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Trial-to-trial adaptation in control of arm reaching and standing posture

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Pienciak-Siewert A, Horan DP, Ahmed AA. Trial-to-trial adaptation in control of arm reaching and standing posture. *J Neurophysiol* 116: 2936–2949, 2016. First published September 28, 2016; doi:10.1152/jn.00537.2016.—Classical theories of motor learning hypothesize that adaptation is driven by sensorimotor error; this is supported by studies of arm and eye movements that have shown that trial-to-trial adaptation increases with error. Studies of postural control have shown that anticipatory postural adjustments increase with the magnitude of a perturbation. However, differences in adaptation have been observed between the two modalities, possibly due to either the inherent instability or sensory uncertainty in standing posture. Therefore, we hypothesized that trial-to-trial adaptation in posture should be driven by error, similar to what is observed in arm reaching, but the nature of the relationship between error and adaptation may differ. Here we investigated trial-to-trial adaptation of arm reaching and postural control concurrently; subjects made reaching movements in a novel dynamic environment of varying strengths, while standing and holding the handle of a force-generating robotic arm. We found that error and adaptation increased with perturbation strength in both arm and posture. Furthermore, in both modalities, adaptation showed a significant correlation with error magnitude. Our results indicate that adaptation scales proportionally with error in the arm and near proportionally in posture. In posture only, adaptation was not sensitive to small error sizes, which were similar in size to errors experienced in unperturbed baseline movements due to inherent variability. This finding may be explained as an effect of uncertainty about the source of small errors. Our findings suggest that in rehabilitation, postural error size should be considered relative to the magnitude of inherent movement variability.

neuromechanics; trial-to-trial adaptation; error; motor learning; anticipatory postural adjustment

NEW & NOTEWORTHY

A key question in motor control is how sensorimotor error drives adaptation on an incremental, trial-to-trial basis. This has not been investigated in the control of standing posture when reaching, despite the significance of postural control deficits caused by various motor control pathologies. Therefore, we examine the relationship between error and adaptation in standing posture and arm reaching concurrently. We find that adaptation scales near proportionally with error in posture, for errors within postural stability limits.

HUMAN MOTOR CONTROL IS HIGHLY complex; we face constantly changing demands and conditions. Despite this, healthy individuals can easily perform skillful movements and interact with objects in our environment. The central nervous system can accomplish this by using a representation of the system's dynamics to plan that motor commands will produce a specified movement. When these dynamics are altered, the control strategy must adapt to compensate (Lackner and Dizio 1994; Shadmehr and Mussa-Ivaldi 1994). Here, "adaptation" refers to any change in predictive control strategy which compensates for a change in task dynamics such that movement kinematics return toward the prior unperturbed performance. More specifically, motor adaptation is defined as a trial-to-trial modification of movement based on error feedback, where error is considered to be a deviation from the expected movement outcome. The movement retains its identity as being a specific action (e.g., reaching), but one or more parameters are changed (e.g., direction or force) (Bastian 2008; Martin et al. 1996). Thus adaptation provides a flexible control that can account for a temporary, predictable change in task demands, allowing a limited number of learned movements to be adapted to a wide variety of tasks (Bastian 2008).

Most adaptation studies have examined the time course of adaptation and/or the final adapted behavior after practicing for a period of time. However, on a more fundamental level, it is important to understand how adaptation is influenced by error on an incremental, trial-to-trial basis because it is this incremental adaptation that leads to more long-term changes in behavior. Classical theories of motor learning hypothesize that adaptation is driven by sensory prediction error, or the difference between the predicted and actual movement outcomes (Jordan and Rumelhart 1992; Kawato et al. 1987; Wolpert and Ghahramani 2004). This is upheld by many previously published findings. Several studies of arm reaching have shown that in adaptation to a constant-magnitude force perturbation, the incremental adaptation for each successive trial is based on the movement error experienced in the previous trial (Franklin et al. 2003; Osu et al. 2003; Shadmehr and Mussa-Ivaldi 1994; Thoroughman and Shadmehr 2000). Other arm-reaching studies, using force and/or visuomotor perturbations of varying magnitudes, have shown that trial-to-trial adaptation scales with perturbation magnitude and/or error magnitude (Fine and Thoroughman 2007; Herzfeld et al. 2014; Marko et al. 2012; Scheidt et al. 2001; Trent and Ahmed 2013; Wei and Kording 2010). In addition, several modeling studies have shown good fits to experimental adaptation data of arm reaching and eye saccades, using models which assume a linear trial-to-trial relationship between movement error and adaptation (Baddeley et al. 2003; Colagiorgio et al. 2015; Donchin et al. 2003; Ethier et al. 2008; Franklin et al. 2008; Smith et al. 2006; Thoroughman and Shadmehr 2000).

With regard to standing postural control, however, there have only been a few studies investigating how we adapt to novel perturbations. This is despite the fact that the ability to

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maintain stable, upright standing is a critical component of many of our daily activities. Furthermore, none of these prior studies examined adaptation on a trial-to-trial basis, and thus the relationship between movement error and adaptation in standing postural control remains unclear. Although this relationship has not been explicitly studied, many studies have investigated anticipatory postural adjustments (APAs) generated in anticipation of impending voluntary movements or external perturbations. For well-practiced voluntary movements, APAs are increased with the amplitude and/or velocity of the impending movement (Bertucco and Cesari 2010; Horak et al. 1984; Kaminski and Simpkins 2001; Lee et al. 1987; Yiou et al. 2007). Similarly, after acclimating to a predictable external perturbation, subjects generate larger APAs for larger perturbation magnitudes (Beckley et al. 1991; Horak and Diener 1994; Horak et al. 1989; Smith et al. 2012). Horak and Diener (1994) found more specifically that APAs increased linearly with both perturbation amplitude and velocity. Together, these results indicate that trial-to-trial adaptation in posture should increase with perturbation and/or error magnitude, similar to what has been observed in arm reaching.

However, previous studies that investigated concurrent adaptation of arm reaching and related postural control have shown specific differences in adaptation between the two modalities, indicating that adaptation occurs via a distinct mechanism in each form of movement. One study found that postural control was adapted at a slower rate than arm control (Ahmed and Wolpert 2009); another found that postural stability limits affected adaptation of APAs but not adaptation of arm movements (Manista and Ahmed 2012). Indeed, there are fundamental differences between whole body and arm reaching movements that lead us to predict that adaptation in response to a given error may differ between these modalities. First, upright standing is inherently unstable, and a person may adapt differently to an error depending on its proximity to postural stability limits [where stability limits are defined using the postural base of support (BOS)]; arm reaching movements, however, are not explicitly subject to the same stability limits. Second, adaptation in arm reaching typically involves visual feedback of the cursor, whereas there is no similarly explicit visual feedback of postural control in these experiments. This may lead to increased uncertainty in postural adaptation compared with arm adaptation and accordingly may lead to different patterns of adaptation. Similarly, in these experiments the reaching movements are voluntary movements to an explicit target, while postural control is a more generalized behavior aimed at maintaining a stable upright posture. This likely also contributes to different patterns of adaptation.

To answer these questions, we will investigate trial-to-trial adaptation of arm reaching and postural control concurrently. To examine the relationship between error and adaptation in each modality, we will measure adaptation in response to a range of evenly distributed error sizes, using a range of perturbation strengths to induce errors concurrently in arm reaching and in related postural control. We predict that adaptation in posture will exhibit error-dependent behavior, similar to adaptation of arm movements. However, we expect that the relationship between error and adaptation will differ between these two forms of movement.

METHODS

Theoretical Development

To examine the relationship between movement error and adaptation in both arm reaching and postural control, we used an experimental paradigm in which subjects make target-directed planar reaching movements while standing and grasping the handle of a force-generating planar robotic arm (Fig. 1*A*). Previous studies have shown that when the robot arm applies perturbing forces to the hand during the reaching movement, movement errors and adaptation are observed in postural control as well as in the reaching movement (Ahmed and Wolpert 2009; Manista and Ahmed 2012; Pienciak-Siewert et al. 2014). These perturbing forces generated by the robot are proportional to the magnitude and perpendicular to the direction of the instantaneous velocity V of the reaching movement (*Eq. 1*), where k is the gain of the force field; we varied the value of k (0, 10, 20, 30, or 40 $N \cdot s \cdot m^{-1}$) in such a way as to induce an evenly distributed range of movement error sizes. This type of perturbation is particularly useful for studying adaptation of postural control, because the component of

