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Williams, Stephan R., and C. Elaine Chapman. Time course and magnitude of movement-related gating of tactile detection in humans. III. Effect of motor tasks. *J Neurophysiol* 88: 1968–1979, 2002; 10.1152/jn.00527.2001. This study investigated the relative importance of central and peripheral signals for movement-related gating by comparing the time course and magnitude of movement-related decreases in tactile detection during a reference motor task, active isotonic digit 2 (D2) abduction, with that seen during three test tasks: a comparison with active isometric D2 abduction (movement vs. no movement) evaluated the contribution of peripheral reafference generated by the movement to gating; a comparison with passive D2 abduction (motor command vs. no motor command; movement generated by an external agent) allowed us to evaluate the contribution of the central motor command to tactile gating; and finally, the inclusion of an active "no apparatus," or freehand, D2 abduction task allowed us to evaluate the potential contribution of incidental peripheral reafference generated by the position detecting apparatus to the results (apparatus vs. no apparatus). Weak electrical stimuli (2-ms pulse; intensity, 90% detected at rest) were applied to D2 at different delays before and after movement onset or electromyographic (EMG) activity onset. Significant time-dependent movement-related decreases in detection were obtained with all tasks. When the results obtained during the active isotonic movement task were compared with those obtained in the three test tasks, no significant differences in the functions describing detection performance over time were seen. The results obtained with the isometric D2 abduction task show that actual movement of a body part is not necessary to diminish detection of tactile stimuli in a manner similar to the decrease produced by isotonic, active movement. In the passive test task, the peak decrease in detection clearly preceded the onset of passive movement (by 38 ms) despite the lack of a motor command and, presumably, no movement-related peripheral reafference. A slightly but not significantly earlier decrease was obtained with active movement (49 ms before movement onset). Expectation of movement likely did not contribute to the results because stimulus detection during sham passive movement trials (subjects expected but did not receive a passive movement) was not different from performance at rest (no movement). The results obtained with passive movement are best explained by invoking backward masking of the test stimuli by movement-related reafference and demonstrate that movement-related reafference is sufficient to produce decreases in detection with a time course and amplitude not significantly different from that produced by active movement.

INTRODUCTION

The detection of tactile stimuli is reduced during movement (Chapman et al. 1987; Coquery et al. 1971; Duysens et al. 1995; Post et al. 1994; Schmidt et al. 1990a). The amplitude of the movement-related reduction in the detection of tactile stimuli depends on many factors, some of which pertain to the stimulus, and some of which pertain to the motor task. Previous papers in this series addressed the importance of stimulus parameters on detection performance. We showed that detection of stimuli during movement is not uniform over time and described the importance of stimulus location (Williams et al. 1998) and intensity (Williams and Chapman 2000) on the reduction in detection during movement.

The relation between factors related to the performance of the motor task and reductions in tactile detection is still unclear. Central signals related to the preparation and performance of the motor task are generally presumed to play an important role in the gating of afferent signals during movement. The evidence for this comes mainly from observations that the amplitude of somatosensory-evoked potentials (SEPs) is decreased prior to the onset of movement and movement-related electromyographic (EMG) activity (Chapman et al. 1988; Cohen and Starr 1987; Coulter 1974; Ghez and Lenzi 1971; Hazemann et al. 1975), i.e., before the generation of peripheral feedback. Consistent with this, there is no modulation of cortical SEP amplitude before the onset of passive movement (Chapman et al. 1988). Further evidence in favor of a central origin for the gating signals was provided by Jiang et al. (1990b), who demonstrated that the time course of the movement-related decrease in cortical SEPs was identical for isotonic and isometric tasks, a result that could be explained by postulating that the modulation was more closely linked to the central motor output than to the peripheral input generated in the two motor tasks. Furthermore, microstimulation of motor cortex can produce a significant reduction in the amplitude of cortical SEPs (Jiang et al. 1990a), possibly via collaterals from the pyramidal tract to the dorsal column nuclei (DCN) (Bentivoglio and Rustioni 1986; Cheema et al. 1985; Jones and Wise 1977; Kuypers 1958, 1960; Martinez et al. 1995) and/or projections to the dorsomedial part of the intermediate zone of the spinal gray matter, the source of postsynaptic input to the

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dorsal column nuclei (Dum and Strick 1996; Hayes and Rustioni 1980; Molenaar and Kuypers 1978).

Peripheral feedback generated by movement is also considered to be an important source of gating signals. The principal evidence for this comes from several studies that have demonstrated that passive movements can also diminish the amplitude of SEPs (Brooke et al. 1997; Huttunen and Homberg 1991; Kakigi et al. 1997; Rushton et al. 1981; Staines et al. 1996) but with a time course that is different from that seen for active movements because the modulation occurs only after movement onset (Chapman et al. 1988). These effects are clearly evident in SEP recordings taken either from the thalamus (ventroposterolateral nucleus) or primary somatosensory cortex (SI) but not at lower levels of the somatosensory system (medial lemniscus) (Chapman et al. 1988).

The results of psychophysical experiments, on the other hand, have provided less indication as to the origin (central and/or peripheral) of gating influences. Although the timing of the earliest decreases in detection for near-threshold stimuli, which precede movement onset by ≤120 ms (Williams et al. 1998), favors the notion that the first changes in detection performance are indeed related to the preparation and initiation of the motor command, the results of previous studies found no differences in the magnitude of reduction in detection during ongoing movements when comparisons were made for active versus passive movement (Chapman et al. 1987) and isometric versus isotonic contraction (Feine et al. 1990). The effects of reducing peripheral feedback are likewise equivocal. Schmidt et al. (1990b) found that local anesthetic blocks of digital nerves had only a modest effect on the movement-induced gating of the magnitude of sensations from the moving digit that were evoked by intraneural microstimulation. On the other hand, larger blocks (median + other nerves) produced larger decreases in the movement-related gating. The main purpose of this study was therefore to quantify and compare the time course and magnitude of movement-related reductions in the detection of weak electrical stimuli, for isotonic, isometric, and passive movement tasks. A preliminary report of some of these data has been presented elsewhere (Williams et al. 1998).

METHODS

Subjects

A total of 10 naive paid volunteers (5 males and 5 females, ages 17–27 yr) participated in the study. All subjects were right handed for writing. The institutional ethics committee approved the experimental protocol, and all subjects or their legal guardian gave their informed consent before participating in the study. Data from each subject were gathered in one to two sessions lasting 1–3 h each. At the beginning of each session, subjects received verbal instructions about the motor task and detection task that they were to perform. This was followed by a small block of practice trials, after which data collection began. Many of the experimental methods have already been published (Williams et al. 1998). A brief recapitulation as well as a description of salient differences is included in the following text.

Motor tasks

Four different motor tasks were tested, all on the right side, involving abduction of the index finger (D2). All tasks were reaction time tasks, i.e., subjects or their helper (passive movements) were instructed to initiate their motor response as rapidly as possible after the illumination of a visual GO cue (a 3 × 3 array of light-emitting diodes (LEDs) placed at eye level, 1 m in front of the subject).

