conventional procedure, while nursing intervention is limited to periodic lifting of the bag containing concentrated proteins. In our experience this technique requires only minimal training of junior doctors and nurses, and it can be safely performed without supervision after being carried out three or four times. Moreover, this part of the procedure can now be automated by using a new dedicated machine (Bellco, Mirandola, Italy).

The use of less expensive albumin substitutes has also been recently suggested.[17 18] Polygeline was recently shown to be a safe replacement for human albumin for re-expanding plasma volume in cirrhotic patients with refractory ascites.[18] However, the efficacy and safety of long term use of these compounds in patients with relapsing ascites is still unknown. Planas *et al* showed that dextran 70 significantly increased plasma renin activity and plasma aldosterone concentrations in patients who had total paracentesis.[17] Their data suggest that dextran 70 is less effective than albumin in protecting patients from a decrease in intravascular volume.

In conclusion, our study indicates that short term treatment with spontaneous ascites filtration and reinfusion is safe and effective and that it is less expensive than total paracentesis plus intravenous albumin infusion for treating tense ascites. Further studies aimed at comparing the efficacy and safety of long term treatment with spontaneous ascites filtration and reinfusion with total paracentesis plus albumin or polygeline infusion in cirrhotic patients with recurrent ascites are warranted.

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Trends in perinatal mortality and cerebral palsy in Western Australia, 1967 to 1985

Fiona J Stanley, Linda Watson

Abstract

***Objective*—To analyse the trends in stillbirths, neonatal deaths, and cerebral palsy in all infants born in Western Australia from 1967 to 1985. To relate these trends to changes in perinatal care, particularly in relation to avoidance of intrapartum asphyxia in term infants and the increased survival of low birthweight infants.**

***Design*—Descriptive epidemiological study calculating population rates for perinatal deaths and cerebral palsy according to year of birth and birth weight.**

***Setting*—Western Australia.**

***Subjects*—All infants born after 20 weeks' gestation or weighing at least 400 g (live and stillborn).**

***Main Outcome Measures*—Stillbirths, neonatal deaths (from perinatal death certificates), and cerebral palsy (from a population based register).**

***Results*—Overall stillbirth rates fell from 12·1/1000 total births in 1967-70 to 8·1 in 1983-5. Early neonatal mortality fell from 13·0/1000 live births to 4·4 over the same period whereas total cerebral palsy rates remained at around 2-2·5/1000 live births. Death rates fell in all birth weight categories, particularly in low birthweight infants between 1975 and 1985, the period when birthweight data were available. In contrast, cerebral palsy rates in infants under 1500 g rose significantly over this period (from 12·1 in 1968 to 64·9 in 1985). The rise was seen in all spastic categories, including severely and multiply handicapped children.**

***Conclusions*—Large increases in the use of interventions aimed at reducing birth asphyxia and handicaps had not (by 1985) resulted in lower rates of cerebral palsy. This suggests that birth asphyxia is not a major cause. The increased survival of low birthweight infants has resulted in more cerebral palsy in this group, due either to postnatal complications of immaturity or prenatal damage to the fetal brain. These findings have implications for planning perinatal care and for litigation for putative obstetric malpractice in cerebral palsy cases.**

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Introduction

As the proportions of stillbirths and neonatal deaths have fallen in most developed countries interest has increased in using the trends of occurrence of cerebral palsy as a measure of the benefits and hazards of obstetric (intrapartum) and neonatal care. Most countries record deaths as part of their vital registration, but few have population data on cerebral palsy, which require special collection.[1]

There is also concern about the increasing expensive litigation in the United States,[2] United Kingdom,[3] and now Australia[4 5] for children born with cerebral palsy,

which is adversely affecting the provision and practice of obstetrics. Lawsuits related to cerebral palsy are rising despite the fact that recent research suggests that most cerebral palsy syndromes are not a result of perinatal asphyxia and therefore could not have been prevented by better obstetric care.[6,7] A recent article intimated that obstetricians had done themselves a disservice by promising that with interventions such as electronic fetal monitoring and caesarean sections most cases of cerebral palsy could be prevented and that few children would be born handicapped.[3]

We present the most recent data on the trends in perinatal deaths and cerebral palsy in Western Australia. These trends are discussed in relation to current practices in perinatal care.

