4-2011

Sensory Information Utilization and Time Delays Characterize Motor Developmental Pathology in Infant Sitting Postural Control

Joan E. Deffeyes
University of Nebraska Medical Center

Regina T. Harbourne
University of Nebraska Medical Center

Wayne A. Stuberg
University of Nebraska Medical Center

Nikolaos Stergiou
University of Nebraska at Omaha, nstergiou@unomaha.edu

Motor Control, 2011, 15, 302-317

Sensory Information Utilization and Time Delays Characterize Motor Developmental Pathology in Infant Sitting Postural Control

Joan E. Deffeyes, Regina T. Harbourne, Wayne A. Stuberg, and Nicholas Stergiou

Sitting is one of the first developmental milestones that an infant achieves. Thus measurements of sitting posture present an opportunity to assess sensorimotor development at a young age. Sitting postural sway data were collected using a force plate, and the data were used to train a neural network controller of a model of sitting posture. The trained networks were then probed for sensitivity to position, velocity, and acceleration information at various time delays. Infants with typical development developed a higher reliance on velocity information in control in the anterior-posterior axis, and used more types of information in control in the medial-lateral axis. Infants with delayed development, where the developmental delay was due to cerebral palsy for most of the infants in the study, did not develop this reliance on velocity information, and had less reliance on short latency control mechanisms compared with infants with typical development.

Keywords: cerebral palsy, developmental delay, infant, artificial neural network, postural sway, sitting

Cerebral palsy is due to a brain injury that occurs early in life, where "cerebral" indicates involvement of the cerebrum, and "palsy" indicates a movement disorder. Thus impairment in motor function is a hallmark of the disorder, but impairments in sensory function are also prevalent, perhaps as a result of injury to thalamocortical pathways (Hoon, et al., 2009). Sensory impairment can include proprioception (Goble, Hurvitz, & Brown, 2009) and cutaneous sensation (Lesny, Stehlik, Tomasek, Tomankova, & Havlicek, 1993; Sanger & Kukke, 2007), and sensory deficits and/or deficits in sensory integration likely contribute both to impairment in motor performance (Bumin & Kayihan, 2001; Bumin & Kavak, 2008; Hadders-Algra, van der Fits, Stremmelaar, & Touwen, 1999) and motor development (Wilke & Staudt, 2009). Sitting is an important motor control skill that infants learn early in

Deffeyes, Harbourne, and Stuberg are with the Munroe-Meyer Institute, University of Nebraska Medical Center; Omaha, NE. Stergiou is with the Nebraska Biomechanics Core Facility; University of Nebraska at Omaha, and the Department of Environmental, Agricultural and Occupational Health Sciences; College of Public Health, University of Nebraska Medical Center; Omaha, NE.

life, at about age 6–8 months. Stable sitting allows the infant to reach for objects in his environment, and allows visual inspection of the environment. In addition, sitting is a major developmental milestone. A strong correlation between ability to sit independently by age 2 years and ability to walk independently by 3–5 years age has been found in children with spastic diplegic or triplegic cerebral palsy (Fedrizzi, et al., 2000). Thus sitting is not only important in itself, but can serve as a window into the sensorimotor system of the developing infant, and provide insight into deficits in motor control in infants with developmental delay.

The control of sitting posture, like standing posture, requires maintaining the center of mass within the base of support. To achieve this goal both in sitting and standing, information from various sensory modalities, including visual information, vestibular information, proprioceptive information, and cutaneous information, is used to provide feedback for various postural control mechanisms (Horak, 2006). Much of the research on postural control in standing is focused on understanding the contributions of these different modes of sensory information, which is accomplished by blocking or altering various sensory modalities, such as closed eyes/open eyes to investigate the importance of vision in postural sway (Kiemel, Oie, & Jeka, 2002), altering visual surround movement to provide false visual information (Peterka, 2002), using vibration to alter touch information to investigate the importance of cutaneous sensory input (Kiemel, Oie, & Jeka, 2002), or use of galvanic stimulation to investigate vestibular function in postural sway (Ali, Rowen, & Iles, 2003). However, when one sensory modality is altered, the information from other modalities is used more for control; i.e., sensory reweighting occurs such that the control dynamics may not be representative of the control dynamics under more typical conditions. For example, in normal adult standing, about a third of the information used for control is from visual information (Peterka, 2002), but in the blindfolded condition used as an experimental manipulation of sensory input for postural control, vestibular information and proprioceptive information become more heavily weighted (Horak, 2006).

A different strategy in the study of postural control is to apply mechanical perturbations to the subject, and characterize the response, to gain insight into the postural control mechanism. Perturbation methods have been applied to adult sitting (Granata, Slota, & Bennett, 2004) and to infant sitting (Harborne, Giuliani, & Neela, 1993; Hedberg, Carlberg, Forssberg, & Hadders-Algra, 2005; Hedberg, Forssberg, & Hadders-Algra, 2004; Hirschfeld & Forssberg, 1994). These studies characterize the response to extreme events that may not represent typical control mechanisms in unperturbed sitting. For example, stretch reflexes might be triggered by a strong perturbation during sitting (Granata, Slota, & Bennett, 2004), but it is not clear from that result whether stretch reflexes are important in control of unperturbed sitting. While understanding sensory reweighting and response to external perturbations are important goals, it is also important to understand normal postural control, i.e., postural control without experimentally altered sensory input or external perturbations. Normal postural control serves as a baseline with which to compare experimental manipulations of postural control, and is relevant to postural control in many everyday situations. Thus it is desirable to develop methods to study normal postural control, and analysis of center of pressure (COP) data from unperturbed sitting with no sensory manipulation is one such method, and it is the method we have chosen to investigate infant sitting.

