Differential Controls Over Tactile Detection in Humans by Motor Commands and Peripheral Reafference

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Chapman, C. Elaine and Evelyne Beauchamp. Differential controls over tactile detection in humans by motor commands and peripheral reafference. J Neurophysiol 96: 1664–1675, 2006. First published June 14, 2006; doi:10.1152/jn.00214.2006. The purpose of this study was to determine the extent to which motor commands and peripheral reafference differentially control the detection of near-threshold, tactile stimuli. Detection of weak electrical stimuli applied to the index finger (D2) was evaluated with two bias-free measures of sensory detection, the index of detectability (d′) and the proportion of stimuli detected. Stimuli were presented at different delays prior to and during two motor tasks, D2 abduction, and elbow extension; both tasks were tested in two modes, active and passive. For both active tasks, the peak decrease in tactile suppression occurred at the onset of electromyographic activity. The time course for the suppression of detection during active and passive D2 abduction was identical, and preceded the onset of movement (respectively, −35 and −47 ms). These results suggest that movement reafference alone, acting through a mechanism of backward masking, could explain the modulation seen with D2 movement. In contrast, tactile suppression was significantly earlier for active elbow movements (−59 ms) as compared with passive (−21 ms), an observation consistent with both the motor command and peripheral reafference contributing to the suppression of detection of stimuli applied to D2 during movements about a proximal joint. A role for the motor command in tactile gating during distal movements cannot be discounted, however, because differences in the strength and distribution of the peripheral reafference may also have contributed to the proximo-distal differences in the timing of the suppression.

INTRODUCTION

The CNS possesses a variety of mechanisms to control the quality and the quantity of sensory feedback at any given moment. One such mechanism, gating of sensory feedback during voluntary movement, has been extensively studied. In brief, it is known that the transmission of somatosensory signals to primary somatosensory cortex (S1) is diminished during movement with the decrease beginning well in advance of movement onset. The latter observation has been widely interpreted as evidence that the motor command (efference copy) plays a key role in gating somatosensory signals (Chapman 1994; Chapman et al. 1988; Coulter 1974; Ghez and Lenzi 1971). Later, during movement, modulation is presumed to reflect the action of both the motor command and the sensory feedback generated by movement.

Perception of tactile stimuli is likewise decreased during voluntary movement (reviewed in Chapman 1994). The underlying mechanisms have been addressed using an experimental paradigm in which tactile detection was measured at various times prior to and after movement onset (Williams and Chapman 2000, 2002; Williams et al. 1998). We used a simple reaction time (RT) motor task, applying near-threshold, tactile stimuli at various delays after the visual GO cue. After each trial, subjects reported the presence or absence of the stimulus. Recently, we reported that the time course for modulation of tactile detection by active and passive abduction of the index finger (D2) is identical (Williams and Chapman 2002) with detection declining before movement onset in both cases. We suggested that the early decrease for the passive movements, prior to the onset of movement, could best be explained by “backward masking” of the test stimulus by movement-related reafference. Backward masking refers to the decrease in sensation that occurs when a second input occurs soon after an earlier test stimulus. Perception is not instantaneous but takes a finite length of time, thus allowing the masker (movement reafference) to interfere with processing of the test input. This phenomenon is not limited to the somatosensory system (Laskin and Spencer 1979) but has also been observed in the visual and auditory systems (Breitmeyer et al. 2004; Brosch et al. 1998; Rolls 2004). Although we believe that backward masking underlies the decreased perception prior to passive movement, we could not exclude the possibility that a systematic change in subject bias occurs, i.e., a change in nonsensory or cognitive factors, most particularly response criterion, and that this is responsible for the modulation prior to movement onset.

The purpose of this study was to determine the extent to which motor commands and peripheral reafference differentially control the detection of stimuli applied to D2. First, we determined whether tactile detection is modified prior to the onset of passive D2 abduction using an experimental design that included equal numbers of trials with and without a stimulus, and was based on signal detection theory (Green and Swets 1988). We calculated two bias-free measures of sensory detection, the index of detectability (d′) and the proportion of stimuli detected, at various times prior to and during movement, so ensuring that changes in bias could not contribute to the results. For comparison, the same subjects were also tested using active D2 abduction. Second, we investigated the generalizability of these findings by including a second motor task involving a proximal movement, elbow extension. The interest here was that this task corresponded to that used by Chapman.
et al. (1988) to study the time course of modulation of the earliest component of S1 somatosensory-evoked potentials (SEPs) during active and passive movements in monkeys. In the latter experiments, we showed that the time course for modulation varies with the mode of movement with SEPs showing a decrease before active movement onset and after passive movement onset. By including this second motor task, we were thus able to determine whether the results varied as a function of the topographical relation between the movement, proximal or distal, and the site of stimulation (distal, D2).

METHODS

Subjects

Four women and four men (21–35 yr of age, all but 1 right-handed for writing) participated in the experiments. All subjects were healthy individuals with no history of neurological or orthopedic disorders affecting the right arm. The experimental protocol was approved by the local human ethics committee, and all subjects provided their informed consent before participating in the study. Data from each subject were collected in two 1-h sessions either on the same day (separated by a 15-min rest) or consecutive days. At the beginning of each session, subjects were provided with a written and verbal description of the motor and perceptual tasks. After a block of practice trials, data collection began. Many of the experimental methods have been previously published (Williams and Chapman 2002; Williams et al. 1998); a brief synopsis is provided in the following text.

