Head Lag in Infancy: What Is It Telling Us?

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MeSH TERMS

- head
- infant, premature
- neurobehavioral manifestations
- patient outcomes assessment
- posture

OBJECTIVE. To investigate changes in head lag across postmenstrual age and define associations between head lag and (1) perinatal exposures and (2) neurodevelopment.

METHOD. Sixty-four infants born ≤ 30 wk gestation had head lag assessed before and at term-equivalent age. Neurobehavior was assessed at term age. At 2 yr, neurodevelopmental testing was conducted.

RESULTS. Head lag decreased with advancing postmenstrual age, but 58% ($n = 37$) of infants continued to demonstrate head lag at term. Head lag was associated with longer stay in the neonatal intensive care unit ($p = .009$), inotrope use ($p = .04$), sepsis ($p = .02$), longer endotracheal intubation ($p = .01$), and cerebral injury ($p = .006$). Head lag was related to alterations in early neurobehavior ($p < .03$), but no associations with neurodevelopment were found at 2 yr.

CONCLUSION. Head lag was related to medical factors and early neurobehavior, but it may not be a good predictor of outcome when used in isolation.

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The mortality rate for preterm infants has substantially decreased over the past few decades as a result of advances in medical care. However, preterm infants remain at high risk for neurological and developmental consequences that persist into childhood. Although cognitive, language, and behavioral problems may result, poor motor development is also common after preterm birth (Doyle, 1995; Lorenz, Wooliever, Jetton, & Paneth, 1998; Salt & Redshaw, 2006).

Postural control, the underlying foundation for motor development (Deborab, 2001), is defined as an infant's ability to maintain the center of his or her body mass or body part over a stable or moving base of support (Massion, 1998; Shumway-Cook & Woollacott, 1995). Postural stability relies on adequate control of the neck and trunk, and it develops rapidly during the first year of life. According to several studies, postural control is impeded by medical conditions such as low birthweight, low gestational age, and cerebral injury and by environmental factors, including prolonged use of mechanical ventilation (Jeng, Yau, Liao, Chen, & Chen, 2000; Samsom, de Groot, Bezemer, Lafieber, & Fetter, 2002; Samsom, Sie, & de Groot, 2002; van Haastert, de Vries, Helders, & Jongmans, 2006). Preterm infants often demonstrate difficulty achieving postural control (Samsom, de Groot, et al., 2002; Samsom, Sie, & de Groot, 2002). This problem can be seen when an infant is being pulled to sit, which involves pulling the infant by the upper extremities from a supine to a sitting position and observing the position of the head and neck.

During pull-to-sit, the normal response is for an infant to right the head and maintain it in line with the shoulders. Head lag is demonstrated when the head is not righted but lags posteriorly behind the trunk as a result of poor head and neck control. The amount of head lag is gauged by how far the head falls behind the shoulders when the infant is pulled into the sitting position (Allen & Capute,

1990; Dubowitz, Dubowitz, & Mercuri, 1999; Larsen, 2006). The ability to right the head is essential to achieving postural control. Preterm infants have a heightened risk for motor problems, and early postural control, related to head lag in pull-to-sit, can be an important predictor of developmental outcome (Samsom & de Groot, 2000; Viholainen et al., 2006).

Persistent head lag beyond age 4 mo has been linked to poor outcomes (Flanagan, Landa, Bhat, & Bauman, 2012), but few studies have investigated head lag during the neonatal period (Barbosa, Campbell, Smith, & Berbaum, 2005; Karmel et al., 2010). Although head lag has been reported to be common in preterm infants up to term-equivalent age (Allen & Capute, 1990), most full-term infants and preterm infants at term-equivalent age can maintain the head in line with the body during pull-to-sit (Dubowitz et al., 1999), making head lag in the neonatal period a marker of poor neurobehavior. However, only one study could be identified that related head lag during the neonatal period to an increased incidence of cerebral palsy (Barbosa et al., 2005). Additionally, another study identified relationships between head lag at term and (1) brain structural alterations and (2) early neurodevelopmental outcome at 12 wk corrected age but not long-term outcomes (Bentzley et al., 2015). Because head lag is commonly assessed in high-risk infants, gaining a better understanding of its early progression in preterm infants can fill important gaps in the literature and aid our understanding of early alterations in function. Better identification of early alterations in function will better inform the appropriate time to begin early intervention services.

