SENSORIMOTOR ADAPTATION IN WHOLE-BODY POSTURAL CONTROL

by

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Thesis directed by Associate Professor Alaa A. Ahmed

Abstract

The ability to maintain stable, upright standing is a critical component of our daily activities. This ability requires that we generate appropriate postural control when making voluntary movements and when responding to perturbations, and appropriately adapt that control to compensate for changing conditions. Despite this, adaptation of whole-body postural control is not well understood.

This dissertation investigates the control strategies involved in the adaptation of whole-body postural control and how well this learning transfers to different environments. We used an experimental paradigm in which subjects made reaching movements while standing and holding the handle of a force-generating robotic arm that could apply novel perturbations to the arm and standing posture concurrently.

First, we sought to identify the signal driving postural adaptation. We examined adaptation in response to varying movement error sizes. Adaptation scaled near-proportionally with error, but was insensitive to very small errors. In a follow-up study, we investigated the effect of small yet consistent errors. Despite the small errors, subjects did adapt, indicating that both error size and consistency play a role in driving adaptation.

Next, we investigated how control strategies are affected by postural stability conditions. Results showed that stability conditions significantly affect how adaptation strategies are used; furthermore, transfer of adapted control between different conditions is affected by the condition in which the task is initially learned.

Lastly, we tested whether postural control can be adapted and transferred independently of arm control. When subjects failed to transfer their adapted arm control between arms, they also failed to transfer their postural control, even though the postural perturbation had not changed. Thus, arm control over-wrote the learned postural control, necessitating re-learning of a previously learned strategy. This suggests that postural control is dependent on information about the arm movement dynamics in this combined task.

Generally, this work demonstrates that postural adaptation manifests many characteristics of general motor adaptation. It also highlights how heavily postural control is influenced by and coordinated with concurrent arm movements. However, postural stability conditions play a significant role in determining how standing posture is controlled, adapted, and transferred between different contexts.

DEDICATION

To my family and my wonderful husband, who have been endlessly supportive of my endeavors.

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CHAPTER 1

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 when responding to perturbations. To generate the appropriate control, we use our knowledge of the mechanical and dynamic properties of our bodies and the environment, including objects with which we interact. For example, when you lift a heavy box, the weight of the box will tip you off balance unless you alter your postural control to compensate for it. Your brain needs to know how much the box weighs in order to compensate smoothly and effectively.

As our bodies and the environment change over time, our brains must learn about the changes and then adapt our movement control patterns accordingly to keep up with the changing conditions. Despite this, how we adapt our standing postural control in response to novel perturbations is not well-understood; one example of adapting to a novel perturbation might be a first-time parent, learning to go about their daily tasks while carrying a baby. Adaptation has been well-studied in arm reaching movements, but there are fundamental differences between arm-reaching movements and whole-body postural control. Most notably, upright standing is inherently unstable. Standing postural control is subject to stability limits; if we move beyond those limits, we may be unable to avoid a fall. Maintaining postural stability 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: what drives adaptation, the process of how control patterns are adapted, how the brain remembers the learned information, and how the brain uses that learned information in different contexts.

This chapter introduces concepts and previous work that serve as background to my dissertation. I provide a general description of motor adaptation, then transition into a discussion of whole-body postural control and previous work examining adaptation of postural control.

1.1 Movement adaptation

1.1.1 Feedforward control and internal models

Voluntary movements are subject to two general types of control: feedback and feedforward. Feedback control is generated in response to sensory feedback information about the current state of the body and environment. However, there is a physiological time delay between when the sensory stimulus is provoked and when the resulting feedback control response is enacted; this delay makes it impossible to make fast, accurate movements using only feedback control. In contrast, feedforward control is generated in a predictive manner, wherein a control signal is generated based on some prediction of what the state of the body and environment will be in the near future.

To generate accurate feedforward control, the brain must be able to relate motor signals and sensory information to actual physical states. This is accomplished using so-called "internal models," or neural representations of the dynamics of the body and environment (Kawato 1999; Lackner and Dizio 1994; Miall and Wolpert 1996; Shadmehr and Mussa-Ivaldi 1994; Thoroughman and Shadmehr 2000; Wolpert et al. 1998; Wolpert and Ghahramani 2004). The brain can use these internal models to plan which motor commands will produce a desired movement outcome.

For example, consider a game of catch. The ball is moving fast enough that it would be impossible to catch if you used only feedback control. If the brain receives sensory feedback about the ball's position at a certain time and then generates a feedback control signal based on that information, the ball will have moved to another position by the time your body responds to that control signal. However, the brain can predict where the ball will be in the future, using its internal model of the ball's movement. In another example, if someone pushes you unexpectedly, you may lose your balance because you can only respond to the push with feedback control after the push has happened. If you expect the push, however, you can use feedforward control to compensate for the push before it happens.

1.1.2 What is adaptation?

In general, we use the term "adaptation" to refer to any change in feedforward control strategy for a specific movement which compensates for a change in task dynamics such that movement kinematics return to their prior unperturbed performance. More specifically, motor adaptation is defined as a trial-to-trial modification of movement based on error feedback. In addition, the following three criteria are met: the movement retains its identity as being a specific

action (e.g. arm reaching), but one or more parameters are changed (e.g. direction or force); the modification occurs with repetition of the movement and is gradual and continuous; and once adapted, subjects cannot retrieve the prior behavior, but must de-adapt the movement with practice in the same manner back to the prior state (Bastian 2008; Martin et al. 1996). This is distinct from motor *learning*, which is a more inclusive term and also includes learning a new skill or movement pattern and/or building a new internal model, rather than modifying an existing movement pattern or internal model (Bastian 2008). Thus, adaptation provides a flexibility of 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).

To revisit the previous example, you may have learned the skill of catching a baseball and have a corresponding internal model of a baseball. If, one day, you find yourself playing catch with an altered baseball that has a much different mass than you expect, you will have to adapt in order to account for the new mass. With practice, you will update your internal model of the ball and adapt your catching movements.

1.1.3 What drives adaptation?

Classical theories of motor control hypothesize that adaptation is driven by sensorimotor error (Jordan and Rumelhart 1992; Jordan and Wolpert 1999; Kawato et al. 1987; Wolpert and Ghahramani 2004). This is upheld by many previously published findings. Several studies of arm reaching have shown that incremental adaptation between subsequent trials is scaled with the error magnitude and/or perturbation magnitude experienced in the previous trial (Fine and Thoroughman 2007; Franklin et al. 2003b; Herzfeld et al. 2014; Marko et al. 2012; Osu et al.

2003; Scheidt et al. 2001; Shadmehr and Mussa-Ivaldi 1994; Thoroughman and Shadmehr 2000; Trent and Ahmed 2013; Wei and Kording 2010). One study of eye saccades found that trial-to-trial adaptation increases with visual error magnitude (Robinson et al. 2003).

Subjects can adapt using either visual or proprioceptive error feedback, or both. When subjects receive both types of feedback, adaptation to visual and proprioceptive errors seems be at least partially independent (Bock and Thomas 2011; Krakauer et al. 1999; Pipereit et al. 2006). In the absence of visual feedback (e.g. vision of the arm is blocked), subjects can adapt to dynamic perturbations using proprioceptive feedback (DiZio and Lackner 1995, 2000; Franklin et al. 2007b; Krakauer et al. 1999; Scheidt et al. 2005; Tong et al. 2002). Vice versa, in the absence of proprioceptive feedback (e.g. in subjects with deafferentation, or loss of afferent input from peripheral nerves to the CNS), subjects can compensate using visual feedback to adapt to visuomotor and dynamic perturbations (Bernier et al. 2006; Ingram et al. 2000; Sarlegna et al. 2010).

1.1.4 Experimental evidence of adaptation

Motor adaptation is commonly studied in arm reaching movements. In the performance of reaching movements, the internal model predicts the forces that act on the arm during the movement. When the dynamics of the movement are altered, the internal model must be adapted to reflect the new dynamics.

Many studies involving target-directed reaching movements have demonstrated the adaptation that occurs when the hand encounters novel dynamics in the environment; for example, subjects reach while holding the handle of a robotic manipulandum which can generate perturbing forces (Figure 1.1). In normal, unperturbed reaching movements, the hand moves

along a smooth, straight-line trajectory to the target, with an associated bell-shaped velocity profile (Flash and Hogan 1985; Morasso 1981; Soechting and Lacquaniti 1981). When subjects encounter the novel dynamics, they experience movement errors due to the perturbing forces; then, with practice, they gradually adapt their control such that movement error is reduced and movement trajectories approach the original straight-line trajectory (Franklin et al. 2003a, 2003b; Lackner and Dizio 1994; Osu et al. 2003; Shadmehr and Mussa-Ivaldi 1994).

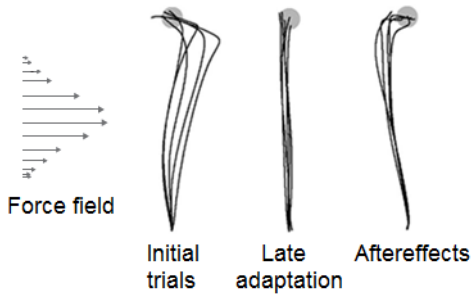

Figure 1.1. Movement trajectories during adaptation. Sample hand trajectories during adaptation to a novel dynamic force field: initial exposure trials, late adaptation trials, and initial aftereffect trials.

Evidence suggests that this adaptation is accomplished by gradually adapting the internal model of the environment; subjects use the adapted model to predict and compensate for the perturbing forces by predictively generating an appropriate time-varying force or torque profile to specifically counter the perturbing forces (Flash and Gurevich 1991; Franklin et al. 2003a, 2003b; Kawato 1999; Ruitenbeek 1984; Shadmehr and Mussa-Ivaldi 1994; Scheidt et al. 2000, 2001). When the novel dynamics are removed following adaptation, subjects make large movement errors, termed "aftereffects," which correlate to the perturbing external forces (Darainy and Ostry 2008; Lackner and Dizio 1994; Osu et al. 2003; Shadmehr and Mussa-Ivaldi

1994). The aftereffects gradually decrease as subjects de-adapt with further practice (Darainy and Ostry 2008; Shadmehr and Mussa-Ivaldi 1994).

Other studies have investigated adaptation of reaching movements to a visuomotor rotation, in which visual feedback of the movement is rotated relative to the actual movement direction (cursor feedback is rotated or subjects wear prism glasses). Subjects initially make movements straight toward the target, but miss the target due to the rotation; with practice, they make movements in the opposite direction of the rotation and are able to reach the target (Buch et al. 2003; Criscimagna-Hemminger et al. 2010; Fernandez-Ruiz et al. 2000; Krakauer et al. 1999; Martin et al. 1996). As above, aftereffects are observed when the perturbation is removed, and subjects gradually de-adapt with further practice. This type of adaptation also seems to involve internal model adaptation (Bock and Schneider 2002; Krakauer 2009; Krakauer et al. 1999).

Adaptation studies commonly also investigate transfer of adapted control from the initial training context to another context: between workspaces (e.g. from a forward reaching movement to a rightward reach), between environments (from reaching while holding the handle of a robot arm to reaching in free space), between limbs (e.g. from right arm to left arm), etc. Examining transfer can provide insight into the representations and mechanisms underlying adaptive behavior. For example, if adapted control is transferred between workspaces, this suggests that subjects adapted their internal model of the general reaching dynamics, and adaptation did not consist merely of a specific association between limb states and forces experienced during initial adaptation (Shadmehr and Mussa-Ivaldi 1994). Transfer from the initial training environment to another environment, especially in the case of an environmentally-driven perturbation (such as a force field generated by a robot arm), can give us information

about the extent to which the brain associated the adapted control pattern with the training environment/device or with the body (Cothros et al. 2006; Kluzik et al. 2008; Torres-Oviedo and Bastian 2012; Reisman et al. 2009). Transfer is especially important in rehabilitation, where it is desirable for adaptation that is acquired in a training context to be generalized beyond that context.

1.1.5 Control strategies in adaptation

Two separate control strategies have been identified in adaptation to novel dynamics: dynamic control (related to internal model adaptation) and impedance control (Osu et al. 2002, 2003; Takahashi et al. 2001). In the dynamic control strategy, which involves the internal model for feedforward control of movement, muscles are activated so as to generate specific torques about the joints and/or a specific net force to counter the novel dynamics. The impedance control strategy involves coactivation of opposing agonist-antagonist muscles; when the coactivated muscles exert equal and opposite torques on a joint, no net torque and thus no movement is produced, but the mechanical impedance of the joint is altered (Darainy et al. 2004; Gomi and Osu 1998; Hogan 1980).

Mechanical impedance is defined as the ratio of force to velocity (Doebelin 1998). In motor control, the term "impedance" is used to describe the forces generated by the limbs or body in automatic response to an imposed motion, before directed voluntary intervention occurs (Hogan 1985; Winters et al. 1988). Impedance is characterized by the stiffness, viscosity, and inertia of the system (e.g., the human arm), and is therefore affected by many factors; however, Milner et al. (1995) showed that subjects can use muscle coactivation to voluntarily modulate a wide range of joint impedances in the arm without changing the net joint torque. In normal

unperturbed movements, coactivation can help to resist the effects of inherent variability due to signal-dependent noise (Osu et al. 2004; Winters et al. 1988); studies have shown that subjects can modulate their arm impedance, using coactivation, to reduce their trajectory and endpoint variability in order to meet specific accuracy requirements (Gribble et al. 2003; Osu et al. 2004).

Many studies have investigated how subjects adapt with repeated exposure to novel but predictable dynamics. Franklin et al. (2003a, 2003b) showed that after adaptation is complete, arm impedance has been modified in a way that correlates with the changes in net joint torque that are required to counteract the external forces. It has also been shown that early in the adaptation process, impedance is increased due to an increase in muscle coactivation levels; however, those coactivation levels decrease as adaptation progresses (Darainy and Ostry 2008; Milner and Cloutier 1993; Thoroughman and Shadmehr 1999; Van Emmerik 1992). This initial increase in impedance is thought to be an important part of the adaptation process in that it provides stability while the internal model is not yet adapted to the novel dynamics; as the internal model is adapted and force control is modified accordingly, impedance can then be decreased without compromising stability or performance (Franklin et al. 2003b; Hinder and Milner 2007; Katayama et al. 1998; Milner and Franklin 2005; Osu et al. 2002). A recent study by Huang and Ahmed (2014b) found that coactivation is also increased early in adaptation to a visuomotor rotation, suggesting that this type of coactivating strategy is generally engaged in an attempt to reduce movement errors, and is used not only in response to dynamic perturbations.

During adaptation to unstable or unpredictable dynamics, coactivation levels and impedance are modified to counteract the instability (Akazawa et al. 1983; De Serres & Milner 1991; Milner 2002; Milner & Cloutier 1993, 1998; Takahashi et al. 2001). Several studies have demonstrated how subjects learn to stabilize unstable dynamics by selectively modifying their

arm impedance; after subjects have adapted, muscle coactivation levels have been modified in such a way that impedance is increased along the axis of instability, proportional to the strength of the perturbing forces, with no significant change in other directions (Burdet et al. 2001; Franklin et al. 2003a, 2003b, 2004, 2007); furthermore, forces and joint torques at the end of adaptation are similar to those made during unperturbed movements prior to encountering the novel dynamics (Burdet et al. 2001; Franklin et al. 2003b). These results indicate that adaptation to unstable dynamics is achieved with impedance control, independent of dynamic control (Burdet et al. 2001; Franklin et al. 2003a, 2003b, 2004). Osu et al. (2003) investigated learning of stable perturbing forces in an unstable field; they demonstrated that subjects adapted to the stable forces by using the dynamic control strategy and adapting their internal model; however, at the same time, subjects also used impedance control to compensate for the field instability. This indicates that dynamic control and impedance control are indeed two separate mechanisms but can function in parallel.

1.1.6 Clinical relevance

Adaptation studies can help to elucidate the neural mechanisms involved in feedforward control, and can also offer insight into specific mechanisms of motor impairments in clinical populations. For example, evidence from adaptation studies has suggested that the cerebellum is important in the formation and adaptation of internal models. Subjects with loss of cerebellar function show impaired adaptation of both arm reaching movements and locomotion to dynamic and visuomotor perturbations (Maschke et al. 2004; Morton and Bastian 2004; Tseng et al. 2007). On a trial-to-trial basis, they fail to adapt their feedforward control in response to movement errors (Smith and Shadmehr 2005). An impaired ability to form and adapt internal

models would also explain why subjects with cerebellar ataxia demonstrate a reduced ability to compensate for complex mechanical properties of the arm (Bastian et al. 1996, 2000; Topka et al. 1998).

Other studies have demonstrated how adaptation-based training paradigms can lead to improved motor performance in various clinical populations. For example, in patients with hemi-neglect due to stroke or other causes, adaptation to a visuomotor perturbation can lead to motor improvements. When subjects with left-side hemi-neglect adapted to a rightward visuomotor perturbation, they displayed aftereffects toward the neglected left side, and this led to improvements in their neuropsychological symptoms of neglect; these improvements lasted for several hours up to several days (Pisella et al. 2002; Rode et al. 2003; Rossetti et al. 1998). Other studies demonstrated that when stroke patients with hemiparesis adapted to dynamic perturbations that were customized to amplify their baseline patterns of movement error, in arm reaching movements and in locomotion, adaptation led to improvements in their movement error patterns compared to baseline (Patton et al 2006; Reisman et al. 2007, 2009). In locomotion, patients' improvements in locomotor symmetry transferred from the treadmill (training environment) to overground walking (normal environment) to a greater extent than in healthy control subjects (Reisman et al. 2009). In order to design effective rehabilitation programs, it is important to understand the mechanisms that drive adaptation and transfer, and how they are affected by varying conditions.

1.2 Control of standing posture

1.2.1 Physics of standing posture

To successfully and safely navigate our environment, humans must be able to maintain a standing posture without falling, while interacting with and moving through the environment. This is a complex control problem, primarily because human standing posture is inherently unstable according to classical definitions. Standing posture is typically modeled as an inverted pendulum (Barin 1989; Camana et al. 1977; Hemami and Golliday 1977; Nashner 1971, 1972; Nashner and McCollum 1985); in a gravitational field, this system is inherently unstable due to the fact that any internal or external perturbation away from equilibrium will result in the development of forces and moments that act to push the system farther away from equilibrium. This condition, along with biomechanical constraints such as muscle strength and foot placement, combine to establish stability limits for standing posture: within stability limits, the body can generate sufficient torque about its joints to generate a desired movement or to recover from a perturbation and return to equilibrium. If the body moves beyond its stability limits, it will be unable to recover and avoid a fall without taking a recovery step or some other corrective action. Thus, larger stability limits constitute a greater capacity to generate large movements or to recover from large perturbations; smaller stability limits constitute a lesser capacity.

A common measure of postural control is center of pressure (COP), defined as the application point of the ground reaction force vector (Murray et al. 1967). This is used because it provides a measure of the net torque at the ankle (an active control variable) and thus represents the active control exerted on the COM (a controlled outcome variable) (Morasso et al. 2014; Winter et al. 1990); it is also easily measured in experiments. Previous studies have shown that

COP displacement (horizontal difference between COP and COM locations) drives horizontal acceleration of the COM for quiet standing (Winter et al. 1998), gait initiation and termination (Jian et al. 1993), and balance recovery after a perturbation (Rietdyk et al. 1999).

1.2.2 Postural stability limits

Stability limits for standing posture were classically defined by the requirement that the center of gravity (planar projection of the center of mass, or COM) remains within the base of support (BOS), typically defined as the area beneath the feet (Dietz et al. 1989; Gollhofer et al. 1989; Horstmann and Dietz 1990; MacKinnon and Winter 1993) (Figure 1.2). However, it was observed that the COM is permitted to move outside the positional limits of the BOS (Murray et al. 1967). Therefore, more recent work has suggested a definition of stability limits which requires that the center of pressure (COP) remains within the BOS. Combined experimental and modeling work has shown that under normal conditions, COP movement is indeed limited to within the BOS; however, in conditions such as reduced ankle strength or a low-friction support surface, stability limits are reduced (Patton et al. 1999; Robinovitch et al. 2002).

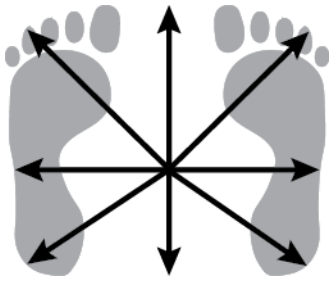

Figure 1.2. Postural base of support. During normal unsupported standing, the base of support (BOS) is defined as the area beneath the feet.

Many studies have shown that subjects will voluntarily restrict their COP movements to a smaller area within the biomechanical stability limits when faced with an increase in postural threat. Here we define postural threat as the consequences of losing postural stability (i.e., falling); for example, standing on an elevated platform constitutes a greater threat than standing on the ground, because the consequences of falling from a height are potentially more severe. Several studies have shown that when subjects are asked to stand quietly on low vs. high platforms, their mean COP position shifts away from the platform edge and their COP movement amplitudes decrease at greater platform heights (Adkin et al. 2000; Carpenter et al. 1999, 2006; Davis et al. 2009; Hauck et al. 2008; Huffman et al. 2009).

1.2.3 Anticipatory postural adjustments

Bernstein (1967) postulated that the control of voluntary movements made while standing must include two feedforward components: a component related to the focal movement, and a component related to maintaining standing posture. The latter component has been identified in many experiments, and is termed an anticipatory postural adjustment (APA). These adjustments are frequently taken for granted as we pursue our daily activities, but they are a fundamental component of our ability to make effective movements.

As postulated by Bernstein, familiar and predictable movements are usually preceded by APAs, which involve the activation of postural muscles and resultant COP movement initiated prior to onset of the focal movement. APAs act to control the whole-body COM against the impending shift in dynamics caused by the movement, thus helping to maintain postural equilibrium (Belen'kii et al. 1967; Massion 1992; Traub et al. 1980; Winter et al. 1990); they can also aid in generating the desired movement (Bouisset et al. 2000; Bouisset and Zattara 1987;

Cordo and Nashner 1982; Crenna and Frigo 1991; Lepers and Breniere 1995; Stapley et al. 1998, 1999).

In order to generate an APA that is appropriate for a given movement or perturbation, one must make an accurate prediction about the impending dynamics; if the prediction is not accurate, the APA will be too large or too small for the actual dynamics, and some further compensatory action will be required. That compensatory action, similarly involving activation of postural muscles and COP movement, is called a reactive postural adjustment (RPA). RPAs are also observed when subjects experience an unexpected perturbation; they can begin as early as 60 ms, and as late as 100-120 ms, after the onset of the perturbation (Aruin and Latash 1995; Cordo and Nashner 1982; Nashner 1976, 1979; Santos et al. 2010). Therefore, any postural control activity observed earlier than that is considered anticipatory.

APAs are usually proportional to the magnitude of the impending movement or perturbation (Beckley et al. 1991; Bertucco and Cesari 2010; Bouisset et al. 2000; Horak et al. 1984, 1989; Horak and Diener 1994; Kaminski and Simpkins 2001; Lee et al. 1987; Smith et al. 2012; Yiou et al. 2007). They can also be affected by changes in postural stability limits (Aruin et al. 1998; Cordo and Nashner 1982; Kaminski and Simpkins 2001; Yiou et al. 2007), postural threat (Adkin et al. 2002), and perturbation uncertainty (Beckley et al. 1991; Horak et al. 1989; Horak and Diener 1994; Smith et al. 2012; Toussaint et al. 1998). However, these effects are not well understood.

1.2.4 Postural adaptation

Previous studies investigated adaptation of arm reaching and postural control concurrently, and showed specific differences in adaptation between the two modalities (Ahmed

and Wolpert 2009; Manista and Ahmed 2012). This suggests that adaptation occurs via a distinct mechanism in each form of movement. Indeed, there are fundamental differences between whole body and arm reaching movements, foremost of which are the presence of postural stability limits and the threat associated with a fall.

Ahmed and Wolpert (2009) first demonstrated concurrent adaptation of arm reaching and related postural control to novel dynamics. Similar to what previous studies of seated arm reaching have shown, subjects adapted their arm reaching to novel dynamics; additionally, when subjects performed the experiment while standing, their postural control as well as their arm control showed adaptation to the novel dynamics. When they first encountered the novel dynamics, they exhibited errors in postural control (large deviations of COP movement in response to, and in the direction of, the perturbing forces) as well as errors in arm movement (large deviations of hand position in the direction of the perturbing forces). With practice, subjects learned to generate anticipatory control to specifically counter the perturbing forces, both in posture (anticipatory COP movement, or APA, to compensate for the postural perturbation) and in the arm (anticipatory forces to counter the perturbation at the hand). Finally, when the novel dynamics were removed, subjects demonstrated large postural errors (aftereffects) which gradually decreased with further practice. The adapted anticipatory postural control was also de-adapted with practice.

This study also demonstrated that after subjects adapt an appropriate arm control strategy, they can then transfer that strategy between different postural contexts, namely from sitting to standing or vice versa. In addition, they found that upon switching from sitting to standing, subjects immediately demonstrated perturbation-specific APAs appropriate to the new posture. This indicates that the postural control system can anticipate the effects of movement dynamics

on a new posture and will control posture accordingly. They did find that postural control was adapted at a slower rate than arm reaching, suggesting that arm and postural control are adapted independently.

Manista and Ahmed (2012) used a similar experimental paradigm, in which subjects adapted their arm reaching to novel dynamics while standing; they 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, 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 smaller stability limits in the backward direction. However, the difference may also have been due to the greater threat associated with a backward fall; one study of arm-reaching demonstrated that the threat, or cost, associated with an error could indeed modify adaptation, independent of the magnitude of the error (Trent and Ahmed 2013). Therefore, it is unclear whether the reduced adaptation observed by Manista and Ahmed was caused by reduced stability limits and/or increased threat. They also found that this difference in adapted APAs was not accompanied by differences in adapted arm control, suggesting that the effects of stability limits and/or threat were localized to postural control only.

1.2.5 Clinical relevance

Older adults and other clinical populations demonstrate various other changes in postural control compared to healthy subjects. Several studies have shown that in older adults and individuals with Parkinson's disease, APAs can be reduced, absent, or otherwise inappropriately scaled compared to healthy control subjects (Beckley et al. 1993; Horak et al. 1996; Rogers et al.

1987; Smith et al. 2012; Traub et al. 1980; Woollacott et al. 1988). These populations also seem to exhibit a trade-off in which they sacrifice movement speed and maneuverability in favor of postural stability (Buckley et al. 2008; Chen and Chou 2013; Hass et al. 2005; Hurt and Grabiner 2015; Martin et al. 2002; Polcyn et al. 1998; Rogers et al. 2001). Some of these observed differences in behavior are further exaggerated in the presence of postural threat (Brown et al. 2002; Gage et al. 2003). These behaviors may be partially due to constraints such as decreased muscle strength, increased neural delays, and/or pathological neural deficits (Fugl-Meyer et al. 1980; Fujimoto et al. 2013; Gross et al. 1998; Horak et al. 1996; Papegaaij et al. 2014; Robinovitch et al. 2002; Schieppati and Nardone 1991; Skinner et al. 1984; Thelen et al. 1996; Woollacott et al. 1988). However, they may also be partially due to these subjects choosing to control their posture more conservatively despite being capable of greater performance (Feldman and Robinovitch 2004, 2005; Jessop et al. 2006). Studying feedforward postural control and adaptation, and how they are affected by factors such as stability limits and threat, can help to elucidate mechanisms of postural deficits in these populations and can aid in the development of effective rehabilitation programs.

CHAPTER 2

THESIS OBJECTIVES

In the previous chapter I presented an overview of how we control our standing posture, how we adapt our movement control, and prior work that investigated the overlap between these two areas. This chapter establishes the motivation, specific aims, and significance of my dissertation work.

2.1 Motivation

There is presently a significant gap in our understanding of how whole-body movement control is adapted in response to perturbations. Although many aspects of sensorimotor adaptation have been studied in seated arm movements, postural control is a distinct motor domain; it is subject to inherent stability constraints and is also significantly affected by other factors. Therefore, the goal of this work is to extend our understanding of motor adaptation in whole-body postural control, by investigating the mechanisms of adaptation in postural control as well as how this adaptation is affected by postural stability limits. I will specifically investigate how movement error drives adaptation, how different control strategies are used throughout adaptation, and how adapted control is transferred between different contexts.

2.2 Specific aims

The main objective of this research is to investigate the mechanisms of postural adaptation in a whole-body movement task. To study postural adaptation, I used an experimental adaptation paradigm in which subjects made reaching movements while standing and holding the handle of a force-generating robotic arm that could apply novel perturbations to arm reaching and to standing posture concurrently.

I will address four specific aims in this dissertation:

Aim 1: Determine the relationship between movement error and adaptation in posture.

Approach: I investigated trial-to-trial adaptation of arm reaching and postural control concurrently. Subjects experienced perturbations of varying strengths. I quantified changes in trial-to-trial error and adaptation with varying perturbation strengths. In a follow-up experiment, I determined the effect of error size and consistency on adaptation. Together, these experiments allowed me to quantify the relationship between adaptation and error magnitude, in both arm and postural control.

Significance: This work demonstrates how adaptation scales with motor error in standing postural control, which is the fundamental relationship that drives adaptation. The results give important insight into how error constitutes a signal that can drive adaptation, and how error size and uncertainty can affect that signal. These findings are directly applicable to designing rehabilitation programs and are also important in understanding adaptation on a theoretical and neural level.

Aim 2: Investigate the time course of postural adaptation strategies and how these are affected by stability limits.

Approach: I investigated the pattern of control strategies that are used at different times during adaptation in both arm and postural control: impedance control via muscle coactivation, vs. anticipatory dynamic control. I also examined muscle activity correlates of adaptation. I investigated how adaptation strategies are affected by postural stability limits and/or postural threat, using perturbation direction to manipulate these factors. I tested two subject groups; one group experienced a perturbation in the forward direction (with larger stability limits and decreased threat), and the other group experienced a perturbation in the backward direction (with smaller stability limits and increased threat). I compared the extent of adaptation between groups, as well as the control strategies used by each group during adaptation, in both arm and postural control.

Significance: This work demonstrates that a muscle coactivation strategy (impedance control) is used to reduce error early in postural adaptation. It also demonstrates that adapted control strategies can be affected by stability limits. The results give insight into how trade-offs between various motor control strategies (e.g. anticipatory vs. reactive control, impedance control vs. dynamic control) are managed under different stability conditions. Understanding these trade-offs is important especially in clinical populations that demonstrate defective and/or inefficient control strategies.

Aim 3: Investigate the effect of stability limits on the extent of postural adaptation and transfer.

Approach: I investigated how adaptation and transfer of postural control are affected by postural stability limits, using stance width to manipulate stability limits without explicitly

changing postural threat. Each subject initially adapted to the dynamics while standing with a wide stance width (larger stability limits) and then switched to standing with a narrow stance width (smaller stability limits), or vice-versa. I quantified the extent of adaptation and transfer in both arm and postural control.

Significance: This work demonstrates that stability limits can affect transfer of adapted postural control between different stability conditions. Specifically, my findings suggest that training in a more stable environment facilitates transfer of learning to new contexts.

Aim 4: Determine the extent to which postural control is dependent on learned arm control.

