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RESEARCH ARTICLE | *Control of Coordinated Movements*

Role of muscle coactivation in adaptation of standing posture during arm reaching

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Pienciak-Siewert A, Horan DP, Ahmed AA. Role of muscle coactivation in adaptation of standing posture during arm reaching. *J Neurophysiol* 123: 529–547, 2020. First published December 18, 2019; doi:10.1152/jn.00939.2017.—The ability to maintain stable, upright standing in the face of perturbations is a critical component of daily life. A common strategy for resisting perturbations and maintaining stability is muscle coactivation. Although arm muscle coactivation is often used during adaptation of seated reaching movements, little is known about postural muscle activation during concurrent adaptation of arm and standing posture to novel perturbations. In this study we investigate whether coactivation strategies are employed during adaptation of standing postural control, and how these strategies are prioritized for adaptation of standing posture and arm reaching, in two different postural stability conditions. Healthy adults practiced planar reaching movements while grasping the handle of a robotic arm and standing on a force plate; the robotic arm generated a velocity-dependent force field that created novel perturbations in the forward (more stable) or backward (less stable) direction. Surprisingly, the degree of arm and postural adaptation was not influenced by stability, with similar adaptation observed between conditions in the control of both arm movement and standing posture. We found that an early coactivation strategy can be used in postural adaptation, similar to what is observed in adaptation of arm reaching movements. However, the emergence of a coactivation strategy was dependent on perturbation direction. Despite similar adaptation in both directions, postural coactivation was largely specific to forward perturbations. Backward perturbations led to less coactivation and less modulation of postural muscle activity. These findings provide insight into how postural stability can affect prioritization of postural control objectives and movement adaptation strategies.

NEW & NOTEWORTHY Muscle coactivation is a key strategy for modulating movement stability; this is centrally important in the control of standing posture. Our study investigates the little-known role of coactivation in adaptation of whole body standing postural control. We demonstrate that an early coactivation strategy can be used in postural adaptation, but muscle activation strategies may differ depending on postural stability conditions.

anticipatory postural adjustment; coactivation; motor adaptation; neuromechanics; postural base of support

INTRODUCTION

The ability to maintain stable, upright standing is a critical component of many of our daily activities. This ability requires that we generate appropriate postural control when making voluntary movements or responding to perturbations; importantly, this includes anticipatory postural control, which precedes the postural disturbance associated with a predictable perturbation or voluntary movement, and helps to maintain postural equilibrium by compensating for the impending shift in body mass and other movement dynamics (Belen'kiĭ et al. 1967; Massion 1992; Traub et al. 1980; Winter et al. 1990). We must also adapt our postural control appropriately when needed. Despite this, there have been only a few previous studies investigating how we adapt our standing postural control in response to novel perturbations. Adaptation has been well studied in arm reaching movements, but there are fundamental differences between whole body and arm reaching movements. Upright standing is inherently unstable; postural movements are subject to stability limits (typically defined using the postural base of support, or BOS), which limit the capacity to generate a movement or to recover from a postural perturbation (Holbein-Jenny et al. 2007; Koozekanani et al. 1980; Pai and Patton 1997; Patton et al. 1999; Schulz et al. 2006). Maintaining postural stability (i.e., maintaining the ability to generate and support whole body movement and to recover from perturbations and avoid a fall) is especially important for older adults and other clinical populations in which poor postural control can be linked to falls and greater mortality risk (Adkin et al. 2003; Duncan et al. 1990, 1992; Feldman and Robinovitch 2004; Holbein-Jenny et al. 2007; Schenkman et al. 2000; Tiedemann et al. 2008). Because effective postural control is critical to performing daily activities and avoiding injury, it is important to understand the mechanisms of postural adaptation.

Previous studies of motor adaptation in arm reaching movements have identified two predictive control strategies that are used in adaptation to novel dynamics: force control and impedance control (Osu et al. 2002, 2003; Takahashi et al. 2001). In the force control strategy, muscle activations are modified in a predictive manner to generate net torques about specific joints and/or a specific net force to counter the novel dynamics. The impedance control strategy is more generalized and involves coactivation of opposing agonist-antagonist muscles; when the coactivated muscles exert equal and opposite torques

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on a joint, no net torque, and thus no movement, is produced, but the mechanical impedance or "stiffness" of the joint is altered (Darainy et al. 2004; Gomi and Osu 1998; Hogan 1984, 1985). Several studies have shown that during adaptation of arm reaching to novel but predictable dynamics, coactivation levels and arm joint impedances are increased early in the adaptation process but decrease later (Darainy and Ostry 2008; Milner and Cloutier 1993; Thoroughman and Shadmehr 1999; van Emmerik 1992). This initial increase in coactivation is thought to be an important part of the adaptation process in that it helps to reduce movement errors and thus provides stability while the novel dynamics are still being learned; as the dynamics are learned and perturbation-specific joint torques are modified accordingly, impedance can then be decreased without compromising stability or performance (Franklin et al. 2003; Hinder and Milner 2007; Katayama et al. 1998; Milner and Franklin 2005; Osu et al. 2002).

The above-mentioned studies clearly indicate that coactivation is a key strategy for controlling movement stability during adaptation. However, despite the central importance of stability in standing postural control, no previous studies have examined the role of coactivation in postural adaptation. Ahmed and Wolpert (2009) first demonstrated that when subjects adapted their arm reaching to novel dynamics while standing, their standing postural control also showed adaptation to the novel dynamics. Postural adaptation was reflected in the emergence of anticipatory postural adjustments, similar to those commonly observed in voluntary arm movements (Aruin and Latash 1995, 1996; Bouisset and Zattara 1987; Cordo and Nashner 1982). They did not measure muscle activity, but they did find that postural error was reduced more quickly than the corresponding anticipatory postural control was developed (analogous to dynamic learning in the arm), which suggests that subjects may have initially used a postural coactivation strategy to reduce errors, despite not yet having learned to counter the perturbation in a predictive manner. Manista and Ahmed (2012) used a similar experimental paradigm, in which subjects adapted their arm reaching to novel dynamics while standing, and adapted in multiple reaching directions, with the direction of the perturbation corresponding to reach direction. They found that after subjects had adapted their arm and postural control, anticipatory postural control for a backward perturbation was significantly smaller than for a forward perturbation. They found no corresponding difference in postural error, which suggests that subjects may have maintained postural stability by using a more coactivated strategy to compensate for perturbations in the backward direction. However, muscle activity was not measured in that experiment, so that explanation remains speculative. Nonetheless, there is additional support for this idea from several studies which found that in conditions of reduced stability limits and/or increased postural threat, subjects generally display reduced postural movement amplitudes and/or increased postural coactivation (Adkin et al. 2002; Gendre et al. 2016; Kaminski and Simpkins 2001; Krishnamoorthy et al. 2004; Yiou et al. 2007).

The directional dependence of adaptation of standing posture has the potential to provide a unique means to probe the effects of stability limits on adaptation strategies. Therefore, in the present study we wanted to capture a more comprehensive picture of how postural adaptation strategies might differ for a forward versus a backward perturbation, and how these strategies are prioritized for adaptation of posture and reaching. We used an experimental paradigm similar to that of Manista and Ahmed (2012). Subjects reached in only one direction and adapted their arm reaching movements and postural control to a novel perturbation in either the forward or backward direction (to reduce any possible uncertainty associated with reaching in multiple directions and to increase subjects' familiarity with the specified movement and perturbation direction). The primary objective was to compare the extent of postural adaptation and coactivation in the forward versus backward directions, to examine the effects of differing postural stability conditions. Based on evidence from previous studies discussed above, we hypothesize that subjects would adapt using reduced anticipatory postural control in the backward direction compared with the forward direction and would compensate using increased postural coactivation to maintain postural stability. If we did not observe this pattern, and/or if subjects used an alternative compensatory mechanism, the results might show a more nuanced effect of stability on adaptation and muscle activation strategies.

METHODS

Twenty healthy young adult subjects (8 men, 12 women; age 22.1 ± 2.2 yr; height 171.1 ± 8.3 cm; mass 64.7 ± 9.8 kg; means $\pm$ SD) 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. All subjects gave written informed consent before participating in the experiment. The University of Colorado Boulder Human Research Committee approved all experimental procedures.

Experimental Apparatus and Setup

Subjects made reaching movements in the horizontal plane with their right hand while grasping the handle of a two-degree-of-freedom planar robotic arm (InMotion2 shoulder-elbow robot; Interactive Motion Technologies Inc.) and while standing barefoot on a six-axis, dual-plate force platform (AMTI dual-top AccuSway; Advanced Mechanical Technology Inc.) (Fig. 1*A*). The subject's 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 122.5 ± 4.9 cm across subjects). A computer monitor, vertically suspended in front of the subject, displayed visual feedback of hand, start, and target positions throughout the movement.

In the experiment, subjects made 15-cm reaching movements to the right ($+x$), using the robot handle to control the cursor on the screen. 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 the location of their center of pressure (COP; represented by a separate 0.6-cm cursor of a different color) anywhere within the start circle (Fig. 1*A*). After a short time delay, the COP cursor 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. After each movement, subjects also received visual feedback about the 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 encourage subjects to complete the reaching movements within a window of 450 to 600 ms.

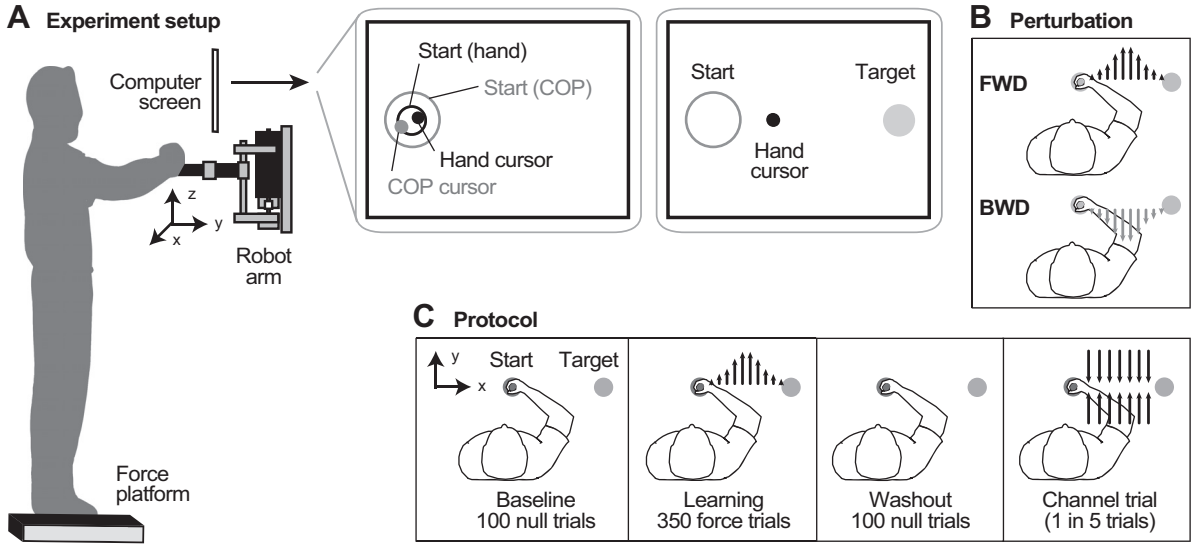

Fig. 1. Experimental setup and protocol. *A*: Experimental apparatus and setup; visual feedback is provided on computer screen. *B*: subjects experienced either a forward (FWD) or backward (BWD) perturbation during the learning block. *C*: experimental protocol; a rightward reach ($+x$) and perpendicular forces ($\pm y$) are illustrated. COP, center of pressure.

