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TIME COURSE AND MAGNITUDE OF MOVEMENT-RELATED GATING OF TACTILE DETECTION IN HUMANS.

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A été évaluée par un jury composé des personnes suivantes:

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SUMMARY

Psychophysical studies have demonstrated that the detection of tactile stimuli is reduced during movement ("gating" of perception). Several mechanisms could explain these reductions. For example, physiological investigations have demonstrated centrally controlled movement-related gating of transmission through the dorsal-column/medial-lemniscal system. Alternatively, it has been documented using physiological and psychophysical methods that tactile afference itself can inhibit the detection of tactile stimuli (masking) by affecting transmission, but also perhaps by reducing the signal to noise ratio in the somatosensory system or by interrupting tactile information processing after transmission is complete. The three series of experiments which form the body of this thesis were conceived to explore the mechanisms underlying movement-related gating of tactile perception.

In order to permit comparisons with reductions in transmission and psychophysical masking results, the first two studies quantified the effect of stimulus timing, localisation and intensity on tactile detection of weak electrical stimuli in human subjects during abduction of the index finger.

The results of the first study demonstrated significant time-dependent reductions in tactile detection over the entire surface of the upper limb ipsilateral to the moving D2. The earliest reductions were seen at the most distal stimulation sites on this limb, about 120 ms before movement onset or 70 ms before EMG onset, and by the time of EMG onset almost no stimuli were detected. Gating of detection exhibited a spatio-temporal gradient, with more proximal sites showing later and smaller reductions in the proportion of stimuli detected. Sites on the

contralateral limb and ipsilateral leg showed no time-dependent reductions in detection.

In the second study, the detection of stimuli of five different intensities during movement was evaluated. At the weakest intensity (which corresponded to the intensity used in the stimulus localisation experiments), approximately 90% of stimuli were detected (P_{90}). Stimuli of 1.25, 1.5, 1.75 and 2 X P_{90} intensity were also evaluated. At all intensities tested, time-dependent reductions in tactile detection were observed. As the stimulus intensity was increased, the reductions in detection were smaller, but the timing of the reductions was invariant across different stimulus intensities, with peak decreases occurring within ± 12 ms of EMG onset (25-45 ms before movement onset).

Once the weakest intensity at which most stimuli were detected during movement had been determined (2 x P_{90}), magnitude estimation experiments were undertaken to evaluate the effect of movement on the scaling of stimuli which remain suprathreshold during movement. Stimuli of intensity 2 x P_{90} and 3 x P_{90}, delivered to D2, were used. Significant reductions in the subjective intensity of suprathreshold stimuli were observed at both stimulation intensities, the magnitude of the reduction being relatively greater at the lower stimulation intensity. The timing of the reduction was similar to that observed for detection experiments (± 20 ms of EMG onset).

The effects of stimulus timing, location, and intensity on tactile detection during movement were successfully modelled using modified logistic functions. The model not only reproduced the experimental results but also provided predictions of detection performance at stimulus parameter combinations that have not yet been experimentally tested. Although reductions in tactile detection during movement probably represent the overall effect of several different

physiological mechanisms controlling the transmission and processing of tactile afference, the model serves to clearly define the resulting perceptual modifications that proposed mechanisms need to explain.

The third study investigated the relative importance of centrally and peripherally originating signals on movement-related reductions in tactile detection. Reductions in detection were compared during active and passive D2 abduction. No significant differences were seen, demonstrating that central signals were not necessary to explain reductions in detection during movement, even those that occur *before* movement onset. These results were confirmed by comparing detection performance during active and passive elbow extension. Reductions in detection during isotonic and isometric D2 abduction were then compared, with stimuli being delivered at two different sites, D2 and the ipsilateral shoulder. At each site, no significant differences were observed between motor tasks, indicating that movement itself was not necessary to explain observed reductions in tactile detection.

In conclusion, it seems probable that the term "gating" encompasses several different processes modulating tactile afference during movement. These modulatory processes exert inhibitory effects at all levels of the somatosensory system, as well as during subsequent cortical processing, and appear to converge functionally to produce similar gating during isotonic, isometric and passive motor tasks.

RÉSUMÉ

La détection des stimuli tactiles est réduite pendant le mouvement («gating » de la perception). Plusieurs mécanismes sont invoqués pour expliquer ces réductions. Il est démontré par exemple que la transmission de l'information tactile est inhibée pendant le mouvement (gating de la transmission), et que cette inhibition est sous contrôle central et périphérique. Par contre, le mouvement génère une grande quantité d'afférence tactile, et il est aussi démontré que tout stimulus tactile accessoire peut empêcher la détection d'un stimulus tactile (masking), présumément non seulement en réduisant la transmission des afférences, mais aussi par simple augmentation du bruit dans le système somatosensoriel, ou alternativement par des mécanismes d'interruption du traitement du stimulus tactile à détecter aux niveaux sous-cortical et/ou cortical. La question se pose donc: est-ce que les réductions dans la détection des stimuli tactiles pendant le mouvement sont le résultat d'une augmentation du bruit de fond dans le système, de réductions dans la transmission de l'information dans le système somatosensoriel, de modifications dans le traitement subséquent du stimulus tactile après son arrivée au cortex, ou d'une combinaison des trois?