Active isotonic D2 abduction, as described in Williams and Chapman (2000), served as the *reference motor task* (10 subjects). Results obtained using the reference motor task were compared with results obtained in *three test tasks*: active isometric abduction of D2 ($n = 7$); passive abduction of D2 (movement generated by a helper; $n = 10$); and "no apparatus" or freehand abduction of D2 ($n = 9$). In the first test task, we eliminated peripheral reafference generated by the movement itself by having subjects perform isometric D2 abductions. The subject's D2 was maintained in a maximally abducted position by a rubber hockey puck 2.5 cm high and 7.5 cm in diameter placed on top of the apparatus (Fig. 1*A*). We monitored the position of D2 by placing the digit on the pivoting plate that was instrumented with a potentiometer. The subject activated the first dorsal interosseous muscle (1st DI) in the same way as in the reference motor task, but D2 could not move as the muscle was already maximally shortened. Careful observation during the experiments indicated that no other fingers were moved during the isometric task. In previous experiments (e.g., Jiang et al. 1990b), comparisons across isotonic and isometric contractions were made by physically blocking the movement so that the isometric contractions were made against the block, generating supplementary cutaneous reafference. Our approach avoided this because the contractions attempted to move D2 away from the puck. Task performance was monitored by inspecting the electromyographic (EMG) activity of 1st DI during the experiment. The second test task was designed to eliminate the central motor command by repeating the testing during passive abduction of D2. In this task, the subject remained relaxed, or passive, while D2 abduction was generated by a helper's D2 and mechanically transmitted, without any form of servo assistance, by a connecting rod to the position detecting apparatus that in turn entrained the subject's D2 (Fig. 1*B*). Helpers received the same instructions relative to the performance of the motor task as those provided to the subjects when they actively produced the movements. The GO cue was clearly visible to both the helper and the subject, but the helper was not visible to the subject. The subject was instructed to remain relaxed. In the third test task, the "no apparatus" task, subjects performed active, isotonic abduction of D2 while their right arm hung unsupported by their side. D2 was in contact only with the stimulating electrodes. This test task aimed to quantify the effects on detection performance of any incidental tactile afference generated by entrainment of the position detecting apparatus during movement. Instructions relative to the performance of the motor task were identical to those in the reference motor task; performance was monitored by inspecting the EMG trace as in the preceding text for the isometric task.

Detection task

The detection task was identical to the one described in Williams and Chapman (2000). Surface electrodes (7 mm diam) were affixed to the glabrous skin of the distal and middle phalanges of the right D2 (position shown in Fig. 1*B*). Stimuli consisted of single 2-ms square-wave electrical pulses at an intensity where ~90% were detected at rest ($1 \times P_{90}$, current range: 0.4–0.88 mA). Stimuli were presented at different delays following the visual GO cue (see following text). The stimulation site used in this study was also employed in our previous studies (Fig. 2 in Williams et al. 1998; see also Williams and Chapman 2000).

Experimental design

The experimental design was described in Williams and Chapman (2000). In brief, stimuli were applied to D2 while subjects performed the reference motor task and the three test tasks. The order of testing for the different motor tasks was randomly determined for each subject, and all trials with a given motor task (~110 trials) were

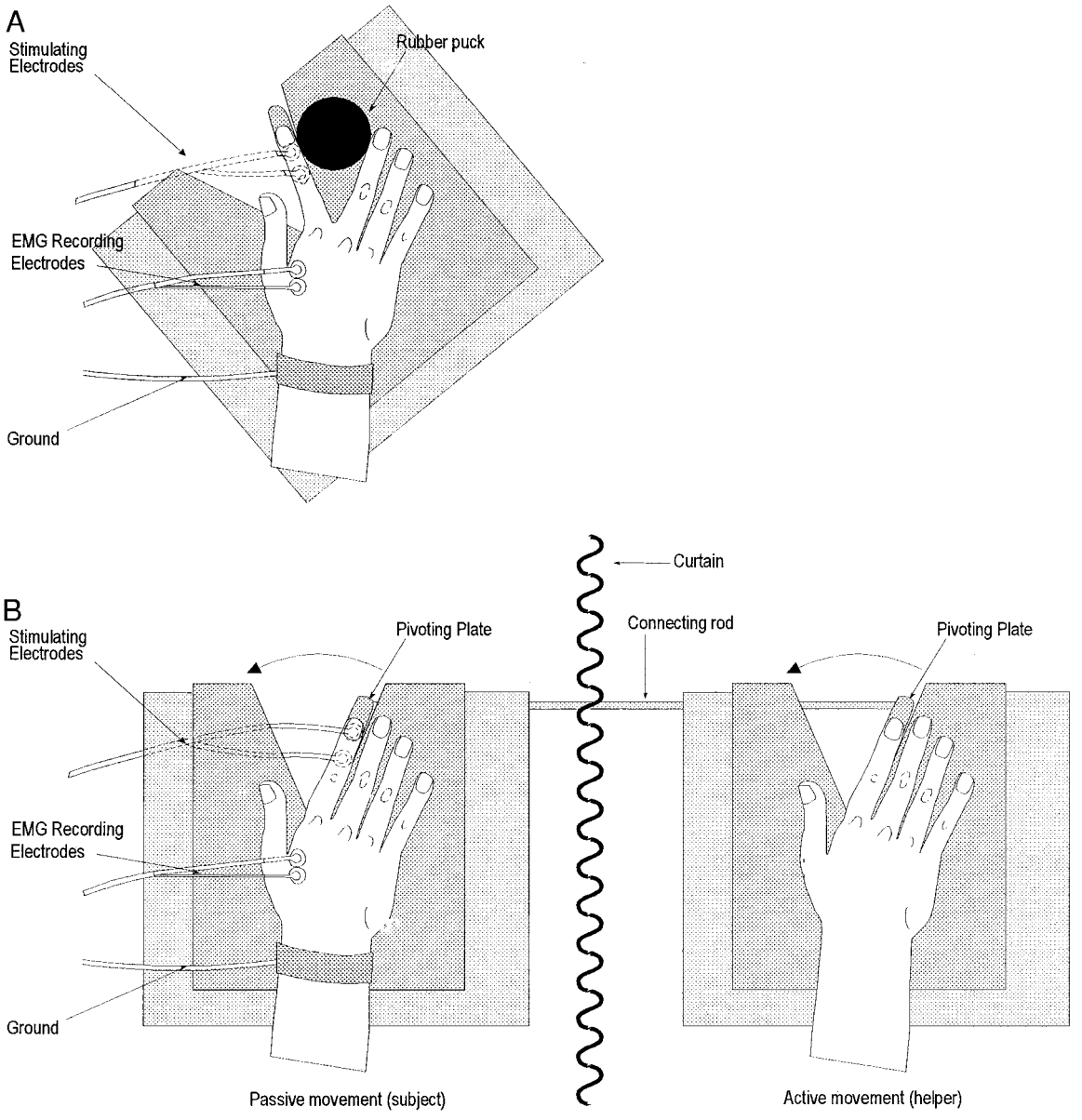

FIG. 1. *A*: experimental position for isometric digit 2 (D2) abduction. A rubber puck was placed between D2 and digit 3 (D3), placing D2 into a maximally abducted position. *B*: representation of the experimental set-up for passive D2 abduction. A helper's right D2 displaced the subject's D2, the force being transmitted through a connecting rod to the subject's position-detection apparatus and thence to the finger.

performed before another motor task was tested. Three trial types were presented: motor task + stimulation (70%), rest + stimulation (20%), and catch trials (no stimulation, 10%: motor task or rest trials). Each trial was preceded by a verbal instruction to move, or not, in response to the visual GO cue. Five different stimulus presentation delays were used, spanning a range of ~200 ms centered on the subjects' reaction time as estimated at the beginning of the session (see Williams and Chapman 2000).

