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PREDICTIVE FACTORS FOR NEUROMOTOR ABNORMALITIES AT THE CORRECTED AGE OF 12 MONTHS IN VERY LOW BIRTH WEIGHT PREMATURE INFANTS

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Abstract – **Background:** The increase in survival of premature newborns has sparked growing interest in the prediction of their long-term neurodevelopment. **Objective:** To estimate the incidence of neuromotor abnormalities at the corrected age of 12 months and to identify the predictive factors associated with altered neuromotor development in very low birth weight premature infants. **Method:** Cohort study. The sample included 100 premature infants. The outcome was neuromotor development at 12 months classified by Bayley Scale (PDI) and neurological assessment (tonus, reflexes, posture). A multivariate logistic regression model was constructed. Neonatal variables and neuromotor abnormalities up to 6 months of corrected age were selected by bivariate analysis. **Results:** Mean birth weight was 1126g (SD: 240). Abnormal neuromotor development was presented in 60 children at 12 months corrected age. **Conclusion:** According to the model, patients with a diagnosis including bronchopulmonary dysplasia, hypertonia of lower extremities, truncal hypotonia showed a 94.0% probability of neuromotor involvement at 12 months.

KEY WORDS: infant, premature, child development, psychomotor performance, risk factors.

Fatores preditivos para anormalidades neuromotoras aos 12 meses de idade corrigida em prematuros de muito baixo peso

Resumo – **Introdução:** O aumento na sobrevida de recém-nascidos prematuros tem suscitado interesse crescente na predição do seu neurodesenvolvimento a longo prazo. **Objetivo:** Estimar a incidência de anormalidades neuromotoras aos 12 meses de idade corrigida e identificar os fatores associados ao desenvolvimento neuromotor alterado em prematuros de muito baixo peso. **Método:** Estudo de coorte. A amostra incluiu 100 crianças prematuras.O desfecho foi o desenvolvimento neuromotor aos 12 meses. Modelo de regressão logística multivariado foi construído. Variáveis neonatais e anormalidades neuromotoras até os 6 meses de idade corrigida foram selecionadas por análise bivariada. **Resultados:** O peso de nascimento médio foi 1126g (DP:240). Aos 12 meses 60% das crianças apresentaram desenvolvimento neuromotor alterado. **Conclusão:** De acordo com o modelo, pacientes com diagnóstico incluindo displasia broncopulmonar, hipertonia de membros inferiores e hipotonia de tronco tinham 94% de probabilidade de comprometimento neuromotor aos 12 meses.

PALAVRAS-CHAVE: desenvolvimento infantil, desempenho psicomotor, criança, prematuro, fatores de risco.

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The increase in survival of premature newborns in recent decades has sparked growing interest in the prediction of overall long-term development of these children. In the follow-up of these infants during the first year of life, various authors have detected abnormal neurological signs, while it has proven difficult to predict whether these signs will be transient or definitive[1-3]. The severity of illness in premature newborns related to lower gestational age and lower birth weight can be associated with abnormalities in muscle tone[4] and reflexes, thereby raising interest among neonatologists in risk factors related to altered infant development.

According to Samson et al.[5] alterations in tonus in the first months of life and inadequate postural control at 12 months appear to have repercussions on neuromotor function at school age. Therefore, the identification of neonatal and post-neonatal characteristics associated with compromised neuromotor development in premature infants allows the earliest possible referral of this group to appropriate early intervention programs, thus optimizing their functional capacity and minimizing their future degree of neuromotor deficit.

This study's objective was to verify the incidence of neuromotor alterations in the first year of life and identify neonatal and post-neonatal predictive factors associated with altered neuromotor development at 12 months corrected age in very low birth weight premature infants, through a predictive model.

METHOD

This was a prospective cohort study of premature newborns with birth weight less than 1500g. We excluded infants with genetic syndromes, congenital malformations, congenital infections, those born in other neonatal units, and deaths before hospital discharge. Gestational age at birth was obtained from date of last menstrual period and obstetric ultrasound performed in the first trimester of pregnancy. In the absence of this information, the estimate was performed using the Ballard method[6].

