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A COMPARISON OF METHODS FOR CALCULATING NEUROLOGICAL DELAYS IN PERTURBED STANCE

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SUMMARY
Research examining the neurological delays between sensory input and motor output has primarily focused on assessing EMG delays to discrete perturbations. However, there is evidence that delayed correlations between the COM motion and joint torques may be used to assess this delay in a static balance task. The purpose of this study was to compare the neurological delays in perturbed stance based on EMG latencies and on delayed correlations between COM motion and ankle joint torques. Calculated delays were 125 ± 11 ms for EMG latencies and 135 ± 6 ms for delays from COM and joint torque correlations. There was a mean difference of 10 ms between the two methods; however, individual differences ranged from 1 to 26 ms, suggesting that there may be significant individual variations in these measures.

INTRODUCTION
The control of balance involves a continuous feedback system of processing visual, vestibular and somatosensory inputs and executing neuromuscular actions to maintain equilibrium [1]. Previous research has examined the delay between these inputs and resulting neuromuscular actions through platform translations to be approximately 65 to 130 ms for EMG [2, 3] and 90 to 164 ms for joint torque [2]. However, these delays may be affected by the velocity and amplitude of the perturbation, and whether a range of perturbations are presented randomly or in series [4, 5]. Furthermore, some research has shown larger EMG responses beginning at 200-300 ms in the absence of ankle cues due to vestibular and visual inputs [6]. Such protocols are useful for determining the minimum delays expected within a balance task; however, these large discrete perturbations may not best represent the neurological delays expected during quiet stance, when the CNS may need to switch between the three main inputs or may be presented with conflicting information.

Yeadon and Trewartha [7] examined the neurological delay in static balance through examining the correlations of joint torques to centre of mass (COM) motion during inverted stance; with estimated latencies of 160 to 240 ms. However, this approach will only give a rough estimate of the average delays over the full duration of the trial, incorporating several delays within it, such as electromechanical delay (EMD), rise time for joint torques to reach maximum and the time for any sensory thresholds to be reached, resulting in an overestimation of the true neurological delay. Based on literature values, Yeadon and Trewartha subtracted an estimated value of 40 ms from all trials to account for these delays, resulting in estimated latencies of 120 to 200 ms. However, these literature values are based on the rise time of joint torques only, and may not best represent the true delay from all three factors.

The aim of the present research was to compare the neurological delays calculated via EMG latencies and correlations of COM and joint torque during perturbed stance.

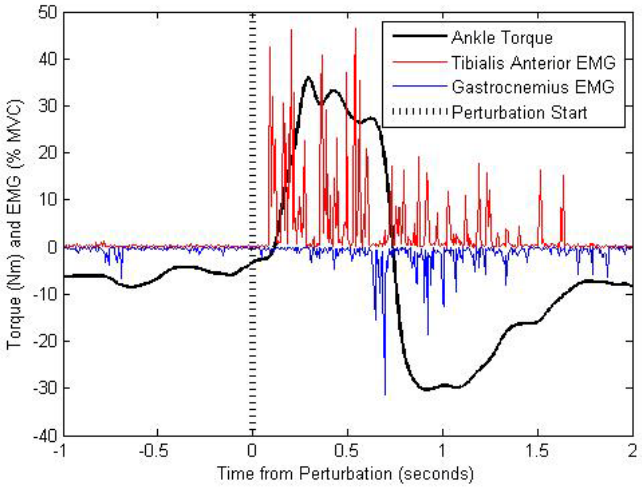

Figure 1: An example of the EMG and ankle joint torque response to an anterior platform perturbation (resulting in posterior sway).

METHODS
Seven healthy subjects were asked to perform 12 trials in perturbed stance, with 3 trials from each of the 4 conditions of small (5cm at 0.1 ms^{-1}) and large (10cm at 0.2 ms^{-1}) platform translations in the anterior and posterior directions. Muscle activity was measured via 4 Trigno wireless EMG sensors (2000 Hz) placed on the muscle bellies of the Medial Gastrocnemius and Tibialis Anterior of the right and left lower leg. Three-dimensional kinematics was collected via a nine camera vicon system

(200 Hz) with a set of 53 markers; and kinetics via two Bertec force platforms (2000 Hz, down sampled to 200 Hz) imbedding into a Stewart platform with six degrees of freedom. All analogue force and EMG data were synchronized with the motion data through the same ADC within the vicon giganet box. All equipment was part of the Motek Medical CAREN system, and platform motions were controlled via Motek's D-Flow software system designed for this purpose.

EMG delays were calculated from the initiation of the platform translation (based on horizontal force) to the first major muscle burst, through visual inspection of the EMG signal (figure 1). Two-dimensional joint torques were calculated for the ankle joint and regressed over time against COM displacement and velocity using the procedures in Yeadon and Trewartha [7].

RESULTS AND DISCUSSION

Calculated neurological delays were 125 ± 11 ms for EMG latencies and 135 ± 6 ms for delays from COM and joint torque correlations (table 1). The mean difference between the two methods was 10 ms; however, the individual differences ranged from 1 to 26 ms, with shorter EMG latencies for all subjects.

It would appear that neurological delays are slightly overestimated when calculated via delayed correlations, suggesting that future research should subtract approximately 10 ms from these values instead of the 40 ms used by Yeadon and Trewartha [6]. However, the relatively large range of differences between these two methods would suggest that it would be more appropriate to first calculate individual values based on perturbed stance, which can then be used to adjust estimated values gained during quiet stance.

Both EMG latencies and torque delays in the present study are similar to those found in previous perturbation studies [2, 3]; however, all responses began before 150 ms, which would suggest that these responses are likely due to long latency reflexes of the ankle plantar- and dori-flexors [3]. Therefore, it remains unclear if these findings can be transferred to the expected delays when balancing in quiet stance, were neurological delays will may be affected by increased sensory reliance on the slower visual and vestibular feedback systems or when the neurological system receives conflicting sensory inputs.

The findings in this study support the theory that delayed correlations between COM displacement and velocity can be used as an estimate of neurological delay during balance tasks. However, it must be noted that these estimated delays will represent an average of all the delays from the numerous corrections expected during continuous balance.

Table 1: Subject and group means and standard deviations for the delays from EMG and COM correlations

	EMG (ms)	COM (ms)	Difference (ms)
Subject 1	131	135	5
Subject 2	116	136	25
Subject 3	109	135	26
Subject 4	125	133	8
Subject 5	129	130	1
Subject 6	125	130	5
Subject 7	142	146	4
Mean	125	135	10
SD	11	6	10

CONCLUSIONS

These findings suggest that delayed correlations between COM displacement and velocity can be used as an estimate of neurological delay during balance tasks once the overestimation of the delayed correlation method is accounted for. However, the relatively large range of individual differences between these two methods would suggest that it may not be appropriate to use a single value for all individuals. It is suggested that researches should first calculate individual values based on perturbed stance, which can then be used to adjust estimated values gained from delayed correlations during quiet stance. Future research may wish to examine these differences further to ensure this relationship between the two methods persists for smaller perturbations or during balance in different postures, such as inverted stance or single leg stance.

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