The mechanism for control of upright posture is not known, but a leading hypothesis is that a control parameter is the time to contact of the perimeter of the base of support (Slobounov, Cao, Jaiswal, & Newell, 2009). To calculate the time to contact parameter, position, velocity and acceleration information must be known. The various sensory modalities provide different types of sensory information. Visual information may include position, velocity and acceleration (Thiel, Greschner, Eurich, Ammermuller, & Kretzberg, 2007). The vestibular labyrinth is particularly suited to sensing acceleration information (Kandel, Jessell, & Schwartz, 2000, p. 802–803). Proprioceptive feedback includes position, velocity and acceleration information (Schouten et al. 2008). Stretch receptors in the skin also contribute information for postural control (Kandel, Jessell, & Schwartz, 2000, p 443). These multiple modes of sensory information must be interpreted and integrated by the central nervous system in order for postural control mechanisms to maintain upright posture (Horak, 2006). While estimations of position information, velocity information, and acceleration information are all available from integrated sensory input, it is not known which information is actually used for infant sitting postural control. Velocity information is thought to be more accurately estimated than position or acceleration from sensory input, and that it is the predominate type of information used for standing postural control in healthy adults (Jeka, Kiemel, Creath, Horak, & Peterka, 2004). It is unclear if infant sitting postural control can benefit from relying more heavily on the more accurately estimated velocity information, compared with position or acceleration information, or if the time-to-contact calculation requires equal use of all three types of information. In addition, it is not known if infants with developmental delay will use the same types of sensory information on a delayed developmental schedule, or if they will adaptively find alternate ways to use sensory information to compensate for sensorimotor deficits.

Postural control, just like any motor control task, is accomplished by contraction of the appropriate muscles at the appropriate time. If sensory information indicates an acceleration in a particular direction is needed, then a motor command is executed to provide that acceleration. At a given point in time, the sensory system may detect position, velocity, and acceleration information, but there is a time lag before that information can be acted upon. The time lag is due to nerve conduction time for the sensory information to flow to the central nervous system (CNS), processing of the sensory signal by the CNS, motor command flow back to the muscle, and muscle activation time. There are a range of delay times that have been measured in adult postural control, including stretch reflex time delay with a latency on the order of about 30 msec and rise time of about 70 msec (Granata, Slota, & Bennett, 2004), vestibular control time delay on the order of 60–100 msec (Ali, Rowen, & Iles, 2003), and visual control time delay on the order of about 500–750 msec (van den Heuvel, Balasubramaniam, Daffertshofer, Longtin, & Beek, 2009). Multiple postural control mechanisms exist (Horak, 2006), resulting in multiple time scales associated with postural control, as the various control mechanisms have different time delays associated with them. Thus time delay is a critical parameter in analysis of postural sway data. In investigating how infants use position, velocity, and acceleration information, it is necessary to also investigate the time delay associated with the utilization of that information.

Conceptually, to maintain upright sitting posture, a control signal is generated by biological neural networks within the central nervous system, with sensory information as the input. The output of the biological controller is a motor control signal that initiates muscle activation. Muscles produce forces and joint torques, which are proportional to accelerations via Newton's second law, often written as F = ma for a system of constant mass. Due to finite nerve conduction velocities and muscle activation response times, there is a time delay between the activation of sensory neurons, and the acceleration of the body that occurs following the sensory input. Thus the biological system has sensory input from which, after sensory integration, includes position, velocity and acceleration information, and the output is a muscle activation that causes an acceleration at time delay τ. As a model of the biological control system in this work, we will use a simple artificial neural network (ANN) controller. The input to the ANN is position, velocity and acceleration at time t, and the output is an acceleration at time $t+\tau$. By training the ANN with position, velocity, and acceleration information form experimental COP data from infant sitting, and then probing the response of the network with a sensitivity analysis, the importance of position, velocity and acceleration information to the postural control can be evaluated. Using this model of the biological control system, we asked the following questions: 1. Was position, velocity, or acceleration information important for infant sitting postural control, and on what time scale is this information used? 2. Did infants with developmental delay use different information, or use information on different time scales, than infants with typical development?

We hypothesized that infants use velocity information more than position or acceleration information for sitting postural control, based upon velocity information utilization in adult standing postural sway (Jeka et al. 2004) We also hypothesized that infants with delayed development use sensory information differently compared with infants with typical development (Hoon, et al., 2009; Goble, Hurvitz, & Brown, 2009; Lesny, Stehlik, Tomasek, Tomankova, & Havlicek, 1993; Sanger & Kukke, 2007), rather than simply being delayed in development (Chen & Woollacott, 2007). This hypothesis was based on sensory deficits in infants with cerebral palsy, which compromise the majority of the sample with atypical development in our study.

Method

Research Methods Overview

The method involved four steps, 1. the collection of postural sway data from infants with typical and with delayed development, once when they could just sit for 10 s (early sitting), and again about 3 months later (late sitting), 2. Calculation of velocity and acceleration from the measured postural sway position data, 3.Training an artificial neural network using the position, velocity, and acceleration information, and probing the train networks for sensitivity to position, velocity, and acceleration, and 4. Repeated-measures ANOVA on the sensitivities. Each of these steps is discussed in more detail below.

Infant Participants and Data Collection

Thirty infants with developmental delay (age = 14.05 months, std = 5.33 months, for early sitting and age = 18.06 months, std = 5.09 months, for late sitting) and 33 infants with typical development (age = 4.92 months, std = 0.57 months, for early sitting, and age = 7.92 months, std = 0.60 months, for late sitting) participated in the study. Recruitment was done through newsletters, flyers, and pediatric physical therapists employed at the University. Infants in the developmentally delayed group were diagnosed with cerebral palsy, or else were developmentally delayed and at risk for cerebral palsy. Obtaining a firm diagnosis of cerebral palsy at this young age is often not possible. Because a definitive diagnosis of cerebral palsy had not always been made, we refer to these infants as developmentally delayed, because all scored greater than 1.5 standard deviations below the mean for their corrected age on the Peabody Developmental Gross Motor Scale (Folio & Fewell, 2000). However, the development is likely not just delayed, but also atypical (Chen & Wollacott, 2007). A consent form was signed by a parent or guardian of all infant participants, and all procedures were approved by the University of Nebraska Medical Center Institutional Review Board.

