Head Lag in Infants at Risk for Autism: A Preliminary Study

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KEY WORDS
- autistic disorder
- infants
- motor skills disorders
- postural balance
- risk factor

OBJECTIVE. Poor postural control during pull-to-sit is a predictor of developmental disruption in cerebral palsy and preterm populations but has not been examined in infants at risk for autism. We examined the association between head lag during pull-to-sit at age 6 mo and autism risk status.

METHOD. High-risk participants were siblings of children with autism. We studied one sample of 40 high-risk infants prospectively from 6–36 mo and obtained diagnostic classifications of autism or no autism. We conducted a subsequent between-group comparison with a new sample of 20 high-risk and 21 low-risk infants.

RESULTS. Head lag was significantly associated with autism spectrum disorder at 36 mo ($p = .020$) and was more frequently observed in high-risk than in low-risk infants ($p = .018$).

CONCLUSION. Head lag with other alterations in early development may be associated with autism risk and may serve as an early indicator of neurodevelopmental disruption. Results have clinical implications for occupational therapists in early intervention practice.

Flanagan, J. E., Landa, R., Bhat, A., & Bauman, M. (2012). Head lag in infants at risk for autism: A preliminary study. *American Journal of Occupational Therapy, 66,* 577–585. http://dx.doi.org/10.5014/ajot.2012.004192

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Autism spectrum disorders (ASD) represent a set of neurodevelopmental disorders defined by qualitatively abnormal social and communication development along with repetitive and stereotyped behaviors and interests (American Psychiatric Association [APA], 2000). Access to early intervention is facilitated by detecting ASD as early in life as possible (Landa, Holman, O'Neil, & Stuart, 2011). Research aimed at improving early detection of autism has largely focused on measurement of social and communication development. The earliest indicators of disruption in the development of infants later diagnosed with ASD, however, may not fall clearly within these conventionally recognized core deficit areas (Bryson et al., 2007; Landa & Garrett-Mayer, 2006). The present study focused on motor delays at age 6 mo in a group of infants at high genetic risk for ASD: infant siblings of children with autism (Ozonoff et al., 2011).

Motor abnormalities distinguishing children with ASD from children with typical development include fine and gross motor impairments and balance impairments (Jansiewicz et al., 2006; Noterdaeme, Mildenberger, Minow, & Amorosa, 2002), reduced environmental exploration (reduced time in active movement exploration and fewer number of explored objects; Pierce & Courchesne, 2001), poor postural stability (Minshew, Sung, Jones, & Furman, 2004; Molloy, Dietrich, & Bhattacharya, 2003), praxis deficits (Dowell, Mahone, & Mostofsky, 2009; Dziuk et al., 2007), imitation deficits (Hobson & Lee, 1999; Rogers, Hepburn, Stackhouse, & Wehner, 2003), unusual gait (Rinehart et al., 2006; Vilensky, Damasio, & Maurer, 1981), poor planning and anticipatory control (Martineau, Schmitz, Assaiante, Blanc, & Barthélémy, 2004), posturing (Rinehart et al., 2006), and irregular locomotor pattern and

poor goal-oriented movement planning (Vernazza-Martin et al., 2005). A recent meta-analysis of motor behavior in 41 studies showed a large effect indicating deficits in motor function in ASD when compared with non-ASD groups at ages ranging from toddlers to adults (Fournier, Hass, Naik, Lodha, & Cauraugh, 2010). Interestingly, few studies on the earliest clinical markers for ASD have assessed motor system variables, including postural control.

Several retrospective studies using analysis of home videos of children with ASD made during infancy have documented disruption in motor development. Hypotonia was reported as one of five early characteristics that significantly differentiated 12 infants with autism from 12 children with typical development (Adrien et al., 1993). Using a detailed coding system, Teitelbaum, Teitelbaum, Nye, Fryman, and Maurer (1998) found atypical movement patterns and delayed motor milestones in home videos of 17 infant participants with autism aged 4–6 mo.

Ozonoff and colleagues (2008), however, reported that children aged 0–24 mo with nonregressive autism exhibited motor delays comparable to those observed in children with developmental delay without ASD. Motor maturity in those groups was lower than in children with typical development or with regressive autism. In a study by Phagava et al. (2008), abnormal general movements (e.g., quality and complexity of movement patterns; Einspieler, Prechtl, Bos, Ferrari, & Cioni, 2004) in the first 4 mo of life distinguished infants with autism from infants with typical development. Esposito, Venuti, Maestro, and Muratori (2009) reported that infants later diagnosed with ASD exhibited less mature motor development—specifically, significantly less static and less dynamic motor symmetry—than infants with typical development and infants with developmental delay. Taken together, several retrospective studies have reported motor delays equal to or greater than developmentally delayed infants.

Prospective studies involving siblings of children with autism have not identified overt impairments during mid-infancy in most infants later diagnosed with ASD compared with infants at low risk for ASD (i.e., who did not have a sibling with ASD). Yet subtle signs of disrupted development related to motor development in siblings of children with autism have been reported. Using a parent-report temperament questionnaire, Zwaigenbaum et al. (2005) identified reduced activity level in some 6-mo-old siblings of children with autism who were themselves later diagnosed with autism at 24 mo. They also identified several behaviors that, at age 12 mo, predicted a diagnosis of autism at 24 mo in this small sample of children with autism ($n = 7$): atypical eye contact; abnormal visual tracking and visual attention disengagement; reduced social smiling, orienting to name, imitation, and reactivity; abnormal sensory-oriented behaviors; and language delays.