Motor tasks

Two simple RT motor tasks were tested in separate experimental sessions. In both cases, movement was initiated after the appearance of a visual go cue (3 × 3 array of green light-emitting diodes located at eye level, 1.1 m in front of the subject). One motor task consisted of abduction of the right D2. The finger rested on a pivoting plate (Fig. 1A) with the axis of rotation under the second metacarpophalangeal joint (Williams et al. 1998). The D2 manipulandum was placed on a small table just to the right of the seated subject. The other motor task required extension of the right elbow. For this task, the right forearm and hand were supported on a manipulandum, with the axis of rotation under the elbow (Fig. 1B); note that D2 (and the rest of the hand) rested on a flat plate attached to the manipulandum. For both motor tasks, the manipulandum was freely moving, i.e., there was no load applied.

Two conditions were tested for each motor task, active movement and passive movement. For the active motor task, subjects were instructed to generate the required movement as soon as possible after the go cue appeared. For the passive motor task, we used the same approach as used by Williams and Chapman (2002): a helper generated the passive movement by means of a mechanical link to the manipulandum, moving the same joint as the subject. The helper received the same instructions as did the subject for the active motor task. The subject was, in addition, instructed to remain relaxed throughout the testing. The helper was not visible to the subject (she was seated behind a curtain), but the visual cue was visible to both the subject and the helper. Availability of the visual cue ensured that subjects were in the same state as regards their expectation of an upcoming stimulus to detect in the active and passive motor tasks. This was an important consideration because it is known that attention has powerful controls over sensory detection (Driver and Spence 1998; Zompa and Chapman 1995).

For each motor task, the initial joint position corresponded to a relaxed, neutral position (0° abduction for D2; ~90° flexion for the elbow). Joint position was displayed on an oscilloscope in front of the subject. Traces displayed on the oscilloscope showed the desired initial position, as well as the minimal amplitude (15° for both tasks). Prior to beginning the experiment, practice trials were performed both by the subject and the helper.

Perceptual task and psychophysical method

The task is described in detail in Williams et al. (1998). Subjects detected the presence of near-threshold electrical stimuli applied via surface electrodes (7 mm diam) affixed to the glabrous surface of the middle and distal phalanges of the right D2 (Fig. 1A). The stimulus was a single, constant-current, square-wave pulse (2-ms duration), eliciting a weak paresthesia localized to the glabrous skin of the finger. At the beginning of the experimental session, and with the subject at rest, we determined the intensity at which 90% of stimuli were detected, and this intensity was used throughout the session (0.37–1.19 mA). Stimuli were presented at different delays after the visual go cue (see following text), and this in separate blocks of 54 trials each.

The signal-detection theory (SDT) rating procedure was used during the experiments, allowing us to generate a bias-free measure of sensory detection, d’ (index of detectability, see following text) (Green and Swets 1988). The stimulus was present in 50% of the trials (signal trials); the other 50% were nonsignal trials (no stimulus). After each trial, subjects were asked to report whether the stimulus was present or absent and to rate their confidence in this decision: 1) certain that the signal was present, 2) rather certain that the signal was absent, 3) unsure whether signal absent or present, 4) rather certain that the signal was present, and 5) certain that the signal was present. No feedback about perceptual performance was given during the experiment.

Data collection

One motor task (both modes) was tested in each session. All trials for one mode of movement (270 trials in 5 blocks of 54 trials), active or passive, were completed before proceeding to testing of the other mode. To concentrate sampling around the onset of movement, RT was estimated for both active and passive movement at the beginning of the session (15 trials for each). These values were used to calculate five delays for stimulus delivery relative to the go cue (Fig. 1C): RT − 150 ms, RT − 100 ms, RT − 50 ms, RT, and RT + 50 ms. All factors were counterbalanced.

Two trial types were presented, motor task (movement, 74%) and rest (no movement, 26%), and equal proportions of each trial type were signal or nonsignal trials (preceding text). Prior to each trial, the subject (and helper when relevant) was instructed whether to move or not in response to the upcoming visual go cue. After each trial, subjects reported whether or not a stimulus was perceived and rated their certainty in this judgment (preceding text). This information was entered into the computer and saved with the trial.

Electromyographic (EMG) activity was recorded from selected muscles using surface electrodes: for D2 abduction, first dorsal interosseous (FDI, agonist), and abductor digitii minimi (synergist); for elbow extension, lateral head of triceps (agonist) and long head of biceps (antagonist). Position and EMG (full-wave rectified and integrated over 5 ms) were recorded during each trial.

Data analysis

As described previously (Williams et al. 1998), various temporal and kinematic parameters were calculated for each trial, including the time of onset of movement and agonist EMG activity, the amplitude and duration of the movement, peak velocity and peak acceleration. Trials in which spontaneous EMG activity was present prior to the onset of the GO cue (Fig. 1C, 500-ms period preceding the onset of the light cue) were eliminated from the analysis. For the passive task, trials were rejected if there was any EMG activity at any time.
Average timing and kinematic values were calculated for each task, and two-tailed t-tests were used for comparison.

