Report

Current Biology

Learning New Feedforward Motor Commands Based on Feedback Responses

Highlights

- Long-latency stretch reflex responses can learn altered arm dynamics
- This learning occurs with minimal engagement of voluntary motor responses
- What reflex responses learn transfers to voluntary motor commands

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In Brief

Maeda et al. show that feedback responses to mechanical perturbations change appropriately when the mechanical properties of the arm are altered and that such changes transfer to the feedforward control of reaching. This finding furthers the notion that feedback and feedforward control processes share common neural circuits.

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Report

Learning New Feedforward Motor Commands Based on Feedback Responses

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SUMMARY

Learning a new motor task modifies feedforward (i.e., voluntary) motor commands and such learning also changes the sensitivity of feedback responses (i.e., reflexes) to mechanical perturbations [1–9]. For example, after people learn to generate straight reaching movements in the presence of an external force field or learn to reduce shoulder muscle activity when generating pure elbow movements with shoulder fixation, evoked stretch reflex responses to mechanical perturbations reflect the learning expressed during self-initiated reaching. Such a transfer from feedforward motor commands to feedback responses is thought to take place because of shared neural circuits at the level of the spinal cord, brainstem, and cerebral cortex [10–13]. The presence of shared neural resources also predicts the transfer from feedback responses to feedforward motor commands. Little is known about such a transfer presumably because it is relatively hard to elicit learning in reflexes without engaging associated voluntary responses following mechanical perturbations. Here, we demonstrate such transfer by leveraging two approaches to elicit stretch reflexes while minimizing engagement of voluntary motor responses in the learning process: applying very short mechanical perturbations [14–19] and instructing participants to not respond to them [20–26]. Taken together, our work shows that transfer between feedforward and feedback control is bidirectional, furthering the notion that these processes share common neural circuits that underlie motor learning and transfer.

RESULTS

In our main experiment (Figure 1), participants (n = 20) sat in a robotic exoskeleton and held their hand in a home target in the presence of short (20 ms) torque pulses applied to the shoulder and elbow joints (for full details, see STAR Methods). The mechanical perturbations were chosen in a way such that they caused pure elbow motion. The perturbations were applied first with the shoulder joint of the robot free to move (baseline phase; normal arm dynamics: forearm rotation causes shoulder torques), then with the shoulder joint locked by the robotic manipulandum (adaptation phase; altered arm dynamics: shoulder torques caused by forearm rotation are cancelled by the robot), and then again with the shoulder joint free to move (post-adaptation phase; normal arm dynamics). In all cases, participants could not predict the perturbation direction or onset and were instructed not to respond to the perturbation. Before and after the adaptation phase, participants also performed occasional probe trials where they self-initiated 20-degree pure elbow extension movements. Probe trials served to test whether learning during feedback control transferred to feedforward control.

We report two key findings. First, locking the shoulder joint leads to a reduction in shoulder reflex responses with a minimal engagement of voluntary motor responses in the learning process. This reduction occurs on a timescale of hundreds of trials and is appropriate for efficient control in the context of the novel arm dynamics. Second, this reduction in feedback responses transfers to feedforward motor commands, as evidenced by (1) a reduction in shoulder extensor muscle activity during self-initiated elbow reaching trials, even though participants never practiced reaching movements with the shoulder locked; and (2) kinematic errors (i.e., aftereffects) after releasing the shoulder joint in the direction predicted if failing to compensate for normal arm dynamics.

Baseline Responses Account for Normal Arm Dynamics

With the shoulder free to move, the mechanical perturbation we applied to the shoulder and elbow joints caused minimal shoulder rotation (Figure 2A) but elicited robust shoulder long-latency stretch reflexes (muscle activity occurring 50–100 ms post-perturbation) in the posterior deltoid (PD) muscle, a shoulder extensor muscle. The presence of a shoulder long-latency stretch reflex response in the absence of shoulder motion is appropriate for countering the underlying joint torques (Figure 2B) [5, 14, 27–32]. Similarly, generating pure elbow extension movements with the shoulder free to move involved substantial PD muscle activity as required to compensate for the torques that passively arise at the shoulder joint when the forearm rotates about the elbow joint (Figures 2C and 2D) [5, 30, 33].

Feedback Responses Learn New Arm Dynamics

Locking the shoulder joint alters the mapping between joint torques and joint motion because torques that normally arise at the

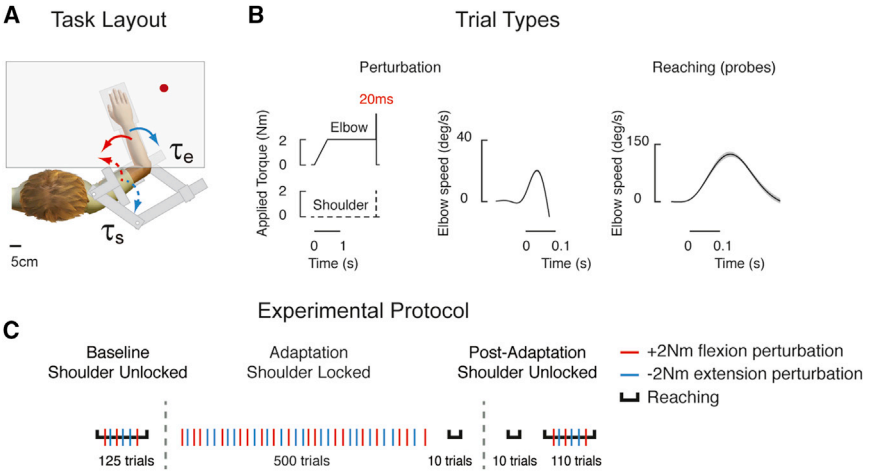

Figure 1. Experimental Setup

(A) Participants either received mechanical perturbations that created pure elbow motion or generated pure elbow extension movements to a dark-red goal target (probes). Red and blue arrows represent the direction of the multi-joint torque pulses applied to the shoulder (dashed) and elbow (solid) joints.

