Automatic postural responses following rapid displacement of a light touch contact during standing.

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Abstract

Responses elicited by the rapid displacement of a light touch contact surface were investigated during standing with the eyes closed. During quiet standing the touch surface was moved with an imperceptible slow (0.5 Hz), small (0.5 cm) oscillation to entrain the participant's sway. Periodically, a rapid displacement (1.25 cm, 12.5 cm/s peak velocity, 187.5 cm/s/s peak acceleration) of the rod was applied, either forwards or backwards, at either the fore or aft position of the entrained sway. Each participant received 10 unexpected displacements of the same direction, with 20 participants receiving forward displacements and 6 participants receiving backward displacements. Electromyographic recordings from 4 arm and 2 ankle muscles were sampled along with center of pressure and joint kinematics. Rapid displacement of the touch surface consistently resulted in short-latency (<120 ms) responses in the muscles of the arm or ankle in 21 of 26 participants. However, the first exposure to the touch displacement resulted in a distinct response in the muscles about the ankle in 13 participants, while responses in arm muscles were observed in 11 participants. Participants that responded with activation of muscles at the ankle displayed a corresponding shift in the center of pressure. Trials 2 through 10 were characterized by an absence of responses in the ankle muscles, but more consistent responses in the arm muscles. The rapid onset of ankle muscle activity following the unexpected slip of a touch surface in some instances suggests that tactile cues provide a potential sensory cue for triggering balance reactions. The importance of this sensory cue in balance control is likely dependent in part on the relevance of the tactile inputs in the context of the perceived task.

Keywords: touch, balance, haptic, standing, human

Abbreviations

AD – anterior deltoid

A-P – anterior-posterior

BB – biceps brachii

CoP – center of pressure

EMG – electromyogram

M-L – medial-lateral

MVC – maximum voluntary contraction

PC – Pacinian corpuscle

PD – posterior deltoid

RA – rapidly adapting

SA – slowly adapting

SOL – soleus

TA – tibialis anterior

TB – triceps brachii

Introduction

In the absence of vision, lightly touching an external reference reduces postural sway during standing (Jeka and Lackner, 1994). The effect of light touch on postural sway is observed with vertical touch forces less than 1 N, which has been shown to be insufficient to create a mechanical benefit (Holden et al., 1994; Kouzaki and Masani, 2008). The effect of light touch on sway is argued to be derived from sensory cues arising from the shear forces at the interface between the fingertip and the contact surface. Cross-correlation analysis shows a positive correlation between the shear force at the fingertip and fluctuations in the body center of pressure (CoP), with the shear force at the finger leading the fluctuations of the CoP suggesting that the sensory feedback from the fingertip was driving the sway (Clapp and Wing, 1999; Jeka and Lackner, 1994). Furthermore, the effect of light touch on sway is reduced when sensory feedback from the hand is impaired by an ischemic block (Kouzaki and Masani, 2008). It is therefore argued that light touch of a stable reference provides a supplemental sensory input to detect the direction, extent and speed of sway, which is then incorporated in the balance control system to regulate postural muscle activity and stabilize the motion of the body.

The light touch sensory cues with contact of a stable surface would therefore seem to encode the relationship between the sway of the body and the reference point at the interface with the contact surface. This proposed role for the tactile cues is further supported by the observation that sway becomes entrained to the motion of the contact surface if the contact surface is not fixed, but slowly oscillates (Jeka et al., 1998; Wing et al., 2011). In other words, the tactile sensory input generated by the motion of the contact surface is interpreted by the balance control system as the body moving relative to the contact surface. This in turn modulates activity of postural muscles to counteract the perceived sway of the body creating an actual sway that is driven by the sensory cues arising from the light touch. This effect also highlights the potency of the tactile cue in regulating postural activity as the entrainment to the touch reference occurs despite the conflicting feedback that would be generated from other sensory cues, including muscle proprioceptors and the vestibular system.

Light touch of a stable reference has also been demonstrated to modulate the responses evoked following sudden, unexpected balance disturbances. For example, Dickstein et al. (2003) reported that light touch influenced the magnitude, but not the latency, of postural responses to unexpected support translation in older adults. Forero and Misiaszek (2013) showed that the amplitude, but not the latency, of responses to pulls at the waist during treadmill walking increased when light touch was provided in the absence of vision. Taken together, these findings suggest that cutaneous feedback from light touch contribute to the scaling of postural corrections to external perturbations to balance. However, the lack of effect on the latency of the evoked responses indicates that the additional cutaneous inputs either are not involved in detecting the disturbance (i.e. triggering the response) or that other sensory cues, perhaps in closer proximity to the source of the disturbance, are better suited for the earliest initiation of a response. What is not clear is if the feedback from the light touch contact point would be capable of triggering rapid, automatic postural corrections. Indeed, it has been demonstrated that electrical stimulation of the median nerve generates interlimb reflexes suggesting the pathways required to trigger short-latency responses to a touch disturbance are present (Delwaide and Crenna, 1984; Kagamihara et al., 2003).

There are many contexts in which the hands and arms are engaged in stabilizing balance, such as when holding a railing or using a cane. It would therefore be logical for the interface between the external support and the body to be a source of sensory information capable of detecting disturbances or threats to the stability that interaction is meant to provide. For example, it has been shown that rapid pulls applied to a set of handles leads to automatic balance corrections during both standing (Cordo and Nashner, 1982) and walking (Nashner and Forssberg, 1986; Forero and Misiaszek, 2014). There are several sensory cues that could be involved in triggering these corrections, including the fast conducting muscle stretch receptors. However, the first point at which a disturbance could be detected is at the skin in contact with the handles. Given the extent of literature demonstrating the powerful effect light touch can have on postural control we speculated that tactile feedback from the hands could be used to detect disturbances to balance and trigger automatic corrective reactions.

Although an attractive option, the temporal relationship between light touch sensory cues and postural adaptations appears to be too slow for such a role. In studies of the effect of a fixed light touch contact the lag between the shear forces at the contact point and the sway of the body are reported to be approximately 350 ms (Jeka and Lackner, 1994). Whereas, when an oscillating light touch reference is entraining sway, the lag is reported to be approximately 176 ms (Wing et al., 2011). The shorter lag was attributed to a closer contact between finger and the touch interface, thereby creating a stronger sensory activation. Although substantially shorter than in the fixed contact paradigm, this delay is still longer than what would be associated with automatic postural corrections which typically range between 80-120 ms (Horak and Macpherson, 1996). Wing et al (2011) used slow oscillations of 0.3 Hz and 0.5 Hz which are appropriate for investigating control of sway. However, automatic postural corrections are typically evoked using rapid, episodic disturbances. Presumably, a faster disturbance at the contact point would activate a more robust sensory signal. We therefore asked, can rapid, episodic displacement of a light touch contact trigger automatic postural corrections?

Specifically, we hypothesized that rapid displacement of a light touch contact would evoke short-latency (<120 ms) responses in a direction specific manner in either tibialis anterior or soleus, consistent with a response to a perceived backward or forward fall.

Experimental Procedures

Participants

Twenty six (14 female, 12 male) healthy young adults ranging in age between 21 and 32 years old volunteered to participate in these studies. The participants ranged in height from 157 to 186 cm and ranged in weight from 54 to 89 kg. All participants were self-reported as right-hand dominant. The volunteers provided written consent of their participation in a protocol approved by the University of Alberta Research Ethics Board.

Set-up and Apparatus

Participants stood on a 6 component force plate (AMTI OR6-7-1000) in normal bipedal stance with the feet shoulder width apart, or what was deemed comfortable by the participant. Participants were instructed to stand quietly, as they would normally, but to try to maintain equal weight distribution between their feet. Participants wore comfortable, flat soled shoes. We were primarily interested in observing responses of the participants to unexpected displacement of a light touch contact. Therefore, in some tasks the participants were asked to lightly touch a touch plate that could be rapidly displaced. The touch plate was a 3.75 cm wide x 7.5 cm long brushed aluminum plate mounted on a pair of steel rods that permitted the height of the plate to be adjusted to the participant's height. The touch plate was positioned so that the participant was able to place only the tip of the index finger in the center of the plate, with the finger bent such that the distal phalanx was vertically aligned so as to avoid the whole finger resting on the plate and detecting the edge (Fig. 1A). When touching the touch plate the right arm of the participant was maintained with a horizontal posture of the forearm and a vertical alignment of the upper arm, such that the elbow was at approximately 90º of flexion. The other fingers of the right hand were curled into the palm to prevent inadvertently coming into contact with the touch plate. When participants were asked to not touch the touch plate the right arm was free to hang in a relaxed posture at their side. The left arm was free to hang at their side throughout. To produce the linear displacement the touch plate was mounted on a square rail acme screw drive positioning stage (LinTech 130 Series), driven by a two-phase stepper motor (Applied Motion Products 5023-124 2-phase hybrid step motor). Stage position was measured using a linear displacement sensor (Penny & Giles SLS130). The entire touch plate positioning apparatus was mounted on a 6 component force transducer (AMTI MC3A-100) to measure the vertical load applied by the participant when touching the touch plate. The relatively mild noise generated by the operation of the motor and the positioning stage was masked by asking the participants to wear over-the-ear noise-cancelling headphones through which white noise was delivered. The participants wore the headphones and received the white noise throughout the testing. In addition, the operation of the motor during the slow oscillations (see section *2.3 Protocol*) introduced a perceptible vibration. To prevent the participants from anticipating the nature of the study vibrations of the touch plate were introduced by the motor in all conditions that included light touch. No safety harness was worn by the participants, but they were made aware that a spotter was present.