The perceptual data from individual subjects were evaluated using two methods, one based on SDT and the other based on the proportion of stimuli detected (signal trials). For each subject, all movement trials from each task and mode were sorted into 40-ms bins relative to the onset of movement or the onset of EMG activity in the agonist muscle (active only). The corresponding rest trials were binned by assigning them the value of the average delay (relative to movement onset) for the block of movement trials recorded at the same time. For the SDT approach, the data from each bin (mean of 29 trials/bin, 50% signal and 50% nonsignal) were used to construct a receiver operating characteristic curve [ROC, hit rate (HR) vs. false alarm rate (FAR) calculated from each of the five rating categories]. From the ROC, the index of detectability ($d'_{H11032}$), a bias-free measure of sensitivity, was derived using a maximum-likelihood procedure applied to a logistic model (Systat version 9.0, SPSS, Chicago, IL); $d'$ was then plotted as a function of stimulus delay relative to movement onset. All subjects showed a time-dependent decrease in $d'$ during the movement trials, and so from this plot we interpolated the delay at which $d' = 1.35$. This corresponds to the delay at which subjects made 75% correct judgments (Johnson 1980), and this time was taken as one measure of the onset of a significant decrease in stimulus detection.

The second analysis, calculating the proportion of stimuli perceived for each bin (signal trials), was included to make a direct comparison with the results of our previous experiments (Williams and Chapman 2002). These proportions were compared with performance during the corresponding immobile trials (rest) using the Fisher one-tailed exact probability test. The data from each subject/task/mode were fit to the following logistic function (Williams et al. 1998): proportion detected $= ([\text{max} - \text{min}] / [1 + \exp^{-4mpk(\text{delay}-tpk)}]) + \text{min}$. Here $mpk$ represents the peak slope, $tpk$ represents the time of the peak slope, $max$ represents the maximum proportion of stimuli detected, $min$ represents the minimum proportion of stimuli detected, and $delay$
represents the stimulus timing relative to movement (or EMG) onset. This latter analysis was repeated after sorting the trials into 20-ms bins, in an attempt to obtain a more precise estimate of the timing of the movement-related decrease in perception.

Repeated-measures analyses of variance (ANOVA) were employed to compare performance across the two tasks (D2, elbow), and two modes of movement (active and passive). The level of significance was fixed at \( P < 0.05 \). Most statistical analyses were performed using Systat, version 9.0.

RESULTS

Global performance

Subjects (\( n = 8 \)) detected weak near-threshold electrical stimuli applied to the right D2 during two motor tasks, right D2 abduction and right elbow extension. The movements were made either by the subjects themselves (active) or by a helper (passive). Detection performance was characterized both by calculating \( d' \) and the proportion of stimuli detected (signal trials).

There were modest differences in the temporal and kine-

matic parameters of the active and passive movements in this study (Table 1) with the latter having lower peak amplitudes (D2 only), peak velocities, and peak accelerations than the corresponding active movements. The potential contribution of these differences to the results is considered in the following text.

Global detection performance during the movement and rest trials is also summarized in Table 1. For the trials in which a stimulus was present, subjects reported detecting a stimulus in 89.5% of the rest trials and 55.3% of the movement trials. All subjects showed a time-dependent decrease in perception during both motor tasks and both modes of movement (active and passive). There was no evidence, on the other hand, that performance across the experimental sessions was modified by either practice or fatigue, as the proportion of stimuli detected (signal trials).

The data here are plotted as a function of the delay of the stimulus relative to the onset of movement. Both measures of detection, \( d' \) (A) and proportion detected (B), showed a time-

dependent decrease that began prior to the onset of movement (\( time 0 \) on the abscissa). When the stimulus was presented \( \pm 100 \) ms prior to the onset of movement (data to the far left of the vertical interrupted line showing movement onset), most of the stimuli presented were detected and \( d' \) values were high (>2.0), indicating that the subject perceived the near-threshold stimuli. These values were similar to those obtained in the rest trials (far left of each graph).

The timing of the decrease that preceded movement onset was characterized using two methods. For the \( d' \) data, the time at which \( d' \) declined to 1.35, a measure corresponding to 75% correct detections, was calculated using linear interpolation. For D2 abduction, \( d' \) reached threshold level at \(-41 \) ms during active movement; a similar result was obtained for passive D2 movements, \(-31 \) ms. For elbow extension, in contrast, the decrease during active movement was earlier than that seen during passive movement, respectively, \(-37 \) and \(-2 \) ms. Similar results were obtained using the proportion detected data. There was a significant decrease in the 40-ms bin that preceded movement onset for both modes of D2 movement and

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Active</th>
<th>Passive</th>
<th>A vs. P P Value</th>
</tr>
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<tbody>
<tr>
<td>D2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RT, ms</td>
<td>212 ± 53</td>
<td>237 ± 40</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>EMG onset, ms</td>
<td>167 ± 53</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>EMG lead time, ms</td>
<td>(-42 ± 14)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Movement duration, ms</td>
<td>156 ± 55</td>
<td>187 ± 57</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>Elbow</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RT, ms</td>
<td>227 ± 48</td>
<td>241 ± 31</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>EMG onset, ms</td>
<td>155 ± 53</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>EMG lead time, ms</td>
<td>(-51 ± 17)</td>
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<td>—</td>
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<tr>
<td>Movement duration, ms</td>
<td>287 ± 76</td>
<td>367 ± 46</td>
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Kinematic

