Archives of Disease in Childhood, 1989, **64**, 848–852

Cerebral palsy in two national cohort studies

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SUMMARY The prevalence of cerebral palsy in the 1958 British Perinatal Mortality Survey and the 1970 British Births Survey remained constant at 2·5/1000 births (40 and 41 cases, respectively). The prevalence at 10 years was higher in the 1970 cohort in which all children with cerebral palsy survived, whereas 22% of the cases in the 1958 cohort died during the first 10 years of life.

A case-control study matched three controls for social class, maternal age, parity and marital state, and a further three controls for the infant's sex, gestation, and birth weight. Comparison of cases and controls showed no consistent differences in social and environmental factors, history of pregnancy, labour, or delivery. Important differences were found in the incidence of respiratory and neurological symptoms in the neonatal period.

These prospective data derived from two whole populations of births support the hypothesis that most cases of cerebral palsy are not associated with adverse obstetric factors, and confirm that neonatal neurological symptoms are associated with subsequent cerebral palsy.

The association between perinatal mortality and childhood impairment, especially cerebral palsy, remains uncertain. It has often been assumed that factors relating to mortality, especially those caused by asphyxia and cerebral trauma at birth, would also be associated with a high risk of impairment among survivors. This hypothesis has recently been challenged by Ounsted who pointed out that the data on which the original concept of a 'continuum of reproductive casualty' was based were open to criticism.[1]

We have examined the prevalence of, and risk factors for, cerebral palsy in two national cohort studies: both were similar in their design and methods but took place 12 years apart. During those 12 years there were reductions both in the rate of stillbirths (from 22 to 13/1000 total births), and in neonatal mortality (from 16 to 12/1000 live births).

Whether or not causally associated with this drop in mortality, obstetric practice had changed and become more interventional. The proportion of births delivered in consultant units increased from 48% to 66%, caesarean section deliveries increased from 3% to 5%, inductions of labour increased from 13% to 26%, and forceps or ventouse deliveries increased from 5% to 9%. In 1958, 8% of women had labours lasting longer than 24 hours for the first stage, and 21% in excess of one hour for the second stage.[2] By 1970 these proportions had dropped to 4% and 12%, respectively, and the mortality associated with cerebral trauma at birth fell from 3/1000 in 1958 to 0·4/1000 total births in 1970.[3]

Subjects and methods

THE BIRTH SURVEYS

The 1958 British Perinatal Mortality Survey aimed to study all births in England, Scotland, and Wales during the week 3–9 March. Detailed questionnaires were completed by the midwives soon after delivery using clinical notes and information obtained by interviewing the mothers. It is estimated that 98·5% of total births were included in the survey.[4 5] The 1970 British Births Study used a similar method, and included all births in the week 5–11 April in the United Kingdom.[3 6] Only 2% of births were thought to have been omitted.

FOLLOW UP

The children born in 1958 were subsequently traced and followed up at the ages of 7, 11, and 16. At each visit the parent was interviewed about the medical history of the child (including admissions to hospital and visits to outpatient departments) and asked whether the child had any impairment, disability, or handicap. At each of these follow up visits the clinical medical officer examined the child and noted any problems. The teacher was also asked to note any problems and to assess the child's ability.

For the 1970 birth cohort, children residing in England, Scotland, and Wales were followed up at the ages of 5 and 10. At the age of 5 a complete medical history was obtained from the mother, and the health visitor concerned with the child. At the age of 10 a complete medical history was again taken from the mother, and the clinical medical officer examined the child. Similar questions were asked to those in the 1958 survey, and once again the child's teacher was asked to participate.

For all children from both surveys who had what could have been cerebral palsy the hospital records were obtained, with the local consultant's permission. In addition, death certificates of all children who had died in the period between birth and the follow up were reviewed. Those with a record of cerebral palsy were investigated in more detail where possible. Cases with unequivocal evidence of postnatal causation were omitted. One case, who seemed to have cerebral palsy in the first year of life but was later normal, was not included.

