# Neonatal Neurodevelopmental Examination as a Predictor of Neuromotor Outcome in Premature Infants

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**ABSTRACT.** There was a marked correlation ($P < .000001$) between neonatal neurodevelopmental examination results and neuromotor outcome at 1 year of age or older in 210 high-risk premature infants (mean birth weight 1,107 g, mean gestational age 28.4 weeks). This neonatal examination consisted of assessment of posture, extremity and axial tone, deep tendon reflexes, pathologic reflexes, primitive reflexes, symmetry, oromotor function, cranial nerve function, auditory and visual responses, and behavior. Premature infants whose neonatal neurodevelopmental examination results were abnormal had significantly higher incidences of both cerebral palsy (38% *v* 6%, $P < .000001$) and minor neuromotor dysfunction (27% *v* 13%, $p < .05$) than did premature infants whose examination results were normal. This correlation continued to be highly significant even with the analysis of subgroups (infants born at or before 27 weeks' gestation, infants with chronic lung disease discharged with oxygen supplementation, infants with periventricular hemorrhage) and when a variety of individual perinatal, demographic, and social variables were used as controls. Normal or nearly normal neonatal neurodevelopmental examination results can be used to reassure parents of high-risk premature infants. Although abnormal neonatal neurodevelopmental examination results cannot be used to diagnose handicap in premature infants, they can be used to select a group of high-risk infants who should be carefully monitored during infancy and childhood. *Pediatrics* 1989;83:498–506; *Neurodevelopmental examination, prematurity, perinatal risk factors, developmental outcome, cerebral palsy.*

Although neurologic handicap is a well-known long-term complication of prematurity, the majority of extremely premature infants do not have major handicaps (cerebral palsy or mental retardation).[1–4] With improved survival of premature infants, increasing numbers of these infants are being discharged from the various neonatal intensive care units throughout the country. Although it is becoming increasingly difficult to closely observe all of these infants throughout infancy, some have suggested that they all be entered into early intervention programs.[5] The ability to select a group of neonates prior to discharge from a neonatal intensive care unit who are at highest risk of handicap would allow close monitoring of their development throughout infancy, assessment of early intervention techniques, and more efficient utilization of existing resources.

In some recent studies, several perinatal factors that carry higher risk of handicaps in premature infants have been identified. These include severe intraventricular hemorrhage,[6–8] ventricular dilation,[6,8] periventricular echodensities,[9] chronic lung disease,[10,11] and poor postnatal head growth.[12–14] Dubowitz et al[15] found that the presence of periventricular hemorrhage was not as good a predictor of developmental outcome at 1 year as the neurologic examination at full term in premature infants.

Although abnormal findings from a neurodevelopmental examination predicted developmental disability in high-risk and/or asphyxiated full-term infants,[16–25] the neonatal neurodevelopmental examination has not been as well studied in premature infants.[12,15,26,27] The premature populations that have been studied to date tend to be more mature and at less risk than the premature infants who are now surviving in most tertiary care centers. In addition, in most studies outcome is classified as abnormal, suspect, or normal with no description of the specific types of disabilities that are associated with abnormal neonatal neurologic examinations.

In this article, we describe a comprehensive neo-

Received for publication Aug 11, 1987; accepted May 12, 1988.
Reprint requests to (M.C.A.) CMSC 210, The Johns Hopkins Hospital, 600 N Wolfe St, Baltimore, MD 21205.
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natal neurodevelopmental examination performed at full term or prior to discharge from a neonatal intensive care unit in a population of high-risk premature infants whose development was sequentially observed for 1 to 5 years. The neonatal neurodevelopmental examination was drawn from work by Capute et al,[28,29] Saint-Anne Dargassies,[30] Amiel-Tison, et al,[31,32] Dubowitz and Dubowitz[33] and Prechtl and Beintema.[34] The relationship between the neonatal examination and later neuromotor outcome (ie, cerebral palsy and minor neuromotor dysfunction) in the total group and in three high-risk subgroups (infants born at or before 27 weeks' gestation, infants with moderate to severe chronic lung disease discharged with oxygen supplementation and infants with periventricular/intraventricular hemorrhage) will be discussed.