The three aims of this study were to (1) investigate the progression of head lag in preterm infants during neonatal intensive care unit (NICU) hospitalization, (2) identify the medical factors that may contribute to head lag, and (3) explore the relationships between head lag and neurodevelopmental outcome. Understanding these factors can inform occupational therapy practitioners on current practice and help improve the clinical evaluation of preterm infants.

Method

This prospective longitudinal study was approved by the Human Research Protection Office at Washington University in St. Louis and took place in the 75-bed Level III-IV NICU at St. Louis Children's Hospital. This study was contained within a larger parent study investigating factors that affect brain development of preterm infants.

Participants were born ≤ 30 wk estimated gestational age (EGA) and were enrolled in the study within the first

72 hr of life. Infants were excluded if they had a known or suspected congenital anomaly or if they were expected to expire within 24 hr of life. Consent was obtained from the infants' parents. As part of the larger study, serial neurobehavioral testing was undertaken during hospitalization at 30 wk postmenstrual age (PMA), 34 wk PMA, and term-equivalent age (37–41 wk PMA). Medical and environmental factors present during the NICU hospitalization were collected from the medical chart. Infants underwent routine cranial ultrasound (CUS) and MRI within the first 2 wk of life and at term-equivalent age, respectively. After discharge, infants underwent neurodevelopmental testing at age 2 yr.

Independent Variables: Medical and Environmental Factors

The following medical and environmental factors were collected from participants' medical records: gender, race (White or non-White), EGA at birth (wk), birthweight (g), Clinical Risk Index for Babies (International Neonatal Network, 1993) score, days of endotracheal intubation, days of continuous positive airway pressure (CPAP), total oxygen hours (including intubation, CPAP, and oxygen administered by nasal cannula), oxygen dependency at 36 wk PMA, days of total parenteral nutrition, exposure to prenatal and postnatal steroids, confirmed sepsis, use of inotropes, length of stay in the NICU, PMA at discharge, presence of patent ductus arteriosus (requiring surgical ligation), presence of necrotizing enterocolitis (all stages), retinopathy of prematurity (all grades), multiple birth, delivery type (vaginal or Caesarean), and cerebral injury (detected by CUS and MRI; defined as the presence of either Grade III or IV intraventricular hemorrhage, cystic periventricular leukomalacia, or cerebellar hemorrhage). In addition, the following social factors pertaining to the mothers were documented: number of prenatal visits, age, marital status, prenatal exposure to illicit drug use (by toxicology screen), insurance type (public or private), and education (college education or less).

Head Lag

Head lag was assessed at 30 and 34 wk PMA using the Premie-Neuro (Daily & Ellison, 2005) and at term-equivalent age using the Dubowitz Neurological Examination (Dubowitz et al., 1999). Both assessments can be used to measure neurobehavioral functioning in the preterm infant, and both assess head lag. The head lag item on each assessment takes less than 1 min to administer and involves the administrator pulling the infant by his or her hands from a supine to a sitting position.

The Premie-Neuro and the Dubowitz Neurological Examination score head lag in the same manner, using pictures depicting the amount of head lag during the exam (Figure 1). Scoring for both is as follows: 0 = *head drops and stays back*, 1 = *tries to lift head but drops it back*, 2 = *able to lift head slightly*, 3 = *lifts head in line with body*, and 4 = *head in front of body*. For this study, head lag scores were dichotomized into *significant head lag* (0 or 1) and *no head lag* (2, 3, or 4). These dichotomized scores were used to investigate associations between head lag and medical conditions, environmental exposures, and neurobehavioral outcome at term-equivalent age and developmental outcome at age 2 yr. Secondary analyses were also conducted that dichotomized scores into *significant head lag* (0, 1, or 2) and *no head lag* (3 or 4).