DEFINITIONS

Cerebral palsy. A non-progressive disorder of movement or posture resulting from damage to the developing brain. 'Acquired' cases (resulting from an insult after the neonatal period) were not included.

Prevalence. This was the number of cases of cerebral palsy surviving the neonatal period/1000 neonatal survivors (excluding acquired cases).

Birth weight. The baby was weighed within a few hours of birth. The birth weights in both surveys were measured in ounces and converted to grams for analysis.

Gestation. This was calculated as the number of completed weeks elapsing from the date of the first day of the last menstrual period to the date of delivery. Women with uncertain dates were analysed separately.

Stillbirth. This was defined as the birth of a dead fetus of 28 weeks' gestation or more (or a weight of 1000 g or more in the absence of an accurate estimate of gestational age).

Parity. The number of previous pregnancies resulting in a live or stillbirth.

Social class. This was coded using the classifications of the husband's occupation developed by the Registrar General for England and Wales.[7,8] For both surveys, all single, widowed, separated, or divorced women who did not have a partner with an occupation coded on the questionnaire were analysed separately—the unsupported mothers.

CASE-CONTROL STUDY

As birth weight and gestation are known to be associated with cerebral palsy, it was felt important to match on these factors to identify other features of cerebral palsy. In order to assess whether possible associations with social class, maternal age, and parity accounted for the birth weight and gestation effects, a second set of controls was chosen. There were thus two sets of three controls for each case, so that each case had six matched controls.

Control series A comprised three infants matched for age of the mother at the birth of her child, parity, social class, marital status at the time of delivery, and whether the child was a single or multiple birth.

Control series B comprised three infants matched for birth weight, gestation, certainty of the date of the last menstrual period, sex of the child, and whether the child was a single or multiple birth.

Results

OVERALL PREVALENCE

Of the children born in the week 3–9 March 1958, 16 751 survived the neonatal period. Of these 40 developed cerebral palsy, giving a prevalence of 2·4/1000. Of the children born in Great Britain in the week 5–11 April 1970, 16 136 survived the neonatal period, and 41 had cerebral palsy (prevalence 2·5/1000). These figures are in remarkable agreement with those of the Isle of Wight (2·6/1000),[9] Watford (2·5/1000),[10] and the United States Collaborative Perinatal Project (which reported a prevalence of 2·5/1000 after excluding the mildly affected and the acquired disorders).[11]

SURVIVAL

All children with cerebral palsy born in 1970 survived until the age of 10, whereas nine of the 40 cases born in 1958 (23%) had died by 10 years. Seven deaths occurred before 7 years and were identified from death certificate records. Of the 33 children who were seen at the age of 7 and described as having cerebral palsy, two subsequently died (one in a road traffic accident) before the 10 year assessment.

Consequently, although the prevalence of cerebral palsy in the neonatal period remained static between the two cohorts, there was a pronounced increase in prevalence at the age of 10 from 1·9/1000 in 1958 to 2·6/1000 in 1980.

BIRTHWEIGHT SPECIFIC PREVALENCE

Only 17 infants weighing under 1500 g at birth from the 1958 cohort survived the neonatal period, compared with 53 from the 1970 cohort. In the study involving the children born in 1958, however, only two children found to have cerebral palsy weighed less than 1500 g at birth, and in the 1970 cohort there was only one (table 1). In 1970 there was an increase in the risk of cerebral palsy among the low birthweight babies, but a slight decrease among those who weighed 3500 g or more at birth.

Table 1 *Prevalence of cerebral palsy by birth weight*

Birth weight (g)	*Prevalence of cerebral palsy/1000 survivors at 28 days*	
	1958	*1970*
<1500	117·6 (2)	18·9 (1)
1500–1999	10·5 (1)	21·1 (4)
2000–2499	6·0 (4)	12·5 (10)
2500–2999	4·1 (12)	2·3 (7)
3000–3499	1·3 (8)	1·4 (9)
≥3500	2·1 (13)	1·4 (8)
Total <2500	9·0 (7)	14·4 (15)
Total ≥2500	2·2 (33)	1·6 (24)
Total including those of unknown birth weight	2·4 (40)	2·5 (41)

Actual numbers are shown in parentheses.