Data acquisition and analysis

D2 position and EMG (full-wave rectified and integrated over 5 ms) activity of 1st DI were recorded during each trial (duration 2 s). Movement timing (movement onset, correlation between EMG onset and movement onset, movement duration) and kinematic (amplitude, peak velocity, peak acceleration) parameters were determined as permitted by the motor task, for each trial, as described in Williams et al. (1998). EMG onset was measured by hand on a trial-by-trial basis, using an interactive, custom-made program. Trials in which spontaneous EMG activity was seen in the 500-ms monitoring period that preceded the illumination of the GO cue (to the left of the vertical line shown in Fig. 2) were eliminated from the analysis. For the passive movement tasks, any trials with EMG activity at any time during the trial were eliminated from the analysis. Average timing and kinematic values were determined for each motor task, and two-tailed *t*-tests were used for comparisons ($P < 0.05$).

All comparisons between motor tasks were made using the same group of subjects for both the test task and reference task. Detection performance data were analyzed as in Williams and Chapman (2000). In brief, the overall proportion of stimuli detected for each of the three trial types (motor task + stimulation, rest + stimulation, catch trials) was first calculated for each motor task (data pooled across subjects). Absolute differences in detection performance between a test task (isometric contractions, passive movements, or movement without the

position detecting apparatus) and the reference task (active isotonic movement) were then evaluated using a Fisher one-tailed exact probability test for the 2 × 2 contingency table [task (reference, test task) × performance in each trial type (*n* stimuli detected, *n* stimuli presented); level of significance, $P < 0.01$]. This same test was also used in all subsequent proportion comparisons. Because all experiments showed a time-dependent decrease in detection performance, this comparison was potentially confounded by sampling differences (differences between tasks in the average timing of the stimuli relative to the motor response).

The time course of the decrease in detection in relation to the motor response (EMG or movement onset, depending on the comparison being performed) was evaluated in two ways. First, trials from all subjects were grouped into 20-ms bins as a function of the delay of the stimulus relative to the motor response, and the proportion of stimuli detected in each bin was calculated along with the 95% confidence interval. The resulting detection performance was plotted as a function of the delay of the stimulus relative to the motor response. Performance in each bin was compared with the performance in the rest + stimulation trials (Fisher exact test). Second, an average detection function (proportion of stimuli detected as a function of the timing of the stimulus relative to the motor response) was calculated for each motor task. For this, the motor task trials from a given subject and motor task were grouped into 40-ms bins relative to either EMG or movement onset, and the proportion of stimuli detected was calculated for each bin. These data were then fitted to a modified logistic function incorporating four parameters: maximum predicted performance, minimum predicted performance, peak slope (measure of the peak rate of decrease in detection performance), and the timing of the peak slope (the time at which performance decreased most rapidly). The average detection function for each motor task was then calculated by averaging the values of the four parameters describing the logistic functions fitted to the individual subject data. Two-tailed paired *t*-tests were used to compare the timing of peak decreases in detection performance. With seven subjects, a 20 ± 15-ms difference in detection performance, assuming an alpha of 0.05, could be detected with >0.8 probability (SISA on-line statistical analysis, Hilversum, The Netherlands).

RESULTS

A total of 36 experiments were analyzed (reference motor task, $n = 10$; test tasks, $n = 26$). Overall, subjects reported having detected a stimulus in 93.3% of rest + stimulation trials, 43.2% of motor task + stimulation trials, and 0.25% of catch trials. There was no significant difference between motor tasks for the proportion of catch trials in which a stimulus was detected (false alarms). Practice or fatigue did not significantly affect detection performance, as detection in the first 10% of rest + stimulation trials was never significantly different from detection in the last 10% of rest + stimulation trials delivered during an experiment. In all motor tasks, significantly fewer stimuli were detected during motor task trials than in rest trials ($P < 0.001$). The results of time course analyses of detection performance as well as comparisons between the reference and test tasks are given in the following text.

Contribution of movement-related reafference to tactile gating

The ability to detect near threshold stimuli applied to D2 was measured in seven subjects during isotonic D2 abduction (reference motor task) and an isometric abduction attempt with D2 already maximally abducted (test task 1). In all subjects, the latency for the onset of EMG activity correlated well with the latency for the onset of movement (both measured relative to the GO cue) for the isotonic task (mean: $r = 0.94$). Figure 2

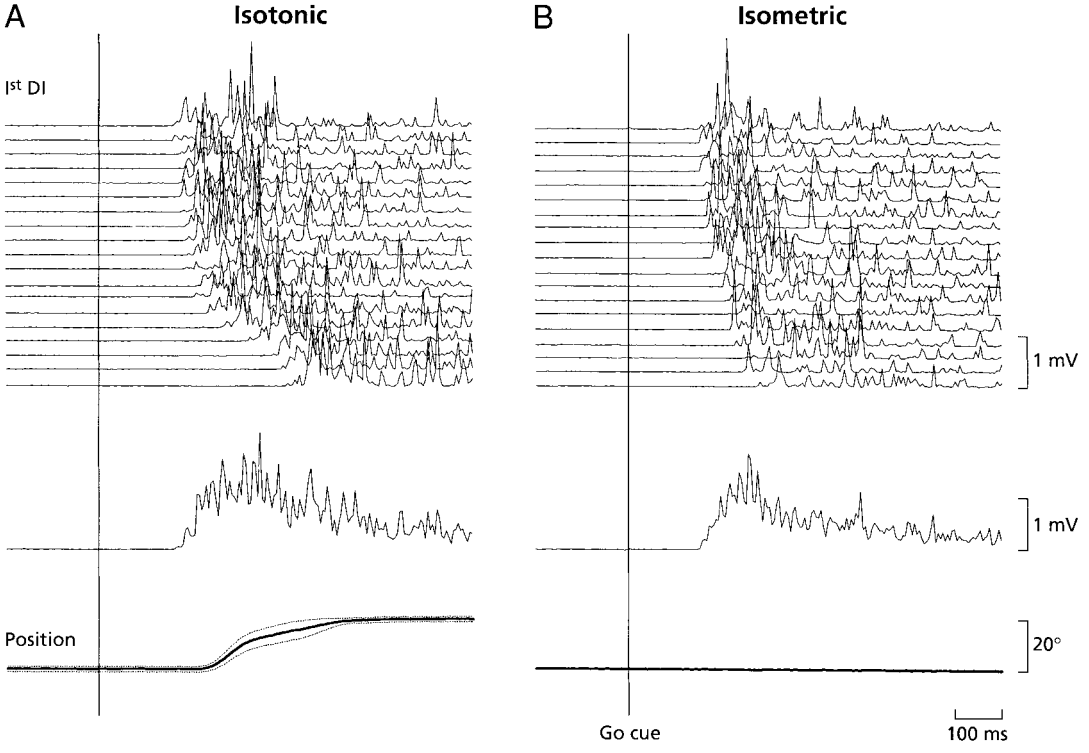

FIG. 2. *A* and *B*: sample electromyographic (EMG) traces for 1st dorsal interosseous muscle (1st DI) aligned on the onset of the GO cue during isotonic (*A*) and isometric (*B*) abduction of the index finger. *Top*: 19 individual trials during each task, rearranged in increasing order of the EMG onset as measured using an interactive program. *Middle*: the resulting average EMG traces. *Bottom*: the average position traces (along with the 95% confidence intervals).