Protocol

In order to emphasize the unexpected nature of the displacement the participants were exposed to a series of testing conditions. In the first task, participants were asked to stand normally with their eyes open and their hands at their sides. In the second task, participants were asked to stand with their eyes open whilst lightly touching (< 1 N) the touch plate with their right index finger. The participants received an auditory cue if the vertical force applied to the touch plate exceeded 1 N. In the third task, participants were asked to stand with their hands at their sides, but with their vision occluded by a pair of darkened goggles. Each of these pre-test tasks were performed for 2 min and served primarily as a deception to the nature of the study.

In the fourth task, participants were asked to stand with their vision occluded whilst lightly touching the touch plate. However, during this condition the touch plate oscillated at 0.4 Hz anterior-posteriorly (A-P) through a peak to peak range of motion of 5 mm. The touch plate was oscillated in this fashion because it is well documented that many reflexes and evoked responses are modulated across a cycle of rhythmic movement. We anticipated that it would be important to ensure the rapid displacements were delivered at the same point in the sway cycle of the participant to ensure a comparable stimulus was delivered across repeated trials and across participants. It has been documented that slow oscillations of <0.5 Hz are capable of subliminally entraining sway (Wing et al., 2011). After approximately 30 s of the slow oscillation the touch plate was rapidly displaced 12.5 mm with a peak velocity of 124 mm/s (Fig. 1B). Following this initial rapid displacement an additional nine displacement trials (in the same direction) were obtained. Between successive trials the touch plate was slowly returned to the starting position whilst the participant continued to touch the plate and then oscillated as above, but only for between 2-8 cycles before the next displacement was presented. The number of cycles was randomized to avoid the participants anticipating the timing of the rapid displacements. The fourth task lasted approximately 10 min.

Initially we anticipated testing responses to the first displacement of the touch plate in both the forward and backward directions. To standardize the timing of the displacements in the sway cycle, the forwards perturbations were delivered at the forward end of the oscillation cycle and the backwards perturbations were delivered at the backward end of the oscillation cycle. In an initial 12 participants, 6 received forward touch displacements and 6 received backward touch displacements. However, the data from these first 12 participants suggested that forward displacements were more likely to induce responses consistent with a balance correction than were backward displacements (see Results). Therefore, a subsequent 14 participants were exposed to only the forward perturbations in an effort to maximize the opportunity to characterize these evoked responses. Consequently, a total of 20 participants received forward touch displacements and 6 received backward touch displacements.

Data Collection and Analysis

In addition to the force plate outputs, electromyogram (EMG) recordings were obtained from 4 muscles of the right arm and 2 muscles of the right leg. EMGs were obtained from the anterior deltoid (AD), posterior deltoid (PD), biceps brachii (BB) and triceps brachii (TB) in the arm; and the tibialis anterior (TA) and soleus (SOL) of the leg. A pair of Ag-AgCl surface electrodes (NeuroPlus A10040) was placed over the bellies of the muscles, aligned to the predicted path of the muscle fibres, with an inter-electrode distance of 2 cm. Ground electrodes were placed over the clavicle and tibia. The EMG signals were amplified and band-pass filtered (30 Hz – 1 kHz, Grass P511 amplifiers, Astro-Med) prior to digitization. Electrogoniometers

(Biometrics, Newport, UK) were placed across the right elbow and ankle joints. All signals were digitized with a sampling rate of 4 kHz and stored directly to hard drive using a 12-bit National Instruments data acquisition card (National Instruments PCI-MIO-16E-4) with a custom-written LabView v8.2 data acquisition routine. Post-processing of the signals was performed offline using custom-written LabView v8.2 routines. The EMG signals were digitally full-wave rectified and then low-pass filtered at 50 Hz (zero-lag 2^{nd}-order Butterworth filter). EMG amplitudes were normalized to the maximum voluntary contraction (MVC). The MVC EMG recordings were obtained by asking participants to perform isolated isometric contractions against resistance. Two attempts were performed for each muscle. The largest amplitude of the rectified and filtered EMG signal averaged over a 50 ms window was taken as the MVC value. The mechanical signals (forces, moments, electrogoniometers, linear displacement) were digitally low-pass filtered at 20 Hz. The anterior-posterior (A-P) and medial-lateral (M-L) positions of the CoP were calculated from the force and moment signals from the force plate.

To determine if the displacement of the touch plate evoked responses in the recorded muscles a two standard deviations band around the mean EMG activity for the 100 ms prior to the perturbation onset was calculated. A response was considered to be present in the EMG of a muscle if following the onset of the perturbation the EMG trace exceeded this band for at least 20 continuous milliseconds. The onset latency of an evoked response was taken as the time following the perturbation onset that the EMG trace first exceeded the two standard deviations band. Given that short latency responses (i.e. <120 ms) in the EMG activity were the primary focus of this study, any responses with onset latencies >200 ms were not considered for further analysis. Response amplitudes were calculated as mean rectified EMG for the first 75 ms following response onset, less the mean background EMG amplitude for the 50 ms prior to the touch plate displacement. The same method was used to determine if the perturbation of the touch plate induced a demonstrable mechanical event in the motion of either the A-P or M-L CoP or the motion at the elbow or ankle, with a limit to the onset latency of 350 ms.

As will be described in the Results, most participants responded to the unexpected touch displacement. However, the participants could be separated into 4 groups based upon whether a response was evoked in the muscles at the 1) ankle, 2) shoulder, 3) both ankle and shoulder, or 4) no response on the first exposure to the perturbation. Moreover, those participants that responded to the first perturbation with a response at the ankle invariably responded at the shoulder with subsequent exposure to the perturbation. Visual inspection of the data traces between the groups suggested when participants responded with a response at the shoulder muscles, the motion of the elbow joint was entrained with the motion of the touch plate. To better characterize the behaviour of the participants leading up to the onset of the perturbations cross-correlation functions were calculated for the touch plate position (driving signal) against each of the mechanical outcome measures for the 2500 ms immediately preceding the perturbation. The peak correlation coefficient (regardless of sign) with a lag time within ± 500 ms was extracted. In addition, the average background EMG activity in each of the muscles, as well as the vertical component of the touch force, was calculated for the 50 ms preceding the perturbations.

Statistics

The primary focus of this study was identifying the presence and describing the characteristics of responses evoked by the touch displacements. The method for identifying the presence of an evoked response was described earlier. Description of the responses included latencies of the responses in a given muscle, relative to other muscles or mechanical events. In all instances, group means are reported with standard deviations. Student *t*-tests were used to compare the temporal characteristics of the responses, response amplitudes, background EMG amplitudes and the vertical component of the touch force between the initial TA and initial AD responders. Comparisons of outcome measures between the 1^{st} and 10^{th} trials from within participants were made using paired *t*-tests. Student *t*-tests were also performed on the peak correlation coefficients after first being transformed to Fisher's *z*. (Note that data are reported in the text and in Fig. 6 as the untransformed correlation coefficient *r* statistic for clarity of interpretation.) All comparisons were made with $\alpha = 0.05$.

Results

The rapid displacement of the touch plate evoked responses in the recorded EMGs of 21 of 26 participants tested. However, responses were not consistent across subjects. Rather, participants could be classified into one of four groups; those that produced responses in muscles at 1) the ankle, 2) the shoulder, 3) both the ankle and shoulder, or 4) no response. In an initial 12 participants, 6 received forward displacements of the touch plate while the other 6 received backward displacements. Of those participants that received a forward displacement, all 6 generated some form of response to the initial perturbation. In contrast, the backward displacements evoked responses in the recorded EMGs of only 2 participants, with 1 activating SOL and 1 activating PD following the initial displacement. Sample data from participants that received forward displacements are depicted in Figs. 2A and B and another participant that received backward displacements is depicted in Fig. 4. Given the high response rate to forward displacement and low response rate to backward displacement, an additional 14 participants were tested with only forward displacements so that a total of 20 participants received forward perturbations. Of these 20 participants, the initial forward displacement evoked responses in 19 participants, with 9 activating TA, 7 activating AD, and 3 activating both TA and AD.

