

## Birthweight specific trends in cerebral palsy

P O D Pharoah, T Cooke, R W I Cooke, L Rosenbloom

### Abstract

**A register of infants with cerebral palsy born to mothers resident in the Mersey region from 1967-84 has been maintained using various sources of information. A total of 1056 patients are registered of whom 331 (31%) have hemiplegia or mixed hemiplegia, 236 (22%) have diplegias or mixed diplegia, and 369 (35%) have quadriplegia or mixed quadriplegia. The remainder have dyskinetic or dystonic forms except for seven, who are unclassified. There has been no significant change in the prevalence of cerebral palsy among infants of normal birth weight (>2500 g). Among low birthweight infants (≤2500 g) there has been a significant increase in prevalence of all the main clinical types. This increase started later among the very low birthweight infants (≤1500 g) than among those weighing 1501-2500 g. These changes in prevalence could be the result of either improved survival of prenatally impaired infants because of improvements in medical care, or a reflection of failure to maintain optimal conditions at or around the time of birth.**

Infants of low birth weight (≤2500 g) are a group at high risk for both neonatal morbidity and death. Major impairments found in the survivors include mental retardation, sensory deficits, hydrocephalus, and cerebral palsy; 25-40% of all cases of cerebral palsy are among the 6-7% of infants who are of low birth weight.<sup>1-6</sup> During the past two decades there has been an appreciable increase in the provision of services for neonatal special and intensive care, and in the admission rates to these units.<sup>7</sup> There has been a coincidental increase in the rate of decline in neonatal mortality, and this has been accompanied by concerns that an increasing number or proportion (or both) of survivors are impaired as a result of these developments.

Several centres have established registers to examine trends in the prevalence of cerebral palsy and, in particular, birthweight specific trends. The periods covered by these registers vary and the birthweight specific trends have not been consistent.<sup>4 8-11</sup> Using a register of patients with cerebral palsy in the Mersey region we have previously reported a significant increase in cerebral palsy among low birthweight infants during the period 1967-77. In contrast, the rate of cerebral palsy among infants of birth weights of >2500 g was unchanged.<sup>12</sup> We now report an extension of the previous study up to 1984 and, in particular,

examine the trends in prevalence in low birthweight infants, subdivided into very low birthweight (≤1500 g) and low birth weight (1501-2500 g).

### Patients and methods

The sources of data used for the compilation of the register have been described previously.<sup>12</sup> Cerebral palsy was defined as: 'a group of disorders of movements and posture due to a defect or lesion of the immature brain'.<sup>13</sup> A child was included if the damage to the brain was judged to have occurred before the 28th postnatal day. Inclusion was also strictly determined by the geographical criterion that the mother was resident within the Mersey region at the time of birth. Thus infants who moved out of the region after birth were included, but those who moved into the region were excluded. This criterion was applied because it could be related to denominator populations that were birthweight specific, and for which data were collected through the standard birth notification procedure. There have been no boundary changes since the 1974 health service reorganisation so that trends since that time are unbiased by anomalies in the numerator/denominator populations.

Classification of the type of cerebral palsy was based on information in the clinical records of each child. Cases for whom the information was equivocal were reviewed by one of us (LR).

The analysis was concerned predominantly with three categories of cerebral palsy: diplegia, hemiplegia, and quadriplegia. Cases with symmetrical or nearly symmetrical spasticity of the legs with lesser impairment of either arm were designated as having spastic diplegia; these included the mixed diplegias—that is, those also having dyskinetic or ataxic features. Cases having unilateral spasticity of an arm and leg were designated as having hemiplegia, and included the mixed hemiplegias. Those with arms and legs equally affected, or with deficits greater in the arms, were designated as having spastic quadriplegia, and included the mixed quadriplegias. A fourth category labelled 'others' included the athetoid, ataxic, or dystonic forms.

### Results

The study population comprised 1056 patients with cerebral palsy born in 1967-84; 100 of the 331 with hemiplegia (30%), 152 of the 236 with diplegia (64%), and 115 of the 369 with quadriplegia (31%) weighed 2500 g or less at birth.

Department of  
Public Health,  
University of Liverpool,  
PO Box 147,  
Liverpool L69 3BX  
P O D Pharoah  
T Cooke

Department of  
Child Health,  
University of Liverpool  
R W I Cooke  
L Rosenbloom

Correspondence to:  
Professor Pharoah.

