

Long-Latency and Voluntary Responses to an Arm Displacement Can Be Rapidly Attenuated By Perturbation Offset

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Kurtzer I, Pruszynski JA, Scott SH. Long-latency and voluntary responses to an arm displacement can be rapidly attenuated by perturbation offset. *J Neurophysiol* 103: 3195–3204, 2010. First published March 24, 2010; doi:10.1152/jn.01139.2009. Feedback control of our limbs must account for the unexpected offset of mechanical perturbations. Here we examine the evoked activity of elbow flexor and extensor muscles to torque pulses lasting 22–152 ms and how torque offset impacts activity in the long-latency (45–100 ms) and voluntary epochs (120–180 ms). For each pulse width, we found a significant attenuation of muscle activity ~30 ms after the offset of torque compared with when the torque was sustained. The brief time between the offset of torque and the attenuation of muscle activity implicates group I afferents acting through a spinal pathway, because this route is the only one fast enough and short enough to be responsible. Moreover, elbow muscle activity in the subsequent 20–45 ms following torque-offset was ~35% smaller than when the torque was sustained. These results show that a fast spinal process can powerfully attenuate corrective responses of the arm to a torque perturbation.

INTRODUCTION

Feedback corrections are a key mechanism for achieving successful behavior when interacting with an uncertain environment. For example, one must stabilize an umbrella against unpredictable gusts of winds to stay dry, pull back against a wrestling opponent's tug to stay upright, and maintain the orientation of steering wheel against the bumps in the road to stay in one's lane. The sustained application of such mechanical perturbations evokes a stereotyped sequence of activity in the stretched arm muscles—R1 response (~20–45 ms), R2/3 response (~45–100 ms), and voluntary response (>100 ms) (Lee et al. 1983; Marsden et al. 1983; Pruszynski et al. 2008). The R1 response (also called the short-latency reflex) can be confidently ascribed exclusively to spinal circuitry because of its highly rapid action (Pierrot-Deseilligny and Burke 2005). In contrast, the later R2/3 response (traditionally called the long-latency reflex) and voluntary response likely include contributions from fast afferent passing information over a distant supraspinal circuit (Day et al. 1991; Everts and Tanji 1976; Lewis et al. 2004) and/or slower afferents (like group II muscle spindles) passing information over a short spinal circuit (Cody et al. 1987; Hendrie and Lee 1978).

Although most research has characterized how feedback corrections are related to the unexpected onset of a perturbation, feedback corrections must also be tailored to the unexpected offset of a perturbation. For example, it would be maladaptive to make

a sustained correction to a gust of wind or preprogram a “triggered reaction” (Crago et al. 1976) because these perturbations have a highly variable duration. Studies that have examined how feedback corrections incorporate perturbation offset consistently report that an imposed displacement lasting under ~30 ms will elicit an R1 response but not an R2/3 response (Ghez and Shinoda 1978; Lee and Tatton 1982; Lewis et al. 2005; Schuurmans et al. 2009). The brief time (15–25 ms) between the displacement's termination (<30 ms) and its impact on muscle activity (~45 ms) implicates group I afferents acting through a spinal pathway because this route is the only one fast enough and short enough to be responsible.

This powerful all-or-none phenomenon does not address how perturbation offsets after 30 ms will influence feedback corrections, which is an important issue for understanding how feedback control handles unexpected perturbation offsets. Possibly, the discrete expression of the R2/3 and voluntary response is maintained throughout the entire epoch so that, once elicited, the responses are unaffected by torque offset over a significant time period, i.e., these events comprise relatively discrete units committed as a whole. Another possibility is that a fast spinal mechanism can significantly attenuate muscle activity at short-latency throughout the R2/3 and voluntary epochs.

This study tested between these two competing possibilities with a straightforward paradigm of applying elbow torques with unpredictable duration (22–152 ms) and examining elbow muscle activity following torque-offset. Accordingly, muscle activity was predicted to either attenuate at a similar rapid latency following each torque offset or show longer periods that were insensitive to torque offset.

METHODS

Subjects

A total of 31 subjects participated in the three experiments (18 males and 13 females; mean age = 26.4 yr) and were paid for their participation. Procedures were approved by the ethics committee at Queen's University.

Apparatus

As described in earlier studies (Kurtzer et al. 2008; Pruszynski et al. 2008; Scott 1999), we used a robotic exoskeleton (KINARM, BKIN Technologies, Kingston, ON, Canada), which permits flexion/extension movements of the shoulder and elbow in the horizontal plane and can selectively apply torques to each joint. Visual targets and a hand-aligned cursor were presented in the same plane as the limb movement while direct vision of the arm was blocked (Fig. 1A).

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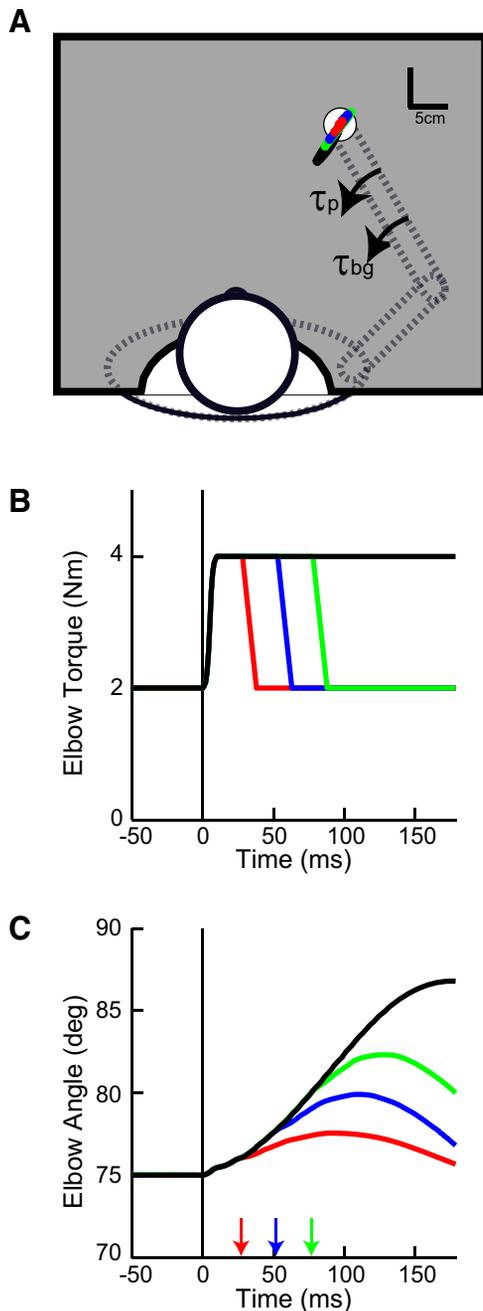


FIG. 1. Summary of experimental design. A: cartoon of subject's initial arm posture with their hand in the small visual target (drawn to scale). The imposed background torque and perturbation torque are indicated by τ_{bg} and τ_p . Colored traces show the mean hand path following 4 different durations of imposed flexor torque in *experiment 1*; red, blue, green, and black traces signify data from torque pulses ending at 27, 52, 77, and 1,500 ms. B: 4 different durations of flexor torque with data aligned to perturbation onset: flexion torque is positive and extension torque is negative. C: pattern of elbow flexion motion for the 4 torque conditions described above with arrows for corresponding time of torque offset; 0° is complete elbow extension.