Responses to the First Forward Displacement

Fig. 2A depicts the data from one participant following an unexpected, rapid forward displacement of the touch plate. The response to the initial perturbation (left column of traces) shows a clearly visible response in TA with an onset latency 102.75 ms, with a subsequent (156.25 ms) appearance of a forward displacement in the CoP_{A-P} trace. In contrast, data from a second participant (Fig. 2B) depicts a clearly visible response in AD with an onset latency of 104.75 ms, no apparent displacement of the CoP, but a later extension of the elbow at 216.5 ms following the initial forward displacement of the touch plate. The first trial responses depicted in Fig. 2A and 2B are characteristic of the responses evoked in the initial TA activating group and the initial AD activating group, respectively. Responses in other muscles of the arm were often observed in the AD activating group, however, which other muscles were activated and their response latencies varied considerably. In 3 participants, the first exposure to the forward touch plate displacement resulted in activation of both the TA and AD (not shown).

Across participants, the onset latencies in TA ranged from 53.5 ms to 115.0 ms for the initial TA activating group. Onset latencies in AD ranged from 48.25 ms to 93.25 ms for the AD activating group. In the 3 participants that responded with activating both TA and AD, the onset latencies for the responses in TA ranged between 61.25 ms and 121.75 ms, whereas the latencies for the AD responses ranged between 55.75 ms and 71.5. Given that there was no apparent difference in onset latency in TA or AD between the groups (those that responded with either TA or AD alone, or those that responded with both TA and AD), the data were combined to compare the onset latencies between TA and AD (Fig. 3A). The mean (±s.d.) onset latency in TA was 85.2 (±25.4) ms, while the mean onset latency in AD was 66.0 (±15.4) ms. The onset latencies in AD were significantly shorter than in TA (t = 1.99, p = 0.049). Each participant that responded with an activation of TA also showed a forward displacement in the CoP, with a mean onset latency of 156.4 (±27.1) ms. In contrast, only 4 of the 10 participants that responded with AD on the initial disturbance exhibited a measurable extension of the elbow within 350 ms of the perturbation, with a mean latency of 188.8 (±54.0) ms. No measurable changes in ankle angle were detected following the perturbations.

Responses to Subsequent Forward Displacements

Responses in TA were only observed in response to the first exposure of the touch plate displacement. Moreover, all participants that responded with an initial TA response subsequently responded with activation of AD. For the participant whose data are depicted in Fig. 2A, by the 10^{th} exposure (right column of traces) to the touch plate displacement the evoked response appears remarkably similar to the 1^{st} trial response of the participant in Fig. 2B. In this instance, the response onset in AD on the 10^{th} trial was 77.75 ms, with a noticeable absence of the forward displacement of the CoP_{A-P}.

The appearance of the AD response on subsequent trials was not immediate in all participants. For the participant depicted in Fig. 2A, the response in AD appeared to emerge over about 4 trials, before a relatively consistent response was expressed in later trials (Fig. 2C). In other participants, the response in AD emerged immediately on the second exposure. Moreover, responses in AD were not expressed in every subsequent trial, both for the initial AD activating group and the initial TA activating group. However, of the 180 non-first trials across all participants (9 trials per participant, excluding the first trial), responses were observed in AD 133 times, or 74% of the time. This includes the 1 participant that never responded to the touch plate displacement during any trial. Of the 10 participants that responded with a burst in AD with the first trial exposure (7 AD alone and 3 AD/TA combined), 7 expressed AD responses in the 10^{th} trial. In these 7 participants, the mean onset latency of the first trial AD response was 62.0 (±14.3) ms, while that of the 10^{th} trial was 97.1 (±34.0) ms (Fig. 3B). A paired *t*-test indicated that for these 7 participants the response onset in AD in the 10^{th} trial was significantly later than the response in the 1^{st} trial (t = -4.12, p = 0.006). However, AD response amplitudes were not different between the 1^{st} and 10^{th} trials in these participants (t = 0.29, p = 0.76) with mean amplitudes of 6.62 (±5.28) %MVC and 5.65 (±5.06) %MVC, respectively. In addition, the mean response onset in AD of the 10^{th} trial for those participants that initially responded with TA alone was 102.4 (±34.0) ms (Fig. 3C). A Student's *t*-test indicated that this was not significantly (t = 0.24, p = 0.76) different from the onset latency for the AD response in the 10^{th} trial for the initial AD responders.

Responses to Backward Displacements

Fig. 4 shows the response in one participant that received backwards displacement of the touch plate. The left column of data represents the response to the initial trial for this participant. This participant responded to the initial, unexpected backward displacement of the touch plate with a short latency (82.0 ms) activation of SOL, with a subsequent (181.75 ms) backward displacement of the CoP_{A-P} position. Similar to the participants that responded with TA activation following forward perturbations, subsequent exposures of the backward perturbation did not evoke a response in SOL. Rather by the 10^{th} trial (right column of traces) a response in

PD was evoked with a latency of 92.5 ms. In addition, 1 other participant responded to the initial backward displacement of the touch plate with an activation of PD at 79.0 ms (not shown).

Background EMG, vertical touch force and cross-correlations

Comparisons of the background EMG in TA and AD prior to the onset of the first exposure to the forward perturbation revealed no differences between those participants that initially responded with TA and those that responded with AD. The mean background activity in the 50 ms prior to the perturbation was 3.53 (±1.49) %MVC and 3.25 (±4.14) %MVC in TA for the initial TA and initial AD responders, respectively ($t = 0.17$, $p = 0.85$). Whereas the mean background activity in AD for the two groups was 4.03 (±1.82) %MVC and 3.23 (±1.56) %MVC, respectively ($t = 0.86$, $p = 0.36$). In addition, the background EMG activity in the TA responders was not different with repeated exposures of the perturbation as the background activity in TA on the 10th trial was 3.60 (±1.24) %MVC and the background activity in AD on the 10th trial was 3.28 (±1.58) %MVC. Paired *t*-tests did not reveal a significant difference between the first and 10th trials for either TA ($t = 0.23$, $p = 0.82$) or AD ($t = -1.03$, $p = 0.33$).

The vertical touch force was monitored throughout the data collection and maintained below 1 N. However, the vertical touch force varied from trial to trial and across participants within a range between 0.25 N to 0.92 N. The mean light touch force at the time of the first exposure to the forward touch displacement in the initial TA responders was 0.54 (±0.17) N and 0.74 (±0.15) N in the initial AD responders. This difference between groups was significant ($t = -2.19$, $p = 0.034$). In addition, a paired *t*-test comparing the vertical touch force at the time of the 1st and 10th trials (0.69 (±0.17) N) in the initial TA responders revealed a significantly heavier touch after repeated exposure to the perturbation ($t = -2.56$, $p = 0.036$). In contrast, a paired *t*-test comparing the 1st trial and the 10th trial (0.70 (±0.18) N) showed no difference in touch force with repeated exposure to the perturbation in the initial AD responders (t = 0.39, p = 0.64).

Fig. 5A depicts data traces from the 1st and the 10th exposures to a forward touch perturbation from a participant that responded with TA initially. Visual inspection of the data suggests that this participant was swaying forwards and backwards with the oscillation of the touch plate prior to the first perturbation (as seen in the CoP_{A-P} and Ankle traces), but displayed a rhythmic motion of the arm in the 10th trial (as seen in the Elbow trace). By comparison, the data from a participant that responded with AD following the initial touch plate displacement (Fig. 5C) appears to show a rhythmic oscillation of the arm (Elbow trace) that is consistent with the motion of the touch plate. To obtain a more objective measure cross-correlation functions were computed for one cycle of touch plate oscillation prior to the onset of the rapid perturbation (Figs. 5B,D). From the cross-correlograms depicted in Fig. 5B positive peaks appear in both the CoP_{A-P} and Ankle functions in the 1st trial, with a slightly positive lag indicating the touch plate motion led the oscillation in these traces. In contrast, in the 10th trial the Elbow function displays a prominent positive peak with dampened peak correlations in the CoP_{A-P} and Ankle traces. In Fig. 5D, the Elbow cross-correlation demonstrates pronounced positive peaks in both the 1st and 10th trials, while the Ankle functions display dampened peak correlations. The CoP_{A-P} functions show negative peak correlations in closer proximity to a zero lag, suggesting oscillations that are anti-phase with the touch plate. (Positive peaks located closer to 1 s lag times reflect the ½ cycle length of the 0.4 Hz oscillation.)

