

How Often Do Perinatal Events at Full Term Cause Cerebral Palsy?

Savas M. Menticoglou, MDCM, FRCSC

Department of Obstetrics, Gynaecology and Reproductive Sciences, University of Manitoba, Winnipeg MB

Abstract

Objective: To determine the contribution of perinatal events to cerebral palsy in children born at full term.

Methods: The delivery records of a cohort of babies born at full term in one tertiary care hospital over an 11-year period were reviewed. The obstetric history and neonatal chart of each baby admitted to the Neonatal Intensive Care Unit was then examined. For those babies whose stay in the NICU was because of encephalopathy, brain injury, asphyxia with organ dysfunction, serious infection, or prolonged respiratory support, a review of their medical records was undertaken to determine how many subsequently developed cerebral palsy.

Results: Of 36 368 babies born at term, 20 were later diagnosed as having cerebral palsy in which the causative insult likely occurred just before, during, or shortly after labour and delivery. This is an incidence of cerebral palsy arising from perinatal events of 0.55 per 1000 deliveries. Only six cases, however, were deemed to have been possibly preventable by better obstetric care.

Conclusion: In our hospital, perinatal events are an important cause of cerebral palsy in children born at full term, but few cases are potentially preventable.

Résumé

Objectif : Déterminer l'apport des événements périnataux en ce qui concerne l'infirmité motrice cérébrale chez les enfants nés à terme complet.

Méthodes : Les dossiers d'accouchement d'une cohorte d'enfants nés à terme complet au sein d'un hôpital de soins tertiaires sur une période de 11 ans ont été analysés. Les antécédents obstétricaux et les dossiers néonataux de chaque enfant admis à l'unité néonatale de soins intensifs ont par la suite été examinés. Dans le cas des enfants dont le séjour à l'UNSI était attribuable à une encéphalopathie, à une lésion cérébrale, à une asphyxie accompagnée d'un dysfonctionnement touchant un organe, à une infection grave ou à un soutien respiratoire prolongé, une analyse de leurs dossiers médicaux a été entreprise afin de déterminer combien d'entre eux ont, par la suite, connu une infirmité motrice cérébrale.

Résultats : Vingt des 36 368 enfants nés à terme ont, par la suite, obtenu un diagnostic d'infirmité motrice cérébrale, pathologie dont l'accident causal s'est probablement produit tout juste avant, pendant ou peu après le travail et l'accouchement. Ce qui donne une incidence d'infirmité motrice cérébrale attribuable à des événements périnataux de 0,55 par 1 000 accouchements. Cependant, il a été estimé que seulement six de ces 20 cas auraient pu être prévenus par la mise en œuvre de meilleurs soins obstétricaux.

Conclusion : Au sein de notre hôpital, bien que les événements périnataux constituent une cause importante d'infirmité motrice cérébrale chez les enfants nés à terme complet, peu de ces cas peuvent être prévenus.

J Obstet Gynaecol Can 2008;30(5):396-403

INTRODUCTION

Labour and delivery can be dangerous for both mother and baby. Catastrophes such as cord prolapse, ruptured uterus, or placental abruption may occur. The repetitive contractions of a long labour can cause hypoxia and metabolic acidosis. The passage of the head through the pelvis can lead to intracranial trauma. Organisms in the birth canal can cause severe infections. Meconium aspiration can result in newborn respiratory failure. These dangers can cause neonatal death or damage the brain, resulting in cerebral palsy.

Obstetricians' interest in the etiology of cerebral palsy is focused on prevention. Historically, it has been assumed that most cerebral palsy is caused by fetal trauma or asphyxia occurring around the time of birth.¹⁻³ In the last two decades, the view has been advanced that cerebral palsy is rarely caused by perinatal events⁴⁻⁸ and that obstetricians can do little to prevent it.^{9,10} Even if perinatal events are only rarely responsible for later cerebral palsy, late pregnancy and labour and delivery are the periods of highest risk in relative terms.¹¹ This is the time when increased fetal surveillance takes place and where prevention may be most possible.

Key Words: Cerebral palsy, perinatal events, preventability, obstetricians

Competing Interests: None declared.

Received on August 3, 2007

Accepted on October 2, 2007

The purpose of this study was to answer two questions:

1. In a cohort of babies born at term, how often do perinatal events contribute to the later development of cerebral palsy?
2. What proportion of such cases might have been prevented by different obstetrical care?

For this study, the term perinatal was used in a general sense to mean around the time of birth—from several hours or days prior to delivery, to the first hours or days after delivery.¹²

MATERIALS AND METHODS

The records from deliveries occurring at Women's Hospital in Winnipeg, Manitoba, over an 11-year period (January 1, 1982, to December 31, 1992) were examined. This hospital is one of the two teaching hospitals for obstetrics and gynaecology at the University of Manitoba. It is one of two centres providing tertiary level obstetric care for the province of Manitoba, and it also provides obstetric care for much of the city of Winnipeg. During the period under review there were about 3500 deliveries per year at the hospital.