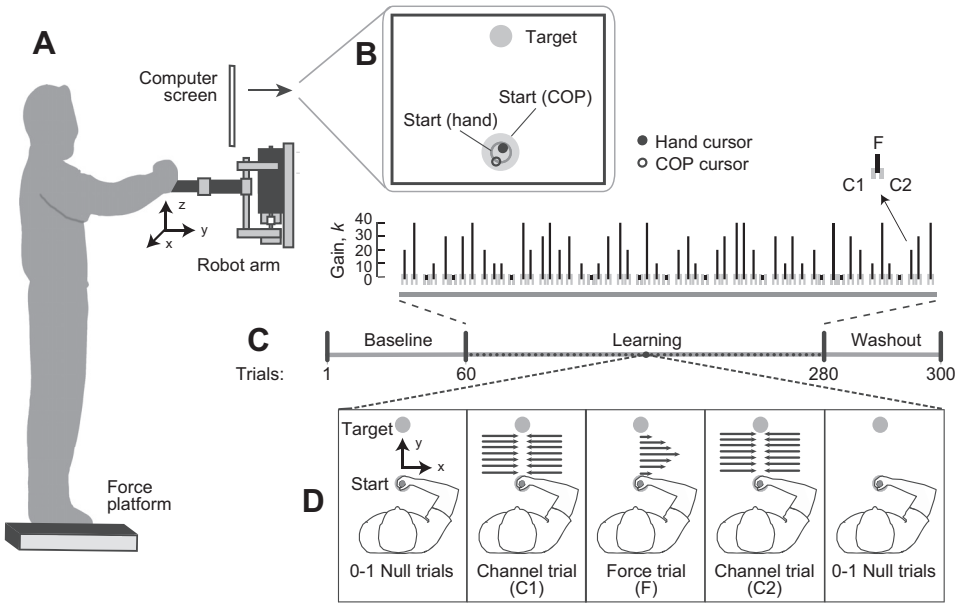

Fig. 1. *A*: apparatus. *B*: visual feedback provided to subjects on computer screen. *C*: experimental protocol and sample trial list. Each black line represents a force trial of a specified gain; each pair of channel trials is represented by the pair of short gray lines bracketing each force trial. *D*: example trial triplet, illustrating forward ($+y$) reaching movements and rightward ($+x$) perturbing forces (force trials). COP, center of pressure.

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postural control that is adapted in response to the force field is perpendicular to, and thus not confounded by, the tangential component related to the focal reaching movement (Ahmed and Wolpert 2009; Manista and Ahmed 2012; Pienciak-Siewert et al. 2014).

$$\begin{bmatrix} F_x \\ F_y \end{bmatrix} = k \begin{bmatrix} 0 & -1 \\ 1 & 0 \end{bmatrix} \begin{bmatrix} V_x \\ V_y \end{bmatrix} \quad (1)$$

Theoretically, when an error is experienced in one trial (where error is considered to be a deviation from the expected movement outcome), the brain responds by changing the motor output, or control, on the next trial to correct that error (Thoroughman and Shadmehr 2000). Thus, the change in output x from trial i to $i + 1$ is determined by a decay factor A and adaptation B as a function of the error e experienced on trial i (*Eq. 2a*). If output x is known and error e is negligible on trials $i - 1$ and $i + 1$ (*Eq. 2b*), as is the case in channel trials (Scheidt et al. 2000) (see next paragraph), then adaptation due to error on trial i is given by *Eq. 2c*.

$$x_{i+1} = Ax_i + B(e_i) \quad (2a)$$

$$x_i = Ax_{i-1} + B(e_{i-1}) = Ax_{i-1} \quad (2b)$$

$$B(e_i) = x_{i+1} - A^2 x_{i-1} \quad (2c)$$

Previous studies have reported values for A ranging from 0.69 to 0.89 (Fine and Thoroughman 2007; Joiner and Smith 2008; Marko et al. 2012; Trent and Ahmed 2013). Our findings were not sensitive to differing values of A, so we set $A = 1$ for simplicity. Thus we quantified adaptation as the change in motor output from the trial before to the trial after the perturbation, $x_{i+1} - x_{i-1}$. To measure motor output before and after each perturbation trial, each force perturbation trial (F) was immediately preceded by one channel trial (C1) and followed by a second channel trial (C2; Fig. 1*D*). We used channel trials to measure motor output because these trials allow us to quantify subjects' predictive, feed-forward control in the arm. In a channel trial, the robot generates a force channel that restricts the hand trajectory to a straight path between the starting position and the target; using the robot, we can then measure the amount of perpendicular force that the subject is exerting into the channel. In addition, because hand error is minimized on channel trials, these trials have a minimal effect on adaptation or de-adaptation (Scheidt et al. 2000). This three-trial arrangement, called a "triplet," was used throughout the experiment. Triplets were separated randomly by 0–2 null field trials (robot forces turned off) to prevent subjects from predicting when the triplets would occur.

Experimental Subjects

Ten young adult subjects (age 21.8 ± 1.7 yr; height 175 ± 10 cm; mass 68 ± 10 kg) participated in the study. All subjects were screened using a health questionnaire and the Edinburgh Handedness Inventory test (Oldfield 1971). All subjects were right-handed, had normal or corrected-to-normal vision, and reported no recent musculoskeletal injuries or history of neurological or musculoskeletal disorders. The University of Colorado Boulder Human Research Committee approved all experimental procedures.

Experimental Apparatus and Setup

Subjects made forward reaching movements in the horizontal plane (in the anterior direction, $+y$) with their right hand while grasping the handle of a two-degree-of-freedom planar robotic arm (InMotion2 Shoulder-Elbow Robot; Interactive Motion Technologies) and while standing barefoot on a six-axis, dual-plate force platform (AMTI Dual-Top AccuSway; Advanced Mechanical Technology; Fig. 1*A*). They stood with one foot placed on each plate of the platform, equidistant from the centerline of the platform. The forearm was supported against gravity by a rigid cradle attached to the handle. The height of the robot was adjusted for each subject so that the robot arm and handle were level with the subject's sternum (mean height: 123 ± 7 cm across subjects), so robot perturbations and the resulting moments and associated center of pressure (COP) movements were dependent on subject height. To ensure that stance width was scaled similarly for each subject, stance width (defined as the distance between the lateral edges of the feet) was fixed at 24% of robot handle height; this scaling was chosen to attain a mean stance width of ~30 cm, based on previous measurements of mean height and relationship to sternum height (Drillis and Contini 1966; McDowell et al. 2008). Mean stance width was 29 ± 2 cm across subjects. Subjects were asked to keep their feet flat on the ground to ensure that the BOS size was not affected by lifting or rotation of the feet. A computer monitor, vertically suspended in front of the subject, displayed continuous visual feedback of hand, start, and target positions throughout the movement.

Before the experiment began, a "start" circle and a cursor representing COP location were shown on the screen. Subjects were asked to stand such that their COP was centered in the start circle when they were standing comfortably straight. Their exact foot position was marked on the force platform to ensure that they always stood in the same location.

In the experiment, subjects were asked to make 15-cm forward reaching movements, using the robot handle to control the cursor on the screen (Fig. 1*B*). At the start of each trial, subjects were required to hold the 0.6-cm-diameter hand cursor in the center of the 1.6-cm start circle and to maintain their COP location (represented by a separate 0.6-cm cursor of a different color) anywhere within the start circle. To facilitate simultaneous performance of the two centering tasks, a second, smaller ring was displayed within the start circle as a guide for centering the hand cursor; the hand cursor was filled in while only the outline of the COP cursor was displayed (Fig. 1*B*). After a short time delay, the COP circles disappeared and a 1.6-cm target circle appeared, and subjects moved the hand cursor toward the target. At the end of the movement, subjects were required to remain within the target circle for 50 ms, after which the robot moved the subject's hand back to the start position to begin the next trial. At the end of each movement, subjects also received visual feedback about movement duration, measured from the time the hand left the start position to the time at which the 50-ms target requirement was fulfilled. This was to ensure that movement durations stayed within a range of 450 to 600 ms. If the duration was within the desired range, the target "exploded"; if it was too long (movement was too slow), the target turned gray and the subject was encouraged to move faster; if it was too short (movement was too fast), the target turned green and the subject was encouraged to move more slowly. With regard to posture, subjects were instructed to not lean on the handle at all, to avoid locking their knees, and to keep their feet flat on the platform.

Experimental Protocol

The protocol was 300 trials long and was divided into three blocks: baseline (60 trials), learning (220 trials), and washout (20 trials) (Fig. 1*C*). The baseline block consisted of null trials, in which robot forces were turned off, to familiarize the subject with the robot and to measure baseline performance. The learning block consisted of mixed null trials and adaptation trial "triplets". Each triplet consisted of three subsequent trials: one channel trial (C1), one force field trial (F), and another channel trial (C2) (Fig. 1*D*). In each force field trial, a viscous curl field was simulated such that the robot exerted a force on the hand that was proportional to the magnitude and perpendicular to the direction of the instantaneous velocity of the robot handle, as described earlier (*Eq. 1*). Thus, for a forward reaching movement (in the anterior direction, $+y$), the robot generated rightward perturbing forces (in the lateral direction, $+x$). In channel trials, stiffness and damping for the force channel were 2,000 N/m and 50 N·s/m, respectively. The learning block consisted of 220 trials total (Fig. 1*C*),

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divided into 11 batches of 20 trials each; each batch contained 5 trial triplets (one for each gain value k = 0, 10, 20, 30, or 40 N·s·m^{-1}, presented in randomized order), randomly interspersed with sets of 0–2 null trials as described earlier. The washout block consisted of null trials to allow subjects to completely de-adapt the previous dynamic environment. Although the sequence of trials throughout the protocol was predetermined, we wished to ensure that our results were not specific to a given trial sequence. Therefore, six subjects experienced one fixed trial sequence, and four subjects experienced another fixed trial sequence, where the order of presentation of gain magnitudes in each batch of the learning block was re-randomized.