To ensure that each subject's COP movements started from a comfortable center position, we performed a centering exercise before the experiment began. The start circle and COP cursor were shown on the screen. Subjects were asked to choose a comfortable stance and adjust their foot placement as necessary while keeping their feet parallel and symmetrical on either side of the center line such that the COP cursor was centered in the start circle when they were standing still. Their exact foot position was marked on the force platform to ensure that they always stood in the same location. (Mean values for foot placement dimensions are included in Table 4.) Throughout the experiment, subjects were asked to keep their feet flat on the ground, to ensure that the size of the base of support (BOS) was not affected by lifting or rotation of the feet.

Experimental Protocol

The protocol consisted of 550 trials and was divided into three consecutive blocks: baseline (100 null trials), learning (350 force trials), and washout (100 null 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. Null trials were also used in the washout block at the end of the experiment to allow the subject to deadapt the previous dynamic environment. The learning block consisted of force trials, in which the robot simulated a viscous curl field by exerting a force F on the hand that was proportional to the magnitude and perpendicular to the direction of the instantaneous velocity V of the robot handle (*Eq. 1*). Thus, for a rightward reaching movement ($+x$), the robot generated forward or backward perturbing forces ($\pm y$) depending on the sign of the field gain $k = \pm 20$ N·s·m^{-1}.

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

In every batch of five trials, one trial was chosen randomly to be a channel trial (i.e., each batch consisted of 5 trials: 4 null or force trials and 1 catch trial). These trials were used to quantify subjects' predictive, feed-forward arm control. In channel trials, the robot generated a force channel that restricted the subject's hand trajectory to a straight path between the start position and the target; the robot could then measure the amount of perpendicular force that the subject was exerting into the channel. Stiffness and damping for the channel were 2,000 N/m and 50 N·s·m^{-1}, respectively. These trials have been shown to have a minimal effect on adaptation or deadaptation (Scheidt et al. 2000).

Subjects were randomly assigned into one of two groups, FWD or BWD, with $n = 10$ per group. The FWD group experienced a force perturbation in the forward direction ($k = -20$ N·s·m^{-1}), and the BWD group experienced a force perturbation in the backward direction ($k = +20$ N·s·m^{-1}) (Fig. 1*B*). The sequence of trial types was identical for all subjects.

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 (Holbein-Jenny et al. 2007; King et al. 1994; 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 eight randomized targets located in different directions, evenly spaced around a 360° circle at 45°angles. Subjects were encouraged to move their COP out as far as possible without lifting their feet, moving their arms, or bending at the waist, hips, or knees.

Data Collection and Analysis

Position, velocity, and force data from the robot handle were sampled at 200 Hz. Center of pressure (COP) position data were calculated from force platform data, which were 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 foot length.

Surface electromyography (EMG) data were collected using a wireless electrode system (Trigno wireless system; DelSys Inc.) with a fixed interelectrode distance of 1 cm on each sensor and a signal bandwidth of 20–450 Hz. EMG data was sampled at 2,000 Hz from the pectoralis major (Pec), posterior deltoid (PDelt), biceps brachii (Biceps), and long head of the triceps of the right arm (reaching arm),

and from the rectus femoris (RF), biceps femoris (BF), tibialis anterior (TA), peroneus longus (PL), medial gastrocnemius (MGas), lateral gastrocnemius (LGas), and soleus (Sol) of the left leg. (The left leg was chosen based on pilot data, which showed that for both forward and backward perturbations, muscle activity adaptations were stronger within individual subjects, and more consistent between subjects, in the left leg compared with the right leg. This supported the expectation that a larger muscle response should be seen in the left leg, due to a greater moment arm about the left leg for force perturbations acting on the right arm.) Electrodes were placed according to SENIAM guidelines (Surface Electromyography for the Non-Invasive Assessment of Muscles, http://www.seniam.org).

All data were aligned to movement onset, such that *time 0* represents movement onset of the arm, and truncated at movement end. Movement onset was defined as 50 ms before the time when tangential hand position and velocity exceed threshold values of 0.25 cm and 2 cm/s, respectively. Movement end was defined as the time the cursor reached the target circle. All data were taken from movement onset to movement end, unless otherwise noted. Data from channel trials were analyzed separately from all other trials.

Trials were excluded from analysis if the movement onset criterion was inaccurate (by visual inspection) or if the data were corrupted. A total of 147 trials were rejected from the entire data set, with 70 trials excluded for the FWD group and 77 trials for the BWD group (of 5,500 total trials per group, with 550 trials per subject). On average, 7.4 total trials (2.5 null trials, 3.8 force trials, and 1.1 channel trials) were rejected per subject.

Arm control. Control of the arm reaching movement was quantified using two metrics: hand error and anticipatory force. Hand error was calculated for each trial, excluding channel trials, as the peak signed value of the perpendicular deviation of the hand trajectory from a straight path between the start and target positions. Anticipatory force was calculated, for channel trials only, as the perpendicular channel force at the time of peak tangential hand velocity. This 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.

Postural control. Control of whole body standing posture was quantified for each trial, excluding channel trials, using two COP movement metrics: reactive postural adjustment (RPA; a measure of postural movement error) and anticipatory postural adjustment (APA; a measure of anticipatory control). These metrics were based on the normalized COP displacement in the direction of the force perturbation (perpendicular to the direction of reaching movement). We observed that COP velocity responses on the first learning trial, when subjects first encountered the unexpected force perturbation, began no earlier than 100 ms after movement onset, and COP displacement responses occurred later than that. Therefore, as a conservative measure of anticipatory control, the APA was calculated as the peak signed value of COP displacement observed between 50 ms before movement onset and 100 ms after movement onset. The RPA was calculated as the peak signed value of COP displacement observed for the remaining duration of the movement (following the APA time period).

Muscle activity. Muscle activity was quantified for each trial, excluding channel trials. EMG data were high-pass filtered at 20 Hz to remove movement artifact, full-wave rectified, and then low-pass filtered at 50 Hz, using a zero-phase fourth-order Butterworth filter. For each subject, EMG data were normalized by dividing by the mean late baseline activity for each muscle, taken as the root-mean-square value of filtered EMG activity from movement onset to movement end (for arm muscles) or from 50 ms before movement onset to movement end (for posture muscles). For arm muscles, muscle activity was quantified as the root-mean-square value of normalized EMG activity from movement onset to movement end. For postural muscles, anticipatory muscle activity was quantified as the root-mean-square value of normalized EMG activity from 50 ms before movement onset to 100 ms after movement onset, and reactive muscle activity was quantified as the root-mean-square value of normalized EMG activity from 100 ms after movement onset to movement end. This timing was based on work by Horak and Nashner (1986), who investigated postural control responses to unexpected backward sway perturbations and observed reactive muscle response latencies varying from 73 to 110 ms; this was after repeated exposure, which is known to reduce the latency of automatic postural responses. Therefore, we chose 100 ms after movement onset as a conservative cutoff for anticipatory muscle activity.

Muscle coactivation. Coactivation was quantified for each trial, excluding channel trials, using methods similar to Thoroughman and Shadmehr (1999) and Gribble et al. (2003). For a given agonist-antagonist pair (e.g., Pec vs. PDelt), at each time sampling point in a given trial, the coactivation value was determined as the minimum value of normalized EMG (for each sampling point i, coactivation trace C is constructed from the individual muscle activity traces A and B such that $C_i = \min[A_i, B_i]$). The resulting time-varying signal represents the magnitude of normalized EMG that is matched by the two opposing muscles (expressed in $\mu V/\mu V$). The representative coactivation value for each trial was calculated as the root-mean-square value of the time-varying coactivation signal from movement onset to movement end (for arm muscle pairs) or from 50 ms before movement onset to movement end (for posture muscle pairs). Arm coactivation was quantified for the opposing muscle pairs about the shoulder (Pec vs. PDelt) and elbow (Biceps vs. Triceps). Postural coactivation was quantified for opposing muscles in the anterior-posterior direction: hip flexor/knee extensor (RF) vs. hip extensor/knee flexor (BF) and ankle dorsiflexor (TA) vs. plantarflexors (MGas, LGas, Sol).

Predictions and Statistical Analyses

Data were compared between groups and across six phases of the protocol: late baseline, first learning, early learning, late learning, first washout, and late washout. The first learning and first washout phases consisted of one trial only; for anticipatory force, these phases consisted of the first channel trial in the block, and for all other metrics, these phases consisted of the first force or null trial in the block. The early learning phase consisted of the first five batches (20 force trials, or 5 channel trials) of the learning block, excluding the first trial (first learning). The late baseline, late learning, and late washout phases consisted of the last five batches (20 null or force trials, or 5 channel trials) of the trial block. All metrics were normalized to the late baseline phase.

Hand error, anticipatory force, RPA, APA, and coactivation data were first analyzed using repeated-measures ANOVAs, with phase (all phases) as a within-subjects factor and group as a between-subjects factor (see Table 1). To test for the expected changes associated with adaptation, we made planned comparisons on the within-subjects results in each group between the late baseline, early learning, and late learning phases (one-tailed paired t tests, $\alpha = 0.05/2$).

To test our main hypothesis, we compared FWD and BWD postural variables: APA, RPA, and postural muscle coactivation. We expected the BWD group, compared with the FWD group, to show reduced APAs and/or RPAs, concomitant with greater postural coactivation. To test for differences between groups, we first compared groups in late baseline to ensure they showed similar behavior; we then made planned comparisons between groups of the magnitude of changes from late baseline to early learning and late learning (independent two-sample t tests, $\alpha = 0.05$).

Additionally, we characterized adaptation by fitting exponential functions to the individual trials t, $y = A + B \cdot e^{-t/k}$, where A and B represent the initial and final values, and k represents the time constant of learning. We fit hand error, anticipatory force, RPA, and APA data from the learning block for each group. To fit the data, we boot-

Table 1. *ANOVA results*

	Phase	Group	Interaction
Hand error	$P = 0.101$, $F = 1.91$	**$P < 0.001$, $F = 30.48$**	**$P < 0.001$, $F = 362.49$**
Anticipatory force	$P = 0.647$, $F = 0.67$	**$P < 0.001$, $F = 705.93$**	**$P < 0.001$, $F = 101.82$**
RPA	**$P < 0.001$, $F = 15.48$**	**$P < 0.001$, $F = 155.73$**	**$P < 0.001$, $F = 132.06$**
APA	$P = 0.771$, $F = 0.51$	**$P = 0.002$, $F = 13.67$**	**$P < 0.001$, $F = 19.94$**
Coactivation			
Pec-PDelt	**$P < 0.001$, $F = 10.78$**	$P = 0.452$, $F = 0.59$	$P = 0.092$, $F = 1.96$
Biceps-Triceps	**$P < 0.001$, $F = 10.48$**	$P = 0.887$, $F = 0.02$	$P = 0.378$, $F = 1.08$
RF-BF	**$P = 0.002$, $F = 4.17$**	$P = 0.916$, $F = 0.01$	$P = 0.760$, $F = 0.52$
TA-PL	**$P = 0.013$, $F = 3.09$**	$P = 0.564$, $F = 0.34$	$P = 0.138$, $F = 1.72$
TA-LGas	**$P < 0.001$, $F = 5.27$**	$P = 0.828$, $F = 0.05$	$P = 0.588$, $F = 0.75$
TA-MGas	**$P < 0.001$, $F = 9.41$**	$P = 0.752$, $F = 0.10$	**$P = 0.009$, $F = 3.26$**
TA-Sol	**$P = 0.004$, $F = 3.73$**	$P = 0.931$, $F = 0.01$	$P = 0.310$, $F = 1.21$

Data are ANOVA results showing effect of phase, effect of group, and interaction effect (phase × group) for hand error, anticipatory force, reactive postural adjustment (RPA), anticipatory postural adjustment (APA), and coactivation metrics. BF, biceps femoris; Biceps, biceps brachii; LGas, lateral gastrocnemius; MGas, medial gastrocnemius; PDelt; posterior deltoid; Pec; pectoralis major; PL, peroneus longus; RF, rectus femoris; Sol, soleus; TA, tibialis anterior; Triceps, long head of the triceps. Significant results are in bold type.

strapped, sampling from each group with replacement, and repeated this 1,000 times per group. The parameters A and B were compared between groups as a secondary analysis of movement control at the beginning and end of learning, and k was compared between groups as a measure of learning rate. Group comparisons were performed based on the 95% confidence intervals of the fitted parameters.