Approach: I investigated whether postural control can be adapted and transferred independently of arm control in a concurrent posture and arm movement task. Subjects initially adapted to the dynamics while reaching with their dominant arm (right), and then switched to reaching with the same dynamics with their non-dominant arm (left). I tested two subject groups; for one group, the dynamics gradually increased in strength throughout the initial adaptation period, such that subjects experienced only very small errors; for the other group, the dynamics were immediately turned on at full strength, such that subjects initially experienced large errors leading to rapid adaptation. Both groups experienced the full-strength dynamics in transfer. In both arm and postural control, I compared the extent of initial adaptation between groups, and then quantified the extent of transfer when subjects switched arms, to determine if adapted postural control can be transferred even if adapted arm control is not transferred.

Significance: This work demonstrates that postural control is not transferred independently of arm control in a whole-body movement task. In contrast, previous findings show that arm

control can transfer independently of postural control. Thus, these transfer results indicate that postural control in this task is dependent on information about arm movement dynamics, but not vice versa. This gives insight into how postural control is coordinated with concurrent arm movements, and can provide more fundamental information about the underlying mechanisms of whole-body movement planning.

2.3 Significance

This dissertation work provides important advances in understanding how we adapt our standing postural control in response to novel perturbations. Our findings carry implications for theoretical as well as practical areas of motor control research. The results provide insight into the representations and mechanisms underlying adaptive behavior; they also provide insight into how we predictively control our movements, and more specifically how postural control is coordinated with concurrent arm movements, and how these are affected by different conditions in movement tasks and in the environment. Understanding these mechanisms and effects may help to elucidate mechanisms of postural deficits in clinical populations and can aid in the development of effective treatment programs. Fundamentally, our findings are applicable to various research fields including sensorimotor control, cognitive neuroscience, and rehabilitation.

2.4 Outline

The remainder of this document is organized into five chapters.

Chapter 3 describes an experiment investigating trial-to-trial adaptation of arm reaching and postural control concurrently (Aim 1).

Chapter 4 describes an experiment investigating the time course of postural adaptation strategies and the effects of stability limits (Aim 2).

Chapter 5 describes an experiment investigating the effect of stability limits on the extent of postural adaptation and transfer (Aim 3).

Chapter 6 describes an experiment investigating whether adaptation can be driven by very small errors, and whether postural control can be adapted and transferred independently of arm control in a standing-and-reaching task (Aims 1 and 4).

Chapter 7 summarizes the main findings of this thesis, discusses their implications, and proposes future directions.

CHAPTER 3

TRIAL-TO-TRIAL ADAPTATION OF POSTURAL CONTROL

The work in this chapter has been submitted for publication as: "Trial-to-trial adaptation of standing postural control." Pienciak-Siewert A, Horan DP, and Ahmed AA. In review, *Journal of Neurophysiology*.

3.1 Introduction

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 (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 using models which assume a linear relationship between error and adaptation (Baddeley et al. 2003; Donchin et al. 2003; Franklin et al. 2008; 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 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 which 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); 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.

To answer these questions, we will investigate trial-to-trial adaptation of arm reaching and postural control concurrently. In order 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.

3.2 Methods

3.2.1 Theoretical development

To examine the relationship between 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 (Figure 3.1A). 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;

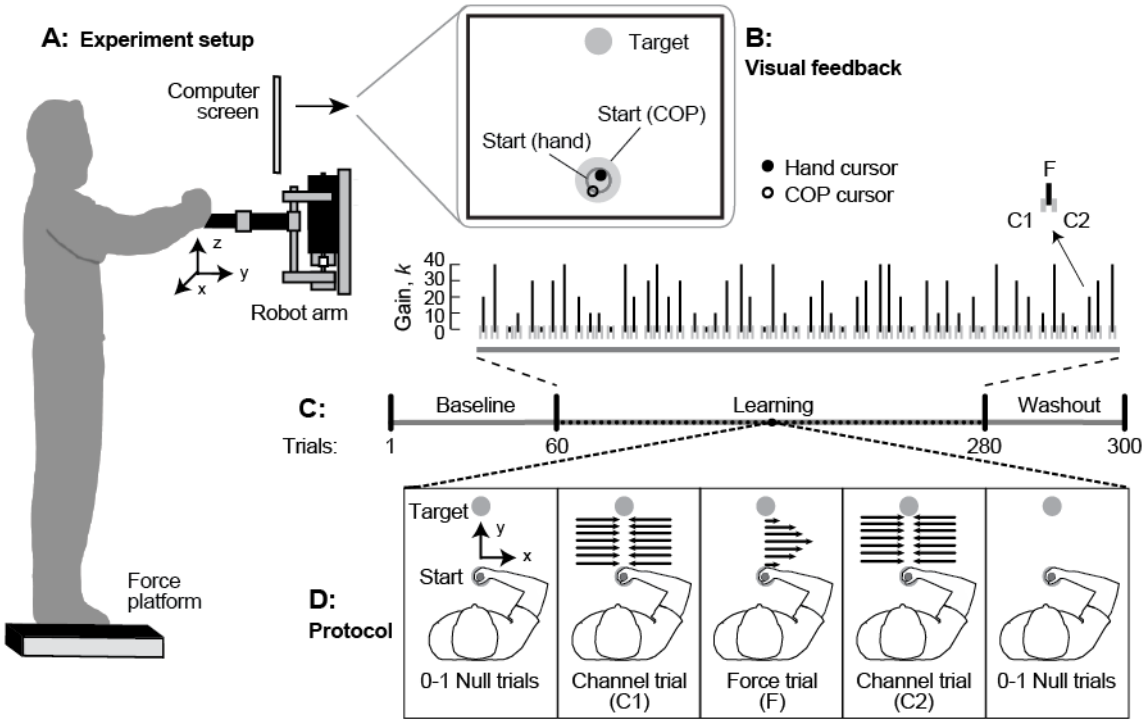

Figure 3.1. Trial-to-trial experiment setup and protocol. (A) Apparatus. (B) Visual feedback provided to subjects on computer screen. (C) Experimental protocol and sample trial list. Each black line represents a force trial of a specified gain; each pair of channel trials is represented by the pair of short gray lines bracketing each force trial. (D) Example trial triplet, illustrating forward (+*y*) reaching movements, perpendicular (±*x*) channel forces, and rightward (+*x*) perturbing forces.

Manista and Ahmed 2012). 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 (Equation 3.1), where *k* is the gain of the force field; we varied the value of *k* (0, 10, 20, 30, or 40 N*s/m) 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 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).

$$\begin{bmatrix} F_x \\ F_y \end{bmatrix} = k \begin{bmatrix} 0 & -1 \\ 1 & 0 \end{bmatrix} \begin{bmatrix} V_x \\ V_y \end{bmatrix} \quad (3.1)$$

Theoretically, when an error is experienced in one trial, the brain responds by changing the motor output on the next trial (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* (Equation 3.2a). If output *x* is known and error *e* is negligible on trials *i*-1 and *i*+1 (Equation 3.2b), 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 Equation 3.2c.

$$x_{i+1} = Ax_i + B(e_i) \quad (3.2a)$$

$$x_i = Ax_{i-1} + B(e_{i-1}) = Ax_{i-1} \quad (3.2b)$$

$$B(e_i) = x_{i+1} - A^2 x_{i-1} \quad (3.2c)$$

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, $x_{i+1} - x_{i-1}$. In order 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) (Figure 3.1D). 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 which 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.

3.2.2 Subjects

Ten young adult subjects (age 21.8 ± 1.7 years; height 175 ± 10 cm; mass 68 ± 10 kg; 5 male, 5 female) 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.

3.2.3 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 Inc.) and while standing barefoot on a six-axis, dual-plate force platform (AMTI Dual-Top AccuSway, Advanced Mechanical Technology Inc.) (Figure 3.1A). 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 about 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 size of their base of support (BOS) was not affected by lifting or rotation of the feet. A computer monitor, vertically suspended in front of the subject, displayed 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 (Figure 3.1B). 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 (Figure 3.1B). 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.

3.2.4 Experimental protocol

The protocol was 300 trials long and was divided into three blocks: baseline (60 trials), learning (220 trials), and washout (20 trials) (Figure 3.1C). 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) (Figure 3.1D). 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 (Equation 3.1). Thus, for a forward reaching movement (in the anterior direction, $+y$), the robot generated rightward perturbing forces ($+x$). In channel trials, stiffness and damping for the force channel were 2000 N/m and 50 N-s/m, respectively. The learning block consisted of 220 trials total (Figure 3.1C), 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, 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. Though the sequence of trials throughout the protocol was pre-determined, 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.

Based on data from pilot testing and previous studies (Ahmed and Wolpert 2009; Manista and Ahmed 2012), we chose the distance and duration of the reaching movement, along with the force field gain values, such that subjects would experience a range of postural perturbations which would be unlikely to exceed postural stability limits, and which would also result in distinct differences in error size between subsequent gain values. The number of trials in the baseline block, the number of triplets per gain value, and the arrangement of interspersing null trials and force trial triplets were chosen based on a previous experiment by Marko et al. (2012) and based on pilot testing data.

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-degree circle at 45-degree 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).

3.2.5 Data collection and analysis

Position, velocity, and force data from the robot handle were sampled at 200 Hz. Center of pressure (COP) position data was 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 was 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 (3000 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 (Figure 3.2A). 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 (Figure 3.2B); 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.

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 (Figure 3.2D). The RPA was calculated as the peak signed value of COP position throughout the remaining duration of the movement (following the APA time period) (Figure 3.2C).

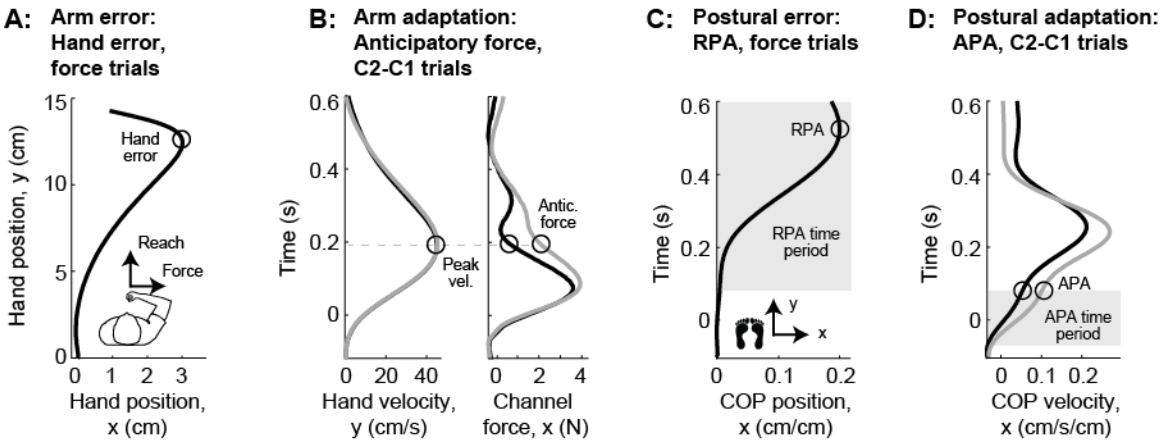

Figure 3.2. Trial-to-trial error and adaptation metrics. (A) Arm error is hand error taken from force trials. (B) Arm adaptation is the change in anticipatory force from C1 to C2 channel trial. (C) Postural error is RPA taken from force trial. (D) Postural adaptation is the change in APA from C1 to C2 channel trial. In (B) and (D), data for C1 channel trial is shown in black and for C2 channel trial in gray.

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 Equation 3.3, where e_i is error as measured on the force trial and e_B is mean baseline error:

(3.3) $$e_i^n = e_i - e_B$$

The baseline error correction was applied to provide similarity with our adaptation metric, where the C1 trial in each triplet acts as a baseline relative to the C2 trial. Based on Equation 3.2c, 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:

(3.4) $$B_i(e_i) = x_{C2} - x_{C1}$$

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}.

3.2.6 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 in order 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 non-zero gain value, we made planned comparisons on the within-subjects results between K0 and each non-zero 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 non-zero 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 ± standard deviation. For all statistical analyses the criterion for significance was set at the level of $\alpha = 0.05$.

3.3 Results

3.3.1 Overview

Group mean trajectory data demonstrate that on force trials with a non-zero force perturbation, hand movement was perturbed rightward, in the direction of the perturbation; stronger perturbations caused a larger deviation in lateral hand trajectory (Figure 3.3A). Similarly, COP trajectories were also deviated rightward (Figure 3.3A). 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 (Figure 3.3C,D) show how subjects adapted their control between C1 and C2 trials. For triplets with a non-zero perturbation, anticipatory force (channel force at time of peak hand velocity, Figure 3.3C) increased in the direction of the hand error experienced on force trials (Figure 3.3A). Similarly, APAs (peak lateral COP velocity during APA period, Figure 3.3D) increased in the direction of

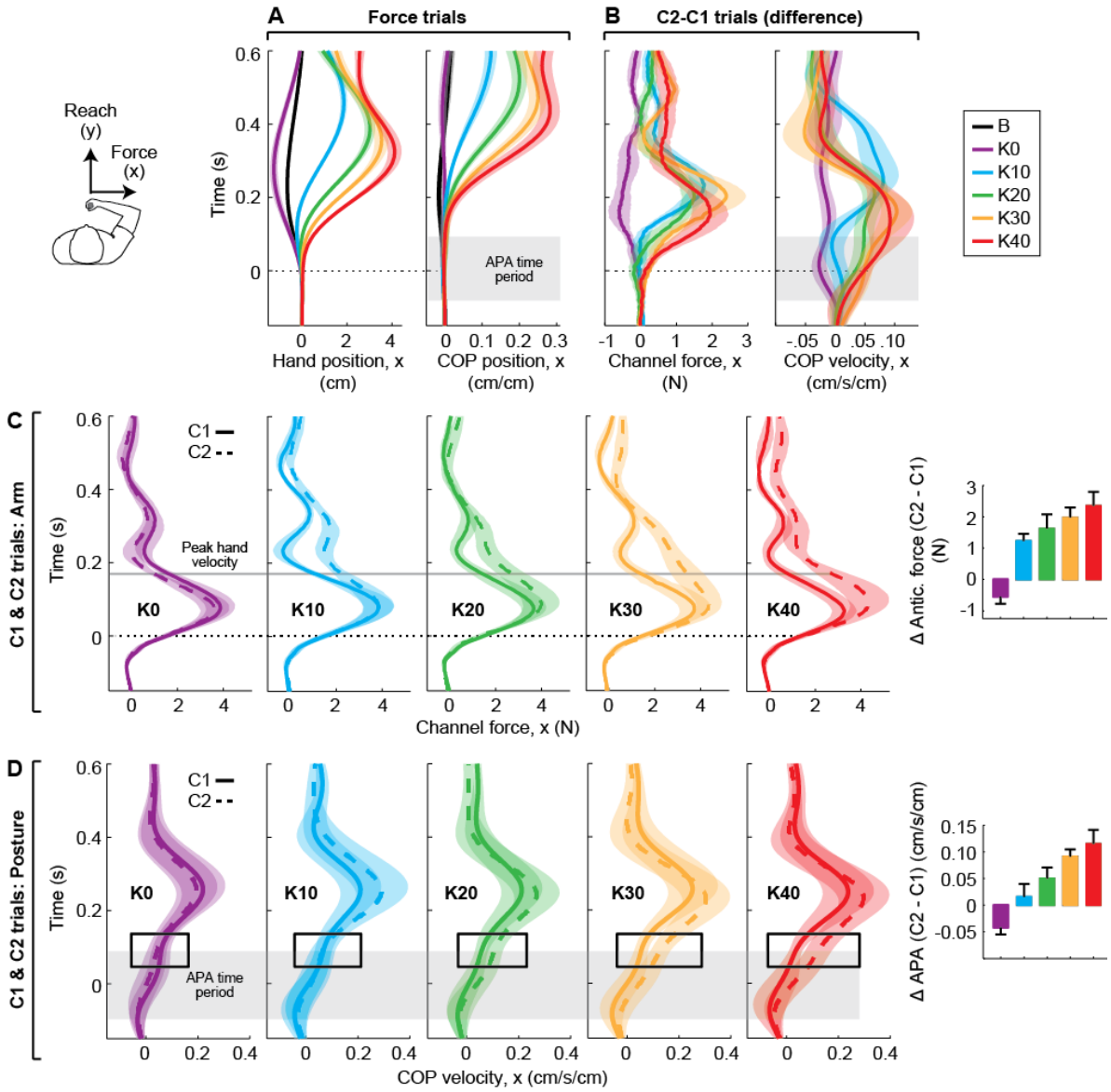

Figure 3.3. Group mean trajectories at each perturbation gain. (A) Group mean trajectories for perpendicular hand and COP position on force trials at each perturbation gain, and on null trials in late baseline (last 10 trials). (B) Group mean difference trajectories (difference between C1 and C2 for 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, 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.

the RPA (maximum lateral COP movement) experienced on force trials (Figure 3.3A). Mean adaptation trajectories (mean difference between C1 and C2 trajectories for each triplet) show how these differences change with perturbation gain (Figure 3.3B).

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, tangential and perpendicular APAs, and mean learning.

3.3.2 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 (Figure 3.4C,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 non-zero 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 non-zero groups (K10 vs. K20, etc.) in the arm (all $p < 0.001$) and in posture (all $p < 0.001$).

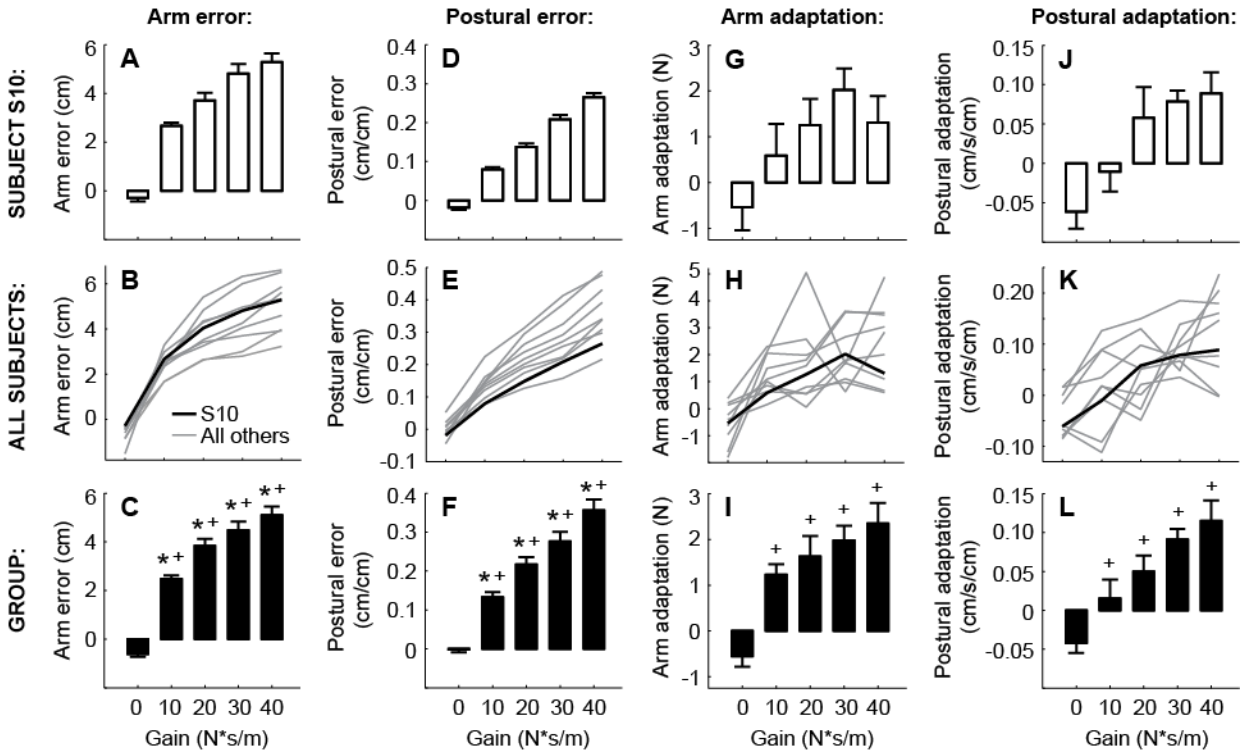

Figure 3.4. Subject and group mean values of error and adaptation. Mean values of arm error (A-C), postural error (D-F), arm adaptation (G-I), and postural adaptation (J-L) at each perturbation gain. Top row: Mean values ± standard error across triplets at each gain for a representative subject (S10). Middle row: Mean values at each gain for all subjects (data for subject S10 is shown in black). Bottom row: Group mean values ± standard error across all subjects at each gain. For group mean values, statistically significant differences ($p < 0.050$) between adjacent gains are denoted by (*), and between K0 and each non-zero gain by (+).

3.3.3 Adaptation

We also examined adaptation at each perturbation gain (Figure 3.4I,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 non-zero 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 non-zero 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.

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 prior to hand movement onset, as has been observed previously (Manista and Ahmed 2012). 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 ($F(4,36) = 0.99$, $p = 0.425$).

3.3.4 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 (Figure 3.5). 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

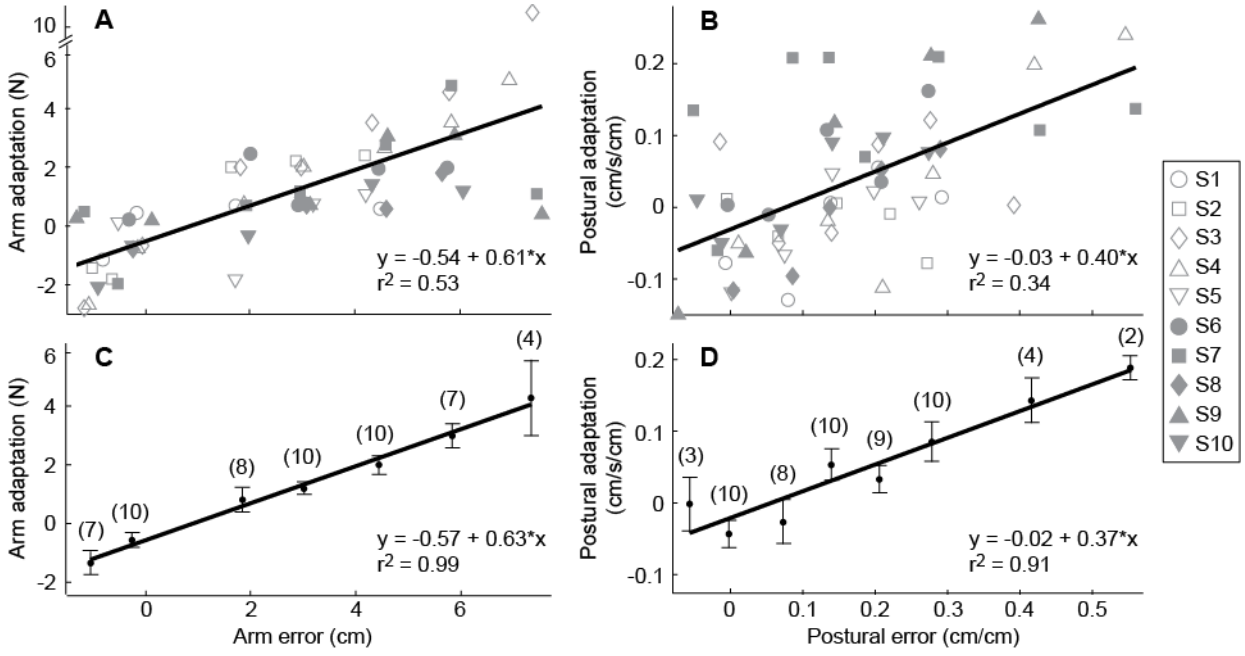

Figure 3.5. Linear regressions of adaptation vs. error. Plots (A) and (C) show arm data; plots (B) and (D) show postural data. Upper plots (A, B) show mean values of adaptation and error at each error bin for each subject (data for each subject is plotted using a unique marker). Lower plots (C, D) show mean value at each bin across subjects (number shown in parentheses above each point indicates number of subjects in that bin); error bars represent standard error for mean adaptation values across subjects (standard error bars for mean error values are too small to be seen on these plots).

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 dominated 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 non-corrected 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) (Figure 3.6). (All COP data was 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

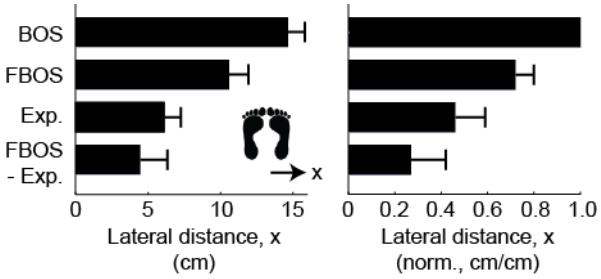

Figure 3.6. BOS limits vs. maximum COP excursion. Group mean values for lateral limit of BOS (50% of stance width, measured from center), lateral limit of functional BOS (FBOS) (maximum lateral COP excursion measured in COP game), maximum lateral COP displacement measured during the experiment, and difference between lateral limit of functional BOS and maximum lateral COP displacement during the experiment. Error bars represent standard deviation across subjects. Left plot shows mean of actual values; right plot shows mean normalized values (normalized to 50% stance width for each subject).

linear relationship between large postural errors and adaptation.

3.3.5 Mean learning over time

We observed that at the K0 gain, both arm and postural adaptation were negative (Figure 3.4I,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 non-zero perturbation magnitude. This is to be expected given the unidirectional force perturbations.

To quantify this mean learning in the arm, we examined non-corrected 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 non-zero gain groups (Figure 3.3A). To quantify similar behavior in posture, we examined APAs and non-corrected RPAs in late baseline and in K0 force trials; RPAs did not significantly change ($p = 0.920$) (Figure 3.3A), 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 (Figure 3.4I,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 (Figure 3.7). 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 approximately 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

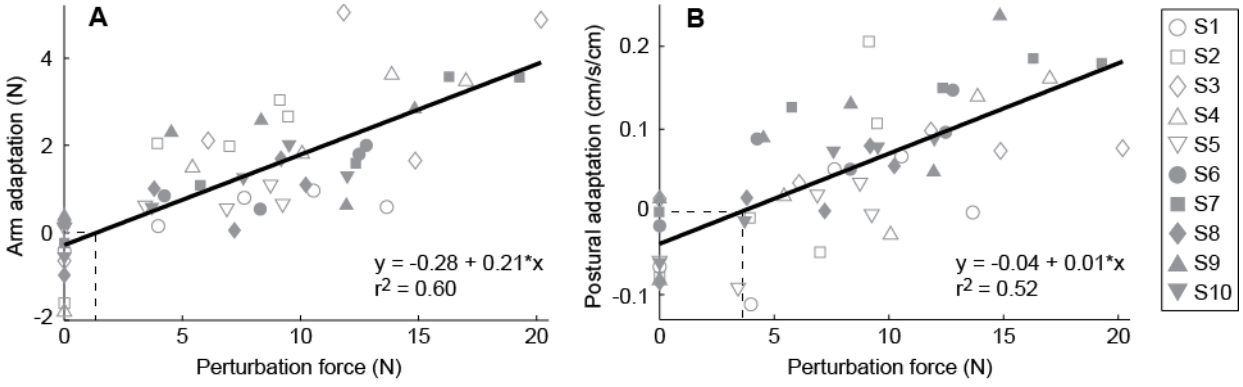

Figure 3.7. Linear regressions of adaptation vs. perturbation force. Plot (A) shows arm data and plot (B) shows postural data, using mean values of adaptation and perturbation force at each error bin for each subject (data for each subject is plotted using a unique marker).

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-values ≥ 0.104).

3.4 Discussion

3.4.1 Summary

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).

3.4.2 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 arm reaching studies which showed good fits to experimental adaptation data using models with a linear relationship between error and adaptation (Baddeley et al. 2003; Donchin et al. 2003; Franklin et al. 2008; 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 which found that adaptation became sub-linear, 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. was 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. perturbation

magnitude became sub-linear 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 about 5 cm (Marko et al. 2012; Fine and Thoroughman 2007) or less than 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.

3.4.3 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 to 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 pre-perturbation 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.

3.4.4 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 in order to remain within torque limits, while hip feedback gains tend to increase in order 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. These findings support the expectation that postural adaptation should increase proportionally with error as long as postural movements don't approach stability limits. In the present experiment, perturbations were not large enough to cause COP movements to approach the limits of the functional 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.

3.4.5 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 non-zero 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 de-adaptation between subsequent trial triplets.

We also noted that subjects adopted a predictive postural control that was targeted to a larger perturbation magnitude (about 3.6 N) than the predictive arm control (about 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).

3.4.6 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 to 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 to 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 to 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 in order to cause errors which are small, in order to promote transfer, but which are large enough to be distinct from baseline performance, in order 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.

Previous studies have shown that adaptation of arm reaching to dynamic or visuomotor perturbations is dependent on the cerebellum (Maschke et al. 2004; Smith & 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.

3.4.7 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.

CHAPTER 4

ROLE OF MUSCLE COACTIVATION IN POSTURAL ADAPTATION

4.1 Introduction

Previous studies of motor adaptation in arm reaching movements have identified two separate control strategies that are used in adaptation to novel dynamics: dynamic control and impedance control (Osu et al. 2002, 2003; Takahashi et al. 2001). In the dynamic 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 involves coactivation of opposing agonist-antagonist muscles; when the coactivated muscles exert equal and opposite torques 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). 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.

2003b; Hinder and Milner 2007; Katayama et al. 1998; Milner and Franklin 2005; Osu et al. 2002).

The above studies clearly indicate that coactivation is a key strategy for controlling movement stability. However, despite the central importance of stability in 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 postural control also showed adaptation to the novel dynamics. 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. This may have been due to reduced postural stability limits (reduced base of support) in the backward direction. They found no corresponding difference in postural error, which could indicate that subjects used a more coactivated strategy to compensate for perturbations in the backward direction.

This study will investigate the role of muscle coactivation in postural adaptation. Do subjects initially rely on a coactivation strategy in standing posture, similar to that observed in seated arm reaching? If so, how are these strategies prioritized for adaptation of posture and

reaching? Subjects will reach in only one direction, and will adapt their arm reaching movements and postural control to a novel perturbation in either the forward or backward direction. The primary objectives are to identify patterns of postural muscle coactivation that develop early in the adaptation process, and to identify changes in muscle activity that correspond to adaptation of arm and postural movements. We will also compare the extent of adaptation and coactivation in the forward vs. backward directions, in order to examine the effects of differing postural stability limits.

Understanding the role of coactivation in adaptation may be especially important with regard to clinical populations such as older adults, who demonstrate increased coactivation and reduced motor adaptation in arm reaching (Huang and Ahmed 2014a). In posture this is of greater concern, given the potentially harmful consequences of losing postural stability (i.e., falling). Older adults show increased postural coactivation in various tasks (Hortobagyi et al. 2009; Manchester et al. 1989; Nagai et al. 2011; Tang & Woollacott 1998), as well as reduced postural stability limits (Binda et al. 2003; Holbein-Jenny et al. 2007; Fujimoto et al. 2013). If we can clarify the role of postural coactivation and how adaptation strategies are affected by stability limits in healthy young adults, this may lead to a better understanding of how these elements might interact in clinical populations.