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<tr>
<td>Peak amplitude (°)</td>
<td>27 ± 10</td>
<td>17 ± 5</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>Peak velocity (°/s)</td>
<td>349 ± 138</td>
<td>168 ± 101</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>Peak acceleration (°/s²)</td>
<td>6509 ± 2762</td>
<td>3167 ± 2239</td>
<td>&lt;0.0005</td>
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Detection performance

<table>
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<th>Passive</th>
<th>A vs. P P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>(proportion detected)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rest</td>
<td>0.98 (0.95, 0.99) *</td>
<td>0.93 (0.89, 0.95)</td>
<td>0.005</td>
</tr>
<tr>
<td>Motor task</td>
<td>0.50 (0.47, 0.54)</td>
<td>0.53 (0.50, 0.57)</td>
<td>0.25</td>
</tr>
<tr>
<td>Detection performance</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(mean confidence rating)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rest</td>
<td>4.9 ± 0.5</td>
<td>4.7 ± 0.9</td>
<td>0.001</td>
</tr>
<tr>
<td>Motor task</td>
<td>3.2 ± 1.7</td>
<td>3.3 ± 1.5</td>
<td>0.086</td>
</tr>
</tbody>
</table>

Parameters (means ± SD) along with detection performance, during active and passive movements (abduction D2, extension of elbow) in eight subjects. Paired comparisons used either t-tests or the Fisher exact test (proportions only). \*95% confidence interval. A, active; EMG, electromyographic activity; P, passive; RT, reaction time.

J Neurophysiol • VOL 96 • SEPTEMBER 2006 • www.jn.org
for active elbow movements (Fisher exact test, movement vs. rest). The earliest significant decrease for passive elbow movements occurred in the first bin after the onset of movement. Each data set (movement trials) was fit to a logistic function, and four parameters derived (see METHODS). The results are summarized in Fig. 2B. In particular, the timing of the peak decrease in slope, tpk, was similar for active and passive digit movements (respectively, −36 and −28 ms) but differed for active and passive elbow movements with the peak decrease occurring before the onset of active movement, −49 ms, and after the onset of passive movement, +11 ms. In addition, significantly fewer stimuli were detected during active compared with passive, elbow movements in the bin that immediately preceded movement onset (Fisher exact test, P = 0.007). No other bins in either task showed a significant difference as a function of the mode of movement.

Figure 3 shows the pooled results from eight subjects. The data for each subject, and each of the four test conditions, were analyzed as shown in Fig. 2. Inspection of Fig. 3 confirms that the results pooled across subjects were similar to the results obtained in the individual subject. Thus for movements involving D2 (Fig. 3, left), the time course for the modulation of perception was virtually identical for active and passive movements with the decrease preceding movement onset in both cases. The pooled data also confirmed that the modulation during passive elbow movements occurred later than that seen during active elbow movements (Fig. 3, right). These observations were confirmed using repeated-measures ANOVAs and post hoc contrasts. For D2 abduction, there was a time-dependent decrease in both perceptual measures (P < 0.0005), but no differences across active and passive movement (d', P = 0.75; proportion detected, P = 0.48). For elbow extension, mode was a significant factor (proportion detected, P = 0.035), as was delay (P < 0.0005), and there was a significant interaction, mode × delay (d', P = 0.029; proportion detected, P < 0.0005). Post hoc contrasts indicated that the differences were concentrated in the critical bins that preceded movement onset (starred values, Fig. 3, right).

For each subject, estimates of the timing of the decrease were obtained from both the d' (time that d' declined to 1.35) and proportion detected data (tpk from the logistic functions, corresponding to the time that subjects detected ~50% of the stimuli), and these are summarized in Table 2. No significant differences in timing were obtained when comparing the two methods of analysis (paired t-test). For both, perception declined prior to the onset of D2 movement, and there was no significant difference according to whether the movement was active or passive. In contrast, both methods revealed a significant difference in timing for the elbow movements with perception decreasing significantly earlier with active compared with passive movements.

We attempted to generate a more precise estimate of the timing of the decrease in the different test conditions by pooling the data of all subjects into smaller, 20-ms, bins. This analysis was restricted to the measure of the proportion of stimuli detected during signal trials (mean number of trials/bin/subject, 7 signal and 7 nonsignal) as there was insufficient data to reliably estimate d'. The results (Table 2) confirmed those obtained with the data binned at 40 ms and were not significantly different (paired t-test).

A repeated-measures ANOVA applied to the timing data (tpk calculated from the logistic functions) revealed a significant interaction between the motor task and the mode of movement (P = 0.001), although neither task nor mode were themselves significant factors. Post hoc contrast analyses indicated that the timing of the peak decrease was significantly different for active and passive movements of the elbow (P = 0.011), but not D2 (P = 0.28). The other logistic parameters...
(max, min, slope) showed no changes across tasks or modes. Similar results were obtained when the ANOVA was repeated with the 20-ms binned data. In contrast, the timing data generated by calculating the delay at which $d'$ declined to 75% correct showed no interaction between the task and mode of movement ($P = 0.138$), suggesting that this method was less sensitive in describing the time-dependent changes in perceptual performance, as compared with tpk from the logistic functions.

