Visual modulation of proprioceptive reflexes during movement

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Abstract

Previous research has demonstrated that feedback circuits such as reflexes can be tuned by setting their gains prior to movement onset during both posture and movement tasks. However, such a control strategy requires that perturbation contingencies be predicted during movement planning and that task goals remain fixed. Here we test the hypothesis that feedforward regulation of reflex circuits also occurs during the course of movement in response to changes in task goals. Participants reached to a visual target that was occasionally jumped on movement initiation, thus changing task goals. Reflex responses were elicited through a mechanical perturbation on the same trial, 100 milliseconds after the target jump. Impedance to the perturbation was tuned to the direction of the preceding jump: reflex responses increased or decreased depending on whether the perturbation opposed or was consistent with the target jump. This modulation, although sensitive to the direction of the jump, was insensitive to jump amplitude, as tested in a follow-up experiment. Our findings thus suggest that modulation of reflex circuits occurs online, and is sensitive to changes in visual target information. In addition, our results suggest a two-level model for visuo-motor control that reflects hierarchical neural organization.

Keywords

Reflexes; Vision; Online Control; Target Jump; Modulation

Introduction

In order to explain how the nervous system selects control signals that are appropriate for a desired movement, conventional models of control have proposed that movements are planned and executed in a sequential order. These models have often proposed that movement planning includes an optimization process, wherein kinematic or kinetic cost functions are minimized. Candidate cost functions have included mean squared jerk (Flash and Hogan 1985), mean squared torque change (Uno et al 1989), peak work (Soechting et al 1995), or muscle energy (Goble et al 2007) among others. Such optimization formulations often require that the entire movement trajectory be computed in advance, reinforcing the idea that biological control incorporates advance planning of movement trajectories. Support for this idea has come from several studies suggesting that the central nervous system accounts for limb and task dynamics when adapting to novel force conditions imposed by robotic manipulanda (Shadmehr and Mussa-Ivaldi 1994; Goodbody and Wolpert 1998), artificial gravity environments (Lackner and DiZio 2003), or novel inertial loads (Sainburg et al 1999). The prediction of such forces can be observed as “after-effects” following adaptation; if the imposed dynamics are removed...
After adaptation, movement errors emerge that mirror image the previously imposed forces, indicating anticipatory control.

According to this primarily feedforward control scheme, the role that sensory feedback plays during the course of movement is largely to correct deviations from the planned or reference trajectory. Such deviations to the movement path are caused by perturbations arising not only from the environment (Colgate and Hogan 1988; Lackner and DiZio 1994; Scheidt et al 2001) but also from within the neuro-musculoskeletal system (Harris and Wolpert 1998; Sainburg et al 1999). When such perturbations can be predicted prior to movement onset, their effects can be partially accounted for during motion planning itself. In such cases, compensation for perturbations has been shown to occur through feedforward, reflex-mediated modulation of limb stiffness and viscosity, i.e. active components of limb impedance (Hore et al 1990; Kimura et al 2006; Lacquaniti and Maioli 1989; Wang et al 2001). Thus, resistance to perturbations has been shown to be selectively tuned based on pre-movement expectations of dynamic events. In addition, when the effects of perturbing forces can be learned over repeated exposure, and more importantly, when task goals remain constant, limb impedance has been shown to vary with the expected timing, and direction of the perturbing forces (Burdet et al 2001; Franklin et al 2003; Kimura et al 2006; Wang et al 2001). An elegant example of such anticipatory impedance modulation was provided by Lacquaniti and colleagues who, in a ball catching task demonstrated not only modulation, but also reversal of the reflex response to ball impact (Lacquaniti et al 1991).

During everyday tasks however, environmental forces and perturbations can rarely be predicted prior to movement. For example, when reaching in a moving vehicle, unexpected disturbances arising from the environment can produce substantial forces on the body. In addition, task goals can often change during the course of a given movement. For example, we frequently make movements toward objects that move en route, such as when reaching for a baby’s hand or chasing a moving pet. We hypothesize that in order to effectively impede such perturbations, sensory feedback circuits are continuously modulated during the course of movement. We therefore predict that during reaching movements, modification of an ongoing movement in response to changes in visual information will also result in modulation of reflex responses to environmental perturbations. In a series of two experiments, using a novel experimental paradigm, we tested these predictions and also assessed the sensitivity of such modulation to changes in goal parameters. Our results confirmed that changes visual information, implemented through target displacements during the course of movement resulted in significant modulation in reflex responses. Remarkably, our findings provided the first confirmation that short latency reflex responses were modulated during goal directed movement, independent of changes in background muscle activation or muscle state. Modulation of the longer latency responses were also consistent with changes in short latency reflex responses. Such impedance modification was sensitive to changes in target direction but surprisingly, insensitive to changes in target distance. This may reflect a fundamental limitation in online modulation of impedance through reflex mechanisms. It should also be emphasized that the observed changes in reflex responses occurred well before responses to changes in target location were initiated. Our results thus provide a demonstration of continuous feedforward tuning of sensory feedback during voluntary movement in response to changes in task goals.

Results

Our main goal was to determine whether limb impedance is modulated online when an ongoing movement had to be corrected in response to changes in target location. During baseline conditions, subjects made 30° elbow extension movements to a 3.5 cm diameter target. We provided changes in target location by jumping the target when subjects first reached the start...
circle boundary (Movement Initiation Point, Figure 1, time zero). The target jump was implemented either further in the direction of the movement (away from the baseline target) or opposite to the direction of the ongoing movement (toward the start position). We refer to these as “Far” and “Close” target jumps respectively. In order to elicit reflex responses in specific muscles, we also occasionally applied a 40 Newton, 30 milliseconds force pulse to the limb, 100 milliseconds after movement initiation. As displayed in figure 2 and described in the following sections, the timing of the mechanical perturbation was close to peak antagonist (flexor) muscle activity, following the first burst in agonist (extensor) activity. On randomized trials, the mechanical perturbation was applied without any target jump while on others the perturbation was preceded by a Far or Close target jump. We ensured that subjects made very consistent movements toward the target under baseline conditions, by introducing target jumps or mechanical perturbations on only 25% of the trials.