The Women's Hospital and the Children's Hospital are part of a larger entity, the Health Sciences Centre. All babies born at the Women's Hospital who need intensive care are admitted to the Children's Hospital. The Children's Hospital NICU also receives babies from other hospitals, but for this study only the records of babies born at the Women's Hospital were reviewed.

In each year of the study period, 1% to 2% of babies born at full term at Women's Hospital were admitted to the NICU. The obstetric history and neonatal course of each infant admitted was reviewed. The main focus of the review was to identify those babies who may have suffered morbidity from events around the time of birth and to determine if cerebral palsy subsequently developed. The following cohorts were reviewed:

1. all babies with evidence of neonatal encephalopathy, such as altered tone for more than a few hours, decreased level of responsiveness, seizures, or feeding difficulties;

2. babies with evidence on imaging of intracranial hemorrhage, infarction, or cerebral edema;
3. babies with non-brain organ dysfunction, possibly due to hypoxic acidemia, that could not be attributed to pre-existing problems;
4. babies with proven or strongly suspected sepsis, meningitis, or herpes infection; and
5. babies who needed ventilation support for whatever reason for more than 48 hours, even if there had not been respiratory depression at birth or thereafter, or any baby who was therapeutically paralyzed for respiratory support.

Babies admitted to the NICU for reasons unrelated to perinatal events (e.g., because of structural anomalies, hydrops, chromosomal abnormalities, genetic syndromes, inborn errors of metabolism, neuromuscular disorders, or congenital infections such as cytomegalovirus infection or rubella) were excluded from the review. Babies admitted primarily for observation (e.g., because of intrauterine growth restriction or maternal drug exposure), or for prophylactic treatment (e.g., after a brief course of antibiotics for possible but unproven sepsis, hypoglycemia in the infant of diabetic mother, hyperbilirubinemia) were also excluded. Babies were also excluded if they were admitted only for respiratory problems (e.g., pneumothorax, transient tachypnea, pneumonia, or meconium aspiration), if the duration of respiratory support was less than 48 hours, if no therapeutic paralysis was required, or if there were no abnormal neurologic findings.

The long-term neurodevelopmental outcome for babies admitted to NICU was mostly accessible from their medical records. The Children's Hospital is the only hospital in the city of Winnipeg that admits children for medical or surgical care. It has the only pediatric emergency department and pediatric subspecialty clinics in the province of Manitoba, and all pediatricians practising in the city admit patients to the hospital. Accordingly, if a child was admitted at some point for surgery or medical care, attended a specialty clinic, or came to the emergency department, it was usually possible to determine from the clinical notes if there was a neurological problem. For babies who had had moderate or severe encephalopathy, follow-up was carried out in the developmental clinic or in the pediatric neurology clinic. Babies with Apgar scores of less than 4 at five minutes of life, babies with neurological findings, or babies with any CNS abnormality (such as intracranial hemorrhage, seizures, or meningitis) were seen in the neonatal high-risk follow-up program. In the few instances in which the medical records were not informative about the neurodevelopmental outcome, information about the child's

ABBREVIATIONS

BD	base deficit
CNS	central nervous system
CS	Caesarean section
DIC	disseminated intravascular coagulation
FHR	fetal heart rate
MRI	magnetic resonance imaging
NICU	neonatal intensive care unit
US	ultrasound

Table 1. Late antenatal, prelabour

Case	Obstetrical event	Condition at birth	Neonatal course	Outcome
1.	Acute onset of abdominal pain at home; fetal bradycardia on admission; CS 50 minutes after onset of pain; placental abruption	3250 g Apgar 0 at 5 minutes	Early onset seizures CT head → cerebral edema EEG → severely abnormal	Spastic quadriplegia Cortical blindness Severe developmental delay Seizures
2.	Admitted in labour; persistently "flat" fetal heart rate tracing; normal vaginal delivery 90 minutes after arrival; true knot in cord.	2930 g Apgar 3 ¹ 5 ⁵ Cord artery pH 7.29	Severe neonatal encephalopathy CT normal ¹³	Choreoathetoid CP Mental retardation Seizures
3.	Induced labour postdates; worrisome fetal heart rate tracing but normal scalp pH; normal vaginal delivery	3985 g Apgar 2 ¹ 5 ⁵ Cord artery pH 7.33	Severe neonatal encephalopathy Seizures CT → cerebral edema ¹³	Spastic quadriplegia Mental retardation Seizures Microcephaly
4.	3 days decreased fetal movement; sinusoidal fetal heart rate tracing; large fetomaternal hemorrhage; CS without labour	3140 g Apgar 2 ¹ 4 ⁵ Cord artery pH 7.39 Hemoglobin 48	Heart failure → transfused Seizures Hyponatremia	Spastic quadriplegia Normal intelligence
5.	24 hours decreased fetal movement; fixed bradycardia; large fetomaternal hemorrhage; CS without labour	3200 g Apgar 2 ¹ 4 ⁵ Hemoglobin 42	Seizures CT head → diffuse severe cerebral edema EEG → severely abnormal	Died at 6 months
6.	Intrauterine growth restriction; biophysical profile score 4/10; CS without labour	2310 g Apgar 5 ¹ 8 ⁵ Cord artery pH 7.08	No encephalopathy Surgical repair small sacrococcygeal teratoma	Spastic quadriplegia Developmental delay
7.	Maternal viral-like illness in days before labour; unremarkable labour and normal delivery	3445 g Apgar 7 ¹ 8 ⁵ Cord artery pH 7.25	Hypotonia; Pleocytosis in cerebrospinal fluid; ? viral meningitis	Ataxic cerebral palsy Developmental delay

