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CEREBRAL PALSY AND NEWBORN CARE. I: SECULAR TRENDS IN CEREBRAL PALSY

THIS set of papers* takes origin from recent publications on the relationship of newborn care to cerebral palsy, which have led to much optimism among those who care for newborns and handicapped children. These three annotations will each treat one of three factors that bear on current trends in the prevalence of neurological handicap; to reach a balanced judgment, all three must be weighed. The first set of publications to be considered are those of HAGBERG and colleagues¹⁻⁴, which document a significant downward trend in the prevalence rate of cerebral palsy among live births in Western Sweden between 1954 and 1970. This striking observation, placed in the context of the birthweight and pregnancy history of the affected cases, led the authors to conclude that the reason for the decline was the improvement in neonatal care for low-birthweight infants.

The second set of publications deals with trends in mortality rates for low-birthweight (LBW) babies over the past decade in Western countries. There is little doubt that there has been an increase in the proportion of survivors.

The third set of publications are studies of the rates of handicap among low-birthweight infants treated in modern neonatal intensive care units. Although, as we will detail, results from the several studies are far from uniform, a spirit of optimism pervades most of them, based on the rather consistent observation that the rates of impairment among survivors are lower than were found in some of the better-known studies of the previous era.

Each of these sets of studies raises important questions. The results of the Swedish workers lead us to expect a decline in cerebral palsy in other countries with well-developed maternity and newborn services. Is there evidence for such trends? If so, can they be

*This is part I of a three-part annotation: part II will be published in the October issue of the journal and part III in the December issue.

interpreted in terms both of birthweight-specific mortality and birthweight-specific rates of neurological impairment in survivors? What is owed to the level of newborn care (which could influence these rates of mortality and impairment in either direction)? Finally, in what ways do changes in mortality rates alter the risk of impairment among survivors, and do they do so in a direction counter to that achieved by level of newborn care?

We consider these questions in our three companion papers. In the first, we analyze secular trends in cerebral palsy in Western populations; in the second, we assess trends in both mortality and neurological impairment for LBW infants; in the final paper, we consider the conditions under which a decline in the prevalence rates of cerebral palsy and mental retardation can be expected.

Secular trends in prevalence rates of cerebral palsy

The frequency of chronic conditions which arise, or are thought to arise, from experiences in the perinatal period are most usefully expressed as a rate per live births. This is usually thought of as a prevalence rate⁵, although some epidemiologists term it incidence, or cumulative incidence. We raise this point here to indicate that we use the term prevalence rate to refer to the rate per live births and *not* per population alive at the time of the study. All the studies of cerebral palsy we will now discuss use this same definition of prevalence rate.

Several studies of time-trends in cerebral palsy have appeared in the recent medical literature. The rates in these studies of cerebral palsy of all types are shown in Figure 1; the rates of spastic diplegia only are shown in Figure 2. In Western Sweden, the prevalence of cerebral palsy syndromes among children born during the period from 1954 to 1970 decreased significantly and steadily, from 2.24 per 1000 in 1954-58 to 1.34 per 1000 in 1967-70¹⁻⁴. After the mid-1960s, the major decline in risk occurred among infants with birthweights of 2000g or less, and was most notable for spastic diplegia. The largest decline by 'cause' occurred in the category of 'perinatal causes'.

HAGBERG and colleagues inferred from these results that advances in newborn care had produced the observed decline in 'perinatal causes'. They inferred further that the residual causal factors in those forms of cerebral palsy which had not shown a decline—for instance cerebral palsy among children born at term—are prenatal in origin, and for that reason had not been influenced by advances in perinatal care. These factors they grouped together under the common heading 'fetal deprivation of supply'. The factors were unchanged in incidence over the period studied.