Experiment 1

Our first experiment employed a design in which we independently varied the direction of the target jump (Far or Close) and the direction of the force pulse (Flexor or Extensor). However, we were only interested in the reflex response to the extensor perturbation condition, since this perturbation resisted the action of the active antagonist muscles (flexors). In contrast, the flexor perturbation simply resisted the silent agonist muscle. This perturbation did not elicit a short latency reflex response in the agonist, nor did it result in any change in the activity of the antagonist muscle. We examined whether the response to the extensor perturbation changed, depending on whether the perturbation assisted or interfered with achievement of the displaced target. We will refer to the condition in which the perturbation assisted achievement of the displaced target as “congruent” and the condition in which the perturbation resisted target achievement as “incongruent”.

Figure 1 shows representative handpaths and tangential velocity profiles from a single trial, as well as ensemble averaged elbow displacement, velocity, torque, and Biceps (antagonist) and Triceps (agonist) EMG for a representative subject. Data have been synchronized to movement initiation, the time at which the target was jumped. In figure 1, baseline trials are shown as thin lines, while target jump trials are shown as thick lines. We will first briefly discuss baseline performance, followed by a description of responses to target jumps. We then detail the reflex responses elicited by the mechanical perturbation and then examine their modulation caused by preceding changes in target location.

Baseline performance—In baseline trials, subjects successfully achieved the 30-degree target as can be seen from the handpaths in figure 1A (thin open circles). These movements were characterized by typical bell shaped tangential velocity profiles (figure 1A, inlay, thin black). Elbow velocity profiles also tended to be smooth and unimodal (figure 1B, thin black). The EMG profiles followed a characteristic reciprocal pattern, with an initial triceps burst that produced the initial extensor muscle torque, followed by activation of the biceps (agonist), and finally a co-activation phase, associated with stopping the movement (figures 1E and 1F, thin black). Across all subjects, the baseline final position error was on average 1.81cm (S.E. ±0.38 cm), which was within the diameter of the target.

Responses to target jumps—Figure 1 shows data for the Far (Left Column) and Close (Right Column) target jumps in bold, overlaying the baseline conditions (thin lines). While baseline trials required 30° extensor movements, the target jumped 50% of that distance in each direction, such that the Close target required 15° extension from the start position, while the Far target required 45° extension. The bold handpaths of figure 1A (left and right) reflect this task requirement. These data reflect movements from a single subject. The statistics
reported below were computed across subjects, and confirm the representative nature of this data.

**Far Target Jump (Figure 1, Left, Bold)**—Prior to the jump, the kinematic, kinetic and EMG profiles overlapped in the baseline and target jump conditions. For all our measures, the first point at which the target jump trials differed statistically from the baseline trials occurred after the target jump, verifying that the subjects did not anticipate the jump. Taking into account an average 10 millisecond delay between the time the target jump command was issued and the time the projection screen was redrawn (see experimental procedures), we estimated that the earliest response in the Far target jump trials was elicited in the triceps as a second burst at a mean latency of 197 milliseconds (S.E. ±9 milliseconds) following the target jump. This latency is consistent with previously observed voluntary response latencies to changes in visual information (Danion and Sarlegna 2007; Day and Brown 2001; Desmurget et al 2004; Sarlegna et al 2003). The increase in triceps activity resulted in a subsequently decreased flexor muscle torque and increased extensor muscle torque, as compared with baseline conditions, at a latency of 212 milliseconds (S.E. ±14 milliseconds) across all subjects. This led to increased extensor velocity and displacement in order to drive the hand towards the farther target. A later increase in biceps and triceps coactivation was associated with movement stopping at the new target location. A final position error of 3.13 cm (S.E. ±0.72 cm) was observed, indicating effective corrections, even though movements were slightly more erroneous than baseline trials.

**Close Target Jump (Figure 1, Right, Bold)**—In the condition where the target jump was opposite to movement direction, the initial kinematic, kinetic and EMG profiles follow the baseline EMG profile prior to the target jump as can be observed from figure 1 (right). The first corrective response to the target jump occurred in the biceps, which showed an increased and prolonged EMG burst at a latency of 214 milliseconds (S.E. ±17 milliseconds). No change in the triceps EMG was observed at this latency. The increase in biceps activity caused an increased flexor muscle torque. This led to a subsequent increase in elbow velocity to drive the limb into flexion in order to bring the hand into the new target location. The final position accuracy achieved in this case was comparable to the baseline movement accuracy with the final position error being 1.61 cm (S.E. ±0.28 cm).

**Responses to Mechanical Perturbations are Modulated by Target Jumps**—In order to determine whether changes in task goals implemented through target jumps modulate limb impedance, we applied the target jump and the mechanical perturbation within the same trial. On certain trials, the mechanical perturbation was also applied without any target jump while on certain others, the perturbation was preceded by a Far or a Close target jump (“target jump + mechanical perturbation” trials). The target jump occurred first, followed by the force pulse 100 milliseconds later. Because the shortest latency response to target jumps was some 200 milliseconds, this allowed approximately 100 milliseconds to assess responses to the mechanical perturbation. We measured reflex responses at latencies that corresponded to the classical components of the response to a rapid stretch of the muscle: a short latency reflex response, often referred to as M1, occurring within 50 milliseconds following perturbation onset (Johnson et al 1993; Matthews 1991; Tatton and Lee 1975), a medium latency response M2, observed some 60 -100 milliseconds following the stimulus (Crago et al 1976; Johnson et al 1993; Kimura et al 2006; Matthews 1991) and likely including transcortical circuits (Matthews 1991), followed by a sustained longer latency response which is thought to reflect a voluntary correction. It should be emphasized that these time values reflect ranges of the latency to initiate a response to a perturbation under different experimental conditions, and do not imply that the reflex components are of that particular duration.
Mechanical Perturbation without Target Jumps—Figure 2A shows the averaged, rectified agonist and antagonist EMG profiles under baseline (thin black) and mechanical perturbation trials (thick black). The time that the mechanical perturbation was applied is shown by a vertical dotted line. As can be seen from these figures, this time was near the peak in antagonist muscle activity under baseline conditions. The effect of this extensor perturbation was thus to stretch the activated biceps muscle.

When this perturbation was applied without any target jump, a rapid increase in the activity of the antagonist biceps muscle was observed (figure 2A, top, thick black). In contrast, there was no change in the agonist triceps activity, which was already inhibited (figure 2A top, thick black). The earliest increase in antagonist response occurred at a latency of 21 milliseconds following the perturbation for one subject. Across all subjects, the increase in antagonist muscle activity occurred at a mean latency 32 milliseconds (S.E. ±3 milliseconds), which was within the range of latencies associated with the M1 response, as described earlier. We then integrated the EMG response in 30 milliseconds bins for each subject starting from their first observed response latency to the mechanical perturbation. These impulses were quantified as the short, medium and long latency components of reflex responses to muscle stretch (see Experimental Procedures). For example, for our subject that showed the earliest response (21 milliseconds following the perturbation), these bins were quantified from 21-51 milliseconds, 51-81 milliseconds and 81-111 milliseconds respectively. It must be emphasized that the division of the typical response to mechanical stretch of the muscle into components such as M1 and M2 originated mainly in studies requiring subjects to maintain a static position. Nevertheless, we observe EMG responses to perturbations during movement at similar latencies suggesting that the same fundamental mechanisms are involved in the early rapid compensatory response production during postural as well as movement tasks.