TABLE 1. *Temporal parameters, kinematic parameters, and detection performance for the reference motor task (active isotonic D2 abduction) and test task 1 (active isometric D2 abduction) in seven subjects*

	Reference Task (Isotonic)	Test Task 1 (Isometric)	*P* Value
Temporal			
Movement onset, ms	239 ± 30	—	—
First DI EMG onset, ms	191 ± 25	194 ± 29	0.85
Movement duration, ms	213 ± 88	—	—
Kinematic			
Peak amplitude, °	32 ± 6.5	—	—
Peak velocity, °/s	375 ± 86	—	—
Peak acceleration, °/s^2	6900 ± 1300	—	—
Detection performance			
Rest	0.93 (0.89, 0.96)	0.94 (0.90, 0.97)	0.49
Motor task	0.43 (0.39, 0.48)	0.55 (0.50, 0.59)	<0.0001

In this and all other tables, *t*-tests were used for temporal and kinematic parameter comparisons, while Fisher exact tests were used for comparisons of detection performance. Continuous variables are given as means ± SD. For proportions, the upper and lower 95% confidence intervals are shown in brackets.

shows an example of the EMG records from one subject during the two motor tasks. In the isotonic task (Fig. 2*A*), 1st DI EMG activity consistently preceded movement onset by an average of 38 ms and showed a strong linear correlation with movement onset (not shown; $P < 0.0005$, $r = 0.99$). In the isometric task (Fig. 2*B*), the latency for EMG activity (measured relative to the GO cue onset) was not different from that during the isotonic contractions (respectively, 223 and 189 ms; $P = 0.10$). As detailed in Table 1, the average timing for all subjects of EMG onset relative to the GO cue was not significantly different between motor tasks. The proportion of stimuli detected at rest was also not significantly different. The overall proportion of stimuli detected during movement + stimulation trials was significantly higher in the isometric task (0.55) than in the isotonic task (0.43). This was probably explained by a difference in the timing of detection performance sampling: the average timing of stimuli relative to EMG onset was 30 ms earlier in the isometric task. When detection performance was plotted over time (Fig. 3, *A* and *B*), both tasks were found to produce similar reductions in detection performance. For both tasks, the timing of the peak decrease in detection performance was close to the time of EMG onset (see Table 3) and was not significantly different between tasks ($P = 0.30$). The estimated minimum proportion of stimuli detected after EMG onset approached 0 for both conditions and was also not significantly different ($P = 0.98$).

In summary, isometric D2 abduction attempts produced reductions in detection performance, the magnitude and timing of which were not significantly different from those produced by isotonic D2 abduction. These results show that central motor preparation and commands as well as peripheral afference related to the muscular contractions are sufficient to decrease tactile detection during movement and that peripheral input generated by limb displacement is not necessary for reductions in detection to occur.

Contribution of the motor command to tactile gating

To evaluate the importance of central motor preparation and commands on reductions in tactile detection, detection performance for stimuli delivered to D2 during passive D2 abduction (test task 2) was compared with the results obtained during active isotonic movements in nine subjects (reference motor task). Table 2 shows that the average time of movement onset as well as the average movement duration and amplitude were not significantly different for the active and passive movements. As could be expected from a consideration of the small maximum torque of 1st DI and the approximate doubling of the mass displaced during passive movements, peak velocity was significantly lower in passive movement, and peak acceleration

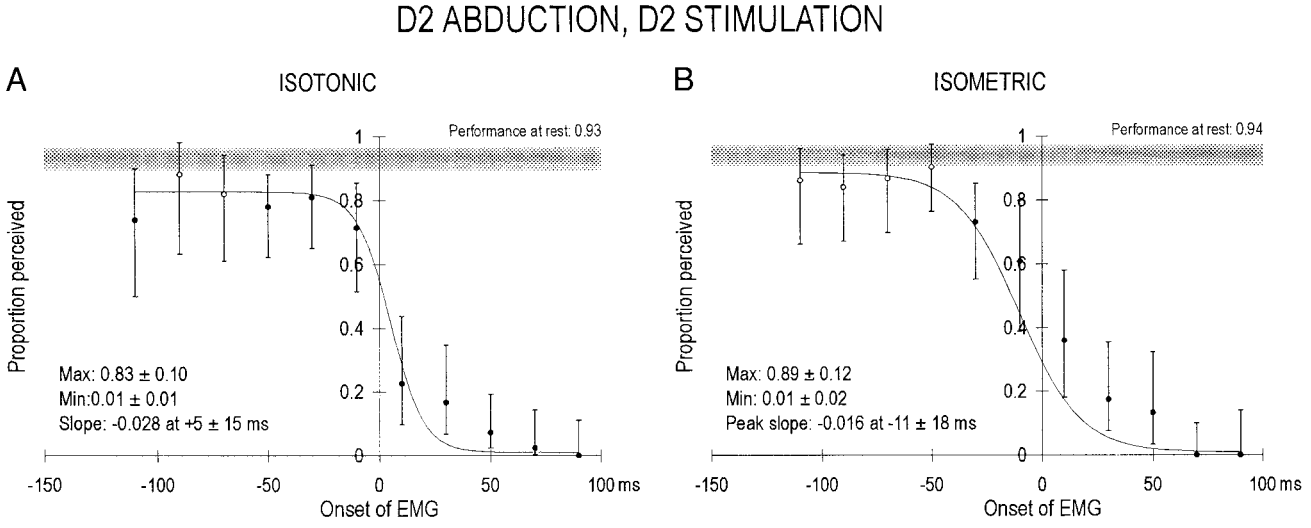

FIG. 3. *A* and *B*: comparison of the effects of active, isotonic D2 abduction and active, isometric D2 abduction on the detection of stimuli applied to the digit in 7 subjects. Detection performance over time is plotted relative to the onset of EMG (20-ms precision). Each point represents the overall proportion of stimuli detected within the bin (error bars, 95% confidence interval for performance across subjects). ▨, the 95% confidence interval for detection performance in the rest + stimulation trials. ●, detection performance during movement + stimulation trials was significantly lower than that observed at rest ($P < 0.01$); ○, no change. The curves represent the logistic functions defined by the averages of the logistic equation parameters fitted to *individual* subject data for each condition. Data using active, isotonic D2 abduction are a subset of data previously published in Williams et al. (1998, Fig. 5).