Subjects and methods

In Western Australia stillbirth is defined as the death before delivery of a fetus of at least 20 weeks' gestation or 400 g birth weight and neonatal death as the death of an infant after birth and up to 28 days of life. Low birth weight was less than 2500 g, very low birth weight less than 1500 g, and preterm birth a delivery before 37 weeks' gestation.

Cerebral palsy is a group of non-progressive disorders of movement and posture due to a defect or lesion of the developing brain.[8] Proportions are calculated as the number of cases diagnosed by age 5 years per 1000 live births or neonatal survivors in that birth cohort.

All stillbirths and neonatal deaths were ascertained from perinatal death certificates, which have been recorded by birth weight and estimated gestational age since the 1960s in Western Australia. The definition of stillbirth changed in 1967 to include all stillbirths from 20 weeks' gestation (previously from 28 weeks); this also defines the lower gestational age limit for live births. Linked files of perinatal and infant death certificates and birth registrations were generously provided by the registrar general on an annual basis for our studies in perinatal epidemiology. This enabled social, demographic, and biological information on the parents to be analysed for all deaths.

Since 1975 the Western Australian midwives' notifications of births attended have also been available for all births from 20 weeks' gestation, or babies weighing from 400 g if gestation was not known. Since 1980 all these data have been linked to create the Western Australian maternal and child health database.

Cases of cerebral palsy were obtained from the Western Australian cerebral palsy register, which has collected basic diagnostic, perinatal, and demographic information on all individuals with a diagnosis of cerebral palsy born or living in Western Australia since 1956. Cases are ascertained from multiple sources and updated to the age of 5 years, by which time most cases will have come to light and a birth cohort can be considered complete. More details of the methods used for the register have been described.[1] In this study only data on children with cerebral palsy born during 1967 to 1985 were analysed, excluding all children not born in Western Australia and those whose cerebral palsy had a definite postneonatal cause. Children born in Western Australia who moved before cerebral palsy was diagnosed were traced to other Australian states by contacting all agencies, paediatric neurologists, and others diagnosing or managing children with these handicaps. Data for children born in 1985 have been recently updated, and we are fairly confident that the register data accurately reflect the cerebral palsy patterns for Western Australia during the study years.

Proportions of all infants who were stillborn, died in the neonatal period, or eventually had cerebral palsy diagnosed were calculated with the appropriate birth denominators (total births, live births, or neonatal survivors) from either birth registrations (1967 to 1974) or the midwives' notifications (1975 to 1985). Proportions specific for birth weight were calculated with the same denominators and standard birthweight categories: accurate data on birth weight were available only from 1975. From 1979 the birth weight was unknown in only a few cases, and we believe from a recent validation study that the recording of birth weight was accurate.[9]

Estimates of gestational age were more difficult. Before 1975 data on last menstrual period were not available for all births. From 1975 to 1979 gestational age could be estimated only from last menstrual period, and in many cases this was either unknown or uncertain. From 1980 onwards the midwives' data recorded last menstrual period, estimated date of delivery (which was updated by ultrasonography and other obstetric measures if available), and an estimate of maturity at birth. However, even the most recent estimates have inaccuracies and must be interpreted with caution. Estimation of completed weeks of gestational age for this analysis used the following hierarchy: menstrual data if accurate, estimated date of delivery, and then estimated maturity at birth.

STATISTICAL ANALYSIS

The effects of calendar time and birth weight on the various rates (stillbirth, neonatal death, and occurrence of cerebral palsy) were examined by Poisson regression[10] with the computer program EGRET (Epixact, version 0.02, 1985-1989, Serc and Cytel, Seattle, United States). This enabled estimation of trends and single category effects, and also of the significance of different trends with time for different birth weight or gestational age groups. For each outcome models were estimated for weight groups alone, then including the trend with time, and finally for the interaction of time with birth weight—that is, assessing the different time trends for each birthweight group. The trends in cerebral palsy subtypes were analysed in the same way. The data were grouped into three periods: 1975-8, 1979-82, and 1983-5 as there were too few children with cerebral palsy, particularly in the <1000 g category, to present data for single years.