Les trois séries d'expériences qui forment le corps de cette thèse furent conçus pour explorer cette question. Dans les deux premières séries, l'effet d'une modification systématique du temps, de la localisation et de l'intensité de stimulation sur les réductions de la détection tactile pendant le mouvement furent quantifiés, dans le but d'obtenir une caractérisation psychophysique adéquatement comparable aux résultats des études de masking et de gating de la transmission

tactile pendant le mouvement. La troisième série d'expériences tenta d'évaluer l'importance relative de signaux d'origine centrale et périphérique dans la genèse des réductions de la détection tactile lors du le mouvement.

Dans la première étude, le décours temporel ainsi que l'étendue spatiale des réductions de la détection de stimuli électriques faibles (d'une intensité ou environ 90% étaient perçus au repos) fut déterminée dans 118 expériences impliquant 47 sujets humains entrainés à produire des abductions rapides de l'index (D2) suite à la présentation d'un signal visuel. Des stimuli électriques furent livrés à dix endroits différents du corps, incluant des sites sur le membre responsable de la tâche motrice (D2, l'auriculaire, la main, l'avant-bras, le bras, l'épaule, et la partie la plus proximale de la ceinture pectorale) ainsi que des sites plus distants (bras controlatéral, jambe ipsilatérale). La détection de stimuli appliqués au doigt déplacé fut diminuée de façon significative. De plus, cette diminution était très dépendante du temps de stimulation: plus la stimulation était tardive, moins le stimuli était percevable. Les premières diminutions significatives étaient environ 120 ms avant le début du mouvement, et 70 ms avant le début de l'activité EMG. Des réductions reliées au mouvement et variant avec le temps de stimulation furent observées à tous les sites situés sur le membre supérieur ipsilatéral au mouvement. Un gradient spatio-temporel important fut observé, selon lequel les réductions dans la détection des stimuli étaient plus précoçes et d'ampleur plus importante aux sites les plus près du doigt en mouvement, et progressivement plus tardives et plus petites aux sites plus distants. Quand les stimuli furent appliqués aux sites sur le membre supérieur

controlatéral ou la jambe ipsilatérale, on n'observait plus qu'une faible diminution de la proportion de stimuli détectés, d'environ 10%. Cette diminution ne dépendait pas du temps de stimulation, mais plutôt était constante pendant la période de temps étudiée. Pour décrire quantitativement l'influence de la localisation du stimulus dans l'espace et le temps sur la réduction de détection due au mouvement, un modèle mathématique basé sur des fonctions logistiques modifiées fut élaboré. Ce modèle reproduit fidèlement les résultats expérimentaux.

Dans la deuxième étude, l'influence de l'intensité du stimulus sur le décours temporel et l'ampleur des réductions de la perception tactile pendant le mouvement fut explorée dans 17 sujets humains. Les sujets furent entrainés à produire des abductions rapides de D2 suite à la présentation d'un signal visuel (c'est à dire la même tâche motrice que dans la première étude, et ce pour faciliter une mise en commun des données lors de l'étape de modélisation). Des stimuli électriques de cinq intensités différentes furent appliqués à D2. L'intensité la plus faible correspondait à une intensité ou environ 90% des stimuli étaient perçus au repos (P_{90}), tandis que les quatre autres intensités correspondaient respectivement à 1.25, 1.5, 1.75 et 2 fois l'intensité de base. A toutes les intensités testées, la proportion de stimuli perçus fut diminuée significativement, les stimuli plus intenses étant proportionellement moins affectés que les stimuli plus faibles. Ces réductions dans la proportion de stimuli perçus étaient dépendantes du temps de stimulation. Les réductions les plus rapides dans la proportion de stimuli détectés étaitent toujours à ± 12 ms du début de l'activité EMG (25-45 ms avant le début

du mouvement); les changements de l'intensité de stimulation n'affectaient donc pas le décours temporel des réductions. Une fois que l'intensité de stimulation la plus faible à laquelle les sujets percevaient la quasi-totalité des stimuli même après le début de l'activité EMG fut connue (2 x P_{90}), des expériences d'évaluation de l'intensité subjective des stimuli furent entreprises en utilisant des stimuli de deux intensités différentes, soit 2 x P_{90} et 3 x P_{90}. Des réductions significatives dans l'intensité perçue des stimuli furent observées aux deux intensités de stimulation, avec un décours temporel qui était semblable à celui des diminutions de la détection des stimuli pour des intensités de stimulation plus faibles. L'ampleur des réductions dans l'intensité perçue variait inversement avec l'intensité de stimulation.