Muscle recording

We recorded surface EMGs (DE-2.1, Delsys, Boston, MA) from two elbow muscles of the right arm, brachioradialis (an elbow flexor) and triceps lateral (an elbow extensor), using procedures fully described in earlier papers (Kurtzer et al. 2008; Pruszynski et al. 2008).

Tasks

All experiments involved the subject stabilizing their right arm against torque pulses of unpredictable duration so we could examine how torque-offset impacted the evolving pattern of muscle activity. The experiments differed in two significant ways. First, the duration of the torque pulses varied across experiments. Second, we only examined the elbow flexor muscle in *experiment 1*, whereas the elbow extensor muscle was examined in all experiments. Other changes in the protocol—different elbow angle (75 and 90°), different comparison torque durations (75 and 1,500 ms), and different trial repeats (30–45)—make the experimental arc less tidy but are unlikely to impact the main results in a significant way.

EXPERIMENT 1. Seventeen subjects participated in this experiment. They were instructed to counter the perturbations quickly and accurately but not anticipate their onset or offset. Visual feedback of the hand cursor was always removed before torque onset so that corrective actions were guided entirely by proprioception.

Each trial proceeded in the same order. 1) An elbow torque was slowly ramped from 0 to ± 2 Nm to elicit steady-state activity of the elbow flexor or extensor muscle, respectively. 2) Subjects stabilized their hand-aligned cursor (0.4 cm radius) within the center of a small visual target area (2 cm radius). The target placement required an elbow angle of 75 (6 subjects) or 90° (11 subjects) and a shoulder angle of 45°; the two configurations did not alter the pattern of results. During a random hold period (between 500 and 3,500 ms), the cursor was extinguished and remained off for the remainder of the trial. 3) An additional elbow torque (± 2 Nm) was rapidly applied in the same direction as the background torque. The duration of this torque pulse was randomly varied from trial to trial. Note that the reported torque duration was determined from an accelerometer tightly coupled to the arm trough to account for any small delays (~ 10 ms) between the commanded torque and induced motion. 4) Subjects needed to return to the target within 500 ms and remain within it. The target circle was filled green or red if their performance was accurate or inaccurate, respectively. 5) Finally, the background load slowly ramped back to zero (500 ms) and remained zero for a brief intertrial period (1,000 ms).

Experiment 1 used three torque pulse durations (27, 52, and 77 ms) that broadly spanned the R2/3 epoch, as well as a step torque lasting 1,500 ms. Thirty repeats were collected for each condition.

EXPERIMENT 2. Eight subjects participated in this experiment, which examined activity of the elbow extensor muscle. Here we used 12 torque durations (ranging between 22 and 77 ms in 5 ms increments) to examine the impact of torque offset on a fine time scale within the R2/3 epoch. Forty repeats were collected for each condition. Target placement required an elbow angle of 90° and a shoulder angle of 45° for all subjects.

EXPERIMENT 3. Ten subjects participated in the final experiment, which examined activity of the elbow extensor muscle. *Experiment 3* used three torque pulses (102, 127, and 152 ms) that broadly spanned the voluntary epoch and a step torque lasting 1,500 ms. Forty-five repeats were collected for each condition. Target placement required an elbow angle of 90° and a shoulder angle of 45° for all subjects.

Data analysis

Angular positions of the shoulder and elbow were low-pass filtered (25 Hz, 2-pass, 6th-order Butterworth). EMG signals were amplified (gain = 1–10 K), band-pass filtered (20–450 Hz), digitally sampled at 1,000 Hz (PCI 6071E, National Instruments, Austin, TX), rectified, and normalized by each muscle's mean activity when countering the background elbow torque before the perturbation (200–0 ms before perturbation onset, preperturbation).

We considered three different time epochs—R1 = 20–45 ms; R2 = 45–75 ms; R3 = 75–100 ms; although researchers often collapse across

the R2 and R3 epochs, we considered them separately to achieve better temporal resolution. A critical difference here is that our time epochs are measured relative to torque offset rather than the relative to torque onset. To avoid confusion, we denote these epochs as $R1_{\text{off}}$, $R2_{\text{off}}$, and $R3_{\text{off}}$.

All analyses contrasted different patterns of muscle activity following torque offset. Importantly, muscle activity with brief torque pulses was often compared with the muscle activity occurring with the longest torque duration: *experiment 1* (1,500 ms), *experiment 2* (77 ms), and *experiment 3* (1,500 ms). Hence, the baseline pattern comprised a complex time-varying pattern rather than a tonic level of activity that occurs in the preperturbation epoch.

For *experiments 1* and *3*, we used ANOVAs to analyze the impact of torque-offset on each postoffset epoch. Repeated two-way ANOVAs compared the muscle activity when the torque was removed versus the baseline activity when the torque was sustained (factor 1: torque off vs. sustained/1,500 ms; factor 2: 3 torque-offset times: 27, 52, and 77 ms). Thereby, we could examine the impact of torque offset on a particular postoffset epoch across all torque-offset times (e.g., main effect for $R1_{\text{off}}$ following torque offsets at 27, 52, and 77 ms) while parsing out the complex time-varying pattern of muscle activity. The impact of torque-offset (relative to the condition with longest torque duration/1,500 ms) was separately determined for each postoffset time epochs ($R1_{\text{off}}$, $R2_{\text{off}}$, and $R3_{\text{off}}$).

Repeated one-way ANOVAs tested whether the change between torque-off and torque-sustained conditions differed between the three postoffset epochs (factor 1: $R1_{\text{off}}$, $R2_{\text{off}}$, and $R3_{\text{off}}$). The impact of torque-offset over time was separately tested for each torque pulse duration (e.g., $R1_{\text{off}}$, $R2_{\text{off}}$, and $R3_{\text{off}}$ following torque offset at 27 ms).

The final epoch-based analysis used a paired *t*-test to determine the impact of torque-offset with a particular torque pulse and postoffset epoch. In all these analyses, we averaged each subject's data across trial repeats. Significant effects were judged at the $P < 0.05$ level. Finally, note that epoch-based analyses were inappropriate for *experiment 2* as the longest torque duration (77 ms) evoked a transient burst of muscle activity, which decays to preperturbation levels by 100 ms. Hence, the changes from this transient baseline following torque-offset were also transient and provided little information for prolonged epochs.