Data on the neonatal period were collected from patient charts, beginning when the newborns were included in the study. Cranial ultrasound was performed in the first week of life and near hospital discharge, and classification of periintraventricular and parenchymatous hemorrhage was based on Papile et al.[7].

After being discharged from the neonatal unit, the children were followed monthly by the High-Risk Newborn Follow-up Outpatient Clinic until they reached 12 months of corrected age. At each appointment, the children's overall development was assessed by a pediatrician and a physical therapist specialized in motor development, including observation of spontaneous movements and acquisition of motor milestones[8,9] in addition to a neurological examination including muscle tone assessment,strength, reflexes, joint angles and posture[10]. Children were examined when they were calm, preferably not crying, and with the parents present during the examination. Maneuvers were conducted to assess cervical, shoulder girdle, trunk, and limb tonus. The examination also included observation of presence of tonus and reflex asymmetries.

Hypertonia was defined as increased tonus in the upper extremities and/or trunk and increased adductor and extensor tonus in lower extremities. Hypotonia was defined as decreased muscle tone and increased joint mobility[11].

Delayed neuromotor development was defined as the child not being able to achieve the appropriate motor milestones for corrected age at the time of examination based on our clinical and neurological assessment[8,10], associated to the results of Denver II test[9]. In the age groups from 0 to 3 months and from 3 months and 1 day to 6 months, the presence or absence of the following were verified: cervical hypotonia, truncal hypotonia, hypotonia of extremities, cervical hypertonia, truncal hypertonia, shoulder girdle retraction; hypertonia of extremities; tonus or reflex asymmetry; delayed motor development.

As the criterion for classifying transient neuromotor alterations, we used postural and muscle tone alterations that resolved before the child reached 12 months corrected age.

Bayley Scales of Infant Development (BSID-II) was administered by a psychologist at 6 and 12 months corrected age. Only the psychomotor development index (PDI) of the BSID-II was considered for this study using the following cutoff points: >85 was considered as normal; 71-85 was considered mild impairment and ≤70 was considered as serious impairment[12.] This assessment was conducted independently of each other (by the pediatrician and the physical therapist).

Both the pediatrician's and physical therapist's decisions regarding neuromotor development and Bayley score were taken into consideration to compute a new variable on final neuromotor development

At 12 months corrected age, the child was classified as having normal or altered neuromotor development (the latter based on at least two alterations[13] in the neurological examination as reflex abnormality, focal neurological signs, any of the tonus alterations mentioned above; delay in motor milestones[13,14] , or PDI score <85 at 12 months corrected age.

Data collection was prospective, and the outcome was defined as altered neuromotor development at 12 months corrected age.

Neonatal variables possibly related to the outcome were: bronchopulmonary dysplasia (BPD), small for gestational age (SGA), abnormal cranial ultrasound (periintraventricular or parenchymatous hemorrhage or leukomalacia), male gender, use of mechanical ventilation, patent ductus arteriosus (PDA), septicemia with positive blood culture, and gestational age categorized as less than versus greater than or equal to 28 weeks, birth weight less than 750g, necrotizing enterocolitis, head circumference at birth below the 10th percentile[15,16].

Candidate post-neonatal variables were delayed motor development and altered tonus in the various body segments and asymmetries, which corresponded to the first identification of

alterations during any consultation from 0 to 3 months and from 3 to 6 months of age. Corrected age corresponding to full term (40 weeks) was defined as zero. Bronchopulmonary dysplasia was defined as the utilization of oxygen therapy for 28 days or longer[17]. SGA was defined as birth weight below the 10th percentile according to the intrauterine growth curve[18].

Sample size calculation

Considering a 50% incidence of motor alterations in the group of exposed premature infants, a relative risk of 2.5, a 95% significance level, and 80% power, a sample of 99 children was needed.