TABLE 2. *Temporal parameters, kinematic parameters, and detection performance for the reference motor task (active isotonic D2 abduction) and test tasks 2 (passive D2 abduction) and 3 ("no apparatus" active isotonic D2 abduction) in 9 subjects*

	Reference Task	Test Task 2 (Passive)	Reference vs. Test Task 2 *P* Value	Test Task 3 (No apparatus)	Reference vs. Test Task 3 *P* Value
Temporal					
Movement onset, ms	252 ± 45	235 ± 23	0.27	—	—
First DI EMG onset, ms	205 ± 50	—	—	175 ± 120	0.33
Movement duration, ms	170 ± 70	218 ± 50	0.14	—	—
Kinematic					
Peak amplitude, deg	29 ± 5	32 ± 4	0.12	—	—
Peak velocity, deg/s	365 ± 90	270 ± 50	0.02	—	—
Peak acceleration, deg/s^2	6200 ± 1650	3300 ± 750	0.002	—	—
Detection performance					
Rest	0.94 (0.90, 0.96)	0.94 (0.92, 0.96)	0.39	0.92 (0.89, 0.94)	0.20
Motor task	0.41 (0.37, 0.45)	0.42 (0.39, 0.45)	0.31	0.41 (0.38, 0.44)	0.50

was almost halved. Overall detection performance at rest and during movement trials was not significantly different for active and passive movement (Table 2). Analyses of the time course of observed reductions in the proportion of stimuli detected during movement + stimulation trials are shown in Fig. 4, *A* and *B*, in this case, plotted relative to movement onset as there was no EMG activity in the passive isotonic movement trials. The magnitude of the decreases in detection was virtually identical for active and passive movements (average minima of 0.01 and 0.03, respectively, $P = 0.25$). Surprisingly, the time course was also similar, with peak decreases preceding movement onset slightly but not significantly earlier in the active (−49 ms) versus the passive (−38 ms) conditions (see also Table 3). The results of these comparisons suggest that the peripheral afference generated by passive D2 abduction may produce reductions in detection not significantly different from those produced by active D2 abduction, i.e., that central motor preparation and commands may not be necessary to explain observed movement-related decreases in tactile detection.

Contribution of central set to tactile gating

The possibility that the early decrease in detection performance observed during passive movement trials could be due to expectation of movement triggered by the visual cue, or central set, was explored in a control experiment incorporating "sham-movement" trials. In two subjects, we repeated the passive test task but included sham- passive movement trials. The instruction to move was given verbally as usual to the helper, but 20% of the time (5 sham-movement trials per block) a second silent cue (a tap on the back) was also given to the helper unbeknownst to the subject. This silent cue indicated that in fact the helper should not perform D2 abduction at the GO cue despite having received the verbal instruction to move. In this way, we were able to determine if central set (expectation of a passive movement) modified subject detection performance. The results of this sham-movement experiment demonstrated that subjects detected 94% of stimuli delivered at rest, only 45% of stimuli delivered during movement trials, but 90% of stimuli delivered during sham movement, a result not significantly different from detection performance at rest (Table 4). Figure 5 shows the distribution of detected stimuli during the sham-movement trials and rest trials, relative to the helper's average reaction time. Inspection shows that there was no difference in performance during the sham trials as compared with performance at rest. Although there was a tendency for nondetected stimuli to be preferentially observed in the interval that preceded the expected onset of movement, this

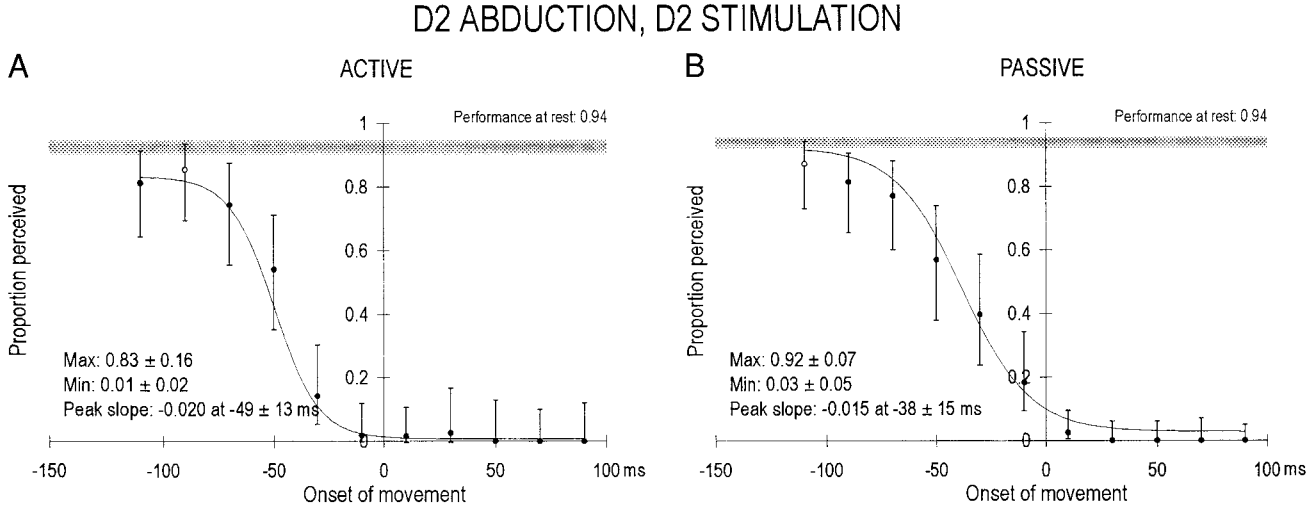

FIG. 4. *A* and *B*: comparison of the effects of active and passive D2 abduction on the detection of stimuli applied to the moving digit in 9 subjects. Detection performance over time is plotted relative to the onset of movement. Data using active, isotonic D2 abduction are a subset of data previously published in Williams et al. (1998, Fig. 5).

TABLE 3. *Comparison of average timing of peak decreases in detection performance*

Motor Task	*n*	Timing, ms	*P* Value
Reference	7	5 ± 15	0.30
Test task 1	7	−11 ± 18	
Reference	9	−49 ± 13	0.26
Test task 2	9	−38 ± 15	
Reference	9	−9 ± 18	0.67
Test task 3	9	−15 ± 33	

Mean time of the peak decrease in detection (±SD) measured relative to the onset of electromyographic (EMG) activity [reference task vs test tasks 1 (isotonic vs. isometric) and 3 (apparatus vs. no apparatus)] or movement onset [reference task vs. test task 2 (active vs. passive)]. Negative values indicate that the peak decrease preceded the event on which the data were aligned; positive values indicate that the decrease followed the event.

was true for *both* trial types (recorded in the same block of trials). No evidence for a transient decrease in tactile detection was seen when (passive) movement was expected—i.e., the results provided no evidence that expectation of movement contributed to the similarity of the results obtained with active and passive movements (Fig. 4).

Contribution of peripheral reafference generated by contact with the position-detection apparatus to tactile gating

In the interest of determining the effect of extraneous cutaneous feedback generated by the digit resting on the position-detection apparatus on detection performance, nine subjects performed active isotonic D2 abductions with D2 resting unsupported by the subjects' side (test task 3; same subjects as for test task 2). Detection performance was compared with results obtained with active D2 abduction using the position-detection apparatus (reference motor task). Although most movement parameters were not available for the test task, the timing of EMG onset relative to the GO cue was not significantly different between the two tasks (Table 2). Overall detection performance in both the rest and the movement trials was not significantly different between conditions. Both *A* (with apparatus) and *B* (without apparatus) in Fig. 6 show similar reductions in detection performance. Nonsignificant differences were observed for both the timing (Table 3) and magnitude of the decreases (to 0.01 and 0.02, respectively, $P = 0.22$). The results of this comparison indicate that the extraneous cutaneous feedback generated by the position-detection apparatus can be eliminated without significantly affecting either the timing or magnitude of the reduction in tactile detection during movement.