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Trends in birthweight specific numbers of live births, neonatal survivors, and cerebral palsy by clinical category

Year	Birth weight (g)	No of live births	No of neonatal survivors	No of hemiplegias	No of diplegias	No of quadriplegias	No of other cerebral palsies	All cerebral palsies
1967:	≤1500	353	118	1	0	3	0	4
	1501-2500	2657	2491	1	5	2	2	10
	>2500	41148	40373	5	8	16	4	33
1968:	≤1500	375	151	0	4	0	0	4
	1501-2500	2989	2784	4	7	4	0	15
	>2500	40309	39594	14	10	16	8	48
1969:	≤1500	346	138	1	4	3	0	8
	1501-2500	2966	2791	3	4	3	0	10
	>2500	38940	38313	20	4	17	5	46
1970:	≤1500	382	164	0	5	1	0	6
	1501-2500	2977	2818	2	3	7	1	13
	>2500	38711	38000	9	6	19	5	39
1971:	≤1500	297	127	0	1	0	0	1
	1501-2500	2534	2370	2	6	5	1	14
	>2500	38385	37673	13	7	16	5	41
1972:	≤1500	289	129	1	0	0	0	1
	1501-2500	2459	2316	4	6	3	2	15
	>2500	35088	34528	23	5	20	7	55
1973:	≤1500	220	76	0	2	0	0	2
	1501-2500	1787	1699	5	6	2	0	13
	>2500	32346	31777	11	7	23	3	44
1974:	≤1500	254	98	2	1	2	0	5
	1501-2500	1964	1862	0	5	8	1	14
	>2500	30297	30153	13	6	15	11	45
1975:	≤1500	204	92	0	1	1	0	2
	1501-2500	1639	1555	6	4	3	1	14
	>2500	29403	29238	12	3	11	4	30
1976:	≤1500	181	105	0	0	0	0	0
	1501-2500	1377	1325	7	6	3	0	16
	>2500	28202	28042	9	2	8	4	23
1977:	≤1500	193	95	0	2	1	0	3
	1501-2500	1252	1213	2	3	3	2	10
	>2500	26985	26838	12	6	8	2	28
1978:	≤1500	191	128	0	3	0	0	3
	1501-2500	1512	1468	5	5	7	1	18
	>2500	28209	28084	13	2	9	4	28
1979:	≤1500	224	139	2	4	1	0	7
	1501-2500	1707	1680	6	9	7	0	22
	>2500	30226	30091	16	2	11	5	34
1980:	≤1500	267	152	4	8	5	0	17
	1501-2500	1923	1872	5	5	2	0	12
	>2500	30054	29978	12	3	13	5	33
1981:	≤1500	269	153	3	8	5	0	16
	1501-2500	1829	1790	3	5	5	0	13
	>2500	29738	29663	11	6	8	3	28
1982:	≤1500	211	140	6	3	4	0	13
	1501-2500	1895	1855	8	4	7	2	21
	>2500	29334	29261	11	3	17	2	33
1983:	≤1500	250	174	7	4	5	2	18
	1501-2500	1794	1759	2	4	9	0	15
	>2500	29803	29743	10	2	12	4	28
1984:	≤1500	249	180	4	6	1	0	11
	1501-2500	1854	1830	5	9	4	0	18
	>2500	29492	29444	16	0	12	5	33

The annual birthweight specific trends with prevalence according to clinical type of cerebral palsy are shown in the table.

To smooth the variation in annual prevalence rates associated with small numbers, the data in the table are used to plot the birthweight specific trends as three year moving averages, which are shown in fig 1 plotted on a logarithmic scale. Among infants of birth weights greater than 2500 g no consistent alteration in prevalence is evident, the rate varying between 0.91 and 1.77/1000 live births. Among infants weighing 1501-2500 g at birth there is a steady rise in the prevalence with a twofold to threefold increase between the start of the study period up to 1977/9, after which the prevalence plateaus or even shows a moderate decline. For very low birthweight infants in the first decade of the study the prevalence of cerebral palsy fluctuates at about 10/1000 live births. Subsequently, in the late 1970s, there is a sharp rise with a fivefold to sixfold increase in prevalence.