Effect of target jump congruence on responses to mechanical perturbations—Figure 2B shows the average biceps EMG response under target jump + mechanical perturbation trials for a representative subject, expanded in time. For this subject, the first response under the mechanical perturbation condition was obtained 26 milliseconds following the force pulse. The dark gray lines in figure 2B show the congruent target jump + mechanical perturbation condition, in which we implemented a Far target jump 100 milliseconds prior to the extensor mechanical perturbation. In contrast, the light gray lines reflect responses in the incongruent target jump + mechanical perturbation trials, when a Close target jump was implemented before applying the extensor perturbation.

Our results indicate significant modulation of all components of the reflex response under congruent and incongruent conditions. From the biceps EMG profiles of figure 2B, we can see that while reflex responses were substantially reduced in the congruent target jump + mechanical perturbation condition (dark gray profile), these responses were significantly increased under incongruent target jump + mechanical perturbation conditions (light gray profile). Figure 2C, which shows the mean change in EMG impulse as a multiple of baseline EMG at short, medium and long latencies, shows the consistency of these observations across all subjects. The light and dark gray bars in figure 2C correspond to the incongruent and congruent target jump + mechanical perturbation conditions respectively, whereas the black bars represent the mechanical perturbation condition without any target jump. Statistical analysis using an ANOVA revealed a significant main effect of target jump direction on the change in EMG response at short (p = 0.0322), medium (p = 0.0024) and long latency (p = 0.0026). Post hoc comparisons showed that this modulation was significantly different for all reflex components between congruent and incongruent target jump + mechanical perturbation conditions (short: p = 0.0061, medium: p = 0.0003, long: p = 0.0004). Across all subjects, the response in the incongruent target jump + mechanical perturbation condition was 1.75 times larger than in the congruent condition. These differences were also sustained at longer
latencies, in which the incongruent condition resulted in responses that were 1.83 and 1.86 times larger than those in the congruent condition at medium and long latencies respectively (figure 2C, compare light gray bars to dark gray bars).

In addition, our post hoc comparisons also revealed that reflex responses in the congruent and incongruent target jump + mechanical perturbation conditions were also significantly different than the responses in the mechanical perturbation trials without any target jump (see table 1). For the congruent conditions, responses were significantly reduced at all latencies, whereas for the incongruent conditions, reflex responses were substantially enhanced at the medium and long latencies. At short latency however, the increase in reflex response was small and did not reach significance. These results are demonstrated in the line plots of figure 2C (compare black bars to dark and light gray bars) and significance levels are given in table 1.

Thus, under the target jump + mechanical perturbation conditions, we observed a significant modulation of reflex responses induced by prior changes in visual target information. It must be emphasized that prior to the time of mechanical perturbation application, antagonist muscle activation and state were similar across all conditions. Thus this modulation cannot be attributed to changes in these parameters.

**Reflex-mediated modulation of reaction force**—We assessed whether changes in reflex responses across the congruent and incongruent target jump + mechanical perturbation conditions were reflected in the reactive force applied by the subject onto the handle of the robotic manipulandum. Our results indicated a direction dependent modulation of reactive force consistent with the direction dependent modulation of reflex responses. Figure 3A shows the average reactive force profile in the X and Y direction for a representative subject. As observed in these profiles, both these components of the total reactive force decreased under congruent (dark gray) but increased under incongruent (light gray) target jump + mechanical perturbation conditions. The total end-point force applied by the manipulandum was 40N. This force was either in the direction of the movement, or opposite to it. Depending on the configuration of the subject, the components of this force in the X and Y directions of the workspace were smaller, as can be seen from the peak values of the time profiles of reaction forces of figure 3A. Assuming a 30-millisecond delay for the muscular excitation-contraction sequence (Della Santina et al 1989), we quantified modifications in total reactive force by examining its value 30 milliseconds after the end of the short, medium and long latency intervals for each subject. For instance, for our representative subject, for whom the short, medium and long latency reflex responses were quantified from 26-56 milliseconds, 56-86 milliseconds and 86-116 milliseconds respectively, reaction force values were quantified at 86, 116 and 136 milliseconds. Average values of these forces across all subjects are shown in the line plots of figure 3B, in which the dark and light gray bars represent the congruent and incongruent target jump + mechanical perturbation conditions respectively. Statistical comparison revealed that reaction forces following the short latency interval in the congruent and incongruent target jump + mechanical perturbation condition were not significantly different (p = 0.0870). Following medium and long latency response intervals however, the difference in reaction force between the congruent and incongruent conditions was substantial, with significant differences emerging at these latencies (medium: p = 0.0424, long: p = 0.0155). As discussed later, this change in reaction force must be due to modulation of reflex responses (see Discussion).

**Experiment 2**

In our second experiment, we tested the sensitivity of reflex modulation to the amplitude of the target displacement. Similar to experiment 1, baseline trials consisted of 30-degree elbow extension movements. The target jump, implemented on movement initiation, could also be
both, farther from the baseline target or closer towards the start location. However, in a given direction, the target could be jumped to two different locations that corresponded to a 15 or a 30-degree corrective response. The mechanical perturbation in this experiment also consisted of a 40 N, 30 milliseconds force pulse applied 100 milliseconds after movement initiation. This perturbation could be either congruent or incongruent with the preceding target jump, or could also be applied on trials without any target jump. Only the extensor mechanical perturbation was used in this experiment and therefore evaluation of reflex response changes was restricted only to the biceps muscle.