Results

Figure 1 shows stillbirth, neonatal death, and cerebral palsy proportions from 1967 to 1985 in

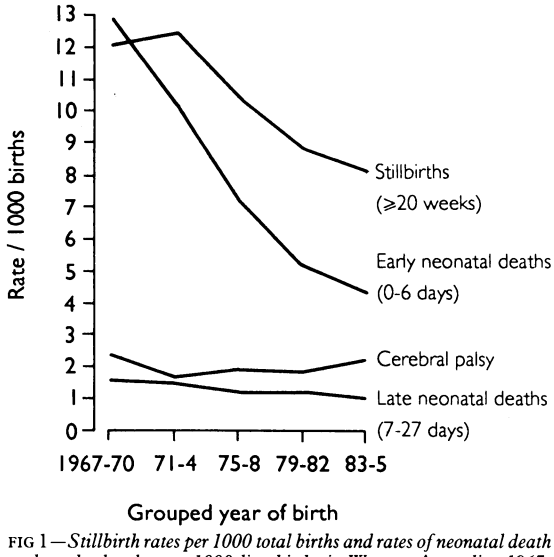

FIG 1—*Stillbirth rates per 1000 total births and rates of neonatal death and cerebral palsy per 1000 live births in Western Australia, 1967-1985 (grouped years)*

Western Australia. Stillbirth rates fell steadily from 12·1 in 1967-70 to 8·1 in 1983-5. Early neonatal mortality (0-6 days) also fell steeply from 13·0 to 4·4 in the same period, but late neonatal mortality fell less steeply (1·6 to 1·1). Proportions of infants with cerebral palsy remained steady at around 2/1000 live births over the 18 years. Significant changes in obstetric and neonatal care have occurred in Western Australia since 1967, including an increase in caesarean section deliveries from 4% in 1970 to 18% of all deliveries in 1985[11]; increased electronic intrapartum fetal monitoring of all births from 2·3% of deliveries in 1974 to 11·5% in 1982[12]; introduction of a neonatal transport service (in 1974); and an increase (from about 55% in 1975 to over 90% in 1985) in the proportion of live babies weighing under 1500 g delivered at the tertiary perinatal centre with access to neonatal intensive care.[13]

Table I and figure 2 show the numbers and proportions of stillbirths, neonatal deaths, and cases of cerebral palsy according to birth weight.

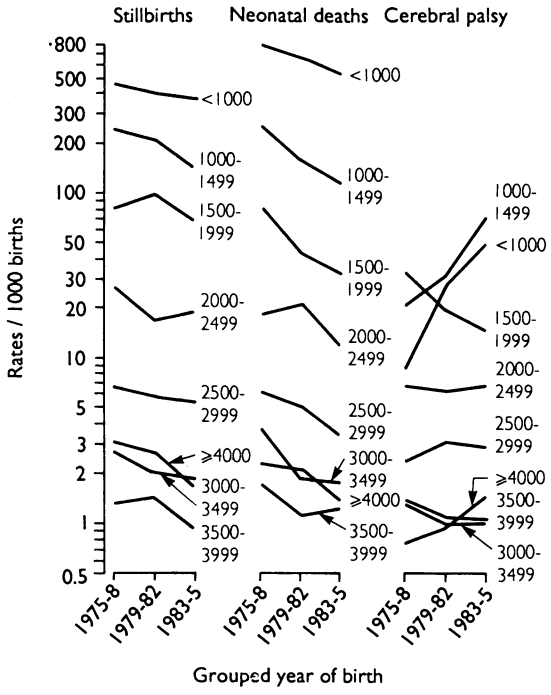

FIG 2—*Stillbirth rates per 1000 total births and rates of neonatal death and cerebral palsy per 1000 live births according to birth weight in Western Australia, 1975-1985 (grouped years)*

STILLBIRTHS

Stillbirth rates fell consistently with increasing birth weight to a constant rate of around 2/1000 total births for infants weighing 3000 g or more. Below this weight the rate in each birthweight group was significantly higher than that in the next highest group ($p<0·01$). The rates also fell significantly with time ($p<0·001$), with an overall fall of about 15% from one period to the next (rate ratio 0·85, 95% confidence interval 0·80 to 0·90). There was no significant difference in the fall with time between birthweight groups ($p=0·63$), the greatest fall being in the groups 1000-1499 g and ≥4000 g.