Some of these early abnormalities appear to be tightly linked to the motor system. In a case series report by Bryson et al. (2007) involving nine 6-mo-old infant siblings of children with autism who were themselves later diagnosed with autism, four infants presented with limited motor control. In addition, in a prospective study of siblings of children with autism and low-risk control participants, Landa and Garrett-Mayer (2006) reported motor delays by age 14 mo in children later diagnosed with ASD that distinguished them from comparison groups of children with typical development and language delays. Similarly, Ozonoff et al. (2010) reported the presence of fine motor delays by 18 mo in siblings of children with autism who had 36-mo outcomes of ASD that distinguished them from a typically developing low-risk comparison sample, supporting Landa and Garrett-Mayer's (2006) finding. These retrospective and prospective studies suggest that disturbance in motor behavior may be an early marker of impairment in children at risk for ASD.

Poor postural control during a pull-to-sit task (also referred to as *traction response* in the international literature) is an early predictor of developmental disability in other populations (e.g., infants with cerebral palsy, preterm infants; Barbosa, Campbell, Smith, & Berbaum, 2005; Samsom, de Groot, Bezemer, Lafeber, & Fetter, 2002; Samsom, Sie, & de Groot, 2002). In the present study, we focused on this aspect of motor development in siblings of children with autism. Postural control in infancy has been associated with self-exploratory behaviors (Rocha & Tudella, 2008), reaching (Thelen & Spencer, 1998), spontaneous motility, symmetry, and hand function (Samsom & de Groot, 2000). Abnormality in postural control in early infancy "disrupts the development of adequate motor behavior and sensorimotor interaction, which can result in a faulty perception–action cycle, thus influencing social and later cognitive development" (de Groot, 2000, p. 65).

The importance of early-developing motor skills for cognitive, language, and social development has been empirically documented (e.g., Campos et al., 2000; Iverson, 2010; Soska, Adolph, & Johnson, 2010). These studies, along with evidence that early motor functioning may provide a mechanism for predicting later impairment, including risk for autism (e.g., Phagava et al., 2008), highlight the need for research focused on early motor development in infants at high risk for autism (Bhat, Landa, & Galloway, 2011).

We examined postural control during pull-to-sit at age 6 mo in infants at high and low risk for ASD. First, we

examined the relationship between head lag and the following outcome variables in infant siblings of children with autism: later diagnosis of ASD and later impairment in motor, language, or social functioning. Next, in a subsequent between-groups comparison involving different participants, we examined whether the presence of head lag distinguished siblings of children with autism and low-risk controls at age 6 mo. We aimed to answer the following research questions:

1. Is postural control in midinfancy, as indicated by the presence of head lag in a pull-to-sit task, associated with diagnostic outcome classification in infants at high risk for ASD?
2. Do infants at high risk for ASD exhibit poor postural control more often than infants at low risk for ASD?

Method

Research Design

We studied two samples of infant siblings of children with autism prospectively in a federally funded longitudinal prospective study approved by the Johns Hopkins Institutional Review Board. In the first sample, we examined only siblings of children with autism using a longitudinal design. In the second sample, we compared a new group of siblings of children with autism with a low-risk group of infants with no family history of autism. All infants were admitted to the study following informed parental consent.

Participants

Participants were recruited through a federally funded longitudinal prospective study focusing on early patterns of development in autism (Landa & Garrett-Mayer, 2006). We followed infants in Sample 1 through 36 mo, when we established outcome diagnostic classification (see Procedures section). The funding for Sample 2 did not support follow-up assessments, so we report only the data from their 6-mo visit. Probands with idiopathic autism through whom the siblings were ascertained in both samples met diagnostic criteria for autism on the Autism Diagnostic Observation Schedule (ADOS; Lord, Rutter, DiLavore, & Risi, 2002) and the Autism Diagnostic Interview–Revised (Lord, Rutter, & Le Couteur, 1994) as well as clinical judgment of autism.

Recruitment sources for both samples included autism advocacy groups, conferences, schools, Kennedy Krieger Institute staff and patients, physicians' offices, caregiver–child play groups, and word of mouth. Low-risk infants were also recruited through mailings to families identified through public birth announcements.

Participants were eligible if English was the primary language of their parents and the infants had none of the following conditions: low birthweight (<2,250 g), severe birth trauma, head injury, prenatal illicit drug or excessive alcohol exposure, gestational age <37 mo, or severe birth defects.

Instruments

We used archived videos of the pull-to-sit task from the Gross Motor scale of the Mullen Scales of Early Learning (Mullen, 1995) to code head lag in all children. (Interrater reliability information is reported in the Procedures section.) For outcome classification at age 36 mo in Sample 1, we used the ADOS and the Mullen Scales, as well as clinical judgment.

