Cerebral palsy—an increasing contributor to severe mental retardation?

Amanda Nicholson, Eva Alberman

Severe mental retardation (SMR), here approximating to an intelligence quotient (IQ) of less than 50, has a prevalence among school entrants of about 3-5 per thousand. The wide range of clinical conditions within the severely retarded population fall into five major groups: Down's syndrome, cerebral palsy, X linked mental retardation, single gene defects, and malformation syndromes. The aim of this paper is to predict the future contribution of the aetologically heterogeneous group of cerebral palsy to the severely retarded population.

The prevalence of cerebral palsy is about 2/1000 in most childhood populations, and up to 35% of those affected (0-7/1000), are also severely mentally retarded (personal communication). This group therefore comprises about one fifth of the overall prevalence of severe mental retardation. For comparison, Down's syndrome contributes one third and X linked mental retardation, single gene defects, and malformation syndromes each contribute one tenth to the overall prevalence.

Background information needed to predict future trends in this group includes a knowledge of the causes of cerebral palsy, and of any trends in their prevalence; and of the effect of changes in the early mortality rates of affected or high risk infants. Although in most cases the cause remains unknown, associated factors are recognised and will be used for prediction.

As only few reports of cerebral palsy distinguish between the characteristics of those with SMR and those without, the literature on trends in cerebral palsy in general will be reviewed, pointing out any factors known to be particularly related to SMR.

Associated factors
One feature which distinguishes the epidemiology of cerebral palsy from that of any of the other causes of SMR is the close association between cerebral palsy and low birth weight, whether in preterm or other births. This relationship acts as an important confounding factor and must be taken into account in any analyses of possible causation, or when predicting trends. It is accepted that many of the adverse obstetric factors known to be associated with cerebral palsy may act primarily by causing preterm delivery, and thus are only indirectly related with the sequelae consequent upon immaturity. Similarly, brain 'damage' may be the cause and not the consequence of asphyxia at birth.

In the following account the likely time of action of different types of insult will be described, but it must be borne in mind that many of these causes may act differently at different ages.

**Known Prenatal Causes**

Environmental factors that are known to cause prenatal brain injury include congenital infections; exposure to toxins including drugs and alcohol; and a suboptimal uterine environment of unknown cause which has been described as 'fetal deprivation of supply', secondary to a number of known and unknown causes.

**Infections**

In the UK cytomegalovirus is the most common cause of congenital infection, leading to impairment in about 0-3/1000 births. Approximately half of the affected children have cerebral palsy or psychomotor delay, but not all will have SMR. Isolated mental retardation is uncommon. The congenital rubella syndrome currently has a birth prevalence of about 0-03/1000, and reported sequelae include cerebral palsy, but again isolated mental retardation is uncommon. Congenital toxoplasmosis is a cause of severe retardation in the absence of cerebral palsy but birth prevalence is estimated to be less than 0-01/1000. The impact of HIV has yet to be assessed. Evidence is growing that a substantial proportion of symptomatic infected children have neurological syndromes which may involve mental retardation, but at present the number of children in the UK known to be HIV positive is very small.

In summary, congenital infections together probably cause no more than an estimated 0-03 cases of SMR/1000 live births, most of which will be associated with cerebral palsy. There is unlikely to be any substantial change in the prevalence of these risks, since it is only congenital rubella, already a rare cause, which is likely to decline further over the next decade. There is a possibility that congenital HIV infections may lead to a small increase.

**Alcohol**

Although many prescription drugs may cause fetal damage, alcohol is the only commonly used fetal toxin that has been linked to mental retardation, although severe retardation is probably not common. The mean IQ for the
Cerebral palsy—an increasing contributor to severe mental retardation?

1051

fetal alcohol syndrome has been stated to be 63,13 Estimates of the prevalence of fetal alcohol syndrome, and more specifically mental retardation associated with maternal alcohol intake, are scanty and inconsistent. It seems that in the UK the number of cases of severe retardation due to alcohol consumption is likely to be very small, although good population based data is scanty.14

Fetal deprivation of supply (FDS)

This is a term introduced in Scandinavia to include cases of presumed prenatal fetal deprivation inferred from either maternal complications or fetal growth retardation. Often it is not known whether the growth retardation is due to fetal or maternal causes. Estimates from Sweden and Canada indicate that 0-3/1000 cases of SMR are linked to FDS,15,16 but it is difficult to tell from the literature how many are associated with cerebral palsy.

