county were a high risk group (25% low birth weight, 26% preterm, 7% multiple births) with a prevalence of cerebral palsy of 13 per 1000 live births. RGSC, taken from birth registration details, was not available for the majority of these children as their births were registered outside West Sussex and therefore these children were omitted from the social class analyses. They were, however, included in the ED quintile analysis. All children born in West Sussex had RGSC recorded. An RGSC was recorded as unclassified for many of the most deprived families, including families with single mothers or unemployed parents, and therefore this group was included in the analysis. Children were excluded from further analysis using birth weight and gestation if this additional data was missing.

RESULTS

A total of 293 children were identified as having cerebral palsy out of 105 760 live births in West Sussex between 1 January 1982 and 31 December 1997, an overall prevalence of cerebral palsy of 2.77 per 1000 live births. Data on birth weight were missing for 58 (0.05%) births and on gestation for 148 (0.14%) births. Three per cent of live births were multiple births, 6.1% low birth weight (<2500 g), and 5.9% preterm. The prevalence of cerebral palsy in singleton births was 2.5/1000 and 12.1/1000 in multiple births. The prevalence of cerebral palsy per 1000 live births increased from 1.7 in

births greater than 2500 g to 12.7 in those with birth weight 1500–2500 g and 64.8 in those with birth weight less than 1500 g. Forty four per cent of children with cerebral palsy were reported to have hemiplegia, 17% quadriplegia, and 33% diplegia.

Registrar General's social class was recorded for 238 (81.2%) children with cerebral palsy and 101 106 (95.9%) without cerebral palsy. Table 2 shows the prevalence of cerebral palsy in singleton births for each social class. Compared with children in social class 1, the odds of having a child with cerebral palsy in social class 5 is 2.57 (95% CI 1.27 to 5.21) and in the unclassified group is 2.77 (95% CI 1.41 to 5.44). The increased odds ratios were observed not only in the most deprived groups but in all groups when compared with the least deprived group.

Postcodes relating to EDs with census data were available for 280 (95.6%) children with cerebral palsy and 100 990 (95.8%) live births who did not develop cerebral palsy. Table 3 shows the prevalence of cerebral palsy in singleton births for each ED quintile. The odds of having a child with cerebral palsy in the most deprived quintile is 1.65 (95% CI 1.14 to 2.39) compared with the odds in the least deprived quintile.

A statistically significant linear association was observed between risk of cerebral palsy and SES for total live births measured by RGSC (χ^2 for linear trend = 5.32, $p = 0.021$) and ED quintile (χ^2 for linear trend = 5.41, $p = 0.020$). When only singleton births were included the linear trend persisted for both RGSC (χ^2 for linear trend = 5.92, $p = 0.015$) and ED quintile (χ^2 for linear trend = 7.57, $p = 0.006$). In infants born at term, those with birth weight greater than 2500 g, and those with quadriplegia, a statistically significant relation was seen for RGSC but not for ED quintile. A linear gradient was observed for the prevalence of hemiplegia, diplegia, and “mild” cerebral palsy with ED quintile but not RGSC. No gradient was noted in the low birth weight group.

After adjustment for known risk factors of birth weight and gestation in the logistic regression analyses, the relation between the risk of cerebral palsy and RGSC no longer reached conventional levels of statistical significance ($p = 0.160$). However, the association between the risk of cerebral palsy and SES measured by ED quintile remained statistically significant ($p = 0.046$). The adjusted odds ratios for each class and ED quintile are given in tables 2 and 3.

Table 2 Prevalence and odds ratios of cerebral palsy in RGSC groups (singleton births)

	% of total singleton births	Children with CP	Total children	Prevalence/1000	OR (95% CI) (RGSC 1 as reference)	OR adjusted for BW and gestation (95% CI)
RGSC 1	14.6	16	15018	1.1	1.00	1.00
RGSC 2	20.4	48	20974	2.3	2.15 (1.22 to 3.78)	2.09 (1.19 to 3.69)
RGSC 3NM	13.6	35	13915	2.5	2.36 (1.31 to 4.26)	2.25 (1.24 to 4.06)
RGSC 3M	29.9	68	30649	2.2	2.08 (1.21 to 3.59)	1.89 (1.09 to 3.26)
RGSC 4	6.2	13	6382	2.0	1.91 (0.92 to 3.98)	1.71 (0.82 to 3.56)
RGSC 5	5.3	15	5478	2.7	2.57 (1.27 to 5.21)	2.11 (1.04 to 4.30)
Unclassified	6.0	18	6112	2.9	2.77 (1.41 to 5.44)	2.15 (1.08 to 4.27)
RGSC missing	4.0	42	4081			
χ^2 for linear trend					5.9 (p=0.02)	
					4.2 (p=0.04) if unclassified group excluded	

Overall 51% and 30% of cases of cerebral palsy were statistically "attributable" to inequality in SES using RGSC and ED quintile respectively.