NEONATAL DEATHS

The pattern for neonatal death rates was similar to that for stillbirths but with a more rapid fall with increasing birth weight and a slightly lower rate for those weighing more than 3000 g. The fall in rate with time was also greater than for stillbirths with a decrease of about 25% per period (rate ratio 0·74, 0·69 to 0·79).

CEREBRAL PALSY

The rates of cerebral palsy also fell with increase in birth weight to a roughly constant rate for weights over 3000 g, but there was no significant trend with time for overall rates of cerebral palsy (rate ratio 1·03, 0·91 to 1·15; $p=0·67$). However, there were significant differences with time in the different birthweight groups ($p<0·001$). The most significant increases with time were in the birthweight groups <1000 g, and 1000-1499 g infants, with a fall in the group 1500-1999 g, and no change in the other birthweight groups.

Table II shows the cerebral palsy rates calculated per 1000 neonatal survivors, which may be more appropriate to compare than rates per 1000 live births. There was no overall significant trend with time (rate ratio 1·00, 0·89 to 1·12; $p=0·95$), but there were significant differences with time in the different birthweight groups ($p<0·005$). Thus, there was an increase of 53% (95% confidence interval −18% to 186%) per period in infants under 1000 g, an increase of 77% (18% to 165%)

TABLE I—*Total births, stillbirths, neonatal deaths, and births of children with cerebral palsy according to birth weight, Western Australia, 1975-85*

	Total births			No of stillbirths (rate/1000 total births)			No of neonatal deaths (rate/1000 live births)			No with cerebral palsy (rate/1000 live births)		
Birth weight (g)	1975-8	1979-82	1983-5	1975-8	1979-82	1983-5	1975-8	1979-82	1983-5	1975-8	1979-82	1983-5
<1000	464	443	368	214 (461·2)	176 (397·3)	136 (369·6)	194 (776)	174 (651·7)	119 (512·9)	2 (8)	7 (26·2)	11 (47·4)
1000-1499	461	539	466	113 (245·1)	110 (204·1)	63 (135·2)	87 (250)	66 (153·8)	45 (111·7)	7 (20·1)	13 (30·3)	27 (67)
1500-1999	876	968	842	71 (81·1)	73 (96·8)	56 (66·5)	64 (79·5)	38 (42·5)	25 (31·8)	25 (31·1)	17 (19·0)	11 (14·0)
2000-2499	2972	3181	2624	80 (26·9)	54 (17·0)	50 (19·1)	53 (18·3)	65 (20·8)	29 (11·3)	19 (6·6)	19 (6·1)	17 (6·6)
2500-2999	12917	13505	11019	87 (6·7)	79 (5·9)	59 (5·4)	78 (6·1)	67 (5·0)	36 (3·3)	29 (2·3)	40 (3·0)	30 (2·7)
3000-3499	30165	31971	26011	82 (2·7)	65 (2·0)	46 (1·8)	110 (3·7)	58 (1·8)	45 (1·7)	38 (1·3)	30 (0·9)	25 (1·0)
3500-3999	23705	26151	20812	31 (1·3)	37 (1·4)	19 (0·9)	40 (1·7)	28 (1·1)	25 (1·2)	31 (1·3)	27 (1·0)	21 (1·0)
≥4000	8152	9062	7088	25 (3·1)	24 (2·7)	12 (1·7)	20 (2·5)	18 (2·1)	8 (1·3)	6 (0·7)	8 (0·9)	8 (1·3)
Unknown	3425	109	190	164	127	124	46	16	1	1		
Total	83137	85929	69420	867 (10·4)	745 (8·7)	565 (8·1)	692 (8·4)	530 (6·2)	333 (4·8)	158 (1·9)	161 (1·9)	150 (2·2)

TABLE II—*Rates of cerebral palsy per 1000 neonatal survivors according to birth weight, Western Australia, 1975-85*