(B) These perturbations lasted 20 ms (load duration, left and middle panels), and reaching trials had speed constraints (100- to 180-ms duration, right panel). Black and shaded areas of the elbow speed profiles represent the mean and standard error of the mean across all participants. In both cases, a servo-controller brought the participant's arm back to the home location within 300–500 ms from completing the trial.

(C) Illustration of the experimental protocol. Participants performed 125 baseline trials with the shoulder joint unlocked (perturbation and reaching probe trials), 500 adaptation trials with the shoulder joint locked (perturbation trials), 10 reaching probe trials with the shoulder locked, 10 reaching probe trials with the shoulder unlocked, and 110 post-adaptation trials with the shoulder joint unlocked (perturbation and reaching probe trials). Black horizontal lines indicate blocks with reaching trials, and red and blue lines indicate trials with the direction of the multi-joint torque pulses as shown in (A).

shoulder joint due to forearm rotation are cancelled by the robotic apparatus. If the nervous system can learn these altered arm dynamics, then it can act more efficiently by reducing shoulder muscle activity without reducing task performance.

Consistent with such learning, Figure 3A illustrates how PD long-latency stretch reflexes slowly (over hundreds of trials) decreased from baseline levels after locking the shoulder joint and then quickly returned to baseline levels when the shoulder joint was unlocked again. This pattern of learning is very similar to what we previously reported for learning feedforward motor commands in this task [5]. A one-way ANOVA comparing PD long-latency stretch reflexes late (last 30 trials) in the baseline, adaptation, and post-adaptation phases revealed a reliable effect of experimental phase on shoulder muscle responses ($F_{2,38}$ = 25.12, $p < 0.0001$; Figures 3B and 3C). Tukey post hoc tests showed that PD long-latency stretch reflexes decreased by 60% relative to baseline ($p < 0.0001$) at the end of the adaptation phase and then returned to levels indistinguishable from baseline at the end of the post-adaptation phase ($p = 0.21$). In general, evoked PD voluntary responses (100–150 ms post-perturbation) were minimal (Figure S1A), and we found no reliable change in PD voluntary responses across the experimental phase ($F_{2,38}$ = 1.58, $p = 0.21$; Figure S1B). We found no corresponding changes in long-latency stretch reflexes of the lateral triceps muscle, an elbow extensor muscle (TR; one-way-ANOVA, $F_{2,38}$ = 2.19, $p = 0.12$; Figures 3D–3F). We also found no change in PD muscle activity prior to perturbation onset (−50–0 ms relative to perturbation onset) across experimental phases (one-way ANOVA, $F_{2,38}$ = 0.83, $p = 0.44$), indicating that the changes we observed in the PD long-latency stretch reflex are not a by-product of systematic changes in the pre-perturbation state of the motor neuron pool (so-called "automatic gain-scaling," [34–36]).

We performed a control experiment to ensure that our main experiment indeed minimized the engagement of PD voluntary responses in the learning process. Participants (n = 20) performed the same experimental protocol as in the main experiment but with a longer (100 ms) perturbation duration. In this case, evoked PD voluntary responses were substantial (Figure S1A), and we found a reliable decrease in PD voluntary responses after locking the shoulder joint (one-way ANOVA, $F_{2,38}$ = 7.209, $p = 0.0022$; Figure S1C). Tukey post hoc tests showed that PD voluntary responses decreased by 74% relative to baseline ($p = 0.003$), demonstrating that when voluntary responses are engaged, they show the same learning pattern.

We performed a second control experiment to rule out the possibility that the changes we observed in the PD long-latency stretch reflex merely reflected extensive exposure to mechanical perturbations rather than learning specific to the shoulder fixation manipulation. Participants performed the same number of trials as in the main experiment with either a 20-ms perturbation duration (n = 10) or 100-ms perturbation duration (N = 10), but the shoulder joint was never locked. For both perturbation durations, we found no reliable decrease in shoulder muscle responses in the long-latency epoch across equivalent experimental phases (20 ms: $F_{2,18}$ = 0.825, $p = 0.45$; see Figures S2A and S2B; 100 ms: $F_{2,18}$ = 0.83, $p = 0.44$; see Figures S2A and S2C). Importantly, the reductions in PD long-latency stretch reflexes at the end of the adaptation phase in our main experiment (i.e., with the shoulder locked; 20-ms load duration) were reliably different than the reductions in this control experiment (t_{11} = −2.81, $p = 0.01$).

Learning New Feedback Responses Transfers to Feedforward Motor Commands

If the nervous system is able to transfer learning from feedback responses to feedforward motor commands, then the reduction in PD long-latency stretch reflexes we observed in response to the shoulder locking manipulation should be expressed during self-initiating reaching. This should be the case even if participants never engaged feedforward control mechanisms under conditions in which the shoulder was locked.