Un modèle mathématique qui décrivait l'effet de l'intensité et du temps de stimulation sur la détection tactile pendant le mouvement fut ensuite créé. Ce modèle reproduisait fidèlement les réductions observées dans la deuxième étude. Ce modèle fut ensuite combiné avec le modèle de l'influence de la localisation et du temps de stimulation sur la détection tactile créé précédemment. Le modèle combiné reflète bien les résultats expérimentaux obtenus, et prédit la performance de détection pour toute combinaison de site, intensité, et temps de stimulation. La réduction de la détection tactile représente probablement la somme de plusieurs mécanismes physiologiques de contrôle de la transmission et du processing de l'information tactile. Le modèle combiné définit clairement le résultat perceptuel que des modèles physiologiques de la réduction de la détection tactile pendant le mouvement devront expliquer.

La troisième étude investiga l'importance des signaux d'origine centrale et périphérique dans le phénomène des réductions de la perception tactile par le mouvement chez l'humain. Dans ce but, le décours temporel et l'ampleur des réductions de la détection tactile furent comparés pendant les tâches suivantes. Premièrement, nous comparâmes le mouvement actif et le mouvement passif, pour des mouvements d'abduction de D2 et d'extension de l'avant bras. L'idée était que les mouvements passifs généraient de l'afférence périphérique reliée au mouvement, mais éliminaient la contribution de la préparation et commande motrice centrale. Dans une deuxième partie, des tâches motrices isotoniques et isométriques d'abduction de D2 furent comparées à deux sites de stimulation, D2 et l'épaule. Le but ici était d'éliminer le mouvement comme tel tout en préservant la préparation et commande motrice centrale. Des réductions significatives dans la proportion de stimuli détectés pendant le mouvement furent observées avec toutes les combinaisons de tâches motrices et perceptuelles étudiées. Quand les réductions perceptuelles pendant les tâches motrices passives furent comparées avec celles évoquées par des tâches motrices actives, aucune différence significative dans les fonctions décrivant la performance perceptuelle au cours du temps ne fut observée. Même dans les tâches motrices passives, les réductions dans la détection tactile *précédaient* nettement le début du mouvement, par 36 ms avec l'abduction de D2 et 97 ms avec l'extension de l'avant bras, et ce malgré l'absence d'une commande motrice ou de réafférence périphérique reliée à la contraction musculaire. Quand les réductions perceptuelles pendant des tâches motrices isométriques furent comparées aux réductions perceptuelles pendant les

tâches isotoniques, encore une fois des résultats semblables furent obtenus, et ce aux deux sites de stimulation étudiés. Ces résultats démontrent que le mouvement seul, sans préparation et commande centrale, est *suffisant* pour générer des réductions de la détection tactile. De plus, ils démontrent que le mouvement comme tel n'est pas pour autant *nécessaire* pour observer des réductions de la détection tactile pendant l'exécution d'une tâche motrice. Finalement, les résultats démontrant des réductions dans la détection des stimuli tactiles *avant* le début du mouvement passif sont réconciliés avec les études de potentiels évoqués qui ont démontré que la transmission des afférences tactiles est diminuée seulement *après* le début du mouvement passif. Nous postulons que l'afférence reliée au mouvement passif interfère avec le traitement de l'information tactile périphérique *après* son arrivée au cortex mais *avant* que la perception consciente du stimulus soit établie.

En conclusion, il semble probable que le terme « gating » englobe plusieurs processus modulatoires de l'afférence tactile périphérique pendant le mouvement. Ces processus modulatoires exerceraient des effets inhibiteurs à tous les niveaux du système somatosensoriel et à toutes les étapes du traitement de l'information tactile afférente, et aboutiraient à une convergence fonctionnelle au niveau du gating de la détection pendant des mouvements actifs isotoniques, isométriques, et passifs.

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List of Abbreviations

General

1st DI	First dorsal interosseous muscle
2AFC	Two alternative forced-choice
ANCOVA	Analysis of covariance
ANOVA	Analysis of variance
CNS	Central nervous system
DC/ML	Dorsal column/medial lemniscal
DCN	Dorsal column nuclei
EMG	Electromyographic
JND	Just noticeable difference
P_{90}	Stimulus intensity at which approximately 90% of stimuli were detected at rest
PAD	Primary afferent depolarisation
RT	Reaction time
SEP	Somatosensory evoked potential
SI	Primary somatosensory cortex
S/N	Signal to noise
VPL, VPLo, VPLc	Ventral postero-lateral nucleus of the thalamus, (o)ral division or (c)audal division

Body parts

cD2	Digit 2 contralateral to the body part in motion
cSH	Shoulder contralateral to the body part in motion
D2	Digit 2, the index finger
iA	Dorsal arm, ipsilateral to the body part in motion
iD2	Digit 2, ipsilateral to the body part in motion
iD5	Digit 5, ipsilateral to the body part in motion
iFA	Doral forearm, ipsilateral to the body part in motion
iHA	Dorsum of the hand, ipsilateral to the body part in motion
iPG	Pectoral girdle, ipsilateral to the body part in motion
ISH	Shoulder, ipsilateral to the body part in motion
ITH	Thigh, ipsilateral to the body part in motion

CHAPTER 1: INTRODUCTION AND LITERATURE REVIEW.