CASE-CONTROL STUDY

Social factors

As those in control series A were matched on social class and marital state there were no striking socioeconomic or environmental differences between them and the index cases. Using control series B, there was no evidence of differences by region, household overcrowding, ethnic group, or educational background.

Maternal health

The mother's age, parity, prepregnancy weight and height, smoking habit, and attendance for antenatal care were not associated with the subsequent development of cerebral palsy in the baby. There was no evidence that mothers of index cases had more complicated pregnancies: in particular there was no increase in the incidence of pre-eclampsia, bleeding, or anaemia during their pregnancies.

History of labour

The mothers of index cases were slightly (but not significantly) more likely to have labour induced. Among the children born in 1958, there were significantly more index children born after the membranes had been ruptured for at least 24 hours when compared with the control series A, but the association was not apparent among the 1970 cases. Neither the first nor second stages of labour differed in duration, nor was there a significant difference in the place of delivery or the type of analgesia given to the mother. There were only four cases of cerebral

Table 2 *Signs of asphyxia, 1970 only. Results are expressed as No (%)*

	Index cases (n=41)	*Control series A (n=121)*	*Control series B (n=121)*
Time taken to establish regular respirations:			
<1 minute	18 (44)	91 (75)	87 (72)
1–3 minutes	14 (34)	27 (22)	25 (20)
>3 minutes	9 (22)	3 (3)	9 (7)
		p<0·0001*	p<0·01*
Resuscitation method:			
None	5 (12)	33 (27)	23 (19)
Positive pressure ventilation	2 (5)	0	1 (1)
Endotracheal tube	4 (10)	2 (2)	3 (3)
Other oxygen	20 (49)	28 (23)	31 (26)
Other physical	10 (24)	60 (49)	63 (52)
		p<0·0001*	p<0·0001*
Administration of oxygen:			
None	15 (37)	91 (75)	85 (71)
By face mask	7 (17)	10 (8)	11 (9)
By face funnel	10 (24)	13 (11)	17 (14)
Other	9 (22)	7 (6)	7 (6)
		p<0·0001*	p<0·001*

*p Values compared with index cases.
Numbers vary in each category according to number of controls on whom data was available.

palsy who had breech deliveries, compared with 13 birth weight/gestation matched controls. In contrast, six index cases were delivered by caesarean section compared with 11 birth weight/gestation controls. Thus there was no evidence that operative delivery affected outcome.

The baby

Although there was a preponderance of boys (26/40) among the cases of cerebral palsy in the 1958 cohort, this difference was not significant and was not confirmed in the children in the 1970 cohort (23/41 boys). Compared with control series A, children with cerebral palsy were significantly more likely to be of low gestation or low birth weight, or both, than controls.

There was a strong association with time to onset of regular respirations, an excess of index cases taking longer than one minute to establish breathing (table 2). Children with cerebral palsy were more likely to have been given oxygen after birth than control cases. Index cases were more often admitted to a special care baby unit, and nursed in an incubator (table 3).

Among specific problems occurring in the first week, highly significant associations existed with 'breathing difficulties', cyanotic attacks, and cerebral irritation. These associations remained significant even when the controls of the same sex, birth weight, and gestation were compared (table 4).

Discussion

There are both advantages and disadvantages in using the two present data sets to identify the possible causes of cerebral palsy. The principle advantages are that they cover a whole population of births, and the data were objective and gathered prospectively. They suffer from none of the biases of retrospective recall, and ascertainment is thought to have been almost complete. The defect lies in the lack of fine detail that might affect interpretation of the results.

We have shown in this study that, although there had been pronounced changes in obstetric practice and perinatal mortality between two periods of time—for example the incidence of cerebral trauma at birth had reduced considerably—the prevalence of cerebral palsy remained static.

Though the overall prevalence of cerebral palsy is unchanged, there is evidence in this and other studies that the birthweight specific risks of cerebral palsy have changed with the increased survival of more low birthweight infants.[12 13] It is possible that the increased contribution of low birthweight babies to the population of infants with cerebral palsy has been matched by a decrease in the contribution from babies weighing more than 3500 g.