Consistent with such a transfer, Figures 4A–4C illustrate that shoulder extensor muscle activity in self-initiated reaching

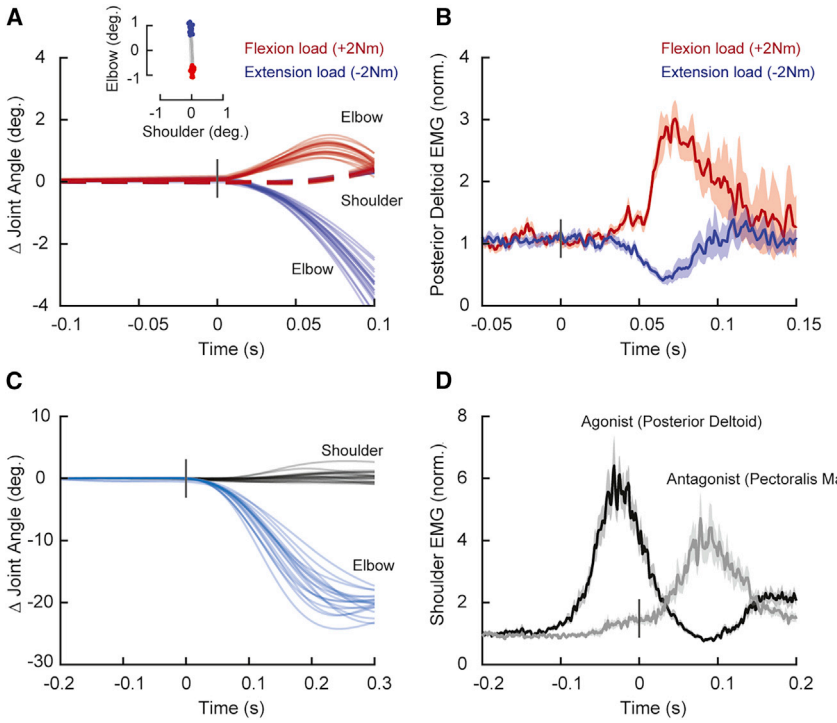

Figure 2. Compensating for Intersegmental Dynamics during Perturbations That Created Pure Elbow Motion and When Generating Pure Elbow Extension Movements

(A) Individual participant's kinematic traces of the shoulder (dashed) and elbow (solid) joints following mechanical perturbations of 20-ms duration. Red and blue traces are from the shoulder and elbow flexor torque and shoulder and elbow extensor torque conditions, respectively. Data are aligned on perturbation onset. Inset shows the amount of shoulder and elbow displacement at 50 ms post-perturbation (data are shown for all subjects).

(B) Normalized shoulder posterior deltoid muscle activity associated with (A). Shaded error areas represent the standard error of the mean.

(C) Individual participant's kinematic traces of the shoulder (black traces) and elbow (blue traces) joints for elbow extension trials. Data are aligned on movement onset.

(D) Black and gray lines represent average agonist (posterior deltoid) and antagonist (pectoralis major) muscle activity associated with the movement in (C). Shaded error areas represent the standard error of the mean.

(i.e., probe trials) performed after shoulder fixation (i.e., at the end of the adaptation phase) is reduced relative to probe trials before shoulder fixation (i.e., in the baseline phase). This reduction is statistically reliable. A one-way ANOVA comparing agonist (−100 to 100 ms epoch aligned on movement onset, see STAR Methods) PD muscle activity in the baseline, adaptation, and post-adaptation phases (10 probe trials) revealed a reliable effect of experimental phase on self-initiated PD muscle activity ($F_{2,38}$ = 20.55, $p < 0.0001$). Tukey post hoc tests revealed that agonist PD muscle activity decreased by 31% relative to baseline ($p < 0.0001$) at the end of the adaptation phase and then returned to baseline levels after unlocking the shoulder joint in the post-adaptation phase ($p = 0.53$). We found no corresponding changes in TR muscle activity (one-way-ANOVA, $F_{2,38}$ = 3.02, $p = 0.06$; Figures 4D–4F).

As expected for such a transfer, we found a reliable correlation between the decrease in a participant's PD muscle activity measured in the long-latency epoch of perturbation trials and their corresponding decrease in agonist PD muscle activity measured for self-initiated reaching trials ($r = 0.48$, $p = 0.03$; Figure S3). Additional evidence that learning new arm dynamics during perturbation trials transferred to reaching trials was the presence of kinematic after-effects. That is, in the early post-adaptation trials, participants generated trajectory errors in the direction predicted if they failed to compensate for the torques that normally arise at the shoulder joint when the forearm rotates about the elbow joint and the shoulder is unlocked (Figure 4G). We quantified these after-effects by performing a one-way ANOVA to compare reach accuracy (measured as distance from the center of the goal target) of trials late in the baseline phase (last 3 trials), trials early in the post-adaptation phase (first 3 trials), and trials late (last 3 trials) in the post-adaptation phase (Figure 4H). Note that we chose a small bin size for this analysis because the return to baseline after unlocking the shoulder joint happens very quickly [5]. We found a significant effect of experimental phase on these trajectory errors ($F_{2,38}$ = 4.14, $p = 0.023$). Tukey post hoc tests showed that movement errors increased by 51% ($p = 0.026$) from the baseline phase to the early post-adaptation phase and returned to levels indistinguishable from baseline ($p = 0.98$) in late post-adaptation phase.