Following the experiment, subjects played a brief COP game for the purpose of measuring the size of their functional BOS, or the limits of the area within the BOS that a person is willing to extend their COP (King et al. 1994; Holbein-Jenny et al. 2007; Lee and Lee 2003). In this game, they controlled the cursor with their COP to make a series of 24 leaning movements from the start circle toward 8 randomized targets located in different directions, evenly spaced around a 360° circle at 45° angles, and at a distance of 13 cm from the central start position (this distance was chosen to encourage subjects to move their COP out as far as possible).

Data Collection and Analysis

Position, velocity, and force data from the robot handle were sampled at 200 Hz. COP position data were calculated from force platform data, which was also sampled at 200 Hz. For each side of the dual-plate platform (right and left), eight voltage signals were collected and converted into three-dimensional ground reaction forces (F_x, F_y, F_z) and moments (M_x, M_y, M_z) which were then low-pass filtered at 10 Hz. COP position data for each force plate (right and left) was calculated from filtered force platform data, relative to the center of the platform [C_x C_y], as [COP_x COP_y] = [C_x C_y] + [M_y M_x]/F_z, where x and y subscripts denote mediolateral and anteroposterior axes, respectively. The net COP was then calculated as a weighted average of the COP for each plate using the method described by Winter et al. (1996). COP velocity was calculated from net COP position using a five-point differentiation algorithm. All COP data for each subject were normalized to 50% of stance width.

All data were aligned to movement onset, such that time zero represents movement onset of the arm, and truncated at movement end. Movement onset was defined as when the cursor crossed the boundary of the start circle. Movement end was defined as when the cursor reached the target circle. All data were taken from movement onset to movement end, unless otherwise noted. Note that for forward reaching movements (+y), the force perturbation is in the rightward direction (+x).

Trials were excluded from analysis if the movement onset criterion was inaccurate (by visual inspection) or if the data were corrupted. If any trial in a triplet was excluded, the entire triplet was excluded from analysis. A total of 47 trials were rejected, out of the entire data set (3,000 total trials, with 300 trials per subject). On average, 4.7 total trials, 1.1 baseline trials, and 2.4 triplets were rejected per subject.

Arm control. To quantify movement error (e) and motor output (x) in the arm, we measured hand error and anticipatory force, respectively. Hand error was calculated on null trials and force trials as the peak signed value of the perpendicular deviation of the handle trajectory from a straight path between the start and target positions (Fig. 2*A*). On channel trials, channel force is the force produced by the robot to maintain the channel when the subject exerts a perpendicular force into the channel and is therefore opposite in direction to the actual force being produced by the subject. Anticipatory force was calculated on channel trials as the channel force at the time of peak tangential hand velocity (Fig. 2*B*); anticipatory force was therefore a measure of the amount of force being exerted by the subject at the time when peak perturbation force would be experienced in the force field.

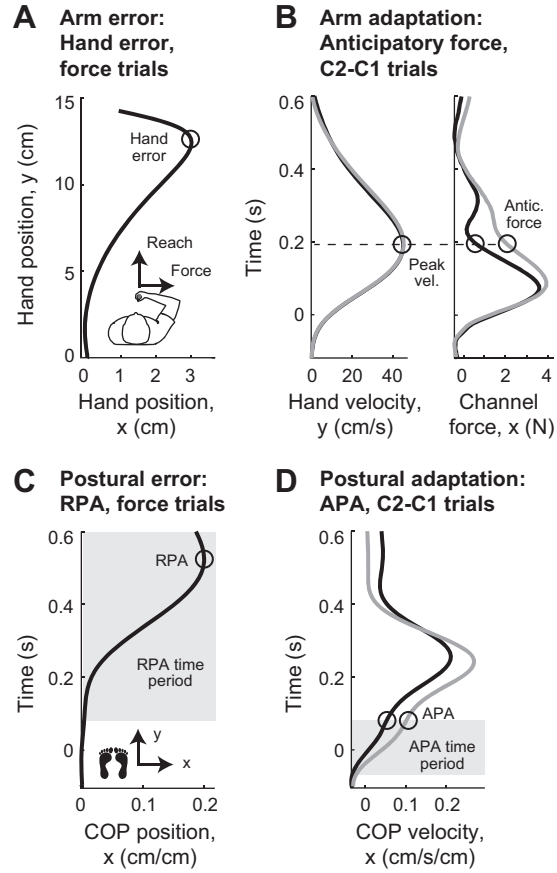

Fig. 2. Error and adaptation metrics. *A*: arm error is hand error taken from force trials. *B*: arm adaptation is the change in anticipatory force from C1 to C2 channel trials. *C*: postural error is reactive postural adjustment (RPA) taken from force trial. *D*: postural adaptation is the change in anticipatory postural adjustments (APAs) from C1 to C2 channel trials. In *B* and *D*, data for C1 channel trial is shown in black and for C2 channel trial in gray.

Postural control. To quantify movement error (e) and motor output (x) in posture, we measured reactive postural adjustment (RPA) and anticipatory postural adjustment (APA), respectively. RPA and APA were based on the normalized COP position and velocity, respectively, in the direction of the force perturbation (perpendicular to the direction of reaching movement), and were calculated on every trial. We observed that COP velocity responses on force trials began as early as 80 ms after movement onset; similarly, Horak and Nashner (1986) observed reaction latencies in the tibialis anterior varying from 73 to 110 ms, in response to unexpected backward sway perturbations. Therefore, as a measure of anticipatory control, the APA was calculated as the peak signed value of COP velocity taken between 70 ms before movement onset and 80 ms after movement onset (Fig. 2*D*). The RPA was calculated as the peak signed value of COP position throughout the remaining duration of the movement (following the APA time period) (Fig. 2*C*).

Metrics. For each triplet, metrics of error and adaptation were defined for both arm and posture. Arm error and postural error were defined as hand error and RPA, respectively, taken from the force trial (F); each error metric was corrected for baseline by subtracting out the mean baseline error (mean across last 10 trials in baseline block), such that the normalized error for each triplet e_i^n is given by *Eq. 3a*, where e_i is error as measured on the force trial and e_B is mean baseline error:

$$e_i^n = e_i - e_B \quad (3)$$

The baseline error correction was applied to remove the bias caused by small lateral deviations of hand and COP position that are inherent

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in unperturbed baseline movements. This correction also provides similarity with our adaptation metric, where the C1 trial in each triplet acts as a baseline relative to the C2 trial. Based on *Eq. 2c*, adaptation to an error on the *i*th trial, B_i, was quantified as the change in motor output x from the first (C1) to the second channel trial (C2) in each triplet:

$$B_i(e_i) = x_{C2} - x_{C1} \quad (4)$$

Motor output for arm and posture was defined as anticipatory force and APA, respectively. For example, postural adaptation for each triplet was calculated as $APA_{C2} - APA_{C1}$.

Statistics

Data were examined across gain groups K0, K10, K20, K30, and K40, with each group consisting of 10 triplets at the specified gain value k. The first batch of learning trials (first triplet at each gain value) was excluded from groups to eliminate the effect of surprise.

Error and adaptation data in arm and posture were analyzed using repeated-measures ANOVAs, with perturbation gain as a within-subjects factor. To test for error and adaptation at each nonzero gain value, we made planned comparisons on the within-subjects results between K0 and each nonzero gain group (K0 vs. K10, K0 vs. K20, K0 vs. K30, K0 vs. K40). We also made planned comparisons on the within-subjects results between adjacent nonzero gain groups (K10 vs. K20, K20 vs. K30, K30 vs. K40).

To directly examine the relationship between adaptation and error, we performed linear regression analyses. We also fit the data to various models to determine the proportionality of the relationship. In these analyses, each data point represents the mean values of adaptation and error for one subject, or the mean values across all subjects, at a specific error size. For grouping by error size, we binned the data from all triplets for each subject; bin size was 1.5 cm for arm error (hand error) and 0.07 cm/cm for postural error (RPA). If a bin contained only one data point for any subject, that data point was excluded from analysis.

All data analyses were performed in MATLAB. Mean data values are reported in the text as mean ± SD. For all statistical analyses the criterion for significance was set at the level of $\alpha = 0.05$.