outcome was obtained after written communication with the child's pediatrician or family doctor.

Review of all hospital records and other written communications was carried out by the author, an obstetrician. In those cases where the follow-up suggested any neurologic disturbance, the hospital chart and other information was reviewed in detail by a developmental pediatrician to determine if cerebral palsy was present.

RESULTS

Between 1982 and 1992, 36 368 babies were born at full term, and 667 of these babies (1.8%) were admitted to the NICU. Twenty-two children had been born at term, had received care in the NICU, and were later found to have cerebral palsy that could not be attributed to etiologies of prenatal origin. Cerebral palsy in two children was attributed to a postneonatal event. One child with a birth weight of 4610g had received care in the NICU for hypoglycemia, possible sepsis, and an Erb's palsy, with complete recovery. There were no neurodevelopmental concerns regarding this child until the age of 14 months, when he sustained a skull fracture and other injuries in a car accident, and at 16

months he was found to have a hemiparesis. The second child required an intravenous glucose infusion for less than 24 hours for hypoglycemia. Development was felt to be normal up to three months of age, at which time he presented with new onset seizures and apnea, and required manual ventilation for 30 minutes in a remote community. At two years of age, this child had severe cerebral palsy and neurodevelopmental dysfunction. Brain imaging, metabolic, genetic, and infectious investigations showed no abnormality. The neurodevelopmental dysfunction was attributed to the prolonged apnea or possibly to a microscopic CNS abnormality.

These exclusions left 20 children with cerebral palsy possibly related to events occurring around the time of birth. In seven of the 20 cases the insult that caused the brain injury almost certainly occurred in the days or hours before labour (Table 1).

In Case 1, the mother was admitted to hospital with an acute placental abruption, and, although Caesarean section was performed 50 minutes after the onset of pain, the baby was born with marked respiratory depression. In Cases 2 and 3 (previously described in detail¹³), the babies had

severe encephalopathy; however, umbilical cord blood gases at delivery were normal, indicating that intrapartum hypoxia was unlikely. No other explanation for encephalopathy was found. In Cases 4 and 5, the cerebral insult was attributed to massive fetomaternal hemorrhage. In Case 6, fetal assessment was carried out because of a maternal perception of decreased fetal movements; a low biophysical profile score, intrauterine growth restriction, and a small sacrococcygeal teratoma were identified, and delivery was subsequently expedited by CS before labour. In this case, the baby had metabolic acidosis but no encephalopathy. In Case 7, the mother had been unwell for some days before the onset of labour with a presumed viral illness; the baby was born in good condition after a normal labour but was hypotonic for several days. Postnatal assessment showed pleocytosis in the cerebrospinal fluid but no specific diagnosis.

In the remaining 13 cases, the inciting insult for cerebral palsy possibly began during labour. In seven cases (Cases 8 to 14) it is unlikely that obstetrical intervention, short of CS before labour, would have changed the outcome (Table 2). In Case 8, the baby was apparently healthy until five hours of age, when the first signs of overwhelming sepsis were noted. In Case 9, the FHR tracing was abnormal, and the baby was born depressed with metabolic acidosis, but delivery occurred 40 minutes after arrival at the hospital. In Case 10, the baby became ill only at 72 hours of age, and thalamic and intraventricular hemorrhage was diagnosed. In Case 11, the baby was the presenting twin during labour. The mother had refused intervention until the FHR tracing had become very abnormal, and the baby was acidotic at delivery. In Case 12, CS was performed because of fetal distress. Neurologic abnormalities were noted in the nursery, and a CT scan confirmed cerebral infarction due to occlusion of the right middle cerebral artery. In Case 13, the mother was an alcohol and solvent abuser and was admitted to hospital at full cervical dilatation with the umbilical cord prolapsed. Delivery was carried out promptly, but the baby did not breathe spontaneously for the first five minutes of life. In Case 14, rupture of membranes had occurred two days prior to admission of the mother to the hospital, and the FHR tracing showed absent reactivity and variability. Profound fetal bradycardia occurred 95 minutes after admission, prompting delivery by emergency CS.

