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Preschool Language Outcomes of Children With History of Bronchopulmonary Dysplasia and Very Low Birth Weight

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Abstract

A prospective follow-up of very low birth weight (VLBW) infants with and without bronchopulmonary dysplasia (BPD) and term control infants was conducted. The effects of BPD and VLBW on speech-language development and specific language impairment at 3 years of age were investigated, controlling for the effects of sociodemographic and other medical risk factors. Groups were compared on cognitive and speech-language outcomes using the Battelle Language and Bayley Mental Scales of Infant Development. Children with a history of BPD had lower receptive language skills than VLBW children without BPD, who in turn had lower receptive skills than term children. Children with a history of BPD also had lower expressive skills than the two comparison groups, whereas VLBW children without BPD did not differ in expressive language from term children. When IQ score was controlled, children with BPD demonstrated specific language impairment in receptive language. The presence of patent ductus arteriosis (PDA) was the best predictor of language deficits and the combined occurrence of PDA and BPD resulted in differentially lower language scores. Neurologic complications, low socioeconomic status, and minority race were also significant predictors of language delay. The findings emphasize the

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importance of considering both medical and sociodemographic factors in evaluating the risk of VLBW infants for poorer speech-language outcomes.

Keywords

very low birthweight; bronchopulmonary dysplasia; patent ductus arteriosis; development; speech and language

Recent progress in medical technology and neonatal intensive care is responsible for dramatic decreases in mortality and morbidity for very low birth weight (VLBW) infants (i.e., infants born weighing less than 1500 g). An estimated 20% to 40% of VLBW survivors are considered language delayed or impaired as toddlers and young children.[1,2] Although VLBW, in and of itself, does not necessarily result in language delays, it is associated with a number of risk factors that can exert both generalized and specific effects on speech and language development.[3,4] School age findings of language impairment in children with VLBW are equivocal. Whereas some studies report continuing deficits in language comprehension, production, and speech abilities,[3,5,6] others report either no differences between VLBW and control children[7-9] or diminishing delays over time.[10] A related issue that has not been well addressed is whether the language delays seen in VLBW samples reflect generalized IQ deficits or more specific language impairments, reflecting disability above and beyond the effects of IQ alone.[3,4]

In recent years, it has become clear that VLBW infants are not a homogeneous group and therefore not at equal risk for developmental delays. Rather, the differential effects of medical risk factors, such as intraventricular hemorrhage (IVH),[11] respiratory distress, and intrauterine growth retardation, have been recognized as important in generating more accurate estimates of risk status among VLBW infants. However, many outcome studies of VLBW fail to consider the number and type of medical complications that may be important predictors of language delay.

Factors associated with VLBW birth that may affect speech and language functioning include prenatal sociodemographic factors (such as socioeconomic status, race, and maternal education),[12,13] birth status factors (such as gestational age, birth weight, and neurosensory deficits of hearing and vision),[12,13] and perinatal and postnatal neurological and medical complications secondary to disruptions in oxygen regulation and metabolic processes associated with VLBW birth.[14,15] This last category of factors includes hypoxic-ischemic events that may cause specific brain damage to language areas, such as IVH and persistent dilation and enlargement of the lateral ventricles. It also includes other brain events and chronic medical conditions that may cause more generalized, diffuse cerebral damage.

Several studies have concluded that IVH is a risk factor for language delays.[14,15] Left ventricular damage in particular may be responsible for the delay in expressive language observed at 1 to 2 years of age in VLBW infants.[16] Vocabulary development and verbal reasoning in 3-year-old VLBW children have been related to the presence and severity of IVH.[17] Ischemic lesions developing in the periventricular region, periventricular leukomalacia (PVL), often occur with IVH in VLBW infants.[18] At 5 years of age, only

VLBW children with IVH or PVL had language and memory deficits in comparison with full-term children.[19]