RESULTS

Group mean trajectory data demonstrate that on force trials with a nonzero force perturbation, hand movement was perturbed rightward, in the direction of the perturbation; stronger perturbations caused a larger deviation in lateral hand trajectory (Fig. 3*A*). Similarly, COP trajectories were also deviated rightward (Fig. 3*A*). These COP deviations were initiated later than hand deviations as COP movement is a control response to the rightward force perturbation. Mean C1 and C2 trajectories for each perturbation gain (Fig. 3, *C* and *D*) show how subjects adapted their control between C1 and C2 trials. For triplets with a nonzero perturbation, anticipatory force (channel force at time of peak hand velocity, Fig. 3*C*) increased in the direction of the hand error experienced on force trials (Fig. 3*A*). Similarly, APAs (peak lateral COP velocity during APA period, Fig. 3*D*) increased in the direction of the RPA (maximum lateral COP movement) experienced on force trials (Fig. 3*A*). Mean adaptation trajectories (mean difference between C1 and C2 trajectories for each triplet) show how these differences change with perturbation gain (Fig. 3*B*).

Data analyses showed that error and adaptation increased in magnitude with perturbation gain across all subjects; furthermore, adaptation showed a significant, linear correlation with error. Results are presented below for error and adaptation, in the arm and in posture. We also present results for arm movement characteristics (reaching velocity, perturbation force), BOS size, overall COP displacements, and tangential and perpendicular APAs, and mean learning.

Error

To compare performance across perturbation gains, we first had to confirm that perturbation force increased with gain. Perturbation force was measured on force trials as the force exerted by the robot at the time of peak tangential hand velocity. Despite the fact that peak hand velocity decreased with increasing gain, perturbation force was found to increase with increasing gain, as expected. There was a main effect of perturbation gain on peak hand velocity [$F(4,36) = 45.15$, $P < 0.001$] and on perturbation force [$F(4,36) = 124.38$, $P < 0.001$].

We then examined error at each perturbation gain (Fig. 4, *C* and *F*). The ANOVA revealed a main effect of perturbation gain on both arm error (hand error, force trials) [$F(4,36) = 133.41$, $P < 0.001$] and postural error (RPA, force trials) [$F(4,36) = 133.41$, $P < 0.001$]. Planned comparisons showed that for each nonzero gain group, error was significantly different from K0 in the arm (all $P < 0.001$) and in posture (all $P < 0.001$). Error was also significantly different between each adjacent pair of nonzero groups (K10 vs. K20, etc.) in the arm (all $P < 0.001$) and in posture (all $P < 0.001$).

Alternative metric. To ensure that our findings were not dependent on a specific postural error metric, we performed an additional analysis using an alternative measure of RPA, taken as peak signed COP velocity during the RPA period (rather than peak signed COP position). With this alternative measure our main postural error finding remained similar; ANOVA showed a main effect of perturbation gain [$F(4,36) = 16.42$, $P < 0.001$]. This indicates that our chosen metric accurately reflects overall changes in postural error.

Adaptation

We also examined adaptation at each perturbation gain (Fig. 4, *I* and *L*). The ANOVA revealed a main effect of perturbation gain on arm adaptation (anticipatory force, C2-C1) [$F(4,36) = 13.59$, $P = 0.001$] and on postural adaptation (APA, C2-C1) [$F(4,36) = 16.05$, $P < 0.001$]. Planned comparisons showed that for each nonzero gain group, adaptation was significantly different from K0 in the arm (all $P \leq 0.003$) and in posture ($P = 0.014$ for K0 vs. K10; all other $P < 0.001$). However, adaptation was not significantly different between any adjacent pair of nonzero groups in the arm (all $P \geq 0.251$) or in posture (all $P \geq 0.124$).

To validate our selection of APA time period, we also examined APAs on force trials. If we observe an effect of current trial gain on APAs, it would indicate that our selected time period included reactive control specific to the current trial and/or that subjects were anticipating the specific perturbation gain of each force trial. However, the ANOVA revealed no main effect of perturbation gain on APAs [$F(4,36) = 0.89$, $P = 0.478$]. This confirms that our selection of APA time period was appropriate and also indicates that subjects were not able to anticipate the specific perturbation gain of each force trial.

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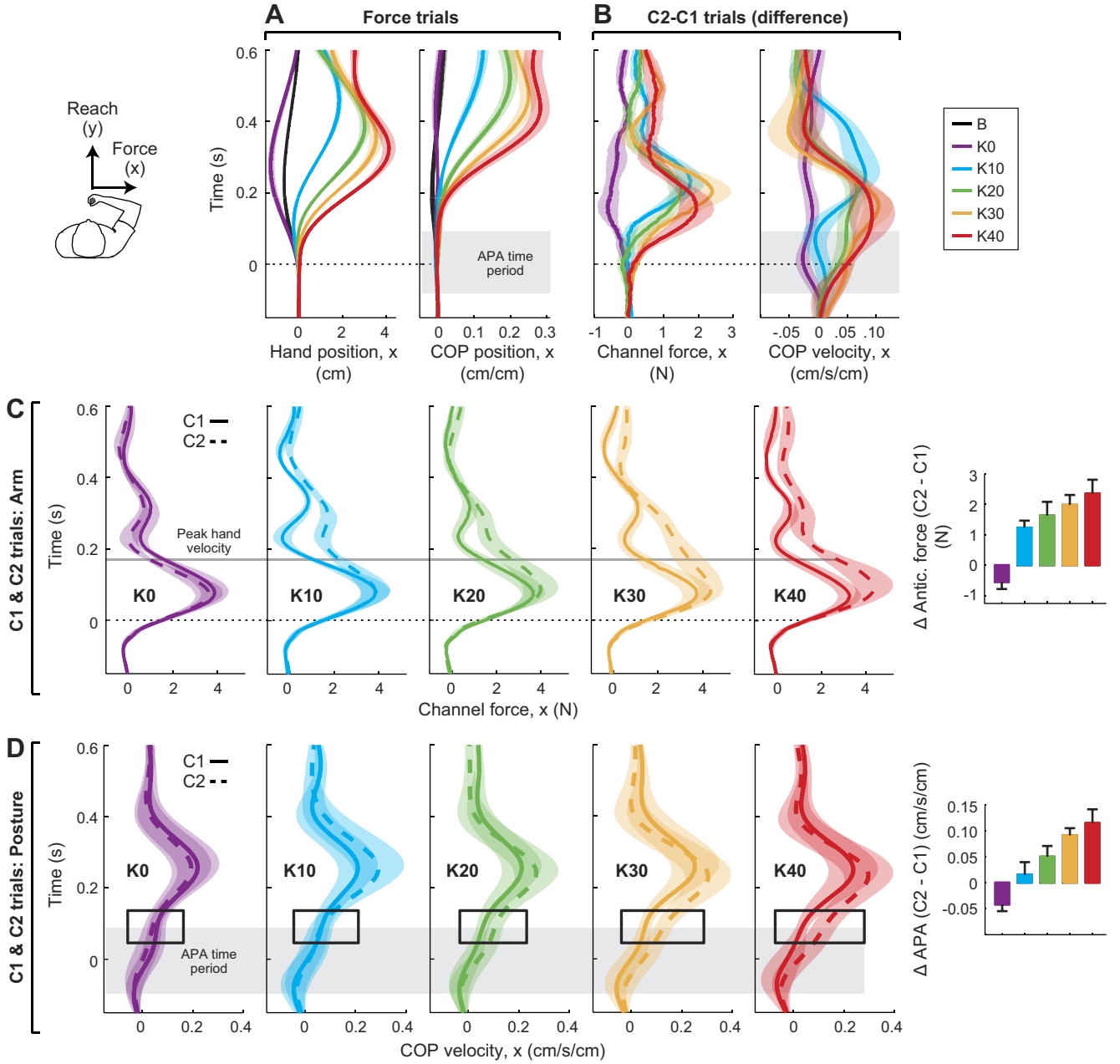

Fig. 3. *A*: group mean trajectories for perpendicular hand and COP position on force trials at each perturbation gain, and on null trials in late baseline (last 10 trials) (denoted "B" in legend). *B*: group mean difference trajectories (difference between C1 and C2 for each triplet) for perpendicular channel force and COP velocity at each perturbation gain. *C*: group mean trajectories for perpendicular channel force on C1 and C2 trials, illustrating the adaptive change in anticipatory force (taken at peak hand velocity) for each perturbation gain. *D*: group mean trajectories for COP velocity on C1 and C2 trials, illustrating the adaptive change in APA (peak COP velocity during APA period) for each perturbation gain. Note: for force trials (*A*) and channel trials (*C* and *D*), trajectories were averaged across triplets in each gain group for each subject, then averaged across subjects. For "C2-C1" difference trajectories (*B*), C1 trajectory was subtracted from C2 trajectory for each triplet; this difference trajectory was averaged across triplets in each gain group for each subject, then averaged across subjects. On all plots, shading indicates standard error across subjects. Time zero represents movement onset of the arm.

