Potentially asphyxiating conditions and spastic cerebral palsy in infants of normal birth weight

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OBJECTIVE: Our purpose was to examine the association of cerebral palsy with conditions that can interrupt oxygen supply to the fetus as a primary pathogenetic event.
STUDY DESIGN: A population-based case-control study was performed in four California counties, 1983 through 1985, comparing birth records of 46 children with disabling spastic cerebral palsy without recognized prenatal brain lesions and 378 randomly selected control children weighing ≥2500 g at birth and surviving to age 3 years.
RESULTS: Eight of 46 children with otherwise unexplained spastic cerebral palsy, all eight with quadriplegic cerebral palsy, and 15 of 378 controls had births complicated by tight nuchal cord (odds ratio for quadriplegia 18, 95% confidence interval 6.2 to 48). Other potentially asphyxiating conditions were uncommon and none was associated with spastic diplegia or hemiplegia. Level of care, oxytocin for augmentation of labor, and surgical delivery did not alter the association of potentially asphyxiating conditions with spastic quadriplegia. Intrapartum indicators of fetal stress, including meconium in amniotic fluid and fetal monitoring abnormalities, were common and did not distinguish children with quadriplegia who had potentially asphyxiating conditions from controls with such conditions.
CONCLUSION: Potentially asphyxiating conditions, chiefly tight nuchal cord, were associated with an appreciable proportion of unexplained spastic quadriplegia but not with diplegia or hemiplegia. Intrapartum abnormalities were common both in children with cerebral palsy and controls and did not distinguish between them. (Am J Obstet Gynecol 1998;179:507-13.)

Key words: Birth asphyxia, tight nuchal cord, maternal infection, Apgar scores, neonatal seizures, cerebral palsy

Approximately 1 of 1500 infants born weighing ≥2500 g has disabling cerebral palsy.[1] Cerebral palsy in infants of normal birth weight has greatly influenced medical practice because of the assumption that this disorder is chiefly the result of asphyxial birth and is preventable if detected early. This assumption has been an important factor in the development and widespread use of electronic fetal monitoring and other forms of antenatal and intrapartum surveillance and has influenced the rate of surgical delivery.[2] In spite of these changes in medical management, the cerebral palsy rate in term infants has not declined in recent decades.[3, 4]

Assessment of the contribution of asphyxial events to cerebral palsy is complicated by the fact that there is no generally available tool for direct measurement of birth asphyxia. In the absence of a validated means for its recognition, birth asphyxia is commonly diagnosed on the basis of fetal or neonatal signs such as abnormalities on electronic fetal monitoring, low Apgar scores, need for respiratory support, and neonatal encephalopathy. The specificity of these signs to asphyxial states has not been established.

It is not known how much of perinatal hypoxia or ischemia, when present, is primary and how much is a downstream consequence of other initiating pathologic features. This distinction is potentially important because identification of factors that initiate pathogenesis may influence strategies for primary prevention.

In a large cohort of births in the mid-1980s we sought to evaluate the contribution of birth asphyxia to

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cerebral palsy. We did not wish to assume that signs of stress in the fetal or neonatal period usually meant that the initiating pathologic feature was asphyxial in nature. Instead, the approach we chose was to identify a priori a number of birth complications that can limit oxygen supply or blood flow to the fetus as a primary pathogenetic event and to investigate the association of these factors, separately and together, with cerebral palsy and with clinical signs in the intrapartum and neonatal periods.

Methods

The outcome of this study was spastic cerebral palsy, which was more likely than purely ataxic or dyskinetic forms to be related to birth events. Cerebral palsy was defined as a chronic disability originating in the central nervous system, characterized by aberrant control of movement or posture, appearing early in life and not the result of progressive disease. Children in whom disability was acquired after the first 28 days of life or through nonaccidental head trauma in the first month and children with mild involvement or pure hypotonia were not included.