Bronchopulmonary dysplasia (BPD) is a significant medical complication of prematurity that has not been well investigated in relation to language outcomes in VLBW children. BPD is a chronic lung disease that develops after preterm neonates with immature lungs are treated with artificial ventilation and supplemental oxygen for severe respiratory distress. Since its initial description,[20] BPD has become the leading cause of lung disease in United States infants, with over 7,000 new cases diagnosed annually.[21]

BPD is associated with a number of risk factors that may affect language development, including an increased incidence of growth failure and cardiopulmonary and central nervous system complications that can affect sensory, motor, and/or cognitive aspects of speech-language outcomes.[22-26] Sauve and Singhal[27] observed a higher rate of hearing loss in BPD (3.8%) versus VLBW controls without BPD (1.7%). Perlman and Volpe[28] described a movement disorder, characterized by rapid, random, jerky movements involving the neck, limbs, trunk, and oral-buccal-lingual structures that may be seen in infants with severe BPD. This may be associated with early articulatory delay and may affect motor aspects of speech. Furthermore, BPD is associated with repeated episodes of hypoxia during sleep and feeding, which may result in diffuse cerebral damage that may affect speech-language development in a global way.[24,25]

Northway[20] originally speculated that BPD could have widespread neurological consequences. More recently, Volpe[29] observed that chronic episodic hypoxia in BPD might lead to nonprogressive white matter injury and cortical loss, affecting a wide range of intellectual functions. Nevertheless, only a few studies have considered the effects of BPD on speech-language outcomes. Sauve and Singhal[27] compared VLBW infants with BPD with preterm controls and found a greater frequency of language deficits at 30 months. Similarly, at school age, children with a history of BPD were found to have more receptive vocabulary deficits than preterm and term controls, but expressive language was not evaluated, and it was not clear whether language delays were specific or reflected more general cognitive delays.[30]

The present study is part of a larger investigation of the effects of BPD on child development.[22] The first question of the current study is whether BPD is associated with negative effects on language development at age 3 years, beyond the effects of sociodemographic and other medical risk factors, and the second, whether language delays, if they occur, are reflections of overall developmental delay or whether they represent specific language impairments occurring over and above the effects of IQ. Finally, among VLBW infants, which medical and sociodemographic factors are most predictive of language delay?

METHODS

Subjects

All infants with very low birth weights and bronchopulmonary dysplasia (BPD) admitted to the neonatal intensive care units of hospitals in the Cleveland region were eligible for the study and were prospectively, consecutively recruited. For the approximately four-county region, all infants with BPD were cared for in the neonatal intensive care units (NICUS) of the three participating hospitals that had the only Level 3 NICUS in the region, providing an exhausting regional sample. BPD infants were preterm, less than 1500 g at birth, requiring supplementary oxygen for more than 28 days with radiographic evidence of chronic lung disease.[20] A partial stratification sampling strategy was adopted to enroll adequate numbers of subjects without socioeconomic disadvantage or severe neurologic risk so that these factors could be investigated in data analyses. Infants diagnosed with BPD who were free of neurologic problems other than grades I to II intraventricular hemorrhage, and who were not socially disadvantaged (i.e., Hollingshead classification IV and V), were exhaustively recruited. The remaining infants were randomly recruited by approaching the family of the next available infant diagnosed with BPD who could be accommodated in the follow-up schedule. Parents of infants with BPD were approached by a research assistant in the NICU as soon as possible after the diagnosis of BPD was made by the attending physician.

For each infant with BPD, the next born very low birth weight (VLBW) comparison infant without BPD of the same race and socioeconomic status, born during the same time period, was recruited. Term infants were recruited from the newborn nurseries. Information about the study and return-addressed postcards were provided to all mothers in the nurseries. For each BPD infant enrolled, the next term infant equivalent in race and socioeconomic status with a returned postcard indicating parental willingness to participate was recruited, providing eligibility criteria were met. Infants with major congenital malformations or drug exposure, or whose mothers had a major psychiatric or physical illness, human immunodeficiency virus, or mental retardation, or who lived more than 2 hours driving distance from the facility, were excluded. VLBW infants without BPD were preterm, less than 1500 g birth weight, requiring oxygen supplementation for less than 14 days. Term infants had no diagnosed medical illnesses or abnormalities at birth, were more than 36 weeks gestational age, and more than 2500 g birth weight for singleton infants.