Alternative metrics. To ensure that our findings were not dependent on specific adaptation metrics, we performed additional analyses using alternative measures of anticipatory force and APA. These included anticipatory force taken as peak signed channel force (rather than channel force at peak tangential hand velocity), and APA taken as mean COP velocity or peak signed COP position during APA period (rather than peak signed COP velocity during APA period). With these alternative measures our findings remained similar; ANOVAs showed a main effect of perturbation gain on arm adaptation [$F(4,36) = 5.93$, $P < 0.001$] and on both alternative metrics of postural adaptation [mean COP velocity, $F(4,36) = 16.17$, $P < 0.001$; peak signed COP position, $F(4,36) = 3.18$, $P = 0.025$]. This indicates that our chosen metrics accurately reflect overall changes in adaptation behavior.

It is also important to note that although lateral hand movements are minimized on channel trials, thus resulting in negligible hand error, the same cannot be said for postural movements. If we do not assume negligible postural error on channel trials, we can solve *Eq. 2a* to quantify postural

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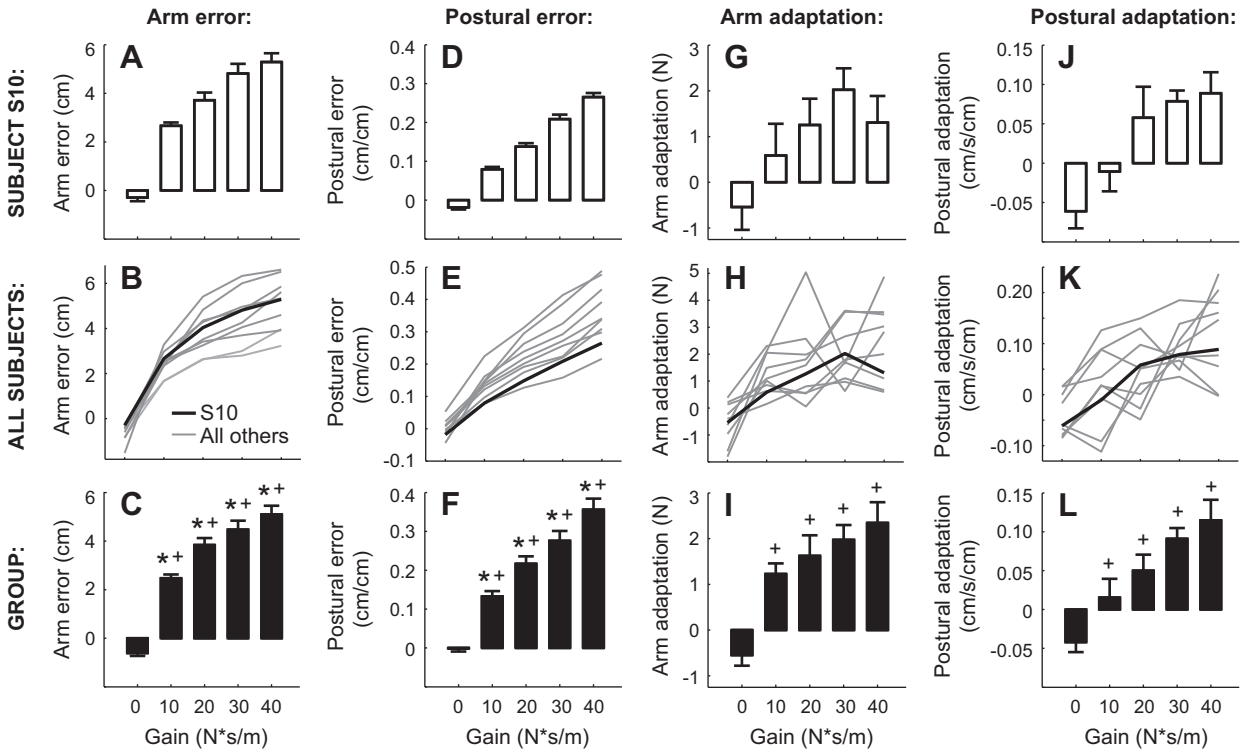

Fig. 4. Mean values of arm error (*A–C*), postural error (*D–F*), arm adaptation (*G–I*), and postural adaptation (*J–L*) at each perturbation gain. *A*, *D*, *G*, and *J*: mean values ± SE across triplets at each gain for a representative subject (*S10*). *B*, *E*, *H*, and *K*: mean values at each gain for all subjects (data for *subject S10* is shown in black). *C*, *F*, *I*, and *L*: group mean values ± standard error across all subjects at each gain. For group mean values (*C*, *F*, *I*, and *L*), $*P < 0.050$, statistically significant differences between adjacent gains and $+P < 0.050$, between K0 and each nonzero gain.

adaptation B using motor output x, or APAs, on trials i and $i + 1$ (*Eq. 5*).

$$B(e_i) = x_{i+1} - Ax_i \quad (5)$$

With $A = 1$, this simply becomes $x_{i+1} - x_i$, or the change in motor output x (APA) from trial F to C2 in each triplet. Our findings remained similar when we performed an additional analysis using this postural adaptation metric; ANOVA showed a main effect of perturbation gain [$F(4,36) = 2.73$, $P = 0.044$]. Therefore, we elected to measure postural adaptation between trials C1 and C2, as described by *Eq. 4*, to maintain similarity between our arm and postural adaptation metrics. (Arm adaptation cannot be measured differently, due to our inability to measure arm motor output on force trials.)

Tangential APAs. Thus far our analysis has focused on APAs that developed to anticipate force perturbations. These APAs were in the same direction as force perturbations and perpendicular to the direction of the hand reaching movement. In the direction tangential to the reaching movement, APAs related to the reaching movement itself were observed consistently on all trials; specifically, the COP moved away from the target before hand movement onset, as has been observed previously (Manista and Ahmed 2012; Pienciak-Siewert et al. 2014). To confirm that tangential APAs were not affected by perturbation gain, we examined tangential APAs on force trials. Tangential APAs were measured in the direction of reaching as the peak signed value of COP velocity, similar to perpendicular APAs, but taken between 100 ms before movement onset and 50 ms after movement onset (Ahmed and Wolpert 2009; Aruin and Latash 1995; Manista and Ahmed 2012). The ANOVA showed no main effect of perturbation gain on tangential APAs [$F(4,36) = 0.99$, $P = 0.425$].

Adaptation vs. Error

To directly examine the relationship between adaptation and error, we performed linear regressions of adaptation onto error for both arm and posture (Fig.5). In both arm and posture, adaptation and error showed a strong linear correlation. In the arm, regression across error bins showed a significant correlation for per-subject mean values [$F(1,55) = 60.66$, $P < 0.001$, $r^2 = 0.53$], and a significant, strongly linear correlation for group mean values [$F(1,6) = 515.16$, $P < 0.001$, $r^2 = 0.99$]. In posture, regression across error bins showed a significant correlation for per-subject mean values [$F(1,55) = 27.85$, $P < 0.001$, $r^2 = 0.34$], and a significant, strongly linear correlation for group mean values [$F(1,7) = 59.24$, $P < 0.001$, $r^2 = 0.91$].

We quantified the proportionality of the relationship between adaptation and error by fitting the data to several different models: linear, quadratic, and cubic. We found that a linear model was the best fit for both arm and postural data. However, in posture, this relationship appears to lose its linearity for very small error magnitudes. To investigate this, we performed separate linear regressions of adaptation onto error, examining only data within the three smallest error bin magnitudes in arm (error absolute value <2.25 cm) and posture (error absolute value < 0.105 cm/cm). In posture, this revealed no significant correlation for per-subject mean values [$F(1,20) < 0.01$, $P = 0.975$] or for group mean values [$F(1,2) = 0.40$, $P = 0.642$]. These results suggest that the postural adaptation vs. error relationship is domi-

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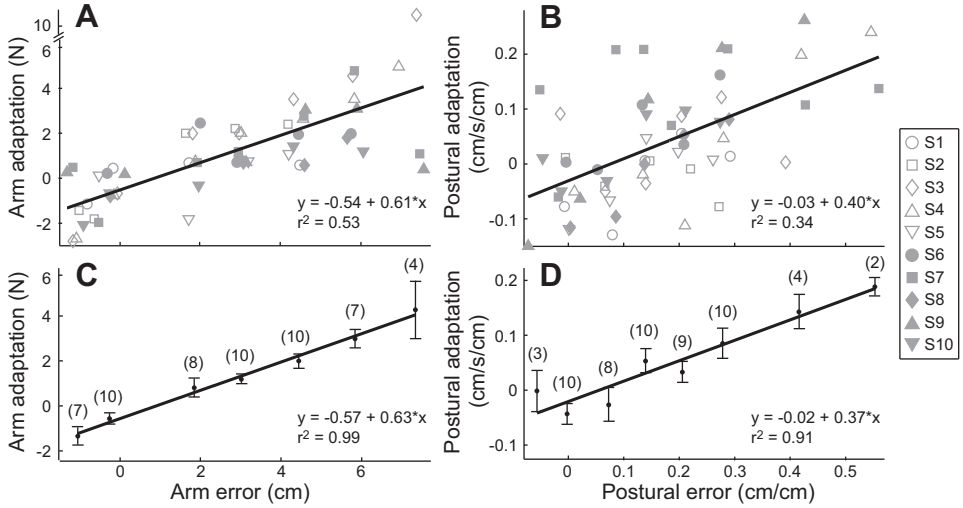