4.2 Methods

4.2.1 Subjects

Twenty healthy young adult subjects (age 22.1 ± 2.2 years; height 171.1 ± 8.3 cm; mass 64.7 ± 9.8 kg; 8 male, 12 female) 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.

4.2.2 Experimental apparatus and setup

Subjects made rightward 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.) (Figure 4.1A). 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). Subjects were asked to keep their feet flat on the ground, to ensure that the size of their base of support (BOS) was not affected by lifting or rotation of the feet. A computer monitor, vertically suspended in front of the subject, displayed visual feedback of hand, start, and target positions throughout the movement.

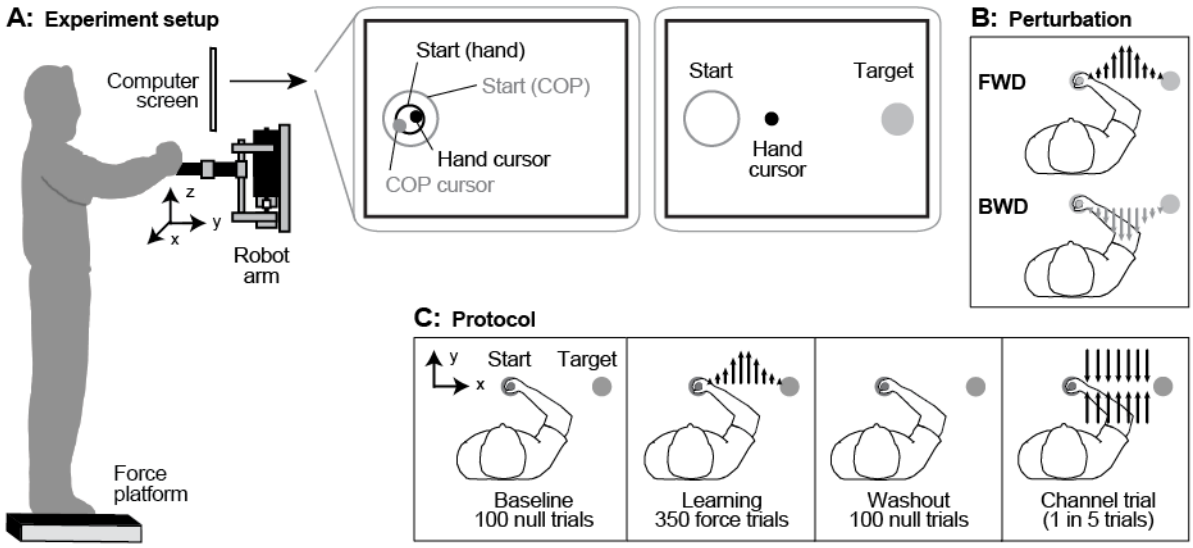

Figure 4.1. Experiment setup and protocol for adaptation to forward vs. backward forces. (A) Experimental apparatus and setup; visual feedback is provided on computer screen. (B) Subjects experienced either a forward (FWD) or backward (BWD) perturbation during the learning block. (C) Experimental protocol.

Before the experiment began, a "start" circle and a cursor representing center or pressure (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 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 their COP location (represented by a separate 0.6-cm cursor of a different color) anywhere within the start circle (Figure 4.1A). 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 duration window of 450 to 600 ms.

4.2.3 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) (Figure 4.1C). 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 de-adapt the previous dynamic environment. The learning block consisted of force trials, in which a viscous curl field was simulated such that the robot exerted 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 (Equation 4.1). Thus, for a rightward reaching movement (+x), the robot generated forward or backward perturbing forces (±y), depending on the sign of the field gain k, where k = ±20 N-s/m.

$$\begin{bmatrix} F_x \\ F_y \end{bmatrix} = k \begin{bmatrix} 0 & -1 \\ 1 & 0 \end{bmatrix} \begin{bmatrix} V_x \\ V_y \end{bmatrix} \quad (4.1)$$

The number of trials in each block was chosen based on pilot testing data, such that subjects had sufficient practice in the baseline and learning blocks that movement metrics and muscle activity reached a near-steady state by the end of each block. The field gain values and the distance of the reaching movement were the same as those used by Manista and Ahmed (2012).

One trial in every batch (5 trials) was chosen randomly to be a channel 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 which the subject was exerting into the channel. Stiffness and damping for the channel were 2000 N/m and 50 N-s/m, respectively. These trials have been shown to have a minimal effect on adaptation or de-adaptation (Scheidt et al. 2000). The sequence of trial types was identical for all subjects.

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), and the BWD group experienced a force perturbation in the backward direction (k = +20 N*s/m) (Figure 4.1B).

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-degree circle at 45-degree 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).

4.2.4 Data collection and analysis

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

Surface EMG data was 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 2000 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 (pilot data suggested that for both forward and backward perturbations, muscle activity adaptations were stronger and more consistent across subjects in the left leg compared to the right leg). 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 zero represents movement onset of the arm, and truncated at movement end. Movement onset was defined as 50 ms prior to when tangential hand position and velocity exceed threshold values of 0.25 cm and 2 cm/s, respectively. 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. 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 was corrupted. A total of 147 trials were rejected, out of the entire data set, with 70 trials excluded for the FWD group and 77 trials for the BWD group (out of 5500 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: Arm control 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 handle 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: Postural control was quantified for each trial, excluding channel trials, using two COP movement metrics: reactive postural adjustment (RPA) (a measure of postural 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 force trials 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 was 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 was 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.

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 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 units of μV/μV. 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*).

4.2.5 Statistics

Data were compared between groups and across 7 phases of the protocol: late baseline (LB), first learning (FL), early learning 1 (EL1), early learning 2 (EL2), late learning (LL), first washout (FW), and late washout (LW). 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 1" phase consisted of the first 5 batches (20 force trials, or 5 channel trials) of the learning block, excluding the first trial ("first learning"). The "early learning 2" phase consisted of the next 5 batches following "early learning 1". The "late baseline", "late learning", and "late washout" phases consisted of the last 5 batches (20 null or force trials, or 5 channel trials) of the trial block.

For the following statistical analyses of hand error, anticipatory force, RPA, and APA data, these metrics were taken as the absolute magnitude of the change from late baseline (to

facilitate the comparison between groups). These metrics were first analyzed using repeated-measures ANOVAs, with phase (all phases) as a within-subjects factor and group as a between-subjects factor. To test for adaptation in each group, we made planned comparisons between the late baseline, first learning, and late learning phases, using paired t-tests. To compare adaptation between groups, we made planned comparisons between groups in early learning 1, early learning 2, and late learning, using independent two-sample t-tests.

We made specific predictions about changes in activity for individual 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 dorsiflexors (TA). In both cases, we might also expect to see decreases in the muscles acting opposite to the primary agonists. To test for changes in 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, using one-sided paired t-tests ($\alpha = 0.05/2$).

We also expected that muscle coactivation would increase early in learning and subsequently decrease. Coactivation metrics were analyzed using repeated-measures ANOVAs, with phase (LB, EL1, EL2, and LL only) as a within-subjects factor and group as a between-subjects factor. To test for changes in coactivation associated with adaptation, we made planned comparisons on the within-subjects results in each group between the late baseline, early learning

1, early learning 2, and late learning phases, using one-sided paired t-tests ($\alpha = 0.05/2$). To test for differences in coactivation between groups, we made planned comparisons between groups of the magnitude of changes from late baseline to early learning 1, early learning 2, and late learning, using independent two-sample t-tests.

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

4.3 Results

4.3.1 Overview

Both groups displayed generally similar arm movement velocities and experienced similar force magnitudes in the learning block. All subjects adapted their arm and postural control as expected, as illustrated in Figure 4.2. When initially exposed to the force field, subjects exhibited movement errors in the direction of the perturbing forces. With practice, they increased their anticipatory control in the direction appropriate to counter the force field. We were able to observe changes in muscle activity that corresponded with these movement adaptations in both arm and posture (Figures 4.4 and 4.7). As expected, we also observed that muscle coactivation levels were increased during early learning and were reduced later (Figures 4.5 and 4.8).

In early learning, anticipatory control metrics (anticipatory force and APA) were similar between groups. However, we did observe differences in error metrics (hand error and RPA): the

BWD group showed greater reduction of hand error and less reduction of RPAs compared to the FWD group. This may be explained by group-related differences in muscle coactivation strategies during early learning. In the arm, the BWD group showed an increase in coactivation in more muscle pairs than the FWD group; conversely, in posture, the FWD group showed an increase in coactivation in more muscle pairs than the BWD group and also showed larger increases in coactivation. Using a linear regression analysis, we were also able to show more

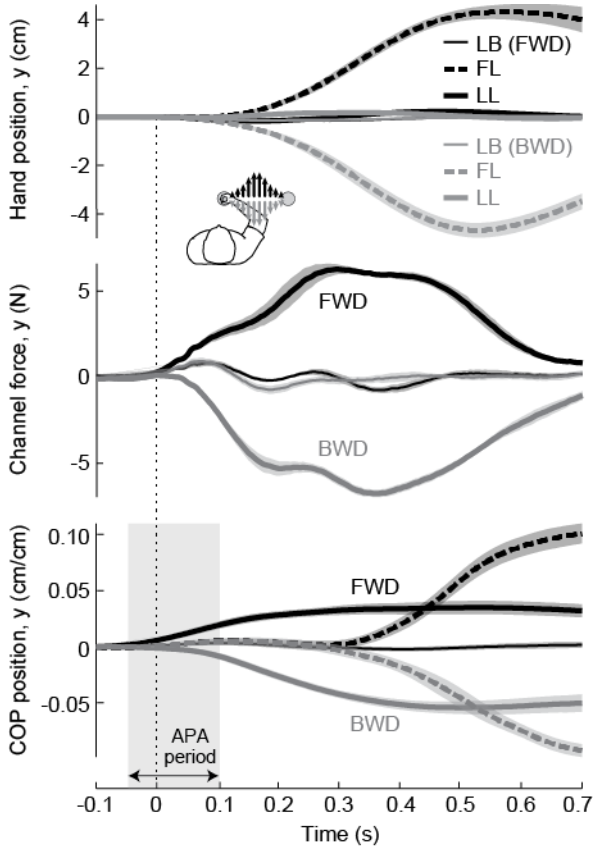

Figure 4.2. Group mean trajectories at key phases of adaptation. Group mean trajectories for late baseline (LB), first learning (FL), and late learning (LL), for FWD group (black) and BWD group (gray). Top: perpendicular hand position (force trials only). Middle: channel force (channel trials only). Bottom: perpendicular COP position (force trials only). Note: Trajectories were averaged across trials in each phase for each subject, then averaged across subjects in each group. Shading indicates standard error across subjects. Time zero represents movement onset of the arm.

directly that reduction of RPAs in early learning was significantly related to increases in coactivation. In late learning, both groups showed similar levels of adaptation in arm control (hand error and anticipatory force). However, in postural control we did observe a trend towards larger RPAs and smaller APAs in the BWD group compared to the FWD group, though this was not statistically significant.

Our main findings were as follows. First, we showed that an early coactivation strategy is used in postural adaptation similar to the coactivation observed in the adaptation of arm reaching movements. We were able to show directly that postural coactivation in early learning is used to reduce error. Second, in muscle activity, we were able to show changes in individual muscles that corresponded to our adaptation findings. Finally, we found differences in adaptation strategies depending on the direction of the perturbation. Results are presented below for movement characteristics (reaching velocity, field force, and COP displacement), arm control (hand error and anticipatory force), posture control (RPA and APA), and muscle activity (arm and postural muscle activity and coactivation).

4.3.2 Arm movement adaptation

To compare adaptation between groups, we had to be sure that both groups made hand reaching movements with similar velocities and experienced similar forces. In the first learning phase, peak velocities and peak field force magnitudes were significantly lower in the BWD group compared to the FWD group ($p = 0.003$). However, at all other learning phases, peak velocities and peak field force magnitudes were similar between groups (all p-values ≥ 0.208).

Hand error and anticipatory force data show that subjects adapted their arm control to the force field during the learning block and de-adapted during the washout block (Figure 4.3A). In

the early portion of the learning block the FWD group made larger hand errors, compared to the BWD group, but showed no differences in adaptation of anticipatory force. In late learning, adaptation of both metrics was similar between groups. For hand error, the ANOVA revealed a main effect of phase ($p < 0.001$, $F = 205.25$) as well as an interaction effect of phase x group ($p = 0.015$, $F = 2.62$), but did not show a main effect of group ($p = 0.494$, $F = 0.49$). For anticipatory force, the ANOVA revealed a main effect of phase ($p < 0.001$, $F = 132.93$), but did not show a

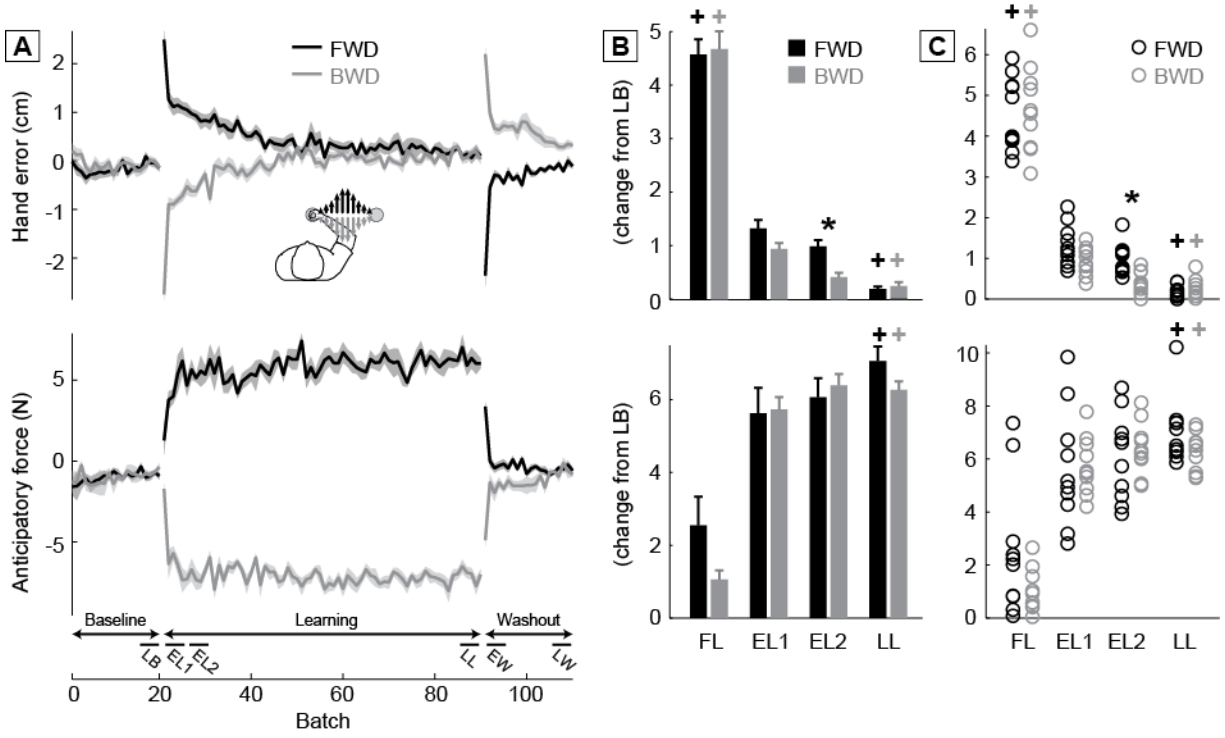

Figure 4.3. Adaptation of hand error and anticipatory force. Upper and lower plots show hand error and anticipatory force, respectively. (A) Left plots show each metric vs. batch (5 trials); in each plot, two traces show group means (solid lines) ± standard error (shading) for FWD group (black) and BWD group (gray). (B) Middle bar plots show the absolute magnitude of the change from late baseline (LB) to first learning 1 (FL1), early learning 1 (EL1), early learning 2 (EL2), and late learning 1 (LL1), for FWD group (black) vs. BWD group (gray); error bars show standard error. (C) Right plots show the absolute magnitude of the change from late baseline (LB) for each subject. Statistically significant differences between groups ($p < 0.050$) are denoted by (*). (+) denotes a statistically significant change within group ($p < 0.050$): hand error, from LB to LL or from FL to LL; anticipatory force, from LB to LL.

main effect of group ($p = 0.472$, $F = 0.54$) or an interaction effect of phase x group ($p = 0.115$, $F = 1.70$).

To test for movement adaptation in the arm, we focused on hand error and anticipatory force in the late baseline, first learning, and late learning phases (Figure 4.3B,C). In both groups, hand error magnitudes significantly increased from late baseline to first learning and significantly decreased from first learning to late learning (all p-values < 0.001). Anticipatory force magnitudes significantly increased from late baseline to late learning (all p-values < 0.001).

To compare adaptation of arm control between groups, we first confirmed that hand error and anticipatory force were similar between groups in late baseline (all p-values ≥ 0.816); peak hand velocity was also similar between groups ($p = 0.539$). Then we compared the magnitudes of the changes in hand error and anticipatory force from late baseline to early learning 1, early learning 2, and late learning (Figure 4.3B,C). These planned comparisons revealed that the changes in anticipatory force from late baseline to early learning 1 and early learning 2 were similar between groups (all p-values ≥ 0.599). However, the changes in hand error from late baseline to early learning 1 and early learning 2 were greater in the FWD group compared to the BWD group (early learning 1 $p = 0.067$, early learning 2 $p < 0.001$), indicating that error was reduced less in the FWD group. This is despite the fact that peak field force magnitudes in these phases were similar between groups (all p-values ≥ 0.208). Changes in both metrics from late baseline to late learning were similar between groups (hand error $p = 0.525$; anticipatory force $p = 0.101$), indicating that the groups adapted their arm control similarly by the end of learning.

4.3.3 Arm muscle activity

To determine which muscles were involved in the unperturbed reaching movement, we focused on arm muscle activity in the late baseline phase. As shown in Figure 4.4, the posterior deltoid and triceps muscles show early activation to initiate arm movement, and the pectoralis major shows inhibition; the pectoralis major and biceps brachii show later activation to decelerate the arm movement. This is consistent with previous descriptions of muscle activity in

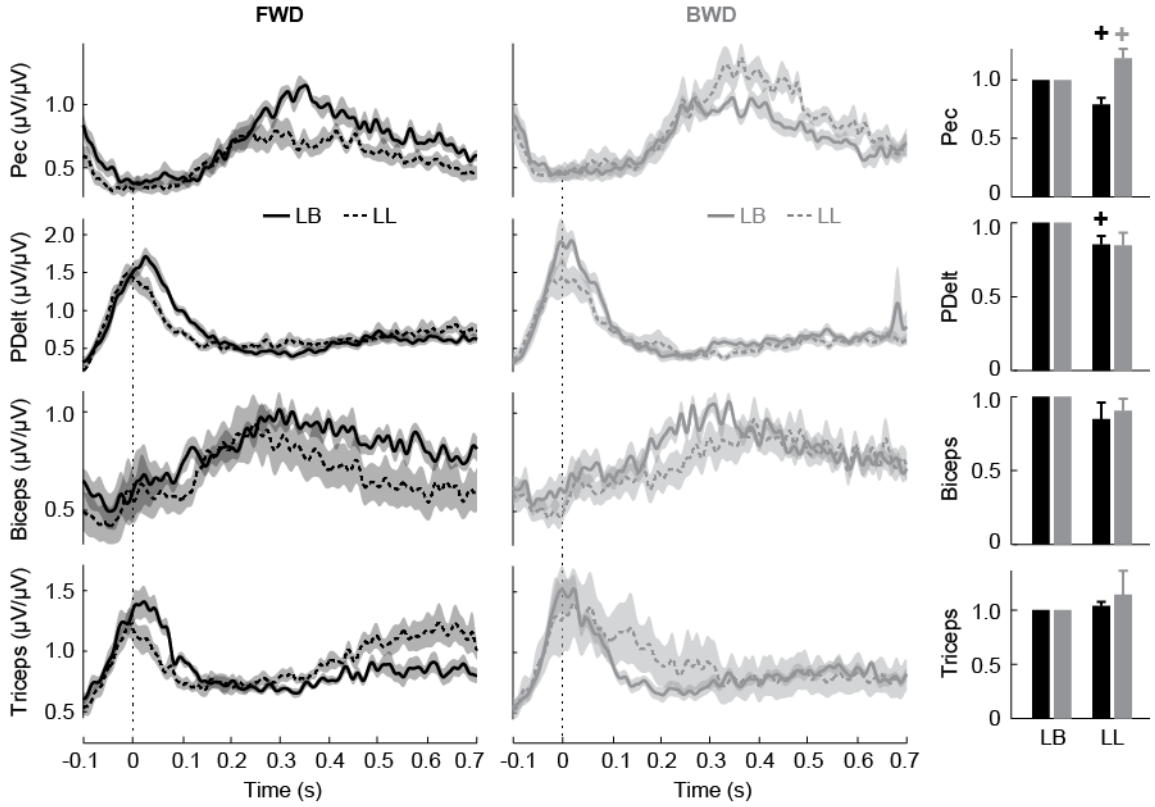

Figure 4.4. Mean arm muscle activity. Mean time traces of muscle activity (normalized EMG) for the pectoralis major (*Pec*), posterior deltoid (*PDelt*), biceps brachii (*Biceps*), and long head of the triceps (*Triceps*), in the late baseline (LB) and late learning (LL) phases, for the FWD group (black) and BWD group (gray); shading indicates standard error across subjects. Time zero represents movement onset of the arm. Far right bar plots show group mean values (root-mean-square value of normalized EMG activity from movement onset to movement end) at late baseline (LB) and late learning (LL), for FWD group (black) vs. BWD group (gray); error bars show standard error. (+) denotes a statistically significant change from LB to LL within group ($p < 0.050$).

rightward arm reaching (Gribble et al. 2003). Because muscle activity is normalized to late baseline, we also confirmed that non-normalized activity in all arm muscles was similar between groups in late baseline (all p-values ≥ 0.091).

To examine adaptation of arm muscle activity, we focused on changes from late baseline to late learning (Figure 4.4). The FWD group showed significant decreases in activity in the pectoralis major ($p = 0.003$) and posterior deltoid ($p = 0.014$). The BWD group showed a significant increase in activity in the pectoralis major ($p = 0.022$) and a trend towards a decrease in activity in the posterior deltoid ($p = 0.054$). Biceps brachii and triceps activity remained similar from late baseline to late learning in both groups (all p-values ≥ 0.203).

For both arm coactivation metrics, the ANOVA revealed a main effect of phase (both p-values < 0.001, $F \geq 8.86$), but did not show a main effect of group (both p-values ≥ 0.795, $F \leq 0.07$) or an interaction effect of phase x group (both p-values ≥ 0.834, $F \leq 0.29$). To examine the role of arm coactivation in adaptation, we focused on changes in coactivation across the late baseline, early learning 1, and late learning phases (Figure 4.5B). In the FWD group, Pec-PDelt coactivation significantly increased from late baseline to early learning 1 and early learning 2 (EL1, $p < 0.001$; EL2, $p = 0.013$), and then significantly decreased from early learning 1 to late learning ($p < 0.001$); Biceps-Triceps coactivation showed a non-significant increase from late baseline to early learning 1 ($p = 0.052$), and then significantly decreased from early learning 1 to late learning ($p = 0.027$). In the BWD group, Pec-PDelt coactivation significantly increased from late baseline to early learning 1 and early learning 2 (EL1, $p < 0.001$; EL2, $p = 0.028$), and then significantly decreased from early learning 1 to late learning ($p < 0.001$); Biceps-Triceps coactivation also significantly increased from late baseline to early learning 1 ($p = 0.016$), and then significantly decreased from early learning 1 to late learning ($p = 0.002$).

To compare arm coactivation between groups, we compared the magnitudes of the changes from late baseline to early learning 1, early learning 2, and late learning (Figure 4.5B). These planned comparisons revealed no significant differences between groups (all p-values ≥ 0.556).

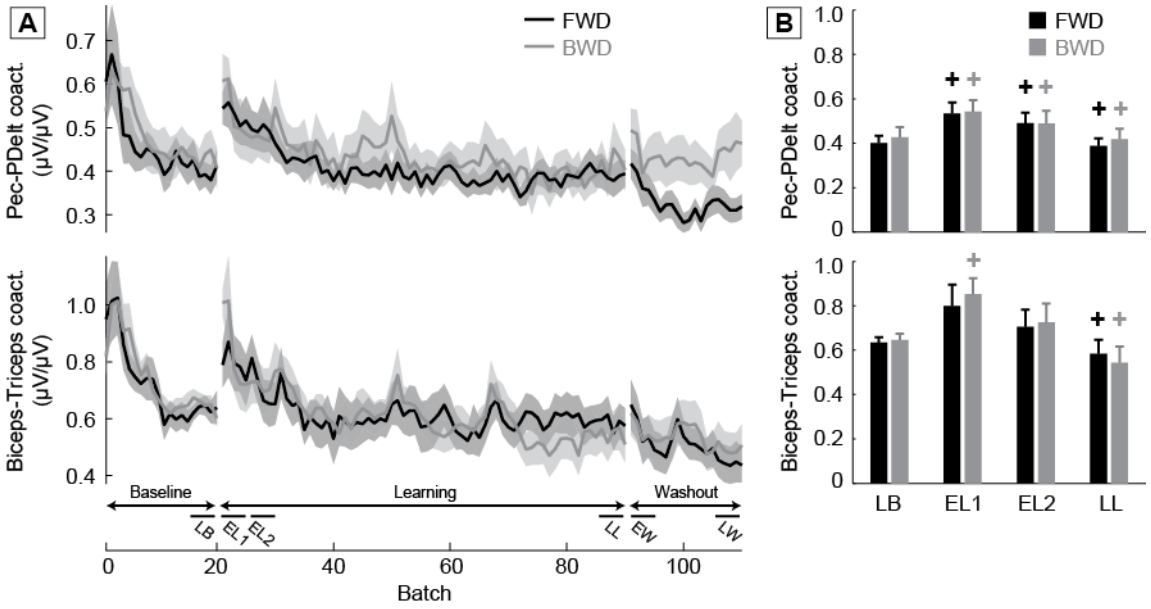

Figure 4.5. Coactivation in arm muscle pairs. (A) Left plots show coactivation vs. batch (5 trials); in each plot, two traces show group means (solid lines) ± standard error (shading) for FWD group (black) and BWD group (gray). (B) Right bar plots show the group mean values at late baseline (LB), early learning 1 (EL1), early learning 2 (EL2), and late learning 1 (LL1), for FWD group (black) vs. BWD group (gray); error bars show standard error. Statistically significant differences between groups ($p < 0.050$) are denoted by (*) (here, no significant differences were found between groups). (+) denotes a significant change within a group from LB to EL1 or EL2, or from EL1 to LL.

4.3.4 Postural movement adaptation

RPA and APA data show that subjects adapted their postural control to the field during the learning block and de-adapted during the washout block (Figure 4.6A). In the early portion of the learning block the BWD group showed larger RPAs, compared to the FWD group, but showed no significant differences in adaptation of APAs. In late learning, we found no

significant differences in adaptation of either metric. However, the data suggest that overall the BWD group made smaller APAs and larger RPAs, compared to the FWD group. For RPAs, the ANOVA revealed a main effect of phase ($p < 0.001$, $F = 101.12$) as well as an interaction effect of phase x group ($p < 0.001$, $F = 8.08$), but did not show a main effect of group ($p = 0.919$, $F = 0.01$). For APAs, the ANOVA revealed a main effect of phase ($p < 0.001$, $F = 19.76$), but did not show a main effect of group ($p = 0.872$, $F = 0.03$) or an interaction effect of phase x group ($p = 0.439$, $F = 0.99$).

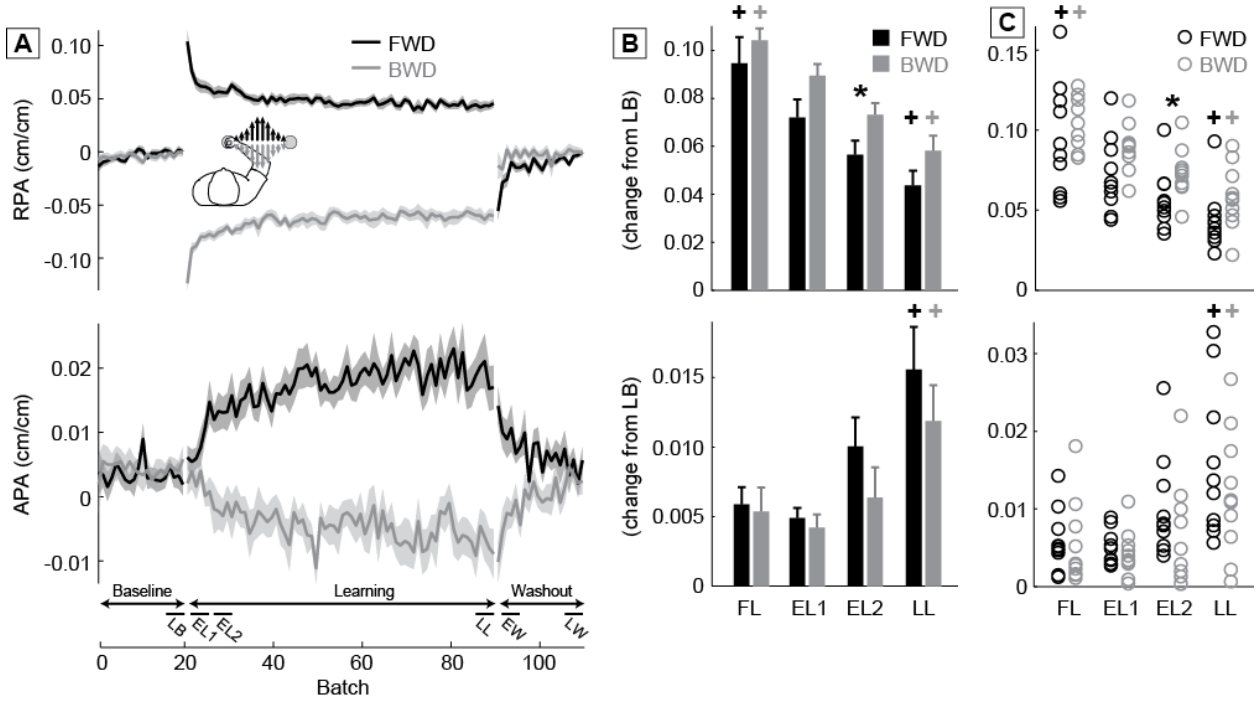

Figure 4.6. Adaptation of RPA and APA. Upper and lower plots show RPA and APA, respectively. (A) Left plots show each metric vs. batch (5 trials); in each plot, two traces show group means (solid lines) ± standard error (shading) for FWD group (black) and BWD group (gray). (B) Middle bar plots show the absolute magnitude of the change from late baseline (LB) to first learning 1 (FL1), early learning 1 (EL1), early learning 2 (EL2), and late learning 1 (LL1), for FWD group (black) vs. BWD group (gray); error bars show standard error. (C) Right plots show the absolute magnitude of the change from late baseline (LB) for each subject. Statistically significant differences between groups ($p < 0.050$) are denoted by (*). (+) denotes a statistically significant change within group ($p < 0.050$): RPA, from LB to LL or from FL to LL; APA, from LB to LL.