From January 2004 to December 2006, 217 very low birth weight newborns were admitted to the Neonatal Intensive Care Unit (NICU). Ninety-one premature newborns were excluded (26 with congenital malformations, 4 with congenital infections, 3 with genetic syndromes, 8 born in other hospitals, 44 that died before hospital discharge and 6 transferred to other hospitals). Two children died after discharge, at 1 month corrected age, and 24 (18.9%) did not complete the follow-up protocol up to 12 months corrected age. The study thus included 100 premature newborns with birth weight less than 1500g, born in the maternity ward of a tertiary public hospital in Rio de Janeiro, Brazil.

Statistical analysis

The independent variables that showed a p value of less than 0.25 in the bivariate analysis were included in the multivariate model as potential factors associated with the outcome. To select the predictive variables for altered neuromotor development at 12 months corrected age, we constructed a predictive model using logistic regression with neonatal variables and developmental alterations observed in the first 6 months of life that remained in the model, selected by the stepwise forward method, with a significance level of $p<0.05$. The model was calibrated by the Hosmer and Lemeshow test[19] using the goodness-of-fit statistic. The model's discrimination in relation to the patients that evolved versus did not evolve to the outcome was verified by the receiver-operator curve (ROC), constructed by sensitivity and specificity estimates. In addition, we estimated the predictive values for developing a neuromotor alteration at 12 months of age for each group of variables in the adjusted predictive model. The statistical analysis used the STATA 8.0 software package (Stata Corp, College Station, TX).

This study is part of a research project that was approved by the Human Research Ethics Committee of the Instituto Fernandes Figueira and the parents signed a free and informed consent form.

RESULTS

Mean birth weight was 1126g (SD:240) and mean gestational age was 29 weeks and 6 days (SD: 2.0 weeks). Forty-seven percent (n=47) were males. Approximately one-fourth of the sample consisted of newborns with gestational age less than 28 weeks, 32% (n=32) newborns developed bronchopulmonary dysplasia, of which 11 cases were moderate and 1 severe. A total of 58 (58%) received assisted ventilation, 40% presented PDA, 42% were SGA, 21% showed altered cranial ultrasound (cerebral haemorrhage and/or periventricular leukomalacia). Grade III cerebral haemorrhages occurred in only 4 preterm infants and all of them showed abnormal neuromotor development at 12 months corrected age. None of the children had grade IV haemorrhage or neonatal seizures. Cerebral haemorrhage (grades I and II) occurred in 15 babies and 60% of them had altered neuromotor development. Necrotizing enterocolitis was observed in two babies. Twelve newborns (12%) presented culture-proven septicemia. Head circumference at birth less than 10th percentile was observed in 28 newborns. Only three premature infants in this cohort received postnatal corticosteroid therapy. Mean hospitalization time was 56 days. As for socioeconomic status, 7% of the families earned less than the minimum wage (approximately U$ 125/month) and 43% earned less than twice the minimum wage (U$ 250/month). Twenty-seven percent of the mothers of these children had not finished elementary school.

The maternal, social, neonatal, and post-neonatal characteristics of the children that were lost to follow-up were compared to those of the children that completed the study, and there was no statistically significant difference between the two groups for these characteristics.

Table 1 shows the principal neuromotor alterations found in the first 6 months of life. One child was dis-

Table 1. Neuromotor alterations identified in the first six months of corrected age in very low birth weight premature children.