TABLE 4. *Proportion of stimuli perceived during passive movement (test task 2), rest trials and sham passive movement trials (2 subjects)*

	Movement	Rest	Sham Movement	Fisher Exact Test (Rest vs. Sham)
Subject 1	38/75	23/25	22/25	0.33
Subject 2	30/75	24/25	23/25	0.38
Total	68/150	47/50	45/50	0.22

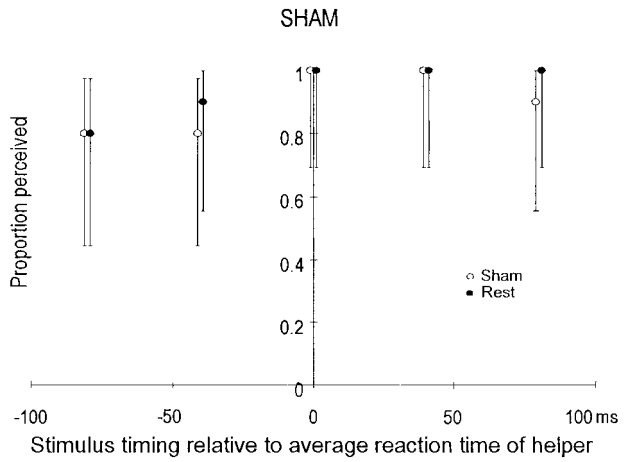

FIG. 5. Comparison of detection performance at rest and during sham (passive) movement trials in 2 subjects. Ten trials of each type were delivered at each of 5 stimulus intervals distributed around the mean reaction time of the helper performing the abduction movements that were imposed on the subject's D2. No significant differences in detection performance were observed when performance in sham movement trials was compared with detection performance in rest trials.

DISCUSSION

The main finding of this study was that time-dependent decreases in the detection of near threshold stimuli show a remarkably similar time course across a variety of motor tasks, including active/passive movement and isotonic/isometric contractions. Moreover, these decreases in detection were not due to some form of central set when movement is expected, as sham-movement detection performance was not significantly different from detection performance at rest.

Methodological considerations

Increases in movement amplitude, peak velocity, and peak acceleration are all associated with decreases in the proportion of stimuli detected (Angel and Malenka 1982; Chapman et al. 1996; Williams et al. 1998). Thus significant differences in movement kinematics could potentially affect comparisons between motor tasks. For all but one comparison, however, there were no significant differences in kinematic parameters. In the one exception (active vs. passive D2 abduction), passive D2 abduction was performed with significantly lower peak acceleration and velocity. The experimental setup in which two test apparatuses were yoked, approximately doubling the mass to be displaced in the passive task by the helper's abduction movement, explained this difference. In both tasks, detection performance during movement fell to almost zero, and reductions in detection were timed similarly in both the active and passive conditions. We suggest that kinematic differences did not contribute substantially to the results.

The choice of stimulus intensity and the magnitude of the reduction in detection may have obscured subtle differences in detection performance in the different experimental conditions. As shown in Williams and Chapman (2000), however, stimulus intensity does not affect the timing of peak reductions in detection for active isotonic D2 abduction. These supplementary findings give an indication that the absence of difference across the various comparisons was most likely a robust observation.

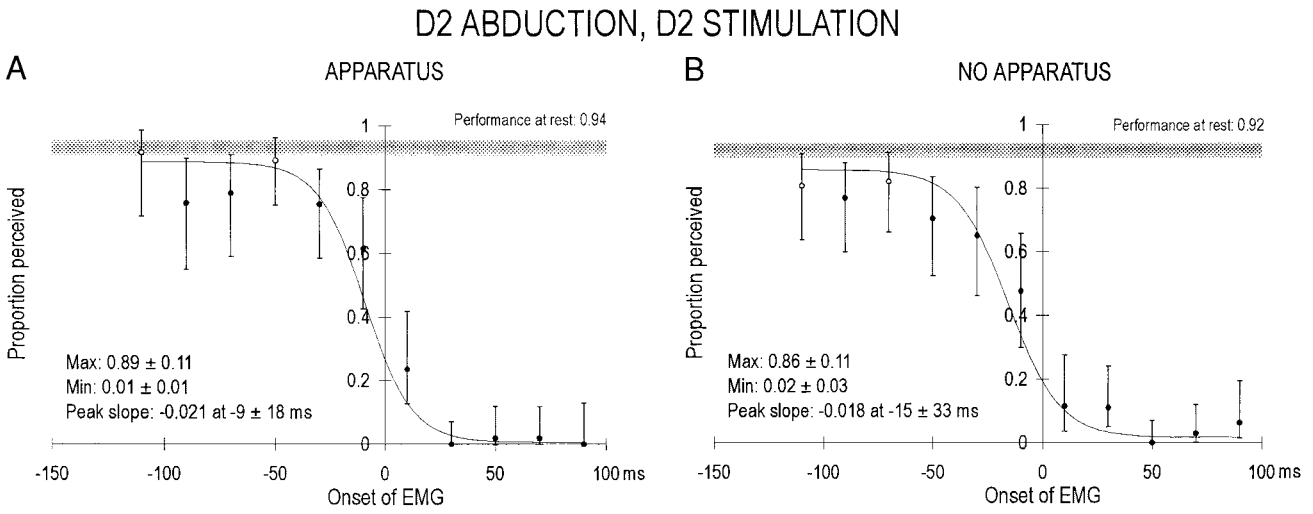

FIG. 6. *A* and *B*: comparison of the effects of active D2 abduction with (data from Fig. 4*A*) and without the position-detection apparatus on the detection of stimuli applied to the moving digit in nine subjects. Detection performance over time is plotted relative to EMG onset, as in Fig. 3.

Non-time-dependent decreases in tactile detection

We previously reported a modest (10%) and sustained decrease in tactile detection in all movement + stimulation trials, including the earliest delays tested. The decrease was only revealed when the stimulus to be detected was applied to sites distant to the D2 movement (contralateral shoulder or D2, ipsilateral leg) (Williams et al. 1998) where the strong time-dependent decrease related to the movement was absent. We attributed the non-time-dependent decrease to attention because subjects had to split their attention between two tasks, the tactile detection task (same as used here) and the motor task, corresponding to the reference motor task in the present study. These results were subsequently extended by Williams and Chapman (2000) to include magnitude estimates of suprathreshold tactile stimuli. We found a very early ($\leq$120 ms before EMG onset) slight but significant decrease in the perceived intensity of relatively weak suprathreshold tactile stimuli ($2 \times P_{90}$), but not stronger suprathreshold stimuli ($3 \times P_{90}$). Superimposed on this was a large time-dependent decrease in perceived intensity linked to movement, similar to that reported here. This same attentional influence is reflected in the current database. Inspection of Figs. 3, 4, and 6 shows that the maximum predicted performance calculated from the logistic functions applied to the data collected during the motor tasks was generally lower than the performance at rest, likely reflecting a modest decline due to divided attention. In addition, performance in the earliest bins tested, at delays well before the steep decline in detection related to movement, was occasionally significantly less than the performance at rest (●). Thus while attentional influences undoubtedly contributed to the reduced tactile detection during the four motor tasks tested here, the effect was small and presumed to be constant across all delays tested. The time-dependent decreases in tactile detection, on the other hand, can best be explained by dynamic signals directly related to the motor task.

Sources and mechanisms of the time-dependent decrease in tactile detection

The signals potentially controlling reductions in the detection of tactile stimuli during movement originate both centrally (motor preparation and command) and peripherally (movement-related reafference). They may affect detection performance either by reducing the transmission of the test stimulus as it courses through the various relays of the somatosensory system on its way to cortex or by influencing cortical processing of the signal.

