Limits in motor control bandwidth during stick balancing

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When studying a system, the system can be parsed into separate subsystems: the plant and the controller. In control terminology, the plant is the isolated subsystem that is controlled; the controller is the subsystem that applies control input to the plant to make it behave in some desired fashion. The behavior of the overall system is governed by the characteristics of both the plant and controller.

In terms of a simple plant with a single rigid mass, movement of the plant without any control input reflects the mode of the system, the tendency of the system to move at a certain natural frequency. A plant with a higher natural frequency will move at a faster rate than a plant with a lower natural frequency. Intuitively, we know that it becomes increasingly more challenging to control an object as its speed of movement increases. Like other physical systems, human motor control has constraints, referred to as bandwidth, which limits the range of frequency over which the system can operate within some tolerated level of error. For instance, a person can track closely a reference signal such as a low-frequency sine wave, but, as the frequency of the sine wave increases, limits in the controller’s bandwidth will result in tracking error. This tracking error occurs as a result of a reduction in amplitude and/or an increase in delay (i.e., phase shift) between the reference and tracking signal. There are a number of factors that affect motor control bandwidth, including transmission delays in the nervous system, and force saturation and low-pass-filtering effects, such as electromechanical delay (Norman and Komi 1979; van Dieen et al. 1991), in the muscular system. In general, a lower force saturation point, longer delay, and/or greater lag from the low-pass filtering effect will lower the bandwidth for motor control.

In the present study, stick balancing was used to explore motor control bandwidth. Stick balancing is a classic system for studying control theory and has been applied in the past to study motor control (Cabrera and Milton 2002, 2004; Milton et al. 2009a, 2009b). To investigate motor control bandwidth, the dynamics of a stick-balancing task was modified by adjusting the height of a mass attached to the stick. By adjusting the mass height, the natural frequency at which the stick falls over changes: as the mass height is reduced, the stick will tend to fall over in a shorter period of time, reflecting that the plant has a higher natural frequency. By adjusting the dynamics of the plant, it is possible to gain insight into the bandwidth of the controller. We hypothesized that, as limits in control bandwidth were approached as the mass height decreased, the probability of successfully balancing would also decrease. In addition, we hypothesized that the level of muscle activation in both agonist and antagonist would increase as the natural frequency of the stick increased. These changes in muscle activation suggest that the central nervous system adapts muscle activation to task dynamics, possibly to improve control bandwidth.

Motor control; biological stability; feedback delay; electromechanical delay; muscle coactivation

Materials and methods

Subjects

Nine subjects (3 women: mean age = 24.3 yr (±6.3), mean height = 165 cm (±2), mean weight = 65.8 kg (±8.2); and 6 men: mean age = 34.0 yr (±11.4), mean height = 179 cm (±8), mean weight = 83.2 kg (±3.6)) were recruited for the study. All subjects were in good physical health and had no reports of neurological, musculoskeletal upper extremity or spinal disorders. Michigan State University’s Institutional Review Board approved the experimental protocol, and subjects gave informed consent prior to participation.

Task

Stick balancing was performed with a 1.9-kg mass attached to a 105-cm, 0.4-kg aluminum rod. To constrain the degrees of freedom,
the stick was connected to a pivot joint on a 1.2-kg cart that translated freely along a track (Fig. 1A). To keep the stick upright, the subject positioned the cart to keep the center of mass of the stick plus mass over the pivot joint. Subjects moved the cart by applying force to the cart handle. To confine the task, the stick balance apparatus was enclosed in a frame that was 85 cm long and 55 cm high (Fig. 1B).

Stick balance was performed at four different mass heights: center of mass of a 1.9-kg mass located at 100 cm, 75 cm, 50 cm, and 25 cm along the stick with the pivot joint representing the origin. Subjects performed stick balancing with their dominant hand, with feet shoulder width apart and with the nondominant hand at their side. At each mass height, the goal was to successfully complete five trials. For each trial, subjects were given 5 attempts to successfully balance the stick, allowing for a maximum of 25 attempts for each mass height. A trial was successful if the subject kept the stick upright for 20 s without making contact with the frame.