During the recruitment period, 250 infants with BPD were identified, of whom 89 were excluded (35 for drug/alcohol exposure, 21 for all other exclusions, 33 who could not be accommodated into the testing schedule and who were, by definition, all of lower socioeconomic status, on public assistance), leaving 161 eligible VLBW infants with BPD. Twenty (12%) refused the study, 14 (9%) died, and 5 (3%) were unable to be contacted. Of 122 enrolled infants, 7 died after enrollment, 1 withdrew before 8 months of age, 4 were lost to follow-up, and 12 did not come for the visit, leaving 98 (89%) at 3 years of age.

Of 214 VLBW infants without BPD, 24 were excluded for drug/alcohol exposure, 34 for oxygen supplementation for 21 to 28 days, and 46 for all other exclusions, leaving 110 eligible VLBW infants without BPD, 8 (7%) of whom were unable to be contacted and 18 (16%) who refused the study. Of 84 recruited, 2 withdrew and 1 was lost. One infant died

at 2 years and 10 did not come for the visit, leaving 70 (80%) at 3 years of age. Of 123 term infants, 6 withdrew, 5 were lost, 16 did not come for the visit, and 95 (85%) were seen at 3 years of age. Groups did not differ by gender, race, socioeconomic status, maternal education, or marital status of parents. At follow-up at 3 years, group demographic and medical characteristics did not differ significantly from the sample recruited at birth.

The following neonatal medical and demographic information was obtained from the hospital chart: infant gestational age (based on both the date of the last menstrual period and the Ballard examination),[31] birthweight (g), length (cm), head circumference (cm), and Apgar scores at 1 and 5 minutes. The presence or absence of respiratory distress syndrome, BPD, patent ductus arteriosis (PDA), necrotizing enterocolitis (demonstrated, with or without surgery), septicemia, retinopathy of prematurity, and failed hearing screening was recorded, as well as the number of days of ventilator support and of supplemental oxygen, and peak bilirubin levels. Also noted was the presence or absence of the following neurologic abnormalities: minor neurologic malformations, seizures, echodense lesions, porencephaly, hydrocephaly, ventriculoperitoneal shunt, meningitis, and periventricular leukomalacia.

Cranial ultrasound studies were performed and reviewed by board-certified radiologists, typically at 3, 10, and 28 days, as well as before discharge. A system for rating the severity of intraventricular hemorrhage (IVH) was devised based on the extent of the lesion. A score of 0 was used to indicate no hemorrhage. Identifiable lesions were graded on a scale from 1 to 4, based on the criteria of Papile.[32] At least one ultrasound study was available for all infants; ratings were based on the most severe lesion diagnosed. A total neurologic risk score was calculated for the above neurologic risk factors, wherein 0 was used to indicate absence and 1 was used to indicate presence.

Procedures

All children recruited at birth were scheduled for the 3-year follow-up visit, with ages corrected for prematurity, at which time language assessments were completed in one or two sessions at the behavioral laboratory of the Department of Pediatrics. Families were reimbursed for transportation to the assessment and given a stipend of $25. This study was approved by the Institutional Review Boards of the hospitals which participated, and maternal informed consent was obtained for all subjects.