Inclusion criteria for entry into the study for the typically developing infants were: a score on the Peabody Developmental Gross Motor Scale of greater than 0.5 *SD* below the mean, age of five months at the time of initial data collection, and sitting skills as described below in beginning sitting. Exclusion criteria for the sample of infants who were typically developing were: a score on the Peabody Developmental Gross Motor Scales greater than 0.5 *SD* below the mean, diagnosed visual deficits, or diagnosed musculoskeletal problems. If a typically developing infant was found to be less than 0.5 *SD* below the mean, and did not qualify for the study, the parents were informed of the score, the possibility of error in the measurement, and advised to have the infant reevaluated within the next 3 months. Operational definitions of beginning sitting were used to determine the infant's readiness for entry into the study. Beginning sitting was defined as (a) head control such that when trunk is supported at the midtrunk, head is maintained for over one minute without bobbing; (b) infant can track an object across midline without losing head control; (c) infant may prop hands on floor or legs to lean on arms, but should not be able to reach and maintain balance in the prop sit position; (d) when supported in sitting can reach for toy; (e) can prop on elbows in the prone position for at least 30 s. Each infant was tested when they entered into the study based on the ability to sit for about 10 s (early sitting), and then again 3–4 months later (late sitting).

For the infants with developmental delay the inclusion and exclusion criteria were as follows. Inclusion criteria were: age from five months to two years, score greater than 1.5 *SD* below the mean for their corrected age on the Peabody Developmental Gross Motor Scales, and sitting skills as described above for beginning sitting. Exclusion criteria were: age over two years, a score less than 1.5 *SD* below the mean for their corrected age on the Peabody Developmental Gross Motor Scale, a diagnosed visual impairment, or a diagnosed hip dislocation or subluxation greater than 50%.

For all data collection sessions, the infants were allowed time to get used to the laboratory setting, and were at their parent's side or on their lap for preparation and data collection. All attempts were made to maintain a calm, alert state by allowing the infant to eat if hungry, be held by a parent for comforting, or adapting the temperature of the room to the infant's comfort level. A blanket was placed over the plate for warmth and was securely adhered with double-sided tape on the ground. The baby was held in the sitting position in the middle of the plate to start. Once the examiner could completely let go of the infant, data were collected for 10 s while the infant attempted to maintain sitting postural control. Trials were performed until we had collected three trials, or until the infant was no longer interested in sitting, i.e., was crying or agitated and could not be calmed. At any time the infant became irritated; the session was halted for comforting by the parent or a chance for feeding, and then resumed only when the infant was again in a calm state. We attempted to collect three trials at each of the two sessions, but could not always get that many, depending on the infant's behavior.

For data acquisition (Figure 1), infants sat on an AMTI force plate (Watertown, MA), interfaced to a computer system running Vicon data acquisition software (Lake Forest, CA). Center of pressure (COP) data were acquired at 240 Hz using the Vicon software. Trials were recorded including force plate data and video data from the back and side views. Afterward segments were selected by viewing the corresponding video. Segments of data with 2000 time steps were selected from these trials by examination of the video. Acceptable segments were required to have no crying or long vocalization, no extraneous items (e.g., toys) on the force platform, neither the assistant nor the mother were touching the infant, the infant was not engaged in rhythmic behavior (e.g., flapping arms), and the infant had to be sitting and could not be in the process of falling.

Figure 1 — Postural sway COP data were collected as an infant sits on a force plate. COP data were used to train the neural network.

Calculation of Position, Velocity and Acceleration from COP Data

The time delay in a sensory feedback system is an important parameter. Since the goal is to model actual infant sitting, the delay from one time step to the next should be appropriate for human motor control. The data in this study was acquired at 240 Hz, meaning there were 240 data points collected each second, or a time lag of 4.2 msec between points. To investigate time lags of different lengths, the time series data were sectioned into nonoverlapping windows sized from 33 msec (8 data points) to 750 msec (180 data points). Position data for each window was calculated as the average position for that window. Velocity data were calculated by differencing the position data in that window, and calculating the average, and similarly differencing the position data twice and averaging gave the acceleration for that window. Thus from the original time series, three time series were calculated: position time series, velocity time series, and acceleration time series.

Because the effect of the three different information types were to be compared, all of the input data to the model needed to be comparable in magnitude for the comparison with be meaningful. Each point of the position data were then normalized by subtracting the mean and dividing by the standard deviation for all position data. Likewise, all the velocity data were normalized using mean and standard deviation for velocity, and acceleration data normalized using mean and standard deviation for acceleration. The normalization process was used in order that each type of data had a mean of zero and a standard deviation of 1, and thus the weights from the ANN would be related to the importance of that type of information, and not influenced by the different units on position, velocity and acceleration.

Neural Network Model

A simple neural network model was created with 3 neurons in the input layer, one each for position, velocity, and acceleration; a hidden layer with 6 neurons, and an output layer with one neuron (Figure 2). All neurons used a simple sigmoidal function for activation (Duda, Hart, & Stork, 2001), which has an output of [0, 1], so the acceleration for comparison with the model output was normalized to be in

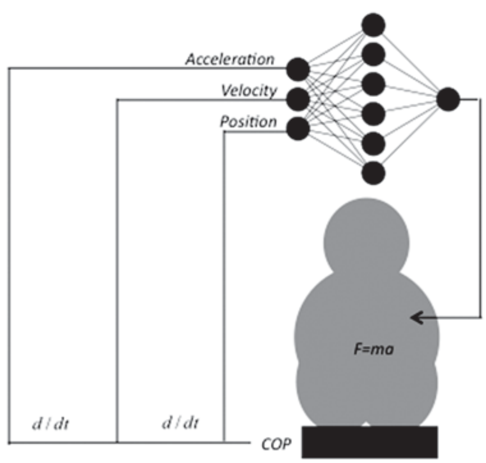


Figure 2 — Model of infant as a sitting on a force plate with a neural network controller. Force plate is indicated as a black box that outputs COP data, which is differentiated to get velocity and acceleration sensory information, the input to the neural network. The output of the network is a control signal that drives muscles to maintain upright sitting posture of the infant. We measured COP with the force plate to derive the position, velocity, and acceleration sensory data for the model, whereas the infant relies on visual, vestibular, proprioceptive, and cutaneous sensory input for this information.

the range [0,1]. Each neuron in the model summed the input from the preceding layer, and the applied the sigmoidal function in Equation 1 to calculate output,

$$f(net_j) = \frac{1}{1 + e^{-\sigma \cdot net_j}}$$

where σ is a steepness parameter, that was set equal to one for this model, and netj is the summation of input to the neuron j. The output of a sigmoid neuron is between zero and one, so all the desired output calculated from the infant posture data were scaled to be between zero and one.