We also made specific predictions about changes in activity for individual postural muscles that might be involved in counteracting the force field. For a forward perturbation (FWD group), which exerts a net forward torque on the COM, we expected that subjects would adapt by increasing activity in muscles, which would generate a net backward torque on the COM to counter the perturbation: specifically, hip extensor (BF) and ankle plantarflexors (PL, MGas, LGas, Sol). Conversely, for a backward perturbation (BWD group), which exerts a net backward torque on the COM, we expected that subjects would adapt by increasing activity in muscles, which would generate a net forward torque on the COM: specifically, hip flexor (RF) and ankle dorsiflexor (TA). In both cases, we might also expect to see decreases in the muscles acting opposite to the primary agonists. To test for the predicted changes in postural muscle activity associated with adaptation, we made planned comparisons on the within-subjects results in each group between the late baseline and late learning phases (one-tailed paired t tests, $\alpha = 0.05/2$). Arm muscle activity was only measured to quantify coactivation, so we did not make any predictions about these data, but for the sake of completeness we made planned comparisons between late baseline and late learning (two-tailed paired t tests, $\alpha = 0.05$).

All data analyses were performed using MATLAB. Unless otherwise noted, within-subjects comparisons between phases were made using two-tailed paired t tests ($\alpha = 0.05$), and comparisons between groups were made using independent two-sample t tests ($\alpha = 0.05$). Mean values in the text are means ± SD unless otherwise noted.

RESULTS

Both groups displayed generally similar arm movement velocities and experienced similar force magnitudes in the learning block. When initially exposed to the force field, subjects exhibited movement errors in the direction of the perturbing forces; subsequently, all subjects adapted their arm and postural control as expected, with increases in error followed by gradual reduction and a concomitant increase in adaptation (Fig. 2). Interestingly, the magnitude of postural adaptation was not affected by the stability condition (forward vs. backward). Despite the similar adaptation, there was an effect of stability on muscle activation strategies. Although we did observe the use of an early coactivation strategy in postural adaptation similar to the coactivation observed in the adaptation of arm reaching movements, the direction of perturbation influenced the degree of coactivation. Counterintuitively, re-

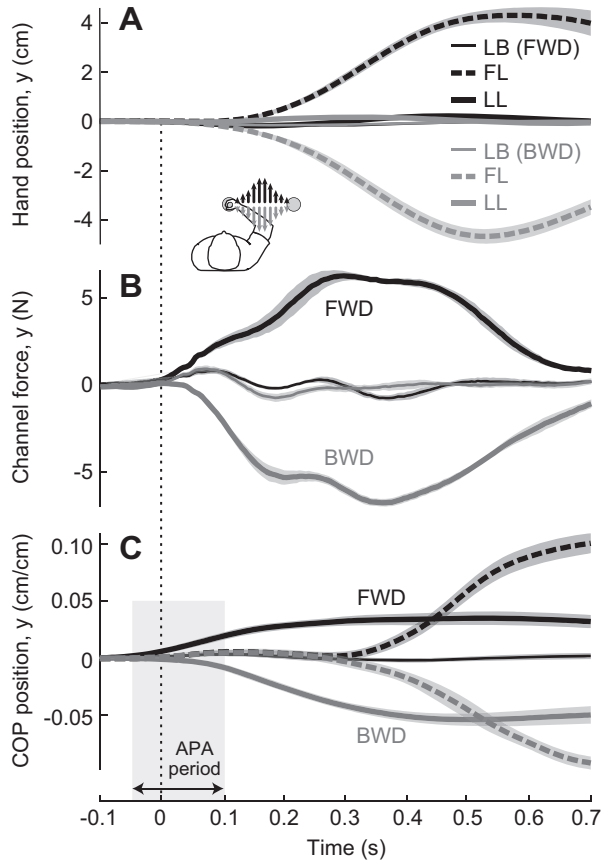

Fig. 2. Group mean movement trajectories. Late baseline (LB), first learning (FL), and late learning (LL) phases are shown for subjects experiencing either a forward (FWD group; black) or backward perturbation (BWD group; gray). *A*: perpendicular hand position (force trials only). *Inset* shows the direction of perturbing forces for each group. *B*: channel force (channel trials only). *C*: perpendicular center-of-pressure (COP) position (force trials only). Note: trajectories were averaged across trials in each phase for each subject and then averaged across subjects in each group. Gray box indicates the anticipatory postural adjustment (APA) period. Line shading indicates SE across subjects. *Time 0* represents movement onset of the arm.

duced stability (associated with the backward perturbation) led to lower levels of coactivation.

Arm Movement Adaptation

Before comparing adaptation between groups, we had to be sure that both groups made hand reaching movements with similar velocities and experienced similar forces in the phases of interest: late baseline, early learning, and late learning. At these phases of interest, peak velocities and peak field force magnitudes were similar between groups (all P values $\geq$ 0.208).

Hand error and anticipatory force data show that subjects adapted their arm control to the force field during the learning block and deadapted during the washout block (Fig. 3, *A* and *D*). In both groups, hand error magnitudes significantly increased from late baseline to first learning and significantly decreased from first learning to late learning (Fig. 3, *B–F*; Table 2). Anticipatory force magnitudes significantly increased from late baseline to late learning.

A comparison between groups revealed that the changes in anticipatory force and hand error from late baseline to early learning and late learning were similar (Fig. 3, *B–F*; Table 2). The parameters obtained from fitting exponential functions (of the form $y = A + B \cdot e^{-t/k}$, as described in METHODS) revealed a significant difference in the learning rate between groups, with the BWD group reducing hand error more quickly but displaying the same amount by the end [Fig. 3*G*; FWD: $A = 0.37$ (0.05), $B = 1.46$ (0.21), $k = 9.28$ (2.01); BWD: $A = 0.37$

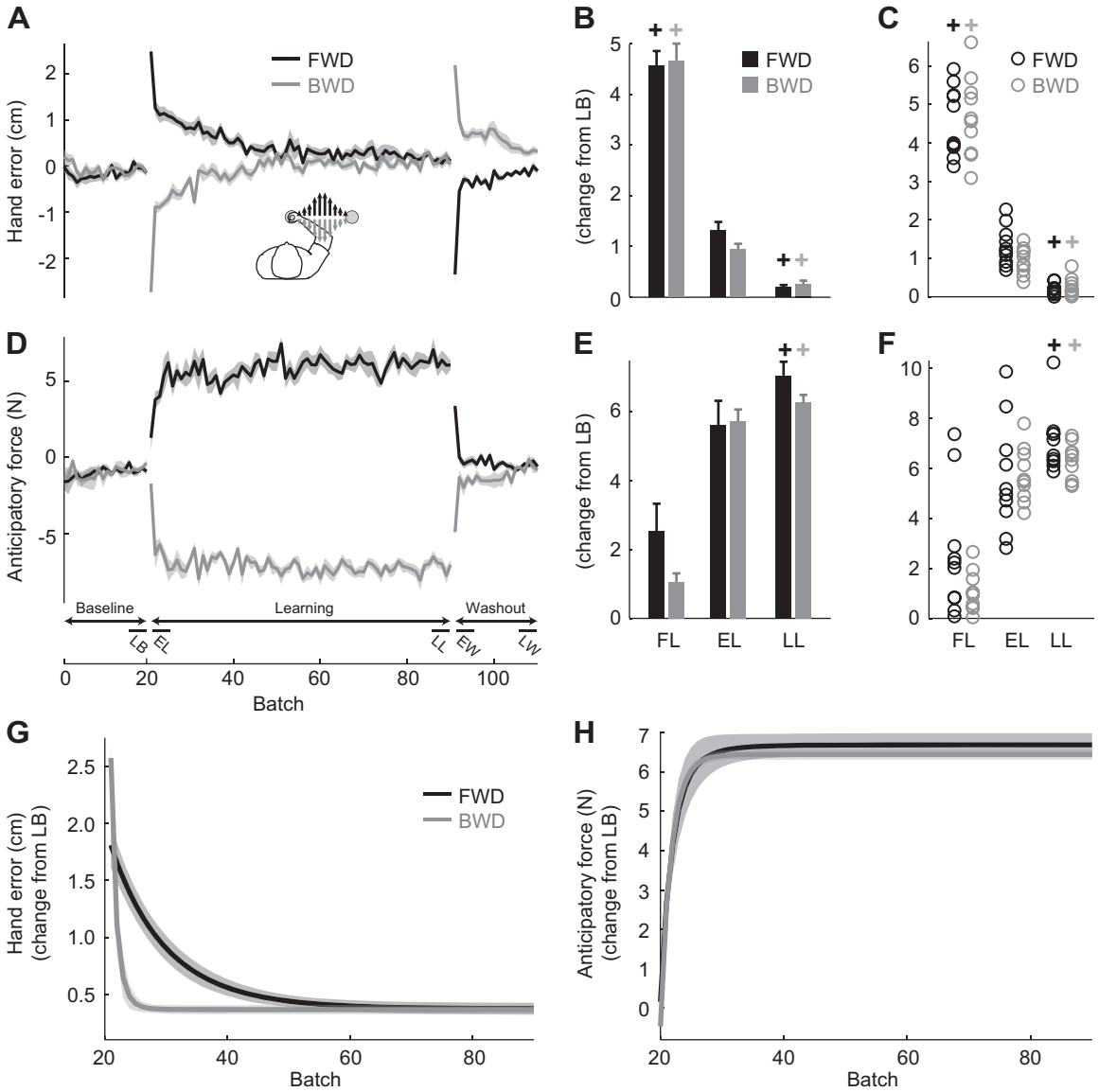

Fig. 3. Hand error and anticipatory force metrics. *A–C* and *D–F* show hand error and anticipatory force, respectively. *A* and *D* show each metric vs. batch (5 trials). In each plot, 2 traces show group means (solid lines) ± SE (shading) for the forward (FWD; black) and backward perturbation (BWD; gray) groups. Bar plots in *B* and *E* show the absolute magnitude of the change from late baseline (LB) to first learning (FL), early learning (EL), and late learning (LL) phases, for FWD group (black) vs. BWD group (gray), with error bars showing SE. Bar plots in *C* and *F* show the absolute magnitude of the change from LB for each subject. $+P < 0.050$, statistically significant change within group for hand error, from LB to FL or from FL to LL, and for anticipatory force, from LB to LL. *G* and *H* show exponential fits of hand error and anticipatory force data from the learning block, respectively. In each plot, 2 traces show bootstrapped mean data for FWD (black) and BWD (gray) across 1,000 bootstrap-resampled groups (solid lines are means, shading indicates ±SE).