Autism Diagnostic Observation Scale. The ADOS is a semistructured set of play-based probes for autism symptoms. Administration and scoring are standardized, and algorithm criteria provide for classifications of ASD or autism. The original Communication–Social Interaction algorithm cutoffs from Modules 1 or 2 contributed to our outcome classification of ASD. In the norming of the ADOS, internal consistency was assessed using Cronbach's alpha. For the Communication–Social Interaction total algorithm score, Cronbach's alphas were .91–.94 for all modules. For Module 1, sensitivity and specificity (autism and ASD vs. non-ASD classifications) were .97 and .94, respectively. For Module 2, sensitivity and specificity were .95 and .87, respectively (Lord, Rutter, DiLavore, & Risi, 2008).

Mullen Scales of Early Learning. The Mullen is a standardized developmental test for children aged 0–68 mo. Scales include Gross Motor, Fine Motor, Visual Reception, Receptive Language, and Expressive Language. The Receptive Language and Expressive Language scales served as the indexes for language delay (defined as a score of at least 1.5 standard deviations below the test mean). Internal consistency reliability, calculated in the normative sample using the median internal consistency split-half coefficients for the five Mullen scales, ranged from .75 to .83 (Mullen, 1995). Test–retest reliability (within a 1- to 2-wk interval) for the Gross Motor scale was .96, and the median correlations on the scale involving the four remaining scales ranged from .71 to .79 for participants aged 25–56 mo. Interrater reliability of the testers ranged from .91 to .99. To assess validity, correlations between the Mullen motor scale and cognitive (including language and nonverbal cognitive) scale scores were calculated using a variety of motor and cognitive measures that were considered to be tools of the trade in clinical psychology. The correlation between scores on the Mullen

Fine Motor scale and Peabody Fine Motor Scale (Folio & Fewell, 1983) in the normative sample 6–36 mo of age was .65–.82. In other comparisons, correlations were .53 and .85 (refer to Mullen, 1995, for details).

Clinical Judgment. Expert clinical researchers made clinical judgments of ASD using criteria for autism and pervasive developmental disorder–not otherwise specified (PDD–NOS) from the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; APA, 2000) or on the basis of delays in social, language, and motor development at the outcome assessment. The clinical researchers providing clinical judgments were blind to head lag status.

Procedures

Experienced pediatric occupational therapists coded videotapes of the Mullen Gross Motor pull-to-sit task; these researchers were blind to outcome classification for Sample 1 and risk status for Sample 2. They assigned a score of 0 when the infant maintained head alignment with the spine or in front of the spine from the beginning to the end of the pull-to-sit task and a score of 1 when any degree of head lag was observed. Interrater reliability was assessed for 57% and 25% of participants in Samples 1 and 2, respectively. Raters agreed on the presence or absence of head lag for 95% of cases (25/26; $\kappa = .898$) in Sample 1 and 100% of cases in Sample 2 ($\kappa = 1.0$). The first author (Flanagan) developed the codes used in the analyses.

Outcome Diagnostic Classification in Sample 1. Participants in Sample 1 were tested at ages 6, 14, 24, and (for outcome diagnosis) 30 ($n = 3$) or 36 mo. Outcome diagnosis was assigned at 30 or 36 mo because a diagnosis of ASD is considered stable at this age (Charman et al., 2005; Stone et al., 1999). The three possible outcome classifications were ASD, social/communication delay, and no communication/social delay ("nondelay"). Outcome classification was determined on the basis of clinical judgment paired with scores on one or more of the standardized measures described in the Instruments section.

Definition of Outcome Grouping Criteria in Sample 1. Participants with ASD ($n = 10$ boys) met ADOS algorithm criteria for autism or ASD in addition to having a clinical judgment of autism or PDD–NOS. Participants with social/communication delay ($n = 13$; 8 boys) met the criterion of ≥1.5 standard deviations below the mean on the Mullen Receptive or Expressive Language scales, met the ADOS algorithm cutoff criteria for ASD or autism on the Communication or Reciprocal Social Interaction domains with clinical judgment, or met such criteria for both ADOS algorithm domains even in the absence of clinical judgment of delay. Participants with nondelay ($n = 17$; 5 boys) did not meet the criteria for social/communication delay or ASD.

Data Analysis

Fisher's exact tests were used to test the associations between outcome classification (ASD vs. non-ASD, combining the social/communication delay and nondelay groups) and the presence of head lag at age 6 mo in Sample 1. A chi-square analysis was used to test the association between ASD risk status and presence of head lag in Sample 2. Statistical significance was set at $p < .05$ in two-tailed tests.