To test for movement adaptation in posture, we focused on changes in RPAs and APAs in the late baseline, first learning, and late learning phases. In both groups, RPA magnitudes significantly increased from late baseline to first learning and significantly decreased from first learning to late learning (all p-values < 0.001). APA magnitudes significantly increased from late baseline to late learning (all p-values < 0.001).

To compare adaptation of posture control between groups, we first confirmed that RPAs and APAs were similar between groups in late baseline (all p-values ≥ 0.534). Then we compared the magnitudes of the changes in RPAs and APAs from late baseline to early learning 1, early learning 2, and late learning (Figure 4.6B,C). These planned comparisons revealed that the changes in RPAs from late baseline to early learning 1 and early learning 2 were greater in the BWD group compared to the FWD group (early learning 1 $p = 0.064$, early learning 2 $p = 0.042$), indicating that RPAs were reduced less in the BWD group. This is a reversal of what was observed in hand error, where error was reduced more in the BWD group than in the FWD group. In late learning, the changes in RPAs continued the trend of being greater in the BWD group than in the FWD group, but this was not significantly different ($p = 0.114$). The changes in APAs from late baseline to early learning 1, early learning 2, and late learning were not significantly different between groups (all p-values ≥ 0.189), though there did appear to a trend toward smaller APAs in the BWD group overall. These results suggest that generally the BWD group adapted their APAs less and instead relied on larger RPAs to compensate for the perturbation. RPA and APA magnitudes in the learning phases (not relative to late baseline) show this more clearly. In all three learning phases, RPA magnitudes were significantly larger in the BWD group than in the FWD group (all p-values ≤ 0.031). APA magnitudes were not significantly different in early learning 1 ($p = 0.368$), but in early learning 2 and late learning

they were significantly smaller in the BWD group than in the FWD group (both p-values ≤ 0.035).

4.3.5 Postural movement characteristics

To verify that the COP movements executed during the experiment were within the limits of the functional BOS (fBOS), we compared maximum COP displacements during the experiment to those measured during the COP game (which established the dimensions of the fBOS). (All COP data was measured from the "start" location, and normalized to foot length. Across all subjects, mean foot length was 25.1 ± 1.5 cm.) In the COP game, averaged across all subjects, maximum forward displacement was 0.41 ± 0.09 cm/cm (normalized), or 10.4 ± 2.3 cm, and maximum backward displacement was 0.31 ± 0.05 cm/cm (normalized), or 7.9 ± 1.3 cm; the magnitudes of these fBOS limits were significantly different between the forward and backward directions (paired t-test, $p = 0.002$). This confirms that the fBOS is larger in the forward than the backward direction. We then compared these limits between groups. In the FWD group, the forward fBOS limit (measured in the COP game) was 0.40 ± 0.08 cm/cm (normalized), or 10.0 ± 2.0 cm; in the BWD group, the backward fBOS limit was 0.31 ± 0.06 cm/cm (normalized), or 7.9 ± 1.6 cm. The magnitudes of these limits were significantly different between groups (independent t-test, $p = 0.008$). Finally, maximum COP displacement magnitudes in the experiment were compared to fBOS limits and were also compared between groups. In the experiment, maximum COP displacement magnitudes in the FWD group were 0.15 ± 0.04 cm/cm (normalized), or 3.8 ± 1.1 cm; in the BWD group they were 0.18 ± 0.05 cm/cm (normalized), or 4.5 ± 1.3 cm. These magnitudes were similar between groups (independent t-test, $p = 0.197$). COP displacements in the experiment did not meet or exceed the

limits of the functional BOS in any subject. In the FWD group, the "safety margin," measured as the difference between the forward fBOS limit and maximum forward COP displacement during the experiment, was 0.26 ± 0.10 cm/cm (normalized), or 6.2 ± 2.5 cm. In the BWD group, the safety margin was 0.13 ± 0.07 cm/cm (normalized), or 3.3 ± 1.8 cm, measured as the difference between the backward fBOS limit and maximum backward COP displacement during the experiment. These safety margins were significantly different between groups (independent t-test, $p = 0.004$). Overall, these results confirm that COP movements developed in response to the force field were well within the limits of the functional BOS 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 prior to hand movement onset, as has been observed previously (Manista and Ahmed 2012). 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 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). Across all subjects, the magnitude of the tangential APA showed no significant differences between phases (all p-values ≥ 0.122); and at all phases, magnitudes did not significantly differ between groups (all p-values ≥ 0.103).

4.3.6 Postural muscle activity

To determine which muscles were involved in the tangential APA related to the reaching movement, we focused on anticipatory postural muscle activity in the late baseline phase. As shown in Figure 4.7, 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. We also confirmed that non-normalized activity in all postural muscles was similar between groups in late baseline (all p-values ≥ 0.185).

To examine adaptation of postural muscle activity, we focused on changes in anticipatory and reactive activity from late baseline to late learning (Figure 4.7). The FWD group showed significant increases in anticipatory activity in the PL ($p = 0.016$), MGas ($p = 0.007$), and Sol ($p = 0.002$), and significant increases in reactive activity in the MGas ($p = 0.031$) and Sol ($p = 0.011$). They also showed a significant decrease in reactive activity in the TA ($p = 0.041$). No other significant changes in activity were observed in the FWD group (all other p-values ≥ 0.111). The BWD group showed a significant increase in reactive activity in the RF ($p = 0.040$), a significant decrease in reactive activity in the MGas ($p = 0.023$), and a trend toward an increase in anticipatory activity in the RF ($p = 0.105$), but no other significant changes in activity (all other p-values ≥ 0.133). The fact that we observed significant changes only in reactive activity aligns with the observation that the BWD group showed a trend toward larger adapted RPAs and smaller adapted APAs in late learning compared to the FWD group. Combined, these results suggest that the BWD group adapted their APAs and anticipatory muscle activity less, and instead relied on a more reactive strategy to compensate for the perturbation.

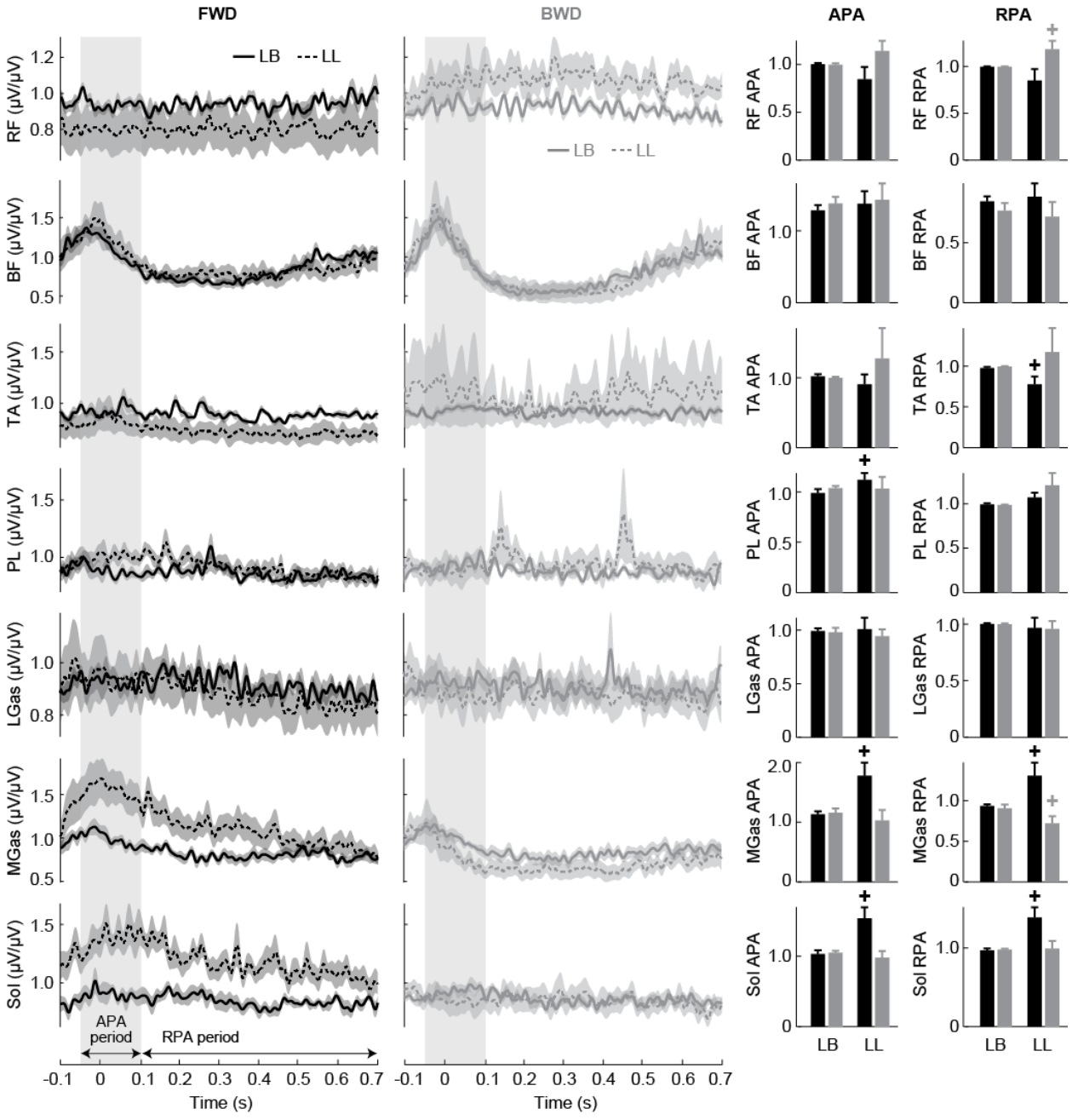

Figure 4.7. Mean postural muscle activity. Mean time traces of muscle activity (normalized EMG) for the rectus femoris (*RF*), biceps femoris (*BF*), tibialis anterior (*TA*), peroneus longus (*PL*), lateral gastrocnemius (*LGas*), medial gastrocnemius (*MGas*), and soleus (*Sol*), in the late baseline (LB) and late learning (LL) phases, for the FWD group (black) and BWD group (gray); shading indicates standard error across subjects. Time zero represents movement onset of the arm. Far right bar plots show group mean values (root-mean-square value of normalized EMG activity over the anticipatory and reactive periods, respectively, left and right bar plots) at late baseline (LB) and late learning (LL), for FWD group (black) vs. BWD group (gray); error bars show standard error. (+) denotes a statistically significant change from LB to LL within group ($p < 0.050$).

For all postural coactivation metrics, the ANOVA revealed a main effect of phase (all p-values < 0.017, $F \geq 3.69$). Coactivation in the TA-PL muscle pair did not show a main effect of group ($p = 0.678$, $F = 0.18$), but did show an interaction effect of phase x group ($p = 0.047$, $F = 2.83$). Coactivation in all other pairs showed no main effect of group (all p-values ≥ 0.204, $F \leq 1.73$) and no interaction effect of phase x group (all p-values ≥ 0.090, $F \leq 2.28$). To examine the role of postural coactivation in adaptation, we focused on changes in coactivation across the late baseline, early learning 1, and late learning phases (Figure 4.8B). 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 1 (all p-values ≤ 0.041) and then significantly decreased from early learning 1 to late learning (all p-values ≤ 0.012); TA-PL, TA-LGas, TA-MGas, and TA-Sol coactivation also significantly increased from late baseline to early learning 2 (all p-values ≤ 0.004). In the BWD group, RF-BF coactivation slightly increased from late baseline to early learning 1 ($p = 0.085$) and then slightly decreased from early learning 1 to late learning ($p = 0.059$); no other postural muscle pairs showed a significant increase in coactivation from late baseline to early learning 1 or early learning 2 (all p-values ≥ 0.142). To further examine coactivation in the BWD group, we looked for changes within individual subjects. Six subjects showed a significant increase from late baseline to early learning 1 and/or early learning 2 in at least one muscle pair (p-values ≤ 0.022), but these increases were found in different pairs across subjects. Four subjects showed no significant increases from late baseline to early learning 1 or early learning 2 in any postural muscle pairs.

To compare postural coactivation between groups, we compared the magnitudes of the changes from late baseline to early learning 1, early learning 2, and late learning (Figure 4.8B). These planned comparisons revealed that the changes from late baseline to early learning 1 were

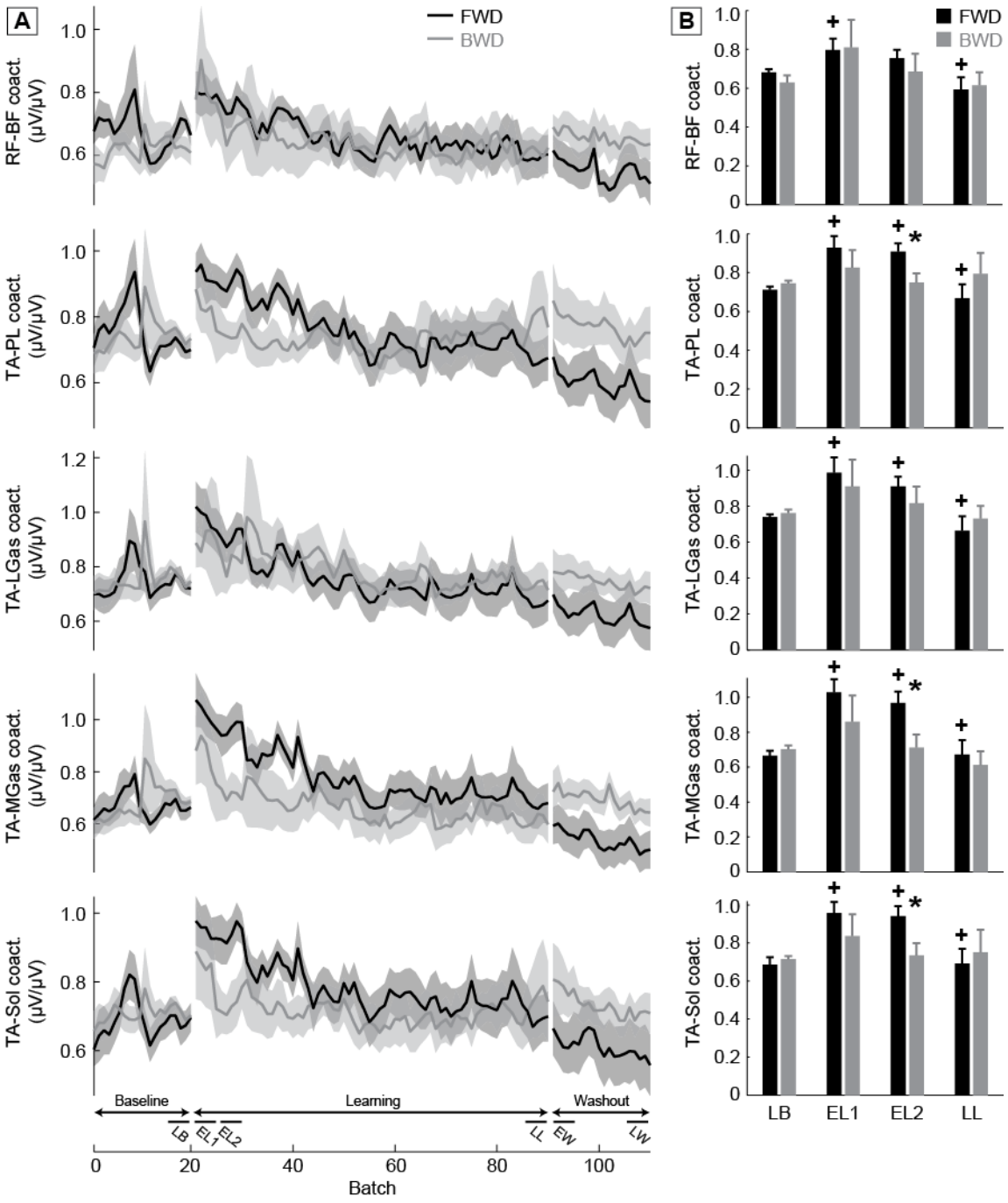

Figure 4.8. Coactivation in postural muscle pairs. (A) Left plots show coactivation vs. batch (5 trials); in each plot, two traces show group means (solid lines) ± standard error (shading) for FWD group (black) and BWD group (gray). (B) Right bar plots show the group mean values at late baseline (LB), early learning 1 (EL1), early learning 2 (EL2), and late learning 1 (LL1), for FWD group (black) vs. BWD group (gray); error bars show standard error. Statistically significant differences between groups ($p < 0.050$) are denoted by (*). (+) denotes a significant change within a group from LB to EL1 or EL2, or from EL1 to LL.

similar between groups for all postural muscle pairs (all p-values ≥ 0.215), and the changes from late baseline to early learning 2 were also similar between groups for the RF-BF and TA-LGas pairs (all p-values ≥ 0.255). However, the FWD group showed a greater increase in coactivation from late baseline to early learning 2 in the TA-PL, TA-MGas, and TA-Sol muscle pairs (all p-values ≤ 0.027). The changes from late baseline to late learning were similar between groups for all postural muscle pairs (all p-values ≥ 0.370).

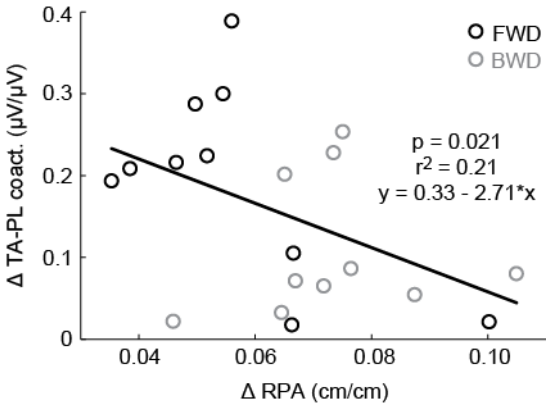

Figure 4.9. Linear regression of TA-PL coactivation vs. RPA. Each data point represents the magnitude of change from late baseline to late learning for one subject (FWD subjects shown in black, BWD in gray). This shows a significant negative correlation between these metrics across all subjects.

Given the significant differences between groups in RPAs and postural muscle coactivation that were observed in early learning 2, we wished to investigate the relationships between RPAs and coactivation. To do so, we performed one-tailed linear regressions between the magnitude changes in RPAs and coactivation from late baseline to early learning 2 (Figure 4.9). Across all subjects, we found a significant negative relationship between the magnitude changes in RPAs and TA-PL coactivation (p = 0.021), such that greater coactivation levels (relative to late baseline) are associated with greater reduction of error (RPAs). This supports the

idea that the FWD group's larger change in coactivation compared to the BWD group may help to explain the FWD group's greater reduction of RPAs.

4.4 Discussion

4.4.1 Summary

In this study we demonstrated changes in muscle activity corresponding to adaptation of postural control. Our results show that an early muscle coactivation strategy is used in postural adaptation similarly to how it is used in the adaptation of arm reaching movements. Specifically, we found that coactivation in both arm and posture is increased in early learning and is later decreased, and that greater postural coactivation in early learning correlates with a larger reduction in error. Our results also suggest that subjects used smaller APAs and larger RPAs overall, and less postural coactivation in early learning only, to compensate for the backward perturbation compared to the forward perturbation.

4.4.2 Early coactivation strategy

Our study demonstrates that during postural adaptation, postural muscle coactivation is increased during early learning and is later decreased. This is similar to what we observed, and what other studies have observed, in arm adaptation (Darainy and Ostry 2008; Milner and Cloutier 1993; Thoroughman and Shadmehr 1999; Van Emmerik 1992). Results from previous studies of postural adaptation suggested the use of an early coactivation strategy, but coactivation was not measured in these 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 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.

Furthermore, we showed a direct correlation between amounts of coactivation and error reduction in a dynamic learning paradigm. While previous studies demonstrated that arm coactivation is increased as hand error is reduced early in adaptation to novel dynamics, these changes were not directly compared. A recent study by Huang and Ahmed (2014b) found that coactivation is also increased early in adaptation to a visuomotor rotation, where no perturbing forces are experienced. This suggests that this type of coactivating strategy is 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 trajectory and endpoint variability in order to meet specific accuracy requirements (Gribble et al. 2003; Osu et al. 2004, 2009; Wong et al. 2009).

4.4.3 Differences in postural adaptation

Throughout learning, we observed data trends indicating that the groups adapted their anticipatory and reactive postural control differently. Overall, it appears that the BWD group adapted to use smaller APAs and larger RPAs than the FWD group. Some differences were not

statistically significant, but that may be caused by large variability between subjects. In fact, the observed differences are supported by our findings on muscle activity adaptation (discussed in the next section). The differences in postural adaptation may be due to the difference in stability limits between the forward and backward directions, where subjects in the BWD group may be less willing to generate appropriately sized anticipatory COP movements (APAs) in the backward direction due to reduced stability limits.

This is in agreement with Manista and Ahmed (2012). That study used a similar experimental paradigm in which subjects adapted their arm reaching to a curl force field while standing; subjects 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 a backward perturbation than for a forward perturbation, despite the fact that subjects had the biomechanical capacity to adapt similarly in both directions. In addition, several other studies have shown that subjects generate smaller APAs when making voluntary arm movements or responding to perturbations in conditions of reduced stability limits (smaller BOS), caused by standing on a wobble board with a narrow beam width (Aruin et al. 1998) or by placing the feet closer together (Kaminski and Simpkins 2001; Yiou et al. 2007). Alternatively, it is also possible that reduced APAs in the backward direction were an effect of increased postural threat, where threat might be influenced by the potential consequences of a backward vs. a forward fall and the uncertainty associated with taking a compensatory step in the backward direction. For example, Adkin et al. (2002) demonstrated that when subjects were asked to rise to their toes while standing, APAs decreased when postural threat was increased (standing on a high vs. low platform).

Manista and Ahmed also found that while subjects adapted their APAs differently, there was no corresponding difference in RPAs. Therefore it seems likely that subjects may have employed more coactivation to compensate for backward perturbations. Similarly, Krishnamoorthy et al. (2004) found that in a condition of restricted stability limits (standing on a wobble board), subjects did not generate optimally-sized APAs to compensate for a postural perturbation, but rather "stiffened" the postural joints using coactivation. However, in the present experiment, we observed a different compensatory strategy. The BWD group compensated for smaller APAs using larger RPAs, and demonstrated larger COP movement magnitudes in general. Consequently, the BWD group also showed smaller margins between their functional BOS limit (measured in the COP reaching game) and the peak magnitude of COP displacement observed during the experiment; this indicates that they moved closer to the edge of their stability limits than the FWD group. Throughout learning, they also showed similar or less postural coactivation compared to the FWD group, rather than more coactivation. This supports the argument that the differences in postural control between the FWD and BWD groups are not explained by the use of a simple "stiffening" strategy.

Rather, our results suggest that the BWD group is using a more cautious, reactive strategy, relying more on reactive control (RPAs) than on anticipatory control (APAs). In contrast, the FWD group uses a more efficient predictive strategy, with larger APAs which allow for smaller COP movements overall (because large RPAs are not needed to compensate for the perturbation). It is possible that Manista and Ahmed did not observe such behavior 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.

In early learning, unlike late learning, we observed significant differences in postural coactivation between groups, in addition to the differences in APAs and RPAs discussed above. The BWD group, compared with the FWD group, showed less coactivation and also showed significantly larger adapted RPAs (magnitude relative to late baseline). Combined with our finding that postural coactivation and error reduction were significantly related in early learning, this suggests that decreased coactivation contributed to the presence of larger RPAs (postural errors) in the BWD group. We believe that the differences in coactivation between groups may be explained by a postural control trade-off between stability and maneuverability that only emerged in early learning.

Previous studies have suggested the existence of a trade-off between stability (ability to reject a perturbation) and maneuverability (ability to quickly respond 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; Jinrich and Qiao 2009; Qiao and Jindrich 2012; Ting et al. 2009; Tirosh and Sparrow 2004). In the present study, the early coactivation strategy that is observed in early learning could cause such a trade-off; when coactivation is increased, stability ("stiffness," used to reject perturbations) is increased and thus errors are reduced, but consequently maneuverability is also reduced. It is possible that the BWD group chose to approach this trade-off differently in posture, given that they already had less postural maneuverability when responding to the perturbation than the FWD group. 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 are less maneuverable, or have less capacity to respond, when responding to the backward perturbation, for two reasons: (1) maximum voluntary ankle torques are greater in plantarflexion than in dorsiflexion (Fugl-Meyer 1981; Fujimoto et al. 2013; Thelen et al. 1996); and (2) BOS limits are smaller in the backward than 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 to the FWD group, the BWD group may have chosen to employ a smaller increase in postural stability (smaller increase in postural coactivation), compared to the FWD group, in order to minimize their loss of maneuverability in the trade-off.

4.4.4 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 in order 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 non-significant change in anticipatory activity. The fact that we did not observe significant changes in anticipatory activity may be explained by two things. First, in adaptation of COP movements from late baseline to late learning, the BWD group seemed to show a smaller change in APAs and a greater change in RPAs, compared to the FWD group. Second, the muscle strategy adaptation 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; 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 in order 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.

4.4.5 Differences in arm adaptation

In late learning, we found no differences in arm adaptation between the FWD and BWD groups; adaptation of hand error, anticipatory force, and arm coactivation were similar between groups. Similarly, Manista and Ahmed (2012) found no differences in arm adaptation between perturbation directions. Generally, these results show that by the end of learning, adaptation of

arm control was not affected by differences in stability limits or by related differences in postural control. These findings, together with findings from Chapter 3 of this dissertation, support the idea that adaptation of arm and postural control occur via similar but distinct mechanisms (Ahmed and Wolpert 2009).

In early learning, however, smaller hand errors than the FWD group. This is the reverse of what we observed in postural control, where the BWD group showed significantly *larger* postural errors (RPAs) than the FWD group. One potential explanation is that the differences in COP movement were mechanically linked to the differences in hand movement. However, this explanation seems unlikely. Several studies have shown that when performance of an arm task is mechanically coupled with postural control, postural control (including quiet standing as well as response to postural perturbation) is altered so as to meet the performance demands of the voluntary arm task (de Lima et al. 2010; Morioka et al. 2005; Papegaaij et al. 2012). However, those studies found that postural movement was restricted in order to provide greater steadiness or stability for the arm task (de Lima et al. 2010; Morioka et al. 2005; Papegaaij et al. 2012). For example, in a steadiness task described by Morioka et al. 2005, postural control became more steady (sway was reduced) with greater steadiness demands at the hand. In contrast, in the present study we found that larger postural errors were associated with smaller hand errors (BWD group, early learning), and vice versa, suggesting that these differences between groups are not due to mechanical coupling of hand and postural errors. In addition, arm control was similar between groups in late learning, despite differences in postural control strategies.

Another possible explanation is that the early learning differences in postural control could have caused an attentional trade-off between arm and postural control, leading to the observed differences in arm behavior. The attentional effect of performing the arm and postural

tasks concurrently would have disappeared by the end of learning, because such effects can be mitigated over time with practice (Pellecchia 2005). In postural control during early learning, larger errors (RPAs) in the BWD group were linked to decreased coactivation; theoretically, this decreased coactivation may have been part of a maneuverability-friendly postural control strategy chosen by the BWD group. If we consider coactivation as an "easy" strategy for minimizing the effects of perturbations, compared to generating rapid compensatory muscle activations as a direct response to a perturbation (Baudry et al. 2010; Dideriksen et al. 2015), then a more coactivated strategy is less attentionally demanding than a less coactivated strategy. Thus, the low-coactivation strategy used by the BWD group would be more attentionally demanding than the greater coactivation used by the FWD group. In addition, several studies have shown that when postural control was more attentionally demanding (e.g. standing with a narrow vs. normal BOS, or standing vs. sitting), performance on a concurrent auditory reaction time task was reduced (reaction times were slower) (Lajoie et al. 1993, 1996; Remaud et al. 2012). Thus the reduced BOS limits in the backward direction could have also contributed to the increased attentional demands of postural control in the BWD group. Consequently, the observed differences in arm behavior between groups could have been a result of attentional resources in the BWD group being allocated more to postural control and less to arm control. It follows that the BWD group might have chosen a less attentionally demanding strategy for the arm, and thus might have used a more coactivated strategy for arm control. We did not observe any significant differences in arm coactivation between groups. However, we did find that the BWD group showed a significant increase in arm coactivation from late baseline to early learning in both pairs of arm muscles, while the FWD group showed a significant increase in only one pair. We

did not measure muscle activity in the brachioradialis or the lateral head of the triceps, but these muscles could have also played a significant role in arm adaptation (Gribble et al. 2003).

4.4.6 Clinical implications

We observed differences in postural adaptation strategies related to differences in stability limits. In the direction of smaller stability limits, subjects adapted smaller APAs and compensated with larger RPAs. However, these COP movements were well within postural stability limits in both directions. Stability limits may be reduced in some clinical populations, such as older adults (Binda et al. 2003; Fujimoto et al. 2013; Holbein-Jenny et al. 2007) and individuals with Parkinson's disease (Jessop et al. 2006; Mancini et al. 2008). This can lead to reduced mobility and a reduced ability to recover from perturbations (Pai and Patton 1997; Robinovitch et al. 2002), thus contributing to poor performance in daily activities and an increased risk of falls. Older adults also show undersized APAs when making voluntary movements (Rogers et al. 2001; Woollacott et al. 1988), as well as slower postural responses to unexpected perturbations (Allum et al. 2002; Singer et al. 2015). The present study showed that healthy young adults can compensate for undersized APAs using larger RPAs; theoretically, larger RPAs could also be used to compensate for a delayed response. However, in individuals with reduced stability limits, it may not be possible to generate sufficiently large RPAs. In the future, the effects of postural stability limits on postural adaptation should be investigated in older adults, in order to learn more about the postural control strategies that these individuals use when compensating for perturbations.

In early learning, we observed group-related differences in postural coactivation and reactive postural control (RPAs). 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 in order to minimize their loss of maneuverability. This highlights an area for concern in populations who exhibit reduced mobility, reduced postural stability, 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 & 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 Parkinson's patients (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 vs. maneuverability strategies is especially critical because both stability and maneuverability are low compared to healthy young adults. For example, these deficits may be a contributing factor to falls in the elderly (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 & Zajac 1993; Robinovitch et al. 2002; Vincent et al. 2002; Taaffe et al. 1999).

4.4.7 Conclusions

The results of this study demonstrate that an early muscle coactivation strategy is used in postural adaptation, similar to how it is used in the adaptation of arm reaching movements. Furthermore, we were able to show directly that postural coactivation in early learning is used to reduce error. We also observed changes in muscle activity corresponding to adaptation of postural control. In the forward vs. backward directions, differences in postural adaptation and

coactivation indicated that postural learning can be affected by differences in postural stability limits. However, by the end of learning, arm adaptation was similar between groups despite the differences in postural adaptation. This demonstrates that differences in postural conditions and associated differences in postural control do not necessarily affect control of a concurrent arm movement.

CHAPTER 5

EFFECTS OF STANCE WIDTH ON POSTURAL ADAPTATION AND TRANSFER

The work in this chapter is also published as: "Transfer of postural adaptation depends on context of prior exposure." Pienciak-Siewert A, Barletta AJ, and Ahmed AA. *Journal of Neurophysiology* 111: 1466-1478, 2014.

