Original Article

Motor Development of Very Low Birthweight Infants with Chronic Lung Disease – A Comparative Study

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Abstract

Introduction: To determine whether chronic lung disease (CLD) influences specific aspects of motor development in infancy. Materials and Methods: Twenty-nine very low birthweight infants with CLD at 36 weeks' post-conceptional age and 31 infants without CLD were evaluated at 8 months' and 24 months' corrected age using the Neurosensory Motor Development Assessment. Perinatal and neonatal characteristics of the infants with CLD and control infants were compared using the chi-square test for categorical variables and Student's *t*-test for continuous variables. The relationship between CLD and adverse outcome was measured by the odds ratio (OR) and its 95% confidence interval (CI). Results: The overall developmental scores of the CLD infants were significantly different compared with control infants at 8 months. By 2 years of age, both groups of infants showed marked improvement in motor performance. However, differences persisted in the area of postural balance and sensory motor skills. Taking periventricular haemorrhage and periventricular leukomalacia into consideration, CLD contributed significantly to the occurrence of motor dysfunction at 8 months of age [odds ratio (OR), 7.4; 95% confidence interval (CI), 2.1 to 26.5]. The impact of CLD on motor development remained substantial, though not statistically significant (OR, 3.7; 95% CI, 0.4 to 37.9) at 2 years of age. Conclusion: CLD has a definite effect on motor development. The pathologic influence of CLD on motor development remains speculative but results of this study emphasise the need for careful neurodevelopmental follow-up of infants with CLD, whether or not these infants suffer intraventricular haemorrhage or periventricular leukomalacia.

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Introduction

New approaches in obstetric management of preterm births as well as the increased availability of tertiary neonatal care have resulted in improved survival rates of very low birthweight infants. The decline in mortality of these high-risk infants has been accompanied by an increase in the absolute number of infants with chronic lung disease (CLD). The incidence of neurological sequelae in infants with CLD was recently reported to be much higher than that of infants without CLD (40% compared with 6% respectively).[1] Impaired development in infants with CLD was linked to intraventricular haemorrhage and/or periventricular leukomalacia, rather than the lung disease per se.[2-5] Singer et al[6] showed that CLD had an unfavourable effect on motor performance at 3 years of age, and neurological risk factors and social class had a negative effect on mental development. Katz-Salamon et al[7] reported poorer eye-hand coordination and proprioception amongst infants with CLD at 5 months and 10 months of age. Reports on the pattern of neurodevelopmental and motor performance difficulties in these infants were limited.

The aim of this study was to determine whether CLD influences specific aspects of motor development in infancy. Using the Neurosensory Motor Development Assessment (NSMDA),[8] the overall motor function as well as the subgroup areas of gross and fine motor performance, posture and balance, neurological and primitive patterns of movement and motor response to sensory input were assessed and compared. This test provides a valuable profile of a child at defined ages and is a useful indicator of outcome up to 6 years of age.

Materials and Methods

The study population comprised infants born in or admitted

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to the neonatal intensive care unit of the Singapore General Hospital in Singapore from August 1997 to May 2000. Subjects eligible were infants with birthweight of less than 1500 g and diagnosed with severe CLD, defined as the need for supplemental oxygen beyond 36 weeks' post-conceptional age, in association with chest radiographic findings of persistent hazy opacification or a cyst-like pattern of density and lucency. Very low birthweight infants with chromosomal or congenital malformations were excluded. For each study infant, a control infant was sequentially selected as the closest gestational age match.

Perinatal and neonatal characteristics of CLD and control infants were collected and coded for computer analysis. Details pertaining to lung disease and pulmonary air leak were noted. Neonatal complications documented included hypotension (mean blood pressure of <35 mm Hg and <30 mm Hg for infants 1000 g to 1499 g and infants <1000 g respectively); significant patent ductus arteriosus included those requiring medical or surgical closure; periventricular-intraventricular haemorrhage, as outlined by Papile et al[9] and identified by cranial ultrasound done at least twice in the first 2 weeks of life; neonatal sepsis defined as clinical signs of sepsis supported by positive blood culture results; and necrotising enterocolitis, defined as radiologic, operative or postmortem evidence of necrotising enterocolitis. Eye examination for retinopathy of prematurity was done for all infants at the corrected age of 34 weeks and stages were recorded using the International Classification of Retinopathy of Prematurity.[10]