Signal Collection and Processing

Surface electromyography (EMG) signals were collected using a Bagnoli EMG system (Delsys, Boston, MA), with 16-bit resolution, common mode rejection ratio of minimally 85 dB, and a frequency response between 20 and 450 Hz. Single differential EMG sensors were used with two 10×1-mm silver bar contacts and contact spacing of 10 mm. EMG signals were recorded from wrist flexors (flexor carpi radialis) and wrist extensors (extensor digitorum) on the dominant hand. Subjects were instructed to resist wrist flexion and extension to identify EMG sensor placement. The following trunk muscles were recorded bilaterally: internal oblique (approximately midway between the anterior superior iliac spine and symphysis pubis, above the inguinal ligament), external oblique (~15 cm lateral to the umbilicus), thoracic erector spinae (5 cm lateral to T₁0 spinous process), and lumbar erector spinae (3 cm lateral to L₁₅ spinous process). All signals were differentially amplified and then analog-to-digital converted at a sample rate of 1,600 samples/s. Postprocessing of EMG signals involved removing DC bias, rectifying, and low-pass filtering with a second-order dual-pass Butterworth filter with a cutoff frequency at 4 Hz.

Prior to stick balancing, maximal voluntary isometric contractions (MVCs) against manual resistance were conducted to obtain reference values to normalize EMG signals. For wrist flexors and extensors, subjects generated MVCs in wrist flexion and extension. For trunk muscles, subjects generated MVCs in trunk flexion, extension, and lateral bending to the right and left. MVCs were achieved during a 3-s trial with ~2 min between each exertion.

To quantify the level of agonist and antagonist muscle activation, peaks in agonist muscle activation levels during the stick-balancing trials were determined (Fig. 2). Using these peaks, the activation levels at these time periods in the antagonist were then determined. The average of the agonist peaks and corresponding antagonist activation levels over the entire trial were used to estimate the average muscle activation level for agonist and antagonist, respectively. For the forearm, wrist flexors and extensors represented agonist-antagonist pairs. For the trunk, the four left-side muscles were combined, and the four right-side muscles were combined to form two distinct groups, representing agonist-antagonist pairs.

![Fig. 1. A and B: setup for stick-balancing experiments. A: the mass attached to the stick can be positioned at different heights (length_{com}) to change the dynamics of the plant. With the stick in the configuration shown on B, the cart would need to be accelerated to the left (from subject’s perspective), which means that wrist flexors and trunk muscles on the left side of the body would act as agonist, and wrist extensors and trunk muscles on the right side of the body would act as antagonist.](image-url)
Statistical Analysis

Hypothesis 1: The probability of successfully balancing decreases as mass height decreases. The probability of successfully balancing the stick was estimated based on the number of successful (s) and failed (f) attempts during a trial. It can be shown that the likelihood function \( L(p) \) to determine the probability of success \( p \) and consequently failure \( 1 - p \) admits the form:

\[
L(p) = p^s(1 - p)^f
\]  

(1)

Similar likelihood functions have been studied in the past (Hastings and Peacock 1975; Johnson and Kotz 1969; Rice 2007). From this likelihood function, the probability of success can be estimated by the following approximation:

\[
p = \frac{s}{s + f}
\]  

(2)

Next, the null hypothesis that the probability of success is the same for all mass heights was tested using the following likelihood ratio test:

\[
\Lambda = \frac{\max L(p_{100}, p_{75}, p_{50}, p_{25})H_0}{\max L(p_{100}, p_{75}, p_{50}, p_{25})H_1}
\]  

(3)

where \( H_0 \) is the reduced model (null hypothesis) under which the probability of successfully balancing the stick is not dependent on mass height, and \( H_1 \) is the full model (alternative hypothesis) where the probability of successfully balancing the stick is a function of mass height. The null hypothesis was rejected if the test score based on the observed \( \Lambda \) was less than \( \alpha_{critical} \) set at 0.05.