Table 2. *Planned comparisons: hand error, anticipatory force, RPA, APA, and coactivation*

	FWD Group			BWD Group			Between Groups		
	LB vs. FL	FL vs. LL	LB vs. LL	LB vs. FL	FL vs. LL	LB vs. LL	LB	EL–LB	LL–LB
Hand error	**<0.001**	**<0.001**		**<0.001**	**<0.001**		0.829	0.067	0.525
Anticipatory force			**<0.001**			**<0.001**	0.816	0.893	0.101
RPA	**<0.001**	**<0.001**		**<0.001**	**<0.001**		0.582	0.064	0.114
APA			**<0.001**			**<0.001**	0.533	0.570	0.369
	LB vs. EL	EL vs. LL		LB vs. EL	EL vs. LL		LB	EL–LB	LL–LB
Coactivation									
Pec-PDelt	**<0.001**	**<0.001**		**<0.001**	**<0.001**		0.648	0.658	0.883
Biceps-Triceps	0.052	**0.027**		**0.016**	**0.002**		0.728	0.744	0.569
RF-BF	**0.041**	**0.012**		0.085	0.059		0.223	0.639	0.412
TA-PL	**0.002**	**<0.001**		0.193	0.403		0.147	0.215	0.477
TA-LGas	**0.006**	**0.001**		0.168	0.143		0.389	0.563	0.658
TA-MGas	**0.001**	**<0.001**		0.142	**0.038**		0.332	0.220	0.370
TA-Sol	**0.002**	**0.002**		0.160	0.232		0.489	0.269	0.838

Data are *P* values for the following planned comparisons: within group [forward (FWD) or backward perturbation (BWD)] for hand error, anticipatory force, reactive postural adjustment (RPA), anticipatory postural adjustment (APA): late baseline vs. first learning (LB vs. FL), first learning vs. late learning (FL vs. LL), and late baseline vs. late learning (LB vs. LL); within group (FWD and BWD) for coactivation metrics: late baseline vs. early learning (LB vs. EL) and early learning vs. late learning (EL vs. LL); between groups: at LB; and between groups: absolute magnitude of change from LB to EL (EL–LB) and late learning (LL–LB). BF, biceps femoris; Biceps, biceps brachii; LGas, lateral gastrocnemius; MGas, medial gastrocnemius; PDelt; posterior deltoid; Pec; pectoralis major; PL, peroneus longus; RF, rectus femoris; Sol, soleus; TA, tibialis anterior; Triceps, long head of the triceps. Significant results are in bold type.

(0.04), $B = 2.21$ (0.11), $k = 0.97$ (0.42); means (SE)]. No group differences were observed for anticipatory force [Fig. 3*H*; FWD: $A = 6.72$ (0.29), $B = -6.56$ (0.59), $k = 2.43$ (1.35); BWD: $A = 6.45$ (0.14), $B = -6.92$ (0.20), $k = 1.74$ (0.25)]. Generally, our results indicate that the groups adapted their arm control to a similar extent.

Arm Muscle Activity

To examine adaptation of arm muscle activity, we focused on changes from late baseline to late learning (Fig. 4*B*; Table 3). As expected, the FWD group showed significant decreases in activity in the pectoralis major and posterior deltoid, whereas the BWD group showed a significant increase in activity in the pectoralis major.

Subjects also modulated arm muscle coactivation over the course of learning (Figs. 5 and 6). In the FWD group, Pec-PDelt coactivation significantly increased from late baseline to early learning, and both Pec-PDelt and Biceps-Triceps coactivation then significantly decreased from early learning to late learning. In the BWD group, Pec-PDelt and Biceps-Triceps coactivation significantly increased from late baseline to early learning and then significantly decreased from early learning to late learning. A comparison of the magnitudes of the changes from late baseline to early learning and late learning (Fig. 5*B*; Table 2) revealed no significant differences between groups.

In summary, arm muscle activity shows that both groups increased their arm muscle coactivation in early learning and, over time, adapted their muscle activity to counteract the perturbation. Coactivation levels were generally similar between groups.

Postural Movement Adaptation

RPA and APA data show that subjects adapted their postural control as expected during the learning block and deadapted during the washout block (Fig. 7, *A* and *D*). In both groups, RPA magnitudes significantly increased from late baseline to first learning and significantly decreased from first learning to late learning. Similarly, APA magnitudes significantly increased from late baseline to late learning (Fig. 7, *B–F*; Table 2).

Our main goal was to compare adaptation of standing postural control between groups. To this end, we compared the magnitudes of the changes in RPAs and APAs from late baseline to early learning and late learning (Fig. 7, *B–F*; Table 2). Overall, RPAs increased on exposure and then decreased with learning, whereas APAs increased with learning. The changes in APAs and RPAs from late baseline to early learning and late learning were not significantly different between groups. The exponential fits corroborate these results [Fig. 7, *G* and *H*; APAs FWD: $A = -0.017$ (0.002), $B = -0.013$ (0.002), $k = 13.28$ (3.00); APAs BWD: $A = 0.012$ (0.001), $B = -0.009$ (0.002), $k = 14.5$ (6.65); RPAs FWD: $A = 0.05$ (0.006), $B = 0.05$ (0.009), $k = 5.01$ (2.01); RPAs BWD: $A = 0.06$ (0.005), $B = 0.05$ (0.006), $k = 6.09$ (1.52); means (SE)]. Thus the magnitude of postural adaptation was not affected by stability condition.

In summary, both groups adapted their postural control similarly to the novel perturbation. Contrary to our expectation and to previous findings, postural adaptation was not reduced in the less stable (backward) direction.

Postural Muscle Coactivation

Subjects can also modulate the magnitude of RPAs via postural muscle coactivation (Figs. 6 and 8). To examine the role of postural coactivation in adaptation, we focused on changes in coactivation across the late baseline, early learning, and late learning phases (Fig. 8*B*; Table 2). In the FWD group, coactivation in the RF-BF, TA-PL, TA-LGas, TA-MGas, and TA-Sol muscle pairs significantly increased from late baseline to early learning and then significantly decreased from early learning to late learning. However, a coactivation strategy was not as evident in the BWD group. RF-BF coactivation slightly increased from late baseline to early learning and then slightly

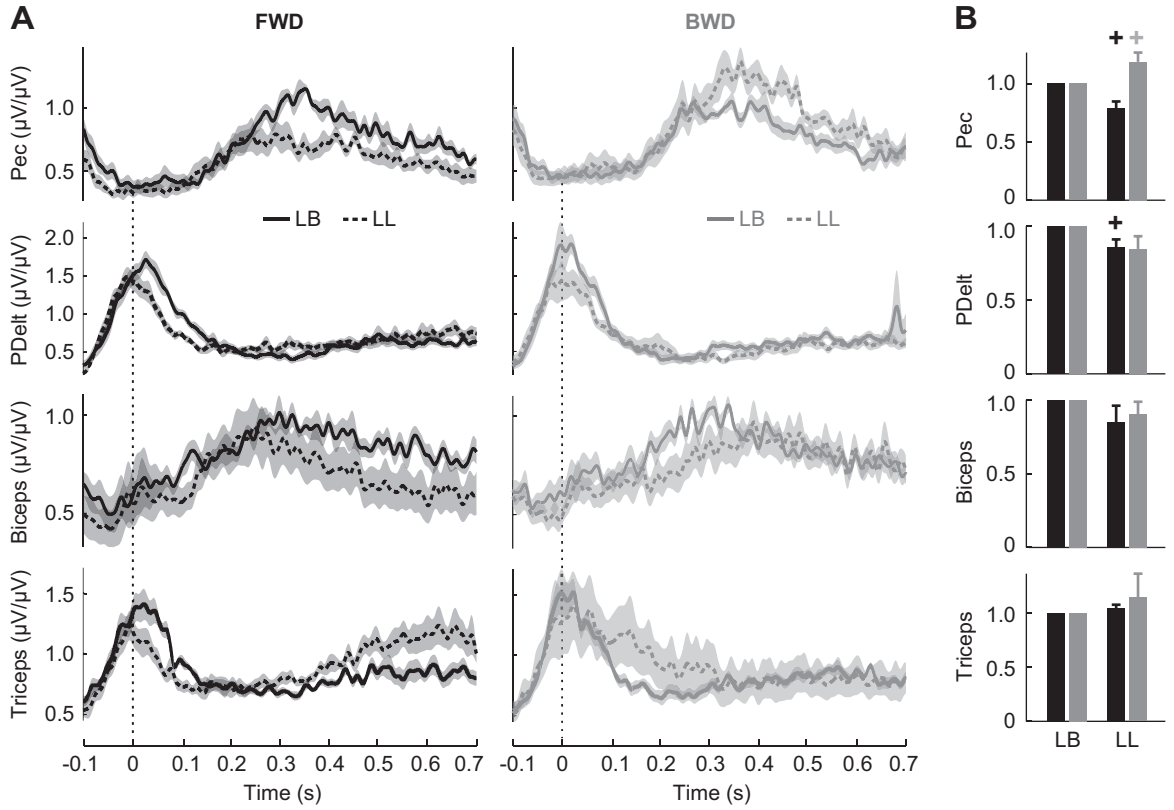

Fig. 4. Arm muscle activity. *A*: average time traces of muscle activity (mean normalized electromyographic activity) for the pectoralis major (Pec), posterior deltoid (PDelt), biceps brachii (Biceps), and long head of the triceps (Triceps), in the late baseline (LB) and late learning (LL) phases, for the forward (FWD; black) and backward perturbation (BWD; gray) groups. Line shading indicates SE across subjects. *Time 0* represents movement onset of the arm. *B*: group mean values (root-mean-square value of normalized EMG activity from movement onset to movement end) at LB and LL for FWD (black) vs. BWD group (gray), with error bars showing SE across subjects. $+P < 0.050$, statistically significant change within a group from LB to LL.

decreased from early learning to late learning, without reaching significance. No other postural muscle pairs showed a trend toward an increase in coactivation from late baseline to early learning. To further examine coactivation in the BWD group, we looked for changes within individual subjects. Five subjects showed a significant increase from late baseline to early learning in at least one muscle pair (one-tailed paired *t* tests, *P* values ≤ 0.022), and two of those subjects showed significant increases in all five postural muscle pairs, but otherwise these increases were found in different pairs across subjects. Five subjects showed no significant increases from late baseline to early learning in any postural muscle pair.

Table 3. *Planned comparisons: muscle activity*

	FWD Group		BWD Group	
PecMaj	**0.005**		**0.044**	
PostDelt	**0.028**		0.107	
Biceps	0.203		0.273	
Triceps	0.292		0.532	
	FWD group		BWD group	
	APA	RPA	APA	RPA
RF	0.112	0.137	0.105	**0.040**
BF	0.737	0.378	0.405	0.341
TA	0.201	**0.041**	0.133	0.139
PL	**0.016**	0.111	0.477	0.925
LGas	0.432	0.632	0.277	0.296
MGas	**0.007**	**0.031**	0.177	**0.024**
Sol	**0.002**	**0.012**	0.248	0.559

Data are *P* values for planned comparisons within each group [forward (FWD) or backward perturbation (BWD)] for muscle activity in late baseline vs. late learning. For postural muscles, results are for reactive postural adjustment (RPA) and anticipatory postural adjustment (APA). BF, biceps femoris; Biceps, biceps brachii; LGas, lateral gastrocnemius; MGas, medial gastrocnemius; PostDelt; posterior deltoid; PecMaj; pectoralis major; PL, peroneus longus; RF, rectus femoris; Sol, soleus; TA, tibialis anterior; Triceps, long head of the triceps. Significant results are in bold type.