Outcome Variables

Outcome measures used were cerebral injury at term-equivalent age (previously described), early neurobehavior at term-equivalent age, and neurodevelopmental outcome at age 2 yr.

Early Neurobehavioral Outcome. At term-equivalent age, neurobehavior was assessed using the NICU Network Neurobehavioral Scale (NNNS; Lester, Andreozzi-Fontaine, Tronick, & Bigsby, 2014). The NNNS is a 115-item test with 13 summary scores: habituation, orientation, hypertonicity, hypotonicity, arousal, lethargy, asymmetry, sub-optimal reflexes, excitability, tolerance of handling, stress, quality of movement, and self-regulation. Evaluations were performed by a single trained and certified occupational therapist (author Pineda).

Neurodevelopmental Outcome. At age 2 yr, infants returned for neurodevelopmental testing. Parents completed the Modified Checklist for Autism in Toddlers (M-CHAT; Robins, Fein, Barton, & Green, 2001) and the Infant-Toddler Social and Emotional Assessment (ITSEA; Carter & Briggs-Gowan, 2006) to determine autism risk and social-emotional development, respectively. Neurodevelopmental testing was also conducted using the Bayley Scales of Infant and Toddler Development, 3rd ed. (Bayley-III; Bayley, 2006).

The M-CHAT, a 23-item screening tool for autism risk in children ages 16–30 mo, is completed by parent report. Parents respond “yes” or “no” to questions representative of characteristics specific to autism. A failed M-CHAT screening indicates the need for in-depth evaluation for autism (Robins & Dumont-Mathieu, 2006). In a recent study, the M-CHAT was sensitive to the presence of autism at 87% and to the absence of autism at 99% (Robins & Dumont-Mathieu, 2006). For this study, M-CHAT pass or fail score was used to operationalize autism risk and was investigated for associations with head lag.

The ITSEA is a 166-item assessment that identifies social-emotional and behavioral problems and delays in the attainment of competencies in children ages 18–36 mo (Carter, Briggs-Gowan, Jones, & Little, 2003). The survey takes approximately 25–30 min to complete. Parents are asked various questions about the types of behaviors their child engages in. The ITSEA has good test-retest reliability (.61–.91) and good interrater reliability (.58–.79; Carter et al., 2003). The ITSEA consists of four domains and 17 subscales: (1) Externalizing (Activity/Impulsivity, Aggression/Defiance, and Peer Aggression), (2) Internalizing (Depression/Withdrawal, General Anxiety, Separation Distress, Inhibition to Novelty), (3) Dysregulation (Sleep, Negative Emotionality, Eating, Sensory Sensitivity), and (4) Competence (Compliance, Attention, Imitation/Play, Mastery Motivation, Empathy, and Pro-social Peer Relations). Additionally, three indexes of the ITSEA include maladaptive behaviors, atypical behaviors, and social relatedness. Relationships among domain scores, index scores, and head lag were explored.

The Bayley-III is a comprehensive, norm-referenced neurodevelopmental assessment for children ages 1–42 mo. Administration requires approximately 45–60 min. The Bayley-III is considered the gold standard in developmental evaluation. Its composite scores for Language, Cognitive, and Motor subscales (as well as whether any composite score was <70, indicating developmental delay) were investigated for associations with head lag (Bayley, 2006).

Figure 1. Head lag scoring on the Dubowitz Neurological Examination.

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Statistical Analysis

All data management and statistical analyses were conducted using IBM SPSS Statistics (Version 20; IBM Corporation, Armonk, NY). Head lag at term-equivalent age was investigated for associations with medical and environmental factors and with neurodevelopmental outcome at term-equivalent age and 2 yr using independent samples *t* tests, χ^2 analysis, and Wilcoxon signed-rank test. All analyses were conducted with an $\alpha < .05$.

Results

This exploratory study included a subset of infants from the parent study who underwent neurobehavioral assessment during NICU hospitalization and who returned for neurodevelopmental testing at age 2 yr ($N = 64$). Table 1 details participant characteristics.