Neuromotor alterations	Age*	Frequency (%)
Cervical hypotonia	0–3 months	54 (54.5)
	3–6 months	10 (10.0)
Truncal hypotonia	0–3 months	46 (46.5)
	3–6 months	40 (40.0)
Hypotonia of lower extremities	0–3 months	4 (4.0)
	3–6 months	5 (5.0)
Hypotonia of upper extremities	0–3 months	0 (0.0)
	3–6 months	1 (1.0)
Overall hypertonia	0–3 months	15 (15.2)
	3–6 months	11 (11.0)
Truncal hypertonia	0–3 months	19 (19.2)
	3–6 months	11 (11.0)
Hypertonia of lower extremities	0–3 months	49 (49.5)
	3–6 months	54 (54.0)
Hypertonia of upper extremities	0–3 months	21 (21,2)
	3–6 months	26 (26.0)
Scapular retraction	0–6 months	67 (67.0)

*age from 0–3 months: n=99 children; from 3–6 months, n=100 children.

Table 2. Association between neonatal and post-neonatal variables and neuromotor development at 12 months corrected age in very low birth weight premature children.

	Relative risk	95% CI	p value
Neonatal variables			
Bronchopulmonary dysplasia	1.63	1.22 - 2.17	**0.003**
SGA	1.06	0.76–1.46	0.74
PDA	1.21	0.88–1.65	0.24
Male gender	1.47	1.06–2.04	0.02
Neonatal pneumonia	1.59	1.20–2.08	**0.04**
Altered cranial ultrasound	1.25	0.90–1.75	0.23
IVH grades III and IV	1.71	1.45–2.03	0.12
Septicemia	1.64	1.28–2.12	**0.01**
Gestational age <28 weeks	1.35	0.99–1.85	0.08
Birth weight <750g	1.61	1.24–2.1	**0.02**
Necrotizing enterocolitis	1.68	1.40–1.9	0.18
Head circumference at birth <10th percentile	0.99	0.90–1.09	0.90
Post-neonatal variables			
Hypertonia of lower extremities at 0–3 months	1.49	1.05–2.08	0.02
Hypertonia of lower extremities at 3–6 months	1.70	1.18–2.45	**0.002**
Truncal hypertonia at 0–3 months	1.18	0.83–1.7	0.38
Truncal hypertonia at 3–6 months	1.25	0.83–1.86	0.28
Hypotonia of lower extremities at 0–3 months	0.40	0.07–2.25	0.18
Hypotonia of lower extremities at 3–6 months	0.65	0.22–1.93	0.31
Overall hypertonia at 0–3 months	1.60	1.19–2.08	**0.02**
Overall hypertonia at 3–6 months	1.42	1.02–1.99	0.10
Cervical hypotonia at 0–3 months	0.75	0.54–1.04	0.08
Cervical hypotonia at 3–6 months	1.18	0.76–1.84	0.37
Truncal hypotonia at 0–3 months	0.79	0.56–1.10	0.16
Truncal hypotonia at 3–6 months	1.40	1.02–1.91	**0.04**
Scapular retraction at 0–6 months	1.35	0.91–2.0	0.09
Asymmetry of tonus at 0–6 months	0.95	0.63–1.46	0.83
Social variables			
Family income <1 minimum wage	0.71	0.29–1.7	0.31
Family income <2 times minimum wage	0.85	0.6–1.2	0.35
Mather's educational level <8th grade	0.83	0.55–1.24	0.33

SGA: Small for gestational age; PDA: Patent ductus arteriosus; IVH: intraventricular haemorrhage; Altered cranial ultrasound (IVH or leukomalacia); CI: Confidence Interval

Table 3. Selected variables for the predictive model for abnormal neuromotor development at 12 months of life.

	OR	95% CI	p
Bronchopulmonary dysplasia	4.1	1.4–12.1	0.01
Hypertonia of lower extremities at 3–6 months	3.4	1.4–8.4	0.008
Truncal hypotonia at 3–6 months	2.9	1.1–7.4	0.03

The model's calibration showed a good fit (C statistic=1.9; degree of freedom=7; p=0.86); OR: odds ratio; CI: confidence interval.

charged from the Neonatal Unit after 3 months of corrected age, so his follow-up began after that date.

In the first three months of life, the most frequent alterations were cervical hypotonia and hypertonia of the lower extremities, while from 3 to 6 months hypertonia of the lower extremities was the most frequent.