Perinatal brain damage

Perinatal brain damage will include the effects on potentially normal babies of complications around the time of birth, including asphyxia and trauma.

For babies of normal birth weight perinatal factors are thought unlikely to be important as a cause of isolated SMR,16 but it had been hoped that improved obstetric care would lead to a fall in prevalence of cerebral palsy in babies of normal birth weight in parallel with the fall occurring in perinatal mortality. Recent work suggests, however, that birth asphyxia is a rare cause of cerebral palsy,17,18 accounting for less than 10% of the total.

In the very low birthweight infant whose chance of survival is increasing, the complications of immaturity may be extremely important in those potentially normal at birth, resulting in periventricular ischaemia and neurological sequelae. However, we do not know in what proportion preterm birth is a consequence of prenatal damage, which itself causes neurological sequelae. Infantile hydrocephalus after cerebral haemorrhage, and its sequelae, is another important cause of SMR and/or cerebral palsy, for which the best recent prevalence data is from Sweden with a rate of 0-63/1000 in the birth years 1979–82.19–22 The ventriculomegaly trial group in England reported that all affected children with developmental delay also had neuromotor impairments.23

Although perinatal damage is traditionally associated with cerebral palsy, some survivors of low birth weight are severely retarded but do not have cerebral palsy. These may have associated sensory deficits such as deafness or blindness or may have isolated SMR. Alberman et al., studying births of less than 1814 g born in the 1970s, found that the total prevalence of severe mental retardation was 17/1000 with 14-5/1000 being associated with cerebral palsy.24 Recent smaller studies report no cases of cerebral palsy,25 although in others it is difficult to extract this information.27

A ‘best guess’ for the contribution of perinatal factors to SMR is about 0-3/1000 births, and this proportion is one that may be rising because of the improved survival of high risk infants.

Postnatal brain injury

Causes of postnatal brain injury reflect the hazards of the older child’s environment. Infections of the nervous system are predominant but trauma, particularly road traffic accidents and non-accidental injury, are also important. Survivors of central nervous system malignancies also contribute but are a small group, most also having motor deficits. There are many problems associated with definitions of postnatal cerebral palsy, reported prevalence of postnatal SMR varying from 0-1 to 0-5/1000,1,15 28 but this group is one where improved standards of living and better medical care should lead to a reduction. Nevertheless preterm births are at increased risk and their increasing survival must be taken into account when assessing likely trends.

Our ‘best guess’ for the overall prevalence per thousand children of SMR with cerebral palsy is 0-03 due to congenital infection, 0-3 due to fetal deprivation, 0-3 due to perinatal factors, and the remainder, perhaps 0-2, associated with postnatal factors, with a small proportion due to the fetal alcohol syndrome or other exposure to fetal toxins.

Trends in the prevalence of cerebral palsy 
PRENATALLY AND PERINATALLY CAUSED CEREBRAL PALSY

Cerebral palsy registers in Mersey Regional Health Authority in England, in south western Sweden, and in Western Australia provide the bulk of the recent trend data,29–32 with additional information from registers in the regional health authorities in the north east and south east of England.

The reported overall prevalence per 1000 live births, excluding postnatal cases, remained fairly stable from 1958 to 1972,2 and then began to rise. Table 1 gives recent data from Sweden, Western Australia and Mersey, and there have been similar reports from elsewhere.33–35

The table gives the trends in different birthweight groups and shows that there has been a fairly constant level over time in babies of normal birth weight in the studies included, although Jarvis et al found an increase in reported live birth prevalence from 1/03 to 1-53/1000 among babies weighing 2500 to 4000 g between birth years 1968–71 and 1972–5.36 In all three registers, apart from the most recent Mersey data, there has been a fall in prevalence rates in the group weighing between 1500 and 2499 g at birth.

However, the most striking finding in the table is the considerable rise in prevalence in neonatal survivors found in all three registers for babies of <1500 g birth weight. The most recent data from Mersey suggest that the increase in prevalence in the very smallest babies is maintained, although there have been some recent reports suggesting that the disability rate in these is stable, or falling.37,38 Nevertheless, the Mersey and Swedish recent prevalence rates of cerebral palsy in babies of under 1500 g of around 90/1000 are confirmed by numerous
smaller, but not always population based, studies.29,30,37,39,40

In many developed countries there has been a sharp fall in neonatal mortality, even though many deliveries previously regarded as miscarriages are being resuscitated and registered as live births. A decrease in neonatal mortality rates with constant cerebral palsy prevalence risks in the survivors will inevitably lead to an increase in both absolute numbers and in prevalence rate.