5.1 Introduction

All of the movements we make while standing are subject to varying stability constraints dependent on the postural base of support (BOS), but the effect of BOS size on postural control remains something of an enigma. If the size of the BOS is reduced, the minimum distance between the center of pressure (COP) and the edges of the BOS, called the "stability margin," is also reduced; this results in a reduced capacity to recover from a postural perturbation (that is, to recover without taking a step, grasping an external supporting object, or otherwise altering the postural configuration) (Koozekanani et al. 1980; Patton and Pai 1997; Patton et al. 1999; Schulz et al. 2006; Holbein-Jenny et al. 2007). In order to generate an anticipatory postural adjustment (APA) that is appropriate for a given movement or perturbation, one must make an accurate prediction about the impending dynamics; if that prediction is not accurate, the APA will be too large or too small for the actual dynamics, and some reactive or corrective control action will be

required in order to recover postural equilibrium. Therefore, decreasing BOS size and thus stability margins could theoretically lead to reduced APAs because of the reduced capacity to recover. However, increasing BOS size could also lead to reduced APAs if the capacity to recover is sufficiently large that APAs are no longer needed to maintain postural equilibrium.

Studies have examined anticipatory postural control using various manipulations of BOS, such as adding postural supports, or changing the size of the BOS by having subjects stand with their feet in different configurations or stand on wobble boards, with ambiguous results. Aruin et al. (1998) used wobble boards to manipulate BOS, and found that APAs were reduced in the direction of the narrower BOS. However, in this study, APAs may have been reduced simply because the reduced BOS imposed by the wobble board was so narrow that it biomechanically constrained the COP movements. Two other studies found that APAs were also reduced, but in various upper-body-supported conditions of very large BOS, where smaller APAs were sufficient or no APAs were required to help maintain balance (Cordo and Nashner 1982; Hall et al. 2010). In all of these studies, however, the forms of postural support used (a balance board or a trunk support) were not very representative of normal daily activities, but rather induced "extreme" BOS conditions -- namely, either very large BOS (trunk support), where APAs were supplanted by other means of maintaining postural equilibrium; or very small BOS (wobble board), where APAs may have been physically limited by the small BOS. Two other studies found that when subjects adapt their APAs to compensate for a postural perturbation in the forward vs. backward directions, APAs were reduced in the backward direction, with reduced BOS limits; however, these results may also have been influenced by the difference in postural threat levels associated with forward vs. backward perturbations (Manista and Ahmed 2012; Chapter 4 of this dissertation). Therefore it is not entirely clear how BOS size influences APAs.

To address certain aspects of this issue, we investigated the effects of BOS size on adaptation and transfer of a novel postural control strategy, using different stance widths to vary BOS size. In standing posture, when the distance between the feet is varied in the mediolateral direction, the width of the base of support and thus the stability limits are increased. Both narrow and wide stances are familiar and everyday postures, with no explicit difference in postural threat, making this an ideal way to manipulate BOS without reducing the familiarity of the support or constraining COP movements biomechanically. Furthermore, we wished to examine a novel task. Some previous studies used tasks such as reaching or pulling on a handle; but in such an overly familiar task, preference for previously established control strategies may have taken precedence over those more appropriate to the specific postural context (de Rugy et al. 2012). Therefore, to better address the question of how BOS size affects postural control, we considered the adaptation of control strategies for a novel task. We also sought to determine how the postural context in which the task was adapted initially would affect the transfer of the adapted control strategy to a different postural context. Such transfer of adapted control is of significant interest, especially in rehabilitation, where it is desirable for adaptation that is acquired in a training context to be generalized beyond that context. In light of studies which question the idea of whether learning in a more challenging environment is beneficial to adaptation and/or transfer (Wulf et al. 1998; Domingo and Ferris 2009), we hope this investigation will lead to a better understanding of how the postural context in which a movement is adapted may influence the strength of adaptation as well as how well the adaptation generalizes to other postures.

We used a well-studied force field learning experimental paradigm in which subjects make arm reaching movements while holding the handle of a robotic arm that generates forces perpendicular to the reaching direction, and with practice they adapt their arm control as well as

their postural control to compensate for those forces (Ahmed and Wolpert 2009; Manista and Ahmed 2012; Chapter 4 of this dissertation). Ahmed and Wolpert (2009) also showed that in this arm-reaching experimental paradigm, subjects adapt an appropriate arm control strategy and can then transfer that strategy between different postural contexts, namely from sitting to standing. In addition, they found that upon switching from sitting to standing, subjects immediately demonstrated perturbation-specific anticipatory COP movements appropriate to the novel posture. This indicates that the postural control system can anticipate the effects of movement dynamics on a novel posture and will control COP movement accordingly.

In the present study, subjects first adapted to the forces while standing in a wide stance and then transferred to a narrow stance, or vice versa. In both stance widths, the same mechanical perturbation is applied to the arm; based on a quasi-static model of standing posture, the same anticipatory COP movement should be biomechanically sufficient to maintain equilibrium, regardless of stance width. Importantly, these postures are familiar and do not biomechanically constrain the required COP movement, and therefore they should not inhibit APAs (unlike the extremely narrow BOS when standing on a wobble board as in Aruin et al. (1998)). Our hypothesis is that anticipatory postural control, reflected in COP movement, is not affected by stance width, as long as the control remains within functional limits.

Based on our hypothesis, we made several predictions specific to this experiment, regarding the adaptation and transfer of COP movements and related muscle control. We predicted that subjects in either wide or narrow stance would show similar COP movements by the end of the adaptation period; however, due to the difference in biomechanical configuration between stance widths, the anticipatory muscle activity required to generate this COP movement would differ (Bingham et al. 2011). We also predicted that subjects would transfer similar COP

movements from one stance to another, with appropriate changes in anticipatory muscle activity to account for the change in configuration. We made two additional predictions about the adaptation and transfer of anticipatory muscle activity related to these anticipatory COP movements; generally, we expected that different muscle activation strategies would be used in each stance width to account for the difference in biomechanical configuration. In adaptation, we predicted that anticipatory COP movements of the same magnitude would require lower levels of anticipatory muscle activity in wide stance compared to narrow stance, and that subjects in wide stance would rely more on hip muscles than subjects in narrow stance (Henry et al. 2001; Torres-Oviedo and Ting 2010; Bingham et al. 2011). In transfer, we predicted that each group would modify their anticipatory muscle activity appropriately in order to transfer the same COP movement between stance widths (e.g., if similar COP movements were transferred from wide to narrow stance, we would predict an increase in anticipatory muscle activity levels as well as a shift from a hip muscle strategy to an ankle strategy).

5.2 Methods

5.2.1 Subjects

Twelve young adult subjects (age 22.1 ± 1.7 years; height 1.7 ± 0.1 m; mass 68.4 ± 13.0 kg) participated in the study. All subjects were screened using a health questionnaire and the Edinburgh Handedness Inventory test (Oldfield 1971). Inclusion criteria included right-hand dominance, normal or corrected-to-normal vision, and no reported history of neurological or

upper-limb musculoskeletal disorders. The University of Colorado at Boulder Human Research Committee approved all experimental procedures.

5.2.2 Experimental apparatus and setup

Subjects made forward reaching movements (+*y*) 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 force-plate (AMTI LG-6-4-1, Advanced Mechanical Technology Inc.) (Figure 5.1A). 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 shoulder joint of the subject's reaching arm. A computer monitor, vertically suspended in front of the subject, displayed visual feedback of hand, start, and target positions throughout the

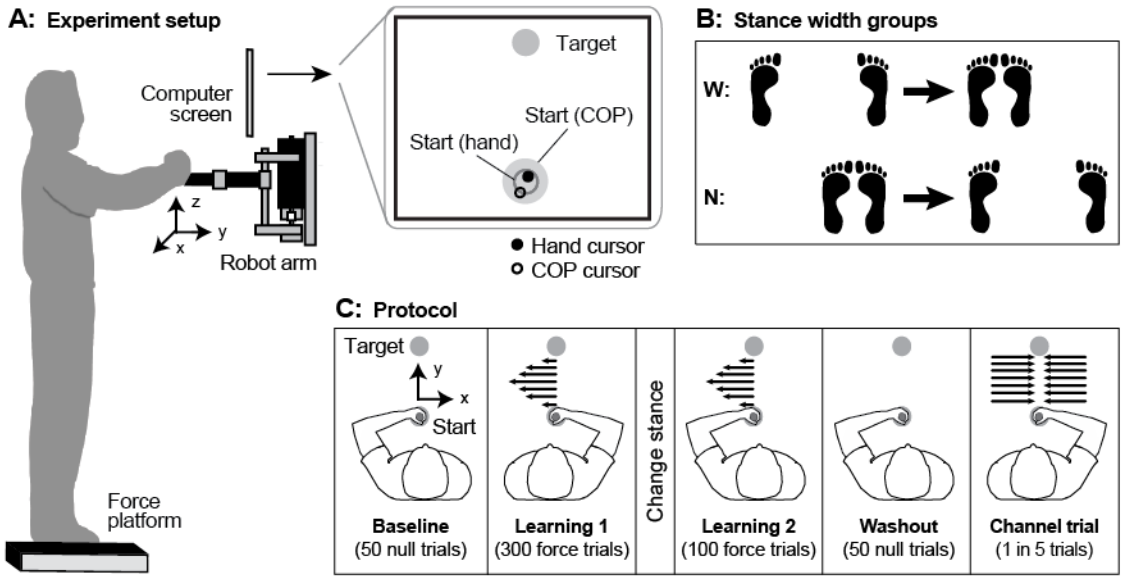

Figure 5.1. Stance width experiment setup and protocol. (A) Apparatus. (B) Subject groups: wide group (W) begins in wide stance and changes to narrow; narrow group (N) begins in narrow stance and changes to wide. (C) Experimental protocol.

movement. Visual feedback about movement duration was provided to ensure that it stayed within a certain range (450-600 ms).

Subjects stood in either a wide stance (150% of hip width, mean 38.6 ± 2.6 cm across all subjects) or a narrow stance (feet placed together, mean 19.6 ± 1.3 cm across all subjects), where stance width was defined as the distance between lateral edges of the feet. These stance widths are comparable to those used in previous studies. Winter et al. (1998) tested stance widths (distance between ankles, measured from joint centers) of 50%, 100%, and 150% of hip width (measured as distance between right and left anterior superior iliac spine); using the same measures, Bingham et al. (2011) tested a range of stance widths from 50% to 200%. Henry et al. (2001) tested stance widths (distance between centers of heels) of 10 and 32 cm, and using the same measure, Torres-Oviedo & Ting (2010) tested 9, 19, and 30 cm (as well as a 60-cm "extreme" stance width). 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.

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 foot positioning was marked on the force-plate to ensure that they always stood in the same location.

Following this, subjects played a brief COP game for the purpose of measuring 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). This game was also used to obtain data for EMG normalization. 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-degree circle at 45-degree angles, and at a

distance of 10 cm from the central start position. This game was first played with feet placed in the wide stance and then repeated with feet in the narrow stance.

After playing the COP game, subjects started the experiment. In the experiment, subjects were asked to make 15-cm reaching movements straight ahead, 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 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. 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. The desired movement time, measured from the time the hand left the start position to the time at which the 50-ms target requirement was fulfilled, was 450 to 600 ms.

5.2.3 Experimental protocol

All subjects encountered the same sequence of trials throughout the experimental protocol (Figure 5.1C). The protocol consisted of 500 trials and was divided into four blocks: baseline (50 trials), learning 1 (300 trials), learning 2 (100 trials), and washout (50 trials). Subjects were randomly assigned into one of two groups, "W" or "N". The W group stood in a wide stance throughout the baseline and learning 1 blocks, and then switched to a narrow stance for the learning 2 and washout blocks; the N group stood in a narrow stance throughout the

baseline and learning 1 blocks, and then switched to a wide stance for the learning 2 and washout blocks (Figure 5.1B).

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 de-adapt the previous dynamic environment. The learning 1 and learning 2 blocks consisted of curl trials, in which a viscous curl field was simulated such that the robot exerted 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 (Equation 5.1). Thus, for a forward reaching movement (in the anterior direction, +*y*), the robot generated leftward perturbing forces (-*x*).

$$\begin{bmatrix} F_x \\ F_y \end{bmatrix} = k \begin{bmatrix} 0 & -1 \\ 1 & 0 \end{bmatrix} \begin{bmatrix} V_x \\ V_y \end{bmatrix}, \text{ where } k = -20 \text{ N*s/m.} \quad (5.1)$$

The number of trials in each block was chosen based on data from pilot testing and the experiment described in Chapter 4, such that subjects had sufficient practice in the baseline and learning blocks that movement metrics and muscle activity reached a near-steady state by the end of each block; however, this was balanced with the desire to minimize the total number of trials, to avoid excessive fatigue due to standing for a long period of time. The force field gain values and the distance of the reaching movement were the same as those used in previous experiments (Chapter 4 of this dissertation; Manista and Ahmed 2012); these parameters were chosen after pilot testing showed that the associated postural perturbations were within the stability limits of both wide and narrow stance.

One trial in every batch (5 trials) was chosen randomly to be a channel trial. 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 perpendicular force which the subject was exerting into the channel. Stiffness and damping for the channel were 6000 N/m and 250 N-s/m, respectively. Channel trials were used to quantify subjects' predictive, feed-forward control. These trials have been shown to have a minimal effect on adaptation or de-adaptation (Scheidt et al. 2001). The sequence of trial types was identical for all subjects.

5.2.4 Data collection and analysis

Position, velocity, and force data from the robot handle were sampled at 200 Hz. Three-dimensional ground reaction forces (F_x, F_y, F_z) and moments (M_x, M_y, M_z) from the force-plate were also sampled at 200 Hz and then low-pass filtered at 10 Hz. Center of pressure (COP) data was calculated from filtered force-plate data as $COP_x = (-M_y - F_x*r_z) / F_z$ and $COP_y = (M_x - F_y*r_z) / F_z$, where x and y subscripts denote mediolateral and anteroposterior axes, respectively, and r_z represents the distance from the top of the force-plate to its origin. COP velocity was calculated using a five-point differentiation algorithm. All COP data for each subject were normalized to foot length.

Surface EMG data was 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 2000 Hz from six right-left pairs of postural muscles: tensor fascia latae (TFL), rectus femoris (RF), biceps femoris (BF), tibialis anterior (TA), peroneus longus (PL), and soleus (Sol). 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 zero represents movement onset of the arm, and truncated at movement end. Movement onset was defined as when the cursor left 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. Data from channel trials were analyzed separately from all other trials. Note that for forward reaching movements (+*y*), the force perturbation is in the leftward direction (-*x*).

For every subject, the same two trials were excluded from analysis, due to a coding error in the sequence of trial types. These were two adjacent trials (a channel trial and a force trial) in the middle of the learning 1 block. Thus a total of 24 trials were rejected, out of the entire data set, with 12 trials excluded for each group (out of 3000 total trials per group, with 500 trials per subject).

Arm control: To confirm adaptation and transfer in the arm, arm control was quantified using two metrics: hand error and anticipatory force. Hand error for each trial (excluding channel trials) was defined as the maximum absolute value of the perpendicular deviation of the handle trajectory from a straight path between the start and target positions. Anticipatory force was taken from channel trials only, and was calculated as the mean of the perpendicular force exerted into the channel over the duration of the movement.

Postural control: Postural control, reflected in COP movement, was quantified for each trial (excluding channel trials) using two metrics: reactive postural adjustment (RPA) and anticipatory postural adjustment (APA). Both of these metrics were based on the normalized COP velocity perpendicular to the direction of reaching movement (where all COP data were normalized to foot length). Horak and Nashner (1986) investigated postural control responses to unexpected backward sway perturbations, and observed reactive response latencies in the tibialis

anterior varying from 73 to 110 ms; this was after repeated exposure, which is known to reduce the latency of automatic postural responses. Therefore, as a conservative measure of anticipatory control, the APA was calculated as the mean velocity from 50 ms before movement onset to 100 ms after movement onset. The RPA was calculated as the maximum positive value of velocity throughout the remaining duration of the movement (following the APA time period). (Positive movement is leftward, in the direction of the perturbation.)

Muscle activity: EMG data was 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. EMG data from one subject in the N group was excluded from analysis on account of excessive noise. For each subject included in the analysis, EMG data was normalized by dividing by the maximum observed activity for each muscle, taken as the maximum filtered EMG activity observed during the pre-experiment COP game in either stance. Anticipatory muscle activity for each trial (excluding channel trials) was quantified as the root-mean-square value of the normalized EMG trace from 100 ms before movement onset to 50 ms after movement onset (50 ms earlier than the time period sampled for COP data, offset to account for the time lag between recorded EMG activity and corresponding COP movement).

5.2.5 Statistics

Data were compared between groups and across 7 phases of the protocol: late baseline, first learning 1, late learning 1, first learning 2, late learning 2, first washout, and late washout. The "first" phases of a trial block consisted of one trial only; for anticipatory force, the "first" phases consisted of the first channel trial. For "late" phases, data was averaged over the last 2 batches (8 non-channel trials or 2 channel trials) of the trial block.

Hand error, anticipatory force, RAPA, and APA data were analyzed using repeated-measures ANOVAs, with phase as a within-subjects factor and group as a between-subjects factor. To test for adaptation, we performed planned comparisons on the within-subjects results between the late baseline, first learning 1, and late learning 1 phases. To compare adaptation between groups, we made a planned comparison in the late learning 1 phase. To test for initial transfer, we performed planned comparisons on the within-subjects results between the late learning 1 and first learning 2 phases. To examine the time course of transfer, we performed additional planned comparisons on the within-subjects results for each group between the first learning 2 and late learning 2 phases. Planned comparisons were also made between groups at specific phases of interest. All planned comparisons were made using independent two-sample t-tests.

Based on previous studies, we made specific predictions about changes in activity for individual muscles that might be involved in development of the anticipatory COP movement (specifically, leftward COP movement): increased activity in the right tensor fascia latae (TFL) and/or decreased activity in the left TFL (Leonard et al. 2009), increased activity in the left tibialis anterior (TA) (Gefen 2001) and/or decreased activity in the right TA (Hopkins et al. 2012) (with similar changes in peroneus longus (PL) activity, because the PL activates with the TA in order to maintain foot-on-ground contact), and also increased activity in the right soleus (Leonard et al. 2009). Directional changes in muscle activity were examined using one-sided paired t-tests ($\alpha = 0.05/2$). To test for differences between groups, we made planned comparisons using independent two-sample t-tests.

ANOVAs were performed using SPSS, and t-tests were performed using the appropriate built-in MATLAB functions. For all statistical analyses the criterion for significance was set at

the level of $\alpha = 0.05$ unless otherwise noted. Mean values are reported as mean ± standard deviation.

5.3 Results

5.3.1 Overview

Both groups displayed similar arm movement characteristics, and also showed similar adaptation of arm control. Across all subjects, similar arm control was transferred between stance widths. Both groups showed similar adaptation of COP movements, by using different anticipatory muscle control strategies to account for the differing stance widths. In transfer, the W group transferred similar COP movements, by modifying their anticipatory muscle activity to account for the new stance. However, the N group showed an increase in COP movements when transferring, related to no change in anticipatory muscle activity.

Results are presented below for movement characteristics (reaching velocity, field force, and COP displacement), arm control (hand error and anticipatory force), COP movements (RPA and APA), and anticipatory muscle activity.

5.3.2 Arm movement characteristics

To compare performance between groups, we had to be sure that both groups made hand reaching movements with similar velocities and experienced similar forces. Average velocities for the reaching movement ranged from 0.31 to 0.36 m/s throughout the experiment; average and maximum velocities were not significantly different between groups at all phases (all p-values ≥

0.133). During learning 1 and learning 2, average and maximum forces were not significantly different between groups at all phases (all p-values ≥ 0.133).

5.3.3 Arm movement adaptation and transfer

Hand error and anticipatory force data (Figure 5.2A) show that subjects adapted to the field during the learning 1 trial block, transferred this adaptation to the learning 2 block with no changes, and de-adapted in the washout block. The ANOVA revealed a main effect of phase (hand error $p < 0.001$, $F = 139.0$; anticipatory force $p < 0.001$, $F = 46.36$), but showed no main effect of group ($p = 0.261$) and no interaction effect of phase x group ($p = 0.261$).

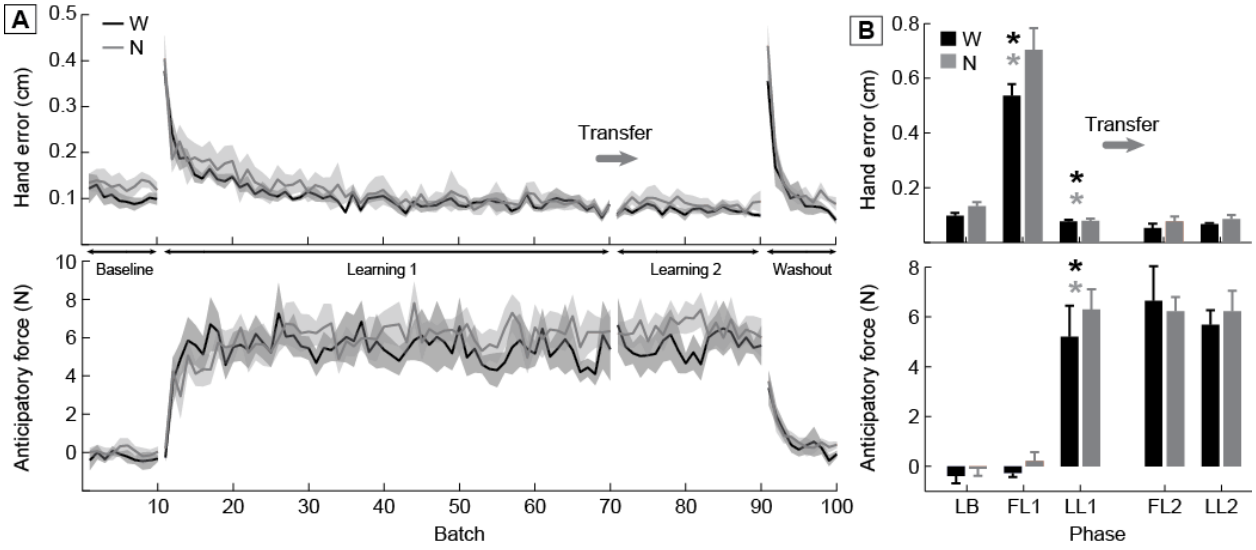

Figure 5.2. Adaptation of hand error and anticipatory force. (A) Upper and lower plots show hand error and anticipatory force, respectively, vs. batch (5 trials); in each plot, two traces show group means (solid lines) ± standard error (shading) for W group (black) and N group (gray). (B) Upper and lower bar charts show hand error and anticipatory force, respectively, at the late baseline (LB), first learning 1 (FL1), late learning 1 (LL1), first learning 2 (FL2), and late learning 2 (LL2) phases, for W group (black) vs. N group (gray), with error bars showing standard error. Statistically significant differences ($p < 0.050$) are denoted by (*) for a change from the previous phase (black for W group, gray for N group).

Adaptation: To test for movement adaptation in the arm, we focused on hand error and anticipatory force in the late baseline, first learning 1, and late learning 1 phases (Figure 5.2B). We found that subjects adapted as expected, and we found no differences between groups. Across all subjects, hand error was significantly increased from late baseline to first learning 1, and reduced from first learning 1 to late learning 1 (all p-values < 0.001). Anticipatory force was not significantly different from late baseline to first learning 1 ($p = 0.380$), and significantly increased from first learning 1 to late learning 1 ($p < 0.001$). Planned comparisons at the late baseline, first learning 1, and late learning 1 phases revealed no significant differences between groups (all error p-values ≥ 0.101, all force p-values ≥ 0.268).

Transfer: To test for transfer of arm control, we focused on the late learning 1 and first learning 2 phases (Figure 5.2B). We found no differences between these phases or between groups. Hand error and anticipatory force did not significantly differ from late learning 1 to first learning 2 (error $p = 0.504$, force $p = 0.610$), and did not significantly differ from first learning 2 to late learning 2 (error $p = 0.402$, force $p = 0.341$). Planned comparisons at the first learning 2 and late learning 2 phases revealed no significant differences between groups (all error p-values ≥ 0.185, all force p-values ≥ 0.587).

5.3.4 Postural movement characteristics

We compared maximum lateral COP displacements during the experiment, in both wide and narrow stance, to those measured during the COP game (which established the lateral dimensions of the functional BOS). (All COP data was measured from the "start" location, and normalized by foot length.) This was done to verify that the COP movements executed during the experiment were within the limits of the functional BOS (see Figure 5.3A). Across all

subjects, mean foot length was 24.0 ± 2.0 cm, mean wide stance width was 38.6 ± 2.6 cm, and mean narrow stance width was 19.6 ± 1.3 cm. In the COP game, averaged across all subjects, the maximum normalized lateral displacement was 0.48 ± 0.09 cm/cm in wide stance and 0.26 ± 0.06 cm/cm in narrow stance, or a lateral functional BOS limit (measured from center) of 11.4 ± 1.6 cm in wide stance and 6.2 ± 1.4 cm in narrow stance; this was significantly different between groups ($p < 0.001$). In the experiment, maximum COP displacements did not exceed approximately 0.15 cm/cm, or 3.6 cm, in either group. In RPAs, COP displacements ranged from

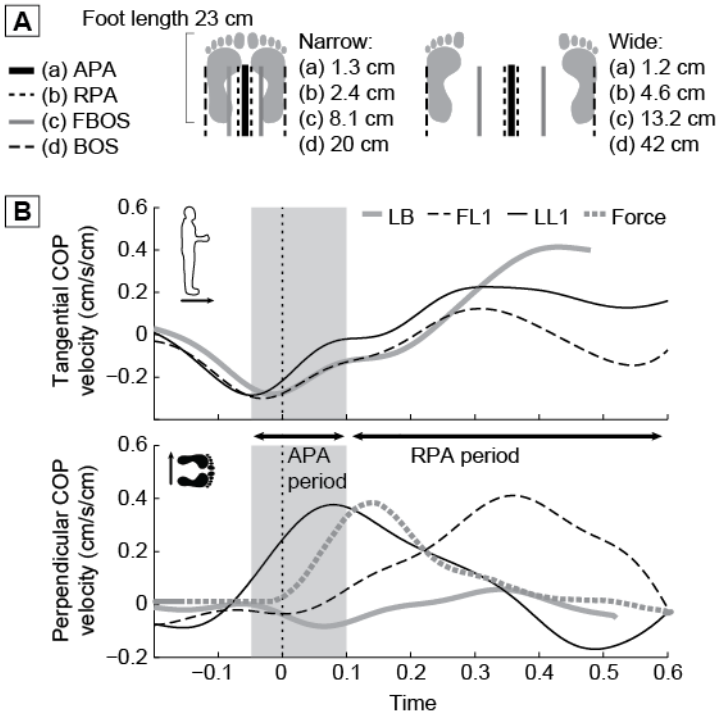

Figure 5.3. COP movements within postural base of support. (A) COP limits and movements for one representative subject: maximum lateral COP excursion observed during APA and RPA movements in the experiment; functional BOS (FBOS), measured as the maximum lateral COP excursion during the COP game; and BOS width for each stance width. (B) Representative tangential and perpendicular COP velocity traces (normalized by foot length) vs. time for the late baseline (LB), first learning 1 (FL1), and late learning 1 (LL1) phases, taken from one subject in the W group. Time zero represents movement onset of the arm. Note that for the perpendicular direction, positive is leftward (same direction as force field); for the tangential direction, positive is forward (same direction as reaching movement).

approximately 0.02 to 0.11 cm/cm in the W group, and 0.02 to 0.15 cm/cm in the N group; in APAs, they ranged from approximately 0.01 to 0.03 cm/cm in the W group, and 0.02 to 0.06 cm/cm in the N group. This confirms that the RPAs and APAs developed in response to the force field were well within the limits of the functional BOS for both stance widths.

APAs that developed to anticipate the force field were in the same direction as the field, 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 (Figure 5.3B); specifically, the COP moved away from the target prior to hand movement onset, as has been observed previously (Manista and Ahmed 2012). Across all subjects, the magnitude of the tangential APA did not significantly differ between phases (all p-values ≥ 0.198); and at all phases, magnitudes did not significantly different between groups (all p-values ≥ 0.263). Hereafter, all RPA and APA results are based on the perpendicular COP velocity.

5.3.5 Postural movement adaptation and transfer

COP movement data (Figure 5.4A) show that all subjects adapted to the field during the learning 1 trial block, transferred this adaptation to the learning 2 block, and de-adapted during the washout block. However, the groups showed differences in transfer. The ANOVA revealed a main effect of phase on both metrics (RPA $p < 0.001$, $F = 22.5$; APA $p < 0.001$, $F = 21.0$), with significant interaction effect of group x phase (RPA $p = 0.001$, $F = 4.4$; APA $p = 0.007$, $F = 3.4$), and with a main effect of group on RPA ($p = 0.019$, $F = 8.1$) but not on APA ($p = 0.087$, $F = 3.7$).

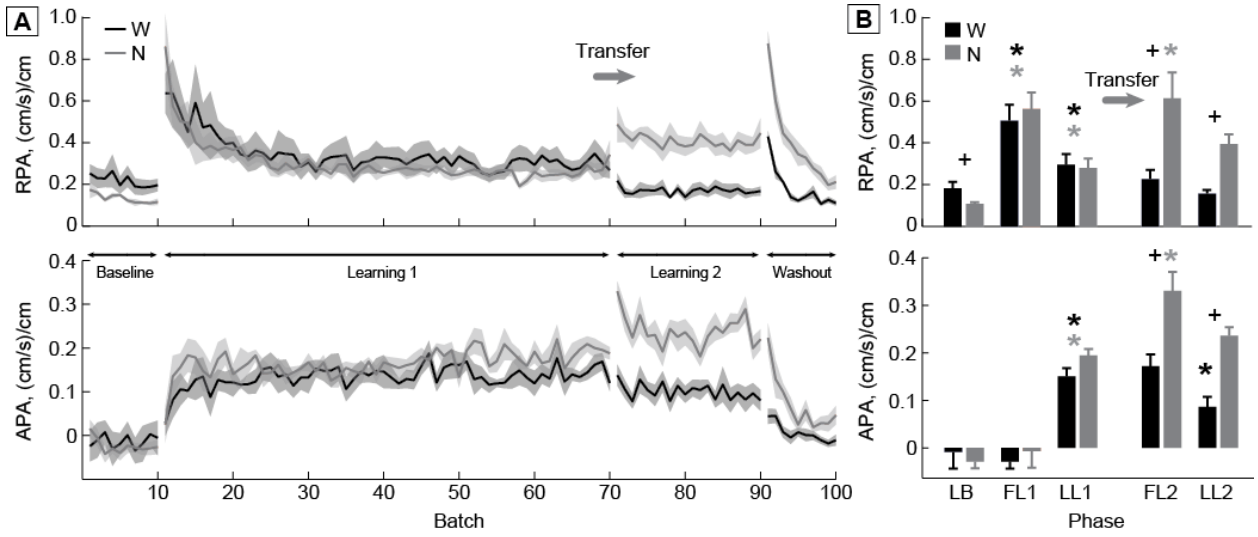

Figure 5.4. Adaptation of RPA and APA. (A) Upper and lower plots show RPA and APA, respectively, vs. batch (5 trials); in each plot, two traces show group means (solid lines) ± standard error (shading) for W group (black) and N group (gray). (B) Upper and lower bar charts show RPA and APA, respectively, at the late baseline (LB), first learning 1 (FL1), late learning 1 (LL1), first learning 2 (FL2), and late learning 2 (LL2) phases, for W group (black) vs. N group (gray), with error bars showing standard error. Statistically significant differences ($p < 0.050$) are denoted by (*) for a change from the previous phase (black for W group, gray for N group) or (+) between groups.

