Developmental Science

Developmental Science 18:4 (2015), pp 622–634 DOI: 10.1111/desc.12238

PAPER

A model to investigate the mechanisms underlying the emergence and development of independent sitting

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Abstract

When infants first begin to sit independently, they are highly unstable and unable to maintain upright sitting posture for more than a few seconds. Over the course of 3 months, the sitting ability of infants drastically improves. To investigate the mechanisms controlling the development of sitting posture, a single-degree-of-freedom inverted pendulum model was developed. Passive muscle properties were modeled with a stiffness and damping term, while active neurological control was modeled with a time-delayed proportional-integral-derivative (PID) controller. The findings of the simulations suggest that infants primarily utilize passive muscle stiffness to remain upright when they first begin to sit. This passive control mechanism allows the infant to remain upright so that active feedback control mechanisms can develop. The emergence of active control mechanisms allows infants to integrate sensory information into their movements so that they can exhibit more adaptive sitting.

Research highlights

- A mathematical model of infant sitting was used to examine the underlying mechanisms contributing to the emergence and maturation of infant sitting.
- The model predicts that newly-sitting infants rely mainly on passive muscle properties to remain upright.
- The model also predicts that upright sitting becomes more stable as infants learn to properly integrate and coordinate active neuromuscular feedback with the appropriate muscle responses.

Introduction

Independent sitting is a critical postural milestone that leads to the eventual development of stance and locomotion (Adolph & Joh, 2007; Thelen, 2000). The development of sitting and other postural milestones results from an improved ability to detect, interpret, and integrate sensory feedback information (i.e. visual, vestibular, and somatosensory) so that corrective movements can be generated within a reasonable time delay (Assaiante & Amblard, 1995; Boker, 2001; McCollum & Leen, 1989). For example, with maturation, infants exhibit improved organization of motor responses (Dusing & Harbourne, 2010; Forssberg & Hirschfeld, 1994; Hadders-Algra, Brogren & Forssberg, 1996; Harbourne, Giuliani & Neela, 1993; Hirschfeld & Forssberg, 1994; Thelen & Spencer, 1998; Woollacott & Shumway-Cook, 1990), the ability to properly coordinate the body's various degrees of freedom (Harbourne & Stergiou, 2003; Latash, Krishnamoorthy, Scholz & Zatsiorsky, 2005), improved sensorimotor integration (Bertenthal, Boker & Xu, 2000; Bertenthal, Rose & Bai, 1997), and a reduced time delay between the detection of sensory stimulation and the initiation of a motor response (Harbourne *et al*., 1993).

Passive mechanisms such as muscle tone are also important in the development of sitting and other

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postural milestones. Muscle tone is the muscle's resistance to displacement and velocity of stretch (Grillner, 1972; Loram & Lakie, 2002; Tokuno, Garland, Carpenter, Thorstensson & Cresswell, 2008; Winter, 1995). A high muscle tone, resulting in a more 'stiff' musculature, provides passive force that could be used to support body weight. However, muscle tone that is too high impairs the ability to perform coordinated goal-directed movements and limits the adaptability of the postural system. In general, muscle tone is referred to as passive because it does not require processing of sensory information from the higher levels of the nervous system (Winter, Patla, Prince, Ishac & Gielo-Perczak, 1998). The contribution of muscle tone to the control of posture has been well studied in standing adults (Winter *et al*., 1998). In fact, some research has suggested that muscle tone alone is sufficient for the maintenance of upright stance (Winter *et al*., 1998).

Interestingly, the contribution of passive control to the development of motor milestones in typically-developing children has not been well studied. Rather, muscle tone is typically only considered in children with developmental delays (de Vries & de Groot, 2002; Hadders-Algra, 2001; Prasad & Prasad, 2003). Thus it appears, in the absence of pathology, the dominant belief is that muscle tone requires little development and is appropriately set and regulated by the upper motor neurons. However, as seen in both hypo- and hypertonic children, too little or too much muscle tone hinders development and the attainment of motor milestones (Papavasiliou, Rapidi, Filiopoulos, Rizou & Skouteli, 2006; Prasad & Prasad, 2003; Shuper, Weitz, Varsano & Mimouni, 1987). Learning to appropriately set muscle tone is likely an important, overlooked mechanism contributing to the development of sitting. Given that newly-sitting infants exhibit non-adaptive and generalized postural movements (Assaiante, 1998; Chen, Metcalfe, Jeka & Clark, 2007; Hadders-Algra, 2002, 2005, 2010; Hadders-Algra *et al*., 1996), it is possible that early sitting is controlled primarily from passive mechanisms such as muscle tone. Passive mechanisms would resist deviations from an equilibrium position, providing infants with sitting experience, which is needed before they can develop and learn to utilize more advanced feedback mechanisms. Although passive mechanisms can be used to maintain posture, more adaptive postural control requires sensorimotor integration so that infants can learn to scale the activation of the appropriate musculature and remain upright while performing various goal-directed movements (e.g., Saavedra, van Donkelaar & Woollacott, 2012).

Given the complexity associated with independent sitting, it is not surprising that its development occurs in multiple stages over a relatively protracted period of approximately 3 months (Harbourne & Stergiou, 2003). Although past studies have provided important information regarding the characteristics associated with the emergence, maturation, and improvements of independent sitting, in general, the specific mechanisms that lead to these developments remain poorly understood. This is largely due to the difficulty of studying neuromuscular mechanisms with non-invasive experimental techniques. Mathematical modeling offers a promising research pathway to identify the specific neuromuscular mechanisms contributing to the development of sitting.