In contrast to analyzing muscle activity in predefined epochs, a different analysis examined the moment-by-moment impact of torque offset on muscle activity. This analysis was used for all three experiments. Receiver operator curves (ROC) curves (Green and Swets 1966; Pruszynski et al. 2008) determined the probability that an ideal observer could discriminate between activity in trials where the torque was removed and trials where the torque was sustained. The longest torque duration was always used for the sustained comparison condition: *experiment 1*, 1,500 ms; *experiment 2*, 77 ms; *experiment 3*, 1,500 ms. This procedure was conducted at each millisecond sample and yielded a time-varying probability of discrimination for each subject. The n probability trajectories for n subjects in each experiment were averaged together to produce a group probability trajectory. The first impact of torque-offset on muscle activity was identified when this average probability of discrimination decreased from 0.5 (equal likelihood) to 3 SD below its nominal variability (25 ms before torque-offset to 10 ms after torque-offset) and remained below this threshold for 5 consecutive samples/ms.

RESULTS

Experiment 1

Our first experiment imposed elbow torques of four different durations ranging from 27 to 1,500 ms. Greater amounts of elbow motion and longer times for reversal occurred with an increased duration of imposed torque (Fig. 1, *B* and *C*). For example, the briefest and longest flexor torque conditions

caused peak flexion displacements of 2.0 (SD 0.3) and 9.6° (SD 2.2), respectively, and times of peak displacement at 84 (SD 3) and 182 ms (SD 19), respectively; a similar pattern was obtained with torques acting in the extension direction.

The torque perturbations reliably elicited stretch-related activity in the elbow muscles. This is most obvious with the longest torque condition, which elicited bursts of muscle activity in successive epochs ($R1$, $R2/3$, and voluntary responses), although the detailed shape of evoked activity differed between elbow flexors and extensors (see black traces in Fig. 2). Figure 2*A* shows the evoked activity of an exemplar elbow extensor. Initially, there are overlapping patterns of activity for the briefest torque duration (red trace) and longest torque duration (black trace) because the nervous system received the same initial pattern of sensory information. However, the torque-offset at 27 ms (see red arrow) led to a decrease in muscle activity ~30 ms later relative to the condition with more prolonged torque. This exemplar muscle also showed a relative decrease in activity ~30 ms after the torque-offset at 52 (blue trace/blue arrow) and 77 ms (green trace/green arrow). Such rapid decreases in muscle activity were visibly present for all torque-offsets and for both elbow muscles (Fig. 2, *A–D*).

To better visualize the pattern of decreasing muscle activity, we realigned the data to the torque-offsets at 27, 55, and 77 ms, respectively. Figure 3, *A–C*, presents data from the exemplar muscle in Fig. 2*A*, and Fig. 3, *D–F*, presents data from the exemplar muscle in Fig. 2*B*. In all cases, torque-offset resulted in a decrease in muscle activity (relative to the condition with

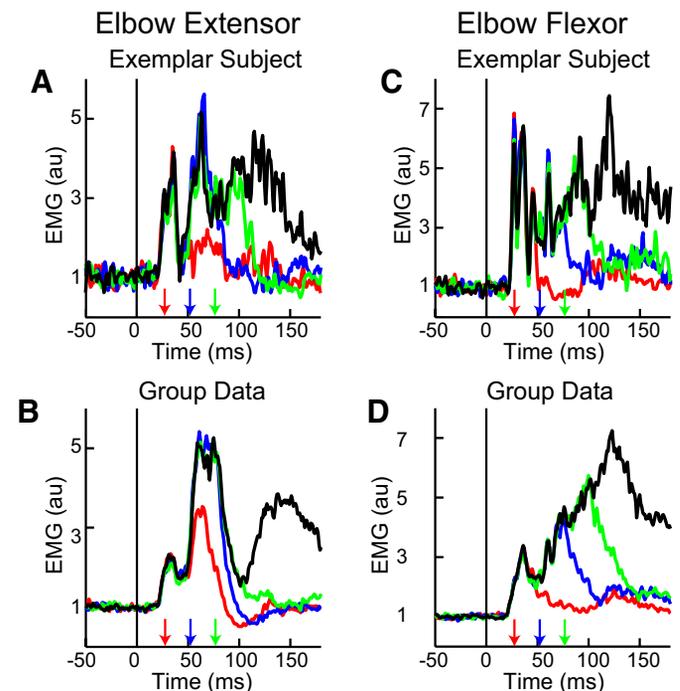


FIG. 2. Muscle activity following the 4 torque perturbations of *experiment 1*. *A*: colored traces show evoked activity in representative subject's elbow extensor muscle: red, blue, green, and black traces signify data from torque pulses ending at 27, 52, 77, and 1,500 ms. Colored arrows are aligned to the different times of torque offset. *B*: activity of the elbow extensor muscle averaged across subjects ($n = 12$). *C* and *D*: evoked activity of the elbow flexor muscle from a representative subject and the group mean ($n = 11$; same format as above).