Adaptation: To test for adaptation of COP movements, we focused on RPAs and APAs in the late baseline, first learning 1, and late learning 1 phases (Figure 5.4B). We found that subjects adapted as expected, and the groups showed similar adaptation. For both groups, RPAs were significantly increased from late baseline to first learning 1 (W group $p = 0.010$, N group $p < 0.001$), and reduced from first learning 1 to late learning 1 (W group $p = 0.034$, N group $p = 0.017$). APAs were not significantly different from late baseline to first learning 1 (W group $p = 0.248$, N group $p = 0.406$), and significantly increased from first learning 1 to late learning 1 (all p-values < 0.001). Planned comparisons between groups showed that RPAs were higher for the W group than for the N group in late baseline ($p = 0.036$); otherwise, RPAs and APAs were not significantly different between groups in late baseline (APA $p = 0.512$), first learning 1 (RPA $p = 0.617$, APA $p = 0.583$), and late learning 1 (RPA $p = 0.789$, APA $p = 0.067$).

Transfer: To test for transfer of COP movements, we focused on the late learning 1 and first learning 2 phases (Figure 5.4B). We found that the W group transferred similar COP movements, but the N group did not; we also found differences between groups related to the amount of transfer. The W group transferred RPAs and APAs that were not significantly different from late learning 1 to first learning 2 (RPA $p = 0.108$, APA $p = 0.203$), but then decreased from first learning 2 to late learning 2 (RPA $p = 0.185$, APA $p = 0.039$), with both metrics in late learning 2 being significantly lower than in late learning 1 (RPA $p = 0.034$, APA $p = 0.041$). In the N group, RPAs and APAs significantly increased from late learning 1 to first learning 2 (RPA $p = 0.012$, APA $p = 0.040$), and then numerically decreased, although not significantly, from first learning 2 to late learning 2 (RPA $p = 0.062$, APA $p = 0.109$), with RPAs in late learning 2 remaining significantly higher than in late learning 1 ($p = 0.004$) and with APAs in late learning 2 not significantly different from late learning 1 ($p = 0.298$). (Note that the N group showed high inter-trial variability in late learning 2.) Planned comparisons at the first learning 2 and late learning 2 phases showed significantly higher RPAs and APAs in the N group than in the W group (first learning 2, RPA $p = 0.024$, APA $p = 0.018$; late learning 2, RPA $p = 0.004$, APA $p = 0.002$).

5.3.6 Anticipatory muscle activity

To determine which muscles were involved in the tangential APA related to the reaching movement, we focused on anticipatory muscle activity in the late baseline phase. As shown in Figure 5.5, the TA and PL muscles showed anticipatory activity bilaterally in this phase, and the soleus muscles showed inhibition, indicating a normal APA for forward reaching. This is consistent with previous findings (Leonard et al. 2009; Manista and Ahmed 2012).

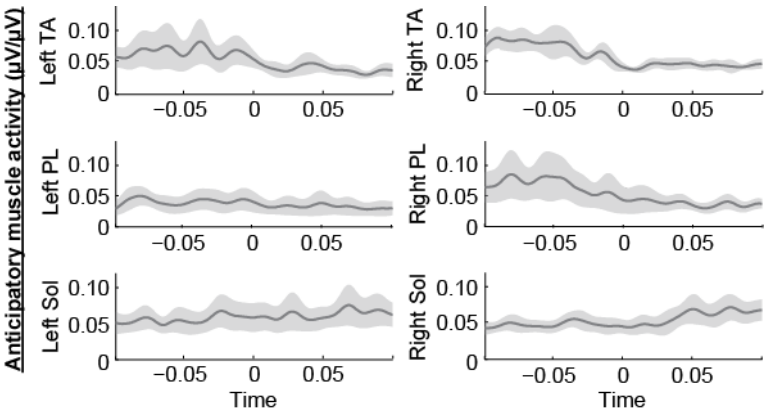

Figure 5.5. Baseline anticipatory muscle activity. Mean time traces of anticipatory muscle activity (normalized EMG) for the TA, PL, and soleus muscles in the late baseline (LB) phase for the N group. Each plot shows group average trace (solid line) ± standard error (shading). Time zero represents movement onset of the arm.

Anticipatory muscle activity data pertaining to adaptation and transfer are shown in Figure 5.6. Bar plots are included for all muscles which showed relevant differences (Figure 5.6A). Plots of anticipatory activity vs. batch (Figure 5.6B), as well as group average traces for the late baseline and late learning 1 phases (Figure 5.6C), are shown for the left and right TA, as these muscles are exemplary of the differences between groups and across phases. To examine adaptation and transfer of anticipatory muscle activity, we limited our focus to the late baseline, late learning 1, first learning 2, and late learning 2 phases.

Adaptation: To determine how muscle activity led to the changes in APAs after adaptation, we focused on changes in anticipatory muscle activity from late baseline to late learning 1 (Figure 5.6A,C). As expected, we found changes in anticipatory activity in the TA, PL, and soleus across all subjects, and changes in the TFL in the W group; also as expected, we observed higher levels of anticipatory activity in the N group. Across all subjects, anticipatory muscle activity from late baseline to late learning 1 was significantly decreased in the right TA ($p = 0.018$) and right PL ($p = 0.039$), and significantly increased in the right soleus ($p = 0.036$).

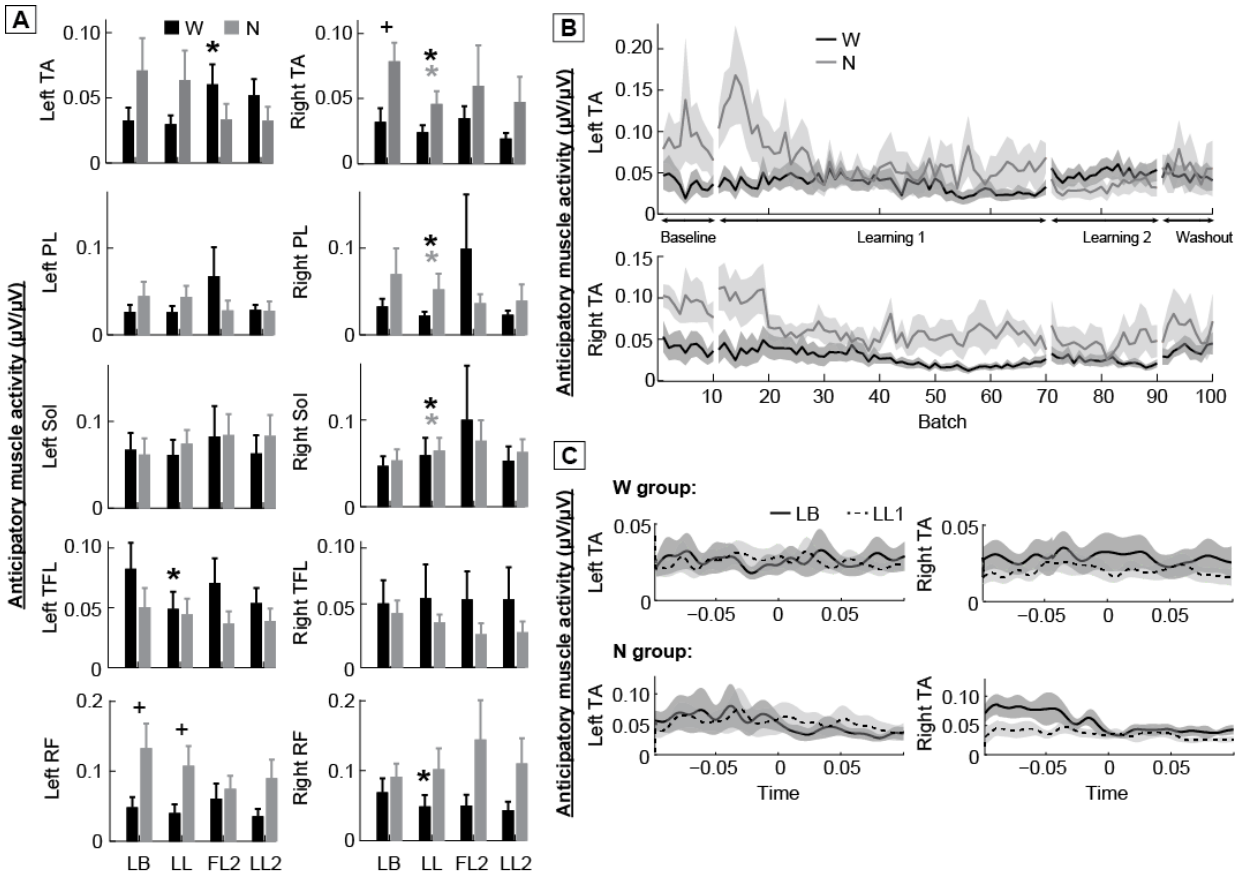

Figure 5.6. Adaptation of anticipatory muscle activity. (A) Bar charts show anticipatory muscle activity (mean normalized EMG) for muscles at the late baseline (LB), late learning 1 (LL1), first learning 2 (FL2), and late learning 2 (LL2) phases, for W group (black) vs. N group (gray), with error bars showing standard error. Statistically significant differences ($p < 0.025$) are denoted by (*) for a change from the previous phase (black for W group, gray for N group) or (+) between groups. (B) Batch plots show anticipatory muscle activity for the left and right TA vs. batch (5 trials); in each plot, two traces show group means (solid lines) ± standard error (shading) for W group (black) and N group (gray). (C) Mean time traces of anticipatory muscle activity for the left and right TA in the late baseline (LB) and late learning 1 (LL1) phases. In each plot, two traces show group mean for LB phase (solid line) and LL1 phase (dashed line) ± standard error (shading). Time zero represents movement onset of the arm.

The W group showed a significant decrease in left TFL activity ($p = 0.037$), and also showed a significant decrease in right RF activity ($p = 0.020$). Activity was higher for the N group than the W group in the right TA and left RF in late baseline (right TA $p = 0.016$, left RF $p = 0.036$) and late learning 1 (right TA $p = 0.052$, left RF $p = 0.038$); the same trends were also seen in the left TA and right RF, but with no significance. The BF muscles showed no significant differences across phases or between groups.

Transfer: To test for transfer of EMG strategies, we focused on changes in anticipatory muscle activity from late learning 1 to first learning 2 (Figure 5.6A). We found a significant change in anticipatory activity in the W group and no significant changes in the N group. In the W group, left TA activity significantly increased from late learning 1 to first learning 2 ($p = 0.024$) and right TA activity did not significantly change ($p = 0.405$), adding to the asymmetry caused by the decrease in right TA activity from late baseline to late learning 1. This increased left TA activity was maintained from first learning 2 to late learning 2, with late learning 2 significantly higher than late learning 1 ($p = 0.036$). Small, non-significant increases were also observed from late learning 1 to first learning 2 in the right PL ($p = 0.147$) and right soleus ($p = 0.190$), but that activity then decreased and was not significantly different between late learning 2 and late learning 1 (all p-values ≥ 0.423). In the N group, no significant changes in anticipatory muscle activity were observed from late learning 1 to first learning 2 (all p-values ≥ 0.112). However, activity slightly decreased in the left TA ($p = 0.068$) and left PL ($p = 0.056$), and then decreased further, with activity being significantly lower in late learning 2 than in late learning 1 (left TA $p = 0.042$, left PL $p = 0.043$).

5.4 Discussion

5.4.1 Summary

The results of this study partially support our hypothesis that anticipatory postural control, reflected in COP movement, is not affected by stance width, as long as the control remains within functional limits. Specifically, we found that adaptation of novel anticipatory postural control is not affected by stance width. However, the transfer of adapted anticipatory postural control between stance widths is affected by the biomechanical context of prior exposure.

5.4.2 Adaptation of postural control

The results confirmed our prediction that subjects in either wide or narrow stance would show similar COP movements by the end of the adaptation period. As expected, lateral functional BOS limits were increased from narrow to wide stance, and RPAs and APAs were well within these limits for both stance widths. Combined, these results indicate that the same COP strategy was sufficient for both stance widths and that adaptation of COP control was not affected by the difference in stance width.

While the N group did show non-significantly higher APAs than the W group in late learning 1, we interpreted this result as "similar" for several reasons. Firstly, the difference between group means in late learning 1 was small and statistically insignificant. More importantly, there is no reason to expect larger APAs in narrow stance, based on previous studies which found that anticipatory COP movements were reduced with smaller BOS size (Manista and Ahmed 2012; Kaminski and Simpkins 2001; Yiou et al. 2007; Chapter 4 of this dissertation).

The results also confirmed our prediction that due to the difference in biomechanical configuration between stance widths, the anticipatory muscle activity required to generate the same COP movement would differ. In late baseline and learning 1, activity levels were higher in the N group compared to the W group. Previous experimental studies found that in general, muscle activity in response to a perturbation is decreased with increasing stance width (Winter et al. 1998; Henry et al. 2001; Torres-Oviedo and Ting 2010). Initially, this was thought to be due to greater passive stiffness in wide stance, which would provide more passive control of COM movement and thus require less active muscle control (Henry et al. 2001). However, an experimentally validated model demonstrated that the body's frontal plane inertia actually decreases with wider stance, meaning that a given joint torque will generate larger COM movement in wider stance; therefore, to generate the same COM movement, joint torques (and muscle activity) must be decreased in wider stance (Bingham et al. 2011).

Both groups adapted their APAs in learning 1, specifically adapting APAs in a leftward direction, by reducing anticipatory muscle activity in the right TA and right PL and increasing activity in the right soleus. Results from several previous studies indicate similarly that higher activity in the left TA relative to the right TA will generate leftward COP movement (Hopkins et al. 2012; Gefen 2001). Anatomically, the PL acts directionally opposite the TA; however, we believe that here the TA muscles were activated as the primary controlling muscles to move the COP, and the PL muscles were activated to stabilize the ankles and keep the feet flat on the ground. This is supported by our data, which shows that changes in left and right PL activity accompany changes in left and right TA activity. Our finding of increased right soleus activity is supported by results from Leonard et al. (2009), who observed anticipatory activity in the right soleus for leftward arm-reaching movements.

Additionally, we found that the W group adapted by also reducing anticipatory activity in the left TFL and right RF. Similarly, Leonard et al. (2009) found that anticipatory activity decreased in the left TFL and increased in the right TFL as reach direction changed from forward to leftward. In a study of reactive postural control, Henry et al. (2001) found that left RF activity was associated with leftward COP movements; it follows that decreased activity in the right RF relative to the left RF should also be associated with leftward COP movements. We observed these strategies in the W group but not in the N group, which agrees with the idea that hip muscles are more effective in wide stance than in narrow stance. Bingham et al. (2011) demonstrated that hip muscle torques have greater leverage on the COM moment in wide stance than in narrow stance, and are therefore more effective in responding to a perturbation.

It is noteworthy that the batch plots for the TA muscles (Figure 5.6B) show that anticipatory activity increased bilaterally early in the learning 1 block; a similar pattern was seen in other muscle pairs as well. These increases occurred after the initial exposure trial (first learning 1), and may be indicative of a "stiffness" strategy using muscle coactivation to help reject the perturbation caused by the force field. Such a strategy has been observed in adaptation to novel dynamics in arm reaching (Katayama et al. 1998; Osu et al. 2002; Franklin et al. 2003b; Milner and Franklin 2005; Hinder and Milner 2007), and also in standing posture (Chapter 4 of this dissertation).

5.4.3 Transfer of postural control

We predicted that subjects would transfer similar COP movements from one stance to another, with appropriate changes in anticipatory muscle activity to account for the change in configuration. The fact that both groups showed similar APAs in late learning 1 confirms that

similar APAs were appropriate for either stance; therefore, this was a reasonable prediction. The W group did transfer similar COP movements, increasing their anticipatory muscle activity to account for the change to narrow stance, which confirms our prediction. However, contrary to our prediction, the N group significantly increased their COP movements upon transfer; this was related to a lack of sufficient modulation of muscle activity. To account for the change to wide stance and transfer similar postural control, a decrease in muscle activity would have been required. This group showed only small, non-significant changes in anticipatory muscle activity upon transfer; thus, the high levels of muscle activity caused an increase in APA magnitudes, and RPAs were also increased.

The observed transfer in the W group suggests that the postural control system can control for movement dynamics based on the known properties of the body and the environment, rather than learn to control a specific movement in the form of a specific muscle activation pattern. Previous studies have shown that subjects can control movement dynamics for arm reaching in unstable environments (e.g. divergent force fields) partially by taking advantage of the inherent mechanical properties of the arm relative to the properties of the environment. Trumbower et al. (2009) found that when reaching in unstable environments, subjects chose to reach using arm postures which maximized endpoint stiffness in the direction of environmental instability. This allowed subjects to minimize energetically costly muscle coactivation that is employed to increase endpoint stiffness and simultaneously provide stability when reaching in unstable environments (Burdet et al. 2001; Franklin et al. 2003a; Franklin et al. 2003b; Franklin et al. 2007). In a similar experiment with fixed arm postures, Krutky et al. (2010) found that subjects preferentially increased stretch reflexes to perturbations applied in the same direction as the environmental instability, only when the magnitude of the instability exceeded endpoint

stiffness in that direction. This showed that stretch reflexes were specifically modulated for the properties of the environment relative to the inherent properties of the arm. These prior results further support the idea that movements can be controlled based on the known dynamics of the body and the environment.

However, this behavior was not observed in the N group. What could be the potential mechanism underlying such asymmetric transfer? One possible explanation relates to the manner in which the task was initially adapted. It is possible that the N group initially adapted to the task in terms of muscle activity rather than COP movement. As this would result in identical COP movement, differences in the representation of the task would not emerge unless upon transfer. The fact that the N group transferred similar muscle activity to the wide stance width therefore suggests that this may reveal the hidden representation of motor adaptation. Other motor learning studies have found similar results. A study of adaptation to a visuomotor rotation found that adaptation in reaching transferred to walking, but transfer was not observed in the opposite direction, likely due to differences in how the visuomotor rotation was represented in the different contexts of walking and reaching (Morton and Bastian 2004). Another study demonstrated that object dynamics are represented along a continuum from object-space to muscle-space, with the least familiar objects represented in muscle-space (Ahmed et al. 2008). Thus, it is possible that the unfamiliarity or reduced BOS of narrow stance may have influenced the representation of the learned task.

There is another possible explanation for the observed asymmetric transfer. The N group may have simply chosen not to change their strategy because it was not required to maintain balance upon transfer to wide stance. Indeed, the larger COP movements observed in the N group upon transfer were acceptable within the larger functional BOS of wide stance. Even after

transfer, the N group did not significantly alter their control. These results may be indicative of "good enough" control strategies, as described by Loeb (2012); in the face of increased functional BOS limits upon transfer from narrow to wide stance, despite the fact that the transferred postural control was overly large and clearly non-optimal, that strategy persisted because it was "good enough." Similarly, de Rugy et al. (2012) found that habitual patterns of muscle coordination in wrist movements were robust to various physical and virtual manipulations of biomechanics, despite the fact that these habitual patterns were not optimal in the face of the altered biomechanics.

Results from a previous study suggest that postural control may be transferred differently between postural contexts, depending on whether the level of postural threat is increased or decreased. Jeka et al. (2008) investigated changes in the amplitude of compensatory postural sway in response to abrupt changes in visual environmental motion. They found that when an experimentally induced change in visual motion threatened balance, subjects responded rapidly with a compensatory change in postural sway to maintain upright stance. However, when the change in visual motion did not threaten balance, subjects responded more slowly, presumably because a rapid adjustment was not required. This is analogous to the behavior observed in the present study, where an increase or decrease in BOS size causes a change in the postural threat level. The W group experienced an increase in postural threat during transfer and immediately modulated their muscle control in order to maintain appropriate COP movements. In contrast, the N group experienced a decrease in threat and did not alter their muscle control, because their existing strategy was sufficient, or "good enough."

With regards to reactive postural control, several other studies have reported findings indicating that the initial postural context in which a task is performed can affect transfer to other

contexts. Horak & Nashner (1986) studied reactive postural control in response to a sagittal-plane platform perturbation in two different BOS conditions, with subjects standing on a beam that was wide or narrow in the direction of the perturbation. They found that subjects used a characteristic control strategy in each condition; interestingly, they also found that when subjects transferred from one condition to the other, they initially used an intermediate control strategy before adapting the characteristic strategy of the new condition. In two other studies, de Lima-Pardini et al. (2012) and Papegaaij et al. (2012) studied the effects of voluntary task stability constraints on reactive postural control. Subjects stood on a platform and held a tray (voluntary task) with a half-cylinder placed flat side down (low stability constraint) or round side down (high stability constraint), and were perturbed with a backward surface translations (postural task). They found that the constraint condition (low or high stability) in the initial trial block affected transfer of postural control strategy to subsequent trial blocks. In these studies it was suggested that subjects chose to use their prior postural control strategy in the new context, where the prior strategy remained "good enough," rather than generate a new control strategy, which would require more attention.

An intriguing implication of the present study is that the postural control system may not always choose to control for movement dynamics or have the ability to do so, as when the N group failed to modulate their muscle activity in transfer. An interesting question for future investigation is what might drive this change in strategy and whether it represents a control choice or a constraint. An extension of this experiment could also provide further insight; if subjects transferred back to their original stance, after transferring from wide to narrow or from narrow to wide, would we observe a similar pattern of asymmetry between the groups? Or would subjects simply revert back to the adapted control patterns that they used in late learning 1?

5.4.4 Effects of BOS size, uncertainty, and threat on postural control

Several earlier studies investigated the effects of postural BOS size on APAs, and reported that APAs were reduced in the direction of smaller BOS size and/or increased in the direction of larger BOS size. However, for various reasons, these studies did not clearly demonstrate that APAs were affected by BOS size alone. In a load-release task performed while standing on a wobble board, Aruin et al. (1998) found that anticipatory muscle activity was reduced with narrower beam widths. However, COP movements may have been biomechanically constrained by the very small BOS of the wobble boards. In the present study, we ensured that both anticipatory and reactive COP movements were not biomechanically constrained, but were well within the functional BOS for both stance widths. Kaminski and Simpkins (2001) asked subjects to make forward-reaching arm movements to a target while standing normally or with one foot placed farther forward (thus extending BOS in that direction); they found that anticipatory COP movement amplitude was increased in the foot-forward condition. Similarly, Yiou et al. (2007) also asked subjects to make forward-reaching arm movements, while standing with their two feet perpendicular to each other, and BOS was varied by increasing the distance between the heels in the forward direction; they also found that anticipatory COP movement amplitude was increased in the direction of the extended BOS. However, Yiou et al. further reported that the velocity of the focal reaching movement was also increased with extended BOS size, which itself would require an increased anticipatory COP movement, thus obscuring the effect of BOS size on COP movements alone. In the present study, we ensured that the characteristics of the focal arm movement were similar between stance widths, and thus the same APA could be expected. Our adaptation results clearly show that anticipatory postural control is

not affected by BOS size, even in a novel task, as long as the COP movement remains within the functional BOS.

Our adaptation results help to further explain the findings of a recent study by Manista and Ahmed (2012), as well as findings from Chapter 4 of this dissertation. Both studies involved a force field adaptation experiment, where subjects made reaching movements while standing. Subjects adapted to a curl field similar to that used in this study, with force field perturbations in the forward vs. backward directions. While the same magnitude of APA was required for a forward vs. a backward perturbation, and the required APA was within the BOS in both directions, both studies found that APAs were reduced in the backward direction. However, it was not possible to determine whether the reduced APAs resulted from the reduced length of the BOS in the backward direction, or the increased threat associated with a recovery step in the backward direction compared to a step in the forward direction. Another previous study demonstrated that the cost, or threat, associated with an error could indeed modify adaptation, independent of the magnitude of the error (Trent and Ahmed 2013). Taken together, the results of Trent and Ahmed (2013) and the present findings suggest that the reduced APAs observed by Manista and Ahmed (2012) and reported in Chapter 4 of this dissertation were not due to the reduced BOS length, but rather due to the increased threat associated with backward perturbations. In the present study, we found no differences in adapted APAs between stance widths, because the BOS size of even the narrow stance is sufficiently large that it is not inherently threatening to healthy young adults.

Other studies have shown that APAs are reduced in conditions of increased threat or uncertainty. Adkin et al. (2002) asked subjects to stand on a platform and rise to their toes; they found that anticipatory COP movement amplitude and velocity were reduced with greater threat,

e.g. when subjects stood on a high vs. a low platform, where the potential consequences of an incorrect APA are greater. Toussaint et al. (1998) asked subjects to lift several boxes repeatedly; they found that anticipatory COP movement amplitude was reduced when the boxes had a less predictable weight (identical boxes of different masses), i.e. when subjects could not accurately predict the required APA and were therefore more likely to make an inappropriate APA that would require a corrective control action. We suggest that in such cases, subjects choose to compensate for the increased threat or uncertainty by decreasing their anticipatory COP movements and thus maintaining a safe stability margin within their existing BOS (Koozekanani et al. 1980).

In the present study, our transfer results support this idea as well. When the W group transferred to narrow stance, they initially modulated their muscle control in order to maintain appropriate COP movements, and then chose to reduce their COP movements, avoiding their functional BOS limits. Interestingly, reduced COP movements in narrow stance were not observed in the subjects who initially adapted in a narrow stance (N group). We suggest that this strategy emerged only after transfer from wide to narrow stance because of the increase in postural threat. It is also notable that RPAs were greater for the N group in wide stance (late learning 2) than for the W group in wide stance (late learning 1), indicating that the N group tolerated larger COP movements after they transferred to wide stance than the W group initially tolerated in wide stance. This suggests that for the N group, excessively large COP movements were acceptable after changing to wide stance due to the decrease in postural threat.

5.4.5 Implications for training and rehabilitation

Our results demonstrate that the postural context in which initial adaptation or training occurs can influence transfer to other contexts. Assuming that the control strategies which were adapted at the end of learning 1 were appropriate strategies for this dynamic task, it would appear that the W group was better at transferring their adapted control to the second stance, due to the fact that the N group, in contrast, showed excessively large COP movements (both RPAs and APAs) as well as high variability in learning 2. This would seem to indicate that it is beneficial to train in a less challenging context. Similarly, Wulf et al. (1998) found that when learning to use a ski simulator, subjects performed better when they trained with ski poles for increased support; furthermore, in subsequent practice sessions without poles, better performance was seen in subjects who had trained initially with poles than in those who had trained initially without poles. However, another study reported differing results. Domingo and Ferris (2009) found that when subjects were trained to walk on a balance beam, either wearing a stabilizing harness or not, performance improvements were greater for subjects who trained without the harness; this result would seem to indicate that it is beneficial to train in a more challenging context.

Taken together with the results of the present study, these findings suggest that in training and rehabilitation, it is important to consider the postural context in which task learning or re-learning occurs, as well as the context in which the task will be performed in the future. However, it remains unclear whether it is beneficial for initial training to take place in a more challenging or less challenging context. Furthermore, these findings demonstrate that the postural context of initial training can influence transfer in healthy young adults; future research directions should expand to include clinical populations.

5.4.6 Conclusions

The results of this study demonstrate that initial adaptation of anticipatory postural control, reflected in COP movement, is not affected by stance width. However, transfer of COP control to another stance width is affected by the context of prior exposure. Generally, these results support the idea that the context in which a task is initially introduced should be taken into consideration, as it can have an effect on the transfer or generalization of the adapted control strategy.

CHAPTER 6

INTERLIMB TRANSFER OF POSTURAL ADAPTATION

6.1 Introduction

An extant question in the field of motor control is how the central nervous system represents and coordinates the concurrent adaptation of movement and posture. Previous studies have investigated concurrent adaptation of arm reaching and standing postural control, using an experimental paradigm in which subjects make arm reaching movements while holding the handle of a robotic manipulandum which can generate perturbing forces. With repeated exposure to a given perturbation, subjects learn to anticipate the perturbation and adapt both their arm control and their postural control to compensate (Ahmed and Wolpert 2009; Manista and Ahmed 2012; Chapters 4 and 5 of this dissertation). These studies showed specific differences in adaptation between the two modalities, suggesting that adaptation occurs via a similar but independent mechanism in each form of movement. Ahmed and Wolpert (2009) also showed that after subjects adapted their arm reaching movements to novel dynamics while sitting, they were able to generate appropriate postural control immediately upon standing. This indicates that the postural control system can use information about arm movement dynamics to plan appropriate postural control, even though the postural system did not directly experience the novel dynamics during adaptation. However, in cases where the perturbation is directly experienced by both arm and posture, it is not clear whether the postural control system is

dependent on information about arm movement dynamics, or whether it can adapt to and controls for novel dynamics independently of arm control.

In this study, therefore, we sought to determine whether postural control is adapted and transferred independently of arm control. Subjects adapted their arm and postural control to a novel force field while standing and reaching with their dominant (right) arm, and then switched to standing and reaching in the same force field with their non-dominant (left) arm. While reaching with the right or left arm clearly required different control, the postural control required to counter the perturbation was similar.

The question of whether learned dynamics transfer from the right to the left arm, or vice versa, has received significant attention. Two previous studies of arm reaching adaptation found that when subjects adapted their arm reaching movements (while seated) to an "abrupt" perturbation, where the force field was experienced at a constant strength throughout the adaptation period, the adapted arm control was transferred from the dominant to the non-dominant arm (Criscimagna-Hemminger et al. 2003; Malfait & Ostry 2004). However, when subjects experienced a "gradual" perturbation, where the force field gradually increased in strength from zero over many trials, they showed no transfer from the dominant to the non-dominant arm (Malfait & Ostry 2004). So an "abrupt" introduction to the force field leads to transfer of learning from right to left in seated reaching movements, but a "gradual" introduction does not. In the present study, we leverage these findings to probe the dependence of postural control on arm control.

We tested two groups of subjects, who adapted their arm and postural control to a novel force field with either an abrupt or gradual development. We then examined the arm and postural behavior upon initial transfer to the non-dominant arm, in order to gain insight into whether

postural control can be adapted and transferred independently of arm control. When subjects switched arms, they experienced the same force field and a nearly identical postural perturbation; thus, the change in reaching arm should not affect the net postural perturbation or the associated postural control. Based on this, we formed two separate hypotheses about how subjects would transfer their adapted control. First, if the postural control system can independently adapt to, anticipate, and control for the novel dynamics, there should be no change in the perturbation-specific postural control when subjects switch arms, regardless of whether or not the adapted arm control is transferred. Conversely, if the gradual group shows no transfer of either arm or postural control, this may support the hypothesis that in this task the postural control system is not only *able* to generate predictive control based on information from the arm, but that planning is also *dependent* on information about the planned arm movement.