Case patients were singleton children born from 1983 through 1985 to residents of 4 San Francisco Bay area counties and included all children who met the following criteria: weighed ≥2500 g at birth, survived to age 3 years, were residents of California to that age, and had moderate or severe congenital cerebral palsy. For initial ascertainment of cases we used records of 2 state agencies known to enroll virtually all eligible children, determining final case status on the basis of standardized clinical examination or extensive record review. Assessment of severity was based on functional ability of the most affected limb: severe meant that that limb had no useful function and moderate that some function was preserved although assistive devices were usually required. Detailed information on ascertainment procedures is available elsewhere.[5] Case children had a median age of 4.9 years at examination. Controls were randomly selected from singleton children who met all the case criteria except for cerebral palsy. Demographic and clinical data were obtained from birth certificates and medical records at >40 hospitals in the 4 counties. Maternal labor and delivery and neonatal records were abstracted by nurses who did not know the purpose of the study or whether the records were those of case or control children. Nurse abstractors noted diagnoses and descriptions recorded by medical caregivers and did not infer diagnoses from clinical signs and symptoms. Information on neonatal characteristics came from the hospital of birth and all other inpatient admissions before infants' first discharge to their homes.

Potentially birth-asphyxiating conditions were defined a priori to include a clinical diagnosis of abruptio placentae (complete or partial), placenta previa, large placental infarctions, prolapsed cord, cord compression, maternal shock, true cord knot, or tight nuchal cord. These diagnoses were as recorded in maternal records. We did not include information on placental vasculopathies because it is not clear that the chief consequence of such lesions, when present, is reduction in oxygenation and because of data (unpublished) indicating that such lesions are not reliably ascertained.

We defined maternal infection, a previously identified risk factor for cerebral palsy,[6] on the basis of diagnoses recorded by clinical caregivers, including a clinical diagnosis of chorioamnionitis, histologic diagnosis of inflammation of placental membranes, gross or microscopic diagnosis of funisitis or cord inflammation, maternal temperature during labor >100.4°F or 38°C, foul-smelling amniotic fluid, renal or urinary tract infection during the admission for delivery, or maternal sepsis.

Gestational age was derived from measures recorded in mothers' charts before delivery, with precedence given to dates established early in pregnancy and to estimates based on ultrasonographic examinations before 19 weeks of gestation.

For children with cerebral palsy, a developmental pediatrician and a geneticist reviewed postneonatal medical and service agency records for documentation of conditions outside the intrapartum period that were judged by both physicians and by us to be probable causes of the motor disability, such as structural malformations of the brain or genetic or other nonprogressive disorders with spastic weakness as a component. Children with such disorders were excluded from the major analyses.

Odds ratios and 95% confidence intervals were calculated to estimate the relative risk between children with cerebral palsy and control subjects with regard to major risk factors. For tables with a 0 cell, 0.5 was added to each cell. Odds ratios were also used to evaluate the association between major risk factors and indicators of neonatal depression, separately for children with cerebral palsy and controls. We assessed potential confounding of the association between the major risk factors and cerebral palsy with Mantel-Haenszel odds ratios for 2 × 2 tables stratified by other factors of interest.[7] All other factors of interest were then jointly considered for potential confounding in a stepwise logistic regression model with use of SAS Logistic (SAS Institute, Cary, NC).[8] Because of the observational nature of this study, we did not make statistical adjustments for multiple comparisons.

Etiologic fraction[9] was used to estimate the proportion of spastic cerebral palsy in term infants that might be attributable to the risk factors under study. The advantage of this measure is that it considers the frequency of risk factors in both affected children and controls.

Results

In a study population of 155,636 children surviving to the age of 3 years, there were 144,167 singletons who weighed ≥2500 g at birth. Of these, 97 children had mod-

Table I. Potentially birth-asphyxiating conditions and risk of unexplained spastic cerebral palsy in singleton infants born weighing ≥2500 g

Conditions	*Total unexplained spastic cerebral palsy (n = 46)*		*Unexplained spastic quadriplegia (n = 19)*		*Control children (n = 378)*
	No.	*OR and 95% CI**	*No.*	*OR and 95% CI**	*(No.)*
Abruptio placentae	1 (2.2%)	4.3 (0.65-39)	1 (5.6%)	11 (1.6-103)	2 (0.53%)
Placenta previa	1 (2.2%)	2.8 (0.52-25)	1 (5.6%)	7.4 (1.3-66)	3 (0.79%)
Maternal shock	0	—	0	—	1 (0.26%)
Prolapsed cord	0	—	0	—	4 (1.1%)
Cord compression	0	—	0	—	4 (1.1%)
Tight nuchal cord	8 (17%)	5.1 (2.1-13)	8 (42%)	18 (6.2-48)	15 (4.0%)
Cord knot	1 (2.2%)	8.6 (0.86-83)	1 (5.6%)	22 (2.1-218)	1 (0.26%)
Placental infarction†	0	—	0	—	0
Any 1 or more	9 (20%)	3.2 (1.4-7.3)	9 (47%)	12 (4.4-30)	27 (7.1%)