The Bayley Scales of Infant Development II[33] and the Communication Domain Subscale of the Battelle Developmental Inventory[34] were administered. The Mental Development Index (MDI) of the Bayley Scales reflects overall cognitive development with a standard score that has a mean of 100 and standard deviation of 15. The Scales are normed for children up to 42 months of age. The Battelle provides a standardized measure of developmental skills in five functional domains for children from birth to age 8 years. Items of the Communication subscale in the receptive subdomain measure discrimination, recognition, and understanding of sounds, words, and gestures, whereas items in the expressive subdomain assess their production and use in communication. Receptive, expressive, and total communication scores were converted to developmental quotients (DQs) for comparison with Bayley standard scores. Battelle DQs have a mean of 100 and a standard deviation of 15.

Data Analysis

Student's *t* tests (for continuous data), χ^2 (for categorical data), or test of proportions, with correction for continuity for proportions, were employed to compare groups on demographic and medical birth characteristics, and to compare the VLBW groups in incidence of medical complications associated with preterm birth.

To assess the effects of BPD and VLBW birth on language outcomes, one-way analyses of variance (ANOVAS) were used to compare the three groups. To assess whether BPD and VLBW birth were related to specific language impairment above and beyond the effects of IQ, analyses of covariance (ANCOVAS) were conducted, using the Bayley MDI as a covariate. Hierarchical multiple regression analyses were used to test the hypothesis that BPD predicted poorer language outcomes after controlling for the effects of socioeconomic (SES) and other medical risk factors. Stepwise multiple regression (MAXR) was used to understand prediction on the basis of cumulative risk and to measure the total predictive power of BPD in comparison with other perinatal risk factors. For regression analyses, only the two VLBW groups were considered because there were no medical risk factors in the term group. All medical and sociodemographic risk factors, i.e., race, socioeconomic status, multiple birth, gestational age, birth weight, neurologic risk score, presence of septicemia, patent ductus arteriosis (PDA), retinopathy of prematurity, BPD, necrotizing enterocolitis, and IVH, as well as peak bilirubin, were included in these analyses.

RESULTS

As previously described,[22] infants with bronchopulmonary dysplasia (BPD) were smaller and more immature than very low birth weight (VLBW) and term control infants (Table 1). The BPD group had a higher neurologic risk summary score, attributable to a higher incidence of neonatal seizures and intraventricular hemorrhage (IVH) but did not differ in the occurrence of other neurologic complications of prematurity (Table 2). The BPD group had a higher incidence of retinopathy of prematurity (ROP), patent ductus arteriosis (PDA), and septicemia, and lower peak bilirubin levels. There were no differences in the incidence of failed hearing screenings as measured by brain stem-evoked response recordings at birth.

At 3 years of age, children with history of VLBW with BPD achieved language standard scores that were significantly lower than the other two groups on receptive, expressive, and total communicative competence scores. VLBW children without BPD performed significantly better than BPD children, but were significantly lower than matched term children on receptive skills. They did not differ from term children in expressive or total communication domains. A high percentage of all children had both receptive and expressive language scores that fell within the impaired range of functioning (developmental quotient [DQ] < 85) (Table 3). However, children with a history of BPD were significantly more likely to score in the impaired range than VLBW and term children. BPD was related to lower receptive language scores at age 3 years, even after controlling for the confounding effect of lower IQ, indicating specific language impairment in this domain. The effect of BPD on expressive and total communication quotients, however, was no longer significant after controlling for IQ.

Hierarchical stepwise multiple regression analyses allowed the assessment of the relationship of BPD to language measures after adjusting for the confounding factors of sociodemographic and medical risk. Because these regression results did not differ among the three domains of the Battelle, only the Total Communication DQ is described in further analyses. Results showed that BPD no longer predicted 3-year Battelle scores after controlling for other medical risk factors associated with BPD.

Rather, presence of PDA, lower socioeconomic status, minority race, and higher neurologic risk were the primary factors predicting language outcome. Table 4 presents the rank order listing of risk factors in the order of magnitude of effect and the number of DQ points lowered by the risk factor. Presence of PDA accounted for a 13-point decrement; minority race, a 6-point decrement; lower socioeconomic status, a 5-point decrement; and higher neurologic risk, a 5-point decrement.