These birthweight specific trends according to clinical type of cerebral palsy are shown in fig 2 (>2500 g), fig 3 (1501-2500 g), and fig 4 (≤1500 g). Figure 2 is unremarkable in that the

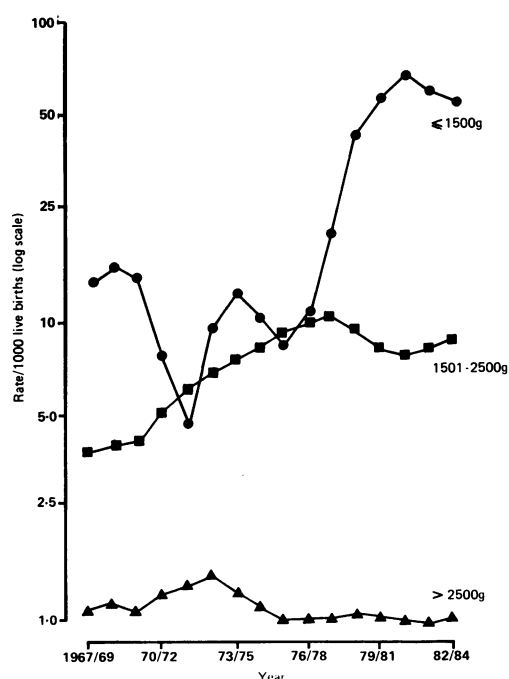


Figure 1 Birthweight specific trends in cerebral palsy.

prevalence rates are unchanged for all types of cerebral palsy. Prevalence rates for quadriplegia and hemiplegia are similar throughout the study period, whereas diplegia shows a consistently lower prevalence than either of the others. Figure 3 shows that the rise in prevalence of cerebral palsy in the birthweight group 1501–2500 g is contributed to by all three clinical categories. Among the hemiplegias there is

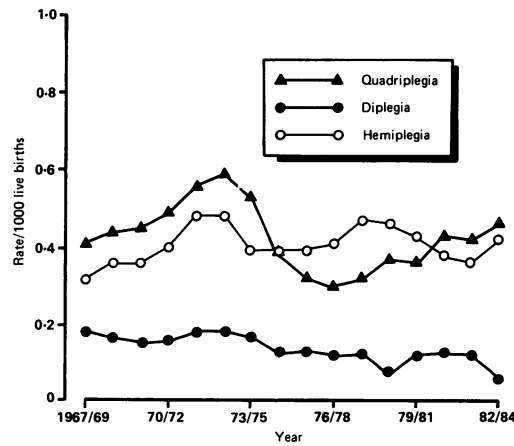


Figure 2 Trends in cerebral palsy: birth weight >2500 g (three year moving average).

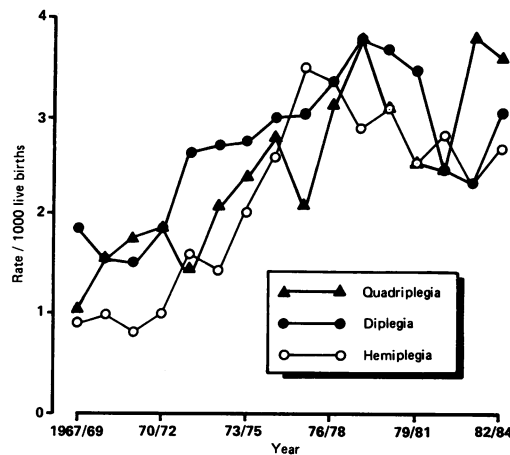


Figure 3 Trends in cerebral palsy: birth weight 1501–2500 g (three year moving average).

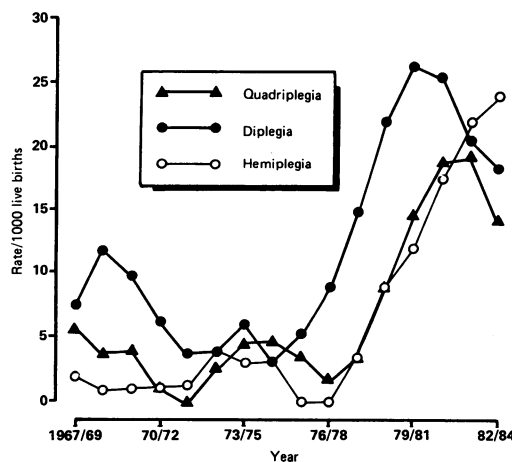


Figure 4 Trends in cerebral palsy: birth weight ≤1500 g (three year moving average).