In adults, mathematical models have been extensively used to determine the mechanisms which govern the control of upright stance, as well as the balance problems that arise with age and disease (Corradini, Fioretti, Leo & Piperno, 1997; Hemami, Weimer, Robinson, Stockwell & Cvetkovic, 1978; Maurer & Peterka, 2005; Peterka, 2000, 2002; Welch & Ting, 2009). Standing posture is typically modeled as an inverted pendulum, where a mass (representing the center of mass) is balanced at some distance above the base of support. A corrective torque, generated through both active and passive control mechanisms, is applied so that (internal or external) perturbations to upright equilibrium are attenuated. In simulations, disturbances to equilibrium are applied to the pendulum in the form of either a continuous series of white noise, or by orientating the pendulum at some angle relative to gravity. The passive stiffness and damping of the pendulum represents muscle tone. Active control (the ability to properly respond to both expected and unexpected environmental stimuli through information gathered by the sensory systems) is modeled utilizing a time-delayed proportional-integral-derivative (PID) controller. A PID controller is a control mechanism that utilizes feedback information to assess the difference between the current postural state and the desired postural position. For example, consider a person trying to remain upright but is oriented at a body angle of 5 degrees relative to the upright. This 5-degree offset would be represented as an error in the intended position and a scaled corrective response would be generated. Learning to scale the appropriate corrective response is essential to development since any over- or under-correction could ultimately prove to be destabilizing. In addition, extending the inverted pendulum model to infants may provide unique insights into how children learn to appropriately calibrate sensorimotor information as they learn to sit and adopt adaptive postural movements. Thus, the PID controller represents the sensorimotor integration necessary to detect possible threats to balance and generate appropriate motor responses (Masani, Vette & Popovic, 2006; Milton, Cabrera, Ohira, Tajima, Tonosaki, Eurich & Campbell, 2009; Vette, Masani & Popovic, 2007). Because feedback processing is necessary for active control, the PID controller acts at some finite time delay. This time delay represents the time it would take for sensory information to be processed in the nervous system and the appropriately-scaled muscle responses to be generated.

Although mathematical models have provided important insights into the control of standing posture in adults, their use has not been widely adopted to examine the development of infant motor milestones, including sitting. Thus, the purpose of this study was to develop a simple mathematical model to investigate the passive and active neuromuscular mechanisms that lead to the development of upright sitting. Specifically, an inverted pendulum model (designed to fit the anthropometrics of sitting infants) was used to examine how upright posture is attained from a non-upright starting position.

Methods

A single-degree-of-freedom, anterior-posterior model of infant sitting was adapted from the classic adult standing models (Maurer & Peterka, 2005). This model captures the forward and backward sway made by infants about the hip joint as they try to remain upright. Sitting balance was represented as a single-segment, inverted pendulum (Figure 1). A corrective torque (T_{hip}), the sum of the instantaneous passive and time-delayed active control processes $T_{hip} = T_{passive} + T_{active}$ was applied about the hip. Thus, muscular action through feedback-driven active muscle properties and passive muscle stiffness provided the force necessary to stabilize and adapt the modeled sitting behavior. Sitting stability was then assessed by examining movement of the center of mass (Equation 1):

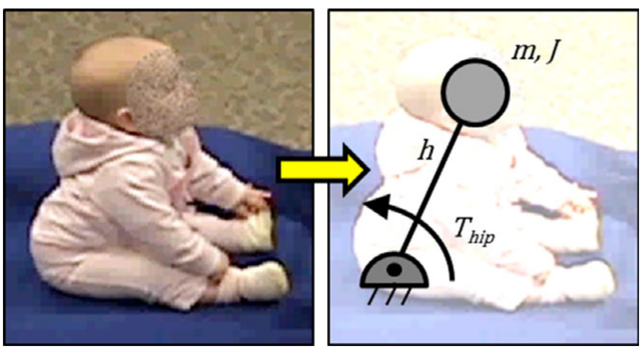

Figure 1 *The representation of a sitting infant as an inverted pendulum. Here, m represents the effective mass of infant's upper body, J represents the mass of moment of intertia of the infant's upper body about its hip joint, h defines the height of the infant's upper body center of mass about its hip joint, and T_{hip} represents the applied hip torque resulting from both passive and active mechanisms (i.e. the infant's skeletal, muscular, and nervous systems).*

$$\ddot{\theta} = \frac{1}{J}(mgh \sin\theta + T_{hip}) \tag{1}$$

Here, J is the moment of inertia about the hip, m is 60% of the total infant mass; the approximate proportion of mass above the hip joint in a newly-sitting infant (Webb Associates, Yellow Springs, Ohio. Anthropology Research Project, 1978), h is the height of the sitting center of mass, g is the local gravitational constant, θ is the angular orientation in the anterior-posterior direction, and $\ddot{\theta}$ is the angular acceleration. The values used for each of these parameters are provided in the following section.

The passive torque is the inherent stiffness and damping of the muscles. Stiffness is the resistance to passive stretch and the damping term effectively acts a shock absorber to attenuate kinetic energy. Collectively, these terms were used to represent the inherent properties of muscle tissue (Equation 2):

$$T_{passive} = -C_m\dot{\theta} - K_m\theta \tag{2}$$

Here, C_m is the muscle damping and K_m is the muscle stiffness. This torque, generated through passive processes, instantaneously changes as a function of the angular position θ and velocity $\dot{\theta}$. A small deviation in angular position and/or velocity results in a small passive torque; and as θ and $\dot{\theta}$ increase the magnitude of the torque also increases (Maurer & Peterka, 2005).