All infants were managed according to neonatal care regimes standard for the Neonatal Intensive Care Unit (NICU) during that period. Artificial surfactant was available for the treatment of respiratory distress syndrome. The use of surfactant was decided by the attending neonatologist. Oxygenation was monitored initially by arterial blood gas analysis, using a transcutaneous oxygen monitor. Later in the hospital stay, pulse oximetry was used for assessment. When assisted ventilation was stopped, the infants were managed with continuous positive airway pressure with positive pressure of 5 cmH_2O and fractional inspired oxygen delivered accordingly to maintain appropriate blood gas values (pH, 7.25 to 7.45; arterial oxygen tension, 50 mm Hg to 80 mm Hg; arterial carbon dioxide tension, 45 mm Hg to 55 mm Hg; base excess, ± 5 mEq/L). When oxygen requirement was persistent at weight of 1600 g, oxygen was given by nasal cannulae. Inspired oxygen was adjusted to ensure oxygen saturation of 92% to 95%. Infants unable to maintain oxygenation in room air were discharged with home oxygen. Oxygen was weaned when normal oxygen saturation was achieved in room air, at rest, and during feeds.

After discharge, the infants were reviewed at 4-month intervals in the 1st year and every 6 months in the 2nd year of life and yearly thereafter. At each visit, growth parameters were measured and a physical examination, including a detailed neurological assessment, was performed. On each of the medical follow-up visits, a physiotherapist reviewed and provided intervention strategies for infants identified to require intervention support. In addition, at 8 and 24 months, the NSMDAs[8] were carried out by a physiotherapist who was unaware of the group classification of any particular child. These ages were selected as previous studies[11-13] had shown a more reliable motor profile at 8 months than at 12 months. Informed parental consent was obtained for these assessments. Psychometric test was also performed at the corrected age of 2 years using the Bayley Scales of Infant Development [Developmental Quotient (DQ) mean, 100; standard deviation (SD) 15].[14]

Using the NSMDA,[8] the overall motor development was classified based on the performance on 6 subsections: gross motor, fine motor, postural/balance, neurological, persistence of primitive patterns of movement and the sensory motor test items. Each subsection involved a number of individual test items graded in a similar manner (Appendix 1). Items in each subsection were scored as normal, suspect/delayed or abnormal and from these individual item scores, a grade for the subsection was derived. With the 6 subsections, the best overall score was 6, while the most abnormal score was 30. Children with scores of 6 to 8 were considered to fall in the normal range for their age while motor dysfunction was graded as minimal (score of 9 to 11), mild (12 to 14), moderate (15 to 19) or severe (20 or more).

A power analysis using a 2-sided significance level of 0.05 and a power of 80% confirmed that a sample of 30 infants was required to show a 37% difference in disability rate. Perinatal and neonatal characteristics of the infants with CLD and control infants were compared using the chi-square test for categorical variables and Student's *t*-test for continuous variables. A P value of <0.05 was regarded as significant. Association between CLD and adverse outcome was measured using stepwise logistic regression analysis. Birthweight, gestation, gender, Apgar scores at 1 and 5 minutes, severe periventricular-intraventricular haemorrhage (grade 3 or 4), duration of mechanical ventilation and CLD were variables included in the logistic regression analysis. Variables staying in the final model with P <0.05 were regarded as exhibiting independent association. Estimate and 95% confidence interval (CI) for the odds ratio (OR) of the risk factors were determined.

Results

Of the total cohort, 29 were study infants and 31 were control infants. Of these, 28 study infants and 28 control

Table 1. Characteristics of Infants with Chronic Lung Disease and Control Infants Assessed at 8 and 24 Months

	CLD infants (n = 28)	Control infants (n = 28)	Significance
Birthweight (g)*	914.5 ± 253.5	1051.8 ± 195.8	0.027
Gestation (wks)*	27.1 ± 2.2	28.8 ± 1.9	0.004
Small for gestational age (%)	14.2	25	0.6
Multiple pregnancy (%)	25	39.3	0.3
Gender (M:F)	13:15	10:18	0.6
Mode of delivery			
Vaginal (%)	67.9	28.6	0.012
LSCS (%)	32.1	71.4	0.012
Apgar scores			
1 minute*	4.4 ± 1.8	5.4 ± 1.5	0.033
5 minute*	6.9 ± 1.5	8.0 ± 0.7	0.001

CLD: chronic lung disease; LSCS: lower segment caesarean section
* values indicate mean ± SD

Table 2. Neonatal Events in Infants with Chronic Lung Disease and Control Infants Assessed at 8 and 24 Months