Because PDA was the best single predictor of 3-year language scores and occurred more frequently in the children with BPD at birth (56%) in comparison with the VLBW controls (18%), the possibility that PDA might differentially affect language outcome within the BPD group was tested. When we compared BPD and VLBW infants with and without PDA on language outcomes, controlling for IQ, BPD was found to exacerbate the effects of PDA on language. Children with BPD and a history of PDA had a 14-point decrement in language score (Table 5) compared with a 7-point decrement in VLBW children with PDA but without BPD. Both BPD- and non-BPD-VLBW children without a history of PDA had equivalent scores.

Within the BPD group, there was a trend for the presence of PDA to be associated with a greater duration of time dependent on oxygen and more severe BPD (r [88] = .19; $p <$ .08). However, the presence of PDA had a detrimental effect on language, even for children within the VLBW group, all of whom were on oxygen for less than 14 days.

DISCUSSION

The findings of this study support previous research suggesting that children with very low birth weight (VLBW) and with a history of bronchopulmonary dysplasia (BPD) are at risk for specific receptive language delays over and above speech/language delays because of lower general intelligence.[27,30] Children with a history of BPD exhibited receptive language delays at 3 years of age, after the variables of IQ, race, gender, socioeconomic status, maternal education, and marital status of the parents were controlled, and were more likely than other VLBW children to fall within the impaired range of language functioning, whereas VLBW children without BPD were not different from term controls. Unlike previous studies, the current study did not find hearing loss to be more frequent in the BPD group. Thus, deficits in receptive language cannot be attributed to lower hearing acuity. However, as of yet, measures of central auditory processing in children with BPD have not been examined. The receptive language deficits observed in this sample may be related to auditory processing difficulties. The presence of BPD was, however, associated with an increase in the presence of neurologic risk factors and an increase in patent ductus arteriosis (PDA), which were the major contributors to speech-language delays. PDA and

the presence of neurologic problems were the only neonatal medical risk factors to predict language development when the relative contributions of all factors were considered. As has been found in previous studies, neurologic complications were associated with poorer language outcomes.[11,13-15] The neurologic factors that were more likely to be found in the BPD group included intraventricular hemorrhage (IVH) and seizures, both of which were related to poorer speech language outcomes in prior studies.[11,14,15,18]

Previous studies have suggested that motor delays in children with BPD may affect the articulatory/motor components of speech development.[27,28] Thus, the differences in expressive language skills between children with BPD and children without BPD may be partially mediated by poorer motor skills.

An unexpected finding was that the presence of PDA was the best predictor of language deficits and that the combined occurrence of PDA and BPD resulted in differentially lower language scores. In contrast, the presence of PDA was not found to be a significant risk factor in the same population when cognitive and motor outcomes of children with BPD and VLBW were evaluated at the same age,[22] and thus, suggests an effect specific to language functions. The mechanisms by which a history of PDA in VLBW children may impact later language skills are unclear and the subject of speculation. PDA results in alterations in blood flow to many different organ systems and is associated with the development of bronchopulmonary dysplasia.[35-38] Thus, PDA may be a marker for the most severe cases of BPD.[39] Indomethacin administration, associated with treatment of BPD, has been shown to increase oxygen and surfactant requirement[35] and may exacerbate the hypoxic effects of BPD. However, the negative effects of PDA on language development were also found in non-BPD VLBW children, although to a lesser extent. Anatomically, the laryngeal nerve, important in language development, crosses the ductus arteriosis and may be negatively affected by PDA. Developmentally, the presence of PDA may also affect vagal tone, an autonomic measure of cardiac efficiency. Vagal tone, as measured by respiratory sinus arrhythmia (RSA), has been associated with measures of physiological self-regulation in early infancy and is thought to play an important role in regulatory processes via communication with brain stem nuclei implicated in attention and communication.[40]