In six cases (Cases 15–20) it is possible or likely that different obstetrical care during labour might have prevented the cerebral palsy (Table 3). In Case 15, rupture of the uterus occurred at full cervical dilatation. In Case 16, there was a 90-minute delay in delivery by CS after fetal acidosis was identified by scalp capillary sampling. In Case 17, the scalp capillary pH initially was normal, but a repeat pH

measurement was not performed despite continued abnormality of the FHR tracing. The baby was severely acidotic at delivery three hours later. In Case 18, CS was performed only after the FHR tracing had been abnormal for some time. In Case 19, the baby suffered a high cervical cord injury after delivery by forceps rotation. In Case 20, neonatal respiratory arrest occurred, likely attributable to an interhemispheric hematoma that may have resulted from a low forceps delivery.

The inclusion of some cases can be disputed.¹⁴ In two children (Cases 5 and 18), the diagnosis of cerebral palsy was made even though they died before one year of age, because it was felt that their degree of brain damage was sufficient to make the diagnosis certain had they lived long enough.¹⁴ In the child with high cervical cord spinal trauma (Case 19), the lesion was not cerebral, but it was included because the child's quadriplegia was clearly attributable to birth trauma,¹⁵ and such cases have previously been included in cerebral palsy cohorts.^{2,16}

DISCUSSION

The contribution of perinatal events to cerebral palsy has been much disputed. In the past, most cases of cerebral palsy were attributed to the events around labour and delivery^{1–3}; then it was claimed that the events around birth were almost never responsible.^{4–10} MRI studies in term newborns with encephalopathy have now indicated that the brain injury usually occurs near the time of birth.^{17,18} Even if late pregnancy and intrapartum events contribute only in a minor way to cerebral palsy, intervention during this period, in which there is increased fetal surveillance, might offer the best chance to prevent some cases.

In the present study, over an 11-year period in a single tertiary care hospital, 20 of 36 368 babies born at full term were later diagnosed with cerebral palsy that was likely caused by an insult occurring just before, during, or shortly after labour and delivery. This is an incidence of cerebral palsy arising from perinatal events of 0.55 per 1000 deliveries. There were at most six cases (0.16 per 1000 deliveries) in which better obstetrical care might have avoided the poor outcome (Table 3).

One obvious consideration in these estimates is that the babies with cerebral palsy were identified only by follow-up of the 1.8% of term newborns who had required admission to the NICU. Babies with complicated births who were born in good condition, or who responded promptly to resuscitation, and who had a smooth neurological course with no need for monitoring or involved respiratory care, were usually not admitted to the NICU; such asymptomatic newborns are not at increased risk of cerebral palsy.¹⁹ Some babies with mild encephalopathy, such as jitteriness or

Table 2. Intrapartum, non-preventable

Case	Obstetrical event	Condition at birth	Neonatal course	Outcome
8.	CS for failure to progress in labour; no fetal distress	3480 g Apgar 9 ¹ 9 ⁵	Well until 5 hours of age; Clinical overwhelming sepsis DIC (all cultures negative)	Spastic quadriplegia Mental retardation Seizure disorder Death at 5 years
9.	Normal vaginal delivery 40 minutes after arrival to hospital; FHR tracing equivocal	2130 g Apgar 1 ¹ 6 ⁵ Cord artery pH 7.07 BD -14	Severe meconium aspiration; Persistent pulmonary hypertension; Seizures; cranial ultrasound→edema CT→ hyperdense changes	Spastic quadriplegia Cortical blindness; Severe developmental delay
10.	Normal vaginal delivery; no fetal distress	2860 g Apgar 8 ¹ 9 ⁵ Cord artery pH 7.27	Well until 72 hrs, then lethargy, poor feeding, seizures. Cranial ultrasound and CT→ right thalamic hemorrhage with intraventricular hemorrhage; Ventriculoperitoneal shunt at 4 weeks	Seizure disorder Left hemiparesis
11.	Presenting twin in labour; maternal refusal of continuous electronic FHR monitoring; abnormal FHR trace at 7 cm; scalp pH 7.04; CS 36 minutes later.	3265 g Apgar 2 ¹ 5 ⁵ First capillary gas at 20 minutes age pH 7.12, BD 9	Seizures day 1 Depressed consciousness for several days	Microcephaly Seizure disorder Cortical blindness Spastic diplegia
12.	FHR decelerations during labour; scalp pH 7.18; CS for fetal distress	3440 g Apgar 7 ¹ 9 ⁵ Cord artery pH 7.14	Slow feeds, abnormal tone; cranial US/CT→ occlusion of right middle cerebral artery and cerebral infarction	Seizures Left hemiparesis Learning disabilities
13.	Alcohol/solvent user; arrival at hospital with feet and umbilical cord outside vulva, palpable pulse, easy breech extraction	2350 g Apgar 2 ¹ 4 ⁵ Head circumference 31 cm; No spontaneous respiration for 5 mins; Extubated at 10–15 min; At 40 mins, capillary gas pH 7.26, BD -7.5	Feeding well at 11 hours of age; No encephalopathy; Normal head US day 4	Microcephaly Seizure disorder Profound mental retardation. Spastic quadriplegia
14.	Ruptured membranes 48 hours; transfer to tertiary hospital; profound fetal bradycardia 95 minutes after arrival; CS performed, marked torsion of umbilical cord noted	2620 g Apgar 0 at 5 min. first arterial gas at 15 minutes age (after 5 mmol HCO ₃) pH 7.02 BD -13	Seizures; coma: prolonged absence gag/suck reflexes; Ultrasound head→ generalized brain edema	Spastic quadriplegia Developmental delay

