

The New England Journal of Medicine

©Copyright, 1996, by the Massachusetts Medical Society

Volume 334

MARCH 7, 1996

Number 10

UNCERTAIN VALUE OF ELECTRONIC FETAL MONITORING IN PREDICTING CEREBRAL PALSY

KARIN B. NELSON, M.D., JAMES M. DAMBROSIA, PH.D., TRICIA Y. TING, B.S., AND JUDITH K. GREThER, PH.D.

Abstract Background. Electronic monitoring of the fetal heart rate is commonly performed, in part to detect hypoxia during delivery that may result in brain injury. It is not known whether specific abnormalities on electronic fetal monitoring are related to the risk of cerebral palsy.

Methods. Among 155,636 children born from 1983 through 1985 in four California counties, we identified singleton infants with birth weights of at least 2500 g who survived to three years of age and had moderate or severe cerebral palsy. The children with cerebral palsy were compared with randomly selected control children with respect to characteristics noted in the birth records.

Results. Seventy-eight of 95 children with cerebral palsy and 300 of 378 controls underwent intrapartum fetal monitoring. Characteristics found to be associated with an increased risk of cerebral palsy were multiple late decelerations in the heart rate, commonly defined as slowing of the heart rate well after the onset of uterine contractions (odds ratio, 3.9; 95 percent confidence interval, 1.7 to 9.3), and decreased beat-to-beat variability of the heart rate (odds ratio, 2.7; 95 percent confidence interval, 1.1 to 5.8); there was no association between the high-

est or lowest fetal heart rate recorded for each child and the risk of cerebral palsy. Even after adjustment for other risk factors, the association of abnormalities on fetal monitoring with an increased risk of cerebral palsy persisted (adjusted odds ratio, 2.7; 95 percent confidence interval, 1.4 to 5.4). The 21 children with cerebral palsy who had multiple late decelerations or decreased variability in heart rate on fetal monitoring represented only 0.19 percent of singleton infants with birth weights of 2500 g or more who had these fetal-monitoring findings, for a false positive rate of 99.8 percent.

Conclusions. Specific abnormal findings on electronic monitoring of the fetal heart rate were associated with an increased risk of cerebral palsy. However, the false positive rate was extremely high. Since cesarean section is often performed when such abnormalities are noted and is associated with risk to the mother, our findings arouse concern that, if these indications were widely used, many cesarean sections would be performed without benefit and with the potential for harm. (N Engl J Med 1996; 334:613-8.)

©1996, Massachusetts Medical Society.

ELECTRONIC fetal monitoring during labor was developed to detect fetal-heart-rate patterns thought to indicate hypoxia. The early recognition of hypoxia would, it was reasoned, alert clinicians to potential problems and enable them to intervene quickly to prevent fetal death or irreversible brain injury. When electronic fetal monitoring was introduced, it was hoped that the use of this technique would prevent the majority of birth injuries due to hypoxia or asphyxia, thus greatly reducing the frequency of cerebral palsy and mental retardation.

The introduction and wide dissemination of fetal

monitoring occurred before randomized clinical trials had evaluated its efficacy. More than 20 years and 11 randomized trials later,¹⁻⁶ electronic fetal monitoring appears to have little documented benefit over intermittent auscultation with respect to perinatal mortality or long-term neurologic outcome. Furthermore, probably in part because of the widespread use of fetal monitoring,⁷ the rate of cesarean section has increased, with a resulting increase in maternal morbidity and costs but without apparent decrease in the incidence of cerebral palsy.

Few of the trials performed so far have been large enough or have lasted long enough to investigate a possible association between findings on fetal monitoring and a relatively rare outcome such as cerebral palsy, which can be confidently diagnosed only years after birth. No randomized trial has explored possible associations between specific heart-rate patterns detected on electronic monitoring and long-term neurologic outcomes.

In a population-based study, we assessed the association of cerebral palsy in children with birth weights of 2500 g or more with specific patterns on fetal monitoring and evaluated the possible confounding effects of

From the Neuroepidemiology Branch (K.B.N.) and the Biometry and Field Studies Branch (J.M.D.), National Institute of Neurological Disorders and Stroke, Bethesda, Md.; the Howard Hughes Medical Institute, Bethesda, Md., and the University of Pennsylvania School of Medicine, Philadelphia (T.Y.T.); and the Birth Defects Monitoring Program, California Department of Health Services, Emeryville (J.K.G.). Address reprint requests to Dr. Grether at the California Birth Defects Monitoring Program, 1900 Powell St., Suite 1050, Emeryville, CA 94608-1811.