## MATERIALS AND METHODS

The population consisted of 210 high-risk premature infants who were born between 1981 and 1986. These infants underwent one or more neonatal neurodevelopmental examinations at The Johns Hopkins Hospital Neonatal Intensive Care Unit at the time of neonatal intensive care unit discharge and were observed at The Johns Hopkins Hospital and/or the Kennedy Institute for Handicapped Children for at least 1 year. Infants selected for follow-up generally had a number of perinatal and socioeconomic risk factors in an effort to obtain a population with a relatively high incidence of cerebral palsy. If the infant was hospitalized beyond term, he or she was examined when stable, by 44 weeks' postmenstrual age (gestational age plus chronologic age). The examinations were all performed and recorded by a neonatologist/developmental pediatrician.

The examination (Appendix) included assessment of posture, extremity and axial tone, deep tendon reflexes, pathologic reflexes, primitive reflexes,[27,28] symmetry, oromotor function, cranial nerve function, auditory and visual responses, and behavior (jitteriness, irritability, lethargy, consolability). The responses were scored on an absolute scale then graded (with respect to appropriateness for postmenstrual age as a normal response, a minor abnormality or major abnormality. Minor abnormalities included mildly decreased extremity, neck, trunk, shoulder, or hip tone for postmenstrual age, intermittent extremity extensor tone, mild neck extensor hypertonia, absent or too strong primitive reflexes for postmenstrual age, asymmetry of posture, tone, deep tendon reflexes, pathologic or primitive reflexes, poor sucking or rooting for postmenstrual age, abnormalities of the cranial nerves (eg, facial nerve palsy, abnormal eye movements), spontaneous clonus, jitteriness, irritability, lethargy, and suspect response to the bell, light, or optokinetic nystagmus drum. Major abnormalities included marked extremity, neck, trunk, shoulder, or hip hypotonia (consistent with at least 4 weeks less than the postmenstrual age of the infant), definite neck extensor hypertonia, sustained clonus, sunsetting of eyes, and absence of habituation or response to the bell or light. Infants were scored as having no abnormalities, subtle abnormalities (≤two minor abnormalities), mild abnormalities (three to four minor abnormalities or mild neck extensor tone and/or intermittent lower extremity extensor tone), definite abnormalities (five or more minor abnormalities or one major abnormality), or marked abnormalities (two or more major abnormalities or one major and five or more minor abnormalities). This classification method was cumbersome and, for the statistical analyses, infants scored as having no, subtle, or mild abnormalities were classified as normal with neonatal neurodevelopmental examination and infants with definite or marked abnormalities (one or more major and/or five or more minor abnormalities) classified as abnormal.

The infants were observed with sequential developmental assessments for 1 to 5 years. Follow-up neurodevelopmental assessment included obtaining a pertinent medical history with the patient's gross motor, fine motor, adaptive, and language milestones and a complete neurodevelopmental examination (including posture, tone, deep tendon reflexes, pathologic reflexes, any persistent primitive reflexes, righting and equilibrium reactions, and motor and problem-solving abilities). Any child who was suspected of a major developmental disability (mental retardation and/or cerebral palsy) was referred to the nearby Kennedy Institute for a complete multidisciplinary evaluation.