1.1 *General introduction.*

The somatosensory system serves to transduce mechanical deformations as well as chemical and thermal stimuli into neuronal activity conveying information helpful to the organisms' survival and reproduction. Perhaps more than in any other sensory system, the function of the somatosensory system is interrelated with motor behaviour. This interrelation is reflected anatomically by the physical proximity and high degree of connectivity between the somatosensory and motor regions of the cerebral cortex (Jones et al. 1978). It is also reflected both physiologically and behaviourally by the high degree of functional interdependence of the two systems. This interdependence can be demonstrated in many ways, and is exemplified by the crucial importance of somatosensory information in the generation of appropriate motor output (Nougier et al. 1996), the dependence of the somatosensory system on movement to gather much of the information it processes (Gordon 1978), and the modifying effects of movement itself on tactile sensation, the latter being the subject of this thesis.

Touch can be defined as "to come or be in contact with; to cause to be in contact" (Chambers W.R.. 1977). As reflected in this definition, tactile experiences can be active, involving self-generated movements in order to better

appreciate the different aspects of the object being studied, or passive, reflecting movement of the object which generates or modifies its physical contact with the perceiver. It has been repeatedly argued that these two ways of touching represent fundamentally different tasks (for example, Gibson 1962; Gordon 1978). Many have also surmised that the somatosensory system could be designed in such a way that information garnered via active touch is processed differently than information gathered by passive touch (for example Chapman 1994). The principal evidence for differential processing of tactile inputs during movement comes from neurophysiological studies which have shown that transmission of tactile inputs to the primary somatosensory cortex (SI) is reduced during movement, and parallel psychophysical studies that have shown a reduction in the perception of tactile stimuli during movement. These physiological and psychophysical findings are generally assumed to be related, and are often grouped under the term movement-related gating.

This thesis explores the relationship between the perception of tactile stimuli and movement using psychophysical methods and human beings. As an introduction to the original experimental work, a review of the extant psychophysical literature which has explored movement-related gating of tactile perception is first presented. In the second part of the literature review, the potential mechanisms underlying psychophysically measured reductions in tactile perception during movement are examined. The experimental objectives and methods are then briefly presented before moving on to the experimental results

and their interpretation. The main body of the thesis consists of 3 papers. The first and second papers deal respectively with the influence of stimulus localisation and intensity on movement-related gating of perception, and present the results in the form of a mathematical representation of gating of tactile detection which accounts for the effects of stimulus intensity, localisation, and timing relative to movement-related peripheral events. The third paper examines the relative importance of central and peripheral factors in determining movement-related gating of detection by varying the motor task. Finally, the findings presented in these studies are further discussed with respect to the insights they provide into the mechanisms underlying movement-related gating of detection.

1.2 *Movement-related gating of tactile perception.*

1.2.1 Introduction

Perception of tactile stimuli is the end result of several sequential processes, including the activation in the periphery of a tactile receptor, followed by the transmission of the neuronal activity generated by the receptor through the dorsal column-medial lemniscal (DC/ML) system to the cortex (Mountcastle 1984), and the further processing of this initial cortical activity into a conscious experience (Gomes 1998; Kulics et al. 1977). Psychophysics was originally defined by Fechner as the branch of science which quantitatively studies the relationship between the physical characteristics of stimuli and our conscious

perception of them. As such, changes in perception measured using psychophysical methods can reflect modifications in the execution of any one of the processes listed above, or several processes simultaneously. Parameters of sensation which are amenable to psychophysical study include stimulus detectability (threshold estimation), discrimination threshold or just noticeable difference (JND), and stimulus magnitude (Stevens 1975). As will be detailed below, psychophysical studies have repeatedly demonstrated that movement modifies the perception of tactile stimuli. Psychophysical characterisation of movement-related gating makes it possible to determine the perceptual consequences of the phenomenon, which in turn should represent the sum of the modifications in the transmission and processing of tactile stimuli by movement. Because changes in tactile perception measured psychophysically probably represent the overall effect of more than one movement-related gating mechanism, psychophysical results cannot always determine the relative contribution of these different mechanisms. The results nevertheless define the effect (or sum of effects) that proposed explanatory mechanisms must produce on the neuronal representation of the tactile stimuli during movement.