A consequence of these changes in pattern is that babies of under 1500 g form an increasing proportion of the total cerebral palsy cases. Between the periods 1975–8 and 1979–82 this proportion rose from 9% to 18% in the Swedish data, from 5·7% to 13·0% in the Australian data, and from 4·6% to 21·3% in the Mersey data, which also shows that between 1983 and 1984 these babies accounted for nearly 24% of the total.

POSTNATAL CEREBRAL PALSY

Population based reports from Mersey and Western Australia give the best epidemiological data on cerebral palsy arising after the neonatal period.41–43 Postnatally acquired cerebral palsy in the industrialised world usually forms approximately 10% of total cases.4 The recent report from Mersey43 was unusual in finding that postneonatal cases were responsible for 18% of the total. In this study, between 1966 and 1977 the prevalence rate of postnatally acquired cerebral palsy varied from 0.2 to 0.6/1000 neonatal survivors. The overall figure was 0.3/1000 neonatal survivors. In Western Australia41,42 prevalence rates varied from 0·19 to 0·27/1000 neonatal survivors between 1956 and 1975.

In Mersey there was evidence that survivors of low birth weight were at higher risk of developing cerebral palsy than babies of normal birth weight, their increased prevalence possibly accounting for the unusually high proportion of postnatal cases. The rates per 1000 neonatal survivors were 0·71 in those of birth weight <1500 g; 0·56 in the birthweight group 1500–2499 g, and 0·31 in those of normal birth weight.

SUMMARY OF PREVALENCE DATA

The prevalence figures from Mersey are the most complete and have been used to estimate future trends (see table 2). The rate of cerebral palsy due to prenatal and perinatal causes in babies of normal birth weight is lower in Mersey than reported elsewhere (table 1) and this may affect results.

SEVERE MENTAL RETARDATION IN CEREBRAL PALSY

Information on mental retardation in cerebral palsy is limited. Table 3 gives data from two studies. Although previously SMR was less common in children of low birth weight and cerebral palsy, recent reports from Sweden indicate that there is a high prevalence of severe retardation among children contributing to the increasing prevalence of cerebral palsy in very low birthweight infants.45 Although Pharoah et al reported a very high incidence of SMR of 57% in postnatal cases,46 other reports of postnatal cerebral palsy give figures that are closer to those given in table 3 for the prenatal and perinatal cases. For instance Arens and Molteno describe 37% of their postnatal cases as having severe or profound retardation.44 The assumption is made that 35% of cerebral palsy cases in all birthweight groups in pre/perinatal, and in postnatal cases will have SMR.

Prediction of future trends

Because of the considerable importance of birth weight as a risk factor any prediction of future trends must take into account current and future likely trends in birthweight distribution and survival rates. Data on birthweight distribution and neonatal mortality rate of live births from 1983 to 1988 was supplied by the Office of Population Censuses and Surveys (personal communication).
### Table 4 Number of cerebral palsy cases expected from births 1983–8 in England and Wales, assuming prevalence rates from table 2, by time of insult and birth weight

<table>
<thead>
<tr>
<th>Year</th>
<th>Total No live births</th>
<th>Total</th>
<th>&lt;1500 g</th>
<th>1500–2499 g</th>
<th>≥2500 g</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NNS</td>
<td>Pre/per</td>
<td>Post</td>
<td>NNS</td>
<td>Pre/per</td>
</tr>
<tr>
<td>1983</td>
<td>639</td>
<td>111</td>
<td>3731</td>
<td>338</td>
<td>3</td>
</tr>
<tr>
<td>1984</td>
<td>636</td>
<td>818</td>
<td>4003</td>
<td>363</td>
<td>3</td>
</tr>
<tr>
<td>1985</td>
<td>656</td>
<td>417</td>
<td>4254</td>
<td>395</td>
<td>3</td>
</tr>
<tr>
<td>1986</td>
<td>661</td>
<td>018</td>
<td>4490</td>
<td>407</td>
<td>3</td>
</tr>
<tr>
<td>1987</td>
<td>681</td>
<td>511</td>
<td>4825</td>
<td>438</td>
<td>3</td>
</tr>
<tr>
<td>1988</td>
<td>693</td>
<td>577</td>
<td>4818</td>
<td>457</td>
<td>3</td>
</tr>
</tbody>
</table>