Lastly, we performed a third control experiment to rule out the possibility that the changes we observed in PD muscle activity for self-initiated reaching reflected extensive exposure to background loads with the shoulder joint locked rather than a transfer of learning from feedback responses. Participants ($n = 10$) performed the same protocol as in the main experiment, but the robotic device did not apply a mechanical perturbation in the adaptation phase when the shoulder joint was locked. That is, in the adaptation phase, background loads were slowly turned on and off as in the main experiment, but no mechanical perturbation was ever introduced. In this experiment, we found no reliable decrease in shoulder muscle activity across equivalent experimental phases ($F_{2,18}$ = 0.55, $p = 0.58$; see Figures S4A and S4B). We also found no reliable decrease in shoulder muscle responses in the long-latency epoch in perturbation trials before, at the end of, and after experiencing repeated background loads with the shoulder locked ($F_{2,18}$ = 2.03, $p = 0.16$; see Figures S4C and S4D). Lastly, we found no reliable effect of experimental phase on self-initiated PD muscle activity during reaching trials ($F_{2,18}$ = 1.81, $p = 0.19$; see Figures S4E and S4F) and no reliable effect of experimental phase on trajectory errors ($F_{2,18}$ = 0.29, $p = 0.74$; see Figures S4G and S4H).

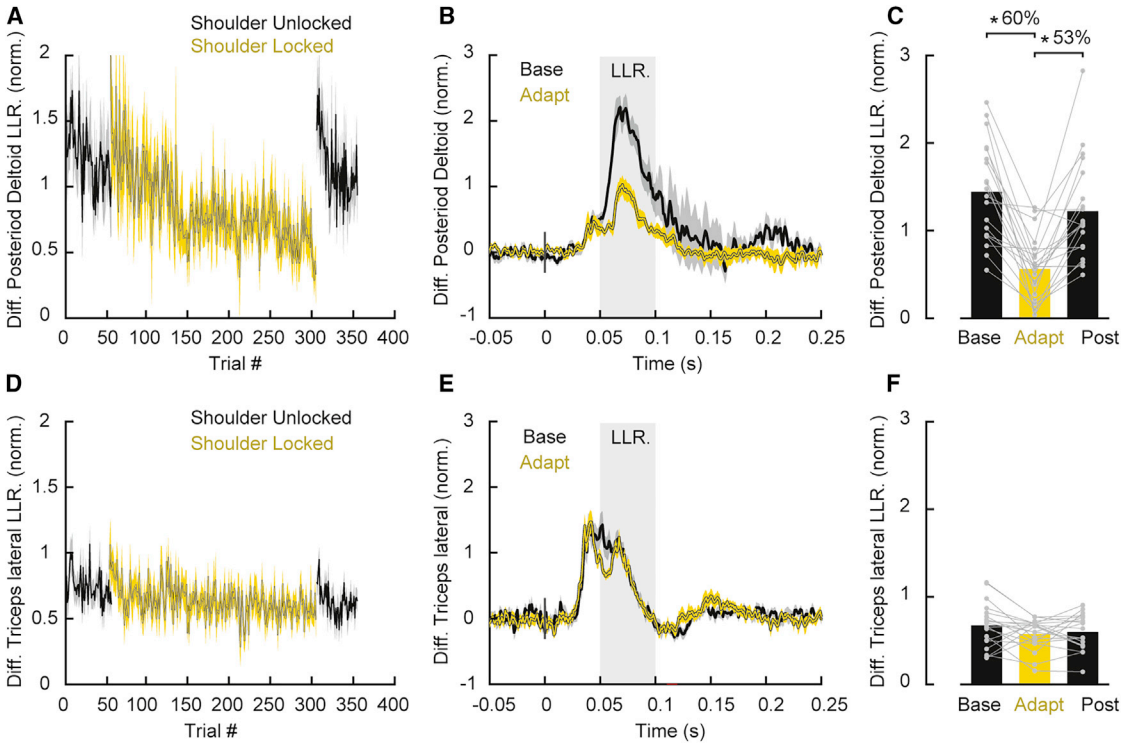

Figure 3. Learning Novel Arm Dynamics by Long-Latency Stretch Reflexes during Perturbation Trials with Shoulder Fixation

(A) Average of the difference of shoulder posterior deltoid muscle activity (flexion minus extension loads) in the long latency epoch (50–100 ms post-perturbation) across trials. Shaded error areas represent the standard error of the mean. Electromyographic signal (EMG) data are normalized as described in the STAR Methods.

(B) Time series of the difference of the posterior deltoid normalized muscle activity averaged over the last 30 baseline and adaptation trials. Shaded error areas represent the standard error of the mean. Gray horizontal shaded areas represent the long-latency reflex epoch (LLR). Data are aligned on perturbation onset.

(C) Average of the difference of posterior deltoid muscle activity in the long-latency epoch associated with trials late in the baseline, adaptation, and post-adaptation. Each dot represents data from a single participant. Asterisks indicate reliable effects ($p < 0.05$, see main text).

(D–F) Data for the difference of the elbow triceps lateral muscle (flexion minus extension loads) are shown using the same format as (A)–(C).

Related to Figures S1 and S2.