As noted by Schmidt et al. (1990a), the psychophysical effects of movement on tactile perception depend on both movement- and stimulus-related parameters. The nature of the perceptual task (detection, discrimination, magnitude estimation) may also play a role in determining observed movement-related gating of perception. Stimulus-related parameters which may influence

movement-related gating include stimulus modality, intensity, location, duration, and timing relative to the movement. Movement-related parameters which could potentially be of great importance include the nature of the motor task (active, passive, isotonic, isometric, ballistic, tracking, exploratory...), the kinematic parameters of the movement being produced, and the body part in motion. The following section critically appraises the existing psychophysical literature, which has to a greater or lesser degree evaluated the effect of some of these factors on the perception of tactile stimuli during movement. Particular attention is paid to whether or not previous studies provided adequate control of the many potentially important stimulus- and motor-task-related parameters which potentially could confound the interpretation of the results, and to what extent results from previous work can be combined into a global understanding of the relative importance of each of these parameters on movement-related gating of perception.

1.2.2 The basic phenomenon.

Coquery et al. (1971) were the first to report that the detection of tactile stimuli is impaired during movement in humans. This oft-cited paper laid the framework for much of the subsequent study of movement-related reductions in the perception of tactile stimuli, and made several important observations. Firstly, they showed that the timing of stimulation relative to movement onset was a critical determinant of movement-related reductions in detection, with stimuli

delivered before the onset of movement-related electromyographic (EMG) activity being less or not at all affected, while stimuli delivered after EMG onset were hardly detected at all. Secondly, using a single near-threshold intensity of stimulation, they presented results which suggested that, in most cases, increasing the distance between the body segment in motion and the site of stimulation lessened the degree of movement-related decreases in the subjective intensity of the stimuli. This study was however, anecdotal in nature (e.g. only 2 subjects, with differing results, for the timing study), and there was no attempt to monitor and/or control the motor tasks. Nevertheless, the results did provide a clear indication that detection and scaling are both decreased during movement.

1.2.3 Influence of stimulus-related parameters.

Nature of the stimulus. In the somatosensory system, movement-related gating of perception has been reported both for stimuli which excite muscular afferents (Collins et al., 1998) and for those activating cutaneous mechanoreceptive afferents. Support for the second part of this assertion comes from many studies that have demonstrated movement-related gating of perception using innocuous electrical surface stimulation (for example Chapman et al. 1987; Milne et al. 1988), as well as natural tactile stimuli including pressure (Angel et al. 1985) and vibration (Dyhre-Poulsen 1978; Post et al. 1994). Consistent with the latter observations, Schmidt et al. (1990a; b) have shown that near-threshold flutter and

pressure sensations evoked using intraneural microstimulation are diminished by movement. Existing evidence thus favours the interpretation that all the tactile submodalities mediated by large-diameter cutaneous fibres are likely decreased during movement, though the range of stimulus parameters over which they are affected may differ (see below). On the other hand, neither pain nor innocuous thermal sensations (both transmitted via smaller diameter cutaneous afferent fibres) seem to be gated by motor activity. Feine et al. (1990) examined the effect of isometric elbow flexion and extension on the perception of electrical, innocuous thermal, and painful thermal stimuli. Both qualitative and statistical assessments of the experimental results unequivocally showed that perception of the near-threshold electrical stimuli was profoundly diminished during the motor task, while perception of innocuous and painful thermal sensations was unaffected.

Stimulus intensity. The influence of stimulus intensity on movement-related gating of tactile perception was first studied as an independent variable by Chapman et al. (1987). Using a detection task, higher intensities of electrical stimulation were necessary during movement in order to match detection performance at rest, i.e. there was a movement-related increase in detection threshold. However, discrimination of small differences in the intensity of suprathreshold stimuli was unchanged during movement, and when examining the scaling of suprathreshold but innocuous electrical stimuli, magnitude estimates at rest and during movement were not significantly different over the range of

stimulus intensities tested. Helminen et al. (1994) and Pertovaara et al. (1994) obtained similar results, again using electrical stimuli: the detection threshold was significantly increased during active movement, but the just noticeable difference (JND), using 3X or 10X T stimuli as anchors, was unaffected by active movement. When the Chapman et al (1987) experimental paradigm was repeated using vibrotactile stimuli (Post et al. 1994), detection thresholds were again found to be elevated, discrimination of small differences in the intensity of suprathreshold stimuli was also unchanged during the motor task, but magnitude estimates were found to be decreased over the range of stimulus intensities tested. In summary, during movement, preliminary evidence indicates that stimulus intensity appears to be an important determinant of observed decreases in the detectability of near-threshold tactile stimuli and the perceived magnitude of suprathreshold stimuli, while relative differences between suprathreshold stimuli are not affected. It is possible that inter-modality differences in the effect of movement on the scaling of suprathreshold stimuli could be the result of differing amounts of movement-related afferent feedback in the different studies. Alternatively, the different stimulation modalities could show intensity-dependent effects of movement on scaling over very different ranges of stimulus intensity.