NNS, neonatal survivor; Pre/per, expected cases of cerebral palsy per pre/perinatal aetiology; Post, expected cases of cerebral palsy of postnatal aetiology.

communication) and suggest that the proportion of total births that are of low birth weight may have reached a plateau, after rising for some years, and neonatal mortality rates of low birthweight infants are also levelling out after sharp falls. For the births of 2500 g or more neonatal mortality rates are continuing to fall. For the present purpose the conservative assumption has been made that birthweight distributions and the neonatal mortality rates over the next 12 years will level out from 1988, and the 1988 rates will be applied to birth projections for this period.

The importance to the overall prevalence of cerebral palsy of the birthweight distribution of neonatal survivors is demonstrated in table 4. This gives for England and Wales the number of neonatal survivors of known birth weight (over 99% of the total) for the years 1983 to 1988, and applies to them the birthweight group specific cerebral palsy prevalence rates derived from the most recent Mersey data (table 2). Although these prevalence rates in neonatal survivors are kept constant there is a 5% rise in crude prevalence largely because of the increase in the number of neonatal survivors of under 1500 g, with their very high risks. The live birth prevalence of perinatal and preinatal cerebral palsy rises to 2:2/1000 and all cerebral palsy cases reach 2:5/1000 (table 5).

It remains unclear whether any children subject to the insults described above are severely retarded but do not have cerebral palsy. Present indications are that this is the case for very few, if any, severely retarded children. No data are available but a notional birth prevalence of 0:2/1000 for these additional cases is suggested. This group is not included in the subsequent predictions.

### DEMOGRAPHIC PROJECTIONS

The total number of live births in England and Wales expected each year from 1990 to the year 2000 was supplied by the Government’s Actuary Department (personal communication). Assuming that birthweight distribution, neonatal survival, and cerebral palsy prevalence remain constant from 1988 levels, the future number of cerebral palsy cases was estimated by applying rates of 2:2/1000 live births for perinatal and perinatal cerebral palsy and 2:5/1000 for all cases including postnatal (table 6).

The number of severely retarded children with cerebral palsy was estimated to be 35% of the total overall. The projected number of cerebral palsy cases with SMR, assuming a steady rate of 35%, would fall from a peak of 648 born in 1993 and 1994 to 599 born in 2000, when the number of births is expected to have dropped.

### Associated impairments and survival

Details of severely retarded children with cerebral palsy are scarce and data from South East Thames and North East Thames cerebral palsy registers give some picture of these children (P Evans, personal communication). Data from the North East Thames register is still being collected and information regarding mental ability is very incomplete so that results must be viewed with caution. Table 7 gives details of associated impairments in the severely retarded children. Approximately one half have epilepsy and almost two thirds have no useful hand function and are immobile.

### SURVIVAL

A recent report from South East Thames indicates that 79% of severely retarded children registered as having a diagnosis of cerebral palsy survived to a mean age of 16 years. Survival rates beyond this age are not available. This is higher than reported previously in an earlier study from the US. Multivariate analysis of deaths in all mental abilities in the South East Thames data showed that immobility was the strongest predictor for mortality, followed by

Table 5 Total number of expected cerebral palsy cases and rate/1000 live births derived from table 4

<table>
<thead>
<tr>
<th>Year</th>
<th>No of cerebral palsy cases</th>
<th>Rate/1000 live births</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre/per</td>
<td>All</td>
</tr>
<tr>
<td></td>
<td>Pre/per</td>
<td>All</td>
</tr>
<tr>
<td>1983</td>
<td>1324</td>
<td>1525</td>
</tr>
<tr>
<td>1984</td>
<td>1361</td>
<td>1564</td>
</tr>
<tr>
<td>1985</td>
<td>1427</td>
<td>1636</td>
</tr>
<tr>
<td>1986</td>
<td>1452</td>
<td>1662</td>
</tr>
<tr>
<td>1987</td>
<td>1503</td>
<td>1719</td>
</tr>
<tr>
<td>1988</td>
<td>1514</td>
<td>1734</td>
</tr>
</tbody>
</table>

Table 6 Predicted number of cerebral palsy cases from births 1990–2000 in England and Wales: constant prevalence rates