**Time-dependent changes in tactile detection relative to EMG onset**

The peak decrease in detection occurred significantly earlier during active elbow as compared with active D2 movements ($P = 0.008$). We reasoned that this might be related to differences in the timing of EMG activity relative to movement onset. In particular, we found that EMG lead time was significantly longer for elbow movements than for digit movements (Table 1, $P < 0.0005$), likely reflecting the higher inertia of the arm as compared with the digit. Figure 4 summarizes the results obtained when the proportion detected was plotted relative to EMG onset in the respective agonist muscle (FDI for D2 abduction; triceps brachii for elbow extension). Inspection shows that the two curves are superimposed. A comparison of the mean timing of the peak decrease relative to EMG onset indicated that there was no significant difference ($P = 0.88$). For both motor tasks, the peak decrease occurred close to the time of EMG onset (D2, $+9 \pm 6$ ms; elbow, $+7 \pm 8$ ms), indicating that active movement had similar suppressive effects on perception of stimuli applied to D2, independent of the moving joint. This contrasted with the differential timing for passive movements.

**Time-dependent changes in tactile detection in relation to peak movement velocity**

Although the global comparison (Table 1) indicated that active movements were faster than passive, detailed analyses

<table>
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<tr>
<th>Motor Task</th>
<th>D2 Abduction</th>
<th>Elbow Extension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active</td>
<td>-46 ± 8</td>
<td>-54 ± 8</td>
</tr>
<tr>
<td>Passive</td>
<td>-40 ± 15</td>
<td>-20 ± 9</td>
</tr>
<tr>
<td>A vs. P</td>
<td>0.756</td>
<td>0.009</td>
</tr>
</tbody>
</table>

**TABLE 2.** Mean estimates of the onset of gating of tactile detection relative to the onset of movement in 8 subjects as a function of the motor task and mode as well as the method of analysis

![Figure 3](http://jn.physiology.org/lookup/suppl/doi:10.1152/jn.00867.2005/-/DC1/f3a.png)

**FIG. 3.** A and B: pooled results from 8 subjects (means ± SE; 40-ms bins), plotted as in Fig. 2. Paired comparisons (t-test, active vs. passive) showed significant differences prior to movement onset (*) for elbow but not digit movements: the decrease in perception was earlier for active elbow extension than passive.
of the results of individual subjects revealed some exceptions. For the D2 abduction task, seven of eight subjects followed the global trend of faster active, as compared with passive, movements. The exception (subject 2, data illustrated in Figs. 1C and 2) had slower active than passive movements, and yet the results were similar to those obtained in the other subjects, with modulation occurring at the same time, prior to movement onset, for both active and passive movement. (Note: for simplicity, we employ peak velocity as a general descriptor of the movement parameters. In these simple RT tasks, the various movement parameters are closely interrelated: peak velocity shows significant positive linear relations with both movement amplitude and peak acceleration, \( r^2 > 0.66 \).) For the elbow extension task, three subjects had slower active than passive movements; the other five subjects had faster active (vs. passive) movements. Nevertheless, the timing difference (active − passive) was similar for both groups of subjects with active modulation preceding that seen during passive movement in both groups (respectively, 44 and 35 ms). Such observations suggest that the timing of the movement-related suppression of detection was not related in any simple manner to movement speed.

To better illustrate the apparent independence between the movement parameters and tactile detection, Fig. 5 summarizes the frequency distribution of detected (gray) and nondetected (black) signal trials as a function of the peak velocity (ordinate) of the subsequent movement and stimulus delay relative to movement onset. Individual data from all trials, and all subjects. A: active and passive D2 abduction. B: active and passive elbow extension.

![Proportion of stimluli detected (20-ms bins) relative to the onset of electromyographic (EMG) activity for active digit (○) and elbow (▲) movements (respectively, FDI and triceps brachii). Data from all subjects (n = 8) were pooled. Logistic parameters show the mean values for the curves fit separately for each subject and motor task. The 2 logistic functions are superimposed, indicating that the timing of the modulation relative to the motor command was identical for both movements.](image-url)
movement onset (abscissa). The data from all subjects are shown. For both tasks, inspection indicates that there was a clear trend for fewer trials to be detected at delays closer to, and after, movement onset. There was no evidence that detected stimuli were preferentially associated with slower movements. In the critical bins that preceded movement onset, one can see that subjects frequently failed to detect stimuli that preceded relatively slow movements or detected stimuli that preceded more rapid movements.