In summary, postural muscle activity shows that both groups adapted their control to counteract the perturbation. However, the groups used differing coactivation strategies. In early learning, the FWD group increased their coactivation in several muscle pairs, as expected, but the BWD group did not significantly increase their coactivation in any muscle pair.

Individual Postural Muscle Activity

Because the BWD group did not modulate muscle coactivation, we sought to compare how the groups changed individual postural muscle activity to ultimately effect adaptation of the APAs measured. To do so, we focused on changes in anticipatory and reactive activity from late baseline to late learning (Fig. 9, *B* and *C*; Table 3). We expected that the FWD group would modulate muscle activity to generate a backward torque on the body, counteracting the forward torque applied by the robot. Indeed, the FWD group showed significant increases in anticipatory activity in the PL, MGas, and Sol muscles, which generate a plantarflexion torque. We also observed significant increases in reactive activity in the MGas and Sol and a

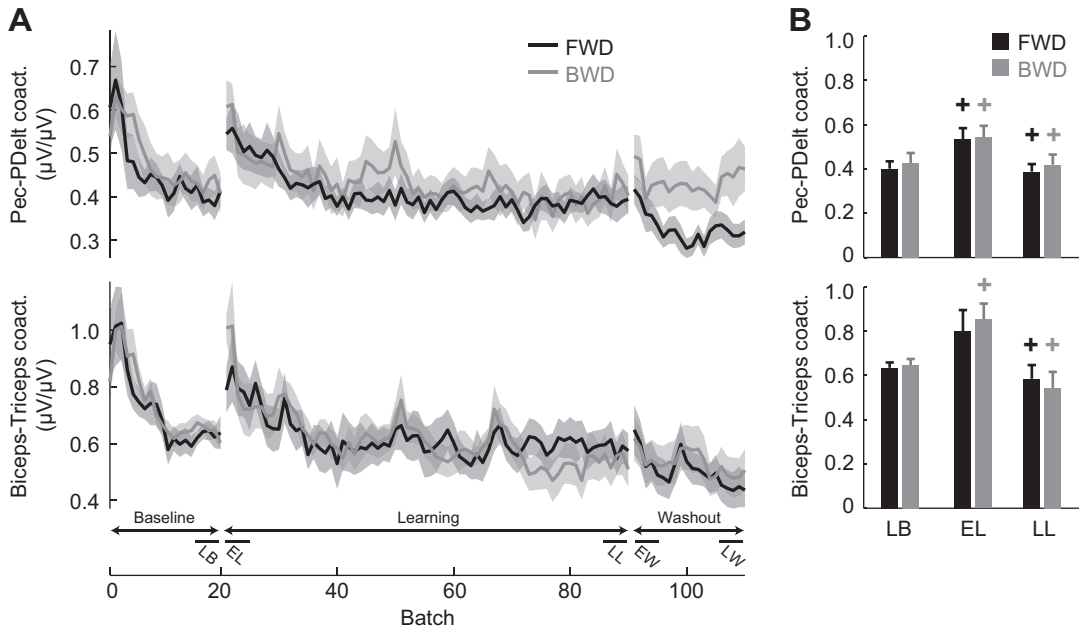

Fig. 5. Coactivation in arm muscle pairs. *A*: coactivation vs. batch (5 trials) for the indicated muscle pairs. In each plot, 2 traces show group means (solid lines) ± SE (shading) for the forward (FWD; black) and backward perturbation (BWD; gray) groups. *B*: group mean values at late baseline (LB), early learning (EL), and late learning (LL) for FWD (black) vs. BWD group (gray), with error bars showing SE. $+P < 0.050$, significant change within a group, from LB to EL or from EL to LL. Biceps, biceps brachii; coact., coactivation; PDelt; posterior deltoid; Pec; pectoralis major; Triceps, long head of the triceps.

significant decrease in reactive activity in the TA. Thus, by the end of the learning phase, the FWD group increased anticipatory activity in muscles contributing to a plantarflexion torque (backward torque on the body) and also increased activity in muscles contributing to the reactive plantarflexion correction, as well.

The BWD group showed a significant increase in reactive activity in the RF, a significant decrease in reactive activity in the MGas, and a weak trend toward an increase in anticipatory activity in the RF. These results suggest that the BWD group adapted their anticipatory muscle activity less, and instead relied on a more reactive strategy to compensate for the perturbation.

Furthermore, we wanted to determine the statistical relationship between changes in anticipatory postural muscle activity and APAs. To do so, we performed one-tailed linear regressions between the changes in anticipatory activity and APAs from late baseline to late learning (Fig. 10). Across all subjects, we found a significant positive relationship between the changes in APAs and anticipatory activity in the MGas ($P = 0.024$) and Sol ($P = 0.009$) such that increases in anticipatory activity tend to be associated with development of positive APAs (COP movement in the forward direction), and decreases in activity tend to be associated with development of negative APAs (COP movement in the backward direction). These findings are in agreement with our initial predictions about how ankle plantarflexor activity (including the MGas and Sol) and COP movements would change with adaptation to the force field.

Other Postural Movement Characteristics

We wanted to verify that COP movements executed during the experiment were within the limits of the functional BOS (measured during the COP game; Table 4). Functional BOS limits in the direction of the perturbation were significantly different between groups (forward limit for the FWD group, backward limit for the BWD group; $P = 0.007$ for normalized values, $P = 0.017$ for nonnormalized values). Despite the difference in functional BOS limits, maximum COP displacement magnitudes in the experiment were similar between groups ($P = 0.197$ for normalized values, $P = 0.160$ for nonnormalized values). Importantly, COP displacements in the experiment did not meet or exceed the limits of the functional BOS in any subject. Overall, these results confirm that COP movements developed in response to the force field were well within the limits of the functional BOS, and therefore well within stability limits, for both groups.

Thus far our analysis has focused on APAs that developed to anticipate the force field. These APAs were in the same direction as the field and perpendicular to the direction of hand reaching movements. 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, 2016). To confirm that tangential APAs were not affected by the perturbing forces and related adaptation in the perpendicular direction, we examined tangential APAs between phases and between groups. Tangential APAs were measured in the direction of reaching as the peak signed value of normalized COP displacement, similar to perpendicular APAs, but were 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; Pienciak-Siewert et al. 2014). Across all subjects, the magnitude of the tangential APA showed no significant differences between adjacent phases (all P values ≥ 0.122); and at all

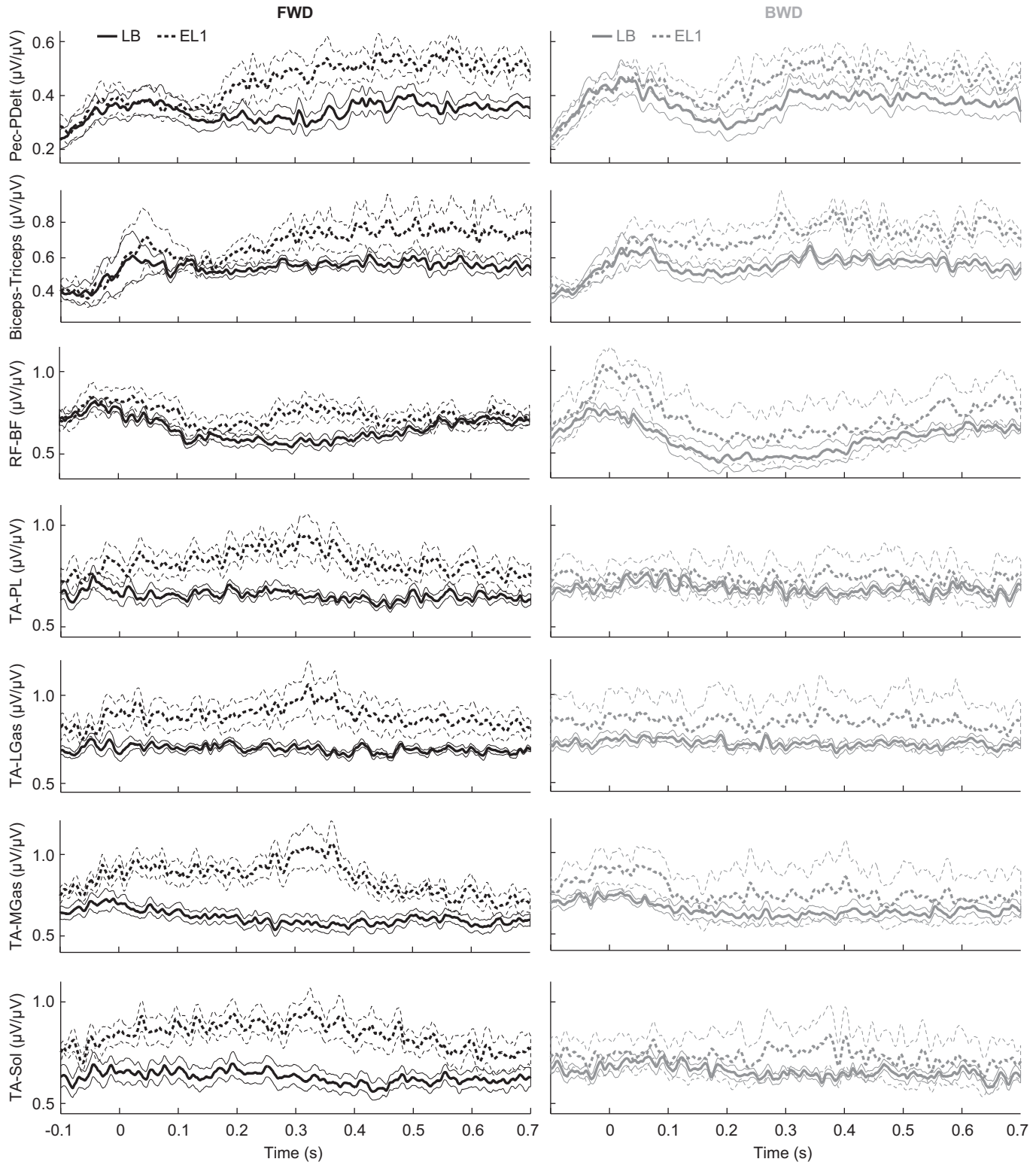

Fig. 6. Muscle coactivation time traces. Average time traces of coactivated muscle activity (magnitude of normalized electromyographic activity that is matched by 2 opposing muscles at each time point throughout the movement) for all arm and postural muscle pairs, in the late baseline (LB) and early learning (EL) phases, for the forward (FWD; black) and backward perturbation (BWD; gray) groups. Thick lines represent group mean values; thin lines indicate SE across subjects. *Time 0* represents movement onset of the arm. BF, biceps femoris; Biceps, biceps brachii; LGas, lateral gastrocnemius; MGas, medial gastrocnemius; PDelt; posterior deltoid; Pec; pectoralis major; PL, peroneus longus; RF, rectus femoris; Sol, soleus; TA, tibialis anterior; Triceps, long head of the triceps.