Results

Sample 1 consisted of 40 siblings of children with ASD aged 5.6 to 10.0 mo. Sample 2 was a new sample of 20 siblings of children with ASD (aged 5.8 to 7.0 mo) and 21 low-risk infants (aged 5.8 to 7.5 mo). Tables 1 and 2 provide demographic data, including age, gender, race, and socioeconomic status (Hollingshead, 1975), for each sample. Fisher's exact tests showed a significant gender difference between the three outcome groups in Sample 1 ($p < .001$), with more boys in the ASD group. This gender

Table 1. Sample 1 Group Differences in Demographics ($N = 40$)

Characteristic	Total[a]	ASD ($n = 10$)	Social/Communication Delay ($n = 13$)	Nondelay ($n = 17$)	*p*
Age, mo, *M* (*SD*)	6.6 (1.07)	6.12 (0.45)	6.68 (1.38)	6.83 (1.02)	.250
Male gender, *n* (%)	23 (58)	10 (100)	8 (62)	5 (29)	.001
Race, *n* (%)					.430
White	33 (83)	7 (70)	12 (92)	14 (82)	
Black/African American	1 (3)	0 (0)	0 (0)	1 (6)	
Multiracial	5 (13)	3 (30)	0 (0)	2 (12)	
Unspecified	1 (3)	0	1 (8)	0 (0)	
SES, *M* (*SD*)[a]	54.47 (10.98)	58.00 (10.29)	52.23 (14.29)[b]	53.58 (7.84)[b]	.470

Note. All comparisons were made using Fisher's exact tests except for age and SES, for which analysis of variance was used. ASD = autism spectrum disorders; *M* = mean; *SD* = standard deviation; SES = socioeconomic status.
[a]Percentages in the Total column do not add to 100 due to rounding. [b]Not all participants in this group had SES data available.

Table 2. Sample 2 Group Differences in Demographics ($N = 41$)

Characteristic	Total	High Risk ($n = 20$)	Low Risk ($n = 21$)	p
Age, mo, M (SD)	6.46 (0.39)	6.30 (0.29)	6.61 (0.43)	.010
Male gender, n (%)	16 (39)	9 (45)	7 (33)	.444
Race, n (%)				1.000
White	37 (90)	18 (90)	19 (90)	
Black/African American	2 (5)	1 (5)	1 (5)	
Unspecified	2 (5)	1 (5)	1 (5)	
SES, M (SD)	56.15 (7.33)	54.58 (7.33)[a]	57.64 (7.32)	.201

Note. Comparisons were made using t test for age and SES, chi-square for gender, and Fisher's exact test for ethnicity because the expected cell count was <5. M = mean; SD = standard deviation; SES = socioeconomic status.
[a]Not all participants in this group had SES data available.

difference is not surprising given that boys are affected by ASD 3 to 4 times more often than girls (APA, 2000). In Sample 2, we found a significant group difference in age, $t(39) = -2.75$, $p = .010$; however, the youngest age in the siblings of children with ASD and low-risk infants was 5.8 mo. Thus, this age difference should not affect performance because at 4 mo typically developing infants experience an increased ability to stabilize the head in midline when pulled to sit, and head lag dissipates (Bly, 1994).

Head lag was associated with ASD at 36 mo and was more frequently observed in siblings of children with ASD than in low-risk infants at age 6 mo. In Sample 1, Fisher's exact test indicated that significantly more infants later diagnosed with ASD exhibited head lag than infants without ASD (combined social/communication delay outcome group and nondelay group; $p = .02$). Within the ASD outcome group, 9 children (90%) exhibited head lag as infants. The 1 infant with ASD who did not display head lag exhibited atypical quality of movement characterized by rigidity and tremulousness in his upper extremities at age 6 mo. Seven (54%) children without ASD who exhibited social/communication delay at the outcome visit had exhibited head lag during infancy, and 1 such child who had not exhibited head lag during infancy exhibited a motor abnormality defined by tremulous movement in the upper extremities at age 6 mo.

In the nondelay group, 6 (35%) children exhibited head lag at age 6 mo. Inspection of the Mullen T scores (normed on a national sample) revealed that 4 of these siblings of children with ASD with head lag had scored at least 1.5 standard deviations below the Mullen Scales mean scores between ages 6 and 36 mo, although that delay may have resolved or was not detected by the Mullen Scales or ADOS at the 36-mo outcome assessment.

In Sample 2, assessed at age 6 mo, three-quarters (75%, $n = 15$) of siblings of children with ASD, compared with one-third (33%, $n = 7$) of low-risk infants, exhibited head lag. Chi-square analysis revealed that significantly more siblings of children with ASD than low-risk infants exhibited head lag, $\chi^2(1, N = 41) = 5.57$, $p = .018$.

Discussion

To our knowledge, this prospective, longitudinal study of early motor development is the first to identify head lag at age 6 mo in siblings of children with ASD and to associate early disruption in motor development with ASD risk. The presence of head lag after age 4 mo is not characteristic of typical development (Bly, 1994). Thus, head lag in siblings of children with autism at age 6 mo (age range = 5.6–10 mo) in a pull-to-sit task is likely to indicate a delay in neuromotor development (Nellhaus, 1983). Head lag during pull-to-sit may suggest low muscle tone and poor postural stability. Such problems involving axial hypotonicity and postural instability have been reported previously in infants with ASD (Adrien et al., 1993; Bryson et al., 2007). Head lag could also be secondary to abnormal sensory functioning. That is, failure to detect change in head position (possibly because of abnormal feedback from proprioception and vestibular–ocular righting systems) could result in failure to make the proper motor adjustments in anticipation of the change in positioning (Bly, 1994). This explanation is plausible, especially considering prior research suggesting deficits in anticipatory postural and motor control (Martineau et al., 2004) and in the integration of sensory input (Minshew et al., 2004; Molloy et al., 2003) in older people with autism.