extreme alertness, might not have been admitted to the NICU, but such neonates virtually never go on to develop cerebral palsy.^{20–24} The diagnosis of neonatal encephalopathy might not have been made clinically in some infants who were therapeutically paralyzed for respiratory care, but all of these infants were followed up to exclude sequelae. The main interest of this study was to determine how frequently cerebral palsy could be ascribed to immediate perinatal events, for which obstetrical intervention was possible. The reasoning was that if a baby was in good clinical condition soon after birth and never developed a problem needing admission to NICU, then it was very unlikely that later cerebral palsy could be ascribed to

acute perinatal events that the obstetrician could have prevented.^{7,25,26}

More than one half of all children who develop cerebral palsy are born at full term.^{27,28} Among children born at term, the incidence of cerebral palsy is reported to be between 1.1 per 1000 and 1.5 per 1000 deliveries.^{29–35} Manitoba has no cerebral palsy register, so the overall cerebral palsy rate in term babies is unknown. However, if it is similar to the rate in developed countries, then the incidence of cerebral palsy due to perinatal events in this study (0.55 per 1000 deliveries) would make perinatal events responsible for one third to one half of all cerebral palsy in babies delivered at full term. This is a higher proportion

Table 3. Intrapartum, possibly preventable

Case	Obstetrical event	Condition at birth	Neonatal course	Outcome
15.	Multipara; induced for hypertension; severe fetal bradycardia at full dilatation; failed forceps delivery; ruptured uterus at CS	4330 g Apgar 1 ¹ 2 ⁵ Cord artery pH 6.66	Early onset seizures Prolonged hypotonia	Spastic quadriplegia Mental retardation Seizures
16.	Morbidly obese mother; late decelerations in labour; scalp pH 7.06; CS 90 minutes later	2500 g Apgar 0 ¹ 3 ⁵ Cord artery pH 6.63	Severe neonatal encephalopathy Seizures	Spastic quadriplegia Developmental delay
17.	Late decelerations at 9 cm; scalp pH 7.30; vacuum delivery 3 hours later	2710 g Apgar 2 ¹ 3 ⁵ Cord artery pH 6.83	Meconium aspiration syndrome Prolonged hypotonia	Predominant left hemiparesis Developmental delay
18.	Poor intrauterine growth; presenting twin, induced labour; bradycardia; emergency CS	2365 g Apgar 0 ¹ 3 ⁵ Cord artery pH 7.04 BD -21	Severe neonatal encephalopathy Seizures	Severe neurologic abnormalities Developmental delay Died before 1 year
19.	Delivery by midforceps rotation	3500 g Apgar 1 ¹ 3 ⁵ Cord artery pH 7.1	High cervical cord injury	C1-C2 quadriplegia Seizure disorder
20.	Low forceps after 20 minute second stage, indication unclear	3200 g Apgar 8 ¹ 9 ⁵ Bilateral cephalhematomas	In normal nursery until 106 hrs of age, when respiratory arrest possibly due to aspiration of feed; hemoglobin dropped 40 gm/L since birth; CT scan day 6→ hematoma in interhemispheric fissure closely related to tentorium and moderate ventricular dilatation	Mild spastic diplegia Developmental delay