Head Lag Progression

Thirty-eight percent ($n = 24$) of infants at 30 wk PMA and 33% ($n = 21$) of infants at 34 wk were too unstable to assess head lag. Infants who were too unstable to test at the earlier time points had lower gestational age at birth and longer periods of intubation than those who could be assessed at 30 and 34 wk. Among infants who were assessed for head lag, 90% ($n = 36$) demonstrated head lag at 30 wk PMA, 60.5% ($n = 26$) at 34 wk PMA, and 57.8% ($n = 37$) at term-equivalent age (Figure 2).

Head Lag and Medical and Environmental Factors

Significant associations were noted between head lag at term-equivalent age and medical and environmental factors (Table 2).

Head Lag and Outcome Measures

Head lag at term-equivalent age was related to cerebral injury ($p = .006$) and alterations in neurobehavior ($p \leq .003$; see Table 2). No associations were found between head lag and autism risk and social-emotional outcome at age 2 yr; head lag and Bayley-III Language, Cognitive, and Motor subscale scores (see Table 2); or head lag and developmental delay.

Discussion

The incidence of head lag changed from 30 wk PMA to term-equivalent age: Most preterm infants demonstrated head lag at 30 wk PMA, and just more than half of the

Table 1. Sample Characteristics ($N = 64$)

Characteristic	<i>n</i> (%), <i>M</i> (<i>SD</i>), or Median (IQ range)
Medical factors	
Female	32 (50)
White	39 (61)
Gestational age at birth, wk	26.5 (1.9)
Birth weight, g	929.0 (250.5)
Clinical Risk Index for Babies score	3.7 (3.3)
Days on ventilator	3 (1–24)
Days on continuous positive airway pressure	3 (1–10)
Oxygen hours	1,572 (912–2,286)
Oxygen requirement at 36 wk PMA	32 (50)
Days on total parenteral nutrition	18 (11–34)
Prenatal steroids	59 (92)
Postnatal steroids	21 (33)
Confirmed sepsis	19 (30)
Inotrope use	21 (33)
Length of stay, days	93.5 (28.9)
Postmenstrual age at discharge, wk	40 (3.5)
Patent ductus arteriosus	36 (56)
Necrotizing enterocolitis	6 (9)
Retinopathy of prematurity	10 (16)
Multiple birth	27 (42)
Vaginal delivery	17 (27)
Cerebral injury	13 (20)
Social factor (mother)	
Prenatal visits	5.4 (2.7)
Maternal age	28.9 (7.1)
Married	28 (44)
Illicit drug use	2 (3)
Private insurance	28 (44)
College education	31 (48)

Note. IQ = interquartile; *M* = mean; PMA = postmenstrual age; *SD* = standard deviation.

sample continued to demonstrate head lag by term-equivalent age. The key findings of this study are as follows:

- More head lag was observed in infants with longer length of NICU stay, more days of endotracheal intubation, and sepsis.
- The presence of head lag was related to cerebral injury and neurobehavioral alterations at term-equivalent age.
- In this cohort of preterm infants, with many comorbidities, no associations between head lag and neurodevelopmental outcome at age 2 yr were found.

Our findings demonstrate changes in the incidence of head lag before and at term-equivalent age. Therefore, head lag may not be a stable marker throughout the perinatal period. The final measure of head lag in this cohort occurred at term-equivalent age; however, defining additional changes in the incidence of head lag beyond term-equivalent age is warranted. Head lag can provide

Figure 2. Percentage of infants with head lag (HL) and without HL (WHL) at 30 wk ($n = 40$) and 34 wk ($n = 43$) postmenstrual age and at term-equivalent age (TEA; $n = 64$).

clinical information about tone of the preterm infant. Underdeveloped muscle tone in the preterm infant may be responsible for the delay in head righting. However, healthy preterm infants with no identified neurological issues have poor motor development compared with full-term infants (Lee, Kak, Oh, & Roh, 2011; Pineda et al., 2013), and persistent head lag in preterm infants at term-equivalent age has been documented (Allen & Capute, 1990).