DISCUSSION

Internal models are a well-established concept in self-initiated reaching [37]. Internal models allow feedforward control mechanisms to compensate for the complex mechanical properties of the arm and delayed and noisy sensory feedback. They also enable adaptation of control signals for movement to changes of the body or the environment [38–43]. Less appreciated is that internal models may also modulate fast feedback responses, so that they may compensate for the same factors when countering external perturbations [2, 4–6, 14, 27–30, 32, 44–48]. For example, an internal model of the arm enables long-latency stretch reflexes, a class of fast feedback responses sensitive to mechanical stretch that include a cortical neural contribution [11, 49], to account for the arm's intersegmental dynamics and respond appropriately to the applied joint torques rather than local joint motion [5, 14, 27–30, 45].

There are two main findings in the present study. First, long-latency stretch reflexes can directly update the internal model that maps joint motion to joint torque during perturbations with little or no engagement of voluntary motor responses. This finding contrasts with previous work showing that when participants are exposed to motor learning paradigms, like visuomotor transformations or force fields without generating voluntary motor commands (i.e., during passive movements), such internal updates do not take place [for review, see 42, 50, 51]. Our finding suggests that evoking reflex responses is sufficient to drive learning, presumably because the nervous system can compare the predicted and actual effects of these responses (with respect to the new underlying torques) on movement outcomes. Second, our results show that the updated internal model learned by long-latency stretch reflexes influences the control of self-initiated reaching. These findings add to a growing body of work demonstrating how internal models updated during self-initiated reaching transfer to fast feedback control [1–9] by showing that this learning and transfer is bidirectional. These findings are also consistent with recent theories of motor control based on optimal feedback control, which posit that motor behavior is achieved via the sophisticated manipulation of sensory feedback [52, 53]. Under this class of models, bidirectional transfer between feedforward and feedback control is expected because feedforward motor commands and transcortical feedback responses are part of the same control system implemented in common neural circuits [10, 11].

An important avenue of future research is determining which neural circuits underlie shared learning during feedforward and

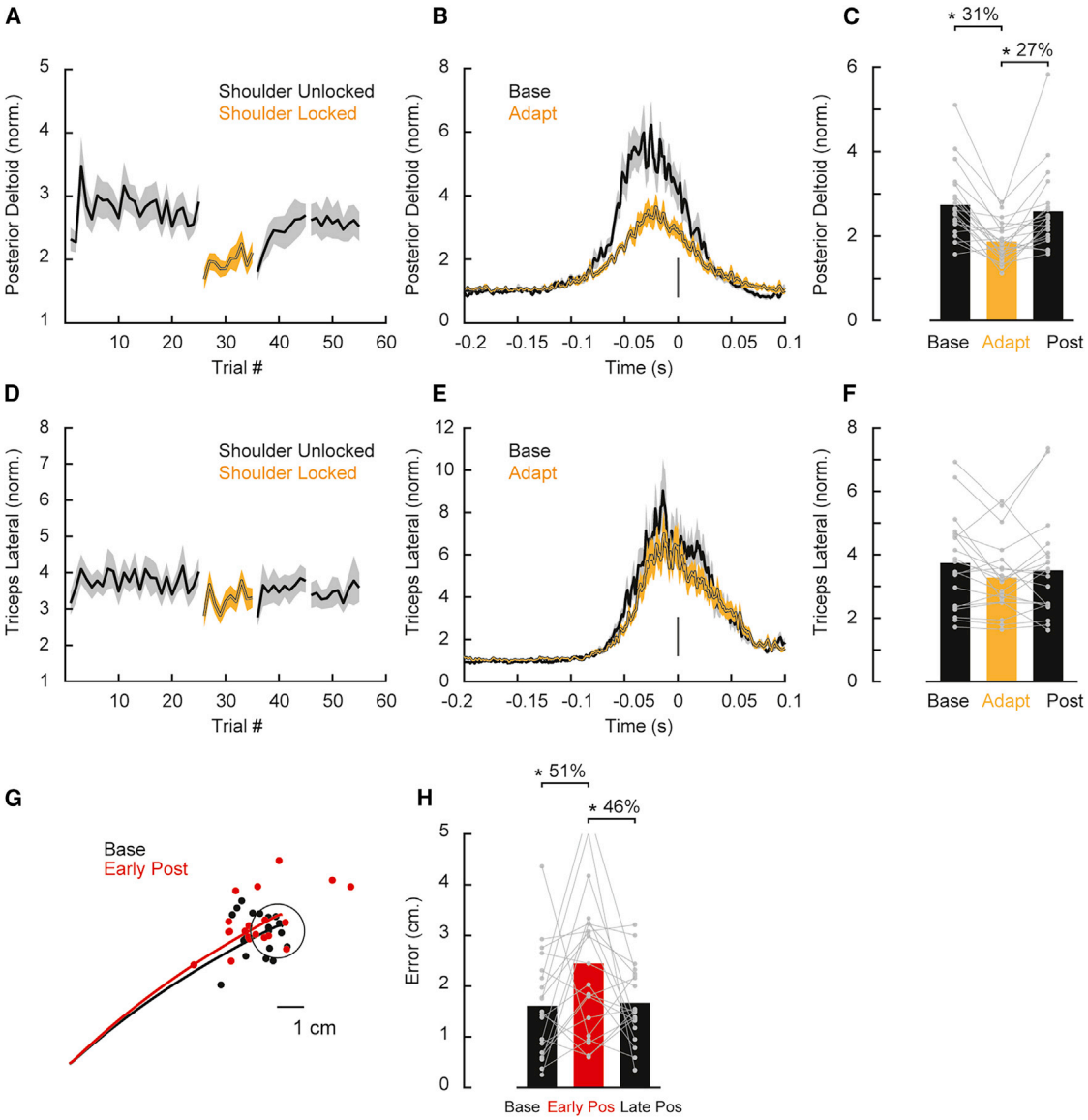

Figure 4. Transfer to Reaching

(A) Average of the shoulder posterior deltoid muscle activity in a fixed time window (−100 to 100 ms relative to movement onset) across reaching probe trials. Shaded error areas represent the standard error of the mean. EMG data are normalized as described in the STAR Methods.