However, we note first that the decrease in the prevalence of spastic diplegia among surviving LBW infants began at least five years before the introduction of such new routines as the Usher regimen⁶ in the care of LBW babies in Sweden. Second, the reported decline in cerebral palsy in Western Sweden halted in the 1970s^{7, 8}. Preliminary data indicate a rate of 1.49 per 1000 livebirths in the period 1971-74, compared with 1.34 per 1000 in the period 1967-70. Moreover, it turns out that the Swedish observations cannot be readily generalized. Figures 1 and 2 do give an impression of a downward trend in other countries, but some populations show stable rates, other fluctuations, and still others rising rates.

Registry data from Eastern Denmark for the period 1950 to 1969 conform with the period of decline in Western Sweden. During the early 1950s the rate was about 2.6 per 1000 live births, and during the 1960s it declined to about 2.0 per 1000^{9, 10}. In an early survey done in Bristol, England, GRACE WOODS¹¹ had also found that the rate of cerebral palsy decreased from 2.5 per 1000 live births in the period 1943-48, to 1.6 per 1000 in the years 1953-58. She suggested that this reduction was owed to improved obstetric and premature-baby care. As

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the date of publication of this report was only five years after the birth of the youngest babies studied, one might worry that the younger cohort had less chance of being ascertained simply because they had lived fewer years than the older. The decline therefore might in part have been the result of an undercount in this cohort. Indeed GRIFFITHS and BARRETT¹² offered data from Birmingham, England, which were somewhat contrary: the rate of cerebral palsy was much the same in children born in 1948 and in 1959.

Rates over time inconsistent with a decline in the prevalence rate of cerebral palsy can also be found in other population studies. In Iceland during the time period 1953 to 1962, GUDMUNDSSON¹³ reported that the prevalence rate for cerebral palsy remained stationary at about 2.2 per 1000 live births. Over the period 1959 to 1966, in the 12 urban teaching hospitals co-operating in the Collaborative Perinatal Project (CPP) in the United States, there was little firm indication of a decline in either cerebral palsy or spastic diplegia¹⁴.

The most recent report is of cerebral palsy rates in Western Australia between 1956 and 1975, and it describes a fluctuating pattern¹⁵. In the period 1956-70, the prevalence rate increased from 2.5 per 1000 live births in 1956-60 to 2.9 per 1000 in 1961-65, to a peak of 3.0 per 1000 in 1966-70, then decreased to 1.6 per 1000 in the period 1971-75. Fluctuations were particularly marked in spastic diplegia. Birthweight data were available for total births