Many investigators have postulated that the dorsal column-medial lemniscal system is subject to descending controls during voluntary motor activity (e.g., Chapman et al. 1988; Cohen and Starr 1987; Coulter 1974; Ghez and Lenzi 1971; Jiang et al. 1990a). Potential anatomical pathways for these controls include intracortical projections from motor cortex to sensory cortex (Jones et al. 1978), back projections from somatomotor cortical regions to the sensory thalamus, either directly or via the thalamic reticular nucleus (Jones and Wise 1977), and somatomotor cortical projections to the DCN and dorsomedial spinal gray matter (Bentivoglio and Rustioni 1986; Cheema et al. 1985; Dum and Strick 1996; Jones and Wise 1977; Kuypers 1958, 1960; Martinez et al. 1995; Walberg 1957). Direct stimulation of motor cortex has complex excitatory and inhibitory effects on neurons at the level of the DCN (Giuffrida et al. 1985; Harris et al. 1965; Jabbur and Towe 1961; Towe and Jabbur 1961). In contrast, uniformly inhibitory actions were observed when SI cortical evoked responses to peripheral stimulation were conditioned by weak, intracortical microstimulation of motor cortex (Jiang et al. 1990a). The timing of reductions in somatosensory system responsiveness observed during both evoked potential and single-unit studies further supports the central control hypothesis. Indeed, during active isotonic or isometric contractions, reductions can precede the onset of peripheral motor activity at the level of the medial lemniscus (Coulter 1974; Ghez and Lenzi 1971), the thalamus (Chapman et al. 1988; Shin and Chapin 1990), and the somatosensory cortex (Cohen and Starr 1987; Coquery et al. 1972; Jiang et al. 1990b, 1991; Rushton et al. 1981). During passive movement, reductions in SEPs occur only after the onset of movement and only at the thalamic relay and above (Chapman et al. 1988). Thus central signals appear crucial to the reductions in responsiveness that precede the onset of movement at all levels and may be entirely responsible for the modulation seen at the level of the DCN. In contrast,

movement-related peripheral reafference can only play a role in reducing the responsiveness of the somatosensory system after the onset of feedback from the moving limb.

If observed reductions in somatosensory system responsiveness during motor tasks were to explain concomitant reductions in tactile detection, then we would have expected peak decreases in detection to occur at the onset of EMG activity during active D2 abduction and that the timing of the peak decrease would shift to *after* the onset of movement in the passive condition. Instead, we found that the peak decrease in detection preceded the onset of passive movement by an average of 38 ms. This observation has been confirmed using another motor task, active elbow extension versus imposed passive elbow extension (3 subjects). In the latter case, the peak decrease in detection occurred almost 100 ms before movement onset, be it active or passive (unpublished observations). These results suggest that central motor commands are not necessary for reductions in detection before the onset of movement. Further, there likely is not a one-to-one link between changes in the amplitude of short latency SEPs in sensory thalamus and SI cortex and changes in detection: thalamic and cortical SEPs show no change prior to passive movement, but our results indicate that tactile detection is decreased prior to the onset of passive movement.

How then to explain the decrease in detection *before* the onset of passive movement? We suggest that the input signal, generated by near-threshold electrical stimulation, was attenuated through an inhibitory action generated by the peripheral feedback from the moving digit (backward masking, see following text). It seems unlikely that central motor commands contributed, because the subjects remained relaxed during the imposed passive displacements (verified with surface EMG recordings from the 1st DI). On the other hand, other central processes (possibly including a change in response criterion) (see Williams and Chapman 2000) might have been triggered by the visual GO cue, which served not only to inform the subject to prepare to detect the tactile stimulus but also to expect a passive movement, depending on the preceding verbal instructions heard by both the subject and the helper. We addressed this possibility by repeating the passive testing and including sham-movement trials, in which case the subject expected, but did not receive, a passive movement. Thus we could evaluate whether expectation of a movement alone (central set) might have contributed to the modulation of detection. It is worth noting that subjects seemed to genuinely expect movement in these sham trials, one of the two subjects going so far as to occasionally castigate the helper for not "paying attention to her job" when the expected movement did not occur. The results of the sham-movement trials showed that the GO cue itself did not trigger any obvious time-dependent decrease in tactile detection: performance was identical to that observed in the rest + stimulation trials acquired at the same time (Fig. 5). Although this observation makes it unlikely that central influences triggered by the GO cue could explain the results obtained in the passive test task, a definitive test of this suggestion would require the use of a bias-free experimental design (Green and Swets 1988). Future experiments will address this possibility.

It seems more likely that the reductions in detection observed prior to and during passive movement are generated by movement-related peripheral reafference. To reconcile the results with observations of no change in the earliest component of the cortical SEP (recorded in areas 3b and 1) before the onset of passive movement (Chapman et al. 1988), it is suggested that gating influenced the response to the test stimulus at some point *after* the stimulus reached areas 3b and 1 but *before* conscious detection of the stimulus was established. This temporal sequence of events has previously been proposed in studies examining "masking" in the somatosensory system, whereby the detection of a weak test stimulus is prevented by near-simultaneous administration of a stronger masking stimulus (Gescheider et al. 1989; Melzack et al. 1963; Schmid 1961). Reductions in the detection of test stimuli that precede the masking stimuli (backward masking) have been reported (Gilson 1969; Laskin and Spencer 1979a; Scherrick 1964; Schmid 1961) and could account for reductions in detection performance occurring before the onset of passive (or active) movement, with movement-related reafference acting as the masking stimulus. Laskin and Spencer (1979b) studied backward masking at the cortical level by examining the modulation of short-latency neural responses to test stimuli by masking stimuli. Backward masking was only observed when neuronal responses in a given cell to test and masking stimuli overlapped in time, and the effects were restricted to the overlapping portion of the response to the test stimuli. The time course of the backward masking effects (~10 ms) was much shorter than seen here (up to ~40 ms prior to movement onset). More recently, Brosch et al. (1998) reported very early backward masking in monkey auditory cortex, in this case of longer-latency responses to auditory stimuli (≤140–180 ms after the onset of the test stimulus). Further to this, there are suggestions in the literature to the effect that conscious detection of tactile stimuli is related more to the amplitude of the longer-latency components of the cortical SEP (Gomes 1998; Kulics et al. 1977; Libet et al. 1964) than to the amplitude of the shorter-latency responses examined in studies of movement-related gating. Our psychophysical results could thus be a reflection of masking of longer-latency responses to the test stimulus. This leads to the prediction that even when the primary component of the SEP occurs before the onset of passive movement, longer-latency components of the neural responses to tactile stimuli should show evidence of gating when they occur after the onset of passive movement. This sequence of events would explain the timing of passive movement-related gating of tactile detection demonstrated in this study. Although the underlying mechanisms of this interaction remain unknown, two hypotheses have been advanced. Scheerer (1973) proposed that the masking stimulus interacts with late responses to the test stimulus to produce a composite representation of both stimuli that does not allow the detection of the test stimulus as a separate event (integration hypothesis). Alternately, Schultz and Eriksen (1977) suggested that the arrival of the masking stimulus interrupts the neuronal processing of the test stimulus before consciousness of the stimulus is achieved (interruption hypothesis).