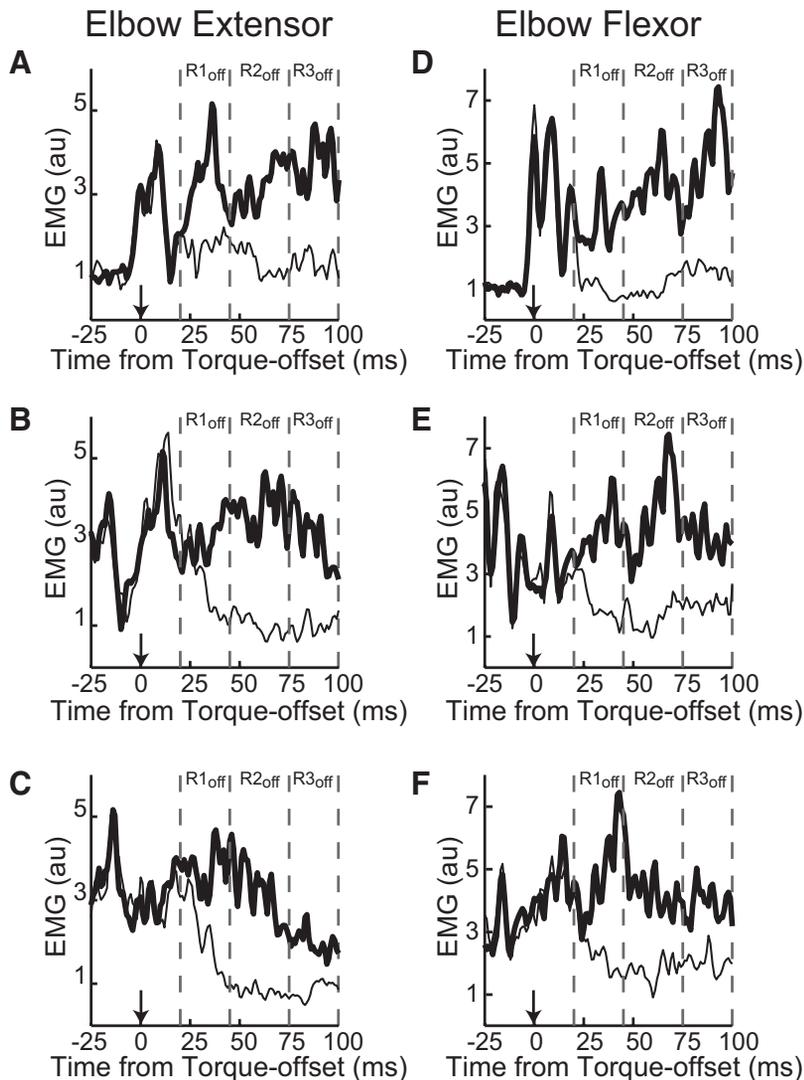


FIG. 3. Muscle activity realigned to torque-offset. A: activity of the exemplar elbow extensor muscle for torque perturbations lasting 27 (thin black line) and 1,500 ms (thick black line) realigned to the perturbation offset; the data are taken from the red and black traces of Fig. 2A, but time-shifted to highlight when the muscle activity differs between conditions. Dashed vertical lines indicate 3 time epochs relative to torque offset: R1_{off}, R2_{off}, and R3_{off}. B and C: muscle activity of the same exemplar extensor muscle realigned to the perturbation offsets at 52 (blue trace of Fig. 2A) and 77 ms (green trace of Fig. 2A), respectively. The data are in the same format as above. D–F: same format is used to plot data from the representative elbow flexor muscle of Fig. 2C.

the longest torque duration/1,500 ms) beginning within the R1_{off} epoch and continuing past the R3_{off} epoch.

We quantified the change in muscle activity by determining the mean activity within each postoffset epoch, R1–R3_{off}. The data for each subject and both muscles are shown in Fig. 4. In almost every case, there is a downward slope connecting the black dots and white dots, which indicates a decrease in activity following torque offset relative to the same time window when the torque was prolonged.

A two-way ANOVA on the R1_{off} epoch yielded a significant main effect of torque presence (off vs. sustained/1,500ms) for the elbow extensor muscle ($P < 0.001$, $F_{(1,22)} = 73.5$). Hence, a reliable short-latency decrease in elbow extensor activity occurred across all three torque-offset times. Similar results were present with later epochs ($P < 0.001$, $F_{(1,22)} = 72.7$ and 60 for R2_{off} and R3_{off}, respectively). The elbow flexor muscle also showed a significant main effect of torque offset for all three epochs ($P < 0.001$, $F_{(1,20)} = 46.6$, 34.5, and 47.8 for R1_{off}, R2_{off}, and R3_{off}, respectively). Moreover, both muscles showed a significant decrease in activity for each torque-offset and postoffset epoch ($P < 0.05$, 1-way t -test): elbow extensor t -values ($df = 11$) ranged between -3.5 and -9.1 ; elbow flexor t -values ($df = 10$) ranged between -4.6 and -6.8 .

The impact of torque offset tended to increase over time where a larger decrease was present in later epochs than earlier epochs. A one-way ANOVA of elbow extensor activity following torque offset at 27 ms showed that the R1_{off}, R2_{off}, and R3_{off} epochs had significantly different decreases in activity ($P < 0.05$, $F_{(2,22)} = 3.9$). Similar results were present for the torque offset at 52 ($P < 0.001$, $F_{(2,22)} = 15$) and 77 ms ($P < 0.005$, $F_{(2,22)} = 9.2$). Significant between-epoch differences were also present for the elbow flexor muscle ($P < 0.001$, $F_{(2,20)} = 11.2$ for R1_{off}; $P < 0.001$, $F_{(2,20)} = 13.1$ for R2_{off}; $P < 0.005$, $F_{(2,20)} = 9.1$ for R3_{off}).

Given the two muscle's similar pattern of torque-offset response, we examined the magnitude of the effect for the combined data set. Accordingly, the evoked activity in the R1_{off} epoch was 280% above the preperturbation activity when the torque was sustained versus 168% of preperturbation activity when the torque was removed ($\Delta = 92\%$). The decrease in activity following torque offset was more prominent for the R2_{off} epoch (320 vs. 40%, $\Delta = 280\%$) and R3_{off} epoch (316 vs. 31%, $\Delta = 285\%$). This can be appreciated in Fig. 4 by examining how the white dots often hover above the preperturbation level (horizontal black line) in the R1_{off} epoch, whereas they often overlap the preperturbation level in the R2_{off} and R3_{off} epochs.

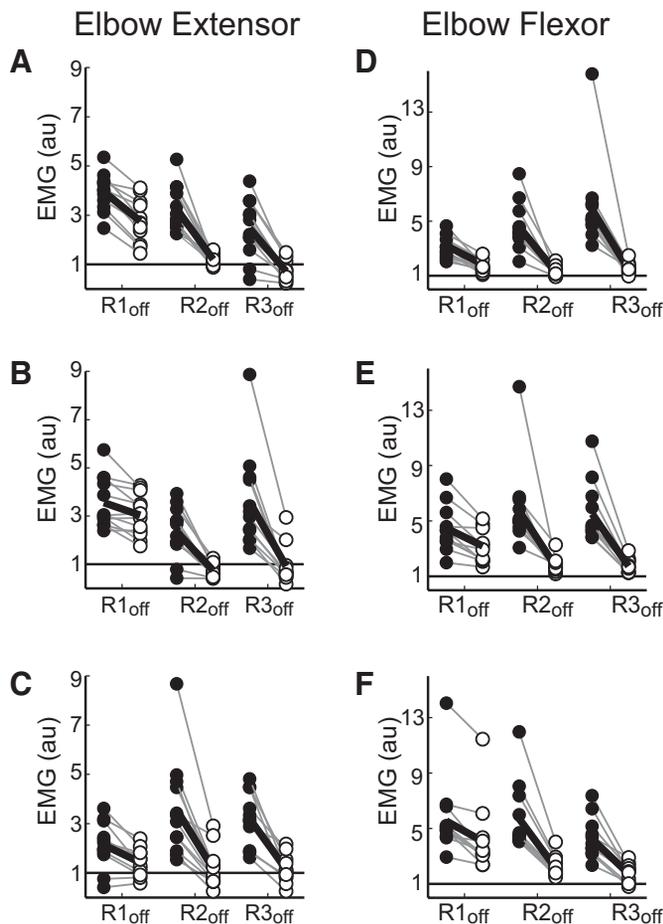


FIG. 4. Average muscle activity following torque offset. *A*: mean activity of the elbow extensor muscle within the R1_{off}, R2_{off}, and R3_{off} epochs if the torque remained on (black dots) or ended at 27 ms (white dots). Small dots and thin lines show each subject's data whereas thick lines show the group average in the 2 conditions. *B* and *C*: mean muscle activity when the torque remained on or ended at 52 and 77 ms, respectively. The data are in the same format as above. *D–F*: data for the elbow flexor muscle.