There are conflicting reports of when head lag should be present during infancy. Some studies have stated that after full-term birth, infants can keep their head in line

with the body during pull-to-sit (Dubowitz et al., 1999), whereas others have maintained that head lag should no longer be present by age 12–16 wk (von Hahn, 2006). In contrast to previous investigations of infant head lag at 4–6 mo, when head control should be stable and trunk control is developing, this study is the first to investigate head lag in preterm infants before and at term-equivalent age, when head-righting responses are developing.

The relationships of head lag to immaturity and other medical factors support the hypothesis that head lag may be an important marker of current developmental status and wellness. Both NICU length of stay and PMA at

Table 2. Relationship Between Head Lag and Medical and Environmental Factors and Neurobehavioral and Developmental Outcomes

Variable	With Head Lag, n (%), M (SD), or Median (IQ Range; $n = 37$)	Without Head Lag, n (%), M (SD), or Median (IQ Range; $n = 27$)	p
Medical or environmental factor			
Days on ventilator	9 (1–32)	1 (1–4)	.008
Total O ₂ , hr	1,824 (1,128–2,400)	1,296 (648–1,920)	.03
Sepsis	15 (83)	3 (17)	.02
Inotrope use	16 (76)	5 (24)	.04
Length of NICU stay, days	100.5 (36.6)	84.5 (18.3)	.009
Postmenstrual age at discharge, wk	40.8 (4.6)	39.1 (2.5)	.03
Cerebral injury	12 (92)	1 (8)	.006
Neurobehavioral outcome (NNNS score)			
Quality of Movement	3.4 (0.8)	3.8 (0.7)	.03
Self-regulation	4.1 (0.7)	4.8 (0.8)	<.001
Bad reflexes	7.6 (2.1)	5.6 (1.7)	.001
Stress	0.4 (0.1)	0.3 (0.1)	<.001
Hypotonia	1.0 (0.8)	0.4 (0.5)	.002
Asymmetry	2.8 (2.2)	0.9 (1.1)	<.001
Developmental outcome (Bayley–III composite score)			
Motor	82.0 (11.9)	86.4 (10.8)	.14
Cognitive	86.2 (10.2)	86.1 (9.7)	.97
Language	88.8 (12.9)	88.7 (11.0)	.98

Note. Bayley–III = Bayley Scales of Infant and Toddler Development (3rd ed.); IQ = interquartile; M = mean; NICU = neonatal intensive care unit; NNNS = NICU Network Neurobehavioral Scale; SD = standard deviation. p values were determined using logistic regression models.

discharge are proxies for illness, because preterm infants who are hospitalized longer have more complicated medical courses. Longer periods of endotracheal intubation also occur when infants have greater medical fragility, and it is understood to be a marker for poor outcome (Engelhardt et al., 2015; Tsai et al., 2014). Medical complications, including long periods of intubation and sepsis, can also result in fewer opportunities for normal sensory and motor exposures, which can affect tonal responses and result in fewer head-righting responses. This could decrease strength, which affects the infant's ability to hold the head in line with the body in pull-to-sit. Our findings, demonstrating relationships between medical complications and head lag, are consistent with other reports (Jeng et al., 2000; Samsom, de Groot, et al., 2002; Samsom, Sie, & de Groot, 2002; van Haastert et al., 2006). Although medical factors can decrease strength and tone, affecting the development of head-righting responses, head lag can also be a marker for altered tonal responses related to brain injury.

The assessment of head lag has long been part of neurological evaluations (Dubowitz et al., 1999; Forslund & Bjerre, 1983). Inability to hold the head in line with the body in pull-to-sit can signal that abnormal tonal responses are present, which could be related to neurological integrity. Our findings support that head lag in the neonatal period is a good marker for cerebral injury, because 12 of 13 infants in the cohort with cerebral injury demonstrated head lag. Head lag also appears to be a good marker of alterations in concurrent neurobehavior, with infants who demonstrated head lag more likely to exhibit hypotonia, poor quality of movements, asymmetry, poor self-regulation, and poor reflex development. This finding coincides with previous findings of head lag being associated with lower motor scores at age 12 wk but not at later time points (Bentzley et al., 2015). Findings from this study differ from those of a previous study that found an association between head lag at age 6 mo and risk for autism (Flanagan et al., 2012) and from other literature showing a relationship between early head-righting responses and poor motor outcome and autism (Barbosa et al., 2005; Karmel et al., 2010).