We considered 1 year to be the minimum length of time necessary to make a diagnosis of cerebral palsy. By then, most of the major gross motor milestones (sitting, standing, cruising) have appeared[35] and neurologic findings suggestive of a diagnosis of cerebral palsy in older infants but not in younger infants (eg, hyperreflexia, pathologic reflexes, primitive reflexes) should no longer be present to a significant degree.[29]

Neuromotor diagnosis was based on gross motor milestones and neurologic examination. Children were classified as having a normal motor outcome if they walked by 18 months of age (or came to a sitting position by 12 months of age) and had no or only a few minor abnormalities when examined neurologically. They were classified as having minor neuromotor dysfunction if they walked at ap-



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proximately 1½ to 2 years of age and definite abnormalities were seen when they were examined (eg, lower extremity hypertonia and hyperreflexia, persistent toe walking, hypotonia). They were classified as having cerebral palsy if they did not walk by 2 years of age (or sit by 1 year from term), had multiple definite neurologic abnormalities, and/or required orthopedic surgery, braces, or aides.

Diagnoses regarding cognition are more difficult to precisely define at 1 year of age, especially because many infants were extremely premature. Nevertheless, half of the infants have been observed until they were preschool aged. The diagnosis of mental retardation is based on results obtained from standardized psychologic tests performed at the Kennedy Institute using the appropriate test norms. The choice of test varied, based on the child's age and abilities (eg, Bayley Scale of Infant Intelligence, Stanford-Binet Intelligence Scale, or Weschler Preschool and Primary Scale of Intelligence).

Only severe persistent sensory impairments (eg, legal blindness, moderate to severe hearing impairment) were reported. The diagnosis of developmental disability includes cerebral palsy, mental retardation, minor neuromotor dysfunction, and/or severe sensory impairment.

## RESULTS

### Study Population

A total of 210 premature infants were examined in the neonatal period and observed developmentally for 12 to 75 months (mean 31.4 months, SD 17). Mean birth weight was 1,107 g (range 460 to 2,280 g, SD 365); mean gestational age was 28.4 weeks (range 23 to 36 weeks, SD 2.8). Of this population, 51% were boys, 38% were white, 61% were black, and 1% were Asian. This population has a number of high-risk perinatal, family, and social risk factors, which are listed in Table 1.

**TABLE 1.** Perinatal and Social Risk Factors

| Factors | Result |
|---|---|
| Gestational age ≤27 wk | 43% |
| Vaginal delivery | 53% |
| 1-min Apgar score 0–3 | 34% |
| 5-min Apgar score 0–3 | 5% |
| Outborn | 9% |
| Small for gestational age | 10% |
| Time connected to ventilator (wk)* | 3.9 ± 5.2 |
| Chronic lung disease (discharged with oxygen supplementation) | 37% |
| Periventricular/intraventricular hemorrhage (grade 1–4) | 45% |
| Intraventricular hemorrhage (grades 3–4) | 16% |
| Maternal age (yr)* | 24.5 ± 6.1 |
| Parental education (<12th grade) | 23% |
| Parents unemployed | 31% |
| Payment by medical assistance | 50% |

* Mean ± SD.

### Neonatal Neurodevelopmental Examination

The neonatal neurodevelopmental examinations were performed at a mean postmenstrual age of 38.0 weeks (SD 2.8 weeks). According to their neonatal neurodevelopmental examinations, 30 infants (14%) had no abnormalities, 45 (22%) had subtle abnormalities, 50 (24%) had mild abnormalities, 57 (27%) had definite abnormalities, and 28 (13%) had marked abnormalities. For data analyses, the 125 infants (60%) with no, subtle, and mild abnormalities were classified as normal, and the 85 (40%) infants with definite and marked abnormalities were classified as abnormal.

### Motor Outcome

After follow-up for 1 year or more, 131 infants (62%) have normal motor outcome. Forty (19%) have cerebral palsy; 15 (38% of those with cerebral palsy) have spastic diplegia, six have spastic diplegia with superimposed spastic hemiplegia, three have spastic quadriplegia, three have spastic hemiplegia, two have extrapyramidal, and 11 (28% of those with cerebral palsy) have mixed cerebral palsy. Three children with cerebral palsy and severe chronic lung disease died just after their first birthday of respiratory failure. Minor neuromotor dysfunction, mild motor delay, and abnormal neurologic examination results were present in 31 infants (16%).