*Versus controls.
†Excludes small or focal infarctions.

erate or severe congenital cerebral palsy, a prevalence of 0.67 per 1000. Of 84 children whose cerebral palsy included spasticity, 48 had disability not explained by brain malformation, prenatal brain infarction, or congenital nonbacterial infection.[6] In 19 children with unexplained cerebral palsy (40%) the subtype was spastic quadriplegia, accompanied by dyskinesia in 6, in 27% spastic diplegia, and in 33% hemiparesis, a distribution similar to that for total spastic cerebral palsy. Intrapartum and neonatal record information was available for 46 (96%) children with unexplained spastic cerebral palsy and for 378 of 391 (97%) control children.

Tight nuchal cord and other potentially asphyxiating conditions. Some degree of neck encirclement by the umbilical cord was reported for 39% of children with unexplained spastic cerebral palsy, 47% of children with quadriplegia, and 19% of control children. In 8 children with spastic quadriplegia and 15 control children the nuchal cord was described in maternal medical records as tight (odds ratio for quadriplegia 18, 95% confidence interval 6.2 to 48; Table I). No child with spastic diplegia or hemiplegia had a tight nuchal cord or any of the other potentially asphyxiating conditions evaluated.

In the history of 1 quadriplegic child there was the abruption of a placenta previa, and 1 child with a tight nuchal cord also had a cord knot. Quadriplegia was severe in all 9 children with potentially asphyxiating conditions and was accompanied by dyskinesia in 4.

Potentially asphyxiating conditions other than tight nuchal cord were uncommon among case and control children. The presence of any 1 or more potentially asphyxiating conditions was associated with a threefold increase in risk for unexplained spastic cerebral palsy, a 12-fold increased risk for quadriplegia.

If the potentially asphyxiating conditions examined here are a sufficient cause of cerebral palsy, then as estimated by etiologic fraction, these might account for approximately 6% of total spastic cerebral palsy in children of normal birth weight (95% confidence interval 0% to 15%), 13% of unexplained cerebral palsy (95% confidence interval 1.0% to 25%), and 43% of otherwise unexplained spastic quadriplegia (95% confidence interval 19% to 67%).

Indicators of intrapartum stress and potentially asphyxiating conditions. We examined indicators of intrapartum fetal stress, including decreased fetal movement, fetal distress, meconium in the amniotic fluid, multiple late decelerations or decreased variability on electronic fetal monitoring, and fetal heart rate (FHR) <80 beats/min for their association with potentially birth-asphyxiating conditions. One or more such indicators were present in 61% of children with unexplained spastic cerebral palsy and in 29% of controls (Table II). (Such indicators were also noted in almost half of children whose cerebral palsy was considered explained, who were excluded from the major analyses of this study.)

Among children with unexplained quadriplegia, indicators of fetal stress were not associated with potentially asphyxiating conditions (odds ratio 0.54, 95% confidence interval 0.10 to 3.4). Similarly, among control children, markers of intrapartum stress were not significantly more common in those with potentially asphyxiating conditions than without them (41% vs 28%, odds ratio 1.8, 95% confidence interval 0.83 to 4.0).

Neonatal illness. We examined indicators of neonatal illness observable immediately after birth and in the neonatal nursery to determine whether these characteristics were associated with spastic cerebral palsy and with the presence of potentially asphyxiating conditions. Apgar scores <6 at 1 or 5 minutes were common in children with unexplained spastic cerebral palsy, especially among those with spastic quadriplegia. Among children with quadriplegia, low Apgar scores were observed in all but 1 child with a potentially asphyxiating condition and also in 67% of those in whom no such condition was recognized (Table II). In contrast, low Apgar scores were

Table II. Intrapartum and neonatal indicators and risk of unexplained spastic quadriplegia in singleton infants born weighing ≥2500 g by presence of potentially birth-asphyxiating conditions*