Responses to target jumps—Overall, the pattern of responses in the target jump trials was similar to that observed in experiment 1. Figure 4 shows elbow angle, muscle torque and the agonist and antagonist EMG response under baseline (black) and small (light gray) and large (dark gray) target jump trials for a representative subject. As can be observed from the profiles in figures 4C and 4D, the Far target jump led to an increase in agonist activity (left panel), while the Close target jump led to an increase in antagonist activity (right panel) to initiate corrective responses. Across all subjects, the peak corrective agonist response was substantially larger for the 30-degree compared to the 15-degree target jump, as plotted in the bar plots of figure 4E, left (p = 0.0130). Similarly, peak activation in the antagonist following the target jump was higher for the 30-degree rather than the 15-degree target jump (p = 0.0322, figure 4E, right). These differences in EMG are also reflected as significant changes in the torque and kinematic profiles. For the Far target jump condition, extensor muscle torques were much larger for the 30-degree jump compared to the 15-degree jump. This led to larger velocities in the 30-degree condition, eventually bringing the hand to the desired target. These trends were also consistent in the Close target jump condition, with the 30-degree target jump resulting in higher flexor muscle torques and higher velocities as compared to the 15-degree target jump. Thus this voluntary mediated response to the target jump was well scaled with the modified target location. The earliest response to the target displacement across all four target jump conditions was 187 milliseconds (S.E. ± 7 milliseconds) on average across all subjects (observed for the Close, 15-degree target jump condition). This was similar to the earliest response in target jump trials in experiment 1 (197 milliseconds). The earliest change in torque due to this change in EMG was observed for the same condition at a latency of 217 milliseconds (S.E. ± 9 milliseconds). As can be seen from figure 4, these changes in torque led to subsequent changes in movement kinematics to bring the limb to the desired location.

Modulation of reflex responses based on direction but not amplitude of the target jump—We assessed amplitude sensitivity of the reflex responses by applying the mechanical perturbation 100 milliseconds after the target was jumped either to the small or large location in a particular direction (target jump + mechanical perturbation trials). Occasionally, the perturbation was also applied on trials in which no target jump was implemented. Figure 5 shows the results from these experimental conditions. The profiles in figure 5A were compiled by ensemble averaging congruent (dark gray) and incongruent (light gray) target jump + mechanical perturbation trials from a representative subject. The dotted profiles in each condition reflect the 15-degree target jump, whereas the solid line represents the 30-degree jump. The bar plots of Figure 5B demonstrate across subject results, as described in the following sections.

Responses to Mechanical Perturbations without Target Jumps—We briefly describe key results from trials in which we applied the mechanical perturbation without any preceding target jump. Broadly, these were very similar to perturbation responses under similar experimental conditions in experiment 1. When the extensor mechanical perturbation was applied, a rapid increase in biceps activation occurred compared to the baseline response. The earliest response for any subject in these mechanical perturbation trials was observed at a
latency of 27 milliseconds following the perturbation. Across all subjects this latency was 34 milliseconds (S.E. ± 2 milliseconds). This latency was similar to that observed in experiment 1 (32 ± 3 milliseconds). The increase in biceps activity at this latency was sustained for a long duration and led to an increase in flexor muscle torque, which acted to bring the extended limb towards the desired target location. Again, for each subject we quantified the reflex components of the perturbation response by integrating the antagonist EMG responses in 30 milliseconds bins following the first change in the EMG amplitude after the perturbation. Our short latency response, for example, for one subject was assessed as the integral from 27-57 milliseconds, while the medium and long latency responses were quantified from 57-87 and 87-117 milliseconds respectively.

**Effect of target jump amplitude on responses to mechanical perturbations**—
Consistent with our results from experiment 1, for both the 15 and 30-degree target jumps we observed significant direction dependent changes in the EMG response to the mechanical perturbation. Our ANOVA revealed a significant main effect for target jump direction on the change in the EMG impulse at all latencies for the 15-degree (short: \( p = 0.0100 \), medium: \( p = 0.0039 \), long: \( p = 0.0067 \)) and 30-degree (short: \( p = 0.0083 \), medium: \( p = 0.0046 \), long: \( p = 0.0077 \)) target jumps. Moreover, also consistent with the results from experiment 1, post-hoc comparisons revealed significant differences between congruent and incongruent target jump + mechanical perturbation conditions for the 15-degree (short: \( p = 0.0014 \), medium: \( p = 0.0006 \), long: \( p = 0.0009 \)) and 30-degree target jumps (short: \( p = 0.0012 \), medium: \( p = 0.0006 \), long: \( p = 0.0011 \)). These trends are demonstrated in figure 5A for a representative subject and across all subjects in figure 5B (compare light gray bars (incongruent conditions) to dark gray bars (congruent conditions)). In addition, when comparisons were made with responses on the mechanical perturbation trials, similar trends as in experiment 1 were observed.

More importantly however, in this experiment, comparison across 15-degree and 30-degree target jump conditions within the Far and Close directions revealed that the magnitude of the modulation was similar. Thus, in case of congruent target jump + mechanical perturbation trials, the decrease in the reflex response was not altered by the amplitude of the target jump at any response latency (short: \( p = 0.2442 \), medium: \( p = 0.4062 \), long: \( p = 0.4903 \)). This can be observed from the overlapping dotted and solid dark gray profiles of figure 5A. In addition, the change in EMG integrals plotted in figure 5B demonstrates that this trend is consistent across all subjects (compare hatched and filled dark gray bars). Similarly, when a Close target jump preceded the extensor mechanical perturbation (incongruent conditions), the exact location of the target jump also did not influence the magnitude of the increase in reflex response to the mechanical perturbation. Thus a similar enhancement was observed irrespective of whether this Close target jump was 15 or 30-degrees. The overlapping dotted and solid light gray profiles of figure 5A demonstrate this trend for our representative subject. This pattern was maintained across all subjects, as can be observed by comparing the hatched and solid light gray bars in figure 5B. Again, statistical comparison yielded no significant differences in the magnitude of reflex response increase at short (\( p = 0.1380 \)), medium (\( p = 0.1059 \)) or long latency (\( p = 0.2430 \)). Statistical significance levels for comparisons across the various experimental conditions in experiment 2 are summarized in table 2.

Thus, taken together our results from both experiments suggest that limb impedance is continuously tuned during movement in response to changing task goals, implemented through random target displacements. This impedance modulation occurs through specific changes in reflex responses and is sensitive to the direction of target displacement, but surprisingly insensitive to its exact amplitude. Moreover, these changes occur at least 100 milliseconds prior to voluntary responses to changes in target location.
Discussion

In the two studies presented here, we investigated whether upper limb reflex responses might be modulated during movement in response to changes in visual target information. We observed significant tuning of reflex responses in a manner consistent with the modified task goal of reaching to the new target. Our results provide the first demonstration that the short latency reflex response to a mechanical perturbation is modifiable during the course of goal directed movement. Previous studies examining task dependent changes in reflex responses in upper limb muscles were unable to demonstrate modulation of the short latency response other than under conditions in which the reflex scaled with changes in voluntary muscle activation or state of the muscle (Abbruzzese et al 1994; Dufresne et al 1980; Mortimer 1981; Zehr et al 2003). This led to the idea that this component of the reflex response might be resistant to descending influences. The remarkable tuning of the short and longer latency components of the reflex response observed in our study however, suggests that reflex circuits are continuously tuned by descending signals to ensure that their output favors task goal achievement. Moreover, the modulation observed in our study is independent of muscle activation or state, because at the time of perturbation application, these parameters were the same under the target jump + mechanical perturbation and mechanical perturbation conditions.