Supported in part by a cooperative agreement with the Center for Environmental Health and Injury Control, Centers for Disease Control and Prevention, in part by funds from the Comprehensive Environmental Response, Compensation, and Liability Act Trust Fund through an interagency agreement with the Agency for Toxic Substances and Disease Registry, Public Health Service, and in part by a training grant from the Department of Health and Human Services, Maternal and Child Health Bureau (MCJ 002001-28-0).

other risk factors. We investigated the usefulness of fetal monitoring as interpreted by the treating physicians at the time of the delivery of infants who would later be given the diagnosis of cerebral palsy.

METHODS

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 resulting from progressive disease. Children in whom cerebral palsy was acquired after the first 28 days of life or through nonaccidental head trauma in the first month and children with mild involvement or isolated hypotonia were not included.

The case patients were singleton children born during the three-year period from 1983 through 1985 to residents of four counties in the San Francisco Bay area; the children weighed 2500 g or more at birth, survived to the age of three years, were residents of California at the age of three, and had moderate or severe cerebral palsy. For the initial ascertainment of cases, we relied on the records of two state agencies known to enroll virtually all eligible children. The inclusion or exclusion of each identified child was determined by means of a standardized clinical examination or extensive review of the medical records. Detailed information about definitions and procedures has been published elsewhere.⁸ Controls were randomly selected singleton children who met all the criteria for the case children except the diagnosis of cerebral palsy.

Demographic and clinical data were obtained from birth certificates and medical records at more than 40 hospitals. Data were abstracted by nurses working at the California Birth Defects Monitoring Program who did not know whether the records were those of case or control children and did not know that the study was about cerebral palsy.

The findings on fetal monitoring that we recorded were those noted in the birth records by the physicians attending the deliveries. No monitoring strips were available for review. We collected data on the highest fetal heart rate above 160 or 180 beats per minute, the lowest fetal heart rate below 100 or 80 beats per minute, and the presence or absence of multiple late decelerations (commonly defined as bradycardia occurring well after the onset of uterine contractions, although in this study the term was recorded as used by the clinicians involved) and decreased beat-to-beat variability in heart rate. Multiple late decelerations and decreased beat-to-beat variability were then combined into a single variable indicating the occurrence of either or both during labor. Inconsistent reporting prevented us from including the duration of monitoring or specific heart-rate patterns in the analyses.

Gestational age was derived from measures recorded in the mothers' charts before delivery, with precedence given to dates established early in pregnancy and to estimates based on ultrasound examinations before 19 weeks of gestation. The level of care provided at the hospital where the delivery occurred was determined according to the criteria of the California Children's Services program.⁹ Level 1 hospitals were those without specialized services for sick or premature infants, level 2 hospitals those that provided care for sick neonates who did not require intensive care, and level 3 hospitals those that provided a full range of services including neonatal intensive care. Standards for birth weight for gestational age were derived from vital-records data (classified according to sex, race, and number of fetuses in the gestation) on more than a million children born in California from 1966 through 1970.¹⁰

Certain factors identified in the literature on fetal monitoring as associated with a heightened risk of cerebral palsy^{3,5,11} were examined for their univariate association with both cerebral palsy and fetal-heart-rate abnormality in this study. These factors were vaginal bleeding during pregnancy, breech presentation, meconium in the amniotic fluid (classified as absent, light, or heavy), gestational age below 37 weeks at delivery, and maternal infection (indicated by a diagnosis of maternal sepsis, chorioinfection, or amnionitis; a maximal temperature during labor of 38°C or higher; or foul-smelling meconium). Factors in terms of which case children and controls differed significantly in the univariate analysis were further evaluated by logistic regression to determine the separate contribution of each to the risk of cerebral palsy, with control for the other factors. These analyses were repeated

for spastic quadriplegia, a subtype of cerebral palsy often linked to asphyxia during delivery.^{12,13}

RESULTS

Among 155,636 children born alive during the period we studied, the overall prevalence of moderate or severe congenital cerebral palsy among singleton children who survived to the age of three years was 1.1 per 1000. Among such children, 95.4 percent weighed 2500 g or more at birth, and in this group the prevalence of cerebral palsy was 0.67 per 1000. Children with birth weights of 2500 g or more made up 56.4 percent of all singleton children with cerebral palsy.