Interestingly, when autism was first introduced as a syndrome, Kanner (1943) noted deficits in anticipatory postural adjustments in infants with autism. Deficits in anticipatory postural and motor control in autism may be attributed to impaired development and use of sensory–motor representations that, in typical development, provide a foundation for the ability to anticipate and flexibly adapt to the ever-changing physical and social environment (Schmitz &

Assaiante, 2008; Schmitz, Martineau, Barthélémy, & Assaiante, 2003).

Multiple factors could contribute to disrupted development of sensory–motor representations in autism. One such factor could involve diminished self-generated action experience. Sommerville, Woodward, and Needham (2005) demonstrated that a child's self-generated actions and sensory–motor representations are linked to their understanding of others' actions and intentions. Children with ASD are at high risk for atypical formation of sensory–motor representations because they exhibit reduced self-generated exploratory behavior (Pierce & Courchesne, 2001), possibly beginning very early in life (Bryson et al., 2007); this reduced exploration may adversely affect the infants' interaction with people and objects in the environment and the subsequent development of foundational play and social occupations.

In addition, head lag during pull-to-sit highlights the need for further investigation into the multisensory processing systems, including the interaction between vestibular, proprioceptive, and visual systems of infants at high risk for ASD. In typical development, infants use visual cues provided by the adult to anticipate being pulled into a sit position and make corresponding postural adjustments. Thus, early ASD indicators may be related to atypical timing and integration of the visually salient information of being pulled into a different posture (sit) and activation of the appropriate motor response (stabilization of the head).

Our assessments of the siblings of children with autism identified many indicators of disrupted neuromotor development, including fine and gross motor delays, poor postural control, poor motor coordination, hypoactivity, and atypical movements. For many of these infants, motor delays persisted into the 2nd year of life and beyond. Social delays often accompanied the motor delays, including decreased frequency and complexity of socially directed vocalization, flat facial affect, and infrequent pairing of eye contact with smiling during social engagement. Hence, an early motor deficit such as head lag could be a preclinical indicator of ASD and a precursor to impaired development of other communication and social aspects of autism, including gestures, reciprocal affect, and production of qualitatively and temporally synchronous movements within interpersonal interactions (Bhat et al., 2011; Flanagan & Landa, 2007).

We also detected head lag in a third of the low-risk group. This finding was unexpected and may indicate that our low-risk group was not representative of the general population. Indeed, Hadders-Algra, Heineman, Bos, and Middelburg (2010) reported that minor dysfunctions may be overrepresented in infants volunteered for participation in child development studies.

Our findings suggest the need for developmental surveillance for infants who have a positive family history for ASD (Johnson & Myers, 2007; Landa & Garrett-Mayer, 2006). Early developmental screening should include a pull-to-sit task for such infants once they reach age 6 mo as well as observation of and inquiry about timing of babble onset and complexity, fine and gross motor development, hypotonicity, postural control, hypoactivity, and atypical movements (Landa, 2011). Such screening will optimize early detection of developmental delays in this high-risk group and promote earlier access to intervention.

The present study had several limitations, including the absence of a comparison group of infants with idiopathic developmental delays. Our samples were relatively small, and the first sample consisted of relatively few children with ASD or impairments in language or social development. Diagnostic outcomes were not available for the second sample. The utility of using a pull-to-sit task for assessing the presence of head lag is a marker of early developmental disruption in siblings of children with autism requires further study in a more systematic manner, including coding severity of head lag and upper-extremity adaptation (or lack of) to task demand during a series of pull-to-sit maneuvers.

Our findings require replication with a larger number of children (at high and low risk for ASD) to better understand the relation between head lag in midinfancy and later developmental delay. Without diagnostic outcomes, we cannot be certain that no participants with ASD are included in the low-risk group, which is a potential confounder. Also, given the prevalence of developmental disabilities in the United States (Boulet, Boyle, & Schieve, 2009), following low-risk infants to an age when outcome diagnosis is possible may be a valuable addition to future studies. A variety of environmental (e.g., limited "tummy time") and genetic factors, may play a role in motor delays in siblings of children with autism.

Implications for Occupational Therapy Practice

This study suggests that head lag with other alterations in early development may be associated with autism risk and may serve as an early indicator of neurodevelopmental disruption. Results have clinical implications for occupational therapists in early intervention practice.

- Occupational therapists, in conjunction with speech–language therapists, physical therapists, and other

professionals, play an important role in early identification and intervention to address sensorimotor and social skills to improve participation in infants showing such red flags.

- Occupational therapists also may play an important role in research on early detection to identify infants exhibiting subtle early sensorimotor deficits that may affect subsequent development of play and social occupations.

Conclusion

Disruptions in early motor development in siblings of children with autism may serve as red flags for later delay in language and social development. The results of this study indicate that a simple pull-to-sit task should be added to existing developmental screenings at pediatric well visits. Further studies should systematically assess both quantitative and qualitative aspects of motor behavior and examine the potential influence of vestibular and ocular systems on head righting prospectively and longitudinally in infants at high and low risk for ASD. Assessing the quality, quantity, and variety of sensorimotor skills in conjunction with social, communication, and emotional regulation behaviors may yield important insights into the very earliest manifestation of the autism-related phenotype. We propose that identifying motor delays in infants at high risk for autism could lead to earlier intervention to facilitate better outcomes in motor, social, and communication development and minimize disabilities and improve participation in early infant occupations. ▲

Acknowledgments

We thank the families who participated in this research and the staff who collected and managed the data, particularly Alison Marvin, Ashley Faherty, and Katelyn Boswell. We especially thank Douglene Jackson and Barbara Benen Demchick, who coded tapes for interrater reliability. Rebecca Landa thanks the National Institute of Mental Health for funding this work through RO1 award MH59630, and Anjana Bhat thanks Cure Autism Now and Karma Foundation for supporting this work with a Young Investigator Award.