A moment-by-moment ROC analysis determined the precise timing of the decrease in muscle activity following torque-offset. For example, Fig. 5*A* shows the individual (thin gray lines) and group average (thick black line) ROC curves that discriminate between elbow extensor activity during torque-offset trials and torque-sustained trials; note that the data are realigned to the torque-offset at 27 ms. The temporally evolving ROC curves hover around 0.5 before torque-offset, which indicates a chance-level of discrimination. Following torque-offset, the individual ROC curves trend below chance level and the mean ROC curve exceeds its statistical threshold 25 ms after torque-offset (white dot). For this condition and muscle, there was a significant decrease in activity 25 ms following torque-offset, which is within the earliest response epoch (R1_{off} = 20–45 ms). Similar results occurred with all other torque offsets for the elbow extensor and flexor muscles; significant decreases in activity occurred within the R1_{off} epoch 24–32 ms after torque-offset.

Experiment 2

Our second experiment tested whether the rapid impact of torque-offset was present on a finer time scale within the R2/3

epoch (45–100 ms) or whether there were periods insensitive to changes in sensory input. The 12 torque perturbations (Fig. 6*A*) elicited the mean patterns of muscle activity shown in Fig. 6*B*. The longest torque pulse (77 ms, black solid trace) elicited the greatest amount of muscle activity in the R2/3 epoch, whereas torque pulses that ended successively earlier elicited successively less evoked activity; the least amount of evoked activity occurred with the briefest torque pulse (22 ms, red dashed trace).

The impact of torque-offset was visually evident throughout the R2/3 epoch. Our ROC analysis further determined that torque-offset consistently impacted the muscle activity at short-latency (range = 25–33 ms; Fig. 6*C*). Note that the apparent impact of torque-offset was quite transient because the longest torque pulse was 77 ms and elicited a transient burst of activity. This transient baseline pattern of activity also precluded the usefulness of ANOVAs based on binned epochs.

Experiment 3

Our final experiment used torque perturbations that lasted 102, 127, 157, and 1,500 ms (Fig. 7*A*) so that we could examine the impact of torque offset throughout the voluntary epoch (120–180 ms). Similar to *experiment 1*, the torque step evoked relatively sustained elbow extensor activity during the voluntary epoch. Also similar to *experiments 1* and *2*, there was a clear decrease in activity shortly after the torque was removed. A two-way ANOVA showed a significant contrast ($P < 0.01$) between the torque offset and torque sustained/1,500 ms condition for all three epochs ($F_{(1,18)} = 22.2, 68, \text{ and } 19.3$ for R1_{off}, R2_{off}, and R3_{off}, respectively). Each epoch also showed a significant decrease in activity following torque offset ($P < 0.05$, 1-way *t*-test; *t*-values (df = 9) ranged between -2.9 and -9.0).

The impact of torque offset became more prominent over time. A one-way ANOVA indicated significant changes between the R1_{off}, R2_{off}, and R3_{off} epochs for torque offsets at 102 ($P < 0.001, F_{(2,18)} = 36.8$), 127 ($P < 0.02, F_{(2,18)} = 5.3$), and 157 ms ($P < 0.01, F_{(2,18)} = 6.8$). On average, evoked activity in the R1_{off} epoch was 195 and 153% above the preperturbation level ($\Delta = 42\%$) when the torque was prolonged and removed, respectively. Decreases in activity were more substantial during the R2_{off} epoch (162 vs. 33% above the preperturbation level, $\Delta = 129\%$) and R3_{off} epoch (150 vs. 45% above the preperturbation level, $\Delta = 105\%$).

The ROC analysis identified a similar decrease in activity shortly following torque-offset. The mean ROC curves exceeded statistical threshold 36, 33, and 38 ms after torque-offsets occurring at 102, 127, and 157 ms, respectively. Hence, torque-offset within the voluntary epoch consistently resulted in a short-latency decrease in evoked activity from the elbow extensor muscle.

Analysis across experiments

In the three previous experiments, we detected a decrease in muscle activity from the time-varying baseline shortly following torque-offset. Here we combine the data across several experiments to determine any overall trends in the timing (Fig. 8*A*) and magnitude (Fig. 8*B*) of the decrease in muscle activity. Regressing the time when torque-offset first impacted muscle activity against the time of torque offset showed a weak