This paradigm also allows us to address the question of how error size affects adaptation in both arm and posture. In general, adaptation of arm reaching movements is found to increase with error and/or perturbation size (Fine and Thoroughman 2007; Herzfeld et al. 2014; Marko et al. 2012; Scheidt et al. 2001; Trent and Ahmed 2013; Wei and Kording 2010). Interestingly, results from an earlier study (Chapter 3 of this dissertation) suggested that people do not adapt their postural control in response to small errors. However, in that experiment people experienced randomly varying perturbations, which resulted in some degree of uncertainty, and may have confounded the results. In the present study, we tested this using the gradual perturbation paradigm. Here subjects experienced very small but consistent errors, which theoretically should lead to less uncertainty. Thus we hypothesized that posture can be adapted in response to very small errors, if those errors are consistently biased so as to minimize uncertainty.

Importantly, the results of this study demonstrate the extent to which postural control is informed by the planned arm control in a concurrent reaching task. This gives us information about how postural control is coordinated with concurrent movement tasks, and can provide greater insight into the underlying mechanisms of whole-body movement planning.

6.2 Methods

6.2.1 Subjects

Fourteen healthy young adult subjects (age 24.6 ± 5.1 years; height 171.7 ± 11.5 cm; mass 68.9 ± 12.4 kg; 7 male, 7 female) 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.

6.2.2 Experimental apparatus and setup

Subjects made forward reaching movements in the horizontal plane 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.) (Figure 6.1A). The subject's forearm was supported against gravity by a rigid cradle attached to the handle (separate right- and left-handed cradles were used during reaching with right and left arms). 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 127.7 ± 7.4 cm across subjects). 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. A computer monitor, vertically suspended in front of the subject, displayed visual feedback of hand, start, and target positions throughout the movement.

Before the experiment began, a "start" circle and a cursor representing center of pressure (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 reaching movements straight ahead (+*y*), using the robot handle to control the cursor on the screen. At the start of each trial,

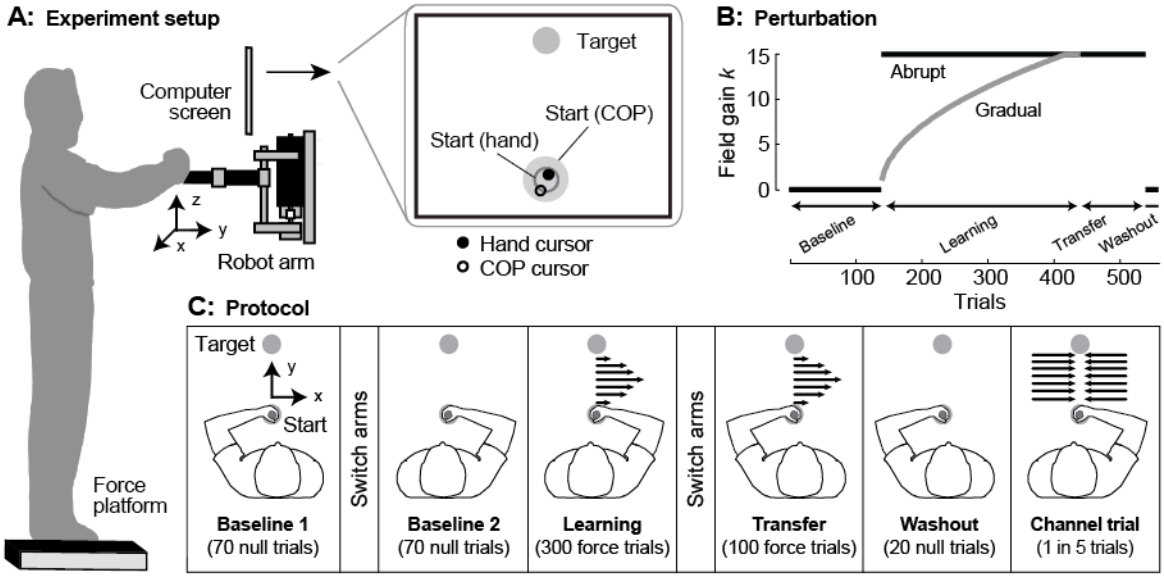

Figure 6.1. Interlimb transfer experiment setup and protocol. (A) Experimental apparatus and setup; visual feedback is provided on computer screen. (B) Force field gain *k* vs. trial; subjects experienced either an abrupt (A) or gradual (G) perturbation during the learning block. (C) Experimental protocol.

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 (Figure 6.1A). 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 duration window of 450 to 550 ms.

6.2.3 Experimental protocol

The protocol consisted of 560 trials and was divided into five consecutive blocks: baseline 1 (70 null trials, left arm), baseline 2 (70 null trials, right arm), learning (300 force trials, right arm), transfer (100 force trials, left arm), and washout (20 null trials, left arm) (Figure 6.1C). The baseline blocks 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 de-adapt the previous dynamic environment. The learning block consisted of force trials, in which a viscous curl field was simulated such that the robot exerted 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, with field strength dependent on the gain k (Equation 6.1). Thus, for a forward reaching

movement (in the anterior direction, $+y$) and a positive value of k, the robot generated rightward perturbing forces ($+x$).

$$\begin{bmatrix} F_x \\ F_y \end{bmatrix} = k \begin{bmatrix} 0 & -1 \\ 1 & 0 \end{bmatrix} \begin{bmatrix} V_x \\ V_y \end{bmatrix} \quad (6.1)$$

Subjects were randomly assigned into one of two groups, "Abrupt" or "Gradual," with N = 7 per group. In the learning block, the force field will be introduced in either an abrupt or gradual manner, depending on group (Figure 6.1B). The Abrupt group experienced force trials with a constant gain of k = 15 N-s/m. For the Gradual group, force field gain k was increased gradually over the first 280 learning trials, with the remaining learning trials at the maximum field strength of k = 15 N-s/m. The gradual increase in gain is dictated by the equation $k = t^a$, where t is learning trial number (excluding channel trials) and $a = \log(15)/\log(T)$; 15 is the maximum final value of k, and T is the learning trial (excluding channel trials) at which k reaches its maximum (Kluzik et al. 2008; Malfait & Ostry 2004).

The number of trials in each block was chosen based on data from pilot testing, such that subjects had sufficient practice in the baseline, learning, and transfer blocks that movement metrics reached a near-steady state by the end of each block. In addition, we wished to ensure that the learning block would be long enough that subjects in the Gradual group would be unable to perceive the gradual increase in field force. However, these factors were also balanced with the desire to minimize the total number of trials, to avoid excessive fatigue due to standing for a long period of time. The parameters of the reaching movement were similar to those used in previous experiments (Chapters 3, 4, and 5 of this dissertation). The maximum value of the force field gain (k = 15 N-s/m) was smaller than that used in previous experiments, in order that

subjects in the Gradual group would be unable to perceive the gradual increase in field force. This was also determined in pilot testing.

Every trial block began with a null or force trial, immediately followed by a channel trial, with the exception of the transfer block; in that block, the first trial was a channel trial. In the rest of each block, one trial in every batch (5 trials) was chosen randomly to be a channel trial. Channel 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 which the subject was exerting into the channel. Stiffness and damping for the channel were 2000 N/m and 50 N-s/m, respectively. These trials have been shown to have a minimal effect on adaptation or de-adaptation (Scheidt et al. 2000). 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 (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-degree circle at 45-degree 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).

6.2.4 Data collection and analysis

Position, velocity, and force data from the robot handle were sampled at 200 Hz. Center of pressure (COP) position data was 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 foot length (mean 25.7 ± 2.2 cm across subjects).

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 50 ms prior to when tangential hand position and velocity exceed threshold values of 0.25 cm and 2 cm/s, respectively. 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. Data from channel trials were analyzed separately from all other trials. 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 was corrupted. A total of 8 trials were rejected, out of the entire data set, with 5 trials excluded for the Abrupt group and 3 trials for the Gradual group (out of

3920 total trials per group, with 560 trials per subject). These were all channel trials. On average, less than one trial was rejected per subject.

Arm control: Arm control 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 handle 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. As a measure of forces experienced in the force field, we quantified field force as the peak signed value of perpendicular force (exerted by the robot arm) on force trials.

Postural control: Postural control was quantified for each trial, excluding channel trials, using three COP movement metrics: reactive postural adjustment (RPA_d) based on COP displacement (a measure of postural error), reactive postural adjustment (RPA_a) based on COP acceleration (another measure of postural error), and anticipatory postural adjustment (APA) based on COP displacement (a measure of anticipatory control). All of these metrics were based on the normalized COP displacement or acceleration in the direction of the force perturbation (perpendicular to the direction of reaching movement). We observed that COP velocity responses on force trials 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, APA was calculated as the peak signed value of COP displacement observed between 50 ms before movement onset and 100 ms after movement onset. Over the remaining duration of the movement (following the APA time period), RPA_d was calculated as

the peak signed value of COP displacement, and RPA_a was calculated as the peak value of COP acceleration.

RPA_d, and similar metrics using COP velocity, are typical measures of postural error used in this experimental paradigm, as seen in previous chapters in this dissertation, and also in earlier studies (Ahmed and Wolpert 2009; Manista and Ahmed 2012); however, by the end of learning, these metrics remain elevated due to the fact that subjects learn to anticipate a perturbation by initiating COP movement near arm movement onset, and that COP movement is then propagated into the reactive portion of the movement. Therefore, we included the additional RPA_a metric as a way to measure the quickness of the COP movement. When subjects experience a large perturbation unexpectedly, COP acceleration will reflect the quickness of their reactive COP movement made in response to the perturbation. However, if subjects are anticipating the perturbation, COP acceleration and thus RPA_a will remain low.

6.2.5 Statistics

Data were compared between groups and across 8 phases of the protocol: late baseline 1 & 2 (LB1, LB2), first learning (FL), late learning (LL), first transfer (FT), late transfer (LT), first washout (FW), and late washout (LW). The "first" 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. One exception is that for both anticipatory force and APA, the first transfer phase consisted of the first trial (channel trial) in the transfer block. For all metrics, the "late" phases consisted of the last 4 batches (16 null or force trials, or 4 channel trials) of the trial block.

Hand error, anticipatory force, RPA_d, RPA_a, and APA data were analyzed using repeated-measures ANOVAs, with phase as a within-subjects factor and group as a between-subjects factor. To test for adaptation during right-handed reaching, we made planned comparisons on the within-subjects results for each group between the late baseline 2, first learning, and late learning phases. To compare adaptation between groups, we made a planned comparison in the late learning 1 phase. To test for initial transfer of adaptation to left-handed reaching, we made planned comparisons on the within-subjects results for each group between the late learning and first transfer phases, and between the late baseline 1 and first transfer phases. To examine the time course of transfer, we made additional planned comparisons on the within-subjects results for each group between the first transfer and late transfer phases. Planned comparisons were also made between groups at specific phases of interest. We used paired t-tests for within-subjects planned comparisons between phases, and independent two-sample t-tests for planned comparisons between groups.

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

6.3 Results

6.3.1 Overview

Both groups adapted their arm and postural control as expected while reaching with the right arm (Figure 6.2). When initially exposed to the force field, the Abrupt group showed large

rightward movement errors, in the same direction as the perturbing forces, but the Gradual group did not. With practice, however, both groups increased their anticipatory control to counter the force field. Despite the difference in the abrupt vs. gradual force field introduction, and the corresponding differences in initial movement errors, both groups showed similar adaptation of arm and postural control by the end of learning. In transfer, neither group showed evidence of

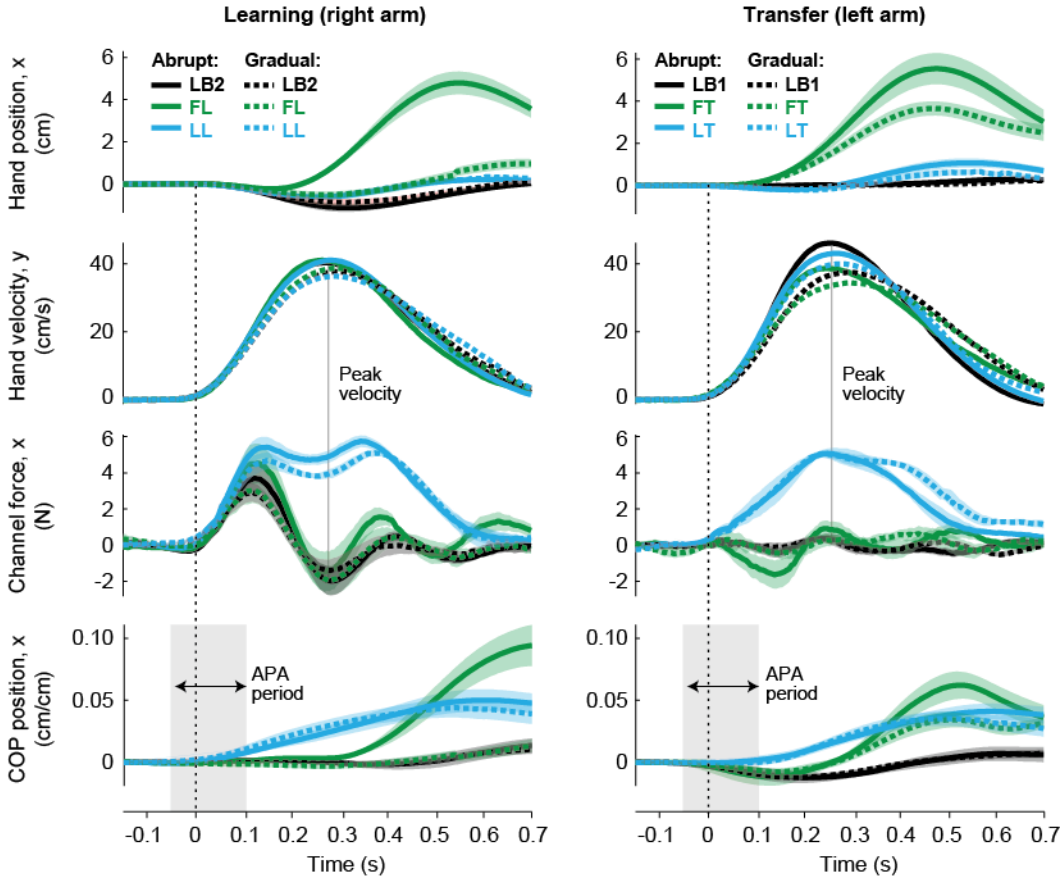

Figure 6.2. Group mean trajectories in learning and transfer. Group mean trajectories for perpendicular hand position (force trials only), tangential reaching velocity and channel force (channel trials only), and perpendicular COP position (force trials only). Left plots show data for right-handed reaching phases: late baseline 2 (LB2), first learning (FL), and late learning (LL). Right plots show data for left-handed reaching phases: late baseline 1 (LB1), first transfer (FT), and late transfer (LT). Note: Trajectories were averaged across trials in each phase for each subject, then averaged across subjects in each group. Shading indicates standard error across subjects. Time zero indicates movement onset of the arm.

transferring the adapted control from the right to the left arm, but both groups showed similar additional adaptation by the end of the transfer block. This is illustrated in the transfer trajectories of Figure 6.2, where upon switching to the left arm, both groups initially showed anticipatory control similar to late baseline and showed large rightward movement errors, but with practice they increased their anticipatory control.

Results are presented below for adaptation and transfer of arm control (hand error and anticipatory force) and postural control (RPA_d, RPA_a, and APA), as well as movement characteristics (reaching velocity, field force, and COP displacements).

6.3.2 Arm movement characteristics

To compare performance between groups, we had to be sure that both groups made similar hand reaching movements, and that they experienced similar forces in late learning and in the transfer block. We first confirmed that hand error and anticipatory force were similar between groups in late baseline 1 and late baseline 2 (all p-values ≥ 0.281). In the first learning phase, peak velocities were similar between groups ($p = 0.814$); as expected, peak field forces were significantly different ($p < 0.001$) due to the difference in field gains. In late learning, peak velocities and field forces were similar between groups (both p-values ≥ 0.289). In the first transfer phase, peak velocities and field forces in the force trial (second trial in transfer block) were significantly lower in the Gradual group compared to the Abrupt group ($p = 0.018$), despite the same field gain. However, in the channel trial, which was the very first trial in the transfer block, peak velocities were similar between groups ($p = 0.202$); therefore, there should be no difference in the magnitude of the anticipated perturbation (proportional to reaching velocity). In

late transfer, peak velocities and field forces were similar between groups (both p-values ≥ 0.360).

6.3.3 Arm adaptation and transfer

Hand error and anticipatory force data (Figure 6.3) show that both groups adapted to the field with the right arm during the learning block. Interestingly, *both* groups then failed to transfer this adaptation to the left arm initially, but subsequently showed further adaptation with the left arm throughout the rest of the transfer block. For hand error, the ANOVA revealed main effects of phase ($p < 0.001$, $F = 148.54$) and group ($p < 0.001$, $F = 80.32$), as well as an interaction effect of phase x group ($p < 0.001$, $F = 13.35$). For anticipatory force, the ANOVA revealed a main effect of phase ($p < 0.001$, $F = 75.54$), but did not show a main effect of group ($p = 0.106$, $F = 3.06$) or an interaction effect ($p = 0.465$, $F = 0.96$).

Adaptation: To test for movement adaptation in the right arm, we focused on hand error and anticipatory force in the late baseline 2, first learning, and late learning phases (Figure 6.3A). In the Abrupt group, hand error significantly increased from late baseline 2 to first learning and then decreased from first learning to late learning (both p-values < 0.001). In the Gradual group, hand error did show a relatively very small, but significant, increase from late baseline 2 to first learning ($p < 0.001$); however, hand error then showed no change from first learning to late learning ($p = 0.429$). Anticipatory force was significantly different from late baseline 2 to late learning in both groups (both p-values < 0.001). To compare adaptation of arm control between groups, we made planned comparisons between the changes in hand error and anticipatory force from late baseline 2 to late learning (Figure 6.3B). These changes were similar between groups

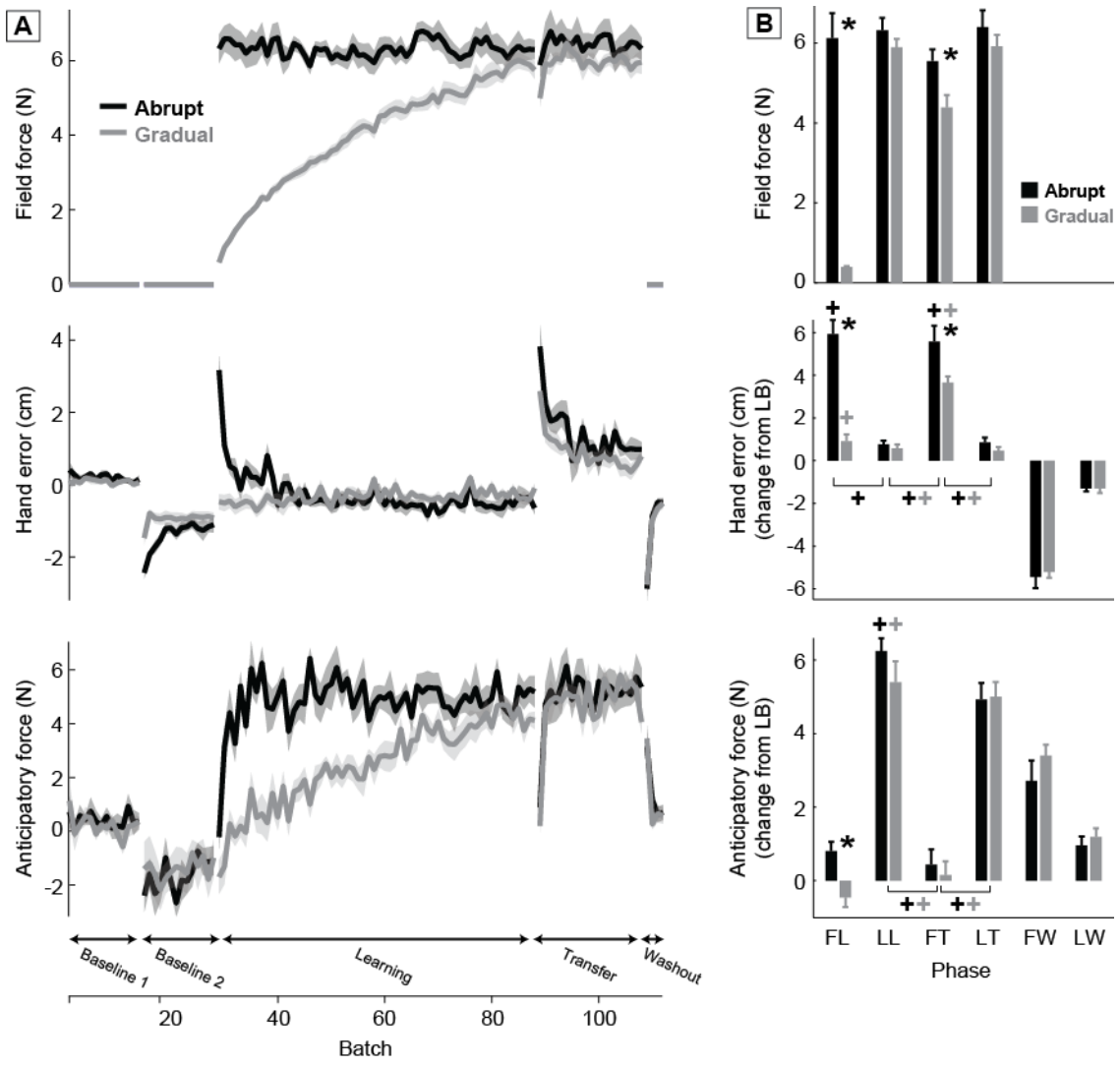

Figure 6.3. Adaptation and transfer of arm control metrics. Upper, middle, and lower plots show peak field force, hand error, and anticipatory force, respectively. Data for Abrupt group is black; Gradual group is gray. (A) Left plots show each metric vs. batch (5 trials); in each plot, two traces show group means (solid lines) ± standard error (shading). (B) Right bar plots show the change from respective late baseline (LB) to first learning (FL, from LB2), late learning (LL, from LB2), first transfer (FT, from LB1), and late transfer (LT, from LB1), with error bars showing standard error. (Because field force is zero in baseline, these are the actual peak force values.) Statistically significant differences between groups ($p < 0.050$) are denoted by (*). (+) denotes a statistically significant difference within group in change from respective baseline, or between other phases where indicated ($p < 0.050$).

(hand error $p = 0.473$; anticipatory force $p = 0.227$), indicating that both groups adapted similarly in the right arm by the end of the learning block.

Transfer: To test for transfer of adapted arm control from the right to the left arm, we focused on the late baseline 1, late learning, and first transfer phases (Figure 6.3A). From late learning to first transfer, both groups showed significant changes in anticipatory force and hand error (all p-values < 0.001). From late baseline 1 to first transfer they showed no significant change in anticipatory force (both p-values ≥ 0.342) and a significant increase in hand error (both p-values < 0.001), indicating that when subjects switched to the left arm they used arm control that was similar to left-handed baseline; they did not anticipate the perturbation in the left arm and thus experienced large errors as a consequence. Subsequently, both groups adapted to the perturbation in the left arm, showing significant decreases in hand error and significant increases in anticipatory force from first transfer to late transfer (all p-values < 0.001). To compare transfer between groups, we made planned comparisons between the changes in hand error and anticipatory force from late baseline 1 to first transfer and late transfer (Figure 6.3B). The changes in anticipatory force from late baseline 1 to first transfer were similar between groups ($p = 0.628$), indicating that both groups showed a similar lack of transfer. The Gradual group did show a smaller change in hand error from late baseline 1 to first transfer, compared to the Abrupt group ($p = 0.035$); however, this is likely due to the fact that the Gradual group reached with a smaller peak velocity and experienced a smaller peak field force than the Abrupt group ($p = 0.018$). The changes in hand error and anticipatory force from late baseline 1 to late transfer were similar between groups (both p-values ≥ 0.184), indicating that both groups adapted similarly in the left arm by the end of the transfer block.

6.3.4 Postural movement characteristics

We compared maximum lateral COP displacements during the experiment, in both groups, to those measured during the COP game (which established the lateral dimensions of the functional BOS). (All COP data was measured from the "start" location, and normalized by foot length.) This was done to verify that the COP movements executed during the experiment were within the limits of the functional BOS. Across all subjects, mean foot length was 25.7 ± 2.2 cm. In the COP game, averaged across all subjects, the maximum normalized lateral displacement was 0.42 ± 0.12 cm/cm, or a lateral functional BOS limit (measured from center) of 10.8 ± 2.9 cm. In the experiment, maximum lateral COP displacements in the Abrupt group were 0.12 ± 0.02 cm/cm (normalized), or 3.1 ± 0.5 cm; in the Gradual group they were 0.08 ± 0.03 cm/cm (normalized), or 2.2 ± 0.5 cm. COP displacements in the experiment did not meet or exceed the limits of the functional BOS in any subject. These results confirm that for both groups, COP movements developed in response to the force field were well within the limits of the functional BOS.

APAs that developed to anticipate the force field 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 prior to hand movement onset, as has been observed previously (Manista and Ahmed 2012; Chapters 3-5 of this dissertation). 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 COP displacement, 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). Across all subjects, the magnitude of the tangential APA showed no significant differences between phases (all p-values ≥ 0.053), except for a reduction in APA magnitude in both groups from late learning to first transfer (Abrupt, $p = 0.036$; Gradual, $p = 0.049$). However, this was likely related to the drop in peak tangential reaching velocity that was observed concurrently (Abrupt, $p = 0.087$; Gradual, $p = 0.002$). Magnitudes did not significantly differ between groups at any phase (all p-values ≥ 0.072). Hereafter, all RPA and APA results are based on perpendicular COP movement.

6.3.5 Postural adaptation and transfer

RPA and APA data (Figure 6.4) show that both groups adapted their postural control to the field during the learning block, when reaching with the right arm; both groups then failed to transfer this adaptation when they switched to the left arm initially, but subsequently showed further adaptation throughout the rest of the transfer block. For RPA_d, the ANOVA revealed main effects of phase ($p < 0.001$, $F = 28.04$) and group ($p = 0.016$, $F = 7.91$), as well as an interaction effect of phase x group ($p < 0.001$, $F = 5.39$). For RPA_a, the ANOVA revealed a main effect of phase ($p < 0.001$, $F = 8.31$), but did not show a main effect of group ($p = 0.328$, $F = 1.04$) or an interaction effect ($p = 0.328$, $F = 1.17$). For APA, the ANOVA revealed a main effect of phase ($p < 0.001$, $F = 8.97$), but did not show a main effect of group ($p = 0.935$, $F = 0.01$) or an interaction effect ($p = 0.947$, $F = 0.31$).

Adaptation: To test for postural adaptation while reaching with the right arm, we focused on RPA_d, RPA_a, and APA in the late baseline 2, first learning, and late learning phases (Figure 6.4A). In the Abrupt group, RPA_d and RPA_a significantly increased from late baseline 2 to first

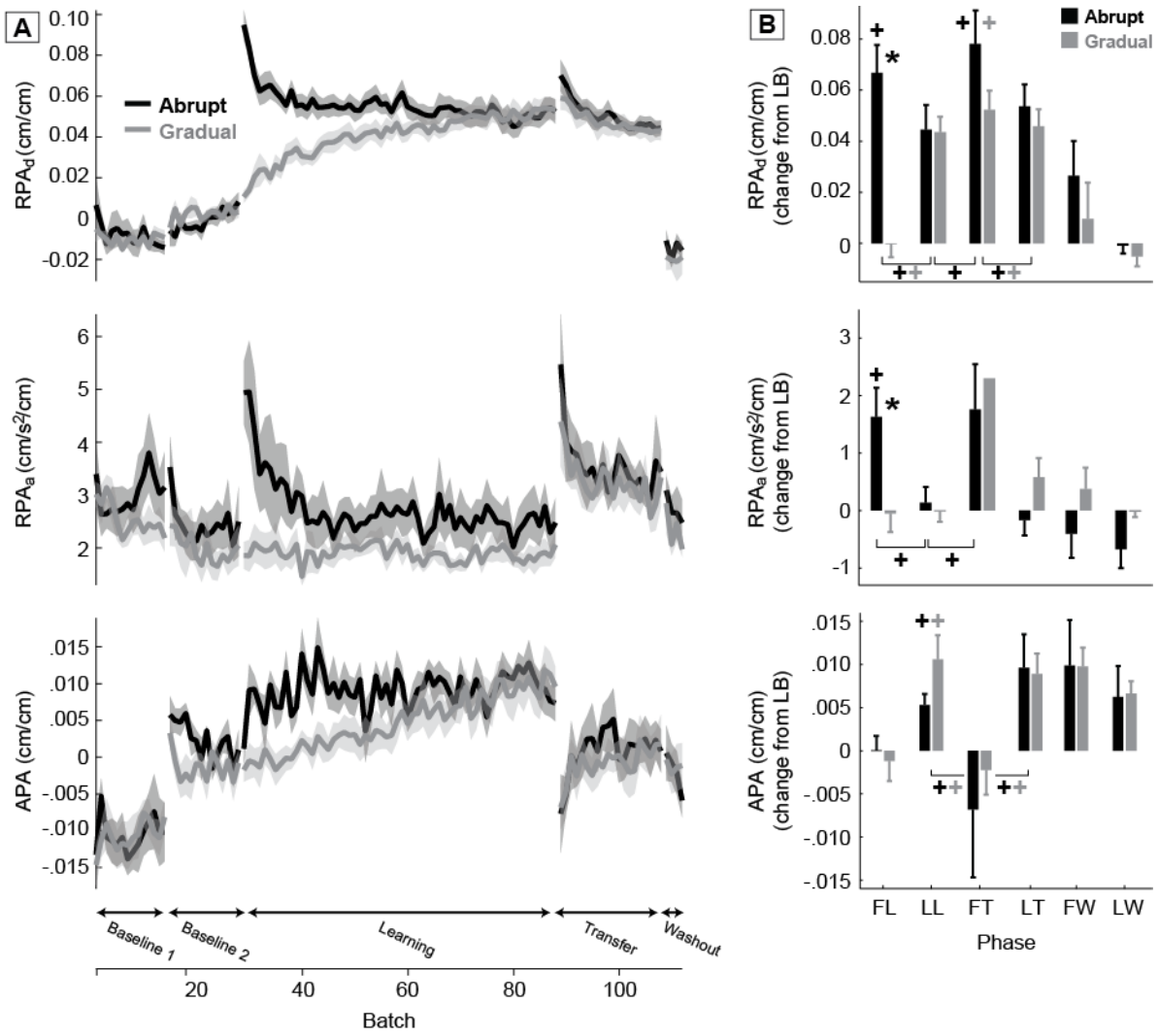

Figure 6.4. Adaptation and transfer of postural control metrics. Upper, middle, and lower plots show RPA_d, RPA_a, and APA, respectively. Data for Abrupt group is black; Gradual group is gray. (A) Left plots show each metric vs. batch (5 trials); in each plot, two traces show group means (solid lines) ± standard error (shading). (B) Right bar plots show the change from respective late baseline (LB) to first learning (FL, from LB2), late learning (LL, from LB2), first transfer (FT, from LB1), and late transfer (LT, from LB1), with error bars showing standard error. Statistically significant differences between groups ($p < 0.050$) are denoted by (*). (+) denotes a statistically significant difference within group in change from respective baseline, or between other phases where indicated ($p < 0.050$).

learning (both p-values ≤ 0.018) and then decreased from first learning to late learning (RPA_d p = 0.055; RPA_a p = 0.008). In the Gradual group, RPA_a showed no significant change from late baseline 2 to first learning or from first learning to late learning (both p-values ≥ 0.870). Similarly, RPA_d did not show a significant change from late baseline 2 to first learning (p = 0.963), but then it significantly increased from first learning to late learning (p = 0.001). However, that increase over the learning block was related to adaptation of APA, which significantly increased from late baseline 2 to late learning in both groups (both p-values ≤ 0.009). (As discussed earlier in the Methods section, RPA_d increased throughout learning because the COP movement that is initiated in the APA is propagated into the reactive time period.) To compare adaptation of arm control between groups, we made planned comparisons between the changes in RPA_d, RPA_a, and APA from late baseline 2 to late learning (Figure 6.4B). These changes were similar between groups (RPA_d p = 0.940; RPA_a p = 0.668; APA p = 0.113), indicating that both groups adapted their postural control similarly by the end of the learning block.