Stimulus location. Since the pioneering work of Coquery et al (1971), others have reinvestigated the influence of the relationship between the site of movement and the site of stimulation on tactile perception during movement. The absence of effect when stimuli are delivered contralateral to the body part in

motion has been repeatedly established (Chapman et al. 1987; Papakostopoulos et al. 1975; Pertovaara et al. 1992; Schmidt et al. 1990b), providing an elegant demonstration that movement-related gating of tactile perception is not due to non-specific attentional factors related to the simultaneous performance of two different tasks, one motor and one perceptual. Based on an anecdotal report that magnitude estimates of electrical stimuli delivered to a digit were only reduced when the same digit produced a movement, and not adjacent or contralateral ones, Papakostopoulos et al. (1975) first suggested that only the body part in motion was subject to gating influences. However Papakostopoulos' report that gating occurs only on the body part in motion is contradicted by later studies. Milne et al. (1988) using electrical surface stimuli delivered during a rhythmic active abduction-adduction of the index finger (D2), and Schmidt et al. (1990b) using intraneural microstimulation of finger afferents delivered during flexion-extension of D2, both explored gating of tactile detection over the extent of the ipsilateral fingers. Both studies qualitatively described a distance-dependent gradient in the gating of tactile detection. This result is interesting but difficult to generalise, given the limited area over which the effect of distance was evaluated. Post et al. (1994) examined the effects of delivering stimuli to sites on the forearm, thenar eminence and digit during an elbow flexion-extension task. They reported significant decreases in detection only at the two sites closer to the elbow; however when the magnitude of the decrease was examined, detection threshold was increased almost as much at the digit site (37%) as at the other two sites (62% at the thenar eminence, 40% at the forearm), suggesting that differences in

response variability may have been more important in determining the proximal-distal gradient than actual differences in the magnitude of the effect. Thus the question of whether there is an actual spatial gradient for detection performance distal to the joint involved in a movement, or whether all sites distal to the joint about which the movement occurs are subject to similar gating influences, remains open to question.

Stimulus timing relative to movement. Although stimulus timing relative to peripheral movement-related events was anecdotally reported (n=2) by Coquery et al (1971) to be a key factor in determining movement-related gating of tactile perception, it has received relatively little attention in the psychophysical literature, with many of the above mentioned studies not even including measures of stimulus onset relative to movement parameters. This is unfortunate, as knowledge of the timing of movement-related decreases in perception can greatly help in identifying possible sources of gating signals. For example, modulation of perception that precedes the onset of movement and EMG activity is generally interpreted as evidence that central signals, related to the preparation and execution of the movement, play a role in gating (Dyhre-Poulsen 1978). Conversely, modulation which follows movement onset is often ascribed to the influence of peripheral feedback generated during movement execution. Psychophysical estimates of the time of onset of gating influences vary widely. In the study of Coquery et al (1971), one subject showed reductions in detection which preceded EMG onset by 50 ms, while the other showed reductions in detection only after the

onset of EMG. Dyhre-Poulsen (1978) looked at the effect of finger movement on the ability to detect electrical stimuli applied to the finger before movement onset, and found reductions beginning 51-100 ms before *movement* onset. This number cannot be compared directly to those obtained by Coquery et al (1971), as Coquery timed his stimuli relative to EMG onset. Angel et al. (1985) studied the time course of recovery for detection of tactile (pressure) stimuli *after* the end of a rapid abduction of the thumb. They found that the ability to detect stimuli was impaired up to 250 ms after the end of the movement, and calculated that suppression of detection faded over time with a half-life of about 100 ms. To summarise, there is anecdotal evidence in the literature that movement-related reductions in detection begin before the onset of movement, and one report of one subject who showed reductions in detection which preceded the onset of movement-related EMG. Also, detection performance is impaired after the end of movement.

1.2.4 Movement-related parameters.

The nature of the motor task. Movement-related gating of tactile perception has been observed in a variety of motor tasks, including active, passive, isometric, ballistic, rhythmic, tracking and locomotor movements. For example, Dyhre-Poulsen (1978) reported that ballistic and tracking movements produced similar reductions in the probability of detecting vibrotactile stimuli during movement. Locomotion also produces decreases in the perceived magnitude of

electrical stimuli, although in this case the changes are phase-dependent in a manner consistent with a central facilitation prior to foot contact with the ground, when peripheral information is most relevant to the performance of the task.

Several studies have examined the impact of changing the nature of the motor task on perception of tactile stimuli. Chapman et al. (1987), using relatively circumscribed rhythmical movements about a single joint, found that both active and passive elbow movement produced similar reductions in the proportion of stimuli perceived during movement. Since there is no motor command during passive movement, the demonstration that passive movement reduces stimulus detection shows that central motor-task related activity is not solely responsible for movement-related reductions in tactile detection. Isotonic and isometric contractions about the elbow or D2 have also been compared and been found to produce similar reductions in perceptual performance (Feine et al. 1990). This result demonstrates that movement itself is not a prerequisite for motor-task-related reductions in detection performance, though interpretation of detection results during the isometric task were made difficult by the additional peripheral feedback generated by the unmoving support which blocked the movement in the isometric task. Observations of reductions in tactile perception during passive and isometric movement were confirmed by the work of Milne et al. (1988), in this case using a stimulus-matching paradigm.