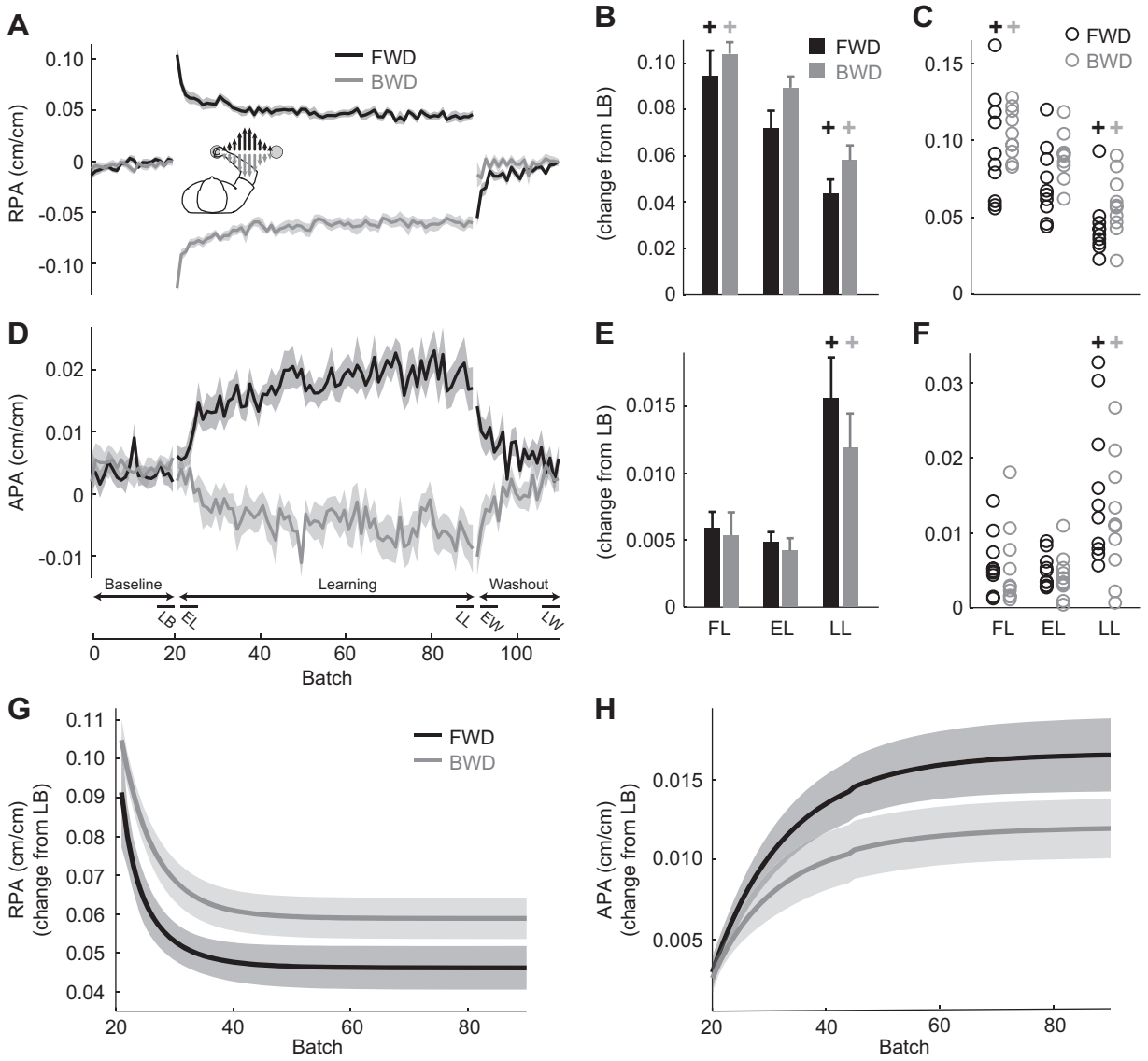

Fig. 7. Reactive postural adjustment (RPA) and anticipatory postural adjustment (APA) metrics. *A–C* and *D–F* show RPA and APA, respectively. *A* and *D* show each metric vs. batch (5 trials). In each plot, 2 traces show group means (solid lines) ± SE (shading) for the forward (FWD; black) and backward perturbation (BWD; gray) groups. Bar plots in *B* and *E* show the absolute magnitude of the change from late baseline (LB) to first learning (FL), early learning (EL), and late learning (LL), for FWD (black) vs. BWD group (gray), with error bars showing SE. Bar plots in *C* and *F* show the absolute magnitude of the change from LB for each subject. $+P < 0.050$, statistically significant change within group for RPA, from LB to FL or from FL to LL, and for APA, from LB to LL. *G* and *H* show exponential fits of RPA and APA data from the learning block. In each plot, 2 traces show bootstrapped mean data for FWD (black) and BWD (gray) across 1,000 bootstrap-resampled groups (solid lines are means, shading indicates ±SE).

phases, magnitudes did not significantly differ between groups (all P values ≥ 0.103).

We also examined the muscles involved in the tangential APA related to the reaching movement, we examined anticipatory postural muscle activity in the late baseline phase. As shown in Fig. 9*A*, the BF and MGas muscles showed anticipatory activity in this phase in both groups, corresponding to an initial leftward and slightly forward COP movement. This indicates a normal APA for rightward reaching with the right arm.

DISCUSSION

In this study we demonstrated that an early muscle coactivation strategy can be used in postural adaptation, similarly to how it is used in the adaptation of arm reaching movements. However, the emergence of a postural coactivation strategy was dependent on the direction of the perturbation. An early postural coactivation strategy was primarily observed during adaptation to forward perturbations. In contrast, and counter to our expectations, backward perturbations led to reduced modulation of postural muscle activity. This suggests that conditions of reduced postural stability and/or increased postural threat may lead to a more nuanced prioritization of postural control objectives, rather than a simple error-reduction or "stiffening" strategy. Yet, despite those differences in postural muscle adaptation, adaptation of arm reaching as well as muscle activation was similar across stability conditions. This finding highlights the flexibility of the relationship between arm movements and postural control: similar control of the

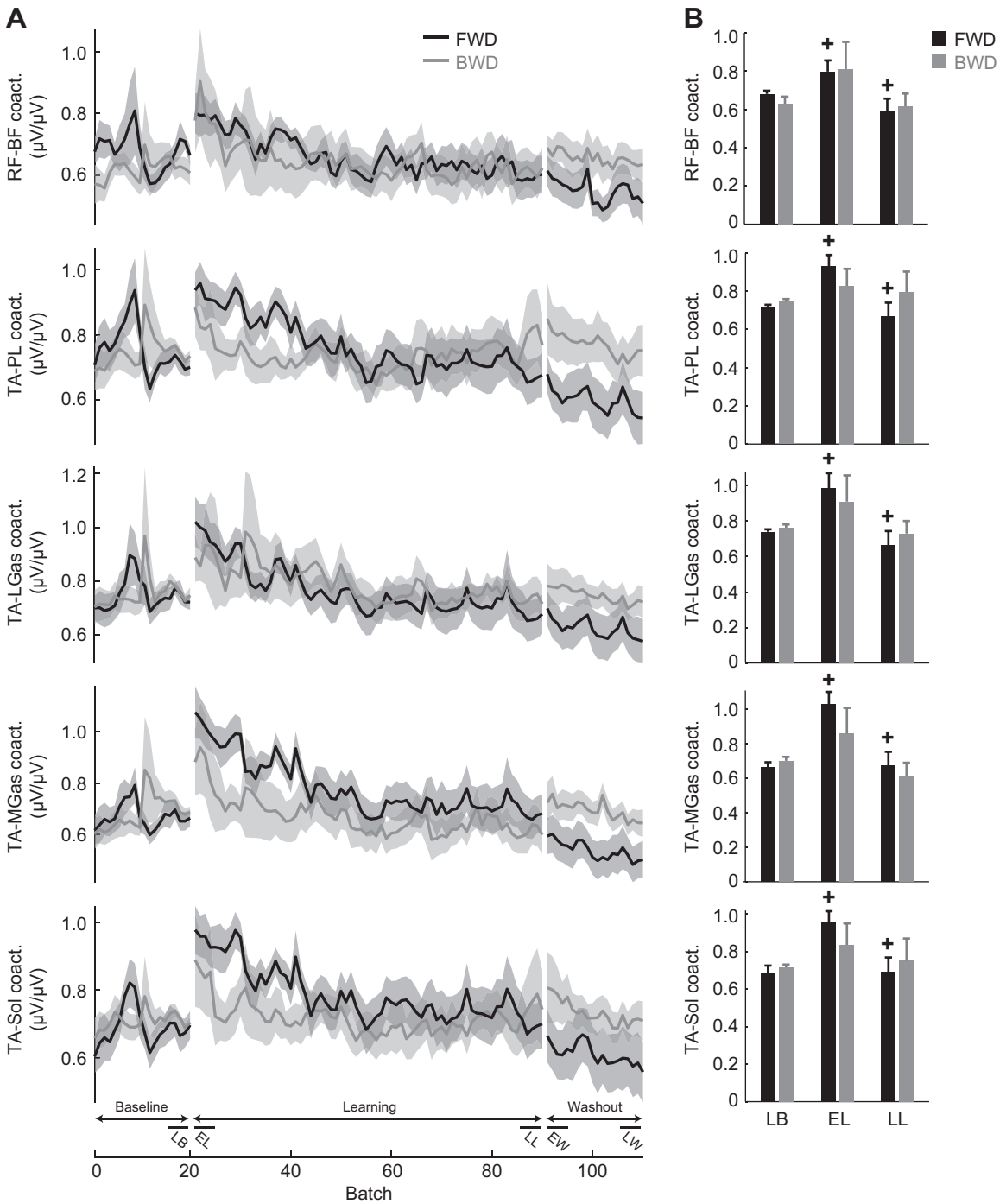

Fig. 8. Coactivation in postural muscle pairs. *A*: coactivation vs. batch (5 trials) for the indicated muscle pairs. In each plot, 2 traces show group means (solid lines) ± SE (shading) for the forward (FWD; black) and backward perturbation (BWD; gray) groups. *B*: group mean values at late baseline (LB), early learning (EL), and late learning (LL), for FWD (black) vs. BWD group (gray), with error bars showing SE. $+P < 0.050$, significant change within a group, from LB to EL or from EL to LL. BF, biceps femoris; coact., coactivation; LGas, lateral gastrocnemius; MGas, medial gastrocnemius; PL, peroneus longus; RF, rectus femoris; Sol, soleus; TA, tibialis anterior.

focal arm movement can be maintained despite different postural control strategies.