	1975-8			1979-82			1983-5		
	No of neonatal survivors	No with cerebral palsy	Rate/1000 neonatal survivors	No of neonatal survivors	No with cerebral palsy	Rate/1000 neonatal survivors	No of neonatal survivors	No with cerebral palsy	Rate/1000 neonatal survivors
<1000	56	2	35·7	93	7	75·3	113	11	97·3
1000-1499	261	7	26·8	363	13	35·8	358	27	75·4
1500-1999	741	25	33·7	857	17	19·8	761	11	14·5
2000-2499	2839	19	6·7	3062	19	6·2	2545	17	6·7
≥2500	74467	104	1·4	79992	105	1·3	64680	84	1·3
Unknown	3243	1		637			68		
Total	81607	158	1·9	85004	161	1·9	68525	150	2·2

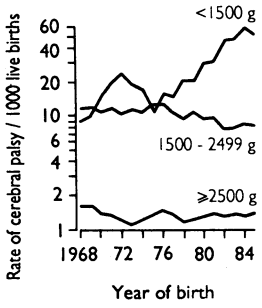

FIG 3—*Rates of cerebral palsy in very low birthweight (<1500 g), low birthweight (1500-2499 g), and normal birthweight (≥2500 g) infants per 1000 live births in Western Australia, 1968-1985, shown as three year moving averages*

per period in those weighing 1000-1499 g, a fall of 36% (8% to 55%) per period in those weighing 1500-1999 g, and almost no change in the other birthweight groups.

As expected the rates of cerebral palsy by birth weight calculated per 1000 neonatal survivors (table II) were higher than when calculated per 1000 live births (table I). The rates rose between 1975 and 1985 but less steeply than when calculated with the live birth denominators. The relative rate for the <1000 g group comparing 1983-5 with 1975-8 was 5·9 (confidence interval 1·3 to 26·7) with live births as denominators and 2·7 (0·6 to 12·3) with neonatal survivors. Similarly, in the 1000-1499 g group the relative rate (comparing 1983-5 with 1975-8) was 3·3 (1·5 to 7·6) with live births and 2·8 (1·2 to 6·5) with neonatal survivors as denominators.

The three year moving averages for cerebral palsy in very low birthweight, low birthweight, and normal birthweight categories from 1968 to 1985 (fig 3) show the changes in rates of cerebral palsy in infants <1500 g that have occurred since 1978, coinciding with falls in neonatal mortality in these infants. The proportion of all cerebral palsy that occurred in very low birthweight infants has increased over the period (in the <1000 g group from 1·3% in 1975-8 to 7·3% in 1983-5; in 1000-1499 g group from 4·4% in 1975-8 to 18·0% in 1983-5).

Though data on gestational age were available from 1979 onwards, the accuracy was uncertain, as described in the methods. With this in mind trends with gestational age were calculated from 1979 in categories 20-27, 28-30, 31-32, 33-34, 35-36, 37-42, and 43-50 completed weeks since last menstrual period. The trends by gestational age were similar to those by weight.

The categorisation of cerebral palsy syndromes into spastic hemiplegia, diplegia, and quadriplegia and other non-spastic syndromes (for example, ataxic, athetoid, or dystonic syndromes) is difficult and not often reliably reproducible between observers.[14] However, each case on the Western Australian register had a main motor handicap recorded, and in 85% of children with cerebral palsy this was spasticity. Table III shows the rates per 1000 neonatal survivors for the spastic syndromes. Interestingly, the main syndromes contributing to the rise in low birthweight cerebral palsy were spastic hemiplegia and quadriplegia.

Hemiplegia—Over all years the highest rates were in infants under 1000 g, and there was a steady falling proportion as birth weight increased. There was no overall trend with time; the 3% fall per period was not significant (p=0·8). However, there was a 50% increase per period in both the <1000 g and 1000-1499 g groups (p<0·03) and a non-significant fall in higher birthweight groups.

Diplegia—Over all years the highest rates were in infants weighing 1000-1499 g with steady falls as birth weight increased. There was a 17% fall per period in total diplegia rates (p=0·06). Although rates in low birthweight groups increased in each period, there was no significant trend (p=0·11). The steady fall in heavy diplegic infants across periods was not significant.