Recent research has emphasized the role of “triggered reactions” as mechanism for modulating responses to perturbations (Crago et al 1976; Hasan 2005; Koshland and Hasan 2000; Lewis et al 2006). Triggered reactions can be defined as task-related, voluntary responses that are stored within sub-cortical structures and that can be released by peripheral stimuli. According to this idea, the stimulus need not be specific and serves only as a “trigger” for the release of the stored response, which results in a well-coordinated action in accord with task goals. For example, an auditory stimulus may trigger early release of a preprogrammed movement (Valls-Sole et al 1999; Carlsen et al 2004). One could postulate that the task-dependent modulation of the reflexes elicited in the current study might actually be a facilitation of such a pre-programmed reaction. However, our results appear inconsistent with this mechanism. This is primarily because the assembly, storage, and release of the response must occur on-line within 130 milliseconds following the visually displayed target jump, which calls into question the idea that the response is stored at all. In addition, in our study, the short latency component of the reflex is modulated by the target jump condition, while triggered reactions have not been shown to cause changes in EMG patterns at such short latencies. Lewis et al. (2006) showed instruction dependent modulation of only the long latency component of the stretch reflex, consistent with previous reports that showed reflex modulation at no less than 70 milliseconds following perturbation onset (Crago et al. 1976). Our results also indicate that modulation of reflex responses varies with the quality of the stimulus. While biceps activity in response to the same extensor perturbation was substantially reduced under the Far target jump + mechanical perturbation condition, it was significantly increased in the Close target jump + mechanical perturbation condition. It should be stressed that triggered reactions, by definition, do not vary with the quality of the stimulus because the function of the stimulus is simply to startle the release of a stored program. Our results thus argue against the idea that the target jump serves as a stimulus for the release of a stored voluntary response. Instead, we suggest that descending commands directly modify the action of reflex circuits in order to regulate limb impedance to perturbations arising during movement.

Reflex modulation as a mechanism for modulating limb impedance

Several previous studies have demonstrated significant contributions of reflex responses to the stiff and viscous components of limb impedance (Crago et. al. 1976; Houk 1979; Nichols and Houk 1976; Houk and Rymer 1981; Wu et. al. 1990). In our study, we cannot obtain a true measure of limb impedance because we applied perturbations only in two directions. However,
our assessment of changes in reaction force to a perturbation that caused similar trajectory displacements across conditions reflects modulation of limb impedance in the direction of the perturbation. Because the state of the muscle prior to the application of the perturbation was the same under all conditions (see Results), this modulation of reaction force across the congruent and incongruent target jump + mechanical perturbation conditions must have resulted from reflex-mediated mechanisms. In agreement with the proposal of Kimura et al (2006), we suggest that such modulation of reaction forces through changes in reflex output is an indication of task dependent modulation of limb impedance. The fact that these resistive forces to the same perturbation were different depending on whether task goals were changed, suggests that limb impedance was selectively modulated.

**Impedance modulation is not limited to pre-movement expectations of dynamic events**

Traditional models of neural control of movement have suggested that entire movement trajectories might first be planned and then executed (Flash and Hogan 1985; Goble et al 2007; Soechting et al 1995; Uno et al 1989). Within such planning-execution models, error feedback that might arise for example, through transient force perturbations has been shown to affect planning of subsequent movements (Scheidt et al 2001; Fine and Thoroughman 2006). Thus, if a dynamic perturbation is expected on a subsequent movement, it has been suggested that reflex gains are set anticipatorily, to tune limb impedance in a manner beneficial for countering the imposed perturbation. Consistent with this idea, several studies have demonstrated tuning of the long latency reflex response in tasks requiring maintenance of static arm positions and application of predictable mechanical perturbations (Hammond 1956, 1960; Lacquaniti and Maioli 1989), and movements made in predictable dynamical environments (Burdet et al 2001; Franklin et al 2003; Kimura et al 2006; Wang et al 2001).

Our current findings extend these results by demonstrating that the ability of the nervous system to modulate limb impedance is not restricted solely to conditions where task dynamics are predictable and perturbation contingencies are known prior to movement onset. Rather, our results show the remarkable ability of the nervous system to alter limb impedance during movement, when perturbations are not predictable and task goals change after movement onset. Furthermore, we show that not only the long latency, but also the short latency component of reflex responses to perturbations can be modulated through visually detected changes in task goals. We conclude that control of limb impedance can occur through changes in both spinal and supraspinal reflex excitability and that this control appears to underlie error reduction mechanisms during goal directed movements.

**Limb impedance regulation as a component of modified control signals**

Instead of imposing a strict distinction between movement planning and execution processes, recent computational theories of motor coordination based on stochastic optimal feedback control suggest that control signals are derived online based on task-specific cost functions (Todorov and Jordan 2002). Although this model is currently limited by simplification of both the physical plant and the structure of the modeled noise, it provides an attractive computational framework for understanding how the nervous system might account for variations in environmental and internal conditions. Such a control structure is advantageous if task goals change during movement; control signals can then be appropriately modified to suit the demands of the modified goals. Our results suggest that modulation of limb impedance is an integral component of formulating the optimal response to changes in task goals. Thus, the nervous system derives optimal control signals not only to respond to the changes in task goals itself, but it also tunes limb impedance to ensure that any perturbations arising after task goals have been modified are countered in a task specific manner. Reflexive resistance to an opposing perturbation is rapidly increased following changes in task goals, whereas resistance to the same perturbation is decreased when it is congruent with changes in task goals. This finding agrees with the “minimum intervention principle” within the optimal feedback control.
framework, in which the controller “intervenes” to counter perturbations that interfere with task goals, but does not resist perturbations that assist goal achievement. We demonstrate that one mechanism to confer such extraordinary specificity is the tuning of reflex circuits in accord with changing task goals.