<table>
<thead>
<tr>
<th>Year</th>
<th>Total No live births</th>
<th>Pre/perinatal cases</th>
<th>All cases</th>
<th>Cerebral palsy and SMR</th>
</tr>
</thead>
<tbody>
<tr>
<td>1990</td>
<td>725 626</td>
<td>1596</td>
<td>1814</td>
<td>635</td>
</tr>
<tr>
<td>1991</td>
<td>732 528</td>
<td>1612</td>
<td>1831</td>
<td>641</td>
</tr>
<tr>
<td>1992</td>
<td>737 681</td>
<td>1623</td>
<td>1844</td>
<td>645</td>
</tr>
<tr>
<td>1993</td>
<td>740 491</td>
<td>1629</td>
<td>1851</td>
<td>648</td>
</tr>
<tr>
<td>1994</td>
<td>740 239</td>
<td>1628</td>
<td>1851</td>
<td>648</td>
</tr>
<tr>
<td>1995</td>
<td>736 834</td>
<td>1621</td>
<td>1842</td>
<td>645</td>
</tr>
<tr>
<td>1996</td>
<td>729 849</td>
<td>1606</td>
<td>1825</td>
<td>639</td>
</tr>
<tr>
<td>1997</td>
<td>719 618</td>
<td>1583</td>
<td>1799</td>
<td>630</td>
</tr>
<tr>
<td>1998</td>
<td>709 397</td>
<td>1561</td>
<td>1773</td>
<td>621</td>
</tr>
<tr>
<td>1999</td>
<td>696 541</td>
<td>1532</td>
<td>1741</td>
<td>609</td>
</tr>
<tr>
<td>2000</td>
<td>684 917</td>
<td>1507</td>
<td>1712</td>
<td>599</td>
</tr>
</tbody>
</table>

SMR, severe mental retardation.

Table 7 Associated impairments in children with cerebral palsy and severe mental retardation. Figures are percentages

<table>
<thead>
<tr>
<th>Impairment</th>
<th>South East Thames (n=189)</th>
<th>North East Thames (n=69)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seizures</td>
<td>51.3</td>
<td>49.2</td>
</tr>
<tr>
<td>Hearing</td>
<td>11.8</td>
<td>16.7</td>
</tr>
<tr>
<td>Vision</td>
<td>68.9</td>
<td>53.0</td>
</tr>
<tr>
<td>Ambulant with or without aids</td>
<td>38.2</td>
<td>24.4</td>
</tr>
<tr>
<td>Chair/bed ridden</td>
<td>62.4</td>
<td>65.9</td>
</tr>
<tr>
<td>(self propelled)</td>
<td>(2-5)</td>
<td>(4-9)</td>
</tr>
<tr>
<td>Severely limited manual dexterity</td>
<td>65.2</td>
<td>59.4</td>
</tr>
</tbody>
</table>

*P Evans, personal communication.*
Severe retardation: 31% of the immobile, severely retarded group had died compared with 3% of the mobile but severely retarded group.

Discussion
Predictions
The most recent data upon which these predictions have been based come from births up to 1984. When calculating future prevalence it has been assumed that prevalence rates will stay at the these levels. It is important to consider any possible changes that may challenge this assumption.

Births Under 1500 G: Prevalence and Survival
The predictions made have taken into account trends in the prevalence of the high risk birth-weight groups up to 1988 only. It is possible that following the alteration in the abortion law, lowering the upper limit at which legal termination is permitted to 24 weeks, the legal distinction between stillbirth and miscarriage will also be changed to 24 weeks. This may have an effect in further increasing the resuscitation of births less than 1500 g.

A focus of recent literature on this question has been how much of the increase in prevalence of cerebral palsy in these babies is due to increased survival of prenatally impaired children, and how much to postnatal damage during their neonatal course.52 The latter situation leaves more scope for improvement with better neonatal care. At present we have little understanding of the prenatal causes leading to preterm birth with permanent neurological sequelae, and no means of preventing them; yet recent work with serial ultrasound has indicated that in one third of children with disabilities, ischaemic brain lesions were noted within two hours of birth, confirming the hypotheses that some of this damage had taken place before or during birth.47 Advances in medical imaging will continue to improve our knowledge of the timing of the insult in this group.