### Relationship of Neonatal Neurodevelopmental Examination to Neuromotor Outcome

Of the 125 high-risk premature infants classified as normal, 100 (81%) have no motor abnormalities, 16 (13%) have minor neuromotor dysfunction, and only eight (6%) have cerebral palsy (Table 2). In contrast, 32 of the 85 (38%) who were classified as abnormal have cerebral palsy and 23 (27%) have minor neuromotor dysfunction. This difference is highly significant ($P < .000001$) using the $\chi^2$ test. Nevertheless, more than one third (35%) of neonates whose neonatal neurodevelopmental examinations showed abnormal results had normal neurologic examination results and walked by 18 months.

A trend can be seen between the increasing number and severity of abnormalities seen in the neo-



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natal neurodevelopmental examination and the increasing incidences of cerebral palsy and minor neuromotor dysfunction (Figure).

### Associated Deficits

The children with cerebral palsy have the largest incidence of associated developmental disabilities: 78% are mentally retarded, 5% have severe hearing impairments, and 5% have severe visual impairments due to retinopathy of prematurity. The children with normal motor outcome and minor neuromotor dysfunction have a relatively small incidence (10%) of mental retardation. The majority (58%) of the children with both cerebral palsy and mental retardation have moderate to severe mental retardation in contrast to 12% of retarded infants who do not have cerebral palsy. One child with normal motor outcome has an isolated severe hearing loss.

**TABLE 2.** Neonatal Neurodevelopmental Examination and Neuromotor Outcome at ≥1 Year of Age (N = 210)*

| Neonatal Examination Results | Cerebral Palsy (n = 40) | Minor Neuromotor Dysfunction (n = 39) | Normal Motor Function (n = 131) |
|---|---|---|---|
| Abnormal (n = 85) | 32 | 23 | 30 |
| Normal (n = 125) | 8 | 16 | 101 |

* Results are given as numbers of infants. $\chi^2 = 48.3$, $P < .000001$.

Almost two thirds (65%) of the mentally retarded children had abnormal neonatal neurodevelopmental examination results and 75% of moderately to severely retarded children had abnormal neonatal examination results (Table 3). Abnormal neonatal examination results thus correlate ($p < .001$) with mental retardation by $\chi^2$ analysis. However, when the Mantel-Haenzel modification of the $\chi^2$ test is used to control for cerebral palsy as a confounding variable, the correlation between an abnormal neonatal neurodevelopmental examination and mental retardation is no longer significant.

The neonatal examination results for the one child with isolated hearing loss were normal, although she did have a suspect response to the bell.

When children with any type of developmental disability (cerebral palsy, mental retardation, minor neuromotor dysfunction, severe sensory impairment) are compared with children with no developmental disability, abnormal neonatal neurodevelopmental examination results correlate highly ($P < .000001$) with development disability.

### Diagnostic Test for Cerebral Palsy

The sensitivity of the neonatal neurodevelopmental examination for detecting later cerebral