	CLD infants (n = 28) (n) (%)	Control infants (n = 28) (n) (%)	Significance
Respiratory distress syndrome	18 (64.3)	11 (39.3)	0.06
Transient tachypnoea of newborn	2 (7.1)	5 (17.6)	0.4
Ventilated	25 (89.3)	13 (46.4)	0.003
Ventilation duration (days)*	18.3 ± 18.7	10.9 ± 14.0	0.1
Pneumothorax	4 (14.3)	2 (7.1)	0.6
Hypotension	15 (53.6)	9 (32.1)	0.2
Patent ductus arteriosus	19 (67.9)	17 (60.7)	0.8
Septicaemia	7 (25)	4 (14.3)	0.5
Necrotising enterocolitis	0	1 (3.6)	0.2
Grade 3/4 intraventricular haemorrhage	3 (10.7)	0	0.2
Periventricular leukomalacia	1 (3.6)	0	0.3
Retinopathy of prematurity			
All stages	18 (64.3)	11 (39.3)	0.1
Stage 3	10 (35.7)	2 (7.1)	0.042

CLD: chronic lung disease
* values indicate mean ± SD

infants were assessed at both 8 months and 2 years of age; hence, analysis and comparisons were made on these infants. One study infant migrated and 3 control infants defaulted assessment at 2 years of age, and were excluded from data evaluation. Study infants were significantly smaller in birthweight and gestational age as compared with control infants ($P = 0.027$ and $P = 0.004$, respectively). The perinatal details of these children, as demonstrated in Table 1, showed that study infants were significantly more compromised than control infants at birth. Mean Apgar scores among study infants were 4.4 ± 1.8 at 1 minute and 6.9 ± 1.5 at 5 minutes in contrast with 5.4 ± 1.5 and 8.0 ± 0.7 respectively in control infants. Being smaller at birth and being more compromised following delivery meant that significantly more study infants were ventilated (89.3%) in contrast with controls infants, where only 46.4% were mechanically ventilated. However, the study group did not experience more adverse neonatal events when compared with the control group (Table 2). None of the control infants had severe periventricular-intraventricular haemorrhage, as compared with 3 study infants who had grade 3 periventricular-intraventricular haemorrhage, of whom 1 also had cystic periventricular leukomalacia. The incidence of severe retinopathy of prematurity of stage 3 and above was significantly higher among study infants (35.7%) than control infants (7.1%) ($P = 0.042$).

At 8 months, 7 (25%) study infants and 5 (17.9%) control infants had growth retardation with weight at less than the 3rd percentile for age. The difference in proportion of infants with growth failure was not statistically significant. The overall developmental scores of the study group were significantly poorer when compared with control group (Table 3). Only 8 (28.6%) study infants were assessed to be developmentally normal; 11 were graded with minimal motor dysfunction, 7 mild, 1 moderate and 1 severe. Twenty-three (82.0%) control infants were assessed to be

Table 3. Grades of Motor Dysfunction of Study and Control Infants at 8 and 24 Months

	8 Months		2 Years	
	Study n (%)	Control n (%)	Study n (%)	Control n (%)
Normal	8 (28.6)	23 (82.0)*	23 (82.0)	27 (96.4)
Minimal	11 (39.2)	3 (10.8)	1 (3.6)	–
Mild	7 (25)	1 (3.6)	1 (3.6)	–
Moderate	1 (3.6)	1 (3.6)	2 (7.2)	–
Severe	1 (3.6)	–	1 (3.6)	1 (3.6)

* indicates $P = 0.001$

functioning within the expected range for age of 8 months, with only 3 assessed with minimal motor dysfunction, 1 mild and 1 moderate (OR, 10.9; 95% CI, 3.3 to 36.6). Study infants performed poorer on all subscales of the neurosensory and motor development skills (Table 4). Logistic regression performed with motor dysfunction as a dependent variable showed that the association between CLD and motor dysfunction remained statistically significant when adjustment for presence of periventricular-intraventricular haemorrhage was made (OR, 7.4; 95% CI, 2.1 to 26.5).