Active control is based on feedback from the vestibular, visual, and proprioceptive systems. Information from the perceptual systems needs to first be processed in the higher nervous system. Then, the appropriate motor action is determined and the motor commands are sent to the musculature controlling the sitting posture. This process takes a finite time to complete, subjecting the active term to an inherent time delay. The active controller is modeled using a time-delayed PID controller. A PID controller is a closed-loop feedback mechanism that is widely used in engineering to assess the difference between the instantaneous state of a variable (sitting orientation in the current project) and a desired setpoint. A PID controller is typically used to model human motor behavior because it nicely represents how movements are controlled based on feedback from the perceptual systems. The PID controller in the current study was represented by Equation 3:

$$T_{active} = -K_p[\theta(t-\tau) - \theta_{ref}] - K_i \int_0^t [\theta(\lambda - \tau) - \theta_{ref}]d\lambda - K_d\dot{\theta}(t-\tau) \quad (3)$$

where K_p is the proportional gain, K_i is the integral gain, K_d is the derivative gain, and τ is the time delay (Maurer & Peterka, 2005). These gains influence the infant's ability to reach the desired angular orientation θ_{ref} (zero when upright), as well as the inherent sway often observed when infants are learning to sit. The proportional gain K_p creates a proportional postural adjustment based on the difference between the intended and current posture (typically set to be upright). The derivative gain K_d impacts the rate of change of orientation and creates an adjustment that will prevent the system from over- or under-correcting. The integral gain K_i forces the system toward the desired set point, and reduces the difference between the desired output and the actual output in a way that accounts for historical (or previous) behaviors. All three parameters must be properly coordinated in order to create the optimal postural control system.

Model simulations

Anthropometric and control parameters (C_m, K_m, K_p, K_i, K_d, & τ) were adjusted from values previously used in adult literature to fit the anthropometric and neuromuscular properties of infants (Maurer & Peterka, 2005; Peterka, 2002). Since it is not appropriate to view infants as parametrically scaled down versions of adults, we chose to not just simply adjust adult model parameters by physical size. Rather, model parameters were non-dimensionalized using the moment of inertia J which is a function of both mass and center of mass height. This method allowed us to account for differences in body morphology and weight distribution that exist between infants and adults. Specifically, each adult parameter was divided by the respective adult-scaled moment of inertia creating a ratio (e.g. $\frac{K_p}{J}$). This process was then repeated for the infant parameters, leaving the unknown parameter in variable form. Like terms were then set equal between adults and infants (e.g. $\frac{K_p}{J_{adult}} = \frac{K_p}{J_{infant}}$). By cross multiplying, the down-scaled infant parameter was calculated (e.g. $K_{p_{infant}} = J_{infant} * \frac{K_p}{J_{adult}}$). This procedure ensured that the underlying postural dynamics of sitting mimic the behavior of a validated model, while accounting for the clear differences that exist between a standing adult and a sitting infant.

Simulations were performed using Simulink in Matlab 7.10. Because the equation of motion used for this model contains an inherent time delay, ode45 with a variable step size was used in conjunction with a transport delay, in lieu of a delayed differential equations solver. Two different groups of simulations were performed. In the first group, the active control and passive control mechanisms were set to zero to examine how each mechanism individually contributes to the development of sitting. In the second group, sets of simulations were performed where each active and passive parameter was systematically varied between plus and minus 5, 10, 25, and 50% of the nominal value which was identified from adult parameters using the scaling procedure discussed above. If the simulation was still stable (was able to adopt the desired upright position) at plus or minus 50%, the parameter value was further increased or decreased until a loss of stability was observed. If the model became unstable before the largest parameter variations, no further simulations were run. Each simulation was run for 20 seconds with the initial orientation and angular velocity set to $\theta = 5$ degrees and $\dot{\theta} = 0$ degrees per second. The six parameters examined in the simulations were muscle stiffness and damping (K_m, C_m), each of the PID controller gains (K_p, K_i, K_d), and time delay (τ). In each simulation the anthropometrics were specified as follows: Mass $m = 4.76$ kg, COM height $h = 0.198$ m, and Moment of inertia $J = 0.9408$ kg·m^2. Anthropometric values were determined using data from a publication maintained on the National Institute of Standards and Technology (NIST) web page (Snyder, Spencer, Owings & Schneider, 1975). This information provides a detailed standard of typical infant morphology. The nominal (or baseline) values, determined after scaling previously-published adult values, for each passive control parameter were as follows: Muscle Stiffness K_m = 0.145 Nm/deg, and Muscle Damping C_m = 0.041 Nm•s/deg. The nominal values for the active control parameters were as follows: Proportional Gain K_p = 0.238 Nm/deg, Integral Gain K_i = 0.008 Nm/s•deg, and Derivative Gain K_d = 0.068 Nm•s/deg. The nominal value of the time delay τ was set to 171 ms.

Each simulation provided a time series of body angle which was determined as the angle of the center of mass relative to vertical. An angle of zero indicated an upright orientation, while a 90-degree angle indicated an orientation parallel to the ground. Each simulation started with the initial conditions listed above. Over the duration of the simulation, body angle would change as the model stabilized (or failed to stabilize). Systematically altering the passive and active control parameters allowed us to examine how each parameter contributed to the development of sitting.

In general, the magnitude of the initial postural correction resulted in an overshoot of the desired upright position (calculated as the difference between the reference angle and the minimum point in the initial correction). When the combination of passive and active parameters created a stable trajectory, the desired orientation was obtained over time, termed the settling time. Specifically, the settling time, as employed here, is the time required for the system to come within 5% of the final orientation. The postural dynamics were then characterized by quantifying the magnitude of the initial overshoot and the length of the settling time.