DISCUSSION

These results show a substantial, statistically significant gradient in the prevalence of cerebral palsy by SES measured using either the RGSC classification or an area based measure. They suggest that, even in this relatively affluent area, 30–51% of cases of cerebral palsy are statistically "attributable" to socioeconomic inequality. These proportions are not directly comparable given the omission of a substantial group of children with cerebral palsy from the analyses of RGSC due to missing data on children born out of the area. The same strong relation is found when the analysis is restricted to singleton births. Whichever measure of SES is employed, the prevalence of cerebral palsy is higher in all groups compared to the most advantaged, not merely in the poorest groups.

Previous studies using measures of SES based on parental occupation^{13–17} or an area based measure of SES^{18, 19} have produced conflicting results (table 1). In some studies the apparent lack of an association with lower SES may be related to study methodology. Emond and colleagues¹⁶ reported a case-control study nested in two population cohort studies (the UK 1958 and 1970 birth cohorts). Children in the control group used to examine for SES effects were matched for birth weight. As at least part of the relation between SES and cerebral palsy is likely to be mediated through birth weight, this control group would inevitably underestimate any association. Lagergren¹⁷ compared the percentage of children with cerebral palsy in the Malmöhus area of Sweden by social group with the percentage of the whole population in Sweden in these groups, and reported an increased risk in the most advantaged group. This comparison may have caused bias as the relation between the distribution of social groups in Malmöhus and the rest of Sweden is not clear. The area based measure of SES employed in the studies by Dolk

and colleagues^{18, 19} is derived from ward of residence rather than enumeration district. Wards are relatively large, socially heterogeneous areas, which leads to a risk of non-differential misclassification of SES and hence a bias towards the null. A significant gradient was shown in the Oxford, UK study¹⁹ but not in the Northern Ireland study.¹⁸

The aetiological mechanisms through which the relation between SES and the risk of cerebral palsy are mediated are complex.⁴ The well documented relations between SES and low birth weight and prematurity are likely to be at least partly responsible. However, in this study the association between SES measured by ED quintile and the risk of cerebral palsy remained statistically significant even after adjustment for birth weight and gestation. The association between RGSC and the risk of cerebral palsy failed to reach conventional levels of statistical significance after adjustment. However, apart from social class IV, the confidence intervals of adjusted odds ratios for each class compared to social class 1 excluded 1 (table 2). This suggests that pathways other than that mediated via prematurity and low birth weight are also implicated.

A major strength of this study is that it is based on prospectively collected, population based data. A comparison of the Child Health Computing system and the Cerebral Palsy Register in Northern Ireland found that neither identified all children with cerebral palsy but that the register using multiple sources for case ascertainment and a standardised assessment was more complete.²⁰ The computerised health system in West Sussex utilises active case ascertainment through the community paediatric service. Although assessment of children is not carried out according to a formalised system, paediatricians regularly examine all children identified as having cerebral palsy and the diagnoses are updated each year. The reported prevalence is high compared to other studies⁴ and may reflect some variability in diagnostic boundaries. However, as the comparisons are internal (between SES groups with a common method of diagnosis) bias should not have occurred.

Table 3 Prevalence and odds ratios of cerebral palsy in ED quintiles (singleton births)

	% of total singleton births	Children with CP	Total children	Prevalence/1000	OR (95% CI) (quintile 1 as reference)	OR adjusted for BW and gestation (95% CI)
Quintile 1	19.2	34	19691	1.7	1.00	1.00
Quintile 2	17.1	41	17563	2.3	1.25 (0.82 to 1.91)	1.39 (0.91 to 2.14)
Quintile 3	17.0	45	17428	2.6	1.38 (0.91 to 2.09)	1.55 (1.02 to 2.35)
Quintile 4	18.8	49	19330	2.5	1.35 (0.90 to 2.03)	1.39 (0.92 to 2.10)
Quintile 5	23.6	75	24255	3.1	1.65 (1.14 to 2.39)	1.55 (1.06 to 2.25)
Quintile missing	4.2	11	4342			
χ^2 for linear trend					7.57 (p=0.006)	

A diagnosis of cerebral palsy cannot be made reliably before the age of 2 years.²¹ In the current study, therefore, a diagnosis of cerebral palsy retained after the age of 2 was accepted. The study does not include children who may have had cerebral palsy but were either stillborn or died before a diagnosis could be made, but does include those in whom a diagnosis was made prior to death or at postmortem examination. All cases of cerebral palsy were included, irrespective of the timing of onset or the underlying aetiology, as it was not possible to analyse separately those who acquired cerebral palsy postnatally. Other studies have reported that in developed countries these cases comprise only 5–17% of the total.⁴ Current postcode is used as a proxy for postcode at birth and may reflect deterioration in SES as a consequence of the cerebral palsy. However, the ED results are consistent with analyses using social class, which are taken at birth.

Although the overall prevalence of cerebral palsy is low, it is the commonest cause of chronic motor disorder in childhood. The association with SES status reported here does not establish a direct causal relation between SES and the risk of cerebral palsy. However, it does suggest that some of the aetiologies for cerebral palsy may be socioeconomically mediated. While tackling poverty and its consequences should not depend on the elucidation of aetiological pathways, researchers should consider whether the social patterns of morbidity can provide aetiological clues. The adequate investigation of these mechanisms will require the use of structural pathways that take account of both proximal and distal antecedents of cerebral palsy.³

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