(B) Time series of shoulder posterior deltoid normalized muscle activity during elbow extension reaching trials averaged over the last 10 baseline and adaptation probe trials. Shaded error areas represent the standard error of the mean. Data are aligned on movement onset.

(C) Average of the posterior deltoid muscle activity in a fixed time window (−100 to 100 ms relative to movement onset) associated with reaching probe trials late (last 10 trials) in the baseline, adaptation, and post-adaptation. Each dot represents data from a single participant. Asterisks indicate reliable effects ($p < 0.05$, see main text).

(D–F) Data for the elbow triceps lateral muscle activity are shown using the same format as (A)–(C).

(G) Average hand trajectories late in the baseline (3 trials) and early in the post-adaptation trials (first 3 trials). Each dot represents data from a single participant at 80% of the movement between movement onset and offset (see STAR Methods).

(H) Average error relative to the center of the target at 80% of the movement between movement onset and offset in the last 3 trials in the baseline, first 3 trials early in the post-adaptation, and last 3 trials late in post-adaptation phases. Each dot represents data from a single participant ($p < 0.05$, see main text).

Related to Figure S3 and S4.

feedback control [for review, see 11]. One likely common node is the primary motor cortex. Previous work has shown that neurons in the primary motor cortex are engaged during both self-initiated reaching actions and following mechanical perturbations applied to the arm [32, 54–63]. In addition, recent studies have demonstrated that the primary motor cortex is causally involved in the compensation for the arm's intersegmental dynamics during both self-initiated reaching actions [64] and in the context of externally applied mechanical perturbations [32]. Primary motor cortex activity is also modified during motor learning in the

context of self-initiated reaching [63, 65–67], although, to our knowledge, no studies have definitively linked the primary motor cortex to the learning process itself. Another likely common node, especially for the learning process, is the cerebellum, which is richly interconnected with the primary motor cortex [68]. Cerebellar circuits are strongly implicated in multi-joint coordination during both feedforward control and feedback responses [68–73] and have long been hypothesized to house the computations related to internal models [38, 74].

It is also possible that the learning itself, although mediated by cortical or cerebellar structures, is implemented at the level of the spinal cord, as both feedforward and feedback motor commands must ultimately pass through spinal interneurons and motorneurons projecting to the muscle. In an elegant set of experiments, Wolpaw and colleagues have studied the operant conditioning of the H reflex, the electrically induced analog of the spinal stretch reflex, and its contribution to rehabilitation following spinal cord injury [for review, see 75]. They have shown that such conditioning produces multisite changes at the level of the spinal cord that drive the observed differences in the H-reflex response, including a shift in motorneuron firing threshold [76] and a change in the number of GABAergic terminals [77]. Importantly, successful operant conditioning of the spinal cord circuit itself requires a functional corticospinal tract and sensorimotor cortex as well as the cerebellum and inferior olive but no other major ascending or descending spinal pathways [78–84], indicating that cerebellar contributions by the sensorimotor cortex (as opposed to the rubrospinal tract) are critical for implementing learning [80, 81]. Given the differences between H-reflex operant conditioning, especially with respect to its development over weeks and months, an extremely long timescale even relative to the slow learning in our paradigm, it is unclear whether the same mechanisms are in play for the type of learning we report here. However, at the very least, the general concept and experimental approach serve as a useful roadmap for examining the plastic changes associated with learning new intersegmental dynamics and how such learning is commonly implemented by feedforward and feedback control systems.

There are two important caveats of this study that should be emphasized and should motivate future work. First, one goal of our experimental design was to minimize or eliminate the engagement of voluntary responses to mechanical perturbation in the learning process so that we could attribute any observed learning to the neural mechanisms that generate feedback responses rather than mechanisms that generate voluntary motor commands. We did this by using very short duration perturbations (20 ms) and instructing participants to not intervene. These methods have been previously shown to elicit long-latency stretch reflexes (50–100 ms post-perturbation) while reducing or eliminating associated voluntary responses (>100 ms post-perturbation) [14–26, 85]. Our paradigm yielded very little muscle activity in the voluntary response epoch, and we found no reliable decrease in muscle activity after 100 ms post-perturbation with shoulder fixation (Figures S1A and S1B). That said, we cannot definitively establish that participants did not engage voluntary responses, as we have no direct or independent measurement of these responses. Second, although we found a reliable correlation between reflex adaptation and feedforward adaptation, causally linking the reduction of the long-latency stretch reflex to the reduction of the feedforward motor commands is not possible without more invasive methods, likely in an animal model.

STAR★METHODS

Detailed methods are provided in the online version of this paper and include the following:

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SUPPLEMENTAL INFORMATION

Supplemental Information can be found online at https://doi.org/10.1016/j.cub.2020.03.005.