Back propagation of error was used to train the network, where error calculated in each time step was back-propagated based on the current weights of the network, allowing new weights to be calculated (Duda, Hart, & Stork, 2001). Initial weights were randomly generated. Iteration was terminated when the error reached below a threshold value, and if the algorithm did not converge, new random weights were chosen, and the training repeated. The network was trained using the inputs position, velocity and acceleration at time (t), and trained to calculate acceleration at the next time window (t+1). The contribution of position, velocity and acceleration were ascertained by propagating [p,v,a], through the trained network, where p is a position value, v is a velocity value, and a is an acceleration value. For example, propagation of [1,0,0] through the trained network results in an output that indicates the response of to a positive position, and neutral velocity and acceleration, i.e., what acceleration would the infant's muscles and gravity provide if the infant were leaning 1 standard deviation away from the mean in the positive direction. The output of the network is in the range [0,1], where a value of 0.5 corresponds to no acceleration, an output close to 0 corresponds to a negative acceleration, and a value near 1 corresponds to a positive acceleration. In this manner, for each time series, the contribution of position, velocity, and acceleration were determined for each time series by propagation of [1,0,0], [0,1,0], and [0,0,1], respectively.

Statistical Analysis

A mixed repeated-measures ANOVA analysis was performed with 2 levels of time (early and late sitting), two axes (anterior-posterior and medial-lateral), 3 levels of ANN input (position, velocity, and acceleration), and 11 window sizes (spanning 33.3 msec to 750 msec). The between subjects factor was the developmental group, delayed versus typical. The significance level for the ANOVA was set at 0.05.

To evaluate whether a control effect was observed, the output of the network was compared with 0.5 for each group and condition combination. For a perturbation, whether it is position, velocity, or acceleration, the correct response is acceleration in the opposite direction to correct for the perturbation. Since we tested the network with a positive perturbation (either [1,0,0] for position, [0,1,0] for velocity, or [0,0,1] for acceleration), the correct response of the network is a value below 0.5, indicating that the acceleration in the next time step is in the opposite direction to the perturbation. Thus one-tailed, independent *t* tests were used test whether the output results were below 0.5. For each window size, infants with typical development and infants with delayed development, at early sitting and late sitting, in each of two axes (anterior-posterior and medial-lateral), are evaluated for the effect of three different types of posture control information (position, velocity and acceleration), resulting in $2 \times 2 \times 2 \times 3 = 24$ comparisons for each window size used. Because 11 window sizes were examined, 24*11= 264 conditions were tested. A conservative Bonferroni correction for multiple comparisons would require setting the $alpha_{critical}$ = 0.05/264 = 0.000189, which is quite difficult to meet. If we had knowledge of the one best window size for posture control in infants sitting, then only 24 conditions would have been examined, and $alpha_{critical}$ = 0.05/24 = 0.0021 would be used. Because of the exploratory nature of this work, we relaxed the criteria for significance from the Bonferroni standard, and we chose to examine two critical values, $alpha_{critical}$ = 0.01 and $alpha_{critical}$ =0.0021. To get an idea of the effect of the relaxed criteria, using an $alpha_{critical}$ value of 0.01 means we expect to reject the null hypothesis when in fact it is true for 1% of the comparisons. For 264 comparisons, we expect about 0.01*264 = 2.64 comparisons to appear as significant, even if the results are actually random. Similarly, for $alpha_{critical}$ = 0.0021, we expect about 0.6 comparisons to be evaluated as significant when in fact they are not.

Results

The repeated-measures ANOVA analysis did not reveal any significant differences for group, nor did it find a main effect for the repeated measures, time, axis, perturbation type, or window size. However, within subject contrasts found significant interactions in perturbation type x group ($p = .044$), window size x axis ($p = .034$), window size x day x axis ($p = .041$), window size x axis x perturbation type x group ($p = .019$), and window size x time x axis x perturbation type x group ($p = .014$), where the p value shown represents the best p value for each type of contrast (i.e., lowest among linear, quadratic, etc). Note that the last interaction with all 5 conditions and group is significant, and has the lowest p value, so there is no simple interpretation of these results, as all interactions must be considered.

To help interpret the interactions, there is an additional consideration about the results that will be helpful, namely the comparison of the neural network output to the neutral value of 0.5 for each condition. As described previously, the output of the network is a normalized acceleration, with a value ranging from 0 to 1, where a value of 0 indicates a maximum acceleration in the negative direction, a value of 1 indicates a maximum acceleration in the positive direction, and a value of 0.5 indicates no acceleration in response to the input. If the network is tested with a positive perturbation, the appropriate response is in the negative direction, i.e., in the opposite direction to the perturbation, which corresponds to an output significantly less than 0.5 If the output of the network for a positive perturbation is not significantly less than 0.5 for that time lag and input perturbation type (i.e., position, velocity, or acceleration), that indicates that the time lag/information type combination is not contributing significantly to control. To statistically test this, one-sample *t* tests were used to compare the output for each condition to 0.5, for inputs designed to test the trained networks sensitivity to position [1,0,0], velocity [0,1,0], and acceleration [0,0,1] (Table 1). The tests were two-tailed *t* tests, but the mean values for all conditions that are significantly different than 0.5 are less than 0.5, consistent with the output of the network having useful function for control. Using a criteria of statistical significance of 0.01, 44 conditions/group combinations were found to be significantly lower than 0.5, out of 264 tested, compared with only about 3 combinations would be expected to be significantly different if

Table 1 Results of One Sample t-Tests with the Output of the ANN Less than the Neutral Value of 0.5