Response bias

Although the $d'$ measure itself is independent of response bias, we performed a further analysis to determine whether the FAR (proportion of nonsignal trials in which subjects reported a stimulus present) showed any sign of a change relative to the motor task, the mode of movement, or the stimulus delay. For each subject, the FAR was calculated for the data from each bin/task/mode. Overall, the FAR was low (1.7% of all nonsignal trials). Inspection of Fig. 6 indicates that the mean FAR showed no evidence for a time-dependent change across stimulus delay for either motor task or mode of movement. This was confirmed by applying a two-way ANOVA (with mode and delay as factors) to the results obtained with each motor task. For the elbow task, FAR did not covary with either factor ($P > 0.8$ in both cases). For the D2 task, FAR also did not vary with delay ($P = 0.90$), but there was a difference according to whether the movements were active or passive ($P = 0.006$); passive D2 movements had a higher FAR (5.5%, passive; 1.5%, active). This effect was limited to the movement trials as the FAR at rest did not vary as a function of the mode of movement (0.36% of trials during testing of each mode, $P = 1.0$). The reason for the higher FAR during passive D2 movements is not obvious but may be related to the fact that the passive movements were applied directly to the stimulated digit as compared with the elbow task in which case the movements were applied to the whole forearm/hand. Consistent with this interpretation, detection performance in the passive rest trials (D2) was significantly lower than for the active rest trials (Table 1).

Measures of sensory detection

We found that the timing values generated using the $d'$ measures and the proportion detected were overall similar. It is notable that the time that $d'$ declined to the criterion level ($d' = 1.35$) was very similar to the time of the peak decrease in the proportion of stimuli detected. The latter, in turn, occurred close to the level at which detection was $\sim 50\%$ (actual proportion detected at tpk varied from 47.8 to 50.3%), often taken as the criterion for defining detection threshold.

The two methods do not, on the other hand, appear to be equally sensitive to small changes in the timing of sensory suppression. First, a significant difference with mode (elbow movements) was obtained only with proportion detected, although both measures showed significant interactions (delay $\times$ mode). Second, the post hoc comparisons (Fig. 3) revealed an earlier difference for active and passive elbow movements when using proportion detected ($-60$ vs. $-20$ ms). Third, only the tpk timing data showed significant differences in a repeated-measures ANOVA looking at the effects of task and mode on the onset of sensory gating. Finally, a differential onset of sensory gating across the two active motor tasks (elbow earlier than D2) was only revealed with tpk ($P = 0.008$); the timing values estimated with $d' = 1.35$ showed no difference ($P = 0.249$). The latter results most likely reflect the fact that all signal trials contributed to calculating tpk from the logistic function, as compared with the restricted data set used to interpolate the time at which $d'$ declined to 1.35 (bins before and after $d'$ declined to 1.35). Altogether, it appears that proportion detected is likely the more sensitive method to use.

Discussion

Using bias-free measures of perception, this study showed that the time course for the suppression of tactile detection during active and passive movements was critically dependent on the motor task. Active and passive D2 (distal) movements yielded virtually identical results with the suppression preceding movement onset in both cases. In contrast, differential time courses for tactile suppression were seen with proximal (elbow) movements with active movement producing an earlier decrease in detection than passive movement, consistent with a role for the motor command in tactile gating. Movement reaference alone could, on the other hand, explain the results obtained with the distal task.

![Fig. 6. Proportion of false alarms (± SE) as a function of the stimulus delay relative to the onset of movement. There was no time-dependent change in FAR for either motor task or either mode of movement. Data from all subjects pooled (n = 8).](Image)
Methodological considerations

In this study, there were modest differences in movement parameters as a function of the mode of movement with active movements often being faster than passive movements. It is known that the magnitude of sensory suppression can covary with kinematics (Angel and Malenka 1982; Chapman et al. 1988, 1996) with faster movements showing a greater relative suppression. Although we do not know whether the timing of the peak decrease (tpk) is sensitive to changes in movement kinematics, it is known that systematic changes in stimulus intensity lead to marked changes in minimal and maximal detection performance. However, tpk, itself, is invariant (Williams and Chapman 2000). Consistent with this, the present study found no evidence that detected trials in the bins immediately before movement onset were preferentially associated with slower movements (Fig. 5).

Although both of the measures of tactile detection that we used were bias-free, we also considered the possibility that response bias (or willingness to report the presence of a stimulus) might have systematically changed in relation to the motor tasks. Given that a rating procedure was used in these experiments, however, any such calculation requires collapsing the data across the rating categories, thus losing some of the power of the experimental design. One such measure is the criterion C (Macmillan and Creelman 1990), corresponding to the midpoint between the z-transformed HR and FAR. Such methods are, however, based on the unstated assumption that signal strength does not vary, an assumption that cannot be met with the current experimental design in which the sensory signals were gated by the movement. Consequently, we restricted our analyses to determining the effects of the various test conditions on the FAR. The results indicated that FAR did not show a time-dependent change relative to movement onset, or any change with the mode of movement, at least for the elbow extension task. The slight increase in FAR during passive D2 movements (from 1.5 to 5.5%), on the other hand, can most likely explained by the distraction caused by the passive movements being applied directly to the stimulated digit. Consistent with this interpretation, tactile detection at rest was also lower in the trials in which passive movements were applied to D2.

Finally, this study did not, as in Williams and Chapman (2002), include “sham-movement” trials within the experimental design. That study included trials during the passive task in which the subject expected but did not receive a passive movement to determine whether central set regarding movement expectancy might have contributed to the results. Sensory detection showed no change (compared with rest), either at short or long delays (i.e., prior to or after the average RT of the helper). Such an observation is consistent with our previous demonstration that tactile detection is diminished during rhythmic, continuous elbow movements, active or passive (Chapman et al. 1987). In the latter study, there was no formal cue to indicate to subjects that a stimulus would be presented; subjects instead reported the occurrence of each stimulus detected (variable interstimulus interval). Taken together, these observations suggest that central set likely did not contribute to the modulation seen prior to or during passive movement.