Nine of the 95 children with birth weights of 2500 g or more who had cerebral palsy (9.5 percent) and 30 of the 378 control children (7.9 percent) were delivered without labor (*P* not significant), indicating that the risk of cerebral palsy in infants born at or near term was not associated with the presence or absence of labor. Of the children born after labor, 9.3 percent of those with cerebral palsy and 13.9 percent of the controls did not undergo intrapartum monitoring — also a nonsignificant difference. A total of 78 children with cerebral palsy, of whom 41 percent had spastic quadriplegia, and 300 controls underwent fetal monitoring.

The children with cerebral palsy did not differ significantly from the controls in terms of a variety of demographic and medical characteristics (Table 1). There was no relation between the highest and lowest fetal heart rates measured by fetal monitoring in each child and the risk of cerebral palsy (Table 2). Multiple late decelerations, decreased beat-to-beat variability, or both were noted in 21 children with cerebral palsy and 28 controls. Multiple late decelerations were associated with nearly a quadrupling of the risk of cerebral palsy (odds ratio, 3.9; 95 percent confidence interval, 1.7 to 9.3), and decreased beat-to-beat variability with nearly a tripling of the risk (odds ratio, 2.7; 95 percent confidence interval, 1.1 to 5.8). The occurrence of multiple late decelerations, decreased beat-to-beat variability, or both abnormalities was associated with a sharp increase in the risk of cerebral palsy (odds ratio, 3.6; 95 percent confidence interval, 1.9 to 6.7). The odds ratios for the association of fetal-heart-rate patterns with spastic quadriplegia were similar to those for all types of cerebral palsy combined. It is notable that 73 percent of the children with cerebral palsy did not have multiple late decelerations or decreased beat-to-beat variability, whereas 9.3 percent of the controls did.

The presence of multiple late decelerations, decreased beat-to-beat variability, or both was associated with similar increases in the risk of cerebral palsy regardless of whether the delivery was the mother's first live birth (Table 3). These heart-rate abnormalities were observed more often when internal monitoring was used than when only external monitoring was used. However, the proportional increase in the rate of these abnormalities was greater for the controls than for the children with cerebral palsy. Thus, the increase in the risk of cerebral palsy associated with these abnormalities on fetal

Table 1. Characteristics of the Deliveries of Singleton Children with Birth Weights of 2500 g or More, According to the Presence or Absence of Cerebral Palsy.*

CHARACTERISTIC	CHILDREN WITH CEREBRAL PALSY (N = 78)	CONTROLS (N = 300)
Mother's race or ethnic group — no. (%)†		
Nonhispanic white	42 (54)	151 (51)
Hispanic	18 (23)	63 (21)
Black	9 (12)	23 (8)
Asian	6 (8)	27 (9)
Other	3 (4)	33 (11)
Mean maternal age — yr	28	27
Mother's parity — no. (%)		
0	42 (54)	144 (48)
≥1	36 (46)	156 (52)
Type of payment for medical care — no. (%)†		
Private	43 (73)	214 (81)
Public	16 (27)	49 (19)
Level of neonatal care at hospital — no. (%)		
1	40 (51)	153 (51)
2	17 (22)	78 (26)
3	21 (27)	69 (23)
Mean gestational age (wk)	40	40
Induction of labor — no. (%)	13 (17)	48 (16)
Augmentation of labor — no. (%)	22 (28)	97 (32)
Internal monitoring — no. (%)	45 (58)	170 (57)
Mean time from admission to delivery (min)	503	590
Mean birth weight (g)	3313	3420
Birth weight — no. (%)		
2500–2999 g	21 (27)	57 (19)
3000–3499 g	29 (37)	117 (39)
3500–3999 g	24 (31)	101 (34)
≥4000 g	4 (5)	25 (8)

*Because of rounding, percentages do not always total 100.