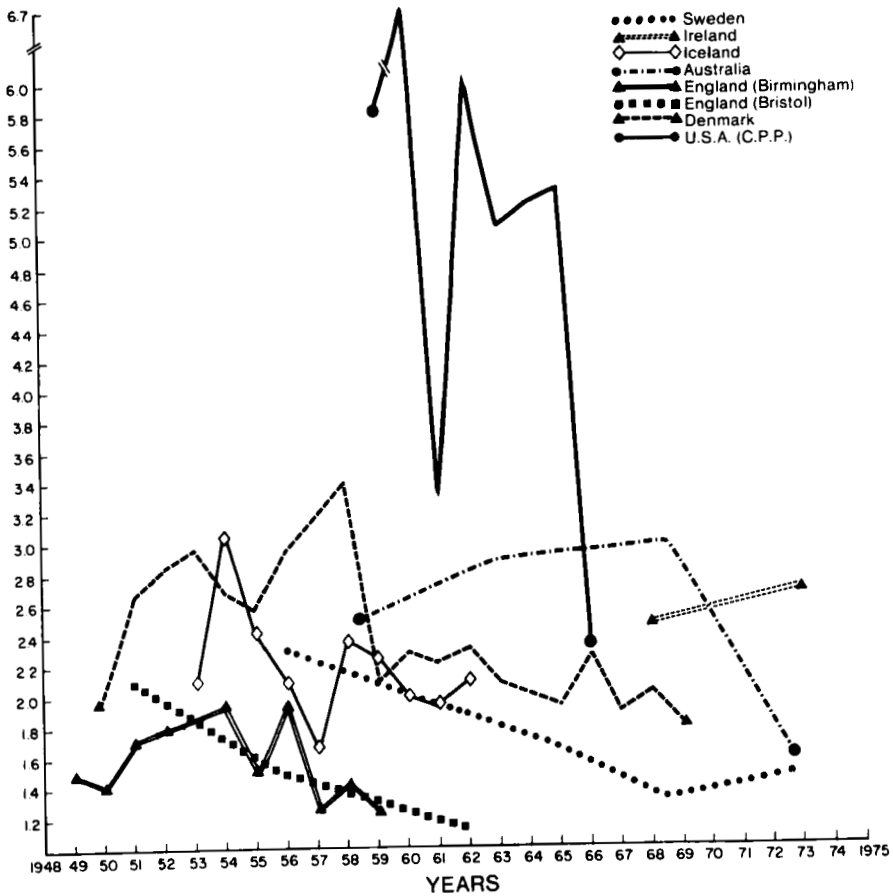


Fig. 1. Prevalence of cerebral palsy per 1000 live births.

in Western Australia only from 1968. The analyses made thereafter (1968-1971 vs 1972-75) do not conform with HAGBERG and colleagues' result in regard to birthweight: the decline in cerebral palsy occurred in normal as well as in LBW groups, and indeed was more marked in infants of normal birthweight (> 2500g).

Factors other than perinatal care may have contributed to these changes. In Western Australia in the time period under study, the birthweight distribution improved somewhat, in contrast to Sweden, the UK, and the USA^{2, 16, 17}. In 1968, 6 per cent of infants weighed less than 2500g compared with 5.3 per cent in 1974-75. The decline in low birthweight, as well as in the rate of multiple births, may have been the result of the decline in maternal age and parity that was also observed.

In Southern Ireland, the prevalence rate of cerebral palsy, about 2.4 per 1000 live births between 1966 and 1970, increased slightly to about 2.7 per 1000 between 1971 and 1975. In this study, CUSSEN *et al.*¹⁸ traced all children with cerebral palsy born in the counties of Cork and Kerry, and re-evaluated them according to HAGBERG and colleagues' definitions. The prevalence of spastic diplegia did in fact decline, although very slightly. An increase in prevalence was especially marked in cases related to 'prenatal' causes, and in those diagnosed as spastic quadriplegia, but at birthweights above 2500g. These changes were concomitant with a falling perinatal mortality rate over the 10-year period.

This Irish report is consistent with that of FRYERS and MACKAY¹⁹ from Salford, England,