Transfer: To test for transfer of adapted postural control from the right to the left arm, we focused on the late baseline 1, late learning, and first transfer phases (Figure 6.4A). From late learning to first transfer, both groups showed a significant change in APA (both p-values ≤ 0.011); the Abrupt group showed significant changes in RPA_d and RPA_a (both p-values ≤ 0.018), but the Gradual group did not (RPA_d p = 0.275; RPA_a p = 0.052). From late baseline 1 to first transfer they showed no significant change in APA (both p-values ≥ 0.421), a significant increase in RPA_d (both p-values < 0.001), and an increase in RPA_a (both p-values ≤ 0.064 but > 0.05), indicating that when subjects switched to the left arm they used postural control that was similar to left-handed baseline reaching; they did not anticipate the perturbation and thus needed to

compensate using large reactive COP movements. Subsequently, both groups adapted to the perturbation while reaching with the left arm, showing significant decreases in RPA_d (both p-values ≤ 0.029), slight decreases in RPA_a (both p-values ≤ 0.086), and significant increases in APA (both p-values ≤ 0.035) from first transfer to late transfer. To compare transfer between groups, we made planned comparisons between the changes in RPA_d, RPA_a, and APA from late baseline 1 to first transfer and late transfer (Figure 6.4B). The changes in all three metrics from late baseline 1 to first transfer were similar between groups (all p-values ≥ 0.111), indicating that both groups showed a similar lack of transfer. The Gradual group did show a trend toward a smaller change in RPA_d from late baseline 1 to first transfer, compared to the Abrupt group ($p = 0.111$); however, similar to hand error, this is likely due to the fact that the Gradual group reached with a smaller peak velocity and experienced a smaller peak field force than the Abrupt group (Figure 6.3B).The changes from late baseline 1 to late transfer were also similar between groups (all p-values ≥ 0.105), indicating that both groups adapted their postural control similarly by the end of the transfer block.

6.3.6 Initial adaptation

We wanted to answer the question of whether adaptation can be driven by very small errors. The study described in Chapter 3 of this dissertation, using a trial-to-trial adaptation paradigm in which perturbations of randomly varying strengths were applied to the hand while subjects stood and made arm reaching movements, found that arm adaptation was sensitive to small error magnitudes but postural adaptation was not. That study compared those small error magnitudes to errors experienced in unperturbed baseline movements, and found that small hand error magnitudes were significantly different from baseline however, small postural error

magnitudes were indistinguishable from baseline. To make a similar comparison, we compared error magnitudes for the Gradual group in first learning and late learning to error magnitudes in late baseline 2. Hand error magnitudes in both phases were similar to late baseline 2 (first learning $p = 0.415$; late learning $p = 0.172$). RPA_a magnitudes were also similar to late baseline 2 (first learning $p = 0.827$; late learning $p = 0.110$). RPA_d magnitudes in first learning were similar to late baseline 2 ($p = 0.617$). RPA_d magnitudes in late learning were significantly increased from late baseline 2 ($p < 0.001$), but as we discussed previously this was related to APA adaptation. Thus, our results show that error magnitudes experienced by the Gradual group during learning were similar to those experienced in unperturbed baseline movements. Despite this, adaptation was able to occur in both arm and posture.

We also wished to make a closer examination of initial adaptation behavior, and to compare it between groups. For each subject, we found the learning batch at which each metric significantly diverged from late baseline 2, using independent t-tests to compare for statistical significance between trials in late baseline 2 and each subsequent batch. Group mean results are shown in Figure 6.5. We then compared between groups using one-tailed independent t-tests, and compared between metrics, within subjects, using one-tailed paired t-tests. For all metrics of arm adaptation (hand error and anticipatory force) and postural adaptation (RPA_d, RPA_a, and APA), the Abrupt group diverged significantly earlier than the Gradual group (all p-values ≤ 0.037). Error metrics (hand error, RPA_d, and RPA_a) diverged earlier in the Abrupt group due to the large initial errors that occurred when the force field turned on at full strength. The difference in anticipatory metrics (anticipatory force and APA) indicates that anticipatory learning progressed faster in the Abrupt group compared to the Gradual group. In both groups, anticipatory force

diverged faster than APA (Abrupt p 0.016; Gradual p = 0.018), indicating that anticipatory learning progressed faster in the arm than in posture.

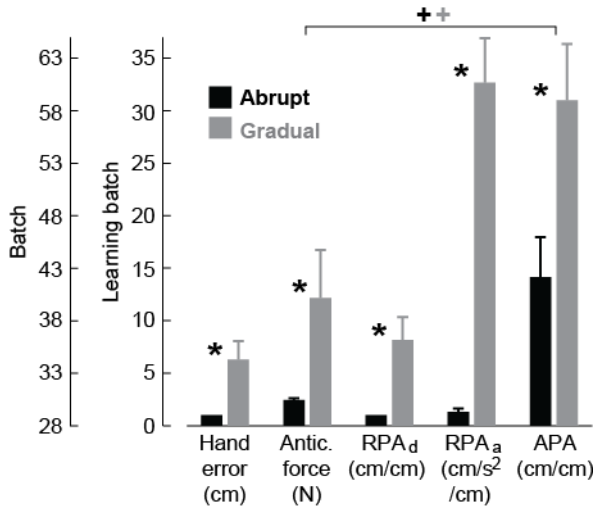

Figure 6.5. Time until arm and postural control metrics diverge from baseline. Bar plot showing group mean values of learning batch at which each metric significantly diverges from late baseline 2 (with batch 29 being equivalent to learning batch 1, the first batch of learning trials). Mean data for the Abrupt group data is shown in black, and the Gradual group in gray. Error bars show standard error. Statistically significant differences between groups ($p < 0.050$) are denoted by (*). (+) denotes a statistically significant difference within group ($p < 0.050$).

6.4 Discussion

6.4.1 Summary

The results of this study show that in a dynamic force learning paradigm with concurrent arm and postural adaptation, postural adaptation cannot be transferred without concurrent arm adaptation. Specifically, we found that when subjects switched to the non-dominant arm after adapting with the dominant arm, neither arm nor postural adaptation was transferred. We also found that, in the Gradual group, arm and postural adaptation was able to occur despite subjects

experiencing very small errors with magnitudes comparable to inherent baseline variability. This suggests that both error size and consistency play a role in driving motor adaptation. While the Abrupt group showed faster anticipatory learning than the Gradual group in both arm and posture, both groups adapted similarly by the end of learning. This indicates that the abrupt vs. gradual perturbation affects learning rate but not the final extent of learning, given sufficient practice.

6.4.2 Adaptation

We found that both groups adapted similarly by the end of learning, in both arm and posture. Several earlier studies reported that in adaptation of arm reaching to a visuomotor or dynamic perturbation, adaptation was similar or greater when the perturbation was introduced gradually rather than abruptly (Kagerer et al. 1997; Kluzik et al. 2008; Malfait and Ostry 2004; Michel et al. 2007). However, the duration of adaptation in these studies varied. Kagerer et al. (1997) tested reaching adaptation to a visuomotor perturbation. They found that after 60 trials with an abrupt vs. a gradually increasing perturbation, followed by 120 trials of the full perturbation (180 trials total), the gradual group exhibited smaller errors than the abrupt group, indicating that they had adapted to a greater extent. However, in our experiment the groups adapted similarly after 300 trials, with the final 20 trials at full perturbation strength. Our findings indicate that, given a sufficiently long adaptation period, subjects will adapt similarly whether the perturbation is abrupt or gradual.

Our results show conclusively that subjects can adapt their movement control to very small but consistent errors. In the Gradual group, the magnitudes of arm and postural errors (hand error and RPA_a) that were experienced during learning were similar to those experienced

in unperturbed baseline movements, but adaptation was still able to occur to a similar extent as in the Abrupt group. Results from an earlier study (Chapter 3 of this dissertation) showed that in a similar standing-and-reaching adaptation paradigm with randomly varying perturbation strengths, subjects did not adapt their postural control in response to small error magnitudes that fell within the range of inherent movement variability. Findings from other studies indicated that in a dynamic learning paradigm, 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). Based on that, in Chapter 3 we suggested that very small postural errors were small enough to be attributed to inherent postural variability rather than to external forces. However, the randomly varying perturbation strengths in that experiment may have contributed a high degree of uncertainty. Other studies have shown that uncertainty can lead to reduced adaptation. Wei and Kording (2010) tested an arm reaching task in which subjects adapted to random visuomotor perturbations over repeated trials; they found that adaptation progressed more slowly when the uncertainty of visual feedback was increased (noise was added to the cursor position). Similarly, Stevenson et al. (2009) tested a standing task in which subjects controlled their COP position in the presence of random visual perturbations to COP cursor feedback; they found that subjects' responses to perturbations were smaller when noise was added to the cursor position. In the present experiment, subjects in the Gradual group were exposed to very small but consistent errors, causing less uncertainty. Despite the small error sizes, subjects did adapt, suggesting that both error size and consistency play a role in driving motor adaptation.

We also found that anticipatory learning progressed faster in the Abrupt group compared to the Gradual group; anticipatory force and APA metrics both diverged from late baseline

significantly earlier in the Abrupt group compared to the Gradual group. This is expected, given the smaller perturbations and smaller error sizes experienced by the Gradual group in early learning. Earlier studies of arm reaching and posture have shown that adaptation from one trial to the next scales with the perturbation magnitude and/or error magnitude experienced in the previous trial (Fine and Thoroughman 2007; Franklin et al. 2003; Herzfeld et al. 2014; Marko et al. 2012; Osu et al. 2003; Scheidt et al. 2001; Shadmehr and Mussa-Ivaldi 1994; Thoroughman and Shadmehr 2000; Trent and Ahmed 2013; Wei and Kording 2010; Chapter 3 of this dissertation). In addition, although error uncertainty was lower for the Gradual group than for subjects experiencing randomly varying perturbations, there was likely greater error uncertainty in the Gradual group than in the Abrupt group, because of the very small error sizes.

In general, anticipatory learning progressed faster in the arm than in posture; anticipatory force diverged from late baseline faster than APA in both groups. In a similar dynamic adaptation experiment, Ahmed and Wolpert (2009) found that anticipatory learning in the arm (anticipatory force) progressed at a faster rate than anticipatory postural control (APAs). These results might be explained as an effect of error uncertainty in arm vs. posture. In both experiments, postural error uncertainty may have been greater than arm error uncertainty due to the fact that subjects received proprioceptive feedback as well as explicit visual feedback (hand cursor) about hand movements, but did not receive explicit visual feedback about postural movements.

6.4.3 Transfer of arm control

Both groups failed to transfer their adapted arm control from right-handed to left-handed reaching. When subjects switched to reaching in the force field with the left arm, they

immediately reverted to a control pattern similar to the left-handed late baseline (late baseline 1), demonstrating no anticipatory force to compensate for the force field and consequently experiencing large hand errors.

Based on previous studies, we expected the Gradual group to show no transfer of adapted arm control. Malfait and Ostry (2004) found that when subjects adapted their seated arm reaching to a similar gradual force perturbation, they showed no transfer from the dominant to the non-dominant arm. They suggested that this was because the very small error sizes experienced during adaptation led to a lack of higher cognitive information about the force field, and thus inhibited transfer. A modeling study by Berniker and Kording (2008) provided further explanation. Their model indicated that during adaptation to novel dynamics, the brain will attribute some of the perturbation dynamics to the environment (e.g. robotic training device) but tends to attribute more to the body (e.g. reaching arm), because the brain prefers to attribute motor errors to a misestimate of body properties rather than to a change in environmental properties. When transferring control from one arm to the other, the brain will assume that the misestimate is localized to the first arm. Thus, only the control for the properties attributed to the environment will be transferred to the other arm. When errors are small and/or uncertainty is high, errors are even more likely to be attributed to the arm than to the environment (Kluzik et al. 2008; Torres-Oviedo and Bastian 2012), and control that is adapted in response to those errors is less likely to be transferred to the other arm (Berniker and Kording 2008; Criscimagna-Hemminger et al. 2003).

However, we expected that the Abrupt group would transfer their adapted arm control between limbs. Two previous studies found that when subjects adapted their seated arm reaching to a similar abrupt force perturbation, they transferred their adapted control from the dominant to

the non-dominant arm (Criscimagna-Hemminger et al. 2003; Malfait and Ostry 2004). In contrast, we found that subjects in the Abrupt group showed no transfer between arms, despite the fact that they experienced large errors during adaptation. It is unlikely that duration of training played a role in this result. In the experiments of Criscimagna-Hemminger et al. (2003) and Malfait and Ostry (2004), subjects adapted to the field over training periods of 450 trials and 15 trials, respectively; in the present study, subjects adapted over a period of 300 trials.

It is possible that subjects in our Abrupt group were prevented from transferring their adapted control between arms because of uncertainty or attentional processing. Movement complexity and variability, and thus overall uncertainty, are likely greater in a standing reaching task compared to a seated reaching task; these factors could have led to a change in error assignment that thus contributed to the lack of arm transfer. The "standing" component of this task likely also had an adverse cognitive effect on the reaching task, compared to the seated reaching task; this effect was further exacerbated by the presence of postural perturbations. Several studies have shown that when postural control is more attentionally demanding (e.g. standing vs. sitting, or standing with a narrow vs. normal BOS), performance on a concurrent mental task is negatively affected (e.g. reaction times are slowed in an auditory reaction time task) (Lajoie et al. 1993, 1996; Remaud et al. 2012). EEG evidence suggests that this is a direct result of competing demands on attentional resources in the brain (Little and Woollacott 2015). Other studies have shown that responding to a postural perturbation can also draw attentional resources away from a concurrent visual or mental task (Brown et al. 1999; Maki et al. 2001; Norrie et al. 2002). These findings suggest that attentional resources, and thus cognitive processing, may be reduced for the arm reaching task when performed concurrently with a

standing postural task, compared to a simple seated reaching task. Theoretically, this might interfere with storage and transfer of motor memories, which could result in reduced transfer.

6.4.4 Transfer of postural control

Both groups also failed to transfer their adapted postural control from right-handed to left-handed reaching. When subjects switched to reaching with the left arm, they immediately reverted to a COP movement pattern similar to the left-handed late baseline (late baseline 1), demonstrating no APAs to compensate for the force field and consequently needing to make large, fast corrective movements (RPA_d and RPA_a).

When subjects switched arms, they were still reaching in the same force field and thus the expected postural perturbation should remain the same. Therefore, if the postural control system was able to adapt to and control for the novel dynamics independently of arm control, there should have been no change in the perturbation-specific postural control pattern, regardless of whether or not the adapted arm control was transferred. However, we found that all subjects failed to transfer their arm control and also failed to transfer their postural control. This suggests that the learned postural control was overwritten by arm control. Ahmed and Wolpert (2009) showed that after subjects adapted their arm reaching movements to a novel dynamics while sitting, they were able to generate appropriate postural control immediately upon standing, even though the postural system did not directly experience the novel dynamics during adaptation. This indicates that the postural control system can plan appropriate postural control based on information about arm movement dynamics; i.e., the postural controller can learn from the arm controller. Our findings indicate that the inverse is not true: the postural controller cannot *teach* the arm controller. Rather, our findings suggest that in a combined postural and arm movement

task, the postural control system is not only *able* to generate predictive control based on information from the arm, but that postural planning is also *dependent* on information about the planned arm movement. Further, whether or not appropriate postural control is *adapted* independently, it can be overwritten by arm control upon transfer to a new context. This is because predictive postural control is generated based on the brain's prediction of how whole-body dynamics will be affected by arm movement dynamics. Therefore, because the brain did not predict novel dynamics at the arm, predictive postural control (APAs) was not generated for those dynamics. Similarly, Flanagan et al. (2003) showed that predictive learning precedes control learning. In a task where subjects were required to manipulate an object with novel dynamic properties, they learned to predict the consequences of their actions before they learned the appropriate control for those actions.

6.4.5 Clinical implications

Our results, combined with findings from Chapter 3 of this dissertation, show clearly that both error size and consistency play a role in driving motor adaptation. Therefore, both of these factors should be considered in the design of training and rehabilitation paradigms. Some clinical populations 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, it is possible that they might be unable to respond to a greater range of "small" errors, compared to healthy individuals, if they are unable to distinguish those errors from their natural errors. However, in light of our present findings, a highly consistent error signal could help to reduce uncertainty and could thus improve adaptation.

In general, it is also desirable for learning that is acquired in one context to be transferred to other contexts. In this study we found that in a standing-and-reaching task, neither arm nor postural learning was transferred when subjects switched reaching arms. In the arm, possible reasons for the lack of transfer included uncertainty and attentional demands. Specifically, the addition of the standing component of the task (compared to a seated reaching task) as well as postural perturbations may have led to greater uncertainty about task performance and error assignment. In addition, they may have drawn increased attentional resources to postural control, thus reducing the amount of resources available for arm control (Brown et al. 1999; Lajoie et al. 1993, 1996; Little and Woollacott 2015; Maki et al. 2001; Norrie et al. 2002; Remaud et al. 2012). Theoretically, any of these factors could have interfered with storage and transfer of the learned dynamics. Therefore, when designing training and rehabilitation paradigms, it is important to consider the possible effects of performing concurrent tasks.

This would be especially important in clinical populations which exhibit poor motor performance in some areas. For example, older adults can demonstrate reduced postural stability (Binda et al. 2003; Campbell et al. 1989; Fujimoto et al. 2013; Holbein-Jenny et al. 2007; Maki et al. 1994; Melzer et al. 2004; Overstall et al. 1977), reduced mobility (Chen and Chou 2013; Hurt and Grabiner 2015; Rogers et al. 2001), and reduced ability to recover from perturbations (Graham et al. 2015; Honarvar and Nakashima 2014; Kuo & Zajac 1993; Robinovitch et al. 2002). These may contribute to the fact that postural tasks can be more attentionally demanding in older adults than in young adults (Brown et al. 1999; Lajoie et al. 1996), even under relatively undemanding conditions (Maylor and Wing 1996; Woollacott & Shumway-Cook 2002). One study also showed that when older adults experienced a postural perturbation, they exhibited delayed attentional switching between postural control and a concurrent visuomotor task (Maki

et al. 2001). This suggests that older adults may have impaired attentional dynamics, which could exacerbate the deleterious effects of concurrent task performance, especially when adapting to novel dynamics which requires additional cognitive resources.

6.4.6 Conclusions

The results of this study demonstrated that in a dynamic force learning paradigm with concurrent adaptation of arm reaching and standing posture, learned postural control was overwritten by the transfer of arm dynamics. In other words, postural movement planning related to a concurrent arm task is dependent on information about arm dynamics, but not vice versa. In contrast to previous studies, no learning is transferred between arms. This may be due to the increased uncertainty and attentional demands caused by the concurrent postural task. Our results also show that adaptation of both arm and postural control can be driven by very small errors, within the range of inherent movement variability, if errors are consistently biased. This suggests that both error size and consistency play a role in driving motor adaptation. Generally, our findings demonstrate that in the design of rehabilitation and training regimens, it is important to consider that while postural control is a separate process from arm control, it is nonetheless dependent on arm control. Further, it is also important to consider both the size and consistency of errors that are used to drive adaptation.

CHAPTER 7

THESIS CONCLUSIONS

I have presented four studies investigating the mechanisms and control strategies involved in the adaptation of whole-body postural control and how well this learning transfers to different environments. Below, I summarize the major findings of these studies, discuss their implications, and suggest directions for future research.

7.1 Summary of findings

1. Error size and consistency both play a role in driving postural adaptation (Chapters 3 and 6).
    - Generally, adaptation scales proportionally with error (Chapter 3).
    - In contrast to arm adaptation, postural adaptation is insensitive to small, randomly varying errors within the range of inherent movement variability (Chapter 3).
    - Adaptation can be driven by very small yet consistent errors (Chapter 6).

2. A muscle coactivation strategy is used early in postural adaptation to reduce errors when novel dynamics have not yet been learned (Chapter 4).

3. Stability conditions can significantly affect postural adaptation strategies (Chapters 4 and 5).
    - Control strategies differ throughout learning depending on stability limits (Chapter 4).
    - The extent of postural adaptation is not affected by differing stability limits only (Chapter 5), but can be affected by a combination of differing stability limits and postural threat (Chapters 4 and 5).

4. Transfer of adapted postural control between different stability conditions is affected by the condition in which the task is initially learned (Chapter 5).
    - Learning in a more stable condition facilitates transfer of postural learning to other conditions.

5. In a concurrent posture and arm movement task, control of the arm movement task is generally not affected by differing postural conditions (Chapters 4 and 5).
    - The extent of arm movement adaptation is not affected by differences in postural adaptation (Chapter 4).
    - Transfer of arm movement control is not affected by differences in postural control (Chapter 5).
    - Learning and/or transfer of an arm movement task may be affected by performing the task while standing, compared to while seated (Chapter 6).

6. Postural control in a concurrent posture and arm movement task is dependent on information about arm dynamics (Chapter 6).
    - Postural adaptation is not transferred between conditions without concurrent arm transfer; i.e., without learned arm movement control, there is no learned postural control.

7.2 Implications of work

The ability to maintain stable, upright standing is a critical component of our daily activities. We are constantly making voluntary movements, responding to perturbations, and adapting our movement control for changing conditions. The experimental findings presented in this dissertation have contributed to our understanding of how we adapt our whole-body postural control in different conditions and how well this learning transfers to new environments. Postural adaptation manifests many characteristics of general motor adaptation (as seen in Chapters 3, 4, and 6). However, in healthy young adults, postural stability conditions play a significant role in determining how standing posture is controlled, adapted, and transferred between different contexts (as seen in Chapters 4 and 5). Postural control is also strongly influenced by concurrent arm movements (as seen in Chapter 6). These findings have important implications for motor control, rehabilitation, and clinical research.

7.2.1 Implications for motor control

The control of standing posture is amazingly complex. The physiology of postural control is similar to that of human motor control in general, and adaptation of postural control manifests many characteristics that have been observed in motor adaptation in eye movements, arm reaching movements, locomotion, and other forms of movement. However, postural control is subject to unique stability constraints that significantly affect how we control and adapt our whole-body posture.

Contributing to the body of research about how sensorimotor error drives adaptation, the findings in Chapter 3 demonstrated that postural adaptation, similar to adaptation in other forms of movement, scales with movement error. Results from Chapters 3 and 6 showed that while adaptation may be insensitive to small errors when uncertainty is high, adaptation can in fact be driven by very small yet consistent errors, suggesting that both error size and consistency play a role in driving motor adaptation.

My results also carry intriguing implications about how and why various control strategies are used in different conditions. Results in Chapter 4 suggested several ways in which postural control strategies in early and late adaptation can be affected by postural stability limits and the inherent control trade-off between stability and maneuverability. Results from Chapters 5 and 6 demonstrated that transfer of adapted control between different conditions (e.g. changes in postural stability or reaching arm) is affected by the condition in which the task is initially learned. Differences in transfer may be due to choices in control strategy related to the change in conditions; the differences could also be related to how the brain represents motor memories under different conditions and subsequently transfers them to other conditions. Those results, combined with previous research findings, suggested that transfer between different conditions

can also be influenced by changes in postural stability, postural threat, and concurrent tasks, and also by a desire to use habitual rather than optimal control patterns.

My experiments involved a coordinated movement task with concurrent control of arm reaching and standing posture. Several findings suggested that in this coordinated task, the focal arm reaching movement is prioritized over postural control. Evidence showed that by the end of learning, adapted arm control was not affected by differences in postural conditions, despite differences in postural adaptation and/or transfer (Ahmed and Wolpert 2009; Manista and Ahmed 2012; Chapters 4-6 of this dissertation). This suggests that postural control may be a secondary consideration to the focal arm movement, such that posture is controlled and adapted in a manner that is specific to different postural conditions, but this is not manifested in arm behavior. Results from a previous study indicated that the postural control system is able to use information about arm movement dynamics to plan appropriate postural control for a novel perturbation (Ahmed and Wolpert 2009). My findings in Chapter 6 indicated, further, that the postural system *depends* on that information about arm dynamics, and does not generate predictive control independent of the planned arm movement.

7.2.2 Implications for training and rehabilitation

Some of my research findings are directly applicable to physical training and rehabilitation. Results from Chapter 3 showed that postural adaptation increases in response to stronger perturbations and larger errors, in agreement with several previous studies of standing posture and locomotion (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). Further, if subjects train in conditions of larger postural stability limits (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 (Wulf et al. 1998; Domingo and Ferris 2009, 2010). In cases where the goal of training is simply to encourage subjects to show stronger adaptation (larger movements, greater muscle force, etc.), such as in some physical rehabilitation programs, these factors could help to maximize performance. This could be especially beneficial in the rehabilitation of individuals who exhibit reduced postural movements, such as older adults and stroke survivors.

However, in most cases it is also desirable for learning to be transferred to other contexts. Two previous studies found that in adaptation of arm reaching and locomotion, smaller errors led to reduced adaptation but also led to increased magnitude and percentage of transfer to other contexts (from reaching with a robotic arm to reaching in free space, or from walking on a treadmill to walking overground), compared to larger errors (Kluzik et al. 2008; Torres-Oviedo and Bastian 2012). This is due to the brain's tendency to assign to smaller errors to the body rather than to the environment (e.g. robotic arm or treadmill) (Berniker and Kording 2008; Kluzik et al. 2008; Torres-Oviedo and Bastian 2012). Combined with findings from Chapters 3 and 6 about how error size and consistency play a role in driving adaptation, this further suggests that rehabilitative paradigms should be designed in order to cause errors which are small, in order to promote transfer outside of the training context, but which are sufficiently large and/or consistent to ensure that errors will drive adaptation.

My transfer findings from Chapters 5 and 6 suggested that it is also important to consider the level of "challenge" that subjects experience in the training context. This may include postural stability or threat conditions, as well as uncertainty and attentional demands. However,

based on these and other findings (Domingo and Ferris 2009; Wulf et al. 1998), it remains unclear whether it is beneficial for initial training to take place in a more challenging or less challenging context. What is clear is that the context in which initial training occurs can influence transfer to other contexts.

7.2.3 Implications for clinical populations

My research carries several important implications for various clinical populations who demonstrate postural control deficits, such as older adults, stroke survivors, and individuals with Parkinson's disease.

Behavioral results from Chapter 4 indicated that healthy young adults altered their postural adaptation strategies in differing postural stability conditions, driven by a trade-off between stability and maneuverability. This highlights an area for concern in populations who exhibit reduced mobility, reduced postural stability, 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 & 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 Parkinson's patients (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 vs. maneuverability strategies is especially critical because both stability and maneuverability are low compared to healthy young adults. These deficits in older adults, for example, 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 & Zajac 1993; Robinovitch et al. 2002; Vincent et al. 2002; Taaffe et al. 1999).

Many clinical research studies are aimed at using adaptation-based training paradigms to improve motor performance in various clinical populations. For example, in patients with hemi-neglect, adaptation to a visuomotor perturbation can lead to improvements in neuropsychological symptoms of neglect (Pisella et al. 2002; Rode et al. 2003; Rossetti et al. 1998). Stroke patients with hemiparesis have experienced improvements in their arm movement and/or locomotor error patterns after adapting to a dynamic perturbation (Patton et al 2006; Reisman et al. 2007, 2009). As discussed in the preceding section, some of my findings suggest ways in which these rehabilitation paradigms can be modified to improve the strength of these adaptation-driven improvements in movement control, as well as to promote transfer of these improvements outside of the training context.

Other findings have suggested that smaller errors can lead to improved transfer, as discussed in the preceding section (Kluzik et al. 2008; Torres-Oviedo and Bastian 2012). In that situation, the definition of "small" could 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 be unable to adapt in response to a greater range of "small" errors, compared to healthy control subjects, if they are unable to distinguish those errors from their natural errors. A similar effect could 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. However, results from Chapter 6 suggest that a highly consistent error signal could help to reduce uncertainty and could thus lead to improved adaptation in these individuals.

7.3 Future directions

The research presented in this dissertation investigated how movement error drives postural adaptation and transfer, how adaptation and transfer of postural control strategies are affected by postural stability conditions, and how postural control is coordinated with a concurrent arm reaching task. The results answered many open questions in the area of postural adaptation, and also raised several other possibilities for future investigation.

In Chapters 4 and 5, I tested the effects of differing postural conditions on adaptation and transfer. In one study I compared between a smaller BOS / higher threat condition and a larger BOS / lower threat condition; in the other I compared between smaller and larger BOS conditions, with no explicit difference in postural threat. This allowed me to examine the effects of BOS size alone, but one area for future investigation would be how adaptation and transfer are affected by postural threat alone.

Results in Chapters 4 and 6 suggested that changes in attentional demands between concurrent movement tasks might affect how movement control strategies are learned and transferred. In one case, more attentionally demanding postural control may have led to a change in arm control strategies. More interestingly, another finding suggested that the added attentional demand of performing an arm reaching task while standing, rather than sitting, may have

contributed to an alteration in motor memory learning and/or transfer. Future studies could more directly investigate the effects of attentional demand in concurrent motor tasks.

In all four experimental studies, the postural control associated with perturbations remained well within postural stability limits. However, it would be expected that postural behavior would change markedly at larger perturbation magnitudes, where postural control approaches stability limits, and/or in conditions of increased postural threat, where subjects may choose to further restrict their postural control. Future research could concentrate more on postural control near these boundaries.

Finally, all of the experiments in this dissertation focused on healthy young adults. Future research directions should expand to include older adults, clinical populations with Parkinson's disease or stroke, and other populations exhibiting motor control deficits. Testing adaptation and transfer of movement control in these individuals can give insight into how various clinical pathologies affect the underlying motor control system. In addition, this would inform the design of more effective treatment and rehabilitation programs for these populations.

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APPENDIX

LIST OF ABBREVIATIONS

APA	Anticipatory postural adjustment
BOS	Base of support
COM	Center of mass
COP	Center of pressure
fBOS	Functional base of support
RPA	Reactive postural adjustment
RPA_a	RPA based on COP acceleration
RPA_d	RPA based on COP displacement