Quadriplegia—Over all weights there was a 22% increase per period (p=0·18) in rates of quadriplegia. In the <1000 g group there was a 70% increase and in the 1000-1499 g group a 150% increase (p<0·04) per period, with a non-significant fall in other groups.

When rates of cerebral palsy by birth weight were calculated per 1000 live births for each subtype similar patterns were observed except that the rise in spastic diplegia in infants <1500 g was significant (p<0·03).

The numbers of cases of ataxic, athetoid, or dystonic cerebral palsy were small and showed no real trend over time, although dyskinetic cerebral palsy rates seemed to have risen in recent years, mainly in infants born at term.

TABLE III—*Trends in spastic cerebral palsy motor categories by birth weight: rates per 1000 neonatal survivors in Western Australia, 1975-85*

	1975-8		1979-82		1983-5	
Birth weight (g)	No	Rate	No	Rate	No	Rate
		Hemiplegia				
<1000	1	17·86	3	32·26	5	44·25
1000-1499	1	3·83	7	19·28	7	19·55
1500-1999	10	13·50	3	3·50	2	2·63
2000-2499	6	2·11	6	1·96	7	2·75
≥2500	36	0·48	47	0·59	28	0·43
		Diplegia				
<1000	1	17·86	3	32·26	3	26·55
1000-1499	6	22·99	4	11·02	14	39·11
1500-1999	10	13·50	10	11·67	8	10·51
2000-2499	9	3·17	6	1·96	4	1·57
≥2500	35	0·47	25	0·31	14	0·22
		Quadriplegia				
<1000	0	—	1	10·75	1	8·85
1000-1499	0	—	2	5·51	6	16·76
1500-1999	5	6·75	2	2·33	1	1·31
2000-2499	2	0·70	2	0·65	2	0·79
≥2500	14	0·19	16	0·20	19	0·29

Discussion

We have presented unique population data on perinatal mortality and cerebral palsy over a period during which obstetric and neonatal care has changed dramatically. Several interesting patterns were observed, some of which were unexpected.

Most developed countries have shown improved perinatal mortality rates similar to those reported here for Western Australia. These improvements have been associated with more widespread use of obstetric and neonatal interventions aimed at detecting and reducing intrapartum morbidity, particularly from intrapartum asphyxia, and treating the complications of extreme immaturity. Changes in the population distribution of maternal demographic and biological characteristics such as falling extremes of parity and age, improved height, and decreases in other risk factors also may have contributed to better perinatal outcomes.

Rates of cerebral palsy remained steady throughout the study period. This is consistent with recent data which indicate that the proportion of cases of cerebral palsy which are due to birth asphyxia, or which could be avoided by perinatal care, is much less than previously thought.[15-17] These findings are most important in relation to litigation as well as to the possible causes and prevention of cerebral palsy. Obstetricians should heed recent advice that they are "shooting themselves in the foot" if they continue to promise a perfect baby in the face of a constant cerebral palsy rate of 2-2·5 per 1000.[3]

The rise in cerebral palsy in low birthweight infants mirrors falls in perinatal mortality and particularly neonatal mortality in such infants. But advances in neonatal intensive care have resulted in gains as well as losses, and there are now many more low birthweight survivors who do not have cerebral palsy.[5]

Rising rates of cerebral palsy in low birthweight infants are being reported in Sweden, the United Kingdom, Ireland, and Finland.[18-21] No population data are yet available from the United States, where obstetric and neonatal interventions occurred earlier and are more widely used than in other developed countries. Perinatal mortality rates in low birthweight infants tend to be lower in the United States than elsewhere although overall mortality is higher.[22] This may reflect their more aggressive policy of improving low birthweight survival.

Though similar methods have been used since the register was established in 1978, our ascertainment of

rural, Aboriginal, postneonatal, and mild cases of cerebral palsy was probably more complete in recent years than in the earlier years. This did not seem to result in selectively better ascertainment of cases in infants of low birth weight. Also, there has recently been considerable effort to reduce interobserver variation in diagnosis of specific motor handicap in the register.[14] Since we believe that this has reduced diagnostic variation in infants born since 1975, the comparison of specific diagnostic categories before and after this time may be difficult.