References

Adrien, J. L., Lenoir, P., Martineau, J., Perrot, A., Hameury, L., Larmande, C., et al. (1993). Blind ratings of early symptoms of autism based upon family home movies. *Journal of the American Academy of Child and Adolescent Psychiatry, 32,* 617–626. http://dx.doi.org/10.1097/00004583-199305000-00019

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Barbosa, V. M., Campbell, S. K., Smith, E., & Berbaum, M. (2005). Comparison of Test of Infant Motor Performance (TIMP) item responses among children with cerebral palsy, developmental delay, and typical development. *American Journal of Occupational Therapy, 59,* 446–456. http://dx.doi.org/10.5014/ajot.59.4.446

Bhat, A., Landa, R., & Galloway, J. C. (2011). Perspectives on motor problems in infants, children, and adults with autism spectrum disorders. *Physical Therapy, 91,* 1116–1129. http://dx.doi.org/10.2522/ptj.20100294

Bly, L. (1994). *Motor skills acquisition in the first year: An illustrated guide to normal development.* San Antonio, TX: Therapy Skill Builders.

Boulet, S. L., Boyle, C. A., & Schieve, L. A. (2009). Health care use and health and functional impact of developmental disabilities among US children, 1997–2005. *Archives of Pediatrics and Adolescent Medicine, 163,* 19–26. http://dx.doi.org/10.1001/archpediatrics.2008.506

Bryson, S. E., Zwaigenbaum, L., Brian, J., Roberts, W., Szatmari, P., Rombough, V., et al. (2007). A prospective case series of high-risk infants who developed autism. *Journal of Autism and Developmental Disorders, 37,* 12–24. http://dx.doi.org/10.1007/s10803-006-0328-2

Campos, J. J., Anderson, D. I., Barbu-Roth, M. A., Hubbard, E. M., Hertenstein, M. J., & Witherington, D. (2000). Travel broadens the mind. *Infancy, 1,* 149–219. http://dx.doi.org/10.1207/S15327078IN0102_1

Charman, T., Taylor, E., Drew, A., Cockerill, H., Brown, J., & Baird, G. (2005). Outcome at 7 years of age of children diagnosed with autism at age 2: Predictive validity of assessments conducted at 2 and 3 years of age and pattern of symptom change over time. *Journal of Child Psychology and Psychiatry, and Allied Disciplines, 46,* 500–513. http://dx.doi.org/10.1111/j.1469-7610.2004.00377.x

de Groot, L. (2000). Posture and motility in preterm infants. *Developmental Medicine and Child Neurology, 42,* 65–68. http://dx.doi.org/10.1017/S0012162200000128

Dowell, L. R., Mahone, E. M., & Mostofsky, S. H. (2009). Associations of postural knowledge and basic motor skill with dyspraxia in autism: Implication for abnormalities in distributed connectivity and motor learning. *Neuropsychology, 23,* 563–570. http://dx.doi.org/10.1037/a0015640

Dziuk, M. A., Gidley Larson, J. C., Apostu, A. A., Mahone, E. M., Denkla, M. B., & Mostofsky, S. H. (2007). Dyspraxia in autism: Association with motor, social, and communicative deficits. *Developmental Medicine and Child Neurology, 49,* 734–739. http://dx.doi.org/10.1111/j.1469-8749.2007.00734.x

Einspieler, C., Prechtl, H. F. R., Bos, A. F., Ferrari, F., & Cioni, G. (2004). *Prechtl's method on the qualitative assessment of general movements in preterm, term and young infants.* London: Mac Keith Press.

Esposito, G., Venuti, P., Maestro, S., & Muratori, F. (2009). An exploration of symmetry in early autism spectrum disorders: Analysis of lying. *Brain and Development, 31,* 131–138. http://dx.doi.org/10.1016/j.braindev.2008.04.005

Flanagan, J., & Landa, R. (2007, April). *Longitudinal study of motor development in infants at high and low risk for autism.* Paper presented at the American Occupational Therapy Association Annual Conference & Expo, St. Louis, MO.

Folio, M. R., & Fewell, R. F. (1983). *Peabody Developmental Motor Scales and Activity Cards.* Chicago: Riverside.

