Limits in motor control bandwidth during stick balancing

N. Peter Reeves¹, Pramod Pathak², John M. Popovich Jr.¹, Vilok Vijayanagar¹

Authors’ contributions
NPR developed the idea of using stick balancing to study control bandwidth.
NPR, PP, JMP, and VV were responsible for developing the study protocol.
JMP and VV collected data.
NPR, PP, and JMP analyzed the data.
All authors aided in preparing the manuscript.

¹College of Osteopathic Medicine, Michigan State University, East Lansing, MI, USA, 48824
²Department of Statistics and Probability, Michigan State University, East Lansing, MI, USA, 48824

Corresponding author
N. Peter Reeves
College of Osteopathic Medicine, Michigan State University,
2727 S. Pennsylvania Ave, Room 230, Lansing, MI, USA, 48910
email: reevesn@msu.edu
Phone: (517) 975-3303
Fax: (517) 975-3305

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Abstract (words 236 max 250)

Why can we balance a yardstick but not a pencil on the tip of our finger? As with other physical systems, human motor control has constraints, referred to as bandwidth, which restricts the range of frequency over which the system can operate within some tolerated level of error. To investigate control bandwidth, the natural frequency of a stick used during a stick balancing task was modified by adjusting the height of a mass attached to the stick. The ability to successfully balance the stick with the mass positioned at four different heights was determined. In addition, electromyographic signals from forearm and trunk muscles were recorded during the trials. We hypothesized that (1) the probability of successfully balancing would decrease as mass height decreased, and (2) the level of muscle activation in both agonist and antagonist would increase as the natural frequency of the stick increased. Results showed that as the mass height decreased the probability of successfully balancing the stick decreased. Changes in the probability of success with respect to mass height showed a threshold effect, suggesting that limits in human control bandwidth were approached at the lowest mass height. Also, the level of muscle activation in both the agonist and antagonist of the forearm and trunk increased linearly as the natural frequency of the stick increased. These changes in muscle activation suggest that the CNS adapts muscle activation to task dynamics, possibly to improve control bandwidth.
Introduction

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, Cabrera and Milton, 2004, Milton et al., 2009b, Milton et al., 2009a). 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. We speculated that the CNS changes muscle coactivation to match the dynamics of the task: if the plant moves faster, the controller becomes more responsiveness by increasing joint stiffness through coactivation.

Materials and Methods
Subjects.

Nine subjects (3 females: mean age=24.3 years (±6.3), mean height = 165 cm (±2), mean weight = 65.8 kg (±8.2); and 6 males mean age=34.0 years (±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. All subjects signed an informed consent form approved by the university’s IRB.

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 4 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 non-dominant hand at their side. At each mass height, the goal was to successfully complete 5 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 seconds without making contact with the frame.

Signal collection and processing.

Surface electromyography (EMG) signals were collected using a Bagnoli EMG system (Delsys, Boston, USA), with 16-bit resolution, CMRR of minimally 85 dB, and a frequency response between 20 – 450 Hz. Single differential EMG sensors were used with two 10 x 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 (approximately 15 cm lateral to the umbilicus), thoracic erector spinae (5 cm lateral to T9 spinous process), and lumbar erector spinae (3 cm lateral to L5 spinous process). All signals were differentially amplified and then A/D converted at a sample rate of 1600 samples per second. Post processing of EMG signals involved removing DC bias, rectifying, and low pass filtering with a 2nd order dual-pass Butterworth filter with a cut-off 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 second trial with approximately 2 minutes 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 4 left-side muscles were combined and the 4 right-side muscles were combined to form two distinct groups, representing agonist-antagonist pairs.

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 \( L \) to determine the probability of success (p) and consequently failure (1-p) admits the form:

\[
L(p) = s^p(1-p)^f
\]  
[eq 1]
Similar likelihood functions have been studied in the past (Johnson and Kotz, 1969, Hastings and Peacock, 1975, Rice, 2007). From this likelihood function, the probability of success can be estimated by the following approximation:

\[
p \approx \frac{s}{s + f}
\]  
\[\text{[eq 2]}\]

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

\[
\Lambda = \frac{\max L(p_{100}, p_{75}, p_{50}, p_{25})_{H_0}}{\max L(p_{100}, p_{75}, p_{50}, p_{25})_{H_a}}
\]  
\[\text{[eq 3]}\]

The null hypothesis was rejected if the test score based on the observed \( \Lambda \) was less than \( \alpha_{\text{critical}} \). \( \alpha_{\text{critical}} \) was 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 \( w_n = \sqrt{\frac{\text{gravity}}{\text{length}_{\text{COM}}}} \) at the 4 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 0. The null hypothesis was tested using a regression ANOVA with \( \alpha_{\text{critical}} \) 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 (Jimenez-Gamero et al., 2006, Babu et al., 1999). For more particulars on the methods, see the appendix.