It is important to note that it is difficult to define the thresholds of overshoot and settling time associated with physical stability, as compared to the mathematical stability of the model. For example, some magnitude of these parameters may be physically destabilizing for one infant and not for another. Physiological properties such as maturation of the sensory and motor control systems, muscular strength, anthropometrics, and neural development can result in large between-infant differences regarding what is physically stable and what perturbation will ultimately result in physical instability. Thus, below we report the absolute and relative changes of these values where appropriate so that comparisons can be made.

Results

Removing active and passive control

When all gains in the PID controller are set to zero, sitting must be maintained by utilizing only passive control. Setting the PID gains to zero means that no postural corrections are made using feedback-driven sensory processes. Thus, when the PID controller gains are set to zero, sitting must be maintained utilizing only the muscle stiffness. When the simulation is run without active control, body orientation slowly changes until it reaches approximately 45 degrees. This orientation is the static equilibrium, where the muscle stiffness is equal to the influence of the anthropometrics and position. In contrast, when the PID controller gains are set to their nominal values, body orientation quickly reaches the desired upright position (Figure 2).

When the passive torque is removed, only active processes are utilized to adopt an upright sitting posture (Figure 3). With the system relying solely on active feedback, there was an approximately 239% increase in the magnitude of the initial overshoot. In addition, there were more corrective movements, and an increase in settling time (Figure 3). These results demonstrate that removing passive muscle properties significantly impairs the attainment of an upright sitting posture.

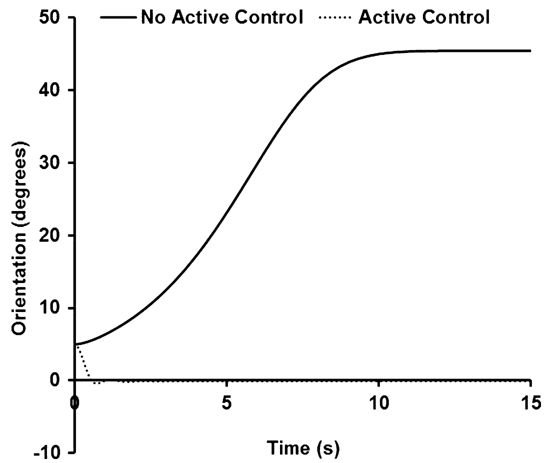

Figure 2 *Postural response when the PID gains (solid) are set to zero compared to when they are set to their nominal value (dotted line).*

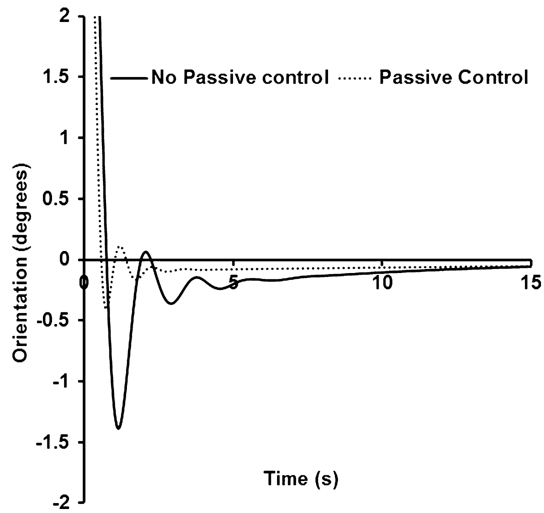

Figure 3 *Postural response when the gains of the passive controller are set to zero (solid line) compared to when they are set to their nominal value (dotted line).*

Because active feedback control occurs over a finite time, we also examined how removing this delay influenced the development of sitting. With no time delay, all postural corrections are made instantaneously and upright sitting is achieved with little overshoot (Figure 4). However, in the presence of a time delay, there is overshoot before an upright posture is adopted. Although zero time delay is not physiologically possible, comparing the zero and nominal time delays demonstrates the importance of feedback processing time in the development of sitting.

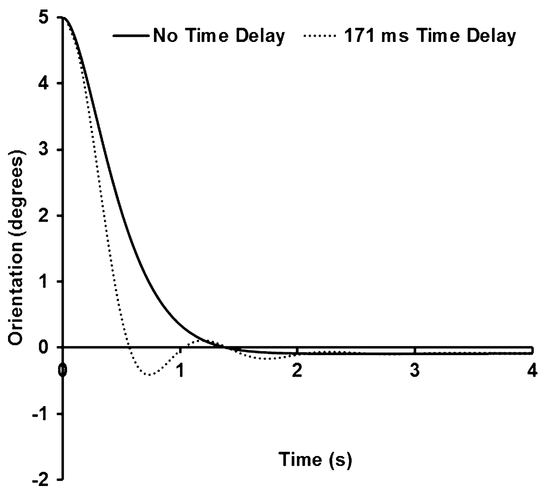

Figure 4 *Postural response when the time delay is set to zero (solid line) compared to when time delay is set to its nominal value (dotted line).*

Systematically varying control parameters

To examine how small- and large-scale changes in the control parameters can influence the development of sitting, each parameter was systematically altered from its nominal value. First, the passive parameters were varied while holding the active parameters constant. Then, the process was reversed. The results of the passive parameter manipulations are presented first and the active parameter manipulations are presented second. Simulations were first run where the parameters were varied by ±5 and 10%. Next, simulations were run where the parameter values were varied by ±25 and 50%. Variations of the proportional gain parameter were however different from what is listed above. Specifically, the proportional gain was varied by ±5 and 10%, and ±10 and 20%. This was done because the system became unstable at higher values. For most of the parameters, only the larger parameter variations resulted in qualitatively different sitting behaviors. The small and large variations are therefore presented separately. The percent change is reported between the extreme values of each set of variation (i.e. the percent difference between −10% and 10%, and −50 and +50%).