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AUTHOR CONTRIBUTIONS

R.S.M. and J.A.P. contributed to the study design. R.S.M. conducted the experiments and analyzed the data. R.S.M. wrote the manuscript with input from J.A.P and P.L.G. All authors revised the final version of the manuscript.

DECLARATION OF INTERESTS

The authors declare no conflict of interest, financial or otherwise.

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STAR★METHODS

KEY RESOURCES TABLE

REAGENT or RESOURCE	SOURCE	IDENTIFIER
Deposited Data		
Data and Code	This manuscript	https://doi.org/10.6084/m9.figshare.11914539
Software and Algorithms		
KINARM exoskeleton	KINARM [86]	https://kinarm.com/kinarm-products/kinarm-exoskeleton-lab/
MATLAB 2018a	Mathworks	RRID:SCR_001622
R version 3.2.1	[87]	RRID:SCR_001905

LEAD CONTACT AND MATERIALS AVAILABILITY

Further information and requests for resources should be directed to and will be fulfilled by the Lead Contact, Andrew Pruszynski (andrew.pruszynski@uwo.ca). This study did not generate new unique reagents.

EXPERIMENTAL MODEL AND SUBJECT DETAILS

Seventy healthy human participants (aged 17–39, 40 females) took part in this study. Participants self-reported that they were right-handed and free from visual, neurological, or musculoskeletal deficits. All participants were naive as to the purpose of the study, were free to withdraw at any time, and provided written informed consent before participating. The Office of Research Ethics at Western University approved this study.

METHOD DETAILS

Apparatus

Participants performed the experiments with a robotic exoskeleton (KINARM, Kingston, ON, Canada). As described in previous studies [34, 85, 86], this device allows for flexion and extension movement of the shoulder and elbow joints in the horizontal plane, and can independently apply torque loads at these joints. Target lights and hand cursor feedback cursor were presented in the same plane as the movement using an overhead LCD monitor and a semi-silvered mirror. Direct vision of the arm was occluded with a physical shield. To ensure a comfortable and tight coupling between each participant's arm and the robot, the two segments of the exoskeleton robot (upper arm and forearm) were adjusted for each participant arm and the spaces were filled with a firm foam. Lastly, the robot was calibrated so that the projected hand cursor was aligned with each participant's right index finger.

Main experimental task and general protocols

Twenty participants used a robotic apparatus and received 20 ms torque pulse perturbations that caused pure elbow motion with the shoulder joint free to move and with the shoulder fixed by the robotic manipulandum (altered arm dynamics). Before and after learning with the shoulder fixed, participants performed twenty-degree elbow extension movements (probes).

In the beginning of a trial, participants were instructed to keep their hand in a home target (white circle, 0.6 cm diameter) which required shoulder and elbow angles of 40 ° and 80 ° (external angles), respectively (Figure 1A). After a random period (250-500 ms, uniform distribution), a background load (+2 Nm) was slowly introduced (rise time = 500ms) to the elbow joint to ensure baseline activation of shoulder and elbow extensor muscles [88]. After an additional random hold period (1-2 s, uniform distribution), a torque pulse of 20 ms duration (i.e., perturbation) was applied to the shoulder and elbow joints (+/−2 Nm at each joint over and above the background torque). At the same time, the home target and hand feedback were turned off and participants were instructed to not intervene with the perturbation. Critically, we chose this combination of shoulder and elbow loads to minimize shoulder motion [see 14, 27, 28, 30]. Within a random period (300-500 ms, uniform distribution), a servo-controller brought the participant's arm back to the home location, and the same procedure was repeated for a new trial. The servo-controller was implemented as a stiff, viscous spring and damper (K = 500 N/m and B = 250 N/(m/s)) with ramp up time of 100ms and trajectory duration of 500ms. Again, participants were instructed to not intervene with the servo (Figure 1B, left and middle columns).

In some trials, participants were required to perform reaching movements (probes). Reaching trials occurred before learning with the shoulder joint unlocked, after learning with the shoulder locked, and after learning with the shoulder unlocked. In these trials, participants started by also keeping their hand in the same home target (red circle, 0.6 cm diameter). After a random hold period (250 –500 ms, uniform distribution), a goal target (white circle: 3 cm diameter) was presented in a location that could be reached with a 20° pure elbow extension movement. Participants were required to remain at the home location for an additional random

period (250 – 500 ms, uniform distribution) so that the goal target turned red, the hand feedback cursor was turned off and participants were allowed to start the movement. Participants were instructed to move to the goal target with a specific movement speed. The goal target turned green when movement time (from exiting the home target to entering the goal target) was between 100 and 180 ms, orange when it was too fast (< 100 ms) and red when it was too slow (> 180 ms). No restrictions were placed on movement trajectories. Participants were required to remain at the goal target for an additional 500 ms to finish a trial. The hand feedback cursor turned back on when the participant's hand entered the goal target or after a fixed time window of 500ms from the cue to start of the movement (Figure 1B, right column). After a random period (300-500 s, uniform distribution), the servo-controller, as described above, moved the participant's arm back to the home location. In ten percent of reaching trials, the background torque turned on, remained on for the same time period (1.0–2.5 s, uniform distribution), but then slowly turned off. In these trials, participants were still required to perform the reaching movements after background load turned off. These trials ensured that background load was not predictive of perturbation trials [5]. The order of all perturbation and reaching trials was randomized in the baseline and post adaptation phases. There were only perturbation trials in the adaptation phase which were randomized in terms of timing and direction.