	Explained spastic cerebral palsy (n = 36)	*Unexplained spastic cerebral palsy (n = 46)*	*Unexplained spastic quadriplegia*			*Controls*		
			With pBA	*No pBA*	*Total*	*With pBA*	*No pBA*	*Total*
Intrapartum indicators†	16 (47)	28 (61)	5 (56)	7 (70)	12 (63)	11 (41)	97 (28)	108 (29)
1- or 5-min Apgar score <6	7 (20)	21 (48)	8 (89)	6 (67)	14 (78)	2 (7.4)	18 (5.3)	20 (5.4)
Other delivery room indicators‡	5 (15)	18 (39)	5 (63)	6 (60)	11 (61)	0	2 (0.58)	2 (0.5)
Neonatal seizures	3 (8.6)	22 (47)	7 (78)	6 (60)	13 (68)	0	0	0
Other newborn nursery indicators§	13 (36)	29 (62)	8 (89)	7 (70)	15 (79)	1 (3.7)	18 (5.3)	19 (5.1)

Values given are number and percent. *pBA*, Potentially birth asphyxiating.
*Percents are given for children with known values.
†Decreased fetal movement, fetal distress, meconium in amniotic fluid, multiple late decelerations or decreased variability on electronic fetal monitoring, or FHR <80 beats/min.
‡Hypotension requiring medication, need for ventilatory support in delivery room, initial pH <7.1, or base deficit exceeding −11.
§Oliguria, neonatal intensive care unit longer than 48 hours, or clinical diagnosis of birth asphyxia or hypoxic-ischemic encephalopathy.

considerably less common among controls with or without asphyxiating conditions.

Other indicators in the delivery room were hypotension requiring medication, need for ventilatory support in the delivery room, initial pH <7.1 or base deficit exceeding −11 (Table II). These findings were common in children with cerebral palsy, especially of the quadriplegic subtype, and were rare in control children (odds ratio for quadriplegia 292, 95% confidence interval 49 to 1070). However, these signs were not associated with potentially asphyxiating conditions, either among children with quadriplegia or among control children.

Neonatal seizures were observed only in children with cerebral palsy. Other indicators of illness later in the newborn period, including oliguria, intensive care nursery stay longer than 48 hours, and a clinical diagnosis of birth asphyxia or hypoxic-ischemic encephalopathy, were also more frequent in children with cerebral palsy than in controls (odds ratio for quadriplegia 70, 95% confidence interval 20 to 197). Among quadriplegic children, 1 or more markers of later neonatal illness were observed more often in children with a potentially asphyxiating condition, but the differences did not reach statistical significance. Of note, hypotension or need for respiratory support in the delivery room was reported for only 1 of 365 control children (0.3%) but for 8 of 17 children with quadriplegia (47%), 4 of whom had potentially asphyxiating conditions.

In summary, intrapartum signs were common both in children with cerebral palsy and in control children and were not associated with potentially asphyxiating conditions. In contrast, indicators of neonatal illness in the delivery room and later were observed frequently only in children with cerebral palsy. However, among children with quadriplegia these signs of neonatal illness did not distinguish well between those who were and those who were not exposed to a potentially asphyxiating condition.

Labor and delivery. To investigate whether characteristics of delivery altered the association between potentially birth asphyxiating conditions and risk of unexplained spastic quadriplegia, we computed adjusted odds ratios for that association by examining the data with and without each potential confounder. Several factors of interest were too uncommon for this evaluation: no quadriplegic child of normal birth weight had a breech presentation, was exposed in utero to magnesium sulfate, or had a birth weight <10th percentile for gestational age. Duration of labor >12 hours was documented for only 1 child with spastic quadriplegia and a potentially asphyxiating condition.

Factors evaluated for confounding included level of care, augmentation with oxytocin, and presence of maternal infection. Adjusted odds ratios observed when data were stratified separately by these factors were not different from the crude odds ratios for the association between potentially asphyxiating conditions and spastic quadriplegia, indicating that these factors did not account for the association observed between asphyxiating conditions and quadriplegia. Stratification by mode of delivery (surgical vs vaginal) also did not alter that association, nor did consideration of emergency cesarean section versus all other modes of delivery, or in 3 strata including cesarean section with and without labor and vaginal delivery, reduce the association.

In a multiple logistic model that controlled simultaneously for level of care, oxytocin augmentation, maternal infection, and mode of delivery, the adjusted odds ratio for the association of potentially asphyxiating conditions with unexplained spastic quadriplegia did not differ significantly from the crude odds ratio. The presence of several 0 cells prevented evaluation of interactions in the logistic model.