Fig. 5. Linear regressions of adaptation vs. error for arm (*A* and *C*) and posture (*B* and *D*). *A* and *B*: mean values of adaptation and error at each error bin for each subject (data for each subject is plotted using a unique marker). *C* and *D*: mean value at each bin across subjects (number shown in parentheses above each point indicates number of subjects in that bin); error bars represent SE for mean adaptation values across subjects (SE bars for mean error values are too small to be seen on these plots).

nated by the response to larger error magnitudes; the linear relationship may hold only for errors above some threshold magnitude. However, this is not the case for arm adaptation vs. error; the regression across only the three smallest error bin magnitudes revealed a significant correlation between arm adaptation and error for per-subject mean values [$F(1,24) = 14.01$, $P = 0.001$] and a near significant correlation for group mean values [$F(1,2) = 111.47$, $P = 0.060$].

This difference between arm and posture prompted us to examine these small error magnitudes relative to movement errors that occur in unperturbed baseline movements due to inherent movement variability and postural sway. Therefore, we compared the magnitudes of noncorrected error in the three smallest error bins to error magnitudes in late baseline (mean across last 10 trials in baseline block) using paired *t*-tests. Hand error showed a significant difference ($P = 0.017$; mean difference 0.31 ± 0.33 cm) but RPAs did not ($P = 0.100$; mean difference 0.01 ± 0.02 cm/cm). This indicates that adaptation was correlated with small errors in the arm because these errors were distinctly different from errors in baseline movements, while postural adaptation showed no correlation with small errors because these errors did not differ from baseline.

To investigate why there was not a strong effect of increasing error magnitude on postural adaptation, we sought to determine the extent to which COP movements executed during the experiment were within the limits of the functional BOS. We compared maximum lateral COP displacements during the experiment to those measured during the COP game (which established the dimensions of the functional BOS) (Fig. 6). Recall that all COP data were measured from the "start" location and normalized to 50% of stance width. Across all subjects, mean stance width was 29.3 ± 2.3 cm (50% of stance width was 14.7 ± 1.2 cm). In the COP game, the lateral functional BOS limit (measured from center) was 0.72 ± 0.08 cm/cm (normalized), or 10.6 ± 1.3 cm, averaged across all subjects. In the experiment, maximum lateral COP displacement on any trial (measured from center) was 0.46 ± 0.13 cm/cm (normalized), or 6.1 ± 1.1 cm, averaged across all subjects. The difference between lateral functional BOS limit and maximum lateral COP displacement during the experiment was 0.41 ± 0.14 cm/cm (normalized), or 4.5 ± 1.9 cm, averaged across all subjects. Maximum lateral COP displacements did not exceed the lateral functional BOS limit for any subject. This demonstrates that the APAs and RPAs developed in response to the force field were well within the limits of the functional BOS as well as the absolute limits imposed by stance width and helps to explain the strong linear relationship between large postural errors and adaptation.

Mean Learning

We observed that at the K0 gain, both arm and postural adaptation were negative (Fig. 4, *I* and *L*). This indicates that subjects were generating anticipatory force and APA that were too large for the K0 perturbation gain, and thus they adapted to this perturbation by decreasing their anticipatory force and APA. This is a result of subjects learning, on average, to compensate for a small but nonzero perturbation magnitude. This is to be expected given the unidirectional force perturbations.

To quantify this mean learning in the arm, we examined noncorrected hand error in late baseline (mean across last 10 trials in baseline block) and in K0 force trials; hand error significantly increased in the negative direction from late baseline to K0 ($P < 0.001$), opposite to the direction of error in all nonzero gain groups (Fig. 3*A*). To quantify similar behavior in posture, we examined APAs and noncorrected RPAs in late baseline and in K0 force trials; RPAs did not

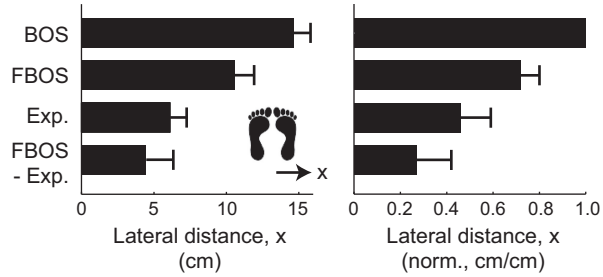

Fig. 6. Group mean values for lateral limit of base of support (BOS; 50% of stance width, measured from center), lateral limit of functional BOS (FBOS) (maximum lateral COP excursion measured in COP game), maximum lateral COP displacement measured during the experiment, and difference between lateral limit of functional BOS and maximum lateral COP displacement during the experiment. Error bars represent standard deviation across subjects. *Left*: mean of actual values; *right*: mean normalized values (normalized to 50% stance width for each subject).

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significantly change ($P = 0.920$) (Fig. 3*A*), but APAs increased in the direction of the force perturbation ($P = 0.025$). These results indicate that on average subjects were anticipating a positive force perturbation and exerted force at the hand along with a small APA to counter the expected perturbation, and in the absence of that perturbation they experienced a hand error in the opposite direction (Darainy and Ostry 2008; Lackner and Dizio 1994; Osu et al. 2003; Shadmehr and Mussa-Ivaldi 1994). The APA was sufficiently small that the resulting RPA was indistinguishable from baseline lateral COP movement. These findings explain why subjects showed negative or zero error for K0 in the arm and in posture, respectively, and negative adaptation for K0 in both arm and posture (Fig. 4, *I* and *L*); when subjects anticipated a force perturbation and encountered no force in K0 force trials, they reduced their anticipatory control in response (negative adaptation).

To estimate the perturbation force that subjects were expecting, on average, we performed linear regressions of adaptation onto perturbation force, using mean subject values at each perturbation gain (Fig. 7). The perturbation force at which the adaptation regression line crosses zero indicates the force for which subjects show zero adaptation, suggesting that this is the force that subjects were expecting. Therefore, the expected force was estimated from the zero intercept of the regression lines. Adaptation and perturbation force showed a significant correlation in the arm [$F(1,49) = 71.13$, $P < 0.001$] and in posture [$F(1,49) = 51.96$, $P < 0.001$]. The zero-intercept of the regression lines occurred at a perturbation force of ~1.3 N for arm adaptation and 3.6 N for postural adaptation, indicating that in each modality subjects learned to compensate for perturbations of that magnitude.

While subjects did demonstrate a change in their mean learned behavior over time, as described above, we wished to be sure that subjects' error and adaptation behavior in response to each perturbation gain were not changing over time. To examine this we performed linear regressions at each perturbation gain of arm error, arm adaptation, postural error, and postural adaptation vs. batch (excluding the first batch). These showed no significant trends at any gain value (all $P \geq 0.104$).

DISCUSSION

The results of this study demonstrate that adaptation scales proportionally with error in the arm and near proportionally in posture. In both modalities, error and adaptation were found to increase significantly with perturbation gain. Adaptation was significantly correlated with error size, and a linear model was the best fit to this data in both arm and posture. However, we did find that in posture only, adaptation showed no correlation with error for small error magnitudes. This finding may be explained as an effect of uncertainty, as discussed below. The observed differences between arm and posture (adaptation response to error, as well as mean learning) provide further support for the idea that adaptation of arm and postural control occur via similar but distinct mechanisms (Ahmed and Wolpert 2009; Manista and Ahmed 2012).

Linear Relationship Between Adaptation and Error

We found that in posture as well as in arm reaching, adaptation generally scales linearly with error. A linear relationship suggests that sensitivity to error, defined as the amount of adaptation normalized by the magnitude of the error, is constant across error magnitudes. This is in agreement with several studies of arm reaching and eye saccades, which showed good fits to experimental adaptation data using models with a linear trial-to-trial relationship between error and adaptation (i.e., a constant adaptation gain) (Baddeley et al. 2003; Colagiorgio et al. 2015; Donchin et al. 2003; Ethier et al. 2008; Franklin et al. 2008; Smith et al. 2006; Thoroughman and Shadmehr 2000). A linear relationship in posture is also supported by a study which found that after subjects practiced responding to a predictable postural perturbation of different magnitudes, they generated compensatory APAs that scaled linearly with the known magnitude of the impending perturbation (Horak and Diener 1994).