Fournier, K. A., Hass, C. J., Naik, S. K., Lodha, N., & Cauraugh, J. H. (2010). Motor coordination in autism spectrum disorders: A synthesis and meta-analysis. *Journal of Autism and Developmental Disorders, 40,* 1227–1240. http://dx.doi.org/10.1007/s10803-010-0981-3

Hadders-Algra, M., Heineman, K. R., Bos, A. F., & Middelburg, K. J. (2010). The assessment of minor neurological dysfunction in infancy using the Towen Infant Neurological Examination: Strengths and limitations. *Developmental Medicine and Child Neurology, 52,* 87–92. http://dx.doi.org/10.1111/j.1469-8749.2009.03305.x

Hobson, R. P., & Lee, A (1999). Imitation and identification in autism. *Journal of Child Psychology and Psychiatry, and Allied Disciplines, 40,* 649–659. http://dx.doi.org/10.1111/1469-7610.00481

Hollingshead, A. B. (1975). *Four Factor Index of Social Status.* Unpublished working Paper, Department of Sociology, Yale University, New Haven, CT.

Iverson, J. M. (2010). Developing language in a developing body: The relationship between motor development and language development. *Journal of Child Language, 37,* 229–261. http://dx.doi.org/10.1017/S0305000909990432

Jansiewicz, E. M., Goldberg, M. C., Newschaffer, C. J., Denckla, M. B., Landa, R., & Mostofsky, S. H. (2006). Motor signs distinguish children with high functioning autism and Asperger's syndrome from controls. *Journal of Autism and Developmental Disorders, 36,* 613–621. http://dx.doi.org/10.1007/s10803-006-0109-y

Johnson, C. P., & Myers, S. M.; Council on Children With Disabilities. (2007). American Academy of Pediatrics Council on Children with Disabilities: Identification and evaluation of children with autism spectrum disorders. *Pediatrics, 120,* 1183–1215. http://dx.doi.org/10.1542/peds.2007-2361

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child, 2,* 217–250.

Landa, R. (2011). Developmental features and trajectories associated with autism spectrum disorders in infants and toddlers. In D. Amaral, G. Dawson, & D. H. Geschwind (Eds.), *Autism spectrum disorders* (pp. 213–228). New York: Oxford University Press.

Landa, R., & Garrett-Mayer, E. (2006). Development in infants with autism spectrum disorders: A prospective study. *Journal of Child Psychology and Psychiatry, and Allied Disciplines, 47,* 629–638. http://dx.doi.org/10.1111/j.1469-7610.2006.01531.x

Landa, R. J., Holman, K. C., O'Neil, A. H., & Stuart, E. A. (2011). Intervention targeting development of socially synchronous engagement in toddlers with autism spectrum disorder: A randomized controlled trial. *Journal of Child Psychology and Psychiatry, and Allied Disciplines, 52,* 13–21. http://dx.doi.org/10.1111/j.1469-7610.2010.02288.x

Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. (2002). *Autism Diagnostic Observation Schedule.* Los Angeles: Western Psychological Services.

Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. (2008). *Autism Diagnostic Observation Schedule.* Los Angeles: Western Psychological Services.

Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism Diagnostic Interview–Revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders, 24,* 659–685. http://dx.doi.org/10.1007/BF02172145

Martineau, J., Schmitz, C., Assaiante, C., Blanc, R., & Barthélémy, C. (2004). Impairment of a cortical event-related desynchronisation during a bimanual load-lifting task in children with autistic disorder. *Neuroscience Letters, 367,* 298–303. http://dx.doi.org/10.1016/j.neulet.2004.06.018

Minshew, N. J., Sung, K., Jones, B. L., & Furman, J. M. (2004). Underdevelopment of the postural control system in autism. *Neurology, 63,* 2056–2061.

Molloy, C. A., Dietrich, K. N., & Bhattacharya, A. (2003). Postural stability in children with autism spectrum disorder. *Journal of Autism and Developmental Disorders, 33,* 643–652. http://dx.doi.org/10.1023/B:JADD.0000006001.00667.4c

Mullen, E. M. (1995). *Mullen Scales of Early Learning* (AGS ed.). Circle Pines, MN: American Guidance Service.

Nellhaus, G. (1983). Review article: Abnormal head movements of young children. *Developmental Medicine and Child Neurology, 25,* 384–389. http://dx.doi.org/10.1111/j.1469-8749.1983.tb13778.x

Noterdaeme, M., Mildenberger, K., Minow, F., & Amorosa, H. (2002). Evaluation of neuromotor deficits in children with autism and children with a specific speech and language disorder. *European Child and Adolescent Psychiatry, 11,* 219–225. http://dx.doi.org/10.1007/s00787-002-0285-z

Ozonoff, S., Iosif, A., Baguio, F., Cook, I. C., Moore Hill, M., Hutman, T., et al. (2010). A prospective study of the emergence of early behavioral signs of autism. *Journal of the Academy of Child and Adolescent Psychology, 49,* 256–266.

Ozonoff, S., Young, G., Carter, A., Messinger, D., Yirmiya, N., Zwaigenbaum, L., et al. (2011). Recurrence risk for autism spectrum disorders: A Baby Siblings Research Consortium study. *Pediatrics, 128,* e488–e495. http://dx.doi.org/10.1542/peds.2010-2825

Ozonoff, S., Young, G. S., Goldring, S., Greiss-Hess, L., Herrera, A. M., Steele, J., et al. (2008). Gross motor development, movement abnormalities, and early identification of autism. *Journal of Autism and Developmental Disorders, 38,* 644–656. http://dx.doi.org/10.1007/s10803-007-0430-0

Phagava, H., Muratori, F., Einspieler, C., Maestro, S., Apicella, F., Guzzetta, A., et al. (2008). General movements in infants with autism spectrum disorders. *Georgian Medical News, 156,* 100–105.