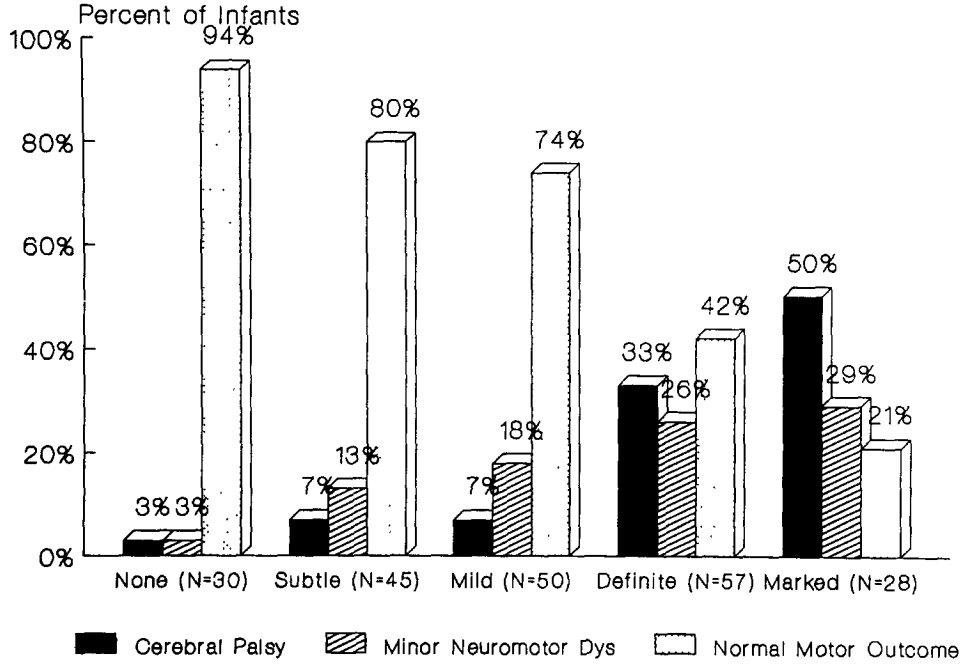


**Figure.** Relationship between number and type of abnormalities seen during neonatal neurodevelopmental examination and later motor development. Subtle abnormality, one to two minor abnormalities; mild abnormality, three to four minor abnormalities or mild neck extensor tone and/or intermittent lower extremity extensor tone; definite abnormality, one major abnormality or ≥ five minor abnormalities; marked abnormality, ≥ two major abnormalities or one major and ≥ five minor abnormalities. Dys, dysfunction.



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palsy is 80% and the specificity is 69% (Table 4). The predictive value for a negative test for cerebral palsy is 94%, whereas the predictive value for a positive test for cerebral palsy is 38%. When calculated for motor dysfunction (both cerebral palsy and minor neuromotor dysfunction), the specificity (77%) and positive predictive value (65%) are greater, but the sensitivity (70%) and negative predictive value (81%) are lesser. When calculated for developmental disability, the specificity (78%) and positive predictive value (69%) are again greater, but the sensitivity (63%) and negative predictive value (73%) are lesser.

### Controlling for Possible Confounding Variables

Because of the possibility of confounding demographic, social, or medical variables, the Mantel-Haenzel modification of the $\chi^2$ test was used to control for a number of individual perinatal, demographic, and social variables. The perinatal variables include gestational age ≤27 weeks, mode of delivery, inborn *v* outborn, one-minute Apgar score of 0 to 3, five-minute Apgar score of 0 to 3, moderate to severe chronic lung disease (discharged home with oxygen supplementation), and periventricular/intraventricular hemorrhage. The demographic and social variables include race, sex, maternal age (<20 *v* ≥20 years), method of payment (medical assistance *v* insurance or HMO), parental education (less than 12th grade *v* completion of a minimum of 12th grade), and parental unemployment *v* employment. When we controlled for each of these variables individually, the correlation between abnormal neonatal neurodevelopmental examination and later cerebral palsy and developmental disability remained significant ($P < .0005$ or better). In addition, length of follow-up (<2 years *v* ≥2 years) did not affect the level of significance.

### Predictability in Selected High-Risk Groups

The predictability of the neonatal neurodevelopmental examination for later cerebral palsy was assessed in three high-risk subgroups of premature infants: (1) 90 extremely premature infants born at or prior to 27 weeks' gestation, (2) 78 infants with moderate to severe chronic lung disease who were discharged from the neonatal intensive care unit with oxygen supplementation, and (3) 84 infants with periventricular/intraventricular (grades 1 to 4) hemorrhage. Abnormal neonatal neurodevelopmental examination results are highly correlated ($P < .001$) with later cerebral palsy in all three high-risk subgroups using the $\chi^2$ or Fisher exact test (Table 5).