Results.

Hypothesis #1. The probability of successfully balancing would decrease as mass height decreased. 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 1 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, $F_{df=1,25}=12.98$, $p=0.001$),
forearm antagonist (Fig. 4A, $F_{df=1,25}=8.82$, $p=0.006$), trunk agonist (Fig. 4B,
$F_{df=1,25}=35.35$, $p<0.001$) and trunk antagonist (Fig. 4B, $F_{df=1,25}=24.06$, $p<0.001$).
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 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 considerably 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 second 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 wrist and the trunk were well below maximum effort levels (see Table 1), suggesting that force saturation was not 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 limitation 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 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 experiments? 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 (Lord et al., 2001, Horak, 2006); 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 failing 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.
Disclosure. There are no potential conflicts of interest, financial or otherwise (e.g., consultancies, stock ownership, equity interests, patent-licensing arrangements, lack of access to data, or lack of control of the decision to publish, or any other potential conflict) associated with the research contained in this manuscript.

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To account for heteroscedasticity in the dependent variables, a sequential bootstrap technique was applied (Jimenez-Gamero et al., 2006, Babu et al., 1999). Briefly, data from the experiment was used to generate 1000 new data sets from the original data but with different frequency of occurrence (weights). These weights were generated from Babu Poisson bootstrap resampling scheme (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, 1000 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 including 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) appears to be linearly related to changes in the natural frequency of the stick.
References.


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 Suppl 2, ii7-ii11.


Table 1: Mean values (standard deviation) of average muscle activation levels in wrist and trunk agonists and antagonists grouped by mass height.
Figure Captions

Figure 1A-B. Set-up for stick balancing experiments. The mass attached to the stick can be positioned at different heights ($\text{length}_{\text{com}}$) to change the dynamics of the system (A). With the stick in the configuration shown on B, the cart would need to be accelerated to the left (from subject 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.

Figure 2. Method for determining agonist and antagonist muscle activation. First, the time periods where peaks in agonist muscle activation levels during the stick balancing trials were determined. Then using these time periods, the activation levels in the antagonist at these instances 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. Right and left lumbar erector spinae muscles are shown.

Figure 3. Probability of successfully balancing a stick with the mass at different heights.

Figure 4A-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 $R^2_{(adj)}$ 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.
<table>
<thead>
<tr>
<th>Mass Height</th>
<th>Natural frequency</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 cm</td>
<td>0.50 Hz</td>
<td>10.6 (7.9)</td>
<td>10.0 (8.2)</td>
</tr>
<tr>
<td>75 cm</td>
<td>0.58 Hz</td>
<td>10.7 (7.8)</td>
<td>9.8 (8.2)</td>
</tr>
<tr>
<td>50 cm</td>
<td>0.71 Hz</td>
<td>11.7 (7.8)</td>
<td>10.5 (8.1)</td>
</tr>
<tr>
<td>25 cm</td>
<td>1.00 Hz</td>
<td>13.8 (8.2)</td>
<td>11.8 (8.1)</td>
</tr>
</tbody>
</table>

Table 1
Figure 1 A-B
Figure 3
Forearm

\[ \Delta \% \text{MVC} = 7.40 \times \Delta \text{Nat Freq} - 0.45 \]
\[ R^2 \text{ (adj)} = 31.5\% \]

\[ \Delta \% \text{MVC} = 4.55 \times \Delta \text{Nat Freq} - 0.50 \]
\[ R^2 \text{ (adj)} = 23.1\% \]

Figure 4A
Trunk

$\Delta \text{MVC} = 3.20 \times \Delta \text{Nat Freq} - 0.21$

$R^2 \text{ (adj)} = 56.9\%$

$\Delta \text{MVC} = 1.88 \times \Delta \text{Nat Freq} - 0.14$

$R^2 \text{ (adj)} = 47.0\%$

Change in Muscle Activation (% MVC)

Change in Stck Natural Frequency (Hz)

Figure 4B