Asymmetry of tonus in the first 6 months of corrected age was present in 19 children (19%) (data not shown in the Table). Transient muscle tone alteration occurred in 58 children, and 7 presented totally normal evolution throughout follow-up. At 12 months corrected age, 35 children (35.0%) in the study sample presented altered neurological examination and 51% presented altered PDI (<85). At 12 months both altered neurological examination and psichomotor development index (PDI) were observed in 26 children. Abnormal neurological examination or altered PDI were presented in 60 children at 12 months corrected age.

Table 4. Probability of altered neuromotor development at 12 months corrected age according to the final model selected.

Variable	Probability
Hypertonia of lower extremities at 3–6 months, bronchopulmonary dysplasia, truncal hypotonia at 3–6 months	94.0
Hypertonia of lower extremities at 3–6 months, bronchopulmonary dysplasia	84.0
Bronchopulmonary dysplasia, truncal hypotonia at 3–6 months	81.0
Hypertonia of lower extremities at 3–6 months, truncal hypotonia at 3–6 months	78.0
Bronchopulmonary dysplasia	60.0
Hypertonia of lower extremities at 3–6 months,	55.0
Truncal hypotonia at 3–6 months	51.0
Absence of the above factors	26.0

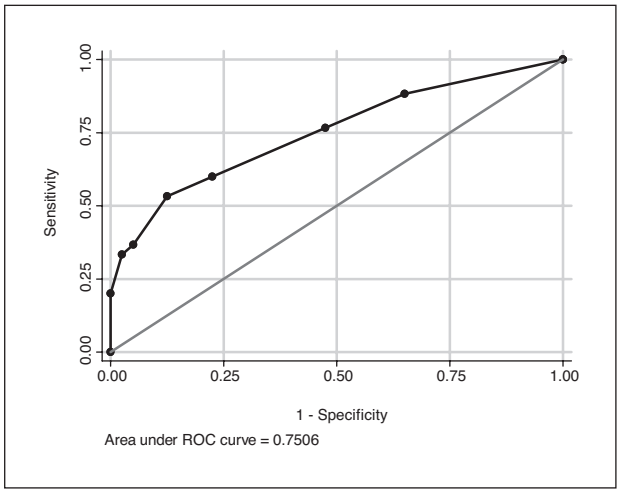

Figure. The discrimination of the model assessed by the area under receiver operating characterist curve (ROC curve).

Children with altered neuromotor development had significantly lower PDI than children with normal neuromotor development at 12 months (73.5±12.1×96.1±13.8 p=0.00001).

The variables that showed a statistically significant association with altered neuromotor development at 12 months were "bronchopulmonary dysplasia", "hypertonia of lower extremities (from 3 to 6 months)", and "truncal hypotonia (from 3 to 6 months)". The variables SGA, PDA, and altered neonatal ultrasound were not associated with altered neuromotor development at 12 months (Table 2).

After adjustment, the following variables remained in the model as predictive factors: hypertonia of lower extremities, BPD, truncal hypotonia (Table 3).

The model's discriminatory capacity is shown in Figure. The model's sensitivity was 88.3% (CI: 78.3–94.7), specificity 35.0% (CI: 21.5–50.6), positive predictive value 67.1% (CI: 56.2–76.7), and negative predictive value 66.7% (CI: 44.9–84.1).

Table 4 shows the probability of altered neuromotor development at 12 months according to the presence of a set of neonatal and post-neonatal variables, based on the proposed predictive model. When the patients presented all the following characteristics: BPD, hypertonia of lower extremities from 3 to 6 months, truncal hypotonia from 3 to 6 months the probability of altered neuromotor development was 94%.

DISCUSSION

The multivariate logistic regression model consisted of three selected variables. The model showed good sensitivity and good positive predictive value. According to the model, patients with a diagnosis including bronchopulmonary dysplasia, hypertonia of lower extremities, truncal hypotonia showed a 94.0% probability of motor involvement We will now discuss the model's significant variables.