To characterize the difference in the signal correlations the peak correlation coefficient, regardless of sign, was extracted from a window within a lag of ± 500 ms. The data from each of the initial TA responders and initial AD responders are depicted in Fig. 6. Note that in this figure the correlation coefficients (*r*), rather than the Fisher *z* transformed data, are shown for each participant. As can be seen, the peak *r* values for Ankle and $CoP_{A\text{-}P}$ data of the initial TA responders show considerable spread with values ranging from -0.70 to 0.82 and -0.78 and 0.90, respectively. The spread of *r* values for the Ankle and $CoP_{A\text{-}P}$ data for the initial AD responders are quite similar, ranging between -0.57 to 0.85 and -0.52 to 0.89, respectively. In contrast, the *r* values for the Elbow correlations are distinctly different between the initial TA responders and the initial AD responders. The Elbow *r* values range between -0.45 to 0.48 for the initial TA responders, with no values greater than 0.5 (absolute). In contrast, the *r* values for the initial AD responders range from 0.61 to 0.90, with no negative correlations extracted. The difference in the Elbow peak *r* values between the initial TA and AD responders was significant ($t = -4.48$, $p < 0.001$). Moreover, the Elbow peak *r* values for the 10th trials of the initial TA responders ranged from 0.62 to 0.89, with no negative correlations. This difference between the 1st and 10th trials in the initial TA responders was also significant (paired $t = -4.77$, $p < 0.001$). The peak *r* values for the Elbow correlations of the AD responders (Fig. 5D) and the 10th trial of the TA responders (Fig. 5B) occurred very close to a zero lag. The average lags for the Elbow peak *r* values were 25.3 (±17.2) ms and 11.3 (±9.4) ms for the 1st and 10th trials respectively for the initial AD responders; and 25.6 (±22.9) ms for the 10th trial for the initial TA responders.

Discussion

The primary question addressed in this study was whether sudden, unexpected displacement of a light touch surface at the fingertip could induce a rapid (< 120 ms) balance correction in standing participants. In short, the answer to the question is yes with approximately half of the study participants demonstrating a short-latency activation of ankle muscles in response to the first exposure of a sudden displacement of a touch plate. However, not all participants responded with activation of the ankle muscles and, moreover, no participant responded with activation of the ankle muscles after the first exposure to the touch plate displacement.

Are the initial responses in TA (and SOL) balance reactions?

In 12 of 20 participants the initial exposure to a rapid forward displacement of the touch plate resulted in a short-latency (< 120 ms) response in TA. We interpret this short latency activation of TA to be part of a balance corrective response initiated by the unexpected displacement of the touch reference. A rapid relative forward displacement of the touch plate could be produced by either the displacement of the touch plate itself, as was used in this study, or by a backward displacement of the finger, as would occur with a backward sway. It is well established that short-latency activation of TA is often observed following perturbations of posture that induce a backward sway (Horak and Macpherson, 1996). In addition, the responses in TA observed in this study occurred with an average onset of 85 ms, well within the cited range for TA onset times for balance corrections. Moreover, the evoked responses in TA typically preceded a subsequent forward sway (Fig. 2A), consistent with what would be expected to correct for backward sway. Alternatively, the activation of TA and the subsequent forward sway could be argued to be a strategy employed to maintain the position of the finger relative to the touch plate. However, in all subsequent trials this TA activation was absent and usually replaced by activation in AD and a concomitant elbow extension, which we suggest is part of a strategy to track the position of the touch plate and maintain the contact of the finger. Although tracking the touch plate position by forward body sway is one plausible solution to the problem, moving only the arm would be more efficient. Moreover, the activation of TA and the subsequent forward sway to accomplish this would itself be potentially destabilizing. We suggest that the evoked responses in TA induced by the disturbance at the fingertip were balance corrections to a perceived backwards sway.

At the conclusion of the experiment we asked each participant to describe what they experienced. All of the participants reported that they realized the touch plate had moved (note that none of the participants were aware of the slow oscillations), but some reported it required more than one trial to reach that conclusion. Two participants reported believing that the force plate had moved beneath them on the first trial and one participant reported, including by vocalizing during the trial, believing they had been pushed, even though we made no physical contact with any of the participants. These three participants all responded with activation of TA on first exposure of the touch plate displacement. Although these participant reports were informal and lack rigour, they provide compelling anecdotes that in at least some of the participants the touch displacement was initially perceived as a balance disturbance.

First Trial Responses

During daily life unexpected threats to balance often occur under unique, single event circumstances. The first exposure to a balance threat is therefore often the only exposure that an individual will experience. Nevertheless, balance reactions in these situations require the production of a response that will prevent a fall, or minimize the potentially catastrophic consequences of an impending fall. Allum et al (2011) point out that due to the unexpected nature of these types of disturbances they are often startling experiences, with exaggerated responses. These exaggerated responses can be seen as larger amplitude muscle activities, or activity in muscles not seen in subsequent exposures to the same disturbance (Nashner, 1976;

Siegmund et al., 2008). A common feature of these first trial responses is habituation, or attenuation, of the responses with repeated exposure to identical disturbances (Allum et al., 2011; Siegmund et al., 2008). In our study, we observed two distinct groups of participants; those that responded to the first exposure of the forward touch plate displacement with activation of TA and those that did not. Invariably, the TA responders only reacted with TA activation upon the first trial. There was not a gradual habituation of the response in TA, it simply was not observed again in any of the subsequent trials. This is different from what has been described in other studies documenting a more gradual habituation of the first trial responses. However, in our study, the disturbance used was not mechanically destabilizing and posed no actual threat to stability. It is therefore likely that after the first exposure to the touch plate disturbance the participants recognized the nature of the disturbance and no longer recruited a balance corrective strategy in response to the disturbance. In other words, subsequent trials in our study no longer recruited an inconsequent strategy. Nevertheless, the results from our study highlight the importance of studying first trial responses to disturbances as clearly some participants responded to the first touch displacement with a very different strategy than they did in later trials or than did other participants.

Given the startling experience that unexpected disturbances can have, it has been suggested that some of the exaggeration of the first trial responses can be attributed to the superimposition of a startle response with a postural response, and that the habituation of the first trial responses is then due to the extinction of the startle component (Siegmund et al., 2008). Could then the response in TA observed in some participants to the initial forward touch displacement arise from a startle response in our study? This seems unlikely. Startle responses are typically associated with more pronounced activation in muscles of the upper body, with less obvious activation of lower body muscles (Brown et al., 1991; Oude Nijhuis et al., 2010). Therefore, the activation of TA initially, with subsequent activation of AD, when extinction of the startle response would be expected to explain the elimination of the TA response, would be contrary to this expectation. Moreover, the initial TA response appears to be replaced by a response in AD that shares common characteristics to the AD response evoked in some participants following the initial disturbance. In addition, the responses appear to be direction specific. In one participant an initial backward displacement of the touch plate resulted in activation of SOL, with activation of PD on subsequent trials (Fig. 4). The directional specificity of the responses evoked at the ankles makes it unlikely that these responses, the components of the responses that extinguish, are a superimposed startle response. We therefore argue that the initial responses evoked in TA (and SOL) in some participants are balance responses evoked by the unexpected relative motion of the finger at the touch plate interface.

Context-dependency of responses

Not all participants responded to the initial rapid forward displacement of the touch plate by activating TA. Rather, some participants responded to the disturbance by activating AD in what appears to be a strategy of tracking the position of the touch plate with a forward arm motion. Indeed, all participants invariably used this arm tracking strategy on subsequent trials. Two observations arising from our data appear to be related to the expression of the AD response. First, in all instances when a response was evoked in AD there was a strong correlation between the motion of the touch plate and movements at the elbow prior to the rapid perturbation. There was a positive lag of less than 30 ms between the motion of the touch plate and the oscillation of the elbow. A lag this short indicates that the correlated motion at the elbow with the touch plate is likely due to mechanical coupling between the touch plate and arm. Moreover, the entrainment of the body sway to the subliminal touch plate oscillations was consistent between the trials that evoked TA responses and AD responses. There was some degree of entrainment of the CoP_{A-P} position in all participants prior to the first rapid displacement, including participants in both groups that swayed out-of-phase. Therefore, although the touch cue appeared to be integrated into the control of the sway to an equal extent in all participants, the distinguishing feature when a response in AD was expressed was the relatively strong correlation of the elbow motion with the touch plate. Second, the coupling of the elbow motion with the touch plate oscillation was related to higher vertical touch forces. Participants that responded to the rapid displacement of the touch plate with a response in AD applied a significantly higher touch force prior to the displacement than when a response in AD was not evoked. Indeed, initial TA responders applied a touch force of close to 0.5 N, while initial AD responders and later trials of initial TA responders applied a touch force of 0.7 N or more. It is likely that these two events are related. A greater vertical force would result in a greater coefficient of friction at the touch plate. This increase in the coefficient of friction could allow for sufficient adherence of the finger to the touch plate to create a mechanical coupling during the slow oscillation of the touch plate prior to the rapid displacements. The resulting motion of the arm would then presumably drive sensory feedback from a variety of sources, including proprioceptors throughout the arm in addition to increased tactile inputs from the finger related to the increase in touch force. In contrast, in the absence of the mechanically coupled arm movement, the touch plate was likely slipping beneath the finger generating a very different sensory profile of the events at the finger-touch plate interface. Presumably, the entrainment of the arm motion provides sufficient sensory input to engage a focal arm response to the displacement of the touch plate, whereas in the absence of the arm motion a presumptive balance reaction is evoked. It is important to note that although all participants became aware of the rapid displacements of the touch plate, none of the participants were aware of the slow oscillations even after the study when they were informed of the details of the protocol.