†Data on this variable were not available for several subjects.

monitoring was larger among those with only external monitoring.

Among children with gestational ages of 40 weeks or less at delivery, the presence of an abnormality on monitoring was associated with a risk almost five times that among children without an abnormality and twice that among children with greater gestational ages (Table 3). Abnormalities on fetal monitoring were more frequent among children — both those with cerebral palsy and controls — whose birth weights were below the 10th percentile for gestational age, but such abnormalities were not associated with a greater risk of cerebral palsy among infants who were small for gestational age as compared with infants whose birth weights were appropriate for gestational age.

The presence of multiple late decelerations, decreased beat-to-beat variability, or both was associated with similar increases in the risk of cerebral palsy whether the delivery was vaginal or surgical (Table 3). Among children with abnormalities on monitoring, 10 of those with cerebral palsy (48 percent) and 10 control children (36 percent) were delivered by cesarean section; this was not a significant difference. Among these children,

cesarean delivery was not associated with a significantly altered risk of cerebral palsy (odds ratio for vaginal vs. cesarean delivery, 1.6; 95 percent confidence interval, 0.52 to 5.2).

We found that the risk factors identified in the medical literature were, on univariate analysis, associated with an increased risk of cerebral palsy in this population (Table 4). These factors were vaginal bleeding during pregnancy, breech presentation, delivery at less than 37 weeks of gestation, the presence of meconium in the amniotic fluid, and infection in the mother. Logistic regression including those five factors along with fetal-monitoring abnormalities (Table 4) provided an excellent fit for the data. The adjusted odds ratios were close to those obtained by univariate analysis, suggesting that each factor contributed independently to the risk of cerebral palsy and that none of the other factors accounted for the increased risk associated with abnormalities on fetal monitoring.

Thirty-seven percent of the children with cerebral palsy and 69 percent of the controls had none of the five risk factors we examined. In this low-risk group, the presence of an abnormality on fetal monitoring was not associated with a significant increase in risk (odds ratio, 1.7; 95 percent confidence interval, 0.53 to 5.4). Among those with one or more risk factors, an abnormality on monitoring was recorded for 35 percent of the children with cerebral palsy and 11 percent of the controls (odds ratio, 4.4; 95 percent confidence interval, 1.8 to 10.5). When the data were expressed in terms of standard measures of association (Table 5), although certain abnormalities on monitoring were associated with an increase in risk, their presence did not provide a reliable indicator of subsequent cerebral palsy.

Extrapolation to the Entire Population

To examine the implications of the findings on electronic fetal monitoring for the assessment of the risk of cerebral palsy, we projected the observations in this

Table 2. Heart-Rate Patterns on Electronic Intrapartum Monitoring of Singleton Infants with Birth Weights of 2500 g or More, According to the Presence or Absence of Cerebral Palsy.*

PATTERN	CHILDREN WITH CEREBRAL PALSY (N = 78)	CONTROLS (N = 300)	ODDS RATIO (95% CI)
	<i>no. (%)</i>		
Tachycardia			
>160 beats/min	22 (28.2)	85 (28.3)	1.0 (0.6–1.7)
>180 beats/min	5 (6.4)	16 (5.3)	1.3 (0.4–3.4)
Bradycardia			
<100 beats/min	27 (34.6)	75 (25.0)	1.5 (0.9–2.5)
<80 beats/min	13 (16.7)	35 (11.7)	1.5 (0.8–3.0)
Multiple late decelerations	11 (14.1)	12 (4.0)	3.9 (1.7–9.3)
Decreased beat-to-beat variability	13 (16.7)	21 (7.0)	2.7 (1.1–5.8)
MLD/DV	21 (26.9)	28 (9.3)	3.6 (1.9–6.7)

*CI denotes confidence interval, and MLD/DV multiple late decelerations, decreased beat-to-beat variability, or both.

Table 3. Association of Multiple Late Decelerations, Decreased Beat-to-Beat Variability, or Both (MLD/DV) with the Risk of Cerebral Palsy in Singleton Children with Birth Weights of 2500 g or More, According to Characteristics of the Delivery and the Presence or Absence of Cerebral Palsy.