ANN input	Axis	Time	Window (msec)	Mean	Standard Deviation	*p*
		Typical development ($n = 33$)				
Position	medial-lateral	late sitting	33.3	0.448	0.116	0.0076
Position	medial-lateral	early sitting	83.3	0.412	0.155	0.0014*
Velocity	medial-lateral	early sitting	83.3	0.407	0.173	0.0021*
Acceleration	medial-lateral	early sitting	83.3	0.410	0.164	0.0018*
Velocity	anterior-posterior	late sitting	83.3	0.431	0.129	0.0023
Position	anterior-posterior	early sitting	133.3	0.431	0.157	0.0084
Position	medial-lateral	early sitting	133.3	0.411	0.189	0.0053
Velocity	medial-lateral	early sitting	133.3	0.411	0.198	0.0074
Acceleration	medial-lateral	early sitting	133.3	0.406	0.191	0.0041
Position	medial-lateral	late sitting	133.3	0.430	0.150	0.0056
Velocity	medial-lateral	late sitting	133.3	0.425	0.166	0.0070
Acceleration	medial-lateral	late sitting	133.3	0.425	0.154	0.0042
Position	medial-lateral	early sitting	187.5	0.410	0.161	0.0016*
Velocity	medial-lateral	early sitting	187.5	0.423	0.160	0.0046
Acceleration	medial-lateral	early sitting	187.5	0.418	0.174	0.0053
Position	medial-lateral	late sitting	250.0	0.438	0.131	0.0053
Acceleration	medial-lateral	late sitting	250.0	0.433	0.152	0.0080
Position	anterior-posterior	early sitting	500.0	0.414	0.153	0.0015*
Velocity	anterior-posterior	early sitting	500.0	0.398	0.172	0.0009*
Acceleration	anterior-posterior	early sitting	500.0	0.391	0.176	0.0006*
Position	medial-lateral	early sitting	500.0	0.412	0.186	0.0054
Velocity	medial-lateral	early sitting	500.0	0.405	0.178	0.0021
Velocity	anterior-posterior	late sitting	500.0	0.423	0.174	0.0082
Position	medial-lateral	late sitting	750.0	0.418	0.154	0.0022
Velocity	medial-lateral	late sitting	750.0	0.418	0.166	0.0040
		Delayed development ($n = 30$)				
Position	medial-lateral	early sitting	133.3	0.420	0.125	0.0008*
Velocity	anterior-posterior	early sitting	187.5	0.406	0.169	0.0025
Acceleration	anterior-posterior	early sitting	187.5	0.419	0.172	0.0075
Acceleration	anterior-posterior	early sitting	250.0	0.428	0.152	0.0070
Velocity	anterior-posterior	late sitting	250.0	0.416	0.167	0.0050
Acceleration	anterior-posterior	late sitting	250.0	0.420	0.158	0.0046
Position	medial-lateral	late sitting	250.0	0.413	0.178	0.0060
Velocity	medial-lateral	late sitting	250.0	0.413	0.179	0.0063
Velocity	anterior-posterior	early sitting	333.3	0.409	0.194	0.0081
Acceleration	anterior-posterior	early sitting	333.3	0.409	0.177	0.0043
Position	medial-lateral	early sitting	375.0	0.410	0.161	0.0023
Acceleration	medial-lateral	early sitting	375.0	0.410	0.163	0.0025
Acceleration	anterior-posterior	late sitting	375.0	0.415	0.153	0.0025
Position	anterior-posterior	early sitting	500.0	0.424	0.124	0.0011*
Acceleration	anterior-posterior	late sitting	500.0	0.432	0.142	0.0068
Velocity	anterior-posterior	early sitting	750.0	0.405	0.171	0.0024
Position	medial-lateral	late sitting	750.0	0.407	0.189	0.0058
Velocity	medial-lateral	late sitting	750.0	0.403	0.160	0.0012*
Acceleration	medial-lateral	late sitting	750.0	0.396	0.174	0.0014*

Note: Only conditions with $p < .01$ are included in the table, and * indicates conditions with $p < .0021$. The p values are for a one-sided t test with null hypothesis mean = 0.5 for each condition/ANN input combination. Comparisons with $p > .01$ are not shown.

the results were random. Using a criterion of statistical significance of 0.0021, 11 conditions/group combinations were found to be significantly lower than 0.5, out of 264 tested, compared with only about 1 combination that would be expected to be significantly different if the results were random.

The significant results (Table 1) were organized by group, day, and axis (Table 2) to facilitate comparisons. Typically developing infants have a wide range of time windows contributing to control in the medial-lateral axis. In addition, position, velocity and acceleration are all contributing to control in the medial-lateral axis for infants with typical development. In contrast, the anterior-posterior axis for late sitting for infants with typical development is very dependent on velocity information. The infants with delayed development have no short time window contributions to control, as there are no significant contributions from time windows less that 100 msec for infants with delayed development, and for late sitting no significant contribution from a time window less that 250 msec.

Table 2 Information Type and Window Times (msec) for Significant Output of Infant Sitting ANN

Delayed development		Typical development	
Medial-lateral	**Anterior-posterior**	**Medial-lateral**	**Anterior-posterior**
	Early Sitting		
P 133*	P 500*	P 83*	P 133
P 375	V 187	P 133	P 500*
A 375	V 333	P 187*	V 500*
	V 750	P 500	A 500*
	A 187	V 83*	
	A 250	V 133	
	A 333	V 187	
		V 500	
		A 83*	
		A 133	
		A 187	
	Late Sitting		
P 250	V 250	P 33	V 83
P 750	A 250	P 133	V 500
V 250	A 375	P 250	
V 750*	A 500	P 750	
A 750*		V 133	
		V 750	
		A 133	
		A 250	

Note. P = position sensitivity of ANN, V = velocity sensitivity of ANN, A = acceleration sensitivity of ANN. Numerical value is window size in msec. * indicates ANN output was significantly different from 0.5 with $p < 0.0021$, and values without * were significantly different from 0.5 with $p < 0.01$, as indicated in Table 1.

In addition, infants with delayed development have more equal lag/information types contributing to control for the anterior-posterior axis and medial-lateral axis, compared with the infants with delayed development who have more in the medial-lateral axis and fewer in the anterior-posterior axis.