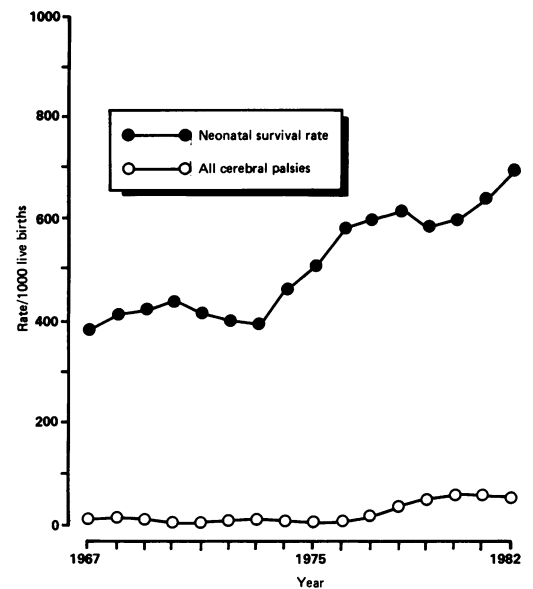


Figure 5 Birth prevalence of cerebral palsy and neonatal survival rates, birth weight ≤1500 g.

more than a threefold increase in prevalence, from about 1/1000 to 3.5/1000 live births in 1977/9. Subsequently the prevalence fell.

Figure 4 shows the trends in prevalence among very low birthweight infants according to clinical category. The diplegias show a sharp rise in prevalence that starts about 1974/6, but from 1979/81 there appears to be a decline. The hemiplegia and quadriplegias also show sharp rises in prevalence that start later than that for the diplegias. It is also pertinent that, in the final period of the study hemiplegia is the commonest form of cerebral palsy among very low birthweight infants.

To put the magnitude of the problem of increasing rates of cerebral palsy into perspective, fig 5 shows the trends in neonatal survival and prevalence of cerebral palsy in very low birthweight infants plotted on the same linear scale. The difference between the two linear graphs represents infants who survive without cerebral palsy. The increase in prevalence is accompanied by an increasing proportion of infants surviving unimpaired.

**Discussion**

The rise in prevalence of cerebral palsy among low birthweight infants is of concern to obstetricians and paediatricians, particularly in the context of an overall improvement in neonatal survival rates. Lack of completeness of data is an acknowledged source of error when examining trends, so several sources of information were used to compile the register to minimise any chance of omitting cases. Evidence that data collection might have been incomplete is given by the fact that 99 cases of cerebral palsy came into the region over the period 1967–84, but only 51 cases left. This represents a shortfall of 48 cases, which may have arisen from those who left the region before the diagnosis was made or before they became known to one of the several sources used to compile the register. As the

shortfall was evenly distributed throughout the study period, however, it will not appreciably alter the general trends.

The report of an earlier study covering the periods 1950–2 and 1970–3 recorded cerebral palsy rates among very low birthweight infants as 6–7% at about the age of 6.<sup>14</sup> These prevalence rates are roughly twice as high as we have observed in this study. The two sets of data are not strictly comparable because different denominators were used (children aged 6 and neonatal survivors) and because the populations and health services provided differed. Nevertheless the possibility of failure of ascertainment of cerebral palsy among very low birthweight infants must be considered. This may occur because some children with cerebral palsy die before the diagnosis is made, which could only affect the trend if such incompleteness differently affected the time period under consideration—for example, if, with improving diagnostic services, children were being diagnosed earlier. If this did occur it is not possible to quantify its effect. Alternatively, incompleteness of registration may have arisen if cases of cerebral palsy were erased from the disability registers when they died. To counteract this possible deficiency, the Office of Population Censuses and Surveys provided death certificates of children which had cerebral palsy mentioned on them. It seems unlikely therefore that there was any important deficit from this source. Even if there has been incomplete registration in the early part of the study period, the magnitude of the failure to record cases would have to be massive to attribute all the six-fold increase in birth prevalence of cerebral palsy among very low birthweight infants to an artefact.