Forward masking could also contribute to reductions in detection that occur after the onset of active and passive movement. Both reductions in detection during active D2 abduction (Williams et al. 1998) and masking (Laskin and Spencer 1979a; Scherrick 1964) show a spatial gradient, with maximal effects occurring closest to the body part in motion or the origin of the masking stimulus. However, the temporal shift

in the timing of peak decreases in detection as distance increases found in Williams et al. (1998) is not apparent in masking studies. Instead, maximum decreases in detection performance are seen at the onset of the masking stimulus, regardless of distance (Scherrick 1964). This difference may be an indication that reductions in detection performance during active movement are not "simply" the result of masking phenomena but that other mechanisms are also involved. It would be interesting to examine whether the temporal shift in the timing of the peak decrease with an increase in distance that was observed with active movement (Williams et al. 1998) is still observed with a passive movement task or alternatively if the timing of the decrease remains relatively constant. Preliminary and unpublished data from this laboratory using shoulder stimulation indicates that the shifts in timing may be preserved.

Do certain sources of peripheral reafference play an essential role in the reduction of tactile detection during movement? Comparisons between isotonic and isometric motor tasks showed no difference in the time course and magnitude of reductions in detection during movement, consistent with a previous study of reductions in SEPs by isotonic and isometric motor tasks that also found no difference (Jiang et al. 1990b). These results indicate that certain types of movement-related afference are not essential for reductions in detection to occur. For example, feedback related to changes in position from muscle spindles, cutaneous mechanoreceptors, and Golgi tendon organs was undoubtedly reduced in the isometric condition. An important advantage inherent to the design of our isometric task was the elimination of added cutaneous discharge generated when a body part exerts force against an immovable object, an important confounding study in most previous studies using isometric contractions. In this study, force was exerted away from the immobile object; no movement could occur because the agonist was already maximally shortened. It can therefore be concluded from the isometric results that movement-related cutaneous afference and afference related to antagonist stretch are not necessary for reductions in detection to occur. This finding is compatible with the weak and irregularly observed effects of digit anesthesia on movement-related reductions in tactile detection (Schmidt et al. 1990b). Furthermore, agonist tendon organ and spindle discharge generated during isometric contractions is qualitatively and quantitatively different from discharges arising during isotonic movement (Edin and Vallbo 1990), but these changes do not appear to influence detection performance in any way. Two possibilities can explain the isometric results. The first is that the agonist-related afference during an isometric motor task is adequate in its nature and sufficient in its quantity to generate observed reductions in detection through processes similar to those explaining the passive movement task results. The second is that centrally originating processes relating to movement preparation and the motor command do in fact reduce the detection of tactile stimuli when they are present with a time course indistinguishable from that of reductions in detection produced by movement-related reafference in the passive movement task. These two mechanisms may or may not coexist. One way to distinguish between these two ideas would be to examine the effects of mental movement imagery on detection of tactile stimuli (see Nelson 1996 for a review), using an experimental paradigm similar to the one used in this study. However, a trial-by-trial objective measure of "imagery onset" would be necessary to analyze the time course of these effects.

In conclusion, reductions in tactile detection during movement are surprisingly resistant to elimination of potential sources of gating signals. In this study, the movement-related decrease in detection performance was not modified by eliminating much of the peripheral reafference related to movement (isometric results) or by eliminating the central processes involved in the generation of movement (passive results). This raises the interesting possibility that several central and peripheral signals may be sufficient in themselves to generate similar reductions in detection, i.e., that redundancy exists in the control of movement-related reductions of tactile detection.

Localization of the putative sites of interactions

While recognizing that movement-related controls over sensory perception involve all levels of the pathway from the DCN up to cortex (above), the results obtained from the passive testing raise the question as to the potential localization of these actions. Specifically, where might proprioceptive input generated by passive movement modulate responsiveness to a cutaneous stimulus? For this to occur, the population of cells activated by the cutaneous stimulus would have to receive inhibitory modulation from movement-related reafference. The latter is likely to be mainly from deep proprioceptors, although cutaneous mechanoreceptive afferents (particularly those supplying the hand) are also sensitive to joint rotation (Edin and Abbs 1991).

It is known that cutaneous and proprioceptive inputs are transmitted along separate pathways from the periphery up to SI cortex. Even within the four cytoarchitectonic regions that comprise SI cortex, these two modalities are largely channelled into different subregions, area 3a for proprioceptive inputs and areas 3b and 1 for cutaneous inputs. It is only in area 2 that substantial proportions of cutaneous and proprioceptive neurons coexist, at least in the primate hand representation (Ageranioti-Bélanger and Chapman 1992; Chapman and Ageranioti-Bélanger 1991; Hyvärinen and Poranen 1978; Iwamura et al. 1993). But even in area 2 the proportion of cells receiving *convergent*, bimodal input is very low so that the opportunity for interactions would appear to be limited. It is not until area 5 in the posterior parietal cortex, which receives much of its input from area 2, that one may encounter substantial proportions of bimodal cells (Sakata et al. 1973; cf. Duffy and Burchfiel 1971; Seal et al. 1982). This raises the interesting possibility that area 5, which is also reciprocally connected with motor cortex (Jones et al. 1978; Strick and Kim 1978), may be the site at which the suppressive effects of movement-related reafference are exerted. Such a suggestion would be consistent with the possibility that backward masking of longer-latency activity underlies the premovement decrease in tactile detection.

On the other hand, it is not possible to rule out the possibility that the suppression occurs within SI cortex itself. Zarzecki and colleagues (Kang et al. 1985; Zarzecki and Wiggin 1982) have shown that at a subliminal level, convergent input from cutaneous and proprioceptive afferents onto individual SI cortical neurons is more widespread than generally believed. Moreover, such inputs may become liminal in conjunction with other factors such as behavioral context (Iwamura et al. 1985;

Tremblay et al. 1996). In addition, even in areas 3b and 1, neuronal responses to discrete cutaneous stimuli can outlast the actual stimulus by several hundreds of milliseconds (Gardner et al. 1984), and these late responses are themselves subject to movement-related suppression (Jiang et al. 1991). Clearly further experiments are needed to localize the site(s) at which movement-related reafference gates tactile detection prior to movement onset.

Functional considerations

The existence of reductions in detection performance during movement cannot be disputed. The functional role for these reductions has not been defined. The existence of pathways originating in sensorimotor cortex and modulating somatosensory relay gain naturally raises the hypothesis that there is an advantage to controlling the flow of afferent information during movement. These pathways could produce gains in processing efficiency by reducing the inflow of afferent information that is either redundant or uninterpretable when buried in movement-related reafference. It is also possible that movement-related reductions in detection performance reflect the limits of somatosensory system performance when noise levels increase and do not serve a functional role. The fact that decreases in detection during passive movement are remarkably similar to those seen during active movement may be a reflection of this reality. On the other hand, the remarkable similarity between the time course of reductions in transmission and detection for active movement and detection in passive movement may not be coincidental. It is possible that the masking effects of movement-related afference are designed by nature to begin influencing the processing of tactile information simultaneously with the onset of centrally mediated reductions in transmission in active movements and that both effects begin at the time that the first peripheral movement-related reafference is expected, that is, before movement onset at the *expected* time of agonist EMG onset (Fig. 4). This combination of centrally and peripherally mediated effects would explain our experimental results as well as why reductions in detection usually coincide with EMG onset for active movement and the time when EMG would have been "expected" in passive movement.

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