Participants first completed a total of 125 baseline trials (100 mechanical perturbations and 25 reaching, randomized), with the shoulder joint free to move. We then locked the shoulder joint with the insertion of a physical pin into the shoulder joint of the robotic manipulandum, and participants completed 500 perturbation trials (adaptation phase) and 10 reaching trials (probes) with the shoulder joint locked. We then unlocked the shoulder joint and participants completed 10 reaching movements with the shoulder joint unlocked. Lastly, participants completed a total of 110 post-adaptation trials (100 mechanical perturbations and 10 reaching, randomized), (post-adaptation phase) (Figure 1C).

The main experiment lasted about 2h. Rest breaks were given throughout or when requested. Before starting, participants completed practice trials until they expressed that they understood the instructions and comfortably achieved ~90% success in reaching trials (approx. 10 min).

Control experiments

Twenty additional participants performed the same version of the main experiment but experienced mechanical perturbations of 100ms load durations. This served as a control to contrast changes in the voluntary response when it is engaged in the learning task with shoulder fixation.

Twenty additional participants performed the same version of either the 20 ms or 100 ms load duration experiments but without locking the shoulder joint. This served as a control to rule out changes that could be caused by extensive exposure to perturbations rather than learning associated with the shoulder fixation manipulation.

Ten additional participants performed the same version of the main experiment with 20 ms perturbation duration but experienced only background loads without perturbations with the shoulder locked. This served as a control to rule out changes that could be caused by extensive exposure to background loads rather than learning and transfer from feedback responses.

All control experiments lasted about 2h. Rest breaks were given throughout or when requested. Before starting, participants completed practice trials until they expressed that they understood the instructions and comfortably achieved ~90% success in reaching trials (approx. 10 min).

QUANTIFICATION AND STATISTICAL ANALYSIS

Kinematic recordings and analysis

We recorded movement kinematics (i.e., hand position, and joint angles) with the robotic device at 1000 Hz and then low-pass filtered offline (12 Hz, 2-pass, 4th-order Butterworth). Data from perturbation trials was aligned on perturbation onset and data from reaching trials was aligned on movement onset, defined as 5% of peak elbow angular velocity [see 5, 30, 33]. We quantified aftereffects of reaching movements following shoulder fixation by calculating hand path errors relative to the center of the target at 80% of the movement between movement onset and offset (also defined at 5% from the peak angular elbow velocity). This window was used to select the kinematic traces before any corrections [5].

EMG recordings and analysis

Electromyographic signals (EMG) were amplified (gain = 10^3) and digitally sampled at 1000 Hz (Delsys Bagnoli-8 system with DE-2.1 sensors, Boston, MA). EMG surface electrodes were used and placed on the skin surface on top of the belly of five upper limb muscles (pectoralis major clavicular head, PEC, shoulder flexor; posterior deltoid, PD, shoulder extensor; biceps brachii long head, BB, shoulder and elbow flexor, Brachioradialis, BR, elbow flexor; triceps brachii lateral head, TR, elbow extensor). Before electrode placement, the participant's skin was prepared with rubbing alcohol, and the electrodes were coated with conductive gel. Electrodes were placed along the orientation of muscle fibers. A reference electrode was placed on the participant's left clavicle. EMG data were band-pass filtered (20–500 Hz, 2-pass, 2nd-order Butterworth) and full-wave rectified offline.

First, we investigated whether feedback responses adapt to the novel arms dynamics following shoulder fixation. To assess whether the long latency stretch response of shoulder and elbow extensor muscles account for and adapt over time to novel arm's dynamics, we binned the EMG data into previously defined epochs [see 85]. These epochs included a pre-perturbation epoch (PRE, −50-0 ms relative to perturbation onset), the long-latency stretch response (R2/3, 50-100 ms), and the voluntary response

(VOL, 100-150ms). We calculated the difference in these epochs between flexion and extension perturbation trials and over all trials. This approach ensured that perturbation direction was unpredictable from trial to trial and allowed us to include all perturbation trials in the analysis [5].

We also investigated whether shoulder and elbow muscles during pure elbow reaching adapt after learning novel arm dynamics following shoulder fixation during perturbation trials. To compare the changes in amplitude of muscle activity before and after learning, we calculated the mean amplitude of phasic muscle activity in a fixed time-window, −100 ms to +100 ms relative to movement onset [see 5, 30, 89]. These windows were chosen to capture the agonist burst of EMG activity in each of the experiments.

Data from perturbation trials were normalized by the pre-perturbation activity, which was the activity required to compensate for a 2 Nm constant load. Data from reaching trials were normalized by a pre-activity, which was the activity required to compensate for a 1Nm constant load in normalization trials performed prior to the experiments [see 5, 30, 85]. Data processing was performed using MATLAB (r2018a, Mathworks, Natick, MA).

Statistical analysis

Statistical analyses were performed using R (v3.2.1, R Foundation for Statistical Computing, Vienna, Austria). We performed different statistical tests (e.g., repeated-measures ANOVA with Tukey tests for multiple comparisons, t tests, and regression analysis), when appropriate for each experiment. Further details of these analyses are provided in the Results. Experimental results were considered statistically significant if the corrected p value was less than < 0.05.

DATA AND CODE AVAILABILITY

Data and code supporting the current study is available at: https://doi.org/10.6084/m9.figshare.11914539.