## DISCUSSION

Neurologically abnormal full-term neonates have a significantly greater incidence of abnormality at 12 to 24 months of age,[16,22,23] of severe neurologic impairment,[25] of minor neurologic dysfunction at 6 years of age,[25] and of cerebral palsy at 7 years of age.[21] Asphyxiated full-term newborns with abnormal neurologic signs and symptoms have a greater incidence of later handicap than those with no abnormal neurologic signs and symptoms and, in general, the handicaps tend to be multiple and severe.[17–20,24] In the premature infant, abnormal neonatal neurologic examination results correlate with neurodevelopmental abnormalities at 1 year of age[15,27] and at 2 to 6 years of age.[12,26,27] Only Dubowitz et al[15] and Parmalee et al[26] used a detailed, comprehensive neonatal neurodevelopmental examination to evaluate premature infants. Only Dubowitz et al[15] discussed their results in terms of cerebral palsy (infants with abnormal neu-

**TABLE 4.** Accuracy of Neonatal Neurodevelopmental Examination*

| | Cerebral Palsy | Motor Dysfunction | Developmental Disability |
|---|---|---|---|
| Sensitivity | 80 | 70 | 63 |
| Specificity | 69 | 77 | 78 |
| Predictive value | | | |
| Negative | 94 | 81 | 73 |
| Positive | 38 | 65 | 69 |

* Values are given as percentages.

**TABLE 3.** Neonatal Neurodevelopmental Examination and Developmental Disability at ≥1 Year of Age*

| Neonatal Examination Results | Developmental Disability† | | Mental Retardation‡ | | | Sensory Impairments§ | |
|---|---|---|---|---|---|---|---|
| | Absent | Present | Absent | Mild | Moderate/ Severe | Hearing | Vision |
| Abnormal | 26 | 59 | 54 | 16 | 15 | 2 | 2 |
| Normal | 91 | 34 | 108 | 12 | 5 | 1 | 0 |

* Results are given as numbers of children.
† $\chi^2 = 34.8$; $P < .000001$.
‡ $\chi^2 = 16.6$; $P < .0005$.
§ NS.



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**TABLE 5.** Neonatal Neurodevelopmental Examination and Neuromotor Outcome in Selected High-Risk Groups*

| High-Risk Group and Neonatal Examination Category | Cerebral Palsy | Minor Neuromotor Dysfunction | Normal Motor Outcome |
|---|---|---|---|
| Gestational age <27 wk† | | | |
| Abnormal | 17 | 16 | 13 |
| Normal | 3 | 7 | 34 |
| Chronic lung disease‡ | | | |
| Abnormal | 17 | 14 | 13 |
| Normal | 2 | 5 | 27 |
| Periventricular/intraventricular hemorrhage§ | | | |
| Abnormal | 27 | 13 | 11 |
| Normal | 6 | 7 | 20 |

* Results are given as numbers of children.
† Fisher exact test, $P < .001$ for cerebral palsy *v* no cerebral palsy.
‡ Fisher exact test, $P < .001$ for cerebral palsy *v* no cerebral palsy.
§ $\chi^2 = 12.5$, $P < .0005$.

rologic examination results at 40 weeks' postmenstrual age had higher incidences of both cerebral palsy and dystonia).

In our study, a highly significant correlation was found between abnormal neonatal neurodevelopmental examination results and cerebral palsy in premature infants who were at greater risk. Of the eight infants who were classified as normal in whom cerebral palsy later developed, half were examined at or before 35 weeks' postmenstrual age and another two were examined at 36 weeks' postmenstrual age. There were not sufficient numbers of infants examined earlier than 36 weeks' postmenstrual age for detailed statistical analysis, but of the six in whom cerebral palsy developed, only two were correctly classified. It is possible that the neonatal neurodevelopmental examination is most accurate when performed at full-term in this premature population. One other child with normal neonatal examination results and cerebral palsy has neurofibromatosis, and although a CNS lesion has not been documented, there was concern that increasing spasiticity was developing in her lower extremities.