It is also important to note that the rapid displacement of the touch plate did not directly lead to a rapid extension of the elbow. The elbow extension observed following the perturbations occurred with a delay and after the onset of the AD burst (Fig. 2). The increase in touch force applied by the AD responders was therefore not sufficient to increase the coefficient of friction to prevent the finger from slipping on the touch plate during the rapid displacements. Consequently, the activation of the response in AD is likely initiated by the tactile cues from the fingertip arising from the slip, just as the presumptive balance reaction in TA is likely initiated by the tactile cues from the fingertip. Thus, tactile cues from the fingertip appear to be sufficient to initiate short latency (<120 ms) reactions of differing functional effect, depending upon the context within which the sensory cue is experienced. The most likely explanation for the context dependent nature of the responses in this study is whether or not the participants anticipated the touch plate could move. Presumptive balance reactions were only ever seen on the first trial in some participants. In contrast, arm reactions were observed on subsequent trials in all participants and on the first trials in some. Arm reactions were related to higher touch forces. Notably, applied touch forces were increased in later trials for those participants that initially reacted with a presumptive balance reaction. The increase in touch force appears then to be a strategy employed by participants to increase the sensory information available about the fingertip-touch contact interface and might indicate that participants were anticipating the touch plate could move. Previously, it has been shown that older adults, with decreased sensitivity of the fingertips, also apply a greater vertical touch force when using a touch reference (Tremblay et al., 2004), suggestive of a strategy to increase the sensory feedback derived from the fingertip-touch contact interface. In this study we utilized four task conditions, the first three (standing normally with eyes open, standing with eyes open whilst touching the stationary touch plate, and standing with eyes closed) were intended as a deception to prevent the participants from anticipating that the touch plate could be displaced. However, we suspect that the deception was incomplete in those participants that responded with an AD burst with the initial disturbance. The similarity of responses in AD across participants once they were aware the touch plate would move suggests that participants then anticipated the touch plate displacement.

Previously, it has been suggested that context-dependency of motor responses, including postural reactions, is a common feature of motor control. Prochazka (1989) defined the concept of sensorimotor set as "… a state in which transmission parameters in various sensorimotor pathways have been adjusted to suit a particular task or context." In other words, identical sensory cues can lead to disparate motor responses due to the specific requirements of the task being performed. Thus there are a number of potential solutions to any problem experienced, but the most probable solution is preselected based upon the available evidence and "tunes" the neural circuitry to execute the corresponding motor responses (Prochazka, 1996; Misiaszek, 2006). In this study, the motor response following the rapid slip of the touch contact at the fingertip is selected based on whether the participants expect a slip at the fingertip is due to their sway relative to a stable reference or if they expect the reference itself moves. The increase in touch force and the related oscillating motion of the arm prior to the perturbations might have been strategies employed in some participants initially, and all participants subsequently, to obtain more information about the environment and context within which they found themselves to provide a more reliable sensory profile. In contrast, the initial TA responders, with their lighter touch force, likely trusted that the touch plate provided a stable reference and therefore obtained minimally required sensory input to achieve what they perceived to be the required task of standing stably.

Neural mechanisms of the presumptive balance reaction

The rapid displacement of the touch plate did not produce a concomitant displacement of the elbow, even when the elbow motion was oscillating with the touch plate prior to the rapid displacement. Therefore, it is likely that the velocity of the rapid displacement overcame the frictional forces and moved the touch plate relative to the position of the finger; a slip. Any responses to the rapid displacement of the touch plate are therefore unlikely to be related to muscle stretch reflexes or other proprioceptive feedback from the arm. Although we cannot specifically rule out muscle receptors from wrist muscles or the intrinsic muscles of the hands (Marchand-Pauvert et al., 2000), the most likely cue is from tactile information related to the slip between the fingertip-contact surface interface. The responses evoked in this study, whether in muscles at the ankle or arm, were direction specific. Srinivasan et al. (1990) demonstrated that only slowly adapting (SA) low threshold mechanoreceptors in the fingerpad responded with a distinct directional bias to slip stimuli. SAII type receptors (Ruffini endings) are sensitive to skin stretch and have been suggested to be important for detection of the direction of slip (Abraira and Ginty, 2013), making them the most likely candidates to trigger the direction-specific responses we observed. However, rapidly adapting type I (RAI) mechanoreceptors (Meissner corpuscles) and Pacinian corpuscles (PC) are very sensitive to the detection of slip onset and the direction of relative motion across the skin can be encoded by the sequential activation of successive receptive fields when a micro-feature (e.g. a microdot) is present on the contact surface (Srinivasan et al., 1990; Abraira and Ginty, 2013). It is therefore possible that SAII, RAI and PC receptors together combine to encode the onset and direction of the relative motion of the touch plate displacement and trigger the evoked responses.

The SAII, RAI and PC mechanoreceptors are served by afferents in the Aβ range, with average conduction velocities in humans estimated at around 45 m/s for SAII (Knibestol, 1975), 55 m/s for RAI and 47 mm/s for PC receptors (Knibestol, 1973). These then are afferents that would be readily excited when cutaneous reflexes are evoked with electrical stimulation. Electrical stimulation of the median nerve has been shown to produce cutaneous interlimb facilitation of the soleus H-reflex beginning with a delay of about 50 ms and reaching a peak of about 100 ms (Kagamihara et al., 2003). The onset latencies of the responses in TA and SOL in our study were comparable to this ranging between approximately 53 and 120 ms. Moreover, onset latencies of around 50 ms for TA in some participants are suggestive of relatively short conduction routes with few interposed synapses. Cutaneous reflexes likely have a role in regulating stability during functional activities such as walking and standing (Zehr and Stein, 1999), and that interlimb cutaneous reflexes might be particularly relevant in coordinating the actions of the arms and legs in response to unexpected disturbances (Zehr and Duysens, 2004). Previously, we showed that interlimb reflexes from the median nerve are facilitated when fingertip light touch is used during walking, suggesting this interlimb reflex is functionally relevant in the control of standing when the finger is providing a supplemental sensory source (Forero and Misiaszek, 2015). Taken together, it is attractive to suggest that interlimb cutaneous reflexes arising from activation of SAII, RAI and PC receptors contribute at least a portion to the earliest components of the presumptive balance reaction evoked following a rapid displacement of the touch plate.

Conclusions

Light touch of a stable reference has been argued to provide additional sensory inputs that are integrated in the postural control system to help stabilize balance. Our findings suggest that tactile cues associated with the rapid slip of the touch reference are a potential sensory cue for triggering balance reactions, at least in the absence of vision. It is also important to acknowledge that the importance of this potential sensory cue to triggering balance reactions is likely dependent in part on the relevance of the tactile inputs in the context of the perceived task. Therefore, when the hands are functionally engaged in providing support for balance, then the tactile cues from the hands would be a useful resource to initiate corrections as the skin of the hand might be the first point of disturbance. For example, we recently demonstrated that the unexpected displacement of a set of handles being used by participants to stabilize their walking on a treadmill leads to rapid responses in the legs to regain balance (Forero and Misiaszek, 2014). The forces applied to the skin of the hands through the handles could provide the earliest cue that a disturbance has occurred. However, it is also important to note that in this study, the displacement of the touch reference did not produce a mechanical disturbance to balance per se. Nevertheless, some participants reacted to the displacement with a presumptive balance reaction with a resultant sway of the CoP. In other words, the response and not the touch plate displacement itself, would be destabilizing in this context and this "false-positive" reaction could potentially have catastrophic consequences (i.e. inducing a fall). Similar balance destabilizing events with sensory stimuli have been observed for vestibular (Day et al., 2002), visual (van

Asten et al., 1988), and muscle proprioceptor (Hayashi et al., 1981) inputs and highlight the multisensory nature of balance control. It is quite possible that “false-positive” reactions contribute to the greater instability observed as individuals age and sensory signals become less reliable. Moreover, this study highlights the importance of applying the correct sensorimotor set, or cognitive awareness, for the sensory cues within a task. The subtle difference of interpreting the relative motion of the contact at the finger (i.e. a slip) as a fall versus a perturbation could have dramatically different outcomes.

Acknowledgements

The authors are grateful to Dr. DF Collins for providing comments on a draft of this manuscript. The work was funded by a grant to JEM from the Natural Sciences and Engineering Research Council of Canada.

References

Abraira VE, Ginty DD. (2013) The sensory neurons of touch. Neuron 79:618-639.

Allum JHJ, Tang K-S, Carpenter MG, Oude Nijhuis LB, Bloem BR. (2011) Review of first trial responses in balance control: Influence of vestibular loss and Parkinson's disease. Hum Mov Sci 30:279-295.

Brown P, Rothwell JC, Thompson PD, Britton TC, Day BL, Marsden CD. (1991) New observations on the normal auditory startle reflex in man. Brain 114:1891-1902.

Clapp S, Wing AM. (1999) Light touch contribution to balance in normal bipedal stance. Exp Brain Res 125:521-524.

Cordo PJ, Nashner LM. (1982) Properties of postural adjustments associated with rapid arm movements. J Neurophysiol 47:287-302.