Our finding of a linear relationship is seemingly in conflict with some studies of arm reaching that found that adaptation became sublinear, or "saturated," at larger error magnitudes (Fine and Thoroughman 2006; Marko et al. 2012; Wei and Kording 2009). Error sensitivity was also reported to decrease with increasing error size (Marko et al. 2012; Wei and Kording 2009). However, those findings may be a result of the fact that each of these experiments used a zero-mean (bidirectional) distribution of perturbation magnitudes, as explained by Herzfeld et al. (2014): "With such a distribution, error-sensitivity declines (and as a consequence, learning from error saturates) for the large errors produced by the perturbations near the bounds. This is because after experiencing an error from a perturbation near one of the bounds, it is much more likely that the next perturbation will produce a change in the sign of the error than not." Here, Herzfeld et al. were referring specifically to the studies by Fine and Thoroughman (2006) and Wei and Kording (2009) and used an adaptation model to demonstrate their argument and replicate the data findings from those two studies. In fact, all three of the above experiments (Fine and Thoroughman 2006; Marko et al. 2012; Wei and Kording 2009) used such a distribution and thus are subject to the same explanation. This argument is supported by the findings of Fine and Thoroughman (2007): the slope of adaptation vs. pertur-

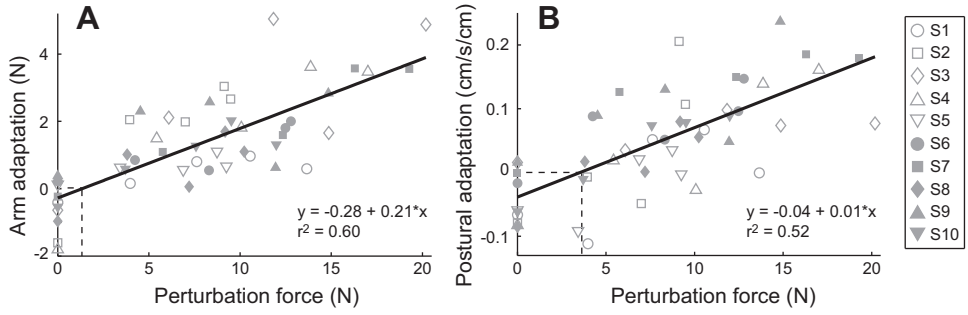

Fig. 7. Linear regressions of adaptation vs. perturbation force for arm (*A*) and posture (*B*), using mean values of adaptation and error at each error bin for each subject (data for each subject is plotted using a unique marker).

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bation magnitude became sublinear at larger perturbation magnitudes, for force distributions with zero bias (bidirectional), but the slope became steeper and more linear as the directional bias of forces increased to strongly unidirectional. These findings were also replicated by the modeling work of Herzfeld et al. (2014). Our experiment used a unidirectional distribution of perturbation magnitudes, which ruled out the possibility of saturation due to this mechanism.

Since we did not directly compare unidirectional vs. bidirectional perturbation distributions, we cannot conclusively state that this is why we observed no saturation. However, in those experiments, which found saturating adaptation with increasing error size, the largest proprioceptive errors that subjects experienced (caused by a force perturbation) were ~5 cm (Marko et al. 2012; Fine and Thoroughman 2007) or <3 cm (Fine and Thoroughman 2006). Our results showed a linear relationship between adaptation and error for a range of arm error sizes, which included and exceeded (>6 cm) those error sizes, ruling out the possibility that our arm error sizes were too small to cause saturation.

Postural Adaptation Does Not Scale with Small Errors

We did find that while postural adaptation scaled linearly with error for larger error magnitudes, there was no correlation between postural adaptation and error at small error magnitudes. We also found that these small errors did not significantly differ in size from postural errors (RPAs) experienced in late baseline. Thus the lack of error-specific adaptation may be explained as an effect of uncertainty, due to the size of those small errors relative to inherent movement variability. We suggest that postural errors below some threshold magnitude (related to inherent postural sway for a given set of conditions) are indistinguishable from inherent postural sway magnitudes, thus causing increased uncertainty about the error signal due to a small signal-to-noise ratio ("signal" being postural errors, and "noise" being inherent postural sway magnitudes).

Torres-Oviedo and Bastian (2012) found that in adaptation to a locomotor perturbation, smaller errors (induced by gradual introduction of the perturbation) led to reduced adaptation, compared with larger errors (induced by abrupt introduction of the same full-strength perturbation). However, the small errors experienced in the gradual case were significantly different from errors experienced in preperturbation baseline conditions and were consistently biased in one direction due to the perturbation; therefore, they could serve as a distinct and reliable error signal for adaptation, unlike the small postural errors in our experiment. It has been suggested that in adaptation paradigms utilizing an external dynamic perturbation, smaller errors are more likely to be attributed to the body, whereas larger errors will be attributed to the environment (e.g., robotic training device) (Berniker and Kording 2008; Kluzik et al. 2008; Torres-Oviedo and Bastian 2012). It is possible that in our experiment, small postural errors experienced on zero-gain force trials and null trials are small enough to be attributed to inherent postural variability and thus may not be attributed to the presence or absence of a perturbing force. Thus the lack of correlation between adaptation and small errors could be explained as an effect of uncertainty about the source of small errors.

Other studies have shown more explicitly that increased uncertainty can lead to reduced adaptation. For example, Wei and Kording (2010) found that in adaptation of reaching movements over repeated trials, adaptation rate was slower when uncertainty of visual feedback was increased (noise was added to cursor position); similarly, Stevenson et al. (2009) found that in a standing task where subjects controlled their COP position in the presence of random visual perturbations to COP cursor feedback, responses to perturbations were smaller when noise was added to the cursor position. More generally, postural error in our experiment may have been subject to greater uncertainty than arm error due to the fact that subjects received explicit visual feedback (hand cursor) as well as proprioceptive feedback about hand movements but did not receive explicit visual feedback about postural control during movements.

Another possible explanation for the lack of specific adaptation to small postural errors could be that subjects chose not to adapt to these errors. The fact that these errors were no larger than those experienced in unperturbed baseline movements indicates a negligible destabilizing effect on posture, and thus perhaps no adaptation was deemed necessary to maintain postural stability. In contrast, errors in arm movements must be corrected to reach the target.

Possible Effects of Postural Stability and Threat

Prior findings suggest that error sensitivity and/or feedback gains may saturate or decrease at larger magnitudes according to stability constraints. Several studies have shown that postural feedback gains are scaled with perturbation magnitude according to postural constraints (Kim et al. 2009, 2012; Park et al. 2004); for example, two studies found that ankle feedback gains tend to decrease with increasing perturbation magnitude to remain within torque limits, while hip feedback gains tend to increase to maintain overall postural stability (Kim et al. 2009; Park et al. 2004). Manista and Ahmed (2012) showed that after subjects had learned a predictable perturbation in various reaching directions, adapted APAs for a backward perturbation were significantly smaller than for a forward perturbation. Because subjects had the biomechanical capacity to adapt similarly in both directions, Manista and Ahmed suggested that APAs were reduced in the backward direction due to the greater threat associated with a backward fall, and also due to smaller stability limits in the backward direction. Thus, sensitivity and adaptation to a given error can be modulated by that error's proximity to stability limits, independent of error size. A later study by Pienciak-Siewert et al. (2014) manipulated stability limits by changing stance width, to remove the confounding effect of postural threat; in that experiment, after subjects had learned a predictable lateral perturbation in either a wide or narrow stance width, COP APAs were found to be similar regardless of stance width. The results of that study suggested that APAs are not affected by changes in postural stability limits as long as the postural control remains within those limits. This finding supports the expectation that postural adaptation should increase proportionally with error as long as postural movements do not approach stability limits. In the present experiment, perturbations were not large enough to cause COP movements to approach the limits of the functional

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BOS; therefore, adaptation of APAs showed a linearly increasing relationship with error magnitude.

Postural threat, which refers to the consequence of moving outside postural stability limits, may be an additional factor that is associated with, but separate from, stability limits. The present experimental setup, where subjects are standing at floor level and being perturbed to the side, is unlikely to have elicited significant responses to postural threat. However, there are a number of studies that have explicitly modulated postural threat and observed differences in behavior. In an arm reaching task where subjects avoided crossing a virtual "cliff," Trent and Ahmed (2013) found that trial-to-trial adaptation was decreased at the largest perturbation magnitudes; therefore adaptation can be modified by the threat associated with a particular error, independent of error magnitude. Manista and Ahmed (2012) showed that postural adaptation can be reduced by increased postural threat (backward vs. forward perturbations). Other studies have shown that under conditions of increased postural threat (standing at greater height), COP movements are reduced in quiet standing (Adkin et al. 2000; Carpenter et al. 1999, 2006; Davis et al. 2009; Hauck et al. 2008) and APAs associated with voluntary movements are reduced (Adkin et al. 2002).

Based on these findings, we might expect postural adaptation to saturate at larger perturbation magnitudes, where COP movements approach the limits of the functional BOS, and/or in conditions of increased postural threat, where subjects choose to further restrict their COP movements.