The sensitivity and specificity of this neonatal neurodevelopmental examination as a test for detecting later neuromotor outcome nevertheless is good, and compares favorably with the previously published studies.[12,15,16,23,25-27] It is not surprising that the usefulness of the neonatal neurologic examination in predicting mental retardation is less than its usefulness in predicting motor impairment. There are no good neonatal predictors of later intelligence. The correlations noted in this study between the neonatal neurodevelopmental examination and mental retardation are related to the fact that CNS insults are generally diffuse rather than focal. In this population, the majority (65%) of children with mental retardation had cerebral palsy. Cerebral palsy, which is more easily defined than mental retardation during infancy, frequently serves as a marker of CNS dysfunction.

Normal neonatal neurodevelopmental examination results are reassuring, because the majority of premature infants who had normal neonatal examination results remained normal at follow-up. The good negative predictive values in this study (94% for cerebral palsy, 81% for motor dysfunction, 73% for developmental disability) compare favorably with those in studies in which the neonatal examination results of full-term or premature infants were compared with outcomes at 1 to 2 years of age.[15,16,23,25] In two studies in which premature infants were observed until preschool age, smaller negative predictive values were found (47% and 62%).[26,27]

The positive predictive value of the examination for cerebral palsy is disappointingly small (38%), although it is comparable with that of other studies of premature and full-term infants (16% to 64%).[12,15,23,25] This relatively small positive predictive value is due to the low incidence of cerebral palsy (19%) even in this high-risk population. Because motor dysfunction and developmental disability are more common (38% and 44%, respectively), the positive predictive values of the test (65% and 69%) are better. It is important to consider those infants with minor neuromotor dysfunction, because there is some evidence that neuromotor abnormalities seen during the first-year examination correlate with school performance at 7 years of age.The more subtle problems of minimal cerebral dysfunction (ie, language disorders, visual-perceptual problems, learning disability, attention deficits, and behavior problems) are common sequelae of prematurity.[36-41] The importance of ob-



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serving this cohort until they reach school age is stressed. It is possible that long-term follow-up to allow diagnosis of the full spectrum of the developmental disabilities will further improve the positive predictive value of the neonatal neurodevelopmental examination.

Having established that the neonatal neurodevelopmental examination is useful in predicting developmental outcome in this selected population of high-risk premature infants, we now focus on its applicability to the sickest and most immature infants. These are the infants who have the highest risk of handicap and about whose outcome there is the greatest anxiety. Dubowitz et al[15] examined the relationship between periventricular hemorrhage and the neonatal examination and found that periventricular hemorrhage was not as good a predictor of outcome as the neonatal examination.

The number of children with various high-risk factors is much larger in this study than in previous studies, and this allows for more detailed analyses. Our high-risk population had sufficient numbers of infants to allow further statistical analysis of the following high-risk groups: (1) extremely premature infants born at or sooner than 27 weeks' gestational age, (2) premature infants with chronic lung disease who were discharged from the neonatal intensive care unit with oxygen supplementation, and (3) premature infants with periventricular/intraventricular (ie, grades 1 to 4) hemorrhage. In each category, the incidence of abnormal neonatal neurodevelopmental examinations and of later cerebral palsy was greater than in the total study population, and the correlation between abnormal neonatal examination results and later cerebral palsy and developmental disability was significant. Thus, the neonatal neurodevelopmental examination can be useful even in extremely premature infants, infants with chronic lung disease, and infants with periventricular/intraventricular hemorrhage.

In addition, when we controlled for a number of possible confounding perinatal, demographic, and social variables individually using the Mantel-Haenzel modification of the $\chi^2$ test, the correlation between abnormal neonatal neurodevelopmental examination results and later cerebral palsy remained highly significant. Thus, the predictiveness of this examination is independent of these variables.