CEREBRAL PALSY AND LOW BIRTH WEIGHT

There are several possible explanations of the rise in cerebral palsy in low birthweight infants. One theory is that as more low birthweight infants survive they suffer the complications of extreme immaturity such as periventricular leucomalacia, intraventricular haemorrhage, respiratory distress, and sepsis, resulting in postnatal brain damage in otherwise normal infants.[23,24] This hypothesis has important implications for neonatal intensive care.

Another hypothesis is that cerebral palsy and preterm birth have similar origins in pregnancy and that more of these children, who were compromised well before birth, are now surviving in increasing numbers.[19] This has implications for further research into the aetiology and future prevention of the cerebral palsies and preterm birth. Another Australian study of cerebral palsy in very low birthweight infants could not identify any obvious preventable factors[25]; though many of the infants with cerebral palsy had perinatal risk factors, they were also present in low birthweight children without cerebral palsy. Other data from the United Kingdom[23] and United States[6,26] support this finding. There are certainly reports now of white matter necrosis having been present well before birth in low birthweight infants dying in the neonatal period.[27-29]

The rise in cerebral palsy in preterm infants was not mainly in spastic diplegia, which might be expected if the first hypothesis accounted for most of the increase. Early studies of low birthweight infants who subsequently were noted to have cerebral palsy reported that most had spastic diplegia, which was often mild and associated with minimal other handicap. In particular, they tended to be of normal intelligence.[30-32] In more recent studies the rise in occurrence of cerebral palsies in low birthweight infants seems to be in all spastic syndromes and associated with other handicaps, many of which are classified as severe.[18,19] This finding tends to support the second hypothesis. However, recent neuropathological studies of white matter necrosis occurring postnatally in very low birthweight babies suggest a much broader distribution beyond that of the periventricular region with widespread necrosis in the subcortex and elsewhere.[33,34] Thus we believe that while it is likely that both antenatal and postnatal events can result in cerebral palsy in very low birthweight infants, a considerable contribution must be coming from the postnatal complications of extreme prematurity.

ANTENATAL CAUSES

Our study, along with other recent published work,[35] also suggests that the intrapartum antecedents of cerebral palsy syndromes may be less important, particularly in infants born at term, than those occurring antenatally. Certainly the central nervous system is known to be vulnerable for longer than many other fetal organs, and neurones are still migrating during the middle trimester and therefore vulnerable to influences which could interfere with migration.[36,37] Genetic factors, early pregnancy teratogens, and mutagens such as viruses and iodine deficiency are known causes of cerebral palsy syndromes.[38,39] We recently reported a strong association between spastic cerebral palsy and intrauterine growth retardation, which results from a poor antenatal environment.[7]

Studies must also continue on the neonatal and perinatal problems encountered by very preterm, low birthweight neonates and the extent to which modern techniques can improve their care and provide an early prediction of their neurological status. In the meantime, data such as ours can provide information on the planning of appropriate services, including family counselling and support, for children with these handicaps. They should also be made widely available to lawyers, judges, parents, and obstetricians to educate them about the putative causes of cerebral palsy.

The Western Australian cerebral palsy register depends on the continued participation of paediatric neurologists and developmental paediatricians in Western Australia. We thank them for their work and the health department and registrar general for access to data on births and deaths. Help with computing was provided by Ms Maxine Walton. Drs Nick de Klerk and Anne Read helped with statistical advice and Peripheral Engineering provided graphical help. The study was funded by the Child Health Research Foundation of Western Australia (previously the TVW Telethon Foundation for Medical Research).