Passive control parameters

As the damping coefficient C_m increased from a 10% reduction to a 10% increase in the nominal value, the magnitude of the overshoot decreased by 65% and settling time decreased by 38%. In the variation from a 50% reduction to a 50% increase in the nominal value, there was a 100% decrease in overshoot, and 54% decrease in settling time. None of these manipulations resulted in the system becoming unstable (Figure 5).

As muscle stiffness increased from a 10% reduction to a 10% increase in the nominal value, the magnitude of the overshoot increased from 0.27 to 0.56 degrees and the settling time increased by 11% (Figure 6). Between −50% and +50% of the nominal value, the overshoot increased from 0 to 0.86 degrees, and the settling time increased 41% from 0.88 seconds to 1.24 seconds. Changes in the overshoot and settling times were small overall. Thus, neither the small- nor large-scale parameter variations resulted in the system becoming unstable. Muscle stiffness is therefore generally robust over a wide range of parameter values and, in fact, needs to be increased by 10 times its nominal value (1.4 Nm/deg) before the system destabilizes.

Although it is counterintuitive that an increased stiffness leads to an increase in overshoot and settling

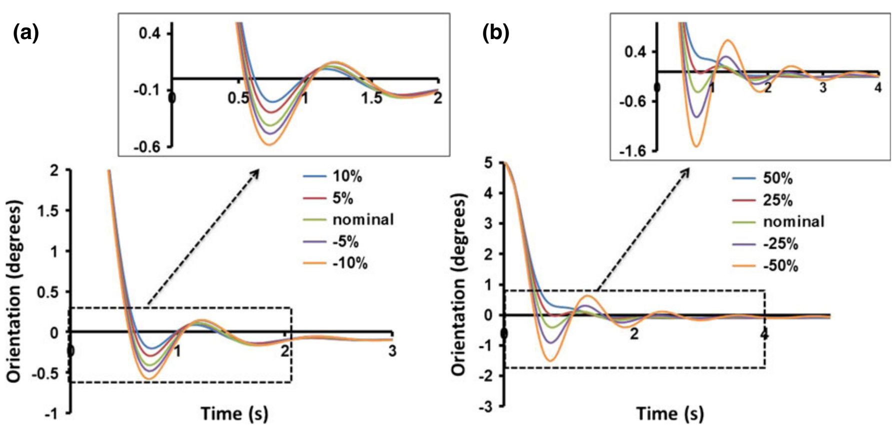

Figure 5 *Response trajectories when muscle damping gain was varied from (a) + 10 to − 10% of the nominal value and (b) + 50 to −50% of the nominal value.*

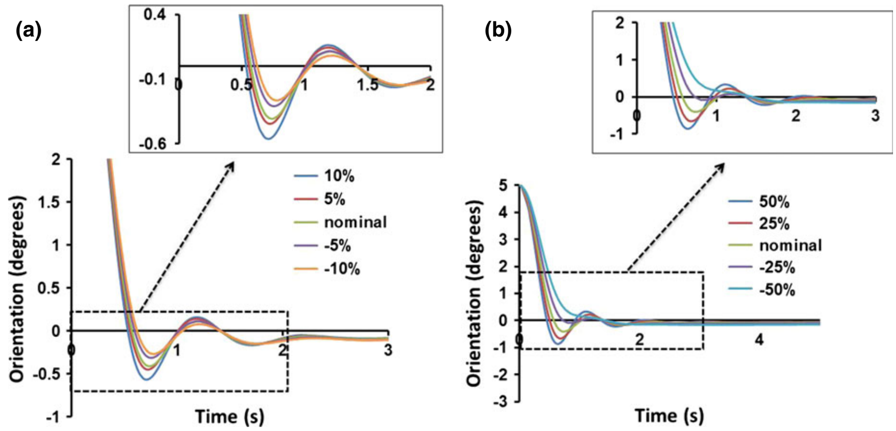

Figure 6 *Response trajectories when muscle stiffness gain was varied from (a) +10 to −10% of the nominal value and (b) +50 to −50% of the nominal value.*

time, it is important to note that these changes were small. Also, this finding emerges because of the way posture is mathematically represented in this system. Specifically, the influence of overall muscle tone can be represented via a damping ratio ($\frac{C_m}{2\sqrt{J(K_m - mgh)}}$), assuming a small angle). This ratio determines how quickly the oscillations will decay; in other words, how long it will take for the system to return to the equilibrium state. The damping ratio may better represent muscle tone than either the damping or stiffness terms, when taken independently, as muscle tone is ultimately the combination of both stiffness and damping within the muscle.

Active control parameters

As proportional gain K_p increased, the magnitude of the overshoot increased. The smaller variation range (−10% to +10%) resulted in a 500% increase in overshoot and a 46% increase in settling time (Figure 7). Because the orientation of the system changed rapidly, the proportional gain could only be varied by ±20% from the nominal value. Between −20% and +20% of the nominal value, the overshoot increased from 0 to 1.13 degrees, while the settling time increased by 27% (Figure 7). The active parameters have a greater impact on the behavior of the system with smaller deviations from the nominal value. Increasing K_p to 0.523 Nm/deg (220% of the nominal value) will force the system to destabilize.

Changing the derivative gain from −10% to +10% of the nominal value resulted in a 77% decrease in overshoot and a 47% decrease in settling time (Figure 8). When the range of values was −50% to +50% of the nominal value, there was no overshoot and a 57% reduction in settling time (Figure 8). At 0.164 Nm•s/deg, approximately 2.5 times the nominal value, the response will change stability.