In conclusion, most evidence supports the contention that isotonic, isometric and passive motor tasks produce similar reductions in tactile perception during movement, although the mechanisms underlying the reductions are not necessarily equivalent in each case (see Section 1.3 *Proposed mechanisms...* below). Behavioural relevance may also affect movement-related gating in more stereotypical movements such as locomotion, but does not seem to affect movement-related gating in a feedback-dependent tracking task.

Movement kinematics. It has been suggested that the magnitude of gating effects covaries with the kinematics of the movement, especially speed. Angel and Malenka (1982) studied the velocity-dependence of movement-related gating of tactile detection. They determined the sensory threshold while subjects attempted to make active finger flexion/extension movements at three different speeds (continuously alternating flexion/extension at 1, 2, and 3 Hz). Although the performance of the motor task was not quantified, the results suggested that perceptual performance varied across the three conditions, with higher attempted "velocities" producing greater reductions in detection. These findings were confirmed using a rhythmic elbow flexion/extension task by Chapman et al. (Fig. 4 in 1996). Schmidt et al. (1990a), again using finger flexion/extension, found that movement velocity also affected the magnitude of both pressure and flutter sensations evoked by near-threshold intraneural microstimulation in a manner similar to that described by Angel et al. (1985) for detection. These studies suggest that velocity is a key kinematic parameter for determining movement-related

reductions in detection. However, correlations between perceptual performance and movement velocity may simply be the reflection of a relationship between perceptual performance and a more fundamental movement-related parameter, such as force or even changes in force such as those occuring in isometric or the onset of isotonic movement. This suggestion is made more credible by the results of Post et al. (1994), who demonstrated in isometric motor tasks that perceptual gating indeed varies with the force produced by muscular contraction.

Several conclusions can be drawn from this review. Available evidence, although far from complete, appears to indicate that the nature of the stimulus, as well as stimulus intensity, timing, and localisation, all contribute to determining the magnitude of movement-related gating of perception. Movement-related parameters also seem to play an important role. The results of all these experiments raise obvious questions about the way the various stimulus parameters, as well as movement parameters, *interact* to determine changes in stimulus detectability during movement. From a methodological standpoint, the accumulated data demonstrate that studies of movement-related gating need to control, or at least measure, the parameters of stimulation (nature, duration, timing, intensity and localisation) and movement-related kinematic and timing parameters. In addition, the nature of the perceptual and motor tasks is important, and both need to be clearly defined. In terms of functional utility, the psychophysical results describing movement-related reductions in tactile perception are compatible with the hypothesis that during movement, low

intensity stimuli are gated in order to reduce the amount of movement-related "noise" afference which reaches higher centres. Possible mechanisms underlying the perceptual gating effects are detailed in the following section.

1.3 *Proposed mechanisms underlying movement-related gating of tactile perception.*

A thorough review of the mechanisms underlying movement-related gating of tactile perception needs to consider the nature and the origin of the movement-related signals which directly or indirectly modulate perception. What is the gating signal, and where does it come from? Also to be considered is the level of the somatosensory system at which these signals influence the various processing steps leading to the conscious perception of tactile stimuli. In other words, where does gating act?

The existing psychophysical literature suggests that the gating signal(s) is characterised by its dependence on movement kinematics and kinetics. The signal also may immediately precede or coincide temporally with the onset of peripheral movement-related events such as EMG onset and the beginning of actual movement, and continues throughout the movement.

The gating signal(s) which is responsible for modifying perception during movement may be either central and/or peripheral in origin. Central signals which fit the characteristics described above and therefore could be mediating movement-related gating include neuronal activity related to motor preparation and especially the motor command. Although some somatosensory neurones begin to discharge before the onset of movement (Soso and Fetz 1980), most of the pre-movement neuronal activity takes place in the pre-motor and primary motor cortices. The notion that corollaries of the signals from motor command centres ("efference copy") can cancel out predictable movement-related reafference by acting on sensory structures was proposed by Von Holst and Mittelstaedt (1950), and has been demonstrated experimentally (Bell 1982). Such a mechanism could account for gating of low intensity tactile afference during movement. A second source of gating is, however, the peripheral afference generated by movement. Peripheral feedback could affect transmission directly via inhibition of transmission at any one of the relays of the DC/ML pathway to SI. Movement-related afference could also affect transmission indirectly once it has reached the parietal cortex, by activating control systems (feedback loops) projecting back to the relay nuclei of the DC/ML system. As reviewed below, existing anatomical and physiological evidence supports the notion that both central and peripheral gating signals may be effective in reducing tactile perception during movement.