Pierce, K., & Courchesne, E. (2001). Evidence for a cerebellar role in reduced exploration and stereotyped behavior in autism. *Society of Biological Psychiatry, 49,* 655–664. http://dx.doi.org/10.1016/S0006-3223(00)01008-8

Rinehart, N. J., Tonge, B. J., Bradshaw, J. L., Iansek, R., Enticott, P. G., & McGinley, J. (2006). Gait function

in high-functioning autism and Asperger's disorder: Evidence for basal-ganglia and cerebellar involvement. *European Child and Adolescent Psychiatry, 15,* 256–264. http://dx.doi.org/10.1007/s00787-006-0530-y

Rocha, N.A.C.F., & Tudella, E. (2008). The influence of lying position and postural control on hand–mouth and hand–hand behaviors in 0–4-month-old infants. *Infant Behavior and Development, 31,* 107–114. http://dx.doi.org/10.1016/j.infbeh.2007.07.004

Rogers, S. J., Hepburn, S. L., Stackhouse, T., & Wehner, E. (2003). Imitation performance in toddlers with autism and those with other developmental disorders. *Journal of Child Psychology and Psychiatry, and Allied Disciplines, 44,* 763–781. http://dx.doi.org/10.1111/1469-7610.00162

Samsom, J. F., & de Groot, L. (2000). The influence of postural control on motility and hand function in a group of "high-risk" preterm infants at 1 year of age. *Early Human Development, 60,* 101–113. http://dx.doi.org/10.1016/S0378-3782(00)00107-9

Samsom, J. F., de Groot, L., Bezemer, P. D., Lafeber, H. N., & Fetter, W. P. F. (2002). Muscle power development during the first year of life predicts neuromotor behavior at 7 years in preterm born high-risk infants. *Early Human Development, 68,* 103–118. http://dx.doi.org/10.1016/S0378-3782(02)00019-1

Samsom, J. F., Sie, L. T. L., & de Groot, L. (2002). Muscle power development in preterm infants with periventricular flaring or leukomalacia in relation to outcome at 18 months. *Developmental Medicine and Child Neurology, 44,* 735–740. http://dx.doi.org/10.1111/j.1469-8749.2002.tb00279.x

Schmitz, C., & Assaiante, C. (2008). Postural control in children with autism spectrum disorder. In M. Hadders-Algra & E. Brogen Carlberg (Eds.), *Postural control: A key issue in developmental disorders* (pp. 253–269). London: Mac Keith Press.

Schmitz, C., Martineau, J., Barthélémy, C., & Assaiante, C. (2003). Motor control and children with autism: Deficit of anticipatory function. *Neuroscience Letters, 348,* 17–20. http://dx.doi.org/10.1016/S0304-3940(03)00644-X

Sommerville, J. A., Woodward, A. L., & Needham, A. (2005). Action experience alters 3-month-old infants' perception of others' actions. *Cognition, 96,* B1–B11. http://dx.doi.org/10.1016/j.cognition.2004.07.004

Soska, K. C., Adolph, K. E., & Johnson, S. P. (2010). Systems in development: Motor skill acquisition facilitates three-dimensional object completion. *Developmental Psychology, 46,* 129–138. http://dx.doi.org/10.1037/a0014618

Stone, W. L., Lee, E. B., Ashford, L., Brissie, J., Hepburn, S. L., Coonrod, E. E., et al. (1999). Can autism be diagnosed accurately in children under 3 years? *Journal of Child Psychology and Psychiatry, and Allied Disciplines, 40,* 219–226. http://dx.doi.org/10.1111/1469-7610.00435

Teitelbaum, P., Teitelbaum, O., Nye, J., Fryman, J., & Maurer, R. G. (1998). Movement analysis in infancy may be useful for early diagnosis of autism. *Proceedings of the National Academy of Sciences of the United States of America, 95,* 13982–13987.

Thelen, E., & Spencer, J. P. (1998). Postural control during reaching in young infants: A dynamic systems approach. *Neuroscience and Biobehavioral Reviews, 22,* 507–514. http://dx.doi.org/10.1016/S0149-7634(97)00037-7

Vernazza-Martin, S., Martin, N., Vernazza, A., Lepellec-Muller, A., Rufo, M., Massion, J., et al. (2005). Goal directed locomotion and balance control in autistic children. *Journal of Autism and Developmental Disorders, 35,* 91–102. http://dx.doi.org/10.1007/s10803-004-1037-3

Vilensky, J. A., Damasio, A. R., & Maurer, R. G. (1981). Gait disturbances in patients with autistic behavior: A preliminary study. *Archives of Neurology, 38,* 646–649. http://dx.doi.org/10.1001/archneur.1981.00510100074013

Zwaigenbaum, L., Bryson, S., Rogers, T., Roberts, W., Brian, J., & Szatmari, P. (2005). Behavioral manifestations of autism in the first year of life. *International Journal of Developmental Neuroscience, 23,* 143–152. http://dx.doi.org/10.1016/j.ijdevneu.2004.05.001