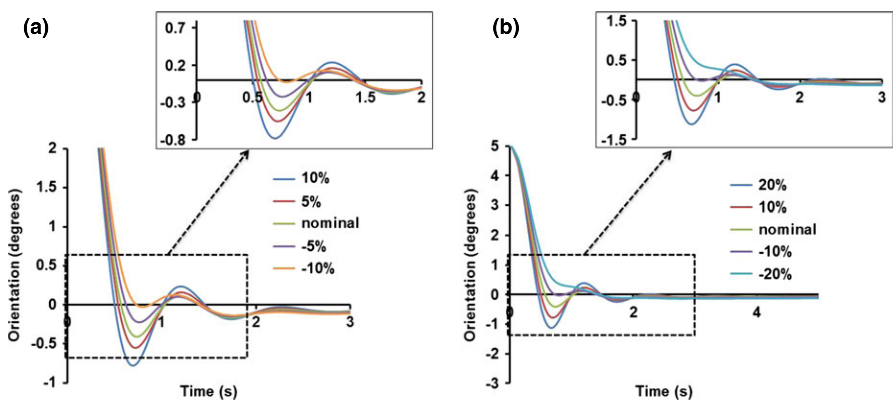

Figure 7 *Response trajectories when the proportional gain (P component of the PID controller) was varied from (a) +10 to −10% of the nominal value and (b) +20 to −20% of the nominal value.*

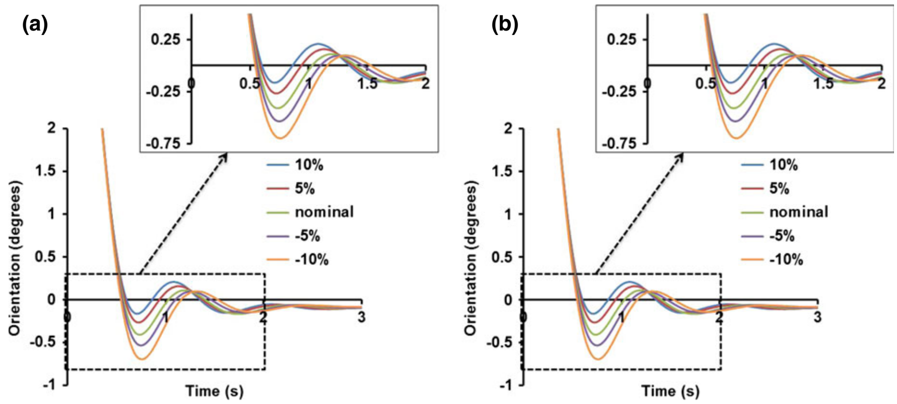

Figure 8 *Response trajectories when the derivative gain (D component of the PID controller) was varied from (a) +10 to −10% of the nominal value and (b) +50 to −50% of the nominal value.*

As the integral gain was varied between a 10% reduction to a 10% increase in nominal value, the overshoot increased by 2.5%, and the steady-state error became greater (the final orientation was further from 0). The settling time decreased by 1% over the smaller range of parameter variations, but increased by 4% over the larger variations (Figure 9). Interestingly, settling time does not drastically change with variations in K_i. Extreme deviations from the nominal value are required to significantly affect the behavior of the system; thus instability occurs at 92 times the nominal value, 0.7975 Nm/s·deg.

Active control is heavily influenced by time delay, which governs how quickly sensory information is processed before the proper motor response can be generated. As time delay decreased, corrections are made more rapidly, with a time delay of zero corresponding to an instantaneous change. Increased time delay resulted in an increased magnitude of overshoot and settling time (Figure 10). The overshoot is increased by 282% from a 10% reduction to a 10% increase in the nominal value and increased 2255% over the larger variation (−50% to +50% of the nominal value). The settling time increases 120% from −10% to +10% of the nominal value, and increases 725% from −50% to +50% of the nominal value. When the time delay is set to approximately 300 milliseconds (175% of the nominal value), the response of the system increased and upright sitting would not be possible.

Discussion

Although extensively used in the adult literature, mathematical models have not been widely adopted to examine the mechanisms underlying the development of posture and balance. Consequently, this is one of the first mathematical models to systematically examine the

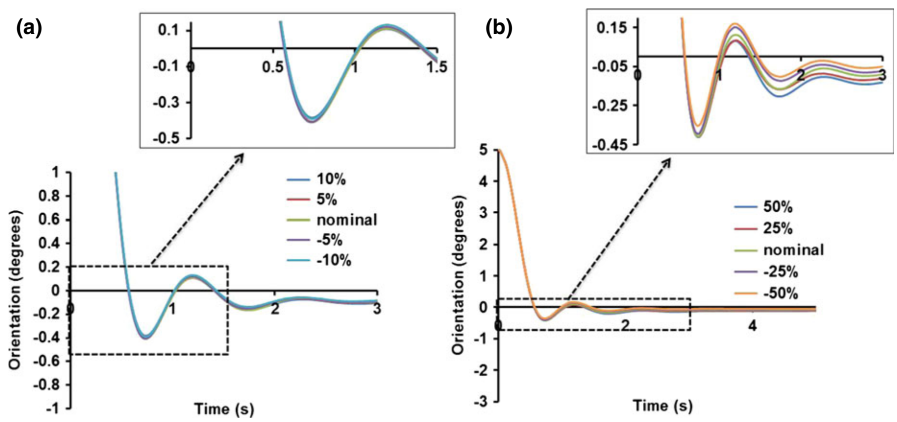

Figure 9 *Response trajectories when the integral gain (I component of the PID controller) was varied from (a) +10 to −10% of the nominal value and (b) +50 to −50% of the nominal value.*