BPD

The contribution of BPD to developmental alterations is controversial. Various studies have concluded that BPD carries an additional risk of developmental deficit[20]. Meanwhile, other investigators have concluded that perinatal factors like intraventricular haemorrhage, low birth weight, and low gestational age are the principal factors[21]. According to other authors, the severity and chronicity of the respiratory disease were the best predictors of motor deficit at 1 to 2 years of age [22] and neuromotor deficit at school age[23]. In the current study, BPD showed an OR of 4.1 for neuromotor deficit at 12 months (CI: 1.4–12.1).

Hypertonia of lower extremities

We found hypertonia of lower extremities at 0 to 3 months in 49 children (49.5%) and in 54% of children at 3 to 6 months, while Samson et al.[2] reported 11.5% at 3 months, while noting that more than 50% of their sample had failed to normalize lower limb tonus at 6 months. The findings of Georgieff et al.[24] were more similar to ours, with a high incidence of hypertonia of lower extremities (62% at 3 months and 71% at 6 months). In the current study, hypertonia of lower extremities at 3 to 6 months

showed an OR of 3.4 for altered neuromotor development at 12 months (CI: 1.4–8.4).

Truncal hypotonia

Premature infants commonly display abnormalities of neuromuscular examination. Truncal tone abnormalities at 3 months were most useful in distinguishing infants at risk for poor developmental status. Infants with truncal tone abnormalities at 3 months, particularly hypotonia are at greatest risk for poor developmental outcome than infants with either hypertonia or normal tone[24]. Samson et al.[2] reported that some 30% of the children in their study had failed to normalize their axial tonus at 6 months,

A structured neurological examination was performed in infants with cystic periventricular leukomalacia at 6–9 months corrected age.Truncal hypotonia and extended arms and legs were associated with unsupported sitting but not walking at 2 years[25]. In the current study truncal hypotonia showed an OR of 2.9 for altered neuromotor development at 12 months (CI:1.1–7.4).

The identification of 35% of children with altered neurological assessment is consistent with findings by other authors. The incidence tends to decrease as the children reach 18 months, as reported by Sommerfelt et al.[26]. These authors found that 28% of children showed altered tonus in at least one of the examinations performed, at 4, 7, 13, and 18 months, with the highest incidence of alterations at 7 months and a drop by 18 months. At early ages (5 to 8 months), it is difficult to determine whether a neuromotor alteration will be transient or definitive (cerebral palsy)[11].

According to some reports, despite resolution of abnormalities before 15 months of life, the group of children that developed transient abnormalities showed a significantly lower mental development index at 24 months as compared to the group without neuromotor abnormalities[1]. In addition, neuromotor alterations identified in early life can lead to inadequate postural control at 12 months that will influence motor function at school age[1, 27].

Although the diagnosis of cerebral palsy should only be confirmed at 2 years of age or older, less severe motor involvement (but which nevertheless requires therapy) can manifest itself at early ages, with deficits in muscle tone, posture, and reflex patterns[25,27]. Hence the importance of identifying the children that still present altered tonus or delayed development at 12 months and of verifying factors associated with these alterations, in order to intervene earlier in children with such risk factors, attempting to prevent and/or minimize these alterations.

The PDI measures the achievement of a border set of motor milestones and in our cohort 51% of the infants had a mildly or significantly delayed psychomotor development. Our results are closer to those of Vohr et al.[13].

Twenty five children for whom pediatrician assigned a normal neurological examination had test score (PDI) in the altered category and nine children with abnormal neurological examination had PDI score in the normal category. Vohr and collaborators[13] verified that seventeen percent of children with abnormal neurologic examination had a normal BSID-II score (PDI >85). Some authors identified similar results in diagnoses by physicians and psychologists[13,28]. Each of these assessment provides unique information about child's neurological findings, developmental motor status and gross motor function[13]. This difference is not surprising because the PDI score reflects a combination of gross motor and fine motor tasks. The BSID-II motor assessment, despite its limitations, has been used as a gold standard for motor performance in many outcome studies of premature infants[13]. However, in medical evaluations of high- risk infants, the best diagnoses and predictions are achieved through a combination of multiple, complementary tools, that is, achieve milestones, neurological examination and assessment of the quality of motor behavior[29].