CHARACTERISTIC	CHILDREN WITH CEREBRAL PALSY (N = 78)		CONTROLS (N = 300)		ODDS RATIO (95% CI)*
	NO. OF CHILDREN	% WITH MLD/DV	NO. OF CHILDREN	% WITH MLD/DV	
Mother's parity					
0	42	35.7	144	15.2	3.7 (1.7–8.1)
≥1	36	16.7	156	5.8	3.3 (1.1–9.9)
Level of neonatal care at hospital					
1	40	30.0	154	9.1	4.3 (1.8–10.2)
2 or 3	38	26.7	146	9.6	2.9 (1.2–7.4)
Type of monitoring					
External only	33	21.1	130	3.9	6.7 (2.0–22.9)
Internal	45	31.1	170	13.5	2.9 (1.3–6.2)
Type of delivery					
Vaginal	55	20.4	252	7.4	3.3 (1.4–7.4)
Cesarean	23	43.5	48	20.8	2.9 (1.0–8.6)
Birth weight					
<3500 g	50	26.0	174	8.6	3.7 (1.6–8.5)
≥3500 g	28	28.6	126	10.3	3.5 (1.3–9.5)
Gestational age					
≤40 wk	45	31.1	177	8.5	4.9 (2.1–11.1)
>40 wk	33	21.2	123	10.6	2.3 (0.8–6.3)
Birth weight for gestational age					
≥10th percentile	71	25.4	285	8.8	3.5 (1.8–6.9)
<10th percentile	7	42.9	15	20.0	3.0 (0.4–21.3)

*For each characteristic, the odds ratios are calculated as the risk of cerebral palsy in children with MLD/DV as compared with children without MLD/DV. CI denotes confidence interval.

case-control study to the entire population of children born during three years in the four counties from which it was drawn. On the basis of the frequency of intrapartum monitoring among the controls, we estimate that in the total population there were 115,511 surviving singleton children with birth weights of 2500 g or more who had undergone electronic fetal monitoring. Assuming that the controls were representative of the total population, 9.3 percent, or 10,770 monitored children without cerebral palsy and 21 monitored children with cerebral palsy, would be expected to have had multiple late decelerations or decreased beat-to-beat variability of the fetal heart rate, or both. Of the estimated total of 10,791 monitored infants weighing 2500 g or more who had abnormalities on monitoring, 21 (0.19 percent) had cerebral palsy — for a projected false positive rate of 99.8 percent. The estimated false positive rate is 99.9 percent among children with none of the other risk factors we examined and 99.6 percent in the high-risk group.

DISCUSSION

We conducted a population-based study relating specific abnormalities in heart-rate patterns detected by electronic fetal monitoring to the risk of cerebral palsy in infants born at or

near term. The records of the children in this study reflect clinical practice during the mid-1980s in a large, diverse, urban area; this study thus represents a cross-section of abnormalities on fetal monitoring as interpreted by clinicians at all levels of expertise. Important strengths of the study are its population-wide nature, the relatively homogeneous range of birth weights that included the large majority of all live births, the examination of specific abnormalities on fetal monitoring, and the assessment of the effects of potential confounding factors. The chief limitations of the study are the absence of standardized definitions of abnormalities or monitoring protocols, including the lack of consistent information on the duration of fetal-heart-rate patterns and the time of their appearance during labor, and our inability to compare examinations performed early in labor with later findings to investigate abnormalities that developed during the course of labor. The absence of information on the duration of severe bradycardia is especially regrettable, although lengthy bradycardias can be recognized without electronic monitoring.

This study was designed as an investigation of the use of fetal monitoring in clinical practice. Monitoring strips were not available for reinterpretation by experts or for an investigation of interrater reliability. In studies from specialized centers, consistency among raters and for individual raters over time has not been very good.^{4,11,14} In this study, interpretations of patterns seen on fetal monitoring by care givers in level 2 and 3 hospitals were not associated with the risk of cerebral palsy any more closely than interpretations by readers in level 1 facilities.