than is generally accepted.⁴⁻⁸ However, the rates in studies published in the last decade are not dissimilar. In a single county in Norway, between 1970 and 1999, the incidence of cerebral palsy among children with birth weight ≥ 2500 g was 1.5 per 1000 deliveries and 39% of these (0.59 per 1000) were deemed to arise from perinatal events.³⁶ In one region of Finland, between 1978 and 1982, the prevalence of cerebral palsy in term babies was 1.28 per 1000 deliveries, of which 24% (0.30 per 1000) were attributed to perinatal events.³⁷ In southwest Germany and western Sweden, between 1975 and 1986, 34% of 208 cases of cerebral palsy in term babies were judged to be perinatal in origin.³⁸ In Western Australia, between 1976 and 1985, 17 of 51 term babies with spastic quadriplegic cerebral palsy (33%) had probable birth asphyxia.³⁹ In western Sweden, between 1991 and 1998, the rate of cerebral palsy among term live births was 1.2 per 1000 births, with 35% (0.41 per 1000) acquired perinatally.^{40,41} In two studies of children with cerebral palsy who were born at full term, children with a history of neonatal encephalopathy represented 24%⁴² and 29%³⁴ of the total cases of term cerebral palsy after exclusion of cases with anomalies and postnatal causes. MRI studies of term neonates with encephalopathy show signs of recent brain injury in most cases.^{17,18} This suggests that

brain injury in most newborns with encephalopathy occurs in the immediate perinatal period.

Obviously, the contribution of perinatal events to the incidence of cerebral palsy is modified by obstetric and neonatal practices and interventions. Many Caesarean sections or operative vaginal deliveries are undoubtedly able to prevent or mitigate asphyxia in the fetus and newborn⁴³ and thus prevent cerebral damage. However, some Caesarean sections and operative deliveries are able to prevent an intrapartum death, but the surviving babies are impaired⁴⁴ (as in Cases 1, 6, 11, 16, and 17). These effects, working in opposite directions, may explain our inability to show that increasing CS rates have decreased the rate of cerebral palsy.^{45,46} The effect of neonatal care is similarly uncertain. Expert neonatal care probably prevents brain damage from developing in some critically ill neonates. In other cases, however, intensive neonatal care, by preventing the death of critically ill babies (such as Cases 2, 3, 4, 5, 8, 9, and 19) may lead to survivors who develop cerebral palsy. Other very ill newborns with severe brain damage may have their care withdrawn, so that the result is a perinatal death instead of a survivor with cerebral palsy.^{32,47}

In this study and others,^{34,36-42} perinatal events have been shown to be more important contributors to the incidence

of cerebral palsy at full term than is generally conceded.^{4–10} Indeed, new speculation about the role of intrapartum infection and the later development of cerebral palsy may indicate an even greater contribution of perinatal events.⁴⁸ Most cases of perinatal stroke leading to cerebral palsy are overlooked in the neonatal period, and so the contribution of perinatal events to the incidence of cerebral palsy may again be underestimated.^{49,50}

Furthermore, it may be wrong to presume that the only adverse outcomes possible from perinatal asphyxia are death or disability—of which cerebral palsy must be a component—as is claimed by some prominent bodies.⁵¹ Cognitive deficits without cerebral palsy have been reported in children following perinatal asphyxia and moderate encephalopathy.^{52–54}

One view currently being advanced is that nothing obstetricians do is likely to affect the occurrence of cerebral palsy.^{10,55} It is true that obstetricians are not blameworthy in most cases of cerebral palsy.^{56–58} It is also unclear whether a substantial proportion of these cases can be prevented by obstetrical intervention without harming many more women than the few babies who benefit. Nevertheless, having a fatalistic view about what obstetricians can or cannot do is not conducive to improving perinatal outcomes.

ACKNOWLEDGEMENTS

The author wishes to thank Diane Moddemann, Department of Pediatrics and Child Health, University of Manitoba, for her assistance in the chart reviews.