Digit movements

The present results confirm that tactile detection is decreased prior to the onset of both passive and active D2 movements with no difference in the timing as a function of the mode of movement [respectively, $-47$ and $-35$ ms in this study, vs. $-38$ and $-49$ ms in Williams and Chapman (2002)]. Moreover, we can now rule out the possibility that a systematic change in response criterion contributed to the similar timing: the present results were based on two bias-free measures of sensory detection, $d'$ and proportion detected, and both showed that perception declined well before the onset of passive movement. This is an important observation because no previous study has controlled for the potential contribution of changes in response criterion to movement-related gating of tactile perception. The results thus provide support for our original hypothesis that backward masking of sensory perception by the peripheral reafference generated by the passive movements can best explain the results (see following text).

Elbow movements

Although active and passive elbow movements produced a time-dependent gating of tactile detection of stimuli applied to the index finger, two important differences in the timing of the modulation were observed as compared with the results obtained with D2 movements. First, modulation with active elbow movements was earlier than that seen with active D2 movements (respectively, $-59$ and $-35$ ms). This was explained by differences in inertia, with the hand + forearm requiring more time to accelerate from rest than the digit alone. Consistent with this, EMG lead time was significantly longer for elbow movements, and the timing difference disappeared when the data were aligned on EMG onset (Fig. 4). This latter observation extends our previous finding that the peak decrease during D2 abduction occurs close to the onset of EMG activity (Williams et al. 1998). Second, the modulation seen with active elbow movements was significantly earlier than that seen with passive elbow movement (respectively, $-59$ and $-21$ ms) and this in a manner consistent with our previous results obtained using intracortical SEP recordings from monkey SI, which showed decreased SEP amplitude prior to active movement onset ($-55$ ms) and after passive movement onset (Chapman et al. 1988). This contrasted with the identical timing seen with the distal motor task of D2 abduction.

Mechanisms contributing to movement-related gating of tactile detection

There are two potential interpretations of our findings. One interpretation is that only motor commands activating proximal muscles gate input from distal skin areas. As a corollary, the results obtained with the D2 motor task could be attributed entirely to peripheral reafference, consistent with the finding of no difference in timing for the two modes of digit movement. If this was correct, then we would be able to reconcile two apparently discordant previous observations. Williams et al. (1998) found a spatial gradient for the timing and magnitude of tactile suppression produced by D2 movement when the stimulation site was shifted from the moving finger to more proximal skin areas on the ipsilateral arm (Williams et al. 1998). The latter observation was not consistent with Jiang et
al.’s (1990) earlier observation that weak intracortical microstimulation (ICMS) of motor cortex in monkeys, essentially mimicking the motor command, decreases transmission of inputs to S1 cortex from skin areas overlying or distal to the activated muscle but not those from more proximal regions. If motor commands only gate inputs from distal (or overlying) skin areas, then the spatial gradient observed by Williams et al. could be attributed to reflecting the spatial distribution of peripheral feedback from the moving digit to cortical sites processing input from more proximal skin regions.

An alternate interpretation of the results is that the earlier suppression associated with passive D2 movements, as compared with passive elbow movements, can be explained by differences in the quantity of sensory feedback from the moving body part fed back to the cerebral cortical regions involved in analyzing the near-threshold tactile stimuli applied to D2. Two factors may have contributed. First, it is well known that the density of sensory innervation shows a proximo-distal gradient with higher densities of cutaneous mechanoreceptors on the digit than elsewhere on the hand/arm (Darian-Smith 1984) and higher muscle spindle densities in the distal muscles (Voss 1971). Thus the quantity of sensory feedback elicited by passive digit movement is likely greater than that elicited by elbow movements. Second, the somatotopic organization of S1 leads to the prediction that the regions involved in processing the near-threshold tactile stimuli applied to D2, the stimulation site in these experiments, receive quantitatively more feedback during digit as compared with elbow movements. Consistent with this suggestion, our unpublished observations (J. Shenasa, S. R. Williams, and C. E. Chapman) indicate that when near-threshold stimuli are applied to the forearm instead of D2, the timing of suppression with active and passive elbow movements is similar and substantially earlier than that seen here (~100 ms prior to movement onset, 3 subjects). A greater timing difference should be obtained if the active and passive movements were more remote from the stimulation site (e.g., shoulder). To summarize, we suggest that the regions involved in processing cutaneous input from the finger likely receive relatively more movement-related feedback from digit than elbow movements.

How can reafference-induced suppression of D2 detection precede movement onset, and so the earliest sensory feedback? We previously suggested that backward masking is responsible for the early suppression seen with passive D2 movements (Williams and Chapman 2002). Perception of a tactile stimulus is not instantaneous but takes time, so allowing a second and stronger input to influence the processing of the weaker and earlier test stimulus. The underlying neuronal mechanisms are likely both complex and varied. In the visual system, masking effects have been reported at very early levels of processing (Felsten and Wasserman 1980) as well as later stages, e.g., temporal cortex (Kovács et al. 1995; Rolls 2004). In addition, Laskin and Spencer (1979) showed that the degree of backward masking is proportional to the intensity of the masking stimulus. Thus the earlier modulation with D2 as compared with elbow passive movements may simply reflect differences in the strength of the masking provoked by sensory feedback.