Although head lag at term-equivalent age may not be a good marker for long-term outcome, there are some alternate explanations for these findings. One explanation could be that early behavioral responses, such as head lag, are affected by concurrent medical factors and that positive strides toward good long-term developmental outcomes can be made by resolution of these medical factors and by therapeutic exposures, which cannot be fully appreciated in a study such as this. This approach to understanding our findings does not support that head lag is a poor predictor

of adverse outcome, but rather that outcome can and should be affected by positive sensory and motor exposures that can alter the pathway to poor or optimal outcomes.

Another explanation could be that the assessment of one isolated response is not adequate to predict neurodevelopmental outcome. Several studies have concluded that a combination of neonatal responses, such as postural reactions, reflex responses, and tone, may have the potential to most accurately predict altered brain development (Futagi, Suzuki, & Goto, 1999; Futagi, Tagawa, & Otani, 1992; Zafeiriou, Tsikoulas, & Kremenopoulos, 1995). Subsequently, investigating whether one isolated neonatal response can predict outcome may not be sensitive enough to discriminate typical from atypical outcome, especially in infants with substantial medical factors and comorbidities. Future research is needed to identify response profiles that have specific associations with cerebral injury and developmental outcome.

Study Limitations

Limitations of this study include the small sample size and large number of comorbidities in the sample. Head lag may be a marker of other types of impairment that could not be clearly identified with the measures used in this study. We were unable to assess head lag in many infants at 30 and 34 wk PMA for medical reasons, which makes it difficult to draw conclusions at these time points. An additional limitation is that head lag was assessed only in the neonatal period and that there was a gap in time between evaluation at NICU discharge and developmental testing at age 2 yr. Almost half of the cohort continued to have head lag at NICU discharge; therefore, it remains unclear whether observing head lag in the months after discharge could have provided additional insights. Future research is warranted to investigate head lag beyond term-equivalent age and associated risks in the preterm infant population.

This study is limited by the use of parent-report measures for assessing autism risk and social-emotional health rather than a formal autism diagnosis or observations of behavior in early childhood. There have been controversial findings about the sensitivity of the M-CHAT in the preterm population (Moore, Johnson, Hennessy, & Marlow, 2012). In addition, this study did not use the M-CHAT follow-up interview (Robins & Dumont-Mathieu, 2006). Instead, we used measures that have been shown to be predictive of an autism diagnosis at age 2 yr, but it would be beneficial to compare these findings with a formal diagnosis of autism. This study also was not able to capture the potential intervening

factors between term-equivalent age and 2 yr, such as home environment, therapeutic interventions, and other sensory and motor exposures.

Implications for Occupational Therapy Practice

The results of this study have the following implications for occupational therapy practice:

- The presence of head lag decreases with advancing postmenstrual age.
- Head lag was related to medical complications.
- Head lag in the neonatal period was related to cerebral injury.
- Head lag was related to early neurobehavior but not to neurodevelopmental outcome at 2 yr.
- Head lag can provide the clinician with clinical information that can support the use of early therapeutic interventions aimed at optimizing outcomes.

Conclusion

Head lag appears to be a marker of increased medical fragility during the neonatal period and is related to concurrent neurobehavioral alterations and cerebral injury. Given that occupational therapists routinely assess postural reflexes in the neonatal period, the findings of this study highlight the importance of evaluating a broad range of reflexes rather than relying on any one in isolation. Assessing head lag in the neonatal period, without accounting for motor and sensory exposures after NICU discharge, could not explain developmental impairment at age 2 yr in this cohort. Future research is warranted to not only ascertain early markers of developmental outcome in preterm infants, but also to identify the importance and evolution of postural control in this high-risk population. ▲

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