Discussion

Our first hypothesis was that velocity information would be more heavily used in infant sitting posture control. We found this to be true, but only for infants with typical development, and then only for control in the anterior-posterior axis for late sitting. That late sitting should use velocity information more heavily is consistent with the sensory integration capabilities of the infants becoming more nearly adult like later in development. In adult standing posture control, Jeka, Kiemel, Creath, Horak, and Peterka, (2004) find that velocity information is more heavily used than position or acceleration. They point out that the proprioceptive, cutaneous, and visual systems are all velocity sensitive, due to the sensor physiology being more sensitive to changes in position rather than absolute position. They mentioned that the vestibular system, a source of acceleration information, is relied on under conditions where sway referenced support has altered normal sensory input. They argue that under normal postural sway conditions, the vestibular system is likely not sensitive enough to contribute greatly to postural control. However, the study by Jeka, et al. (2004) only examined control in the anterior-posterior axis and not in the medial-lateral axis. Just because velocity is more heavily used for control of adult standing posture in the anterior-posterior axis, does not imply that the same is true in the medial-lateral axis, as sensory information is used differently for control in the two different axes. For example, a study by O'Connor and Kuo (2009) found that normal adult standing postural sway is more influenced by visual perturbations in the anterior-posterior axis than in the medial-lateral axis, while the sensitivity is higher in the medial-lateral direction if the feet are placed in tandem rather than side-by-side. As infants learn to sit they must learn to appropriately use sensory information based on task demands.

Our second hypothesis was that infants with developmental delay use sensory information differently than infants with typical development. The infants with developmental delay were found to lack the short time delay contributions to posture control that the infants with typical development demonstrated. Infants with developmental delay were found to not simply be delayed in the development of sitting, but were less able to use short latency sensory information in postural control than infants with typical development, instead relying on longer delay time mechanisms for postural control. One short delay time control mechanism that might be used in postural control is the stretch reflex (Granata, Slota, & Bennett, 2004). Infants with spastic cerebral palsy have altered stretch reflex activity and greater stiffness of the musculoskeletal system, and thus this mechanism may not be as useful for postural control for infants with cerebral palsy compared with infants with typical development. Perhaps an adaptive strategy for maintaining upright posture for infants with altered short latency control, possibly altered stretch reflexes, is a more complete reliance on higher level control mechanisms, which necessarily have a longer delay time. With a reduced number of postural control strategies available, the motor control system has fewer synergies to invoke, so the motor control development becomes atypical as well.

Children with cerebral palsy have been found to have an increased time to produce a given amount of force in lower extremity movements (Downing, Ganley, Fay, & Abbas, 2009), and adults with dystonia have slower reaction times in a visual stimulus and button-pushing task (Jahanshahi, Rowe, & Fuller, 2001). The slow response time of the neuromuscular control system, and the necessary reliance on longer time lag control mechanisms, has important implications for postural control. One model of postural control is the inverted pendulum model, where a mass remains positioned above the ground on a vertical rod due to actuators controlled by a feedback controller. If the delay time of the feedback controller exceeds a critical time delay, then the upright position cannot be maintained. The critical time is given by: t_c= sqrt(2*L/3*g), where L is the distance from ground to the center of mass of the pendulum, and g is the acceleration of gravity, which works out to 260 msec for adult standing (Milton et al. 2009) . From this formula, the critical delay time for control of an inverted pendulum depends on the size of the pendulum, with taller pendulums able to be controlled using slower response times. For an infant, with a center of mass about 20 cm above the ground, the critical control time is 117 msec. None of the significant control time delays for infants with delayed development meet this criterion (Table 2). While the inverted pendulum is a very crude model of infant sitting postural control (Kyvelidou, Stuberg, Harborne, Deffeyes, Blanke, & Stergiou, 2009), and ignores what are likely important contributions from the viscoelastic properties of the infant's body as well as the pelvis and spine joints, the inverted pendulum model suggests that an infant who is not able to use fast latency control mechanisms may have a more difficult control problem to solve than infants with typical development.

This study investigated control of normal postural sway, where no external mechanical perturbations are applied, and no sensory alteration is used. While an important feature of this study is that the results apply to normal, unperturbed posture control with normal sensory weighting, a weakness of this study is that the specific sensory modalities involved in estimation of position, velocity, and acceleration cannot be identified. This study used a very simple ANN to model postural control, which is a complicated control process with multiple control mechanisms interacting to maintain upright posture (Horak, 2006). The ANN topology might be improved by inputting position, velocity and acceleration information for multiple time delays information simultaneously (larger ANN input layer), or by having more processing nodes (larger ANN hidden layer), or by having output to multiple muscles with various different time delays (larger ANN output layer). The probes that we use to test the network sensitivity to position, velocity, and acceleration were also very simple, but more complex, nonlinear combinations of inputs might be important for posture control, as might be expected if the time-to-contact hypothesis (Slobounov, Cao, Jaiswal, & Newell, 2009) is correct. A combination of velocity and acceleration may also be useful for infant sitting postural control, as muscle activity in adult standing postural control has been shown to correlate with perturbation acceleration and velocity (Welch & Ting, 2009). Additional work is needed to address these issues.

Dynamic system theory, as used in the field of developmental psychology, accepts that an important aspect of motor development is the development of perception-action coupling, as a result of exploring a wide variety of coordination patterns, and eventually selecting those best suited to a particular motor task. Thelen (2000) has emphasized the close relationship between cognition and action-perception during development. An important aspect of perception is the cognitive task of sensory integration that must occur to use the information content of the sensory input. Visual, vestibular, proprioceptive, and cutaneous sensory data must be integrated to estimate position, velocity, and acceleration information to be used for posture control. Although there is no theoretical guidance on whether position, velocity, or acceleration information would be most useful for postural control, work with adult standing anterior-posterior postural control indicates that velocity information is most useful (Jeka, Kiemel, Creath, Horak, & Peterka, 2004), and we have noted in this work that infants with typical development use velocity information more heavily in posture control in the anterior-posterior direction. Thus the infants with typical development appear to develop toward using sensory information in a manner similar to adult posture, with the underlying assumption that the infant is developing on a trajectory that will eventually led to the adult pattern of use of sensory information. However, this analysis may be overly simplistic. There is no reason to assume a linear trajectory in infant development (Adolph, Robinson, Young, & Gill-Alvarez, 2008). Development of proprioceptive sensory integration is not mature even in adolescents (Viel, Vaugoyeau, & Assaiante, 2009), so attainment of a fully adult response in infants, even in later sitting, is not likely. Instead, the use of velocity information for control in the anterior-posterior direction may emerge independently in both infant sitting and adult standing, as an efficient means of control for those particular postures, given the anatomical and physiological constraints of each of those systems. In discussing the anterior-posterior and medial-lateral differences in sensory information utilization in adult standing, O'Connor and Kuo (2009) stated that the task direction with the greatest instability requires more feedback, and applying this logic to our results suggests that control in the medial-lateral axis is less stable than the anterior-posterior axis, as more types of sensory information are used for control in that axis, for infants with typical development.