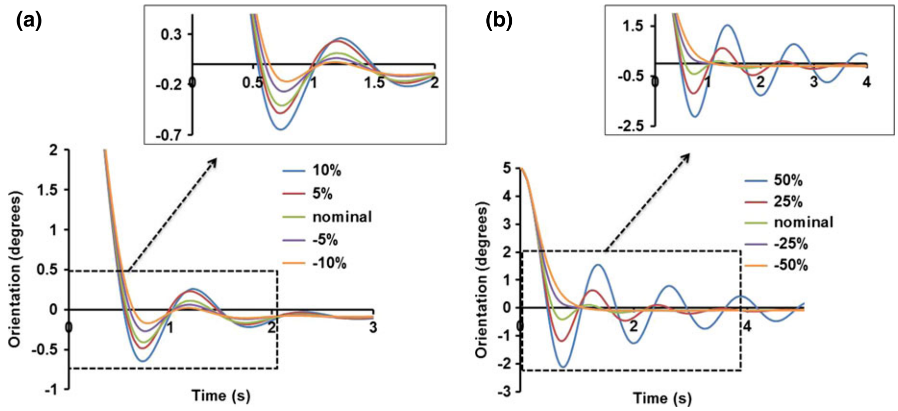

Figure 10 *Response trajectories when time delay was varied from (a) +10 to −10% of the nominal value and (b) +50 to −50% of the nominal value.*

specific active and passive neuromuscular mechanisms that contribute to the onset of and improvements in, independent sitting. The roles of active and passive control are further discussed below.

Removal of active and passive control

To effectively interact with the world, sitting must be adaptive, meaning that the infant must integrate sensory information so they can ultimately attenuate both internally- and externally-induced perturbations. When infants first sit, they are not adaptable and therefore lack the ability to offset perturbations to balance. The lack of adaptability ultimately means that they do not integrate the sensory information needed to adapt posture to task or environmental constraints (Dusing & Harbourne, 2010; Hadders-Algra, 2005; Hirschfeld & Forssberg, 1994) and therefore have difficulty remaining upright when performing goal-directed actions (Harbourne *et al.*, 1993). In order to simulate the lack of sitting adaptability previously reported in the literature, we explored a scenario in which active control was removed. Without active control, sitting posture could not settle to an upright posture. Rather, sitting posture settled to an orientation at 45 degrees relative to vertical. This orientation is the location where the forces produced by the inherent passive muscle properties and gravity balance. Thus, the passive control mechanism only allowed the model to adopt a non-adaptive, static forward lean.

Interestingly, newly-sitting infants have been observed sitting with a leaning posture (McGraw, 1932). Based on our current findings, it appears that early sitting infants rely on passive muscle properties to remain upright while learning to integrate sensory feedback into their postural movements. In this regard, passive control mechanisms may provide a foundation upon which the infant can first sit upright. This would allow the infant to gain critical experience in the sitting posture that would ultimately allow the more adaptive active control mechanisms to become properly calibrated. Utilizing passive elements to remain upright is important because it provides infants with experience stabilizing their head and trunk. Passively-supported sitting would provide the nervous system with the time necessary for the maturation of sensorimotor integration and would allow the infant time to learn the affordances of the new sitting posture.

We also examined the dynamics of sitting when only active control is available. Although the analogue to this manipulation is less clear in typically-developing children, infants with developmental disorders often have low muscle tone. The current simulations demonstrate that when no passive control is available, it is still possible for infants to stabilize to an upright position; however, the magnitude of the overshoot increases. Thus, the infant takes longer to stabilize and could easily lose balance. The passive mechanism, when properly adjusted, therefore acts to dampen movements (Figure 3). These findings may explain why hypotonic children demonstrate overly-exaggerated and uncoordinated movements (Brogren, Hadders-Algra & Forssberg, 1998). In addition, as mentioned above, lack of muscle tone may eliminate the early sitting experiences provided by passive mechanisms. If these early (albeit non-adaptive) experiences are indeed critical to the emergence of sitting, a delay in reaching the sitting milestone is expected when these experiences are absent.

Our model therefore predicts that if infants are provided with early sitting experiences through the support of a therapist or parent, sitting would improve. This prediction is supported by the literature. For example, when children with cerebral palsy are supported in a sitting posture, sitting stability improves (Harbourne, Willett, Kyvelidou, Deffeyes & Stergiou, 2010). Arguably, this method places emphasis on developing the passive control. However, it was also found that training incorporating error learning (i.e. allowing the infant to initiate movements with minimal guidance) further improved sitting abilities as well as causing an increase in sway complexity (Harbourne *et al*., 2010). This intervention provided infants with movement and sensory experience so that they may begin integrating feedback into their postural control strategy. In addition, by giving at-risk infants training and practice integrating sensory feedback, they are better able to react to perturbation, improve their adaptability, and improve their overall motor abilities (Deffeyes, Harbourne, Kyvelidou, Stuberg & Stergiou, 2009; Harbourne *et al*., 2010; Woollacott, Shumway-Cook, Hutchinson, Ciol, Price & Kartin, 2005). It therefore appears that training the passive mechanisms and slowly integrating sensory experiences provides a beneficial training paradigm when applied to children with developmental disorders.

Systematically manipulating the control parameters

In addition to removing either passive or active control, we systematically altered the parameter values of each mechanism. Systematically altering parameter values allowed us to examine how adjustments in control parameter values influenced sitting ability. We chose to perform both large- and small-magnitude manipulations. The large-scale manipulations were chosen because infants tend to exhibit large amounts of variability as they first learn a skill (e.g. Hadders-Algra, 2002, 2010; Saavedra *et al*., 2012), suggesting large changes. However, with practice, movements are refined, suggesting that infants are making small parametric changes as they attempt to discover optimal movement patterns based on the constraints of the task and individual (Thelen, Corbetta, Kamm, Spencer, Schneider & Zernicke, 1993).