Day BL, Guerraz M, Cole J. (2002) Sensory interactions for human balance control revealed by galvanic vestibular stimulation. Adv Exp Med Biol 508:129-137.

Delwaide PJ, Crenna P. (1984) Cutaneous nerve stimulation and motoneuronal excitability. II: evidence for non-segmental influences. J Neurol Neurosurg Psychiatry 47:190-196.

Dickstein R, Peterka RJ, Horak FB. (2003) Effects of light fingertip touch on postural responses in subjects with diabetic neuropathy. J Neurol Neurosurg Psychiatry 74:620-626.

Forero J, Misiaszek JE. (2013) The contribution of light touch sensory cues to corrective reactions during treadmill locomotion. Exp Brain Res 226:575-584.

Forero J, Misiaszek JE. (2014) Balance-corrective responses to unexpected perturbations at the arms during treadmill walking. J Neurophysiol 112:1790-1800.

Forero J, Misiaszek JE. (2015) The amplitude of interlimb cutaneous reflexes in the leg is influenced by fingertip touch and vision during treadmill locomotion. Exp Brain Res 233:1773-1782.

Hayashi R, Miyake A, Jijiwa H, Watanabe S. (1981) Postural readjustments to body sway induced by vibration in man. Exp Brain Res 43:217-225.

Holden M, Ventura J, Lackner JR. (1994) Stabilization of posture by precision contact of the index finger. J Vestib Res 4:285-301.

Horak FB, Macpherson JM. (1996) Postural orientation and equilibrium. In: Handbook of Physiology: Section 12, Exercise Regulation and Integration of Multiple Systems. (Rowell LB, Shepard JT, eds) pp 255–292. New York: Oxford University Press.

Jeka JJ, Lackner JR. (1994) Fingertip contact influences human postural control. Exp Brain Res 100:495-502.

Jeka JJ, Oie K, Schoner G, Dijkstra T, Henson E. (1998) Position and velocity coupling of postural sway to somatosensory drive. J Neurophysiol 79:1661-1674.

Kagamihara Y, Hayashi A, Masakado Y, Kouno Y. (2003) Long-loop reflex from arm afferents to remote muscles in normal man. Exp Brain Res 151:136-144.

Knibestol M. (1973) Stimulus response functions of rapidly adapting mechanoreceptors in human glabrous skin area. J Physiol 232:427-452.

Knibestol M. (1975) Stimulus-response functions of slowly adapting mechanoreceptors in the human glabrous skin area. J Physiol 245:63-80.

Kouzaki M, Masani K. (2008) Reduced postural sway during quiet standing by light touch is due to finger tactile feedback but not mechanical support. Exp Brain Res 188:153-158.

Marchand-Pauvert V, Mazevet D, Nielsen J, Petersen N, Pierrot-Deseilligny E. (2000) Distribution of non-monosynaptic excitation to early and late recruited units in human forearm muscles. Exp Brain Res 134:274-278.

Misiaszek JE. (2006) Neural control of walking balance: IF falling THEN react ELSE continue. Exerc Sport Sci Rev 34:128-134.

Nashner LM, Forssberg H. (1986) Phase-dependent organization of postural adjustments associated with arm movements while walking. J Neurophysiol 55:1382-1394.

Nashner LM. (1976) Adapting reflexes controlling the human posture. Exp Brain Res 26:59-72.

Oude Nijhuis LB, Allum JHJ, Valls-Solé J, Overeem S, Bloem BR. (2010) First trial reactions to unexpected balance disturbances: A comparison with the acoustic startle reaction. J Neurophysiol 104:2704-2712.

Prochazka A. (1989) Sensorimotor gain control: A basic strategy of motor systems? Prog Neurobiol 33:281-307.

Prochazka A. (1996) The fuzzy logic of visuomotor control. Can J Physiol Pharmacol 74:456-462.

Siegmund GP, Blouin J-S, Inglis JT. (2008) Does startle explain the exaggerated first response to a transient perturbation? Exerc Sport Sci Rev 36:76-82.

Srinivasan MA, Whitehouse JM, LaMotte RH. (1990) Tactile detection of slip: surface microgeometry and peripheral neural codes. J Neurophysiol 63:1323-1332.

Tremblay F, Mireault A-C, Dessureault L, Manning H, Sveistrup H. (2004) Postural stabilization from fingertip contract: I. Variations in sway attenuation, perceived stability and contact forces with aging. Exp Brain Res 157:275-285.

van Asten WN, Gielen CC, van der Gon JJ. (1988) Postural movements induced by rotations of visual scenes. J Opt Soc Am 5:1781-1789.

Wing AM, Johannsen L, Endo S. (2011) Light touch for balance: influence of a time-varying external driving signal. Philos Trans R Soc Lond B Biol Sci 366:3133-3141.

Zehr EP, Duysens J. (2004) Regulation of arm and leg movement during human locomotion. Neuroscientist 10:347-361.

Zehr EP, Stein RB. (1999) What functions do reflexes serve during human locomotion? Prog Neurobiol 58:185-205.

Figure legends

Figure 1: A. Diagram of the experimental set-up. Participants touched a touch plate with only the pad of their right index finger. The touch plate was mounted on an acme screw driven by a stepper motor to produce linear displacements in the fore-aft direction. **B**. During the perturbation trials the touch plate was first oscillated at 0.4 Hz through 5 mm. Rapid, episodic displacements were then unexpectedly applied to induce a slip at the fingertip.

Figure 2: A. Data traces from one participant demonstrating an initial response in TA following the first exposure to a forward displacement of the touch plate. The 1^{st} trial data are depicted in the left column of traces and the 10^{th} trial data from the same participant are depicted in the right column. The EMG traces are full-wave rectified and filtered, with the horizontal dashed lines representing the zero baseline. **B**. Example response from one participant demonstrating an initial response in AD following the first exposure to a forward displacement of the touch plate. **C**. Individual responses in AD for each successive touch plate displacement for the same participant displayed in **A**. The vertical dashed lines indicate displacement onset. EMG scale bars represent %MVC.

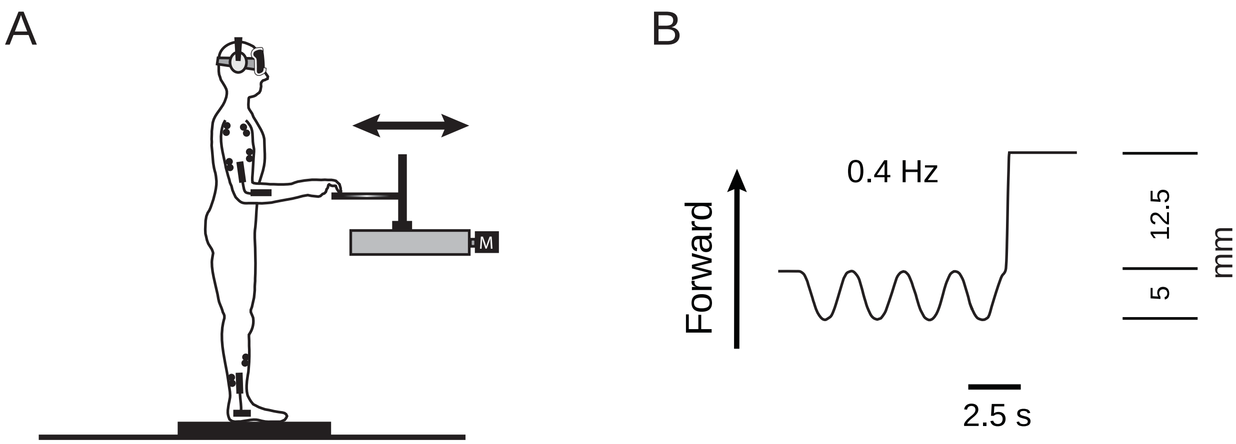
Figure 3: Onset latencies of responses following forward displacements of the touch plate. **A**. First trial responses for initial TA responders (TA_1) compared with initial AD responders (AD_1). **B**. 1^{st} trial responses in AD for initial AD responders (AD_1) compared with their 10^{th} trial responses (AD_{10}). **C**. 10^{th} trial responses in AD from initial TA responders ($AD_{10(TA)}$) compared with 10^{th} trial responses from initial AD responders ($AD_{10(AD)}$). Individual data points are

displayed as the open circles. The horizontal bars indicate the means and the error bars the standard deviations.