**Does hierarchical organization give rise to distinct responses to changes in task goals?**

Apart from illustrating the striking online modulation of reflex responses, our data also distinguish between two responses to the changes in target location. The early response to the target displacement results in modulation of reflex output, whereas the second response, occurring about 100 milliseconds later, likely reflects voluntary behavior. Moreover, results from our second experiment reveal that the modulation of reflex responses, although specific to the direction of changes in visual information, was not stratified based on the amplitude of those changes. The late response observed on trials on which the target was jumped, but no mechanical perturbation was applied, was scaled with the amplitude of the target displacement. We suggest that these two responses occurring at distinct latencies and their specificity to different features of the target change likely reflects two distinct hierarchical processing systems within the visuo-motor system.

It is well known that retinal information is transmitted primarily to the superior colliculus and the lateral geniculate - visual cortical areas. Both these structures have previously been implicated in online trajectory corrections to changes in visual target location (Day and Brown 2001). In fact, Day and Lyon (2000) proposed a “dual pathway” model by which the superior collicular pathway and the cortical pathway might differentially modify motor responses to changing target information. They suggested that the collicular pathway might produce an early, but rudimentary modification in response to the imposed change in visual information. Alstermark and colleagues previously demonstrated that this low-level subcortical pathway is exploited for movement corrections in cats (Alstermark et al 1987, 1990). They showed that lesions to tectospinal and tectoreticulospinal neurons disrupted a short latency response, occurring within 100 milliseconds of a target jump. However, this lesion did not disrupt a longer latency response that was presumably mediated by higher-level visual cortical circuits (Alstermark et al 1987). According to the dual pathway model, this cortical mediated response is more accurate and better adapted to the spatial characteristics of the new target. In our current studies, the early response, measured by direction but not amplitude specific changes in spinal reflex response, is consistent with the tectospinal pathway dependent early response to target location changes in cats demonstrated by Alstermark et al (1987). We thus speculate that lower-level visuo-motor pathways might modulate spinal excitability to cause early changes in reflex output, as demonstrated in our study. In contrast, we expect that higher-level visual cortical pathways might underlie the longer latency corrections to the target jump, which are scaled with the amplitude of the new target.

**Conclusion**

In conclusion, our findings strongly suggest that spinal and higher order reflex circuits are under continuous influence of descending commands. Reflex modulation is not limited to pre-movement expectations of dynamic perturbations, but can occur rapidly through the earliest visually detected changes in target location. This modulation, although specific only to direction of the visual changes is critical for ensuring that perturbations encountered after movement onset are compensated in a manner consistent with task goals. Later responses appear to be better calibrated with specifics of changes in task goals. These distinct responses likely result from distinct hierarchical neural visuo-motor pathways.
Experimental Procedures

Participants

14 healthy subjects were recruited for the study (6 for experiment 1 and 8 for experiment 2). Only right-handers were selected; handedness was determined using a 12-item version of the Edinburgh inventory (Oldfield 1971). All participants gave informed consent prior to participation. Informed consent had been approved by the Institutional Review Board of the Pennsylvania State University.

Experimental Setup

Subjects sat facing a table with their hand supported over the horizontal surface positioned just below shoulder height (adjusted to subjects’ comfort) by an air sled system, which minimized the effects of friction and gravity. A cursor representing finger position, a start circle (1 cm diameter) and a target were projected on a horizontal back projection screen positioned above the arm. The refresh rate of the projector was 75 Hz. A mirror placed parallel and below this screen reflected the visual display so as to give the illusion that the display was in the same horizontal plane as the fingertip. Calibration of the display assured that this projection was veridical. The air sled was attached to a MIT IMT2 robot arm with an ATI 6 degree of freedom (DOF) force transducer to measure interface forces (Krebs et al 1999). The upper arm was stabilized using an adjustable brace such that no movement of the upper arm was allowed. Movements were thus restricted to the elbow joint only. Position and orientation of the forearm and upper-arm segments were sampled using a Flock of Birds (Ascension Technology) electromagnetic 6-DOF movement recording system. The position of the index fingertip, the lateral epicondyle of the humerus and the acromion, directly posterior to the acromio-clavicular joint were recorded using a stylus that was rigidly attached to a 6-DOF Flock of Birds (FOB) sensor. One 6-DOF sensor was then attached to the upper arm segment by means of a plastic arm cuff, while the other sensor was attached to the air sled upon which the forearm was fixed. The sensors were positioned at approximately the center of each segment. As sensor data were received from the FOB, the 3-D position of the above mentioned landmarks was computed by our custom software, with the X-Y plane parallel to the tabletop. We thus used our computed X-Y coordinates of the fingertip to define the projected cursor position. Digital data was collected at 103 Hz using a Macintosh computer, which controlled the sensors through separate serial ports. This data were stored on disk for further analysis. Custom computer algorithms for experimental control and data analysis were written in REAL BASIC (REAL Software) and Igor Pro (Wavemetrics, Inc) respectively. Electromyographic (EMG) activity was recorded from four muscles crossing the elbow joint: biceps brachii, brachioradialis (elbow flexors) and long and lateral heads of the triceps (elbow extensors). EMG was recorded using active stainless steel electrodes with a built-in reference electrode (B & L Engineering). EMG signals were digitized at 1 kHz using a Macintosh computer equipped with an A/D board (National Instruments, PCI-MIO-16XE-50). The EMG signals were full wave rectified and subsequently low-pass filtered at 30 Hz using a third order dual-pass Butterworth filter to eliminate high frequency noise components. Temporal resolution of the EMG data was maintained at 1 kHz.

Experimental task

The experimental task required subjects to make rapid single joint 30-degree elbow extension movements from the defined start circle to the target in response to an audio-visual go signal. All the movements were made without any visual feedback of the subject’s arm or on-screen cursor position. Points were awarded based on the accuracy of these trials. Final position errors of < 1 cm were awarded 10 points, errors between 1 and 2 cm were awarded 3 points and errors between 2 and 3 cm were given 1 point. Points were displayed on the screen following each trial. These “baseline” single joint extension movements comprised 75% of all trials.
On random trials, after movement initiation, we “jumped” the target to a new location, or applied a mechanical perturbation, or implemented a combination of both, the target jump and mechanical perturbation. For experiment 1, the target jump occurred when the subject first breached the start circle boundary and required a 15-degree extensor or flexor correction. 10 trials of each type were randomly implemented, thus yielding 20 target jump trials in all. Subjects were instructed to move toward the new target location as soon as they perceived the jump. Points were awarded based on the accuracy in achieving this new target. For experiment 2, similar extensor and flexor target jumps were implemented. However, in a particular direction, the target jump required either a 15-degree or a 30-degree corrective response. In this case, the target was jumped randomly 7 times for each direction and amplitude combination, thus yielding 28 target jump trials in all.