Maternal infection and potentially asphyxiating conditions. Three of 9 quadriplegic children with potentially

Table III. Potentially birth-asphyxiating conditions, maternal infection,* and risk of unexplained spastic cerebral palsy in singleton infants born weighing ≥2500 g

	Total unexplained spastic cerebral palsy (n = 46)		*Unexplained spastic quadriplegia (n = 19)*		*Control children (n = 378)*
	No.	*OR and 95% CI*	*No.*	*OR and 95% CI*	*(No.)*
No pBA or infection	30 (65%)	Reference	6 (32%)	Reference	340 (90%)
pBA only	6 (13%)	2.5 (1.0-6.7)	6 (32%)	13 (3.9-39)	27 (7.1%)
Infection only	7 (15%)	7.2 (2.7-20)	4 (21%)	21 (5.4-78)	11 (2.9%)
pBA and infection	3 (6.5%)	78 (4.8-406)†	3 (16%)	367 (19-1974)†	0 (0%)

OR, Odds ratio; *CI*, confidence interval; *pBA*, potentially birth-asphyxiating.
*Clinical diagnosis of chorioamnionitis, inflamed placental membranes, funisitis, or cord inflammation, maternal temperature during labor >100.4°F or 38°C, foul-smelling amniotic fluid, renal or urinary tract infection during admission for delivery, or maternal sepsis.
†A value of 0.5 was added to each cell of 2 × 2 table because table contained 0 cell.

asphyxiating conditions were also exposed to maternal infection, whereas no child with diplegia or hemiplegia and no control was exposed to both these risk factors. The risk of unexplained spastic quadriplegia associated with exposure to both these conditions was considerably greater than the risk associated with either condition alone and was more than 300 times greater than the risk in children without exposure to either of these conditions (Table III, 0 cell in table).

Base deficit. In children not recognized to have any of the potentially asphyxiating conditions evaluated, we examined base deficit as an additional screen for possible asphyxia. Ten infants without known asphyxiating conditions had a base deficit >−11; 4 of them had spastic quadriplegia, 5 had diplegia or hemiplegia, and 1 was a control. One of these quadriplegic children had a birth complicated by severe fetal-maternal hemorrhage, in another there was chorioamnionitis, maternal pelvic thrombophlebitis, and thrombosed umbilical cord. The 8 other children had a variety of complications, including maternal infection in 2, a hypothyroid mother with a coagulopathy whose infant had severe transient neonatal hypothyroxinemia, a placenta with hemorrhagic endovasculitis, a mother who used amphetamine and cocaine, had no prenatal care, and was admitted with severe pre-eclampsia and pneumonia, and an infant who was large for dates and had hepatosplenomegaly. If asphyxia played a role in the neurologic morbidity in these children, it did so against a background of other problems that might have contributed to vulnerability or determined outcome.

Comment

In this large population-based study, we examined complications capable of limiting fetal oxygen supply as an initiating pathogenetic event and investigated the association of such conditions with spastic cerebral palsy. Our goal was to evaluate these primary conditions separately from hypoxia or ischemia arising later in a pathophysiologic cascade initiated by other factors, and separately also from conditions that may produce similar clinical manifestations for other reasons.

Of the potentially asphyxiating conditions assessed, only 1—tight nuchal cord—was significantly associated with otherwise unexplained spastic cerebral palsy. Tight nuchal cord is a risk factor for fetal death,[10] anemia,[11] hypovolemia,[12] and other signs of neonatal morbidity,[13, 14] but we found no previous evidence that tight nuchal cord increases risk for cerebral palsy. In fact, we found only two studies of risk factors for cerebral palsy in infants of normal birth weight that included nuchal cord,[15, 16] neither of which evaluated cord tightness.

Tight nuchal cord and other potentially asphyxiating conditions were associated with increased risk of the quadriplegic subtype of spastic cerebral palsy. This association could not be accounted for by characteristics of labor and delivery such as birth in a community hospital, labor lasting >12 hours, oxytocin administered for augmentation of labor, or mode of delivery. We did not find evidence that aspects of medical management of delivery were important in determining why some fetuses with potentially asphyxiating conditions became quadriplegic and most did not.

There was no observed association of potentially asphyxiating conditions with spastic hemiplegia or diplegia.

Some potential causes of asphyxial birth played a surprisingly small role. Umbilical cord prolapse or compression and maternal shock or uterine rupture were not reported for any normal birth weight child with cerebral palsy, and abruptio placentae for only 1, in a birth cohort equivalent in number to 1 in 23 births in the United States in a study year, indicating that these factors are uncommon causes of cerebral palsy in children of normal birth weight.