Early intervention programs for premature children have a positive impact on short and medium-term cognition. However, more research is needed to determine which early developmental interventions are most effective for improving cognitive and motor outcomes and the long-term effects of these programs[30].

There is a paucity of information on neurodevelopmental outcomes of preterm infants in low-resource settings[28]. Since Brazil is a developing country with a large demand for public physical therapy services, it often proves impossible to initiate treatment promptly, and we need to prioritize the earliest possible referral of children at risk to early intervention programs in order to recover their motor function.

Study limitations

One of the most frequent methodological issues in cohort studies involves losses to follow-up. Stoll et al.[15] reported a 20% loss-to-follow-up rate in very low birth weight premature children at 18 to 22 months corrected age. The children we studied belong to a low-income population, and many of the families live on the outskirts of the city of Rio de Janeiro and have difficulty paying for transportation to the hospital. The percentage of losses at different moments in the first year of life was 19%, but there was no significant difference in either the neonatal characteristics or the development variables, compared to the losses among children that remained in the study. Another aspect that deserves attention is the need to validate this model for use in other groups of very low birth weight children, in order to allow the use of this predictive model in other contexts.

In conclusion, the predictive model developed in our

hospital-based study population was able to identify children with increased risk of neuromotor alterations.

Bronchopulmonary dysplasia, truncal hypotonia and hypertonia of lower extremities remained predictive of altered neuromotor development at 12 months corrected age after adjusting for confounding variables.