Overall, the proportion of children with cerebral palsy who had low birth weights was lower in these two British cohorts than that reported in the hospital

Table 3 *Nursing dependency, 1970 only*

	Index cases	*Control series A*	*Control series B*
No (%) admitted to special care unit at any time during first week	17/41 (42)	10/121 (8)*	25/122 (20)*
No (%) nursed in incubator on first day	15/41 (37)	10/117 (9)*	24/121 (20)*

*p<0·001 compared with index cases.

Table 4 *Signs and symptoms in first week of life, 1970 only. Results are expressed as No (%)*

	Index cases	*Control series A*	*Control series B*
Breathing difficulties	13/40 (32)	3/121 (3)***	6/122 (5)***
Cyanotic attacks	9/41 (22)	0/121***	6/119 (5)**
Fits or convulsions	2/39 (5)	0/122	0/120
Cerebral irritation	10/40 (25)	3/122 (3)***	3/118 (3)***
Jaundice	12/40 (30)	18/121 (15)	37/121 (31)
Congenital defect	4/41 (10)	3/122 (3)*	5/123 (4)
Cephalohaematoma	0/41	1/122 (1)	1/122 (1)
Sticky eyes	0/40	9/120 (8)	10/119 (8)
Umbilical discharge	2/40 (5)	1/120 (1)	2/120 (2)
Other illness	4/40 (10)	9/123 (7)	0/119

*p<0·05; **p<0·01; ***p<0·001.

based United States National Collaborative Perinatal Project,[14] in which 22% of the cases of cerebral palsy were born weighing less than 2000 g. These national cohort studies, however, were undertaken before the widespread introduction of intensive care in the 1970s. Evidence now suggests that there has been an increase in cerebral palsy among survivors with low birth weights since 1970, but this increased survival has not made a significant contribution to the overall rate of cerebral palsy. Rates of cerebral palsy are thus not a good index of the impact or quality of neonatal intensive care.[15]

This study lends no support to the contention that infants with cerebral palsy were born after abnormal pregnancies, labours, or types of delivery. Once delivered, however, they were more likely to have an abnormal neonatal course. In both cohorts, asphyxia (measured as time to onset of regular respiration) seemed to be a strong predictor of cerebral palsy. The lack of association with intrapartum factors recognised to lead to asphyxia (for example, a prolonged second stage), however, suggests that the delay in onset of respiration reflects a baby compromised by events or developmental aberrations in early fetal life.

Similar conclusions on the causes of cerebral palsy have been reached from other population studies, using multivariate analyses of risk.[16] Data from both Australia[17] and the United States[16] have led to similar estimates (3–13%) of the proportion of cerebral palsy that might have been caused by intrapartum damage.

There is no evidence from these cohort studies to indicate that changes in obstetric practice would alter the prevalence of cerebral palsy. Few children with cerebral palsy are damaged during birth[17]; improvements in obstetric and neonatal care can be expected to reduce the perinatal mortality rate, but not the prevalence of cerebral palsy in the population.[12 18] In particular there is no evidence that increases in the numbers of caesarean sections will reduce the number of cases of cerebral palsy.

In conclusion, most cases of cerebral palsy are not associated with perinatal factors, though neurological symptoms in the neonatal period are strongly associated with subsequent cerebral palsy. This study offers no evidence to support the concept of a 'continuum of reproductive casualty'; indeed, it lends support to Freud's assertion in 1897 that 'difficult birth in itself is merely a symptom of deeper effects that influence the fetus'.[19] We hope that research over the next 90 years will throw more light on the nature of these deeper effects.

We thank all who took part in these national cohort studies: the midwives, health visitors, medical officers, administrators, teachers, and especially the parents. For this particular study we also thank the records officers and consultants for giving us permission to view the children's clinical records, Richard Belli for carrying out the computing, and Yasmin Iles for typing the manuscript.

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Accepted 18 January 1989