Early Coactivation Strategy

Our study demonstrates that during postural adaptation, subjects used a compensation strategy in which postural muscle coactivation is increased during early learning and is later decreased. This was strongly observed in the group that adapted to a forward perturbation. This early coactivation strategy is similar to what we, and other studies, have observed in arm adaptation (Darainy and Ostry 2008; Milner and Cloutier 1993; Thoroughman and Shadmehr 1999; van Emmerik 1992). A recent study by Huang and Ahmed (2014b) found that arm coactivation is also increased early in adaptation of arm reaching to a visuomotor rotation, where no perturbing forces are experienced. This suggests that this type of coactivation strategy is

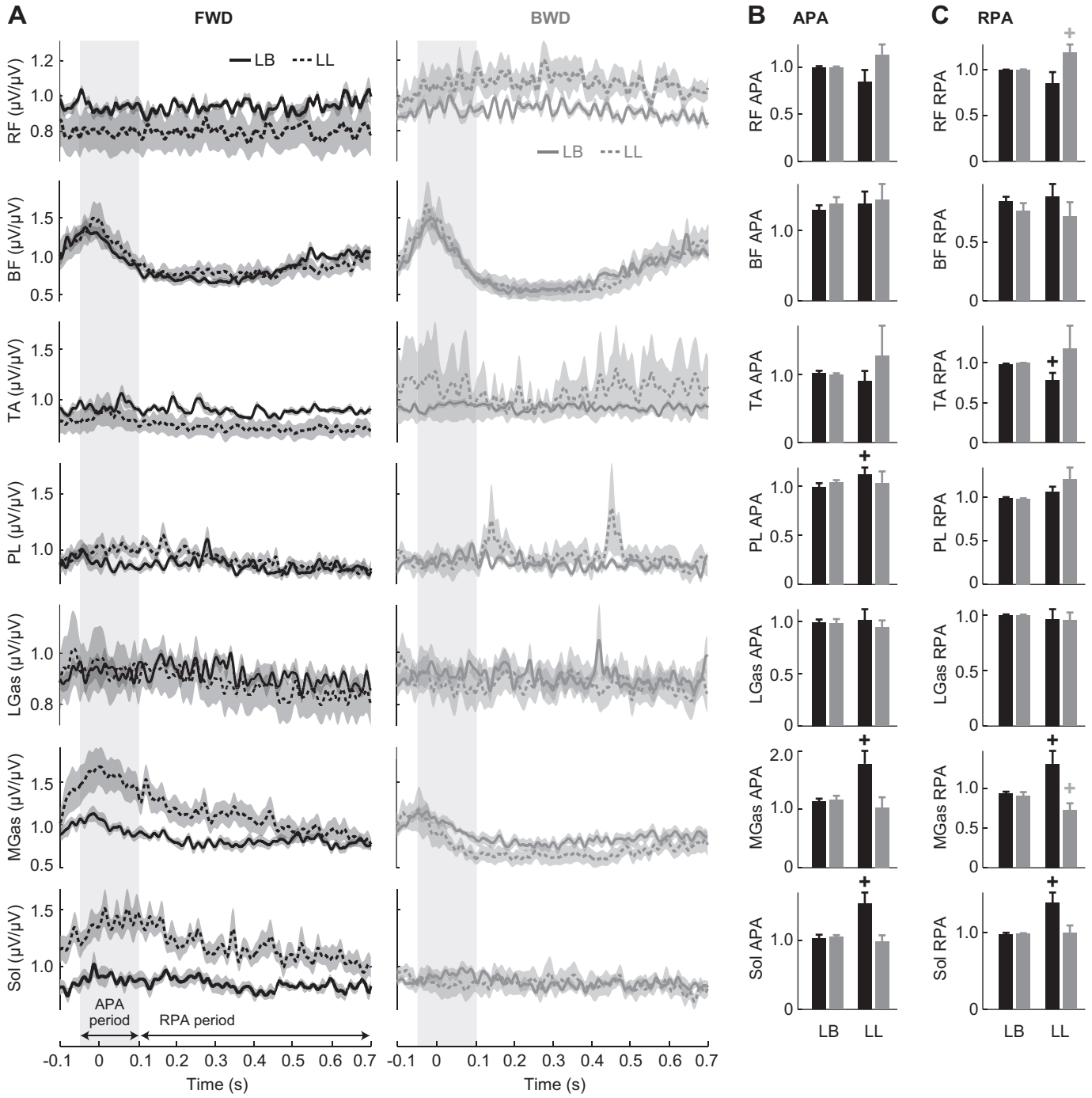

Fig. 9. Postural muscle activity. *A*: average time traces of muscle activity (mean normalized electromyographic activity, EMG) for the rectus femoris (RF), biceps femoris (BF), tibialis anterior (TA), peroneus longus (PL), lateral gastrocnemius (LGas), medial gastrocnemius (MGas), and soleus (Sol), in the late baseline (LB) and late learning (LL) phases, for the forward (FWD; black) and backward perturbation (BWD; gray) groups. Line shading indicates SE across subjects. *Time 0* represents movement onset of the arm. Shading and arrows indicate reactive (RPA) and anticipatory postural adjustment (APA) periods. *B* and *C*: group mean values for anticipatory and reactive muscle activity (root-mean-square value of normalized EMG activity over the APA and RPA periods, respectively) at LB and LL, for FWD (black) vs. BWD group (gray), with error bars showing SE across subjects. $+P < 0.050$, statistically significant change within a group from LB to LL.

generally engaged during adaptation in an attempt to reduce movement errors and is not only used in response to dynamic perturbations. Other studies have shown that in the absence of novel kinematics or dynamics, subjects can modulate their arm impedance, using coactivation to reduce their movement error, to meet specific arm reaching accuracy requirements (Gribble et al. 2003; Osu et al. 2004, 2009; Wong et al. 2009).

Results from previous studies of postural adaptation suggested that such a strategy was being used, but coactivation was not measured in those studies. Ahmed and Wolpert (2009) found that COP RPAs were reduced faster than COP APAs were developed, suggesting that subjects may have used a postural coactivation strategy to quickly reduce RPAs in early learning. Manista and Ahmed (2012) found that after subjects

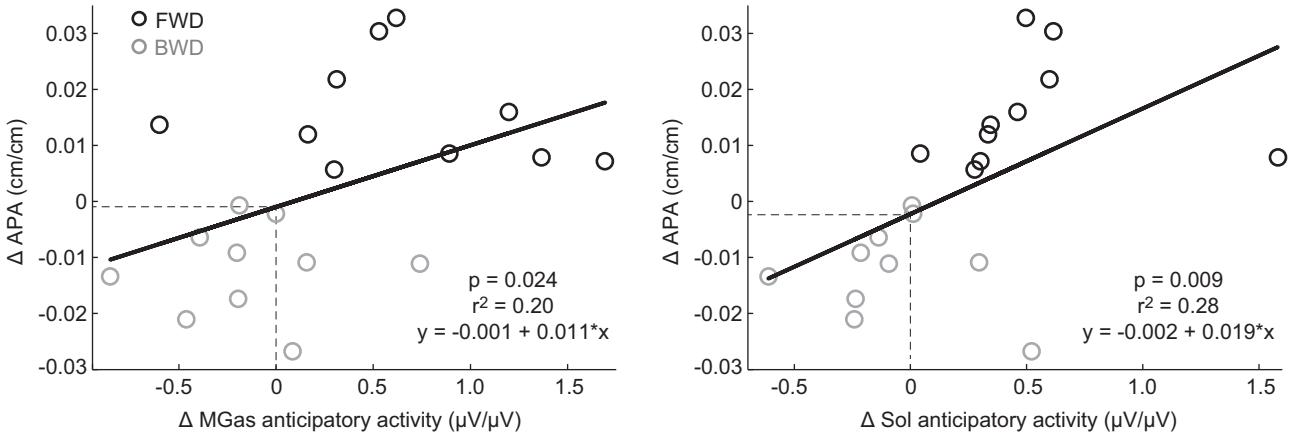

Fig. 10. Medial gastrocnemius (MGas) and soleus (Sol) anticipatory activity vs. anticipatory postural adjustment (APA). Each data point represents the change from late baseline to late learning for 1 subject experiencing either a forward (FWD; black) or backward (BWD; gray) perturbation. Dashed lines indicate *y*-axis intercept. Plots show significant positive correlations between these metrics across all subjects.

adapted to perturbations in multiple directions, APAs for a backward perturbation were significantly smaller than for a forward perturbation; however, there was no corresponding difference in RPAs, suggesting that subjects used smaller APAs and a more coactivated strategy to compensate for perturbations in the backward direction. However, neither of these studies measured muscle activity. Pienciak-Siewert et al. (2014) found that during adaptation to a lateral perturbation, anticipatory muscle activity was increased bilaterally in early learning in several left-right postural muscle pairs. This suggests that a coactivation strategy was being used, but muscle coactivation was not quantified in that experiment.

Whereas an early coactivation strategy was clear in the FWD perturbation group, the BWD perturbation group only exhibited weak increases in postural coactivation, despite the fact that five individual subjects showed a significant increase in at least one postural muscle pair and two subjects showed significant increases in all five postural muscle pairs. It is possible that other muscles (e.g., trunk muscles or right leg muscles) were involved in responding to the force perturbation in our experiment, especially in the BWD group, where we observed only a weak change in postural coactivation. However, we were only able to collect EMG data from a limited number of muscles, and these were not included. A study by Horak and Nashner (1986) found that when subjects experienced a forward or backward platform perturbation while standing, they initially responded using ankle and thigh muscles. That response shifted to the thigh and trunk muscles only when subjects experienced the same perturbations while standing on a shortened 9-cm-long platform, which may be regarded as a higher threat condition due to the abnormally small base of support. In our experiment, where subjects stood on a normal-sized platform, we expected both groups to respond to the postural perturbation using primarily ankle and thigh muscles. However, if we regard the BWD perturbation as more threatening, it is possible that a similar pattern of response may have developed in our experiment (i.e., ankle and thigh muscles in normal condition vs. hip and thigh muscles in higher threat condition), in which case the BWD group also may have relied on trunk muscles.

Additionally, muscles in the right leg may have been involved in the coactivation response. In rightward reaching movements during standing, similar to the movement in our experiment, Leonard et al. (2009) observed increases in generalized muscle activity in the right leg in the later portion of the movement. This suggests that muscles were coactivated to help stabilize the body at the end of the movement; the same muscles may have been involved in the early coactivation response in our experiment. However, we chose to collect EMG from the left leg rather than the right leg, to better capture individual muscle adaptations, for the reasons described in METHODS.

It also may be the case that the BWD group chose to use less coactivation in early learning compared with the FWD group. We address this possibility in *Different Strategies for Different Perturbations*.

Table 4. *BOS data*

	FWD Group	BWD Group
Foot length, cm	24.8 ± 1.5	25.5 ± 1.4
Stance width, cm	31.1 ± 2.6	33.6 ± 3.3
fBOS limit, cm	10.0 ± 2.0 (0.40 ± 0.08)	7.9 ± 1.6 (0.31 ± 0.06)
Maximum COP displacement, cm	3.8 ± 1.1 (0.15 ± 0.04)	4.5 ± 1.3 (0.18 ± 0.05)

Data are group mean values for foot length and stance width (distance between lateral edges of the feet), forward and backward limits of the functional base of support (fBOS), and maximum forward and backward center-of-pressure (COP) displacement magnitudes observed during the experiment. Values for fBOS limit and maximum COP displacement are in the forward direction ($+y$) for the forward perturbation (FWD) group and in the backward direction ($-y$) for the backward perturbation (BWD) group; values were measured from the "start" location as defined in METHODS. Normalized data (in parentheses) were normalized to foot length for each subject.

Similarities in Postural Adaptation

Throughout learning, the data indicated that the groups adapted their postural movements similarly, despite differences in adaptation of muscle activity. Overall, it appears that the BWD group adapted to use similar APAs and RPAs compared with the FWD group, contrary to our hypothesis.