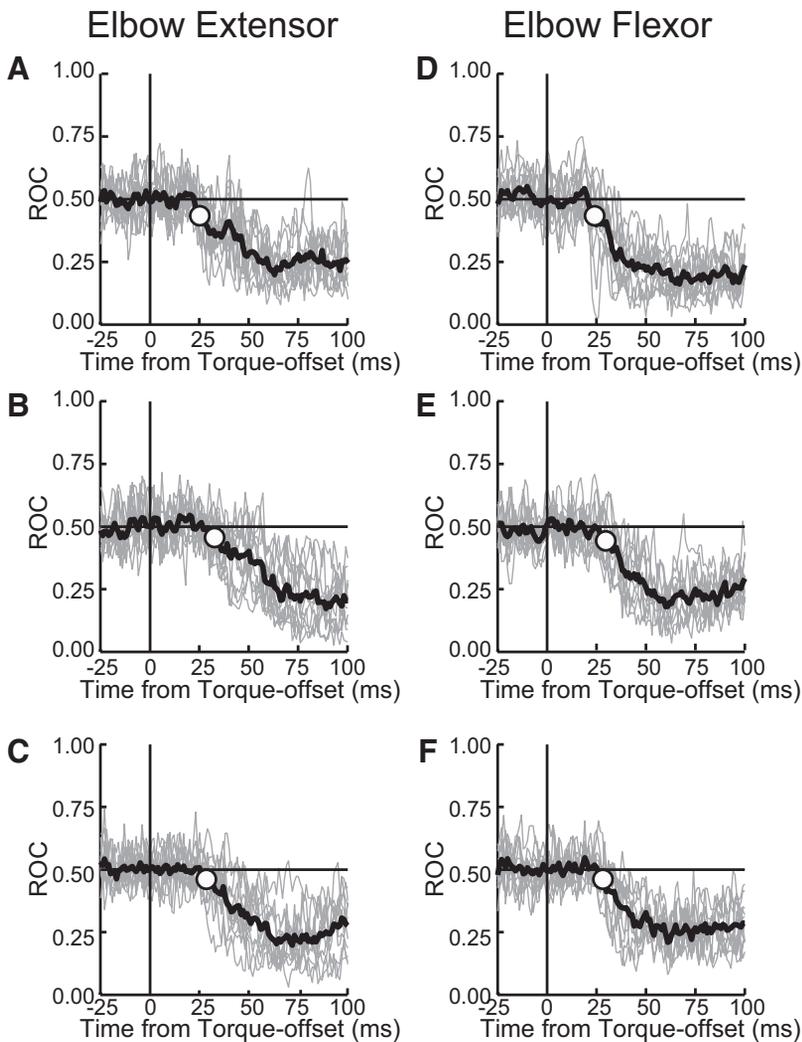


FIG. 5. Receiver operating characteristic (ROC) analysis of muscle activity. *A*: moment-by-moment statistical discrimination of elbow extensor activity between the torque perturbations lasting 27 and 1,500 ms; data are realigned to perturbation offset at 27 ms. Individual and group average ROC curves are depicted by thin gray lines and the thick black line, respectively. Chance discrimination occurs at 0.5, perfect discrimination occurs at 1.0, and the white dot shows when the mean ROC curve 1st reached threshold. *B* and *C*: ROC analysis of the elbow extensor muscle for torque perturbations lasting 52 and 77 ms (same format as above). *D–F*: ROC analysis of the elbow flexor muscle for torque perturbations lasting 27, 52, and 77 ms, respectively.

positive slope (linear regression: $\text{ROC}_{\text{thresh}} = 23 \text{ ms} + 0.1 \text{ per ms}$; $P < 0.001$). Later times for torque offset led to slightly later times for the initial decrease in muscle activity, although the attenuation always began within the short-latency window (range of the $\text{ROC}_{\text{thresh}} = 21\text{--}38 \text{ ms}$ vs. $\text{R1}_{\text{off}} = 20\text{--}45 \text{ ms}$).

A second important point is that the decreases in activity following torque offset could be powerful as well as rapid. Considering all the torque durations in *experiments 1* and *3*, the impact of torque-offset led to $\sim 35\%$ less evoked activity in the subsequent 20–45 ms; *experiment 2* was not included as the decrease in activity from the comparison condition was quite transient. A regression of the R1_{off} activity following torque offset (normalized by the activity when the torque was sustained) against the time of torque offset showed a weak positive trend that did not pass statistical significance (linear regression: percent of activity with sustained torque = $48\% + 0.2 \text{ per ms}$; $P = 0.11$). Across these two experiments, the mean evoked activity following torque offset ranged between 38 and 81% of its value in the R1_{off} epoch when the imposed torque was sustained, i.e., the decrease ranged between 19 and 62%.

DISCUSSION

To successfully counter mechanical perturbations, feedback corrections must be sensitive to the perturbation's unpredict-

able onset and offset. Studies examining the impact of perturbation offset have consistently reported that joint displacements lasting under $\sim 30 \text{ ms}$ will elicit a R1 response but fail to elicit a R2/3 response in the wrist (Lee and Tatton 1982; Schuurmans et al. 2009) and elbow muscles (Lewis et al. 2005) of the human arm; a similar pattern has also been shown in the cat's forelimb (Ghez and Shinoda 1978). This rapid impact of perturbation offset indicates that a fast spinal mechanism can quench the expression of the R2/3 response, and visual inspection of certain figures from these studies also suggests that a short delay between the perturbation offset and decrease in muscle activity remains present (e.g., Figs. 2 and 3 of Lee and Tatton 1982 and Fig. 2 of Lewis et al. 2005). However, this information is insufficient to determine when muscle activity decreases relative to perturbation offset, whether the timing is conserved across a range of perturbation durations or the magnitude of this effect. We undertook this study to address these outstanding questions.

Our first important finding is that the torque pulses we used (22–152 ms) resulted in a rapid decrease in evoked elbow activity following torque-offset. When the torque was unexpectedly removed before, during, and near the end of the R2/3 epoch (27, 52, and 77 ms), there was a decrease in activity $\sim 30 \text{ ms}$ later. Hence, *experiment 1* showed that a fast attenuation of

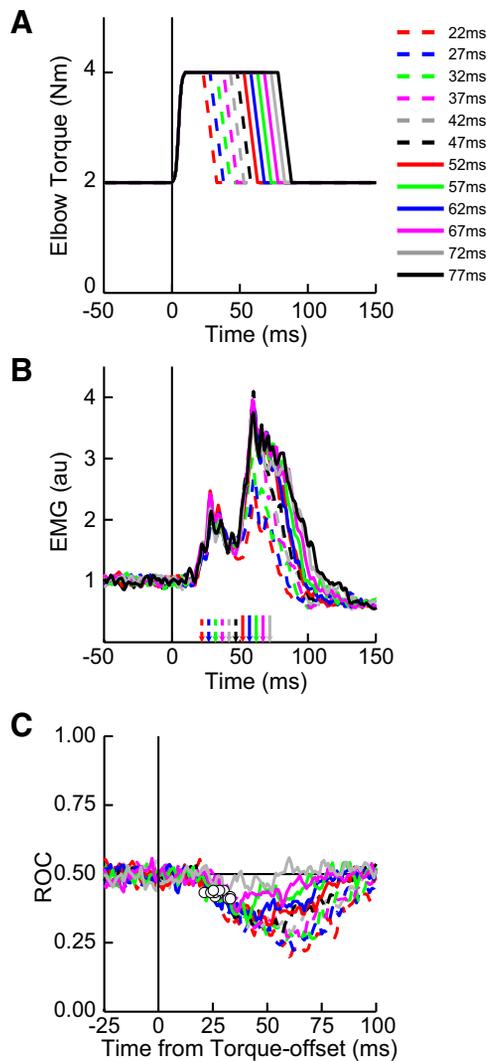


FIG. 6. Imposed torque and evoked muscle activity in *experiment 2*. *A*: 12 elbow extensor torques that lasted between 22 and 77 ms (see *inset* for color code). *B*: group mean activity ($n = 8$) of the elbow extensor muscle following the 12 torque conditions; the time of each torque offset is shown with an arrow with color and line type corresponding to the designated muscle activity. *C*: the 11 mean ROC curves are aligned to perturbation offset; white dots show when the mean ROC curves 1st reached threshold.

elbow muscle activity could take place within the R2/3 epoch and at the beginning of the voluntary response. *Experiment 2* addressed whether there were subperiods within the R2/3 epoch that were insensitive to offset and coincidentally skipped over in *experiment 1*. We found that fast attenuation of elbow muscle activity (~ 30 ms) was present on the fine time scale of 5 ms intervals throughout the R2/3 epoch. *Experiment 3* showed that elbow muscle activity throughout the voluntary response could also be rapidly attenuated by torque offset. Across all experiments, we consistently found response attenuation within the short-latency window (range of the $ROC_{\text{thresh}} = 21\text{--}38$ ms vs. $R1_{\text{off}} = 20\text{--}45$ ms), implicating group I afferents acting through a spinal pathway, because this route is the only one fast enough and short enough to be responsible. Accordingly, a fast spinal mechanism was always capable of attenuating the elbow's ongoing R2/3 response and voluntary response.