Hypothesis 2: The level of muscle activation in both agonist and antagonist increases as the natural frequency of the stick increases. To account for correlation between repeated measures and between subject differences at baseline, a difference method was used. To clarify, the average muscle activation in the agonist and antagonist during the trial at the mass height of 100 cm was set as a baseline. Then the differences in muscle activation between baseline and subsequent mass heights were determined. The null hypothesis that no significant relationship exists between muscle activation and stick natural frequency was tested using regression analysis. Changes in muscle activation between each of the three height differences (100–75 cm, 100–50 cm, 100–25 cm) represented the dependent variable. In terms of the independent variable, the natural frequencies of the stick \( \omega_n = \sqrt{\text{gravity/length}_{COM}} \) where \( \omega_n \) is the natural frequency and \( \text{length}_{COM} \) is mass height at the four mass heights were estimated, and the difference in natural frequencies calculated. The null hypothesis was rejected if the slope of the regression line was significantly different than zero. The null hypothesis was tested using a regression ANOVA with \( \alpha_{critical} \) set at 0.05.

These statistical methods were applied to assess muscle activation for agonist and antagonist of the forearm and trunk, separately.

Exploratory analysis revealed that variability of forearm and trunk agonist and antagonist muscle activation increased with increasing natural frequency of the stick. To account for this heteroscedasticity, a sequential bootstrap technique was applied (Babu et al. 1999; Jimenez-Gamero et al. 2006). For more particulars on the methods, see the APPENDIX.

RESULTS

Hypothesis 1: The Probability of Successfully Balancing Decreases as Mass Height Decreases

The likelihood ratio test produced a \( \Lambda = 1.3 \times 10^{-25} \), which was significant at the alpha-level of 0.05. Thus the null hypothesis that the probability of successfully balancing the stick is the same at all levels was rejected. As shown in Fig. 3, changes in probability of success displayed a threshold effect, indicating limits in stick-balancing ability were approached. Several subjects had difficulty completing trials at the 25-cm mass height, and one subject was unable to complete any of the trials at the 25-cm height.

Hypothesis 2: The Level of Muscle Activation in Both Agonist and Antagonist Increases as the Natural Frequency of the Stick Increases

Regression analysis confirmed that a linear relationship existed between increases in muscle activation and increases in natural frequency of the stick for forearm agonists [Fig. 4A, \( F(1,25) = 12.98, P = 0.001 \), forearm antagonist [Fig. 4A, \( F(1,25) = 8.82, P = 0.006 \), trunk agonist [Fig. 4B, \( F(1,25) = 35.35, P < 0.001 \), and trunk antagonist [Fig. 4B, \( F(1,25) = 24.06, P < 0.001 \). Muscle activation in the agonists was slightly higher than the antagonists, and muscle activation in the forearm was considerably higher than the trunk but well below maximum activation (Table 1).

Bootstrap procedures indicated that the results from the regression analysis were not affected due to heteroscedasticity. For more details on the bootstrap results, see the APPENDIX.

DISCUSSION

The goal of the paper was to investigate limits in motor control bandwidth using a stick-balancing task. As expected, stick balancing became more challenging as the natural frequency of the stick increased (decreased mass height). At the lowest mass height (25 cm), several subjects had difficulty completing the trial, which indicates that limits in motor control bandwidth were approached. The ability to balance the stick with the mass in the lowest position varied considerably between subjects, ranging from being successful 0–83% of the time.

The secondary purpose of the paper was to explore changes in muscle activation with changes in plant dynamics. As the task became more challenging, the level of activation in the agonist muscles increased proportionally. Given that the force applied to the cart handle is regulated by the stick’s angular position and velocity (Reeves and Cholewicki 2010), it is not surprising that muscle activation in the agonist increased as the task became more challenging. More agonist muscle activation is required in accelerating the cart when the task becomes more
challenging, but this is only true for the agonist. More antagonist muscle activation would act to reduce cart acceleration by reducing the torque produced by the joint. However, as the natural frequency of the stick increased, the level of antagonist muscle activation also increased proportionally.