The rise in prevalence of cerebral palsy among low birthweight infants observed in the Mersey region has been associated with developments in obstetrics and neonatology that have been accompanied by continuous improvement in neonatal survival rates. Crucial to the interpretation of these results, however, is whether the increase in prevalence of cerebral palsy is a result of an increase in the incidence of the condition or its duration. If it is postulated that there has been an increase in incidence in recent years, the inference must be that the cerebral damage has occurred at or around the time of birth and that an optimal cerebral environment has not been maintained. This interpretation is of relevance to obstetricians or paediatricians, because they could possibly prevent the condition by modifying clinical management.

This view has been advanced by a Swedish group who found that in half the preterm infants with cerebral palsy that they studied the aetiology was obviously perinatal.<sup>4</sup> Perinatal origin was, however, assumed if there was an intracerebral haemorrhage, or a need for mechanical ventilation, or a sequence of intrauterine asphyxia, severe birth asphyxia, severe neonatal hypoxia, and cerebral irritation, all in the perinatal period. The definition is then tautological and begs the question of what caused the prematurity.

An alternative explanation is that the duration of disease has increased. As by definition cerebral palsy is not progressive, the duration of the condition can only increase by postponing death. This implies that the impairment is prenatal in origin and that obstetric and neonatal improvements are now keeping infants alive who would previously have died. Several lines of evidence support this explanation. A high proportion of infants with cerebral palsy have retarded growth at birth, indicating that there must have been adverse factors affecting the development of the fetus.<sup>5 15–18</sup> Furthermore, infants with cerebral palsy have a higher proportion of congenital anomalies or other dysmorphic features than do normal children, which suggests that prenatal development has been compromised.<sup>5 19–23</sup> In a study of dermatoglyphics in cerebral palsy it was considered that a disturbance of fetal growth had taken place about the third or fourth month,<sup>24</sup> and in a specific type of spastic diplegia caused by maternal iodine deficiency, the fetal brain is impaired early in pregnancy.<sup>25</sup> When the antecedents of cerebral palsy were examined, the inclusion of information about the events at birth and the neonatal period accounted for a proportion of cerebral palsy only slightly higher than that accounted for when consideration was limited to characteristics identified before labour began,<sup>5</sup> and a comparison of cerebral palsy in two national cohort studies concluded that most cases are not associated with adverse obstetric factors.<sup>6</sup> In an analysis of a cohort of infants weighing 2000 g or less, important determinants of diplegia could not be identified—that is, there was no association with either maternal characteristics or markers of intrapartum stress. It was concluded that factors that influence the rate of fetal development may be implicated in the aetiology of diplegia.<sup>26</sup> In contrast, variables known at the time of birth were able to identify most cases of hemiplegia and it was concluded that intrapartum events were closely related to the pathogenesis of hemiplegia though these effects may have been mediated by postnatal events.<sup>27</sup>

If the increase in prevalence of diplegia in very low birthweight infants in the 1970s is the result of better survival of prenatally impaired infants, the decline in prevalence in the 1980s needs explaining. In a survey in Scotland a difference in prevalence between preschool and school aged children was attributed to more cases coming to light as a result of the greater demands placed on the child in the early school years.<sup>28</sup> One possible explanation, therefore, is that there has been an incomplete registration of the youngest children, which will be made more complete should the survey continue. Alternatively, the early recognition of severely impaired infants may have improved and less strenuous efforts made to keep them alive.

The recent birthweight specific changes in prevalence of cerebral palsy are of importance to obstetric and neonatal management but it is not possible to determine whether intrapartum or early postpartum medical management is sub-optimal and thereby initiates the cerebral impairment, or whether the improvements in

management now ensure survival of impaired infants who would previously have died. In most cases, particularly those with diplegia, the evidence is in favour of the latter explanation. For some of the low birthweight infants with hemiplegia the medical management at or around the time of birth may have had a crucial effect. Improvements in the recording and clinical classification of types of cerebral palsy are mandatory for further epidemiological analysis and the development of a standard recording form is a big step in the right direction.<sup>29</sup> The use of prospective clinical studies and the application of newer imaging techniques to enable more precise clinical classification should also be of value in the assessment of possible aetiological factors.

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