Lower socioeconomic status and minority race also predicted poorer language outcomes although caution regarding interpretation of the relationship between race and scores on the language measure should be taken. African-American children from low socioeconomic status families often use language systems that differ from standard American English, which place them at a disadvantage on standardized tests of language development.[41,42] The fact that a large percentage of all groups of children scored one standard deviation or more below the mean on this test supports this view and also reflects the large proportion of lower socioeconomic families found disproportionately in VLBW cohorts. Moreover, because race and socioeconomic factors covary in the United States, it is difficult to specify the extent to which they are independently associated with developmental performance.[43] Family income and parental educational level have been shown to be more significant influences on development than race or ethnicity.[44] In the present study, however, whereas minority race and low socioeconomic status were confounded, medical and neurologic complications were not associated with socioeconomic status or race.

The findings of this study also suggest that medical factors associated with BPD have a greater impact on receptive than on expressive language skills. Significant differences between groups were found on the receptive language quotient but not on the expressive language quotient after controlling for IQ, supporting a previous study of school age VLBW children.[30] Several explanations may be proposed for the discrepancy between receptive and expressive language scores. First, the language test itself may be more sensitive to receptive language difficulties than expressive problems. The scores of all three groups reflected poorer performance on the receptive subscale than the expressive. A second explanation may be that receptive and expressive language skills differ in the underlying genetic, neurological, and cognitive processes that they use and that both BPD and receptive language processes may share similar genetic bases.[36] Third, receptive language abilities may be more closely tied to auditory processing skills and the speed of the processing of sensory input, than are expressive language abilities.[40,45] The effects of BPD and other concomitant sequelae of prematurity, such as IVH, seizures, and hypoxia, may have specific negative effects on language comprehension, whereas the effects of BPD on expressive language may be mediated through the effects of BPD on overall intelligence.

This study highlights the need to consider both the medical complications of prematurity and the sociodemographic factors in the follow-up of VLBW infants.[22,46] Neurologic complications and a history of BPD and PDA conferred substantial additional risk for language delays in VLBW children. As has been found for other developmental outcomes,[22] severity of medical complications rather than lower birth weight and shorter gestation, per se, predicted outcomes within the VLBW cohort.

Early speech and language surveillance of VLBW children with these neurologic and medical complications is indicated, especially when combined with risk from low socioeconomic status and minority status. Early intervention programs should focus on the development of receptive language skills, auditory processing abilities, and speech-sound production, as well as more general language, cognitive, and motor development. Future prospective longitudinal studies that follow children with a history of BPD into early school age are needed to explore how early medical complications of prematurity interact with environmental risk factors to impact language development. Delineating relationships between specific medical complications of prematurity and specific child outcomes may lead to better identification of VLBW children who are at greater risk for later learning problems and may illuminate the biological mechanisms implicated in the adverse effects of VLBW and prematurity.

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Table 1.

Sociodemographic Characteristics at Birth

	VLBW with BPD (n = 122)			VLBW (n = 84)			Term (n = 123)				
	Mean	**SD**	**%**	**Mean**	**SD**	**%**	**Mean**	**SD**	**%**	**F**	***p***
Birthweight (g)	956	248		1252	178		3451	526		1633	.001*
Gestational age (wk)	27	2		30	2		40	1		1416	.001*
Social class	3.5	1		3.6	1		3.6	1		.7	.71
Total oxygen (days)	106	149		5	5		0	0		44.5	.001**
Race (white)			55			48			51	1.1	.58
Gender (male)			52			43			50	1.9	.39
Multiple birth			21			43			10	31.5	.001***

VLBW, very low birth weight; BPD, bronchopulmonary dysplasia.

*BPD < VLBW < Term, $p < .05$

**BPD and VLBW > Term, $p < .05$

***VLBW > BPD and Term, $p < .05$.

Table 2.