Mean Learning

When exposed continuously to perturbations of randomly varying strengths (e.g., without interference from multiple null trials or channel trials), subjects tend to adopt a predictive control that would effectively compensate for a perturbation representing the approximate mean of the distribution; this has been seen in arm reaching (Fine and Thoroughman 2007; Scheidt et al. 2001; Trent and Ahmed 2013) and in posture (Horak and Diener 1994; Horak et al. 1989). We observed that subjects adopted a predictive control compensating for a very small perturbation, roughly equivalent to or smaller than the smallest perturbations experienced on nonzero force trials, despite experiencing an even distribution of larger perturbations. This is most likely due to the fact that in our protocol, subjects were not exposed to a force perturbation on every trial but encountered multiple channel trials as well as a varying number of null trials in between force trials; this prevented continuous learning and resulted in a varying amount of deadaptation between subsequent trial triplets.

We also noted that subjects adopted a predictive postural control that was targeted to a larger perturbation magnitude (~3.6 N) than the predictive arm control (~1.3 N). This may be due to the inherent risk associated with postural perturbations, leading to a more conservative strategy (i.e., anticipating a slightly larger perturbation). A previous study found that when exposed to randomly varying postural perturbations of two possible magnitudes, subjects generated APAs that were scaled to the larger magnitude rather than the smaller or an intermediate magnitude; it was suggested that the larger APA was generated because a smaller or intermediate sized APA might be insufficient for the larger perturbation and thus might result in a fall (Beckley et al. 1991).

Clinical Implications

Some clinical populations demonstrate undersized APAs, such as elderly adults (Woollacott et al. 1988) and Parkinson's patients (Beckley et al. 1993; Traub et al. 1980). For example, when anticipating a predictable perturbation, Parkinson's patients scale their APAs with predicted perturbation magnitude for smaller magnitudes, but at larger magnitudes their APAs saturate and no longer scale with magnitude, despite these subjects having the capability to generate larger COP movements (seen in RPAs) (Horak et al. 1996; Smith et al. 2012). Our findings on the relationship between postural adaptation and error may offer some insight about rehabilitation of postural control in these and other cases.

Results from this and previous studies demonstrate that adaptation can be increased in postural control and in locomotion when subjects experience larger errors and/or stronger perturbations (Beckley et al. 1991; Green et al. 2010; Horak and Diener 1994; Horak et al. 1989; Smith et al. 2012; Torres-Oviedo and Bastian 2012). Therefore, adaptation might be increased by having subjects train in conditions of increased BOS size (e.g., by using external supports) and/or reduced postural threat (e.g., standing at ground level or otherwise reducing the threat/risk of a fall); these factors could encourage subjects to not restrict their postural movements and would thus allow for larger postural errors and may lead to greater adaptation. For example, Wulf et al. (1998) found that when learning to use a ski simulator, subjects performed better when they trained with ski poles, which increase the size of the BOS and facilitate larger amplitude, higher frequency movements. Similarly, Domingo and Ferris (2009, 2010) found that when subjects were trained to walk on a balance beam (which restricts the BOS), subjects who trained on a wider beam (slightly larger BOS and reduced postural threat) showed greater improvements in performance compared with those who trained on a narrower beam.

However, greater transfer of learning outside the training environment can arise from smaller errors (closer to the range of errors caused by natural variability) compared with larger errors. As described earlier, smaller errors may be more likely to be attributed to the body rather than to the training environment (e.g., robotic training device), and therefore the adaptation associated with those errors will also be linked to the body and will be better transferred to other contexts outside the training environment; larger errors will be attributed to the environment and will not be transferred as well (Berniker and Kording 2008; Kluzik et al. 2008; Torres-Oviedo and Bastian 2012). For example, two previous studies found that in adaptation to a dynamic perturbation, smaller errors (induced by gradual introduction of the perturbation) led to reduced adaptation but also led to increased magnitude and percentage of transfer, compared with larger errors (induced by abrupt introduction of the same full-strength perturbation) (Kluzik et al. 2008; Torres-Oviedo and Bastian 2012). Based on our finding that very small postural errors (indistinguishable from baseline performance) did not correlate with adaptation, we further suggest that rehabilitative paradigms should be designed to cause errors that are small, to promote transfer, but that are

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large enough to be distinct from baseline performance, to ensure that errors will drive adaptation.

This would be of particular importance in populations who make larger and more variable baseline errors in their postural control, such as older adults (Campbell et al. 1989; Overstall et al. 1977; Maki et al. 1994; Melzer et al. 2004) and stroke survivors (Reisman et al. 2009). Because of their larger baseline errors, these populations might tend to associate larger environmentally induced errors with themselves rather than with the environment and thus may transfer their adaptation more than subjects who make smaller baseline errors (Reisman et al. 2009; Torres-Oviedo and Bastian 2012). Conversely, these populations might demonstrate a lack of error-specific adaptation to a greater range of "small" errors if they are unable to distinguish those errors from their natural errors. Such an effect might also be caused by a decline in proprioception and/or increased reliance on visual rather than proprioceptive feedback, which can occur in older adults (Seidler-Dobrin and Stelmach 1998; Skinner et al. 1984) and Parkinson's patients (Jacobs and Horak 2006). In such cases there might be increased uncertainty about postural control, which could lead to reduced adaptation.

Anticipatory Postural Control is Cerebellum Dependent

Previous studies have shown that adaptation of arm reaching to dynamic or visuomotor perturbations is dependent on the cerebellum (Maschke et al. 2004; Smith and Shadmehr 2005; Tseng et al. 2007). This is related to the idea that, more generally, the cerebellum is important in the formation and adaptation of internal models (Maschke et al. 2004); subjects with cerebellar ataxia demonstrate a reduced ability to compensate for complex mechanical properties of the arm (such as interaction torques), suggesting that cerebellar damage inhibits the use of internal models to generate appropriate feedforward control (Bastian et al. 1996, 2000; Topka et al. 1998). Similarly, Horak and Diener (1994) found that subjects with cerebellar damage generate APAs that are inappropriately sized for a predictable perturbation of known magnitude, and they do not scale their APAs to the magnitude of the impending perturbation. However, the same study showed that healthy subjects generate APAs that increase linearly with both amplitude and velocity of the impending perturbation. This agrees with our finding that postural adaptation increases linearly with error, within the range where uncertainty and stability limits do not have a significant altering effect. Taken together, these findings indicate that the cerebellum is critical to the process of appropriately generating and adapting anticipatory control of standing posture, similar to what has been observed in arm reaching.

Limitations

Subjects were explicitly instructed not to lean on the handle of the robot arm for support. To ensure that they were not leaning on the handle, we measured the vertical forces exerted on the handle during the experiment, using a force transducer in the handle. The average peak vertical forces were 1.15 ± 0.27 N ($<1\%$ of bodyweight). These forces are sufficiently low to indicate that subjects were not leaning on the robot handle as a postural support. Additionally, the handle was free to move in the horizontal plane and therefore was not likely to provide significant postural support. However, it should be noted that the motion and forces related to the arm movement do have an impact on balance. Experimentally, it is not possible to completely separate out the components of postural movement that are attributable to anticipatory control, reactive control, and arm movement. In the future, modeling work could help to disambiguate the contributions of these factors.

Several previous studies have shown that sensitivity to error can be altered by several factors including the frequency and predictability of perturbations, and the duration of exposure (Fine and Thoroughman 2007; Herzfeld et al. 2014). These and other findings demonstrate that error sensitivity is highly dependent on the context and history of errors. However, this does not detract from the strength of our findings, as our focus was on comparing arm and postural adaptation. We also controlled for those factors by ensuring that they did not change between subjects or between modalities (arm and posture).

Conclusions

The results of this study demonstrate that trial-to-trial adaptation scales proportionally with error in the arm and near proportionally in posture. Interestingly, in posture only, adaptation showed no correlation with error for small error magnitudes, similar in size to errors that were experienced in unperturbed baseline movements due to inherent postural sway. This finding might be explained as an effect of uncertainty about the source of small errors. It is also noteworthy that perturbations in this experiment were not large enough to cause COP movements to approach the limits of the functional BOS; in the future, it would be interesting to investigate how the relationship between postural adaptation and error changes at larger error sizes approaching those limits. Generally, our findings suggest that in the design of rehabilitation and training regimens, postural error size should be considered relative to the magnitude of inherent movement variability.

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DISCLOSURES

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

AUTHOR CONTRIBUTIONS

A.P.-S. and A.A.A. conception and design of research; A.P.-S. and D.P.H. performed experiments; A.P.-S. analyzed data; A.P.-S. and A.A.A. interpreted results of experiments; A.P.-S. and D.P.H. prepared figures; A.P.-S. drafted manuscript; A.P.-S. and A.A.A. edited and revised manuscript; A.P.-S., D.P.H., and A.A.A. approved final version of manuscript.

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