REFERENCES

1. D'Eugenio DB, Slage TA, Mettelman BB, Gross SJ. Developmental outcome of preterm infants with transient neuromotor abnormalities. AJDC 1993;147:570-574.
2. Samsom JK, de Groot L, Bezemer PD, Lafeber HN, Fetter WPF. Muscle power development during the first year of life predicts neuromotor behavior at 7 years in preterm born high-risk infants. Early Hum Dev 2002;68:103-118.
3. Fallang B, Oien I, Hellem E, Saugstad OD, Hadders-Aldra M. Quality of reaching and postural control in young preterm infants is related to neuromotor outcome at 6 years. Pediatr Res 2005;58:347-353.
4. Silva ES, Nunes ML. The influence of gestational age and birth weight in the clinical assessment of the muscle tone of healthy term and preterm newborns. Arq Neurpsiquiatr 2005;63:956-962.
5. Samson JF, de Groot L. The influence of postural control In motility and hand function in a group of high-risk preterm infants at 1 year of age. Early Human Dev 2000;60:101-113.
6. Ballard JL, Khoury JC, Wedig K, Wang L, Eilers- Ealsman BL, Lipp R. New Ballard score, expanded to include extremely premature infants. J. Pediatr 1991;119:417-423.
7. Papile LA, Burstein J, Burstein R, et al. Incidence and evolution of subependymal and intraventricular hemorrhage: a study of infants with birth weights less than 1500g. J Pediatr 1978;92:529-534.
8. Holt KS. Development in the first year. In: Holt KS (ed) Child development- Diagnosis and assessment. London: Butterworth-Heinemann, 1991:70-102
9. Frankenburg WK, Dodds JB, Archer P, Sharir H, Breusnich. The Denver II: a major revision and restandartization of the Denver developmental screening test. Pediatrics 1992;89:91-97.
10. Amiel Tyson C, Grenier A, Neurological assessment during the first year of life. New York: Oxford University Press, 1986.
11. Pedersen SJ, Sommerfelt K, Makest T. Early motor development of premature infants with birthweight less than 2000 grams. Acta Paediatr 2000;89:1456-1461
12. Bayley N. Bayley Scales of Infant Development. 2nd Ed. San Antonio, Tx: The Psychological Corporation, Harcourt Brace and Co; 1993.
13. Vohr BR, Msall ME, Wilson D, Wright L, McDonald S, Poole WK. Spectrum of gross motor function in extremely low birth weight children with cerebral palsy at 18 months of age. Pediatrics 2005;116:123-129.
14. Restiffe AP, Gherpelli JLD. Comparison of chronological and corrected ages in the gross motor assessment of low-risk preterm infants during the first year of life. Arq Neuropsiquiatr 2006;64:418-425.
15. Stoll BJ, Hansen NI, Adams-Chapman I, et al. Neurodevelopmental and growth impairment among extremely low-birth-weight infants with neonatal infection. JAMA 2004;292;2357-2365.
16. Fenton TR. A new growth chart for preterm babies;Babson and Benda's chart updated with recent data and new format. BMC Pediatr 2003;3:13.
17. Jobe AH, Bancalari E. Bronchopulmonary dysplasia. Am J Respir Crit Care Med 2001;163:1723-1729.
18. Alexander GR, Himes JH, Kayman RB, Mor J, Kogan MA. United States national reference for fetal growth. Obstet Gynecol 1996;87:163-168.
19. Lemeshow S, Hosmer DW Jr. A review of goodness of fit statistics for use in the development of logistic regression models. Am J Epidemiol 1982;115:92-106.
20. Hack M, Wilson-Costelo D, Friedman H, Taylor GH, Schluchter, Fanaroff A. Neurodevelopment and predictors of outcomes of children with birth weights of less than 1000g 1992-1995. Arch Pediatr Adolesc Med 2000;154:725-731.
21. Leonard CH, Clyman RJ, Piecuch RE, Juster RP, Ballard RA, Behle MB. Effects of medical and social risk factors on outcome of prematurity and very low birthweight. J Pediatr 1990;116:620-626.
22. Davidson S, Schrayer A, Wielensky E, Krikler R, Lilos P, Reisner SH. Energy intake, growth and development in ventilated very low birth weight infants with and without bronchopulmonary dysplasia. Am J Dis Child 1990;144:553-559.
23. Majnemer A, Riley P, Shevell M, Birnbaum R, Greestone H, Coates AL. Severe bronchopulmonary dysplasia increases risk for later neurological and motor sequelae in preterm survivors. Dev Med Child Neurol 2000;42:53-60.
24. Georgieff MK, Bernbaum JC, Hoffman-Williamson M, Daft A. Abnormal trunk muscle tone as a useful early marker of developmental delay in low birth weight infants. Pediatrics 1986;77:659-663.
25. Rici D, Cowan F, Pane M, et al. Neurological examination at 6 to 9 months in infants wirh cystic periventricular leukomalacia. Neuropediatrics 2006;37:247-252.
26. Sommerfelt K, Pedersen S, Ellertsen B, Markestad T. Transient dystonia in non-handicapped low birthweight infants and latter neurodevelopment. Acta Paediatr 1996;85:1445-1449.
27. Voss W, Neubauer AP, Wachtendorf M, Verhey JF, Kattner E. Neurodevelopmental outcome in extremely low birth weight infants: what is the minimum age for reliable developmental prognosis? Acta Paediatr 2007;96:342-347.
28. Khan NZ, Muslima H, Parven M,et al. Neurodevelopmental outcomes of preterm infants in Bangladesh. Pediatrics 2006;118:280-289.
29. Heineman KR, Hadders-Algra M. Evaluation of neuromotor function in infancy-A systematic review of available methods.J Dev Behav Pediatrics 2008;29:315-323.
30. Spittle AJ, Orton J, Doyle LW, Boyd R. Early developmental intervention programs post hospital discharge to prevent motor and cognitive impairment in preterm infants. Cochrane Database Syst Rev 2007;18:CD 005495.