In summary, the present results provide clear evidence that the motor command contributes to movement-related gating of sensory input from the stimulated digit. The results obtained with active and passive elbow movements reflect the combined action of both central and peripheral signals acting to suppress tactile feedback with the earlier modulation with active elbow movement reflecting the added action of the motor command. On the other hand, we cannot completely discount the hypothesis that movement-related reafference was entirely responsible for the modulation seen with active and passive digit movements and that there may be functional specializations of the gating mechanism(s) in relation to movements of distal versus proximal joints, at least for this experimental paradigm in which stimuli are imposed on the moving body part by an external agent.

Structures involved in gating

In this study, the test stimulus was a near-threshold electrical pulse applied to the index finger, activating mainly cutaneous afferents. It is known that both central and peripheral influences contribute to gating the transmission of cutaneous inputs through the lemniscal pathway during voluntary movement. As reviewed in Chapman (1994), the central influences likely originate from both S1 and primary motor cortex via their descending projections to the various relay sites in the dorsal column-medial lemniscal pathway. The modulation seen at the first major relay, the dorsal column nuclei (DCN), or earlier (spinal cord) is generally considered to be largely central in origin (Chapman et al. 1988; Coulter 1974; Ghez and Lenzi 1971). As this modulation can precede the earliest EMG activity, motor cortex likely plays an important role. Consistent with this suggestion, Jiang et al. (1990) showed that discrete activation of motor cortex (subthreshold intracortical microstimulation) can diminish the amplitude of S1 cortical SEPs. The underlying mechanisms are likely complex, and recent evidence confirms that inhibitory effects (in the form of presynaptic inhibition) prior to the onset of voluntary movement are present even at the level of the spinal cord (Seki et al. 2003). Modulation after movement onset, on the other hand, may be partly dependent on the corticofugal projections of S1, given that this has strong projections to all levels of the dorsal column-medial lemniscal pathway, including sensory thalamus, and the depth of modulation of cutaneous transmission during movement increases at higher levels of the pathway (Chapman et al. 1988). The latter observation is consistent with afferent feedback (direct or via back projections from S1) playing a major role in sensory gating.

Movement, active or passive, undoubtedly activated various muscle and joint receptors although the degree of activation may have varied with the mode of movement since their sensitivity is modulated by fusimotor and/or skeletomotor activity. In addition, finger movements likely activated cutaneous mechanoreceptors (Edin and Abb5 1991). Although neither Seki et al. (2003) nor Ghez and Pisa (1972) found evidence for presynaptic inhibition of cutaneous afferents during passive movements (respectively, wrist and vigorous movements of the whole forelimb), the latter noted that cutaneous stimulation depressed lemniscal transmission, consistent with previous observations (Eccles et al. 1963; reviewed in Rudomin and Schmidt 1999). Neither study tested the effects of digit movements, but it is known that cortical SEPs are decreased by cutaneous stimulation (Jones et al. 1989). To summarize, multiple structures likely contribute to movement-related gating, and the relative contribution of each appears to
vary across the chain of events that ultimately leads to perception.

Functional significance of movement-related gating

Movement-related gating of tactile detection has been shown to vary with both movement kinematics (Angel and Malenka 1982; Chapman et al. 1996; Schmidt et al. 1990) and kinetics (Pertovaara et al. 1992; Post et al. 1994). These observations are consistent with the motor command playing an important role. Moreover, the effects are not limited to the near threshold stimuli used here: the perceived intensity of suprathreshold tactile stimuli is also decreased prior to active movement, with the same time-course as reported here (Williams and Chapman 2000), and yet relative differences (discrimination threshold) are preserved (Chapman et al. 1987; Post et al. 1994).

Recently, Wolpert and collaborators (Bays et al. 2005, 2006; Blakemore et al. 1999; Shergill et al. 2003) provided evidence that the motor command itself gates the perception of the intensity of self-induced tactile sensations. They suggested that the motor command is tightly linked in both time and space to unexpected or novel stimuli is enhanced (Coulter 1974; Wil-
netive touch”, reducing the flow of afferent information that can predict a predictive mechanism of Wolpert and colleagues during “ac-

In 50% of trials, no stimulus was present. We suggest that support the notion that the motor command contributes to externally imposed stimuli during contralateral movements (Williams and Chapman 1998). Such findings suggest that self-induced and externally imposed tactile sensations may be differentially controlled. Bays et al. (2006) suggested that their results can best be explained by a predictive mechanism that serves to enhance perception of externally generated inputs, i.e., those that cannot be predicted from the motor command. Consistent with this, they found that self-induced inputs are suppressed only when the subject expects to generate sensory input. In our case, the subjects also expected to receive a stimulus, although in 50% of trials, no stimulus was present. We suggest that despite obvious differences in the underlying mechanisms, the functional role of the movement-related suppression of perception of externally applied stimuli is the same as for the predictive mechanism of Wolpert and colleagues during “act- active touch”, reducing the flow of afferent information that can be predicted from the motor command so that detection of unexpected or novel stimuli is enhanced (Coulter 1974; Will-

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