A previous study suggested that a backward perturbation, associated with reduced postural BOS, can lead to reduced APAs. Manista and Ahmed (2012) used an experimental paradigm similar to ours, in which subjects adapted their arm reaching to a curl force field while standing; each subject

adapted in multiple reaching directions, with the direction of the perturbation corresponding to reach direction. At the end of learning, subjects showed less adaptation of APAs for backward perturbations than for forward perturbations, despite the fact that subjects had sufficient biomechanical capacity to adapt similarly in both directions. It is possible that the findings of Manista and Ahmed differed from ours because their subjects were required to adapt their movement control for varying reaching directions and perturbation directions, which could have resulted in greater uncertainty. In contrast, each of our subject groups adapted to a constant perturbation in a single direction, which allowed them to fine-tune their adapted control strategy for that specific direction and the associated stability limits. Similarly, in a study by Pienciak-Siewert et al. (2014), subjects adapted to an unchanging lateral perturbation while standing with either a wide or narrow stance width (i.e., wide or narrow BOS); in that case, they found no difference in adaptation of RPAs or APAs between stance widths, despite the difference in BOS size. It would be interesting for future studies to examine the role of movement repetition in the development of anticipatory control in novel stability conditions.

Muscle Activity Correlates of Postural Adaptation

Both groups (FWD and BWD) adapted their postural control by developing anticipatory COP movements (APAs) in the direction of the perturbation. We also observed changes in individual muscle activity that corresponded to adaptation of COP movements.

From late baseline to late learning, the FWD group increased ankle plantarflexion and decreased ankle dorsiflexion to generate a net backward torque on the COM to counteract the forward perturbation. Specifically, they increased anticipatory muscle activity in the left PL, MGas, and Sol; increased reactive activity in the left MGas and Sol; and decreased reactive activity in the left TA. These changes correspond to the development of a forward COP movement (Henry et al. 2001; Imagawa et al. 2013; Krishnamoorthy et al. 2004).

In the BWD group, we observed significant changes in reactive muscle activity but only a nonsignificant change in anticipatory activity. The fact that we did not observe significant changes in anticipatory activity may be explained by several things. First, in adaptation of COP movements from late baseline to late learning, the BWD group seemed to show a trend toward a smaller change in APAs and a greater change in RPAs, compared with the FWD group, although these differences were not significant. These trends suggest the possibility that the BWD group adapted a more reactive postural control strategy, whereas the FWD group adapted a more anticipatory strategy. Second, the muscle adaptation strategy in the BWD group was more complex than in the FWD group. In the FWD group, the COP movement associated with unperturbed reaching APAs included a slight forward component, and adaptation to the forward perturbation required only a magnification of this component. In the BWD group, however, adaptation to the backward perturbation required this component of COP movement to be reversed in direction, which likely involved a more complex change in muscle strategy. To counteract the backward perturbation, the BWD group increased hip flexion and decreased ankle plantarflexion to generate a net forward torque on the COM. Specifically, they increased reactive activity and slightly increased anticipatory activity in the left RF, and decreased reactive activity in the left MGas. The changes in RF activity correspond to the development of a backward COP movement (Saito et al. 2007). Decreased MGas activity, with no associated change in TA activity, should also be associated with backward COP movement. Finally, related to the second point, the BWD group may have adapted using other muscles that were not recorded in our experiment, such as trunk muscles (as discussed earlier in *Early Coactivation Strategy*).

Different Strategies for Different Perturbations

Despite similar APA adaptation, a greater reliance on a coactivation strategy may still be present in the BWD group if the perturbation was a greater threat to stability. However, in early learning, the BWD group did not show clear evidence of a postural coactivation strategy, whereas the FWD group showed very clear evidence of such a strategy. The BWD group also showed similar RPAs compared with the FWD group, contrary to other findings of restricted COP movements in reduced-BOS conditions (Kaminski and Simpkins 2001; Yiou et al. 2007), which indicates that the BWD group did not prioritize restricting their COP movements within the smaller backward BOS limits. Clearly, the differences in postural control between the FWD and BWD groups cannot be explained by the use of a simple "stiffening" strategy. Rather, our results suggest that the BWD group used a reactive strategy, relying more on adaptation of reactive muscle activity than anticipatory activity. In contrast, the FWD group used a more efficient predictive strategy, with more adaptation of anticipatory muscle activity.

We believe that the difference in postural coactivation strategy between groups may be explained by a postural control trade-off between stability and maneuverability. Previous studies have suggested the existence of a trade-off between stability (ability to reject a perturbation) and maneuverability (ability to quickly change direction in response to perturbations) in various forms of motor control, including locomotion and standing posture. In such a trade-off, stability can be increased but at the expense of maneuverability, and vice versa (Chen and Chou 2013; Hasan 2005; Huang and Ahmed 2011; Jindrich and Qiao 2009; Qiao and Jindrich 2012; Ting et al. 2009; Tirosh and Sparrow 2004). In the present study, an early coactivation strategy in early learning could cause such a trade-off; when coactivation is increased, stability ("stiffness;" used to reject perturbations) is increased, and thus movement errors are reduced, but consequently, maneuverability is also reduced. However, the BWD group already had less postural maneuverability when responding to the perturbation, compared with the FWD group, for reasons explained below. Thus it is possible that the BWD group chose to approach this trade-off differently. To respond to the backward perturbation, the BWD group used a net forward torque on the COM (e.g., ankle dorsiflexion) and backward COP movement; conversely, the FWD group responded to the forward perturbation using a net backward torque on the COM (ankle plantarflexion) and forward COP movement. Subjects were less maneuverable, or had less capacity to respond, when responding to the backward perturbation, for two reasons: *1*) maximum voluntary ankle

torques are weaker in dorsiflexion than in plantarflexion (Fugl-Meyer 1981; Fujimoto et al. 2013; Thelen et al. 1996); and 2) BOS limits are smaller in the backward than in the forward direction (Holbein-Jenny et al. 2007; King et al. 1994; Manista and Ahmed 2012). Therefore, because the BWD group was already at a postural maneuverability disadvantage compared with the FWD group, the BWD group may have chosen to employ less of a stabilizing coactivation strategy to minimize their loss of maneuverability in the trade-off. It might be interesting to test this idea further, by challenging stability more (larger perturbations or more extreme reduction of BOS size) or by challenging maneuverability (e.g., random changes in target position during movement).

As mentioned earlier, our results suggest that the BWD group used a more reactive strategy, and relied more on adaptation of reactive muscle activity than anticipatory activity, whereas the FWD group used a more anticipatory strategy, showing more adaptation of anticipatory muscle activity. A similar pattern of behavior was seen in a study by Gendre et al. (2016), in which subjects performed lateral leg raises while standing on an elevated platform. One group stood at the edge of the platform such that their anticipatory COP shift was directed toward the edge, and later in the movement the COP shifted to the opposite side away from the edge; another group stood at the opposite edge of the platform such that their anticipatory COP shift was directed away from the edge, and later the COP shifted toward the edge. In the former group, the anticipatory COP movement toward the edge was slightly reduced in amplitude but was prolonged later into the overall movement duration. However, in the late-movement COP shift toward the edge, there was no difference between groups. There was also no difference in overall performance of the focal leg raise task. Clearly, the postural threat of moving toward the raised platform edge only had an effect on anticipatory COP movement, but not on later COP movement. In our study, it is possible that the reduced BOS in the backward direction was sufficiently threatening to induce a shift away from anticipatory activity toward more reactive activity, but not sufficient to induce a significant change in overall postural movement. This possibility could be investigated in the future by adding a higher threat and/or lower stability condition, such as standing on an elevated platform, experiencing larger perturbations, or standing on a BOS-restricting balance board.

Clinical Implications

Based on our results, we posited that the postural threat induced by the smaller BOS in the backward direction was insufficient to cause a significant change in postural movement behavior. However, this might change in an elderly population, because older adults alter their postural behavior more than young adults in response to postural threat (Brown et al. 2002; Gage et al. 2003; Young and Williams 2015).

We also observed differences in postural adaptation strategies related to differences in perturbation direction. In the BWD direction, subjects modulated their anticipatory muscle activity and degree of coactivation to a lesser extent. Those differences may be explained by the presence of a trade-off between stability and maneuverability, where the FWD group chose a more coactivated and more stable strategy because they were initially more maneuverable than the BWD group; in contrast, the BWD group chose a less coactivated and less stable strategy to minimize their loss of maneuverability. This highlights an area for concern in populations who exhibit reduced postural stability, reduced mobility, and/or reduced ability to recover from perturbations, such as older adults (Binda et al. 2003; Campbell et al. 1989; Chen and Chou 2013; Fujimoto et al. 2013; Graham et al. 2015; Holbein-Jenny et al. 2007; Honarvar and Nakashima 2014; Hurt and Grabiner 2015; Kuo and Zajac 1993; Maki et al. 1994; Melzer et al. 2004; Overstall et al. 1977; Robinovitch et al. 2002; Rogers et al. 2001; Singer et al. 2015) and patients with Parkinson's (Buckley et al. 2008; Hass et al. 2005; Horak et al. 1996, 2005; Jessop et al. 2006; Kim et al. 2009; Mancini et al. 2008; Martin et al. 2002). In individuals with such postural deficits, any trade-off between stability and maneuverability strategies is especially critical because stability, maneuverability, or both might be low compared with those in healthy young adults. For example, in the elderly, these deficits may be a contributing factor to falls (Alexander et al. 2001; Campbell et al. 1989; Fiatarone et al. 1990; Fujimoto et al. 2013; Graham et al. 2015; Honarvar and Nakashima 2014; Kuo and Zajac 1993; Robinovitch et al. 2002; Sample et al. 2016; Taaffe et al. 1999; Vincent et al. 2002).

Of particular interest, and complexity, is the issue of coactivation. Older adults often exhibit higher levels of muscle coactivation than young adults; the increased coactivation may be employed to reduce movement variability and achieve greater accuracy (Darling et al. 1989; Hortobágyi and DeVita 2006; Seidler-Dobrin et al. 1998), but it also may be caused involuntarily by age-related alterations to the central nervous system control of coordination between antagonist muscle pairs (Hortobágyi and DeVita 2006). In either case, it seems likely that in a case such as our experiment, older adults would coactivate more than young adults, despite the consequent loss of maneuverability. This might be accompanied by slower and/or reduced adaptation. A study by Huang and Ahmed (2014a) found that during adaptation to novel arm reaching dynamics, older adults showed greater coactivation and reduced adaptation compared with younger adults. Furthermore, they found that the amount of adaptation was negatively correlated with coactivation, suggesting that high levels of coactivation may be detrimental to motor adaptation. Similarly, Darainy et al. (2009) found that greater inherent "stiffness" (end point impedance at the hand, dependent on arm reaching direction) was correlated with reduced dynamic adaptation. However, in another dynamic adaptation study by Heald et al. (2018), subjects were pretrained to coactivate their muscles more or less; their results showed that greater coactivation levels were correlated with increased adaptation. Thus, in older adults and other clinical populations that may exhibit changes in muscle coactivation and motor adaptation, there may not be a simple causative relationship between those behaviors. To understand those behavioral interactions in such populations, further research is required.

Conclusions

The results of this study confirm that an early muscle coactivation strategy can be used in postural adaptation, similarly to how it is used in the adaptation of arm reaching movements. However, use of a postural coactivation strategy

was dependent on perturbation direction, being largely specific to forward perturbations. Backward perturbations led to less modulation of postural muscle activity and did not lead to smaller postural movements, suggesting that adaptation strategies for this reduced-stability condition were not ruled by the impetus to restrict postural movements. Finally, despite the differences in postural adaptation, arm adaptation was similar between groups by the end of learning. This demonstrates that differences in postural conditions and associated differences in postural control do not necessarily affect control of a concurrent arm movement.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

A.P.-S. and A.A.A. conceived and designed 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. 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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