In general, systematically altering the value of the control parameters simulates the exploration of neuromuscular properties that lead to improvements in sitting ability. Our results demonstrated that sitting was stable over a wide range of passive control parameters. Specifically, when varying the values of the muscle damping and stiffness terms, it was found that large deviations were required to destabilize the model. In essence, almost any amount of muscle tone will facilitate an infant's ability to attain upright sitting. Relying on passive control is beneficial to infants because minimal learning or calibration is needed. In contrast, to utilize active control, infants must develop and appropriately calibrate and integrate feedback to maintain and control sitting posture. Interestingly, small changes in the active parameters had large effects on the behavior of the system. For example, two of the active parameters (K_p, K_d) will cause the model to destabilize at just over twice the nominal value. The increased response is caused by energy being added to the system created by the combination of an increased (derivative) gain and time delay (Silva, Datta & Bhattacharyya, 2005). The sensitivity of the active parameters suggests that infants must learn (with sitting experience) how to modulate and coordinate active control so that they can perform a variety of goal-directed tasks within a dynamically-changing environment. Our findings are consistent with recent experimental findings. Specifically, Saavedra *et al*. (2012) examined how infants learn about gravity as they develop the ability to sit independently. They examined muscular activation patterns and kinematics of sitting infants as external support was provided at different locations of the trunk. They posited that their findings suggest that adaptive and mature sitting requires infants to develop an internal representation of sitting. Once an internal representation is developed, infants should properly activate muscles and utilize the appropriate degrees of freedom so that they can generate the proper body movements to remain upright. Our findings agree with their experimental data that passive control mechanisms could be important in forming this internal representation. Once an internal representation is formed, infants can properly scale active parameters so that postural movements are adaptive and scaled to the constraints of the task and environment.

Scaling parameters allowed us to simulate the process likely employed by infants as they learn to sit. The exaggerated movements that resulted at some of the parameter values may ultimately be functional. Specifically, learning the appropriate parameter values is likely aided by extraneous movements. Extraneous movements, sometimes called exploratory movements, do not directly aid in the performance of a goal, but allow the infant to determine the appropriate movement 'solutions' (Gibson & Pick, 2003). It has previously been suggested that exploratory postural movements aid in the learning of motor milestones such as reaching (e.g. Berthier, Rosenstein & Barto, 2005; Haddad, Rietdyk, Claxton & Huber, 2013), sitting (Harbourne & Stergiou, 2003), and standing posture (Claxton, Melzer, Ryu & Haddad, 2012). Exploratory movements result in infants performing movements in many different ways utilizing different degrees of freedom and muscular activation patterns. This variation of movements ultimately allows the infant to learn how to optimally move given a specific goal and environmental context.

The current model also helps explain some of the classic findings of Adolph (1997) that found information infants learn in one posture does not extend to new postures. For example, after experience crawling, infants learn what slopes are passable and which slopes are too steep to navigate. However, once the same infants begin to walk, they will attempt to walk down slopes they avoided when crawling. Thus, infants must re-learn the control strategies needed to successfully move in a new posture. In essence, our model demonstrates that feedback-driven control is sensitive to small parametric changes that must be calibrated to specific postures. Therefore, generating the appropriate response ultimately means that the spatial and dynamic properties of the response must be correctly scaled. For example, the magnitude of the needed postural correction [modeled as the P term], the speed of the correction [modeled as the D term], and finally the error [modeled as the I term] all need to be appropriately calibrated for each new posture and to the demands of the task and environment.

Because sensory information is not acted upon instantly, time delay is another critical parameter when investigating the development of active control. If time delay is too long, sensory information cannot be acted upon and incorporated into movements. Time delay is likely slower in young infants. This long time delay, as indicated in our simulations, ultimately makes the effective use of the active control mechanism difficult. Thus, in addition to learning the effective active control parameters needed to adaptively control sitting, the neuromuscular system of infants must develop to the point where they can integrate active control. In essence, with a long time delay, a static sitting orientation is not possible, even with properly calibrated control parameters.

Model limitations and future directions

As with all mathematical models, there are limitations worth noting. First, this model examined movement about only a single degree of freedom (a simulated hip joint). While single-degree-of-freedom models are likely adequate for sitting, extending this model to other postures, such as standing, would require the additional degrees of freedom. For example, modeling both the hip and ankle would better capture the movements of early walking. Second, noise was not included in the model. Noise was purposefully omitted here because we thought it was important to keep the model as simple as possible at this stage given that it was the first behavioral model of infant sitting. Third, similar to other postural models, the role of the environment was not included. Including an environmental context (e.g. surface compliancy) into the model could be an important future direction given that learning the dynamical interaction between body and environment is a critical component of development.

Conclusion

The results included herein provide basic insight into the control systems used by the body and how they affect the behavior of the postural system in infancy. The passive mechanism supported the achievement of early (stage 1–2) sitting, which provided experience needed to calibrate active control mechanisms so that the appropriate parameters are selected based on the context of the task and environment. With the emergence of active control, more adaptive sitting behaviors (e.g. reaching while sitting) are supported. Understanding the mechanisms involved in the development of sitting in typically developing infants is a necessary first step towards understanding the factors contributing to motor delays. This could lead to important advances in the ability to evaluate the outcome of therapy, and aid in the development of improved therapeutic interventions for individual developmental motor disorders. Furthermore, our model can be easily refined in future work to examine the development of other motor milestones, such as independent stance.

Acknowledgement

This project was partially supported by a Purdue Research Foundation Grant to Laura J. Claxton.

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Received: 30 June 2013
Accepted: 13 July 2014