Medical and Neurologic Complications by Group

	BPD			VLBW				
	Mean	**SD**	**%**	**Mean**	**SD**	**%**	χ^2	***p***
Minor neurologic malformations			1			1	0.1	1.00
Seizures			7			0	5.7	.02
Porencephaly			3			4	0.0	1.00
Hydrocephalus			9			5	1.3	.28
Ventriculoperitoneal shunt			3			1	0.4	.64
shunt								
Meningitis			2			0	1.4	.51
Intraventricular hemorrhage			43			18	13.9	.01
Grades I–II			28			14	5.3	.02
Grades III–IV			15			4	6.8	.01
Periventricular leukomalacia			8			7	0.2	.78
Neurologic risk score[a]	1.3	2		.58	1		3.34[b]	.001
Necrotizing enterocolitis			4			6	.4	.74
Retinopathy of prematurity			43			4	39.2	.001
Patent ductus arteriosis			56			18	29.7	.001
Hearing abnormal			16			11	.7	.52
Peak bilirubin	8.3	3		9.1	3		1.8[b]	.05
Septicemia			47			24	11.2	.001

BPD, bronchopulmonary dysplasia; VLBW, very low birth weight.

[a]Neurologic risk score is a summary variable, calculated by summarizing the number of zeros and ones, where zero was used for absence and one for presence of the above listed neurological risk factors. The neurological risk score could range from 0–8 for an individual infant.

[b]*t* value.

Table 3.

Language, Mental, and Motor Outcomes at Three Years

	BPD (n = 90)			VLBW (n = 65)			Term (n = 91)				
	Mean	**SD**	**%**	**Mean**	**SD**	**%**	**Mean**	**SD**	**%**	**F/x^2**	***p***
Battelle Scale											
Receptive DQ	84.7	17		91.5	17		97.3	18		11.1	<.02 *
Expressive DQ	92.8	22		99.7	17		101.3	20		4.3	<.02 **
Communication DQ	89.4	21		96.4	19		99.8	20		5.9	<.005 **
Receptive DQ < 85			49			34			30	3.7	<.05 **
Expressive DQ < 85			44			25			25	4.1	<.05 **
Communication DQ < 85			43			31			28	2.3	NS
Bayley Scales	(n = 98)			(n = 70)			(n = 95)				
MDI	83.7	24		90.0	16		96.4	12		11.2	<.0001 *
PDI	84.1	28		97.4	19		102.8	14		17.6	<.0001 **

BPD, bronchopulmonary dysplasia; VLBW, very low birth weight; DQ, developmental quotient; MDI, Mental Development Index; PDI, Psychomotor Development Index; NS, not significant.

* BPD < VLBW < Term, $p < .05$

** BPD < VLBW and Term, $p < .05$.

Table 4.

The Best Four-Variable Model for Predicting the Effects of Medical and Demographic Factors on Communicative Competence

Criterion	Communication DQ	beta[a]	Unstandardized R	R^2[b]	F	*p*
Step 1	Patent ductus arteriosis	−.31	−13.14	.09	16.1	.001
Step 2	Socioeconomic status	−.25	−4.58	.15	14.0	.001
Step 3	Neurologic risk	−.19	−5.42	.19	11.8	.001
Step 4	Race[c]	−.15	−6.27	.21	9.4	.001

[a]Standardized regression coefficient.

[b]Cumulative R^2.

[c]No other risk factor added significantly to the prediction model. These included multiple birth, necrotizingenterocolitis, septicemia, peak bilirubin, retinopathy of prematurity, gestational age, and birthweight.

Table 5.

Communication Developmental Quotients of BPD and VLBW Children With and Without a History of PDA

	BPD		VLBW	
	Mean	**SD**	**Mean**	**SD**
+ PDA	83.6	19	89.4	20
− PDA	97.8	19	96.8	19

BPD, bronchopulmonary dysplasia; VLBW, very low birth weight; PDA, patent ductus arteriosis.