Response attenuation was found to occur at slightly later delays with longer torque pulses. This effect could reflect two

different factors that develop over time: decrease in muscle spindle sensitivity and increasing variability in joint motion. Muscle spindles are known to exhibit history-dependent changes in sensitivity following a preceding muscle stretch because of their passive mechanical properties (Proske et al. 1993). Similarly, a progressive decrease in spindle sensitivity could develop at different times within a single stretch. The second factor is a progressive increase in joint motion variability, which makes it increasingly difficult to detect a change between conditions. An increase in motion variability over

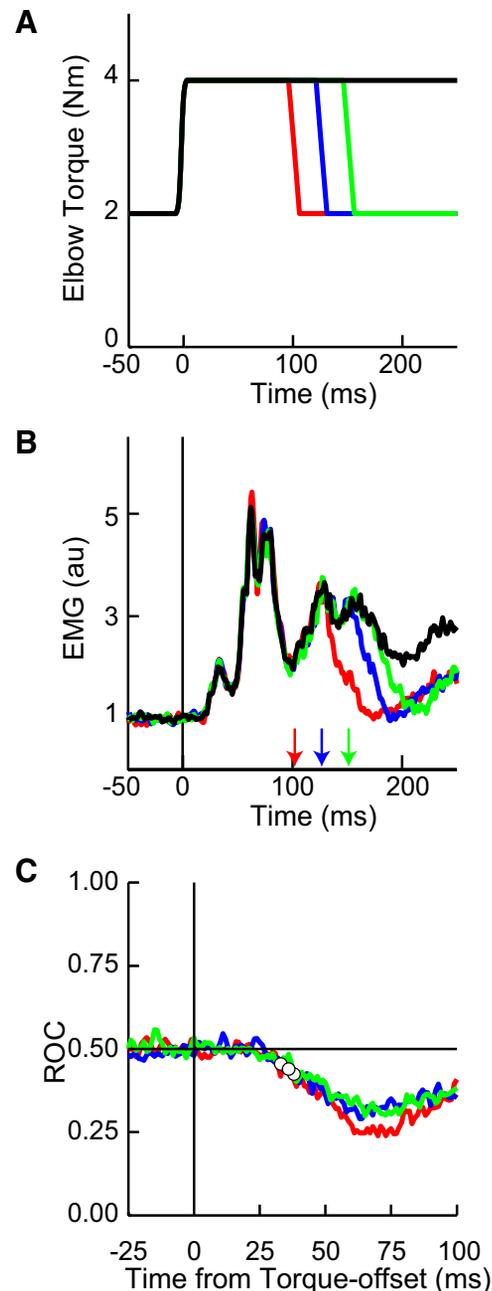


FIG. 7. Imposed torque and evoked muscle activity in *experiment 3*. *A*: elbow extensor torque lasting 102 (red), 127 (blue), 157 (green), and 1,500 ms (black). *B*: group mean activity ($n = 10$) of the elbow extensor muscle following the 4 torque conditions; the time of each torque offset is shown with an arrow with similar color. *C*: the 3 mean ROC curves are aligned to perturbation offset; white dots show when the mean ROC curves 1st reached threshold.

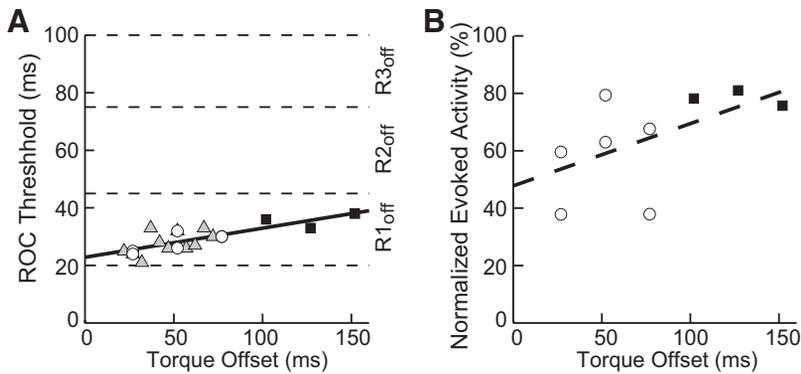


FIG. 8. Trends across experiments. A: timing of muscle activity decrease following torque offset. Data are shown for all 3 experiments. Each icon depicts when the mean ROC decreases from chance following torque offset. White circles, gray triangles, and black squares correspond to the torque offsets in *experiments 1, 2, and 3*, respectively. A significant linear regression is fit through the data (solid line): $23 \text{ ms} + 0.1 \text{ per ms}$; $P < 0.001$. B: magnitude of decrease in the evoked muscle activity during the R1_{off} epoch. Data are shown for *experiment 1* (white circles) and *experiment 3* (black squares). Each icon depicts the mean evoked activity when the torque was removed divided by the activity when the torque remained on: 0 and 100% indicate no evoked activity and no decrease in evoked activity, respectively. A non-significant linear regression is fit through the data (dashed line): $48\% + 0.2 \text{ per ms}$; $P = 0.11$.

time resulted from our use of torque-based perturbations, because any trial-by-trial differences in evoked muscle activity would cumulatively influence the joint motion. A high-gain servo device could enforce a particular joint motion, but its drawback is that corrective actions would have no influence on the perturbations.

A second important finding is that the decrease in elbow muscle activity following torque-offset was substantial. Considering all the torque durations in *experiments 1* and *3*, the impact of torque-offset led to $\sim 35\%$ less activity on average in the subsequent 20–45 ms ascribable to spinal processing. Because spinal mechanisms can powerfully attenuate evoked activity following torque offset, they may play a significant role in naturalistic conditions like stabilizing an umbrella against brief gusts of wind, controlling one's limb position when wrestling a competitor, or maintaining the orientation of steering wheel along a bumpy road.

One issue that was not examined is whether a similar pattern of results occurs with unloading pulses. If the pattern was symmetrical to these findings, the muscle activity would initially fall during the unloading period and rapidly resume with the reintroduction of the load. The results of this experiment, which has not been undertaken to our knowledge, could help shed light on the underlying neural mechanisms.