Strengths of this study are its large population, well ascertained for cerebral palsy, a relatively homogeneous outcome limited to unexplained spastic cerebral palsy and its subtypes in normal birth weight singletons, and

use of maternal records to establish the presence of potentially asphyxiating conditions to minimize bias based on unsatisfactory neonatal condition. Most important, we sought to identify primary asphyxiating complications as opposed to hypoxia or ischemia occurring later in a pathogenetic cascade initiated by other primary causes. However, the tools available for making these distinctions are fairly blunt instruments, limited by the quantity and quality of information in existing medical records and the small numbers of children with these low prevalence outcomes. The markers by which we sought to identify potentially asphyxiating conditions are not an exhaustive list but probably include most such factors likely to be reasonably well documented in maternal records of birth. In spite of the use of maternal birth records for documentation of the occurrence of asphyxiating conditions, we cannot exclude the possibility that poor condition of the infant immediately after birth might sometimes have influenced recording of tightness of the nuchal cord.

Intrapartum indicators such as meconium in the amniotic fluid and fetal monitoring abnormalities were common in all groups and when present were not specific to potentially asphyxiating conditions and thus were not helpful in identifying fetuses in need of an intervention addressed to oxygen supply. Extrapolating the observed figures to the total birth population suggests an enormously high rate of false-positive identification.[17]

Unlike the intrapartum signs, markers of illness observed in the delivery room and nursery were present in most quadriplegic children with potentially asphyxiating conditions, as has been observed in previous experimental[18] and clinical investigations.[19] Such signs were scarce in controls. However, these clinical signs were neither statistically nor clinically significantly more frequent among children with quadriplegia who had potentially asphyxiating conditions than in those in whom no such condition was recognized.

It is possible that medical records did not contain evidence of other asphyxiating conditions that were causes of cerebral palsy in some children. Scrutiny of the records of children with substantial base deficits led us to identify 1 or 2 such instances. Another explanation for neonatal symptoms in children without recognized asphyxiating conditions is that disorders other than primarily asphyxiating ones may be capable of causing both irreversible brain damage and signs of neonatal illness. Exposure to maternal infection, for example, is associated with many signs of neonatal illness in babies of normal birth weight and is a risk factor for cerebral palsy.[6, 20, 21] The fact that children whose cerebral palsy was considered explained by prenatal lesions or nonbacterial perinatal infections also had higher rates of neonatal signs than controls suggests that intrinsic defects in the fetus or nonasphyxial illnesses can cause neonatal symptoms.

Neonatal signs of illness were much more frequent in quadriplegic children with potentially birth-asphyxiating conditions than in control children with such conditions, perhaps related to differences in severity or duration of an asphyxial insult. No child with cerebral palsy was described as having extended fetal bradycardia, but we have no other measures that might reflect severity or duration of asphyxia. It is also possible that other complications, such as intrauterine exposure to infection, may have increased the vulnerability of some infants to asphyxiating conditions. It would not be surprising if two potentially injurious conditions are more harmful than one, and mechanisms for an interaction between inflammatory and ischemic processes have been suggested.[22, 23]

In this normal birth weight group, hypotension or need for ventilatory support in the delivery room and neonatal seizures were noted almost exclusively in children with cerebral palsy. Because they marked high levels of risk, these characteristics may be useful for prognosis and in identifying candidates for trials of therapeutic interventions. However, the specificity to asphyxial states of a proposed intervention would require consideration because about half of infants with these neonatal signs may have them for reasons other than birth asphyxia.

In experimental models of birth asphyxia, the investigator inflicts a known asphyxial insult on a previously normal young animal. The observations we report suggest that such models are chiefly relevant to human spastic quadriplegia, rather than other forms of cerebral palsy, and that tight nuchal cord may be the most common clinical analog of the experimental procedures. However, even in some quadriplegic children with tight nuchal cords the medical history was more complicated. Furthermore, many infants with large base deficits who lacked a history of a cardinal asphyxiating condition had other prenatal problems. Our experimental and conceptual models of birth asphyxia may be incomplete: in human infants with spastic cerebral palsy pre-existing conditions and potentially injurious intrapartum events may sometimes or often interact to influence outcome.

Dr Susan K. Cummins of the California Lead Program, California Department of Health Services, and Dr Cynthia J. Curry, Medical Genetics and Prenatal Detection, Valley Children's Hospital, Fresno, California, investigated the postneonatal records.

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