REFERENCES

- Raju TNK. Historical perspectives on the etiology of cerebral palsy. *Clin Perinatol* 2006;33:233–50.
- Eastman NJ, DeLeon M. The etiology of cerebral palsy. *Am J Obstet Gynecol* 1955;69:950–61.
- Quilligan EJ, Paul RH. Fetal monitoring: is it worth it? *Obstet Gynecol* 1975; 45: 96–100.
- Nelson KB, Ellenberg JH. Antecedents of cerebral palsy. Multivariate analysis of risk. *N Engl J Med* 1986;315:81–6.
- Blair E, Stanley FJ. Intrapartum asphyxia: a rare cause of cerebral palsy. *J Pediatr* 1988; 112:515–9.
- Nelson KB. What proportion of cerebral palsy is related to birth asphyxia? *J Pediatr* 1988; 112:572–4.
- Freeman JM, Nelson KB. Intrapartum asphyxia and cerebral palsy. *Pediatrics* 1988; 82:240–9.
- Badawi N, Kurinczuk JJ, Keogh JM, Alessandri LM, O'Sullivan F, Burton PR, et al. Intrapartum risk factors for newborn encephalopathy: the Western Australian case-control study. *BMJ* 1998; 317:1554–8.
- The origins of cerebral palsy—a consensus statement. The Australian and New Zealand Perinatal Societies. *Med J Aust* 1995;162:85–90.
- Strijbis EM, Oudman I, van Essen P, MacLennan AH. Cerebral palsy and the application of the international criteria for acute intrapartum hypoxia. *Obstet Gynecol* 2006;107:1357–65.
- Gardosi J. Monitoring technology and the clinical perspective. *Baillieres Clin Obstet Gynaecol* 1996;10:325–39.
- Prenatal and perinatal factors associated with brain disorders. U.S. Department of Health and Human Services. Public Health Service. National Institutes of Health. NIH Publication No. 85–1149. April 1985:10.
- Menticoglou SM, Manning FA, Harman CR, Morrison I. Severe fetal brain injury without evident intrapartum asphyxia or trauma. *Obstet Gynecol* 1989;74:457–61.
- The definition and classification of cerebral palsy. *Dev Med Child Neurol* 2007;49:1–44.
- Menticoglou SM, Perlman M, Manning FA. High cervical spinal cord injury in neonates delivered with forceps: report of 15 cases. *Obstet Gynecol* 1995;86(4Pt 1):589–94.
- Mutch L, Alberman E, Hagberg B, Kodama K, Perat MV. Cerebral palsy epidemiology: where are we now and where are we going? *Dev Med Child Neurol* 1992;34:547–55.
- Cowan F, Rutherford M, Groenendaal F, Eken P, Mercuri E, Bydder GM, et al. Origin and timing of brain lesions in term infants with neonatal encephalopathy. *Lancet* 2003;361:736–42.
- Miller SP, Ramaswamy V, Michelson D, Barkovich AJ, Holshouser B, Wycliffe N, et al. Patterns of brain injury in term neonatal encephalopathy. *J Pediatr* 2005;146:453–60.
- Nelson KB, Ellenberg JH. The asymptomatic newborn and risk of cerebral palsy. *Am J Dis Child* 1987;141:1333–5.
- Robertson C, Finer N. Term infants with hypoxic-ischemic encephalopathy: outcome at 3.5 years. *Dev Med Child Neurol* 1985;27:473–84.
- Robertson CM, Finer NN, Grace MG. School performance of survivors of neonatal encephalopathy associated with birth asphyxia at term. *J Pediatr* 1989;114:753–60.
- Hill A. The predictive significance of clinical measures of brain injury in the newborn. *Clin Invest Med* 1993;16:141–8.
- Levene MI, Sands C, Grindulis H, Moore JR. Comparison of two methods of predicting outcome in perinatal asphyxia. *Lancet* 1986;1(8472):67–9.
- Hull J, Dodd KL. Falling incidence of hypoxic-ischaemic encephalopathy in term infants. *Br J Obstet Gynaecol* 1992;99:386–91.
- Towbin A. Obstetric malpractice litigation: the pathologist's view. *Am J Obstet Gynecol* 1986;155:927–35.
- Volpe JJ. Hypoxic-ischemic encephalopathy: clinical aspects. In: Volpe JJ, ed. *Neurology of the newborn*. 4th ed. Philadelphia: WB Saunders; 2001:367.
- Thorngren-Jerneck K, Herbst A. Perinatal factors associated with cerebral palsy in children born in Sweden. *Obstet Gynecol* 2006;108:1499–505.
- Bax M, Tydeman C, Flodmark O. Clinical and MRI correlates of cerebral palsy: the European Cerebral Palsy Study. *JAMA* 2006;296:1602–8.
- Colver AF, Gibson M, Hey EN, Jarvis SN, Mackie PC, Richmond S. Increasing rates of cerebral palsy across the severity spectrum in north-east England 1964–1993. The North of England Collaborative Cerebral Palsy Survey. *Arch Dis Child Fetal Neonatal Ed* 2000;83: F7-F12.
- Rosen MG, Dickinson JC. The incidence of cerebral palsy. *Am J Obstet Gynecol* 1992;167:417–23.
- Cummins SK, Nelson KB, Grether JK, Velie EM. Cerebral palsy in four northern California counties, births 1983 through 1985. *J Pediatr* 1993;123:230–7.
- Topp M, Uldall P, Greisen G. Cerebral palsy births in eastern Denmark, 1987–90: implications for neonatal care. *Paediatr Perinat Epidemiol* 2001;15:271–7.
- Surveillance of cerebral palsy in Europe: a collaboration of cerebral palsy surveys and registers. Surveillance of Cerebral Palsy in Europe (SCPE). *Dev Med Child Neurol* 2000;42: 816–24.