So why does the CNS increase antagonist muscle coactivation? We suspect that the CNS is adapting the controller’s responsiveness to match plant dynamics. Muscle agonist-antagonist coactivation increases joint stiffness, which allows for better coupling between the controller and the plant. By having a stiffer coupling, the controller can apply higher frequency control input to plant, thus improving the controller’s bandwidth. An alternative hypothesis that may explain why the CNS increases muscle coactivation is that this increased coactivation may reduce noise in motor control. There is some evidence to suggest that kinematic variability may decrease with higher levels of coactivation (Selen et al. 2005). Decreased kinematic variability with muscle coactivation would mean that positioning the cart would be more precise. Improved precision in the controller would decrease the size and rate of stick displacement during balancing. Given the limits in control bandwidth, improved controller precision may be important for ensuring the stick stays within the tolerated level of error defined by the experimental constraints (cage dimensions). As with plant dynamics, if impairments in the controller result in task dynamics (i.e., high-velocity stick movements) that approach or exceed the controller’s bandwidth, then the probability of success will decrease and eventually converge to zero.

As previously reported, there was considerable variability in the ability to balance the stick at the lowest mass height. Some balanced the stick while keeping the stick close to upright position for most of the trial, while others struggle to successfully complete stick balancing for the 20-s trial. And others were not able to keep the stick upright for even a short period of time, demonstrating control instability. So what produced the instability in some but not others? One factor that can be ruled out is force saturation in the controller. It is possible that subjects were not able to generate sufficient force to accelerate the cart to keep the stick upright. But the level of muscle activation in both the forearm and the trunk was well below maximum effort levels (see Table 1), suggesting that force saturation was not a factor. Another source for instability could be differences in cognitive and neuromuscular delays between subjects. Even small differences in delays between subjects could be important, if the task is close to the boundary for instability. But other factors could also affect task stability. For instance, the control logic for keeping the stick upright could be different between subjects. Some subjects could use more aggressive control, meaning, for a given displacement, more force could be applied to return the stick to the upright position. Qualitatively, we noticed that some subjects moved the cart with more abrupt movements than others. This aggressive control coupled with delays could lead to instability (Reeves et al. 2011). Perhaps delays between subjects were similar, but control logic varied, resulting in stick-balancing failure in some at the lower height. And finally, because noise is inherent in the neuromuscular system, it is possible that some individuals have more noise in their controller, making it more difficult to keep the stick within the tolerated level of error.

One of limitations of the study is that not all muscles, and perhaps not the primary muscles used to move the cart, were assessed. During stick balance, subjects tended to use a lot of

![Fig. 4. A and B: change in forearm and trunk muscle activation with respect to change in natural frequency of stick. Because the difference method was used, 3 points reflecting the differences between baseline (scores at 100 cm) and the other mass heights (scores at 75 cm, 50 cm, and 25 cm) are shown. The regression equation and $R^2$ value were obtained from Minitab analysis. Note that the change in natural frequency for the agonist and antagonist are the same and were offset in the chart to improve visual clarity.](http://jn.physiology.org/)

![Table 1. Average muscle activation levels in wrist and trunk agonists and antagonists grouped by mass height](http://jn.physiology.org/)

<table>
<thead>
<tr>
<th>Mass Height, cm</th>
<th>Natural Frequency, Hz</th>
<th>Wrist, %MVC</th>
<th>Trunk, %MVC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Agonist</td>
<td>Antagonist</td>
</tr>
<tr>
<td>100</td>
<td>0.50</td>
<td>10.6 (7.9)</td>
<td>10.0 (8.2)</td>
</tr>
<tr>
<td>75</td>
<td>0.58</td>
<td>10.7 (7.8)</td>
<td>9.8 (8.2)</td>
</tr>
<tr>
<td>50</td>
<td>0.71</td>
<td>11.7 (7.8)</td>
<td>10.5 (8.1)</td>
</tr>
<tr>
<td>25</td>
<td>1.00</td>
<td>13.8 (8.2)</td>
<td>11.8 (8.1)</td>
</tr>
</tbody>
</table>