We observed an association of multiple late decelerations and of decreased beat-to-beat variability in the fetal heart rate, but not of the highest or lowest recorded fetal heart rate for each child, with the risk of cerebral palsy overall and in the high-risk group, but not in the low-risk group. Leveno et al.¹¹ concluded, on the basis of other outcome variables, that the outcome of low-risk pregnancies was not improved by electronic monitoring. The association of abnormalities on fetal monitor-

Table 4. Crude and Adjusted Odds Ratios for Cerebral Palsy According to Literature-Identified Risk Factors and Specific Heart-Rate Patterns on Electronic Fetal Monitoring in Singleton Children with Birth Weights of 2500 g or More.*

FACTOR†	UNIVARIATE ANALYSIS			MULTIVARIATE LOGISTIC ANALYSIS		
	CHILDREN WITH CEREBRAL PALSY (N = 78)	CONTROLS (N = 300)	CRUDE ODDS RATIO (95% CI)	ESTIMATES	P VALUE	ADJUSTED ODDS RATIO (95% CI)
	no. (%)					
Bleeding during pregnancy	13 (16.7)	21 (7.0)	2.7 (1.3–5.6)	1.05	0.01	2.9 (1.3–6.4)
Breech presentation	8 (10.3)	10 (3.3)	3.3 (1.3–8.7)	1.06	0.05	2.9 (1.0–8.1)
Gestational age <37 wk	8 (10.3)	12 (4.0)	2.7 (1.1–7.0)	1.01	0.05	2.7 (1.0–7.6)
Meconium	26 (33.3)	52 (17.3)	1.9 (1.3–2.9)	0.45	0.05	1.6 (1.0–2.5)
Maternal infection	14 (18.0)	13 (4.3)	4.8 (2.2–10.8)	1.21	0.01	3.3 (1.4–8.1)
MLD/DV	21 (26.9)	28 (9.3)	3.6 (1.9–6.7)	1.01	0.01	2.7 (1.4–5.4)

*CI denotes confidence interval, and MLD/DV multiple late decelerations, decreased beat-to-beat variability, or both. Odds ratios are calculated as the risk of cerebral palsy among children with the risk factor as compared with those without it. Estimates are the coefficients of the fitted logistic regression, and P values are for the comparison between case children and controls.

†Each factor was analyzed as either absent or present, except meconium, which was coded as absent, light, or heavy.

Table 5. Measures of the Association between Multiple Late Decelerations, Decreased Variability in Heart Rate, or Both on Electronic Fetal Monitoring with Cerebral Palsy in Singleton Children with Birth Weights of 2500 g or More, According to Risk Group.*

RISK GROUP	% OF POPULATION	PREVALENCE OF CEREBRAL PALSY (PER 10,000)	SENSITIVITY (%)	SPECIFICITY (%)	POSITIVE PREDICTIVE VALUE (%)
Low	69	3.6	13.8	91.3	0.05
High	31	13.8	34.7	89.1	0.25
Total	100	6.8	26.9	90.7	0.14

*The risk groupings were based on the presence or absence of five factors identified in the literature as associated with an increased risk of cerebral palsy: bleeding during pregnancy, breech presentation, gestational age of less than 37 weeks at delivery, maternal infection, and the presence of meconium in the amniotic fluid. Low risk was defined as the absence of the five risk factors and high risk as the presence of one or more of them. Sensitivity and specificity were calculated within the case-control study, whereas the percentage of the population in each risk group, prevalence of cerebral palsy, and positive predictive value were obtained by projection onto the entire population of children born during the three-year study period in four counties.

ing with cerebral palsy in our study persisted after we controlled for other factors known to be related to the risk of cerebral palsy. However, the proportion of pregnancies erroneously identified as at risk because of these monitoring abnormalities (the false positive rate) was extremely high.

Electronic fetal monitoring was developed to detect intrapartum asphyxia associated with death or cerebral palsy. The causative factors in cerebral palsy are heterogeneous. Perhaps 3 to 20 percent of cases of cerebral palsy in infants born at term are due to intrapartum asphyxia¹⁵⁻¹⁸; the lower estimates include consideration of risk factors present before labor began, whereas the higher estimates do not. Thus, the prevalence of the target disorder for fetal monitoring among children who survive to three years of age — cerebral palsy related to asphyxia during delivery — is much lower than the overall prevalence of cerebral palsy.