Previous researchers have considered two contrasting mechanisms for the rapid impact of perturbation offset on evoked muscle activity: a single spinal pathway that additively contributes to both the R1 response and R2/3 response or a dual spinal pathway where delayed descending motor commands interact with ongoing afferent activity (Lee and Tatton 1982; Lewis et al. 2005; Schuurmans et al. 2009). Our experiment cannot test between a single or dual pathway organization, but two recent studies are relevant to this issue.

One study modeled how a realistic system of muscle, muscle spindles, and motor neurons responded to simulated ramp displacements having different duration (Schuurmans et al. 2009). A simulated ramp displacement and spindle stretch caused a near synchronous discharge of the noisy motor neurons (R1 response). Critically, this burst of activity was followed by a brief refractory period so that motor neurons could only express a subsequent synchronous discharge (R2/3 response) if they continued to receive spindle input; if not, the neurons would remain quiet. Therefore a single pathway could exhibit the all-or-none sensitivity of R2/3 to brief displacements.

Evidence against the dual spinal pathway hypothesis was provided by the second study using a novel paradigm of two

successive joint displacements (Lewis et al. 2005). The first step displacement ended early enough to quench the R2/3 response, whereas the second step displacement began before the critical duration of 30 ms. A hypothetical interaction between delayed descending motor commands and ongoing afferent activity would predict that activity during the R2/3 epoch would reflect the summation of two events through two pathways: an R1 response elicited by the second step (pathway 1) and an R2/3 response elicited by the first step and enabled by ongoing afferent activity from the second step (pathway 2). However, the observed activity was significantly smaller than this prediction, which argues against the hypothesis of a dual spinal pathway. Taken together, these two studies suggest that our observations reflect the impact of group I afferents on a single spinal pathway.

The group I afferents responsible for the attenuation of muscle activity could either reflect intramuscular receptors (spindles and golgi tendon organs) and/or cutaneous receptors. Both can convey information about a changing limb configuration (Hulliger 1984; Jami 1992; Macefield 2005), and cutaneous receptors can obviously convey information about the imposed contact force (Johansson and Flanagan 2009). Evidence for the role of these receptors in the long-latency reflex is mixed. For finger muscles, activity in the long-latency epoch can be evoked by contact forces alone (Corden et al. 2000), and anesthesia of the skin has been reported to sometimes depress long-latency activity elicited by muscle stretch (Loo and McCloskey 1985). In contrast, anesthesia of the skin does not seem to affect the long-latency response of wrist muscles (Bawa and MacKenzie 1981; Cody et al. 1987). A definitive answer will require experiments focused on the elbow muscles and will likely implicate a multitude of sources interacting in a more subtle way.

Muscle activity during the R2/3 epoch is also thought to include contributions from supraspinal structures like primary motor cortex (M1). This issue is complicated by differences between the various muscles of the upper limb. For example, patients exhibiting mirror movements exhibit mirroring long-latency reflexes for their hand muscles—stretching a hand muscle will evoke a local long-latency response and a similarly timed response in the corresponding muscle of the unperturbed hand—which implicates a cortical contribution (Capaday et al. 1991; Fellows et al. 1996; Matthews et al. 1990). However, this bilateral pattern of long-latency reflexes is not present for the elbow muscles of patients exhibiting mirror movements (Fellows et al. 1996), suggesting a difference in the cortical role and/or neuropathology of these two upper limb muscles.

Despite the ambiguity in clinical data, a number of nonclinical studies support a cortical contribution to the elbow's long-latency reflex. One strong piece of evidence involves applying transcranial magnetic stimulation (TMS) concurrently with muscle stretch (Lewis et al. 2004; for similar experiments on the hand and wrist, see Day et al. 1991 and Lewis et al. 2004; respectively). When TMS is timed to evoke a muscle response during the long-latency epoch, the total response is larger than the summed activity of stretch alone and TMS alone, which indicates an interaction within M1; this pattern is not observed when TMS is applied during the short-latency epoch. Additional evidence for a cortical role comes from single-unit recordings in the M1 of awake behaving monkeys. Neurons activating muscles at a particular joint give very quick responses to muscle stretch (~20 ms), which may contribute to long-latency reflexes at the elbow (Evarts and Tanji 1976; Flament and Hore 1988; Herter et al. 2009), shoulder (Herter et al. 2009), and wrist (Cheney and Fetz 1984); scalp potentials in humans also indicate cortical changes fast enough to contribute (Abbruzzese et al. 1985; Crammond et al. 1985; Mac Kinnon et al. 2000). Finally, group II muscle spindles traveling slowly through a short spinal pathway could play a role in the elbow's R2/3 period. Evidence for group II afferents includes a selectively depressed R1 response with vibration preconditioning (Hendrie and Lee 1978) and a selectively depressed R2/3 response with cooling (Cody et al. 1987; Lourenço et al. 2006) and a muscle relaxant (Lourenço et al. 2006; Meskers et al. 2010), but these studies have only been conducted for the wrist muscles of the upper arm.

From a functional standpoint, the different circuits for the R2/3 and voluntary response could also support their observed increase in flexibility compared with the R1 response. Unlike the R1 response, R2/3 and voluntary responses of hand, wrist, elbow, and shoulder muscles can be modulated with explicit task requirements (Calancie and Bawa 1985; Crago et al. 1976; Hammond 1956; Mutha et al. 2008; Pruszynski et al. 2008; Rothwell et al. 1980; Soechting et al. 1981). Likewise, R2/3 and voluntary responses of hand, elbow, and shoulder muscles can account for mechanical interactions across multiple joints (Cole et al. 1984; Gielen et al. 1988; Koshland et al. 1991; Kurtzer et al. 2008, 2009; Latash 2000; Soechting and Lacquaniti 1988) and regulate limb stiffness in parallel with environmental instability (Doemges and Rack 1992; Kimura et al. 2006; Perreault et al. 2008); note that the long-latency reflexes of elbow muscle also express a consistent output across background load conditions (Pruszynski et al. 2009). This evolving muscular response to a mechanical perturbation reflects an intuitively reasonable trade-off between speed and complexity, where the earliest epoch is the least sophisticated, and later epochs are increasingly sophisticated and likely reflects the temporal overlap of multiple neural mechanisms.

In this study, we found that the nervous system can quickly attenuate the evolving response of elbow muscles if the perturbation is unexpectedly removed, a common occurrence in naturalistic situations. The ability to develop increasingly sophisticated responses that can be quickly attenuated likely reflects the temporal overlap of multiple neural generators acting with different delays. This organization is also broadly consistent with feedback laws designed to balance multiple costs like speed and accuracy (Scott 2004; Todorov and Jordan 2002).

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DISCLOSURES

S. H. Scott is associated with BKIN Technologies which commercialized the robot that was utilized.

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