In summary, we find that late sitting for infants with typical development is characterized by a high reliance on velocity information in control in the anterior-posterior axis, as is adult standing posture control (Jeka, Kiemel, Creath, Horak, & Peterka, 2004), with relatively more complicated control in the medial-lateral axis utilizing a wider range of information types. Infants with delayed development did not show the same reliance on velocity information within the time limits of our study, although this may occur later in development. Infants with delayed development have less reliance on short latency control mechanisms compared with infants with typical development, perhaps due to altered stretch reflexes or generally slower sensorimotor dynamics, necessitating an adaptive switch to other longer latency control mechanisms.

Acknowledgments

This work was supported by NIH (K25HD047194), NIDRR (H133G040118), the Nebraska Research Initiative, the University of Nebraska Presidential Graduate Fellowship, grant T73MC00023 from the Maternal and Child Health Bureau, Health Resources and Services Administration, Department of Health and Human Services and in part by grant 90DD0601 from the Administration on Developmental Disabilities (ADD), Administration for Children and Families, Department of Health and Human Services. The authors would also like to acknowledge the assistance of Stacey DeJong and Anastasia Kyvelidou in data collection.

References

Adolph, K.E., Robinson, S.R., Young, J.W., & Gill-Alvarez, F. (2008). What is the shape of developmental change? *Psychological Review, 115*(3), 527–543.

Ali, A.S., Rowen, K.A., & Iles, J.F. (2003). Vestibular actions on back and lower limb muscles during postural tasks in man. *The Journal of Physiology, 546*(Pt 2), 615–624.

Bumin, G., & Kavak, S.T. (2008). An investigation of the factors affecting handwriting performance in children with hemiplegic cerebral palsy. *Disability and Rehabilitation, 30*(18), 1374–1385.

Bumin, G., & Kayihan, H. (2001). Effectiveness of two different sensory-integration programmes for children with spastic diplegic cerebral palsy. *Disability and Rehabilitation, 23*(9), 394–399.

Chen, J., & Woollacott, M.H. (2007). Lower extremity kinetics for balance control in children with cerebral palsy. *Journal of Motor Behavior, 39*(4), 306–316.

Downing, A.L., Ganley, K.J., Fay, D.R., & Abbas, J.J. (2009). Temporal characteristics of lower extremity moment generation in children with cerebral palsy. *Muscle & Nerve, 39*(6), 800–809.

Duda, R.O., Hart, P.E., & Stork, D.G. (2001). *Pattern Classification* (2nd ed.). New York: John Wiley & Sons.

Fedrizzi, E., Facchin, P., Marzaroli, M., Pagliano, E., Botteon, G., Percivalle, L., et al. (2000). Predictors of independent walking in children with spastic diplegia. *Journal of Child Neurology, 15*(4), 228–234.

Folio, M.R., & Fewell, R.R. (2000). *Peabody Developmental Motor Scales (2nd ed.).* Austin, TX: Pro-ed, Inc.

Goble, D.J., Hurvitz, E.A., & Brown, S.H. (2009). Deficits in the ability to use proprioceptive feedback in children with hemiplegic cerebral palsy. *International Journal of Rehabilitation Research. Internationale Zeitschrift fur Rehabilitationsforschung. Revue Internationale de Recherches de Readaptation, 32*(3), 267–269.

Granata, K.P., Slota, G.P., & Bennett, B.C. (2004). Paraspinal muscle reflex dynamics. *Journal of Biomechanics, 37*(2), 241–247.

Hadders-Algra, M., van der Fits, I.B., Stremmelaar, E.F., & Touwen, B.C. (1999). Development of postural adjustments during reaching in infants with CP. *Developmental Medicine and Child Neurology, 41*(11), 766–776.

Harbourne, R.T., Giuliani, C., & Neela, J.M. (1993). A kinematic and electromyographic analysis of the development of sitting posture in infants. *Developmental Psychobiology, 26*(1), 51–64.

Hedberg, A., Carlberg, E.B., Forssberg, H., & Hadders-Algra, M. (2005). Development of postural adjustments in sitting position during the first half year of life. *Developmental Medicine and Child Neurology, 47*(5), 312–320.

Hedberg, A., Forssberg, H., & Hadders-Algra, M. (2004). Postural adjustments due to external perturbations during sitting in 1-month-old infants: evidence for the innate origin of direction specificity. *Experimental Brain Research, 157*(1), 10–17.

Hirschfeld, H., & Forssberg, H. (1994). Epigenetic development of postural responses for sitting during infancy. *Experimental Brain Research, 97*(3), 528–540.

Hoon AH, J.r., Stashinko, E.E., Nagae, L.M., Lin, D.D., Keller, J., Bastian, A., Campbell, M.L., Levey, E., Mori, S., & Johnston, M.V. (2009). Sensory and motor deficits in children with cerebral palsy born preterm correlate with diffusion tensor imaging abnormalities in thalamocortical pathways. *Developmental Medicine and Child Neurology, 51*(9), 697–704.

Horak, F.B. (2006). Postural orientation and equilibrium: what do we need to know about neural control of balance to prevent falls? *Age and Ageing, 35*(S2), ii2–ii11.

Jahanshahi, M., Rowe, J., & Fuller, R. (2001). Impairment of movement initiation and execution but not preparation in idiopathic dystonia. *Experimental Brain Research, 140*(4), 460–468.