Estimates derived from this study indicate that among 100,000 singleton children born at term, 9.3 percent, or 9300, would be expected to have multiple late decelerations or decreased beat-to-beat variability on fetal monitoring. Of those with these abnormalities, about 18 children (0.19 percent of 9300) would be expected to have cerebral palsy. If in 20 percent of these children cerebral palsy might be related to asphyxia during delivery and if there were an intervention that could prevent asphyxia-related cerebral palsy once monitoring abnormalities were recognized, then approximately 4 ($18 \times 0.2 = 3.6$) of 100,000 full-term infants might benefit. However, the intervention would be administered in 9296 ($9300 - 4$) additional deliveries (2324 nonbeneficial interventions for each child in whom cerebral palsy was prevented).

If it is assumed that all the cases of cerebral palsy in children with abnormalities on fetal monitoring are due to asphyxia during delivery and, further, that there is a perfectly effective intervention to prevent cerebral palsy (neither assumption is warranted given currently available evidence), then the number of interventions performed during delivery in which there was no benefit would be 516 ($[9300 - 18] \div 18$) for each child saved from cerebral palsy. Once monitoring abnormalities are noted, cesarean section is often performed. In evaluat-

ing the wisdom of this policy, it is necessary to evaluate the risks — as well as costs — to the 516 or 2324 mother-baby pairs exposed to surgical delivery for each infant who might benefit. In one large study, 11.6 percent of cesarean sections were associated with intraoperative complications.¹⁹ In 2.1 percent, the complications were major, chiefly serious hemorrhage; in surgical deliveries performed during labor, the rate of major complications was 4.1 percent.¹⁹ Postoperative complications of surgical delivery are also relatively common, occurring in 13 to 65 percent in different studies.²⁰

So far as we know, neither cesarean section²¹ nor any other intervention during delivery has been shown to prevent cerebral palsy in infants born at term. In this study, children who had abnormalities on fetal monitoring during labor and were delivered by cesarean section did not have a lower frequency of cerebral palsy than children with these abnormalities who were delivered vaginally.

The increase in the rate of cesarean section in the United States and elsewhere during recent decades has not been reflected in a decrease in the incidence of cerebral palsy.²²⁻²⁴ Whether electronic fetal monitoring is helpful in reducing perinatal mortality remains uncertain,^{5,25,26} and most randomized trials have not found low Apgar scores, acidosis, neonatal apnea, or the need for intubation to be less frequent among infants who were monitored electronically.^{5,27}

It is likely that technological approaches to fetal surveillance are here to stay.²⁸ The same issues that confront us with regard to fetal monitoring and intervention by means of cesarean delivery are likely to continue to confront us as new methods are developed for the assessment of the risk due to asphyxia during delivery and as new therapeutic interventions appear. We need to consider how to balance the possibility of helping the small number of babies threatened by death or irreversible brain injury as a result of asphyxiating conditions during labor against the possibility of harm to some of the very large number of mothers and babies who, despite similar heart-rate patterns and other clinical findings during delivery, would not benefit from intervention. What trade-offs are reasonable?

The focus on a relatively rare but severe outcome, cerebral palsy caused by asphyxia during delivery, may have diverted clinical and research attention from an exploration of factors other than birth asphyxia that can contribute to maldevelopment or injury of the infant's brain.^{16,29-31}

We are indebted to the children and parents who participated in this study.

REFERENCES

1. Shy KK, Larson EB, Luthy DA. Evaluating a new technology: the effectiveness of electronic fetal heart rate monitoring. *Annu Rev Public Health* 1987; 8:165-90.
2. Lumley J. Does continuous intrapartum fetal monitoring predict long-term neurological disorders? *Paediatr Perinat Epidemiol* 1988;2:299-307.
3. Grant A. Monitoring the fetus during labour. In: Chalmers I, Enkin M, Keirse MJNC, eds. *Effective care in pregnancy and childbirth*. Vol. 2. Oxford, England: Oxford University Press, 1989:846-82.
4. Paneth N, Bommarito M, Stricker J. Electronic fetal monitoring and later outcome. *Clin Invest Med* 1993;16:159-65.