34. Badawi N, Felix JF, Kurinczuk JJ, Dixon G, Watson L, Keogh JM, et al. Cerebral palsy following term newborn encephalopathy: a population-based study. *Dev Med Child Neurol* 2005;47:293–8.
35. Wu YW, Croen LA, Shah SJ, Newman TB, Najjar DV. Cerebral palsy in a term population: risk factors and neuroimaging findings. *Pediatrics* 2006;118:690–7.
36. Meberg A, Broch H. Etiology of cerebral palsy. *J Perinat Med* 2004;32:434–9.
37. Riikonen R, Raumavirta S, Sinivuori E, Seppala T. Changing pattern of cerebral palsy in the southwest region of Finland. *Acta Paediatr Scand* 1989;78:581–7.
38. Krageloh-Mann I, Hagberg G, Meisner C, et al. Bilateral spastic cerebral palsy—a collaborative study between southwest Germany and western Sweden. III: Aetiology. *Dev Med Child Neurol* 1995;37:191–203.
39. Stanley FJ, Blair E, Hockey A, Petterson B, Watson L. Spastic quadriplegia in Western Australia: a genetic epidemiological study. I: Case population and perinatal risk factors. *Dev Med Child Neurol* 1993;35:191–201.
40. Hagberg B, Hagberg G, Beckung E, Uvebrant P. Changing panorama of cerebral palsy in Sweden. VIII. Prevalence and origin in the birth year period 1991–94. *Acta Paediatr* 2001;90:271–7.
41. Himmelmann K, Hagberg G, Beckung E, Hagberg B, Uvebrant P. The changing panorama of cerebral palsy in Sweden. IX. Prevalence and origin in the birth-year period 1995–1998. *Acta Paediatr* 2005;94:287–94.
42. Gaffney G, Flavell V, Johnson A, Squier M, Sellers S. Cerebral palsy and neonatal encephalopathy. *Arch Dis Child Fetal Neonatal Ed* 1994;70:F195–200.
43. Low JA, Pickersgill H, Killen H, Derrick EJ. The prediction and prevention of intrapartum fetal asphyxia in term pregnancies. *Am J Obstet Gynecol* 2001;184:724–30.
44. Hagberg B, Hagberg G, Zetterstrom R. Decreasing perinatal mortality—increase in cerebral palsy morbidity. *Acta Paediatr Scand* 1989;78:664–70.
45. Scheller JM, Nelson KB. Does caesarean delivery prevent cerebral palsy or other neurologic problems of childhood? *Obstet Gynecol* 1994;83:624–30.
46. Clark SL, Hankins GD. Temporal and demographic trends in cerebral palsy—fact and fiction. *Am J Obstet Gynecol* 2003;188:628–33.
47. Ryan CA, Byrne P, Kuhn S, Tyebkhan J. No resuscitation and withdrawal of therapy in a neonatal and a pediatric intensive care unit in Canada. *J Pediatr* 1993;123:534–8.
48. Wu YW, Escobar GJ, Grether JK, Croen LA, Greene JD, Newman TB. Chorioamnionitis and cerebral palsy in term and near-term infants. *JAMA* 2003;290:2677–84.
49. Lynch JK, Nelson KB. Epidemiology of perinatal stroke. *Curr Opin Pediatr* 2001;13:499–505.
50. Wu YW, Lynch JK, Nelson KB. Perinatal arterial stroke: understanding mechanisms and outcomes. *Semin Neurol* 2005;25:424–34.
51. American College of Obstetricians and Gynecologists. American Academy of Pediatrics. Neonatal encephalopathy and cerebral palsy: defining the pathogenesis and pathophysiology. Washington, DC:ACOG;2003:xvii.
52. Robertson CMT. Long-term follow-up of term infants with perinatal asphyxia. In: Stevenson DK, Benitz WE, Sunshine P, eds. *Fetal and neonatal brain injury*, 3rd ed. Cambridge: Cambridge University Press; 2003:829–58.
53. Lindström K, Lagerroos P, Gillberg C, Fernell E. Teenage outcome after being born at term with moderate neonatal encephalopathy. *Pediatr Neurol* 2006;35:268–74.
54. Gonzalez FF, Miller SP. Does perinatal asphyxia impair cognitive function without cerebral palsy? *Arch Dis Child Fetal Neonatal Ed* 2006;91:F454–9.
55. Nelson KB. Can we prevent cerebral palsy? *N Engl J Med* 2003;349:1765–9.
56. Illingworth RS. Why blame the obstetrician? A review. *Br Med J* 1979;1(6166):797–801.
57. Niswander KR. The obstetrician, fetal asphyxia, and cerebral palsy. *Am J Obstet Gynecol* 1979;133:358–61.
58. Stanley FJ, Blair EM. Obstetrical responsibility for abnormal fetal outcome. In: Chamberlain G, Steer P, eds. *Turnbull's Obstetrics*. 3rd ed. London: Churchill Livingstone; 2001:709–19.