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Alcohol consumption as a risk factor for poor outcome after aneurysmal subarachnoid haemorrhage

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Abstract

***Objective*—To evaluate the effect of factors existing before aneurysmal subarachnoid haemorrhage on outcome of haemorrhage.**

***Design*—Prospective follow up study.**

***Setting*—Helsinki University Hospital.**

***Patients*—291 consecutive patients (149 men) aged 15 to 65 years admitted within 96 hours after the bleeding.**

***Main outcome measures*—Potential risk factors (baseline characteristics, health habits, and clinical variables) for poor outcome after haemorrhage (dependent state in the activities of daily living, or death) were studied using multiple logistic regression.**

***Results*—One year after haemorrhage, 179 (62%) patients were independent in the activities of daily living and 28 (10%) dependent; 84 (29%) had died. Risk of poor outcome was predicted, after adjustment for sex and age, by clinical condition at admission according to the Glasgow coma scale ($p<0.0001$); occurrence of rebleeding (relative risk 7·1, 95% confidence interval 2·8 to 18·0, $p<0.0001$) or delayed cerebral ischaemia (10·3, 4·2 to 25·4, $p<0.0001$); surgery on an aneurysm (0·13, 0·05 to 0·35, $p<0.0001$); and heavy consumption of alcohol (4·5, 1·8 to 11·0, $p=0.0014$). Heavy drinking remained a significant risk factor after additional adjustment for hypertension, body mass index, and presence of intracerebral haematoma. Heavy drinkers had a more unfavourable outcome after rebleeding and delayed ischaemia than did others with rebleeding or ischaemia. Those who had salicylates in urine on admission had delayed ischaemia with fixed neurological deficits less commonly than others.**

***Conclusions*—Heavy drinking impairs outcome mainly through severe rebleeding and delayed ischaemia and to a lesser extent through a poor initial condition and presence of intracerebral haematoma.**

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Introduction

Aneurysmal subarachnoid haemorrhage is a serious disease with high mortality and morbidity. Delayed cerebral ischaemia and rebleeding are the most important causes of death and disability, after the primary bleed.[1-3] The risk of delayed ischaemia is best predicted by the amount of subarachnoid[4-6] and intraventricular[5] blood, as well as by previous hypertension,[6] irrespective of clinical condition on admission or hydrocephalus. There are no uniformly accepted predictive risk factors for rebleeding except the administration of antifibrinolytic drugs, which reduce this risk.[4,5] The overall outcome can best be predicted by the initial clinical condition and the amount of subarachnoid blood as a poor initial condition is associated with mortality due to the primary bleed.[4,5]

Besides these independent risk factors, which are associated with the severity of haemorrhage, there may also be pre-ictal factors other than hypertension that can impair the outcome. It is not known whether alcohol consumption, smoking, or the use of non-steroidal anti-inflammatory drugs influence outcome. Alcohol consumption, especially heavy drinking, has been shown to increase the risk of all types of stroke,[7-11] while smoking increases the risk of subarachnoid haemorrhage[12-15] and cerebral infarction.[10,11,15] Use of non-steroidal anti-inflammatory drugs seems to increase the risk of haemorrhagic stroke.[16] Hence, these factors might also impair outcome after subarachnoid haemorrhage by making patients prone to cerebral ischaemia, rebleeding, or a severe initial bleed. Awareness of factors worsening the outcome could be important in the management of subarachnoid haemorrhage. This prospective study investigated the influence of these factors on the outcome after subarachnoid haemorrhage.

Patients and methods

From January 1985 to September 1986 a total of 291 patients with aneurysmal subarachnoid haemorrhage were admitted to the Helsinki University Hospital. After they had been admitted, I interviewed patients, their family members, and fellow workers, as appropriate, about smoking and alcohol habits, use of medicines, and previous diseases of the patient. Data were obtained solely from the patients themselves in 42 cases and from family members only in 55 cases or fellow workers only in six cases when the patient was in poor condition. In the remaining 188 cases information was obtained from both patients and family members. In about a quarter of these cases either the patient or the family member underestimated or overestimated alcohol intake. Because underestimation is more likely in alcohol intake, the greater amounts reported in these cases were registered.

Alcohol consumption, both recent and long term, was calculated from the amount of alcohol consumed before haemorrhage. Patients with two or more positive answers to the four questions in the CAGE interview were considered to be "CAGE positive," a classification that correlates well with heavy drinking.[17,18] On admission, blood samples were obtained for measurement of markers of alcohol intake (erythrocyte mean cell volume, γ-glutamyltransferase concentration, and platelet count). Cigarette smoking was categorised as never smoked, former smoker, and current smoker—≤ 10, 11-20 >20 cigarettes a day, respectively. Use of