## IMPLICATION

Although normal neonatal examination results are reassuring, abnormal examination results cannot be used to diagnose cerebral palsy with certainty in the neonatal period. The neonatal examination can be used to select infants who are at greater risk for later developmental disability so that they can be more carefully observed during infancy and childhood. Perhaps these are the infants who should be the focus of early intervention studies to determine the efficacy of an early neurodevelopmental program concerning neuromotor outcome.

This examination has served well with one experienced examiner in a high-risk population of premature infants. In the author's experience, it can be easily taught to residents and fellows. The focus of further research will be refining this examination and establishing its reliability with other examiners, evaluating the validity of this examination in another population of premature and full-term infants, and evaluating its efficacy in predicting the more subtle handicaps of minimal cerebral dysfunction/attention deficit disorder/learning disability.

## APPENDIX

### Neonatal Neurodevelopmental Examination

Auditory (record state):
- Alert to bell,[42] habituate to bell[42]

Visual (record state):
- Blink to light,[43] habituate to bell[42]
- Fixate, follow, estimate of visual attention
- Blink to a visual threatening gesture
- Optokinetic nystagmus to an optokinetic nystagmus drum[42]

Posture:
- In prone, degree of flexion at elbows, hips and knees, and hip adduction
- In supine, degree of flexion at elbows, hips and knees, and hip adduction
- In supine, presence of space behind neck and shoulders[32]

Extremity tone:
- Subjective assessment of flexor or extensor tone at elbows and knees
- Subjective assessment of hip adductor tone
- Popliteal angle maneuver, [30,31,33] heel to ear maneuver[30,31,33]
- Recoil of upper and lower extremities[30,31,33]
- Slipthrough at the shoulders, anterior and posterior scarf signs[30,31,33]

Axial tone:
- Neck tone on pull to sit from supine[30–33]
- Neck tone when rocked in sitting[32]
- Trunk tone in ventral suspension[30–33]
- Estimate of appropriateness of neck and trunk tone for gestational age

Deep tendon reflexes:
- Brachioradialis, biceps, pectoralis
- Knee and ankle jerks

Pathologic reflexes:



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Babinski, Chaddock sign, Hoffman sign
Mass reflex, crossed adduction

Primitive reflexes:

Asymmetric tonic neck reflex, Galant, tonic labyrinthine in supine and prone, symmetric tonic neck reflex and positive support graded in the manner of Capute et al[28,29]

Moro, upper and lower extremity grasp, lower extremity placing and stepping in the manner of Allen and Capute[42]

Cranial nerve function:

Facial symmetry
Eye movements (eg, dysconjugate gaze, sunsetting)

Oromotor function:

Root (one to four quadrants)
Suck (jaw closure, stripping action of the tongue, lip seal, number of sucks per burst)
Gag

Behavior:

State at beginning and end of examination, lowest and highest state attained, transition from state to state, range of states (states as discussed by Brazelton[44])
Lethargic, jittery, irritable
Consolability, cuddliness

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## WHO IS AT FAULT?

Doctors, lawyers, architects, and other professionals strike a bargain with society: Leave us alone, they say, and we will take care of you . . .

But do professionals warrant the trust placed in their hands?

Headlines shout of bridges and buildings toppling. Health care cost escalate. Our children compare poorly in knowledge of science, math, and foreign languages with those of other countries. Malpractice suits skyrocket. Greed corrupts Wall Street. America has lost the industrial muscle that was once the envy of the world.

Has our army of experts, whom we entrust to take care of us, let us down?

Or, on the other hand, have we let *them* down, shackling them in regulation, keeping them from doing their jobs, impeding them in the free exercise of their expertise?

Submitted by Student

From Kanigel R: Angry at our gods. *Columbia Magazine,* Oct 1988.



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