By 2 years of age, 1 study infant and 2 control infants persisted to have growth failure, with weight remaining at less than the 3rd percentile for age. Three study infants required glasses for correction of astigmatism. None of the infants had significant visual impairment. Both the study and control infants showed marked improvement in their motor performance. Eighty-two per cent of the study group, as compared with 96.4% of the control group, was assessed to be normal (OR, 5.9; 95% CI, 0.6 to 53.9) (Table 3). This difference was not statistically significant. When adjustment for the presence of periventricular-intraventricular haemorrhage was made, the association between CLD and motor dysfunction remained, though it was not statistically significant (OR, 3.7; 95% CI, 0.4 to 37.9). The most marked difference between study and control infants at 2 years was documented in the area of fine motor skills (35% compared with 57% normal, Table 4). The change in performance category from 8 to 24 months was most striking among study infants (Table 5). Of the infants in the study group, the majority of those with minimal to mild dysfunction at 8 months of age were functioning normally by 2 years of age. Only 1 infant in each group developed progressive motor dysfunction (study group, from mild to moderate dysfunction; control group, from moderate to severe dysfunction) from 8 to 24 months.

Discussion

The influence of CLD on neurodevelopment in very low birthweight infants has been reviewed with contradictory

Table 4. Grades of Dysfunction on the Subscales of the Neurosensory and Motor Development Assessment (NSMDA) for Study and Control Infants at 8 months and 24 months

	8 Months		24 Months	
	Study n (%)	Control n (%)	Study n (%)	Control n (%)
Gross motor				
Normal	7 (25)	15 (54)	24 (86)	27 (96)
Minimal	11 (39)	9 (32)	0	0
Mild	9 (32)	3 (11)	1 (4)	0
Moderate	0	0	2 (7)	0
Severe	1 (4)	1 (4)	1 (4)	1 (4)
Fine motor				
Normal	8 (28)	18 (64)	10 (35)	16 (57)
Minimal	14 (50)	8 (29)	14 (50)	11 (39)
Mild	4 (14)	2 (7)	1 (4)	0
Moderate	1 (4)	0	2 (7)	1 (4)
Severe	1 (4)	0	1 (4)	0
Neurological				
Normal	22 (78)	27 (96)	24 (85)	27 (96)
Minimal	4 (14)	1 (4)	1 (4)	1 (4)
Mild	1 (4)	0	2 (7)	0
Moderate	0	0	1 (4)	0
Severe	1 (4)	0	0	0
Primitive movement patterns				
Normal	23 (82)	27 (96)	25 (89)	28 (100)
Minimal	4 (14)	1 (4)	2 (7)	0
Mild	0	0	0	0
Moderate	0	0	1 (4)	0
Severe	1 (4)	0	0	0
Postural				
Normal	10 (36)	20 (71)	20 (71)	25 (89)
Minimal	10 (36)	5 (18)	5 (18)	2 (7)
Mild	7 (24)	3 (11)	1 (4)	0
Moderate	0	0	2 (7)	0
Severe	1 (4)	0	0	1 (4)
Sensory motor				
Normal	16 (57)	25 (89)	20 (71)	25 (89)
Minimal	11 (39)	3 (11)	7 (25)	2 (7)
Mild	0	0	0	1 (4)
Moderate	0	0	1 (4)	0
Severe	1 (4)	0	0	0

results. Bryne et al[15] evaluated motor development at term of very low birthweight infants using the neurological assessment of Dubowitz and Dubowitz[16] and found that infants with BPD are no different from infants without BPD and suggested that motor development was not affected by environmental factors. Luchi et al[17] reviewed the influence of severity of pulmonary disease on neuro-developmental outcome at 3 years and concluded that lung disease is not a predictor of neurodevelopment by the age of 3 years. A contrasting report[6] showed that CLD per se had an unfavourable effect on motor performance at 3 years of age. Burns et al[18] evaluated motor performance in a

Table 5. Categories of Motor Performance at 8 Months and 24 Months in Study and Control Infants

8 Months	24 Months					
	Total	Normal	Minimal	Mild	Moderate	Severe
Study infants						
Normal	8	8				
Minimal	11	11				
Mild	7	4	1	1	1	
Moderate	1				1	
Severe	1					1
	28	23	1	1	2	1
Control infants						
Normal	23	23				
Minimal	3	3				
Mild	1	1				
Moderate	1					1
Severe						
	28	27				1

similar group of very low birthweight infants with and without bronchopulmonary dysplasia (BPD) and found BPD infants to be at risk of gross motor dysfunction at the age of 8 months. Katz-Salamon et al[7] evaluated the effect of CLD on development in the first year of postnatal life using the movement assessment of infant's scale and Griffiths' developmental test.[19] Results of these reports concur with our findings of significantly poorer motor performance among CLD infants at 8 months of age.