Jeka, J., Kiemel, T., Creath, R., Horak, F., & Peterka, R. (2004). Controlling human upright posture: velocity information is more accurate than position or acceleration. *Journal of Neurophysiology, 92*(4), 2368–2379.

Kandel, E.R., Jessell, T.M., & Schwartz, J.H. (2000). *Principles of Neural Science* (4th ed.). New York: McGraw-Hill.

Kiemel, T., Oie, K.S., & Jeka, J.J. (2006). Slow dynamics of postural sway are in the feedback loop. *Journal of Neurophysiology, 95*(3), 1410–1418.

Kyvelidou, A., Stuberg, W.A., Harbourne, R.T., Deffeyes, J.E., Blanke, D., & Stergiou, N. (2009). Development of upper body coordination during sitting in typically developing infants. *Pediatric Research, 65*(5), 553–538.

Lesny, I., Stehlik, A., Tomasek, J., Tomankova, A., & Havlicek, I. (1993). Sensory disorders in cerebral palsy: two-point discrimination. *Developmental Medicine and Child Neurology, 35*(5), 402–405.

Milton, J., Cabrera, J.L., Ohira, T., Tajima, S., Tonosaki, Y., Eurich, C.W., et al. (2009). The time-delayed inverted pendulum: implications for human balance control. *Chaos (Woodbury, N.Y.), 19*(2), 026110.

O'Connor, S.M., & Kuo, A.D. (2009). Direction-dependent control of balance during walking and standing. *Journal of Neurophysiology, 102*(3), 1411–1419.

Peterka, R.J. (2002). Sensorimotor integration in human postural control. *Journal of Neurophysiology, 88*(3), 1097–1118.

Sanger, T.D., & Kukke, S.N. (2007). Abnormalities of tactile sensory function in children with dystonic and diplegic cerebral palsy. *Journal of Child Neurology, 22*(3), 289–293.

Schouten, A.C., de Vlugt, E., van Hilten, J.J., & van der Helm, F.C. (2008). Quantifying proprioceptive reflexes during position control of the human arm. *IEEE Transactions on Bio-Medical Engineering, 55*(1), 311–321.

Slobounov, S., Cao, C., Jaiswal, N., & Newell, K.M. (2009). Neural basis of postural instability identified by VTC and EEG. *Experimental Brain Research, 199*(1), 1–16.

Thelen, E. (2000). Grounded in the World: Developmental Origins of the Embodied Mind. *Infancy, 1*(1), 3–28.

Thiel, A., Greschner, M., Eurich, C.W., Ammermuller, J., & Kretzberg, J. (2007). Contribution of individual retinal ganglion cell responses to velocity and acceleration encoding. *Journal of Neurophysiology, 98*(4), 2285–2296.

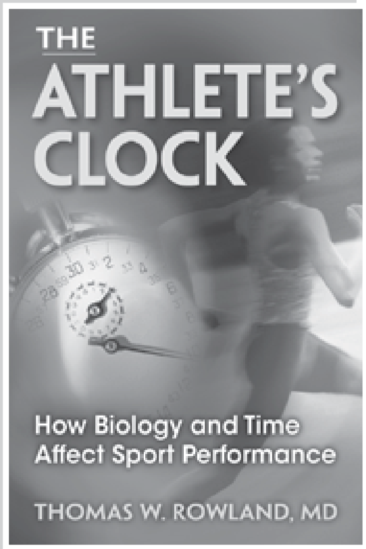
van den Heuvel, M.R., Balasubramaniam, R., Daffertshofer, A., Longtin, A., & Beek, P.J. (2009). Delayed visual feedback reveals distinct time scales in balance control. *Neuroscience Letters, 452*(1), 37–41.

Viel, S., Vaugoyeau, M., & Assaiante, C. (2009). Adolescence: a transient period of proprioceptive neglect in sensory integration of postural control. *Motor Control, 13*(1), 25–42.

Welch, T.D.J., & Ting, L.H. (2009). A feedback model explains the differential scaling of human postural responses to perturbation acceleration and velocity. *Journal of Neurophysiology, 101,* 3294–3309.

Wilke, M., & Staudt, M. (2009). Does damage to somatosensory circuits underlie motor impairment in cerebral palsy? *Developmental Medicine and Child Neurology, 51*(9), 686–687.

Study the effects of time on sport performance



©2011 • Paperback • 224 pp
Print: ISBN 978-0-7360-8274-7
E-book: ISBN 978-0-7360-8765-0

eBook format also available

Author Interview

Find out more about these intriguing topics by watching one of our video interviews with author Thomas Rowland at **www.HumanKinetics.com**.

Audiences: *A text for students in exercise physiology and other disciplines within kinesiology; a resource for sport science researchers and academic libraries as well as athletes and coaches seeking information on improving sport performance through an understanding of the physiological factors that affect athletes.*

The Athlete's Clock: How Biology and Time Affect Sport Performance offers an engaging, interdisciplinary consideration of some of the most compelling questions in sport and exercise science. This unique text takes a broad look at the physiological clock, offering students, researchers, coaches, and athletes a unique approach to understanding how various aspects of time affect sport performance.

The Athlete's Clock explores ways in which time and its relationship to athletic effort optimize sport performance. Readers can investigate challenging questions such as these:

- What is the optimal time of day for training?
- If a coach thinks that a high stroke count leads to a better time in a particular swim event, should the athlete go with it? Or is it better to stick to a more intuitively normal cadence?
- Do athletes consciously control sport performance, or are athletes under the control of unconscious processes within the central nervous system?
- When and how can young, promising athletes be safely and successfully nurtured to greatness?

Exceptionally readable, Rowland's intelligent insights and humor provide an engaging discovery of a unique and wide-ranging topic. *The Athlete's Clock* is sure to bring readers closer to both an understanding of and appreciation for the intricacies of human potential.

For a complete description or to order, visit **www.HumanKinetics.com** or call:
(800) 747-4457 US • (800) 465-7301 CDN • 44 (0) 113-255-5665 UK
(08) 8372-0999 AUS • 0800-222-062 NZ • (217) 351-5076 International

HUMAN KINETICS
The Information Leader in Physical Activity & Health

1219 3/11