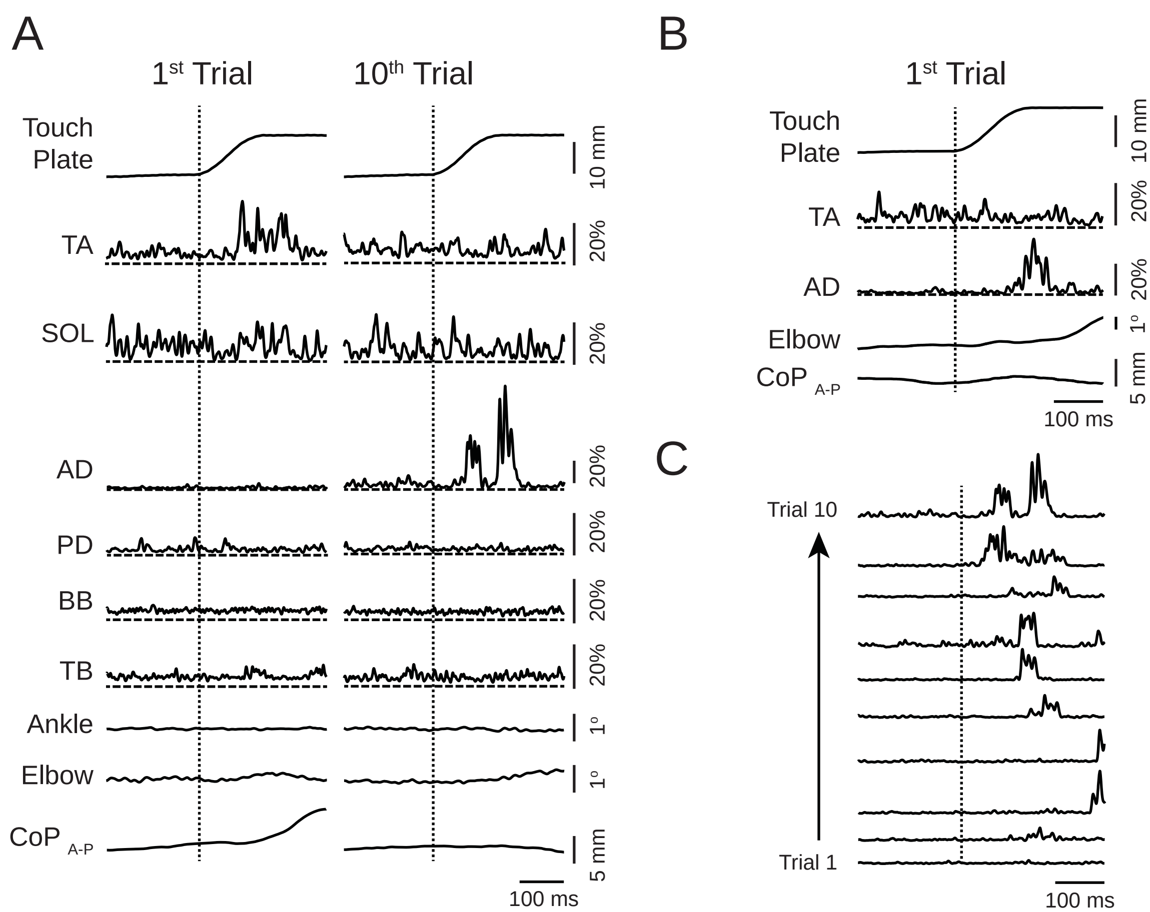
Figure 4: Data from one participant that received an initial backward displacement of the touch plate, demonstrating a clear response in SOL on the first trial (left column) and subsequent response in PD on the 10th trial (right column). The vertical dashed lines indicate displacement onset. EMG scale bars represent %MVC.

Figure 5: **A, C**. Data displaying one full cycle of oscillation prior to the onset of a forward touch plate displacement for a participant that initially responded with TA (A) and a participant that initially responded with AD (C). For both A and C the 1st trial is displayed in the left column and the 10th trial the right column of traces. The vertical dashed lines indicate displacement onset. EMG scale bars represent %MVC. **B, D**. Cross-correlations for the data displayed in A and C, respectively. The vertical dashed lines are aligned to 0 lag.

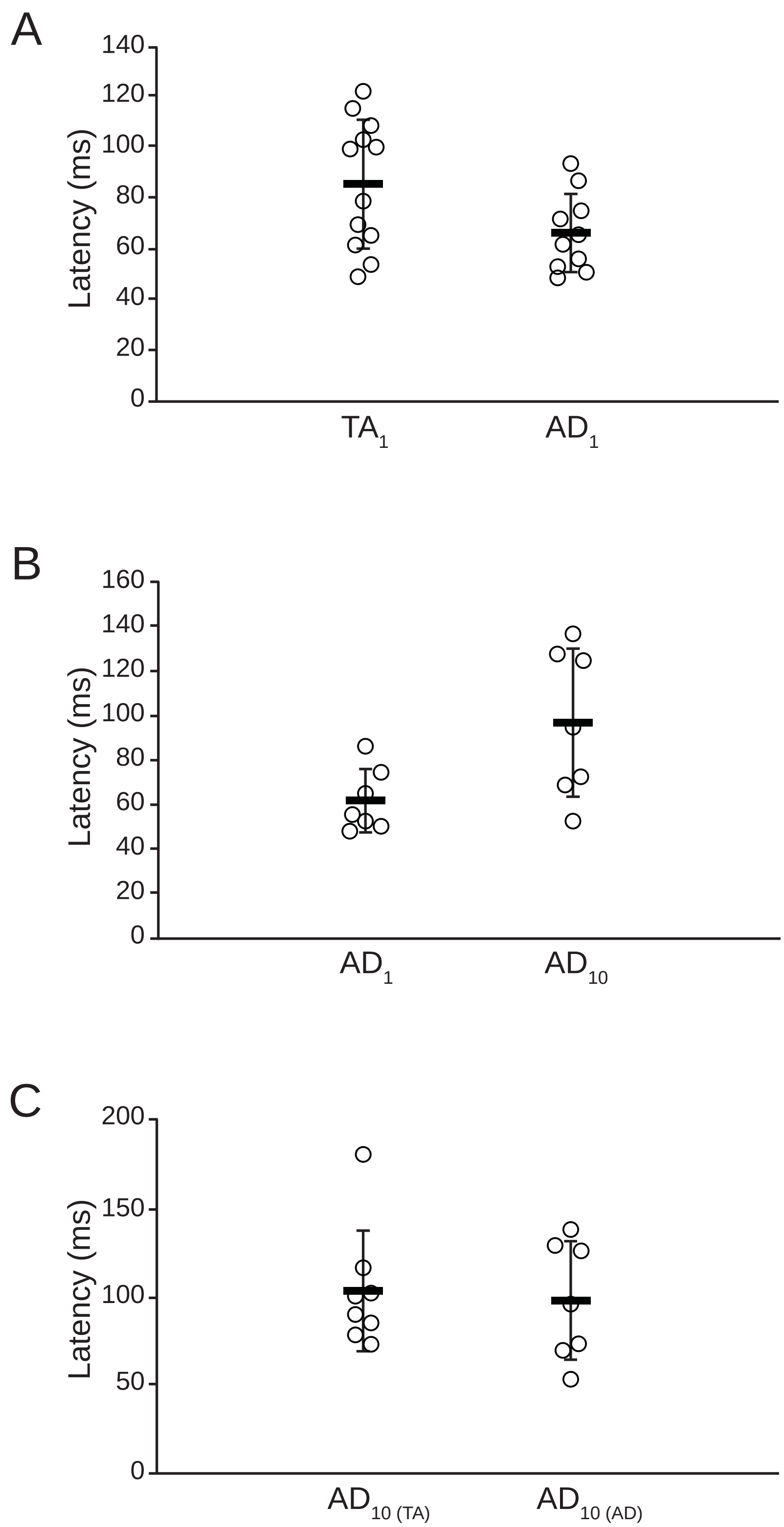
Figure 6: Peak cross-correlation coefficients within ±500 ms for each of the 1st trials from each of the initial TA responders (TA) and initial AD responders (AD). The cross-correlations are of the touch plate position related to each of the **A**, Ankle, **B**, Elbow and **C**, $COP_{A\text{-}P}$. Individual data points are displayed as open circles.