Because we used a projector with a refresh rate of 75 Hz, we had to take into account the delay between the time that the target jump command was issued and the time that the screen was refreshed to display the displaced target. This time could range from a minimum of 0 to a maximum of 13 milliseconds. Assuming a distribution of such times, we chose a relatively conservative value of 10 milliseconds for this delay period. This time was then subtracted out from the estimated response time to the target jump.

A 40 N direction dependent force pulse lasting 30 milliseconds was used to apply a mechanical perturbation to the moving limb. This force was applied 100 milliseconds after the trigger using the robot arm. For experiment 1, we used both, extensor and flexor perturbations (10 random trials of each type, without any target jumps). However, for reasons described earlier, our main interest was to examine perturbation responses only on the extensor forces trials. For experiment 2, given the large number of trials for the various combinations of experimental conditions and to avoid subject’s fatigue, we used only the extensor mechanical perturbation. 7 such trials were randomly implemented in experiment 2. Subjects were instructed to correct for the perturbation and achieve the target on the screen.

On trials where both the target jump and the mechanical perturbation were applied (target jump + mechanical perturbation), the target was jumped first on movement initiation, followed by the force pulse 100 milliseconds later. Thus we provided 100 milliseconds between these events for visual information to be processed. On these trials, the direction of the target jump and the force pulse was randomly chosen, yielding different combinations of these perturbations: congruent conditions in which an extensor target jump preceded the extensor force pulse, or incongruent conditions in which the target jump was flexor but the perturbation remained extensor. 5 trials of each type were implemented for experiment 1, thus yielding 10 target jump + mechanical perturbation trials in all. For experiment 2, we used 7 trials for each target jump and extensor perturbation combination, thus yielding a total of 28 target jump + mechanical perturbation trials. No perturbation was applied during the first 20 trials in either experiment.

**Kinematic Data Analysis**

Elbow and shoulder angles were calculated from the 3-D positions of the finger, elbow and shoulder. All kinematic data were filtered at 8 Hz using a 3rd order dual-pass Butterworth filter and angular data was differentiated to yield velocity and acceleration values. The first 20 trials were considered practice trials and were not considered for analysis. Trials on which the subject failed to make a corrective response to any perturbation condition were also excluded from analysis. At the beginning of each trial, hand velocity was close to zero, but small oscillations occurred within the start circle. Movement onset was defined as the point at which subjects first breached the start circle. The small diameter of the start circle ensured that this time of movement initiation was fairly consistent across trials. Movement termination was defined as the first minimum (below 5% maximum tangential velocity) following peak tangential finger velocity. Peak angular velocity and peak angular acceleration were calculated using custom
algorithms. Final position accuracy was calculated as the distance between the final hand position and the center of the target.

**Kinetic Data Analysis**

Although the motion of the upper arm was constrained using an arm cuff, small displacements of the upper arm segment could still occur, especially when the mechanical perturbation was applied. In order to accurately account for the effects of such motion on the motion of the forearm, we used a planar two-segment rigid body model to calculate joint torques at the elbow. The arm was modeled as consisting of two interconnected rigid links with frictionless joints at the shoulder and elbow with forces from the robot manipulandum acting at the distal end of the forearm link. The torque at the elbow was calculated as:

\[
\tau_e = A\ddot{\theta}_e + (A + B\cos(\theta_e))\dot{\theta}_e + B\sin(\theta_e)\dot{\theta}_e^2 - C\sin(\theta_s + \theta_e)\ddot{x}_s + C\cos(\theta_s + \theta_e)\ddot{y}_s - I_e\sin(\theta_s + \theta_e)F_x + I_e\cos(\theta_s + \theta_e)F_y
\]

where

\[
A = I_e + m_er_e^2, \quad B = m_uls, \quad C = m_ur_e
\]

The suffixes “e” and “s” refer to the elbow and shoulder respectively, \(m\) represents mass of the segment, \(I\) represents moment of inertia, \(l\) is the length of the segment and \(r\) is the distance of the center of mass from the proximal joint. In addition, \(\ddot{x}_s\) and \(\ddot{y}_s\) represent shoulder accelerations in the horizontal \(XY\) plane and \(F_x\) and \(F_y\) are components of the total external force \(F\) applied by the robot manipulandum along the \(X\) and \(Y\) axes. \(\theta_s\) is the external angle between the upper arm segment and the horizontal \(X\) axis, while \(\theta_e\) is the external angle between the forearm segment and the upper arm. For implementation in the equations of motion, the limb segment inertia, center of mass and mass were computed from the regression equations using the subjects’ body mass and segment lengths (Winter 1990). It must be pointed out that we obtained interface force data starting only at the trigger (point when the start circle was first breached). Therefore joint torques were calculated from the time of the trigger to movement end. This explains the sharp transition seen in the muscle torque profiles in figures 1 and 4 at the very beginning of the movement (up to time 0).

**EMG Data Analysis**

EMG was recorded from 500 milliseconds prior to our movement initiation and was normalized to percent of maximum of EMG for each muscle, within subjects. In order to compare the timing of the EMG responses during mechanical perturbation and target jump trials to baseline EMG, we first calculated the average baseline EMG response from 10 baseline trials. These baseline trials were randomly selected, but we ensured that they were not trials either directly before or after a target jump or mechanical perturbation trial. As an example, figure 6 (top) shows the average baseline biceps response for an individual subject (black profile) along with the response in the same muscle for a mechanical perturbation trial (gray). For the muscle of interest, we then subtracted this average baseline response from each accepted EMG response in the target jump and mechanical perturbation trials to yield a difference profile. Figure 6 (bottom) shows the difference between the exemplar baseline and perturbation responses. The maximum difference between the baseline and the target jump/mechanical perturbation trials...
was then calculated as the peak in the difference profile. This peak difference is marked as the dark gray cross on the difference profile of figure 6 (bottom). Starting from the time of this peak, we searched backwards for the first time point that was below 3% of the peak in the difference profile. In our example, this point is shown by the light gray cross. For the mechanical perturbation trials, this time was taken as the response time. In case of target jump trials, as mentioned earlier, we subtracted 10 milliseconds from this calculated time value to yield the latency of the response to the to jump. This latency in each particular condition was then averaged to yield the average response initiation latency for one subject for a perturbation/target jump condition. This analysis was also performed to detect changes in kinematic and kinetic measures across the different target jump and mechanical perturbation conditions. All values were confirmed by subsequent visual inspection.