Values are means (SD). MVC, maximal voluntary isometric contractions.
internal and external rotation about the shoulder. Unfortunately, the activity of these muscles is difficult to assess with surface EMG. Instead, we decided to record more superficial muscle in the forearm and the trunk. However, it is important to remember that the various body segments must all work together to transfer forces to the cart to keep the stick upright. Therefore, we felt confident that adaptive changes in muscle activation seen in one part of the body would be reflected in other body segments. Our data of muscle activation in two body segments, the forearm and the trunk, support this belief.

So what are the clinical implications for the stick-balancing experiment? In the future, assessment of postural control bandwidth may provide insight into falling in at risk population, such as elderly (Lord et al. 2001; van Dieen and Pijnappels 2008). Risk factors for falls in elderly appear to be multifactorial (Horak 2006; Lord et al. 2001); however, control bandwidth for postural control could represent an umbrella assessment that captures the consequences of these various risk factors, which then could be used to target intervention. For instance, do elderly people have longer delays in response to postural perturbations, or, alternatively, do they have similar delays, but those at risk have more aggressive control strategies, possibly from a fear of falling? Perhaps some elderly people may have limited force-generating capacity, making them less robust to sudden, significant perturbations, such as slips. Or possibly, those at risk of falling have less precise controllers, resulting in movement dynamics that fall outside their control bandwidth. Assuming that the underlying mechanism for limits in control bandwidth can be identified, the next question will be: can they readily be modified?

APPENDIX

To account for heteroscedasticity in the dependent variables, a sequential bootstrap technique was applied (Babu et al. 1999; Jimenez-Gamero et al. 2006). Briefly, data from the experiment were used to generate 1,000 new data sets from the original data, but with different frequency of occurrence (weights). These weights were generated using the Poisson bootstrap resampling scheme (Babu et al. 1999). For instance, a particular observation could receive a weight ranging from 0, 1, 2 . . . representing that the observation was not present, present once, or present multiple times with the multiple reflecting the weight. The new data sets ranged from 6 to 18 observations. Note that the original experiment contained 9 observations, which reflected the average score for each subject at a given height. Using the new data sets, 1,000 experiments were simulated to yield a distribution of dependent variables. In this case, the dependent variables were the regression coefficients relating forearm and trunk agonist and antagonist activation level to changes in natural frequency of the stick. Using the observed distribution of dependent variable, the confidence interval ranging from the 2.5–97.5 percentile was determined. If the confidence interval for the coefficients for the “slope” of the regression line did not contain a zero value, then it was assumed that a regression model that included the independent variable, was a better predictor than a simple mean model (slope = 0), thus indicating that the dependent variable is affected by changes in mass height.

Bootstrap procedures indicated that the results from the regression analysis were not affected due to heteroscedasticity. Bootstrap distributions of the dependent variables reflecting the regression slopes showed that the zero value was not contained in the 95% confidence interval. In fact, the zero value was not present in any of the bootstrap distributions (minimum regression slope for forearm agonist = 3.1; forearm antagonist = 2.4; trunk agonist = 2.0; trunk antagonist = 1.2). Therefore, bootstrap procedures support the findings that changes in the dependent variables (forearm and trunk agonist and antagonist muscle activation) appear to be linearly related to changes in the natural frequency of the stick.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

Author contributions: N.P.R. conception and design of research; N.P.R. analyzed data; N.P.R., P.P., J.M.P., and V.V. interpreted results of experiments; N.P.R. prepared figures; N.P.R. drafted manuscript; N.P.R., P.P., J.M.P., and V.V. edited and revised manuscript; N.P.R., P.P., J.M.P., and V.V. approved final version of manuscript; J.M.P. and V.V. performed experiments.

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