28.6% of our study group and 82% of controls were developmentally normal at the corrected age of 8 months. In comparison, Burns et al[18] documented normal development in 42% of their study group and 62% of control population at the corrected age of 8 months. The difference in the proportion of our study children with motor dysfunction as compared with that of Burns's could be explained by the variation in study populations. Our study group comprised infants who were oxygen-dependent at the corrected age of 36 weeks. Study infants in Burns's review remained on supplemental oxygen at 28 days of life. Better motor performance documented in our control group, compared with that in Burns's study, could not be explained by any neurological events as none of the infants in both control groups had any severe periventricular-intraventricular haemorrhage or periventricular leukomalacia identifiable on neonatal ultrasounds. However, it was evident that a greater proportion of their control infants had respiratory distress syndrome (60.4% compared with 39.3% in our control cohort) and similarly, a higher percentage of their control infants needed mechanical support as compared to our control group (73.6% versus 46.4%). This difference suggested that the control infants in Burns's study were probably sicker and hence had worse adverse neonatal experiences than our control infants.

Katz-Salamon et al[7] reported deviant volitional movement at 10 months of corrected age in infants with CLD as compared to infants without CLD. In their review, CLD infants were infants who required ventilatory support in the first week of life, continued to exhibit signs of chronic respiratory disease and remained on supplemental oxygen at 28 days of postnatal life, supported by chest radiograph showing persistent strands of density in both lung fields. The disparity in motor performance between infants with CLD and control infants reported by Katz-Salamon et al was comparable with our findings. Our study group showed weakness in the area of gross/fine motor, postural balance and sensory motor skills at 8 months of corrected age with fine motor, postural balance and sensory motor dysfunction persisting at the corrected age of 2 years. Bowen et al[20] also reported impairment in hand and eye coordination in neurologically normal extremely preterm infants at the age of 3 years. The impairment in hand and eye coordination may be related to damage in both corticospinal motor pathways and visual pathways. Cytokine-mediated inflammatory processes leading to CLD and subtle recurrent hypoxia in infants with established CLD contributes to damage in these vulnerable areas of the brain. Postural balance was achieved through the integration of proprioceptive, visual, vestibular and somatosensory inputs together with an efficient process of muscle activation and stabilisation. The link between muscle tone, motor performance and sensory motor deficits explained the coexistence of poorer postural balance in these study infants.

Marked improvement in overall motor performance among study infants from age 8 months to 24 months suggests that these vulnerable and medically fragile preterm infants have the potential for developmental catch-up. Hence, an adequate period of developmental catch-up must be allowed for these infants before making outcome conclusions and prognostication. Performance in the subscales of the NSMDA between study and control infants at both 8 months and 24 months could not be compared with the restricted population enrolled in our study. Future studies should include more infants so as to permit comparisons in the various components of motor development.

It is encouraging that the majority of our infants, both study and control infants, were free of major motor abnormalities at 2 years of age. Follow-up into school age is necessary to establish if subtle coordination disorders and schooling difficulties persist till then, particularly in infants with CLD.

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Appendix 1. Neurosensory Motor Development Assessment Test Items Conducted at the Corrected Ages of 8 and 24 Months.

At 8 months

Gross motor: Posture when prone and supine, head control, rolling, creeping, crawling, sitting, taking weight when held standing and weight transfer or cruising along furniture.

Fine motor: Ability to grasp, type of grasp used and transfer and gross co-ordination.

Postural/balance: Placing and supporting, head righting to gravity and protective/parachute reactions.

Neurological testing: Muscle tone during quiet activity, deep tendon reflexes and clonus, with evidence of tremor, persisting primitive patterns of movement such as Moro, galant, extensor thrust, tonic labyrinthine and neck reflexes.

Sensory motor: Response to touch, ability to adapt position, ability to visually follow and locate moving objects and post-rotatory vestibular response.

At 24 months

Gross motor: Ability to climb onto and rotate to sit on an adult chair, to kneel upright and stand, the quality of gait, stair-climbing and ability to stand on one leg.

Fine motor: Quality of grasp and performance on items of coordination and pronation/supination of forearm while manipulating a screw top.

Postural/balance: Righting reactions to gravity, the child's balance in sitting and standing.

Neurological testing: Muscle tone during quiet activity, deep tendon reflexes and clonus, with evidence of tremor, persisting primitive patterns of movement such as Moro, galant, extensor thrust, tonic labyrinthine and neck reflexes.

Sensory motor: Child's use of tactile sensitivity, joint and position sense, ability to visually follow a small bead and rapidly moving objects and post-rotatory vestibular response.

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