Although we used filtered EMG data to estimate onset of the response to the mechanical perturbation and target jumps, filtering did not affect response latency estimation. We repeated our analysis on mechanical perturbation trials for experiment 1 without filtering the EMG data. A comparison of response times obtained with and without filtering showed no significant differences between the conditions (p = 0.903). In fact, the earliest response following the perturbation was at exactly the same time irrespective of whether the filtered or the unfiltered data was used (21 milliseconds following the perturbation). Thus, the filtering the EMG data did not affect evaluation of response onset. Moreover, as demonstrated by Hodges and Bui (1996), low-pass filtering the data might in fact lead to better inferences of response onset.

For each subject, following the evaluation of the onset of the response to the perturbation, we calculated the EMG impulse over the first three 30 milliseconds intervals following response onset and qualified these as the short, medium and long latency responses. We chose this time interval based on the observation that changes in EMG should be sustained for greater than 25 milliseconds to cause appreciable changes in joint torque (Bagesteiro and Sainburg 2005; Hodges and Bui 1996). These analyses were carried out separately for experiment 1 and 2. In order to assess changes in reflex responses based on visual information, we compared the percentage change in the EMG impulse relative to baseline under the mechanical perturbation and target jump + mechanical perturbation conditions for each of the short, medium and long time intervals. In order to do this, we first calculated the difference between the EMG impulse for the particular perturbation condition and the impulse for the baseline trials. We divided this difference by the baseline EMG impulse and multiplied the result by 100 to yield a percentage value. Statistical analysis was performed on this measure. For illustration purposes, we expressed the change in EMG impulse as a multiple of the EMG impulse under baseline trials at short, medium and long latencies. This measure was simply obtained by dividing the EMG impulse under the mechanical perturbation or target jump + mechanical perturbation conditions by the baseline EMG impulse for each of the short, medium and long latency intervals.

**Statistical Analysis**

We compared the percent change in reflex response to the mechanical perturbation under the various conditions using one-way ANOVA, the factor being the direction of the target jump. Subjects were treated as a random factor for this analysis. The ANOVA was performed independently for the short, medium and long latency reflex intervals. Statistical significance levels were set to 0.05.

**Acknowledgments**

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References


Figure 1. Responses to Target Jumps

**Left Panel:** Far Target Jump. **Right Panel:** Close Target Jump. Baseline trials are shown in thin black, whereas target jump trials are shown in thick black. **1A.** Representative handpaths and velocity profiles (inlay) from a single trial. **1B.** Ensemble averaged Elbow Displacement. **1C.** Ensemble averaged Elbow Velocity. **1D.** Ensemble averaged Muscle Torque. **1E.** Ensemble averaged Agonist (Triceps) EMG. **1F.** Ensemble averaged Antagonist (Biceps) EMG. All profiles are for a representative subject. Vertical dotted line indicates onset of target jump.
Figure 2. Reflex Response and its modulation by preceding target jumps

2A. Ensemble averaged antagonist (top) and agonist (bottom, inverted) EMG profiles under baseline (thin black) and mechanical perturbation (thick black) trials. 2B. Average EMG response under congruent target jump + mechanical perturbation (dark gray) and incongruent target jump + mechanical perturbation trials (light gray) for a representative subject, expanded in time. 2C. Change in EMG integrals as a multiple of baseline EMG integral under mechanical perturbation (black), congruent (dark gray) and incongruent (light gray) target jump + mechanical perturbation trials at short, medium and long latencies across all subjects. C = Congruent, I = Incongruent, M = Mechanical Perturbation.
Figure 3. Changes in reaction force

3A Ensemble average reaction force profiles under mechanical perturbation (thick black), congruent target jump + mechanical perturbation (dark gray) and incongruent target jump + mechanical perturbation (light gray) trials in the X and Y direction for a representative subject.

3B Reaction force measured 30 milliseconds following the short, medium and long latency reflex intervals under congruent target jump + mechanical perturbation (dark gray) and incongruent target jump + mechanical perturbation (light gray) conditions across all subjects. Data represented are Mean ± SEM.
Figure 4. EMG responses to 15-degree and 30-degree target jumps

**Left Panel:** Far Target Jump. **Right Panel:** Close Target Jump. Baseline trials are shown in thin black, 15-degree target jump is shown in light gray and 30-degree target jump is shown in dark gray. **4A:** Ensemble averaged Elbow displacement. **4B:** Ensemble averaged muscle torques. **4C:** Ensemble averaged Agonist (Triceps) EMG. **4D:** Ensemble averaged antagonist (Biceps) EMG. **4E:** Peak activity in the antagonist (left panel) and agonist (right panel) following the corrective response across all subjects in the 15-degree (light gray) and 30-degree (dark gray) target jump conditions. Data represented are Mean ± SEM.
Figure 5. Direction but not amplitude based modulation of reflex responses

5A: Ensemble averaged antagonist EMG responses under congruent 15-degree (dotted dark gray) and 30-degree target jump + mechanical perturbation (solid dark gray) trials in the Far direction for a single subject, expanded in time. Also shown are ensemble averaged antagonist EMG responses in the incongruent 15-degree (dotted light gray) and 30-degree (solid dark gray) target jump + mechanical perturbation condition. 5B Change in EMG integrals as a multiple of baseline EMG integrals across all subjects under mechanical perturbation (black bars), congruent 15-degree (hatched dark gray bars) and 30-degree (filled dark gray bars) target jump + mechanical perturbation conditions. Change in EMG integrals under incongruent target jump + mechanical perturbation conditions are also shown for the 15-degree (hatched light gray bars) and 30-degree (filled light gray bars) target jumps. Data are shown for short, medium and long latencies. C = Congruent, I = Incongruent, M = Mechanical Perturbation.
Figure 6. Calculation of response onset
Top panel shows the rectified biceps EMG in a mechanical perturbation trial (gray) overlaid on a baseline response (black) in the same muscle. Bottom panel shows the difference between the two profiles in the top panel. The dark gray cross represents the peak difference, whereas the light gray cross represents the time of onset of the response to the mechanical perturbation using our calculation procedures (see Experimental Procedures)
**Table 1**

P-values for comparisons across conditions for experiment 1

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<th>Comparison</th>
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<td>Congruent target jump + mechanical perturbation - Incongruent target jump + mechanical perturbation</td>
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