5. Neilson JP. Electronic fetal heart rate monitoring during labor: information from randomized trials. *Birth* 1994;21:101-4.
6. Thacker SB, Stroup DF, Peterson HB. Efficacy and safety of intrapartum electronic fetal monitoring: an update. *Obstet Gynecol* 1995;86:613-20.
7. Rostow VP, Osterweis M, Bulger RJ. Medical professional liability and the delivery of obstetrical care. *N Engl J Med* 1989;321:1057-60.
8. Grether JK, Cummins SK, Nelson KB. The California Cerebral Palsy Project. *Paediatr Perinat Epidemiol* 1992;6:339-51.
9. 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.
10. Cunningham GC, Hawes WE, Madore C, Norris FD, Williams RL. Intrauterine growth and neonatal risk in California. Santa Barbara: Community and Organization Research Institute, University of California, 1979.
11. Leveno KJ, Cunningham FG, Nelson S, et al. A prospective comparison of selective and universal electronic fetal monitoring in 34,995 pregnancies. *N Engl J Med* 1986;315:615-9.
12. 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.
13. 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.
14. Cohen AB, Klapholz H, Thompson MS. Electronic fetal monitoring and clinical practice: a survey of obstetric opinion. *Med Decis Making* 1982;2:79-95.
15. Nelson KB, Ellenberg JH. Antecedents of cerebral palsy: multivariate analysis of risk. *N Engl J Med* 1986;315:81-6.
16. Blair E, Stanley F. When can cerebral palsy be prevented? The generation of causal hypotheses by multivariate analysis of a case-control study. *Paediatr Perinat Epidemiol* 1993;7:272-301.
17. Naulty CM, Long LB, Pettett G. Prevalence of prematurity, low birthweight, and asphyxia as perinatal risk factors in a current population of children with cerebral palsy. *Am J Perinatol* 1994;11:377-81.
18. Yudkin PL, Johnson A, Clover LM, Murphy KW. Assessing the contribution of birth asphyxia to cerebral palsy in term singletons. *Paediatr Perinat Epidemiol* 1995;9:156-70.
19. Nielsen TF, Hokegard K-H. Cesarean section and intraoperative surgical complications. *Acta Obstet Gynecol Scand* 1984;63:103-8.
20. Nielsen TF. Cesarean section. In: Sachs BP, Beard R, Papiernik E, Russell C, eds. *Reproductive health care for women and babies*. New York: Oxford University Press, 1995:279-90.
21. Scheller JM, Nelson KB. Does cesarean delivery prevent cerebral palsy or other neurologic problems of childhood? *Obstet Gynecol* 1994;83:624-30.
22. Hagberg B, Hagberg G, Olow I, von Wendt L. The changing panorama of cerebral palsy in Sweden. V. The birth year period 1979-82. *Acta Paediatr Scand* 1989;78:283-90.
23. Stanley F, Watson L. Trends in perinatal mortality and cerebral palsy in Western Australia, 1967 to 1985. *BMJ* 1992;304:1658-63.
24. MacGillivray I, Campbell DM. The changing pattern of cerebral palsy in Avon. *Paediatr Perinat Epidemiol* 1995;9:146-55.
25. Vintzileos AM, Nochimson DJ, Guzman ER, Knuppel RA, Lake M, Schifrin BS. Intrapartum electronic fetal heart rate monitoring versus intermittent auscultation: a meta-analysis. *Obstet Gynecol* 1995;85:149-55.
26. Bader TJ, Morgan MA. Intrapartum electronic fetal heart rate monitoring versus intermittent auscultation: a meta-analysis. *Obstet Gynecol* 1995;85:643.
27. MacDonald D, Grant A, Sheridan-Pereira M, Boylan P, Chalmers I. The Dublin randomized controlled trial of intrapartum fetal heart rate monitoring. *Am J Obstet Gynecol* 1985;152:524-39.
28. Paul RH. Electronic fetal monitoring and later outcome: a thirty-year overview. *J Perinatol* 1994;14:393-5.
29. Nelson KB, Ellenberg JH. Antecedents of cerebral palsy. I. Univariate analysis of risks. *Am J Dis Child* 1985;139:1031-8.
30. Torfs CP, van den Berg BJ, Oechsli FW, Cummins S. Prenatal and perinatal factors in the etiology of cerebral palsy. *J Pediatr* 1990;116:615-9.
31. Nelson KB, Grether JK. Can magnesium sulfate reduce the risk of cerebral palsy in very low birthweight infants? *Pediatrics* 1995;95:263-9.