Regardless of timing, many studies have shown that periventricular leucomalacia and ventricular enlargement are predictors of future handicap.22 26 48 49 Early recognition of severely damaged infants may lead to a reconsideration of their neonatal management, with a reduction in effort to maintain their survival. This may lead to a fall in both neonatal survival and cerebral palsy prevalence in those survivors.

Births Weighing Between 1500 and 2499 G
This is a group about which little is known in regard to timing of insult, although their survival has improved considerably. It is likely, although not certain, that in many such affected births there has been a prenatal insult, but they probably include a group which resembles the very low birthweight group in their stormy neonatal course. It is difficult to predict whether there are likely to be changes in risk exposure affecting the prevalence of SMR, other than purely demographic, which have been taken into account. The assumption has been that there will be no such changes over the next decade.

Births Greater Than 2499 G
Since imaging is less often carried out in larger babies, and many will develop cerebral palsy after an apparently normal obstetric and neonatal course, the literature on the timing of the insult is usually based on inference only. It is now recognised that birth asphyxia plays only a small part, and probably 90% are due to prenatal causes, most of which are not preventable in our present state of knowledge. Fetal deprivation of supply is so poorly understood that substantial reduction seems unlikely. HIV infection may prove increasingly important but further reduction in the number of cases of rubella or of infants damaged by cytomegalovirus is unlikely. Increasing public awareness may reduce the problems of alcohol but this is unlikely to have a major impact on total prevalence of SMR.

The assumption has been made that over the next decade the risk in these babies is likely to stay constant, and that changes are likely to be accounted for by demographic changes only.

Postnatal Cerebral Palsy and SMR
Work published on postnatally acquired cerebral palsy is largely based on data from the 1950s to 1970s. The population studies on SMR giving prevalence of postnatal SMR are of the same era. Changes in medical care and child safety may make these results unreliable as a basis for future prediction. Infection is the leading cause of postnatal cerebral palsy in all studies, particularly meningitis and encephalitis. Gastroenteritis and subsequent dehydration was responsible for up to 15% of cases. Trauma is the second most common cause accounting for a quarter of cases. Road traffic accidents alone cause almost 10% and non-accidental injury between 3% and 9% of postnatal cerebral palsy. Improvement in parental education so that children’s illnesses are recognised and treated sooner may mean that infectious causes are diminished. It seems feasible to assume that the widespread use of safety seats in cars will have reduced the contribution of road traffic accidents to the total. On balance it seems most likely that postnatal causes will be less common than they were, and any fall in this group would imply a reduction in our projected prevalence of 2.5/1000.

Summary
It is estimated that the prevalence of non-genetic SMR associated with cerebral palsy has risen from 0.7 to about 0.9 per 1000 live births in the last decade. This is due to the predicted rise in total cerebral palsy prevalence to 2.5/1000 live births. This predicted prevalence of cerebral palsy is similar to that given for Western Australia in 1979–82, allowing for postnatal causes, but is higher than prevalence data from England and Sweden for that period. The estimated rise is due largely to improved survival and increased proportion of low birthweight babies since 1983 and also reflects the use of prevalence rates based on Mersey data.

Improvements in prenatal diagnosis, and a parental choice of selective termination may.
Cerebral palsy—an increasing contributor to severe mental retardation?

lead to reductions in other causes of SMR, such as Down's syndrome and neural tube defects, so it seems that children both physically and mentally handicapped due to brain damage will contribute a greater proportion of the SMR population. The careload of these children is greater than that associated with many other causes of SMR and most survive into adult life. The implications for planning future services will need to be recognised.

Unpublished data and advice from Dr G Hagberg, Dr F Stanley, Dr J Fertig, Dr P Watson, and Dr F Evans is gratefully acknowledged. The Office of Population Censuses and Surveys and the Government Actuary's Department kindly provided unpublished demographic data. The study has been supported by the Department of Health.

Addendum

The recent publication of F J Stanley and L Watson (Trends in perinatal mortality and cerebral palsy in Western Australia, 1967 to 1985, BMJ 1992;304: 1656-60) has shown that there are minor errors in table 1. For Western Australia total prevalence of cerebral palsy per 1000 live births (postnatal causes excluded) for the years 1975-8 and 1979-82 should read 1.92 and 1.88 (table 1: lines 19 and 20). Individual birthweight prevalences are correct. Thus our predicted prevalence for all cerebral palsy of 2.5/1000 live births is higher than any previous reports.


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