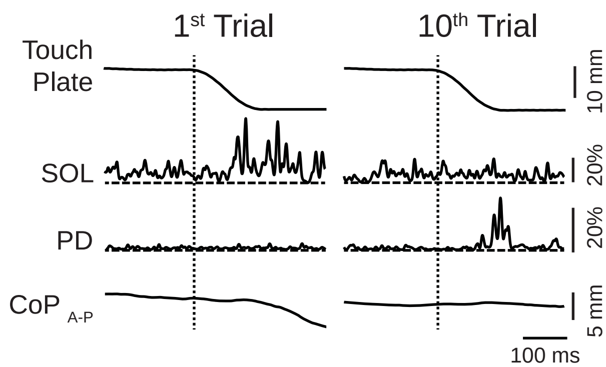


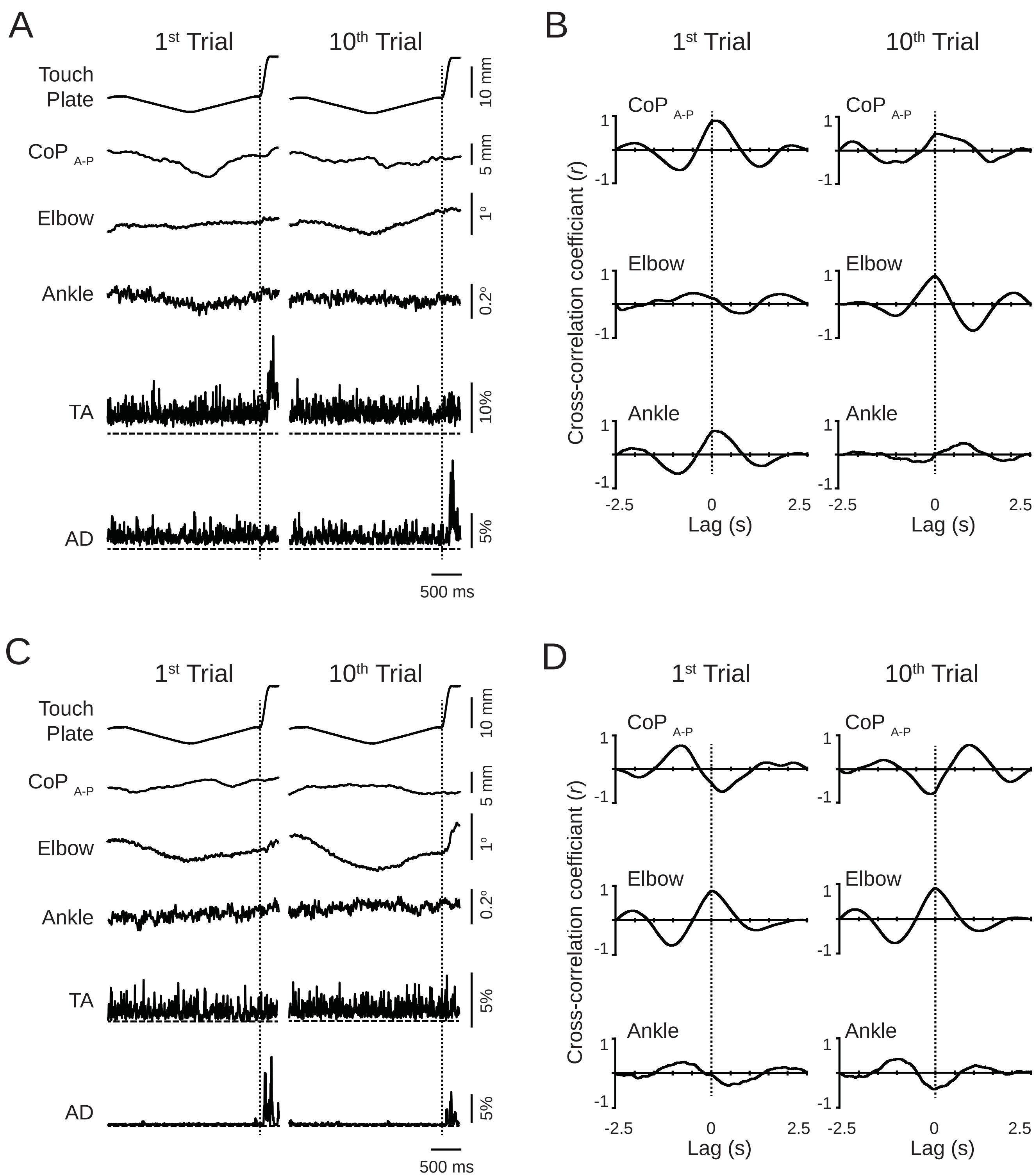
A
M
B
Forward
0.4 Hz
12.5
5
mm
2.5 s



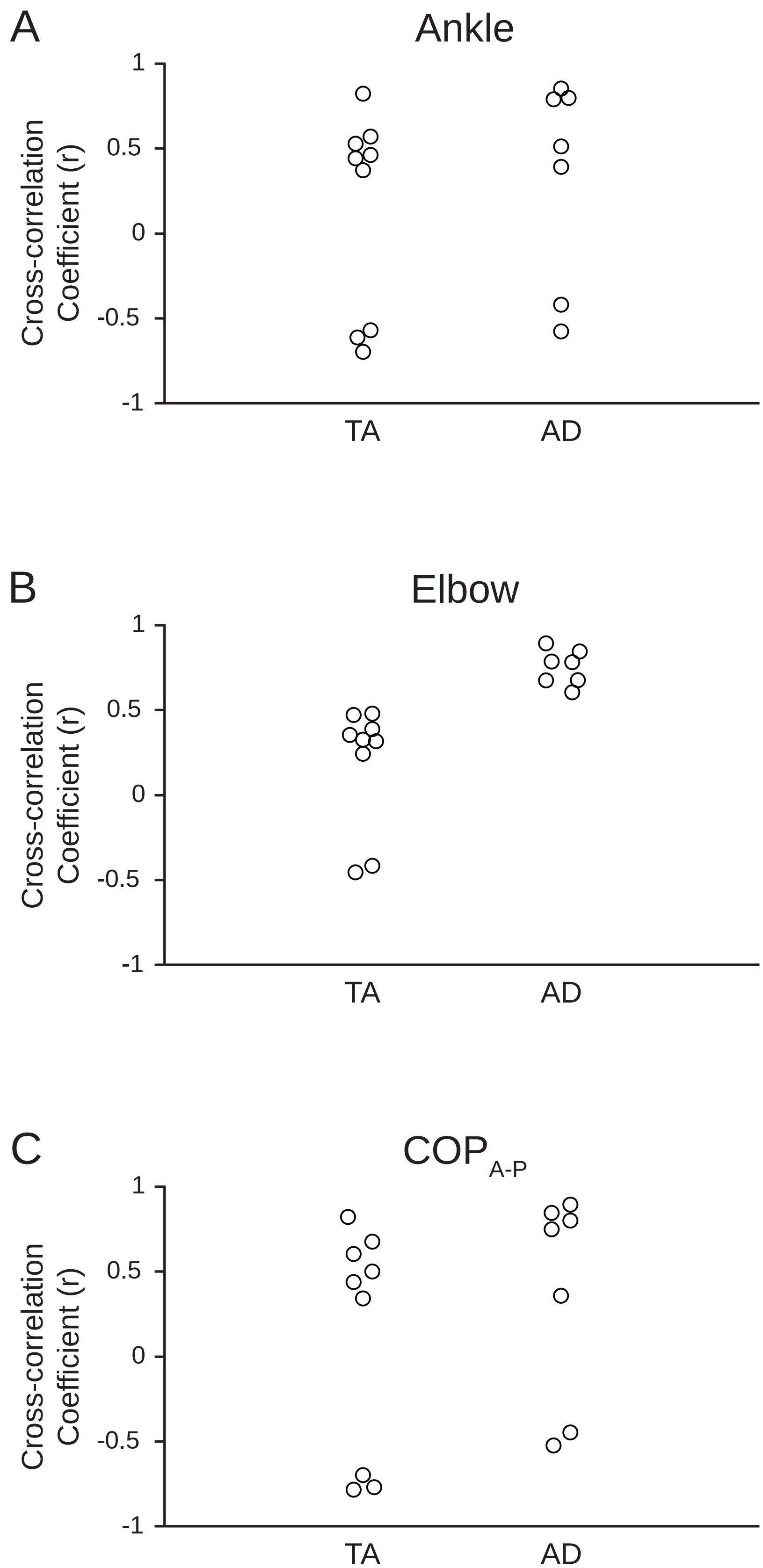
A
1st Trial
10th Trial
Touch Plate
TA
SOL
AD
PD
BB
TB
Ankle
Elbow
CoP A-P
10 mm
20%
1°
5 mm
100 ms
B
1st Trial
C
Trial 10
Trial 1


A
Latency (ms)
140
120
100
80
60
40
20
0
TA1
AD1
B
Latency (ms)
160
140
120
100
80
60
40
20
0
AD1
AD10
C
Latency (ms)
200
150
100
50
0
AD10 (TA)
AD10 (AD)


1st Trial
10th Trial
Touch Plate
SOL
PD
CoP A-P
10 mm
20%
20%
5 mm
100 ms



A
1st Trial
10th Trial
Touch Plate
CoP A-P
Elbow
Ankle
TA
AD
10 mm
5 mm
1°
0.2°
10%
5%
500 ms
B
1st Trial
10th Trial
CoP A-P
Elbow
Ankle
Cross-correlation coefficient (r)
1
-1
-2.5
0
2.5
Lag (s)
C
1st Trial
10th Trial
Touch Plate
CoP A-P
Elbow
Ankle
TA
AD
10 mm
5 mm
1°
0.2°
5%
5%
500 ms
D
1st Trial
10th Trial
CoP A-P
Elbow
Ankle
Cross-correlation coefficient (r)
1
-1
-2.5
0
2.5
Lag (s)



A
Ankle
Cross-correlation Coefficient (r)
1
0.5
0
-0.5
-1
TA
AD
B
Elbow
Cross-correlation Coefficient (r)
1
0.5
0
-0.5
-1
TA
AD
C
COPA-P
Cross-correlation Coefficient (r)
1
0.5
0
-0.5
-1
TA
AD