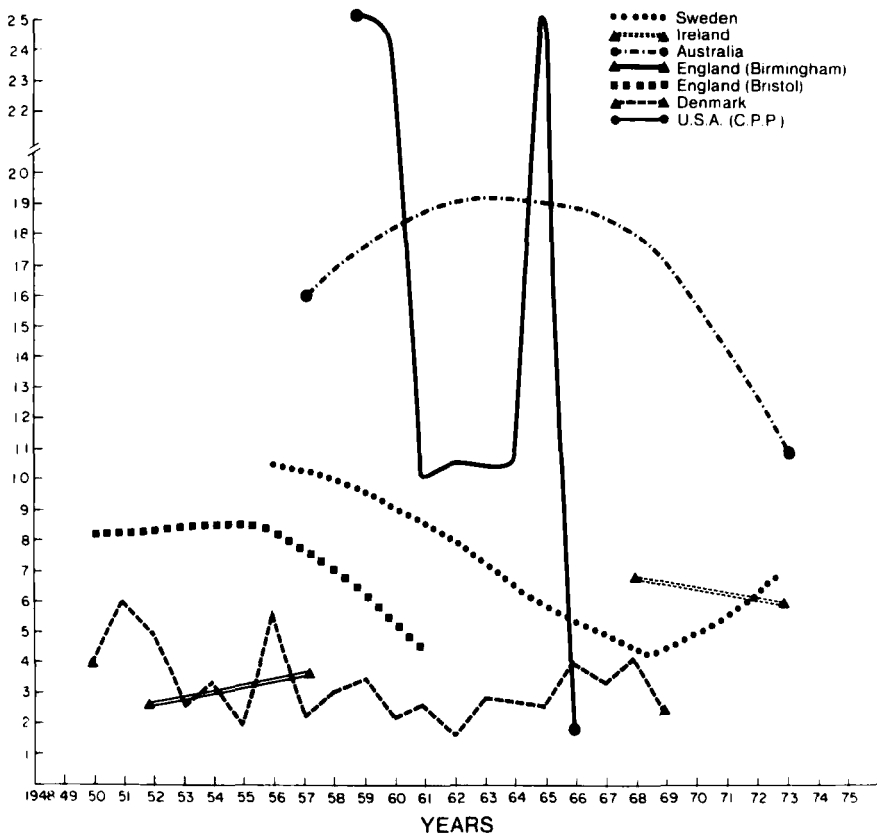


Fig. 2. Prevalence of spastic diplegia per 1000 live births.

on the prevalence rate for severe mental retardation. The Salford Mental Handicap Case Register routinely collected data on all mentally retarded children and adults²⁰. A marked shift in the age-specific prevalence of severe mental retardation to older age-groups had previously been observed in this register, and attributed to improved survival in the first years of life²¹. FRYERS and MACKAY¹⁹ studied temporal trends in severe mental retardation (IQ < 50) from 1951 to 1975. Rates were calculated for three five-year periods: 1961-1965, 1966-1970 and 1971-1975. The rate of severely mentally retarded cases with 'perinatal metabolic problems' more than doubled, from 0·5 per 1000 births in the first period, to 0·8 per 1000 in the second period, to 1·05 per 1000 in the third period. 'Perinatal metabolic problems' included only cases who in the newborn period had experienced prolonged apnea, cyanosis and obstetric difficulty. At the same time, the perinatal death-rate declined from 40 per 1000 births in 1963 to 25 per 1000 in 1973. FRYERS and MACKAY suggest that the increase in severe mental retardation associated with perinatal metabolic problems was the result of improved perinatal survival because of better obstetric and neonatal management.

Comparisons *between* countries in the prevalence of cerebral palsy should not be made by using the data presented in Figure 1. Different definitions of cerebral palsy are used; for example most exclude congenital malformations and postnatal causes, but some include one or the other or both. The CPP data from the United States are based on neurological examination of an entire cohort, hence many more mild cases are likely to be included than in series built on cases reported to treatment facilities. In addition, the age of ascertainment varies in different studies.

Despite the initially encouraging Swedish data, and the over-all impression of a declining trend, the notion of a steady reduction in cerebral palsy prevalence rates corresponding to improved neonatal care is not sustained by the available evidence. The Swedish decline, though chiefly involving cases of perinatal origin, does not decline over a time-period entirely coinciding with changes in perinatal care. In Ireland and in Salford, England, the rates of neurological impairment associated with perinatal causes actually increased. In sum, the pattern of time trends in cerebral palsy in western populations is a complex one. At the least, we must reserve judgment at this stage on the association of the trends with advances in perinatal care.

Summary

Reports of cerebral palsy prevalence rates per live births in recent decades in western nations show a mixed pattern. Declining rates were noted in Bristol, England, and in Denmark; but more recently a rising rate was noted in Ireland. In Western Sweden a decline has been followed by a recent rise, and in Western Australia a rise has been followed by a decline. Rates with not statistically significant changes have been found in Iceland and Birmingham, England, and over the seven-year span of the US Collaborative Perinatal Project, although their over-all direction was downward. No single factor is likely to explain the trends observed.

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PLASMA EXCHANGE IN ACUTE POST-INFECTIOUS DEMYELINATION

BETWEEN 1827 and 1836, Paris hospitals alone used five to six million leeches annually to suck 1,680,000 litres of blood from French people in order 'to suck all deleterious matter from the body'¹. There were certain to be successes; a parallel example is that improvement now is seen in as many as 60 per cent of multiple sclerosis sufferers receiving placebo therapy. Comparison may be drawn between the motives for leeching and its modern-day successor, plasma exchange (PE), of which there are now many reports claiming success in