The gating signals may interact with the neuronal activity elicited by the test stimulus (i.e. the stimulus to be perceived) at multiple levels beginning as early as the primary afferent neurone. Transmission through the various relays of the DC/ML system may also be affected by gating signals. Finally primary somatosensory cortical responsiveness and the later processing of tactile information into a conscious experience can also be affected.

While the function of gating is not known, movement-related gating appears to be a widespread phenomenon, being observed not only in the DC/ML system but also for example in the spino-olivo-cerebellar (Apps et al. 1997) and visual (Latour 1962) pathways. Modulation of the access of sensory information to the central structures involved in the further processing of these signals by gain control mechanisms appears to be a general and important process in sensori-motor control (Apps et al. 1997; Brooke et al. 1997; Prochazka 1989). Indeed, selective inhibition of afferent information during transmission has long been postulated to serve a vital purpose in the processing of afferent input (Von Békésy 1967).

1.3.1 Gating at the level of the primary afferent neurone.

Many sensory systems incorporate efferent controls that modify the response of a receptor to a given external stimulus. One simple example is found

in the visual system, where pupil diameter is adjusted to optimise the amount of light falling onto the retinal photoreceptor cells. Efferent control of sensory systems at the level of the sensory receptor becomes more complex when receptors are scattered over a wide area, such as is the case in the somatosensory system. For instance, regulation of muscle spindles requires that each spindle be wired to a motor control system dedicated to this task. Tactile receptors do not seem to possess a separate neural pathway exclusively for efferent control at the level of the peripheral receptor. There are nevertheless central controls over transmission of information at a very early level in the somatosensory system. In the DC/ML system, this can be seen as early as the dorsal horn, where stimulation of SI produces reductions in cord dorsum potentials (Hagbarth and Kerr 1954). These reductions are thought to be mediated by direct and indirect projections from SI to the dorsal horn of the spinal cord (Coulter and Jones 1977), where gamma-aminobutyric acid (GABA)ergic interneurones in turn inhibit transmission at the spinal afferent terminals via primary afferent depolarisation (PAD). This mechanism is supported by a study which demonstrated that stimulation of SI does in fact produce PAD in cutaneous nerve afferents (Andersen et al. 1964a). In addition to these cortical controls, strong evidence exists to support the notion that spinal central pattern generators can also modulate the response of primary afferent neurones to peripheral stimuli, at least in decerebrate and/or spinal preparations. Indeed, during both real (Yakhnitsa et al. 1989) and fictive (Gossard et al. 1990; Gossard and Rossignol 1990) locomotion, dorsal root potentials evoked by peripheral cutaneous nerve

stimulation are phasically reduced during the locomotor cycle. Peak reductions of up to 80% as compared to maximal evoked activity are observed during ipsilateral swing phase. PAD of sufficient intensity can also produce antidromic discharge in cutaneous afferents (Dubuc et al. 1985). This antidromic activity may travel outwards to the periphery and modulate receptor responsiveness (Gossard et al. 1999). Alternatively, PAD-produced activity could selectively affect only certain branches of the primary afferent neurone. Indeed, control of transmission at the level of the main branches of the various arborisations of the primary afferent neurone has been demonstrated by Wall (1994), and could serve to selectively permit the transmission of antidromic discharge only to certain areas of the primary afferent neurone.

Notwithstanding the role of central structures in the modulation of primary afferent responsiveness, PAD of cutaneous afferents can also be evoked by peripheral stimuli such as muscle stretch (Devanandan et al. 1965) and natural stimulation of the hair and skin (Schmidt et al. 1966). Thus, during movement, reductions in primary afferent neurone responsiveness could also be mediated by movement-related peripheral reafference (Burke et al. 1977; Edin and Abbs 1991; Hulliger et al. 1985; Hulliger et al. 1979). This peripherally evoked PAD may be modulated by centrally controlled PAD. For example Gossard suggests that during locomotion peripherally evoked PAD could be greater during stance than during swing. This would occur because during swing central PAD reduces responses to peripheral stimuli (and therefore peripherally evoked PAD). In any

case, clear evidence for both peripherally and centrally controlled PAD exists, and may represent the first level at which the nervous system modifies afferent influx during movement, modifications which potentially could lead to the reduced perception of tactile afference during movement.

1.3.2 Gating of tactile transmission at the somatosensory relay nuclei and primary somatosensory cortex.

"...the cerebral cortex is not a passive recipient of the sensory information transmitted up the spinal cord and brain stem; it exercises powerful inhibitory controls...at the synaptic relays on the pathways."
(Eccles, 1964)

The simplest conception of the somatosensory system views it as a series of relays whose input signals remain relatively unprocessed until they reach the regions of cortex concerned with primary somatosensory transformations, leaving little room for control of transmission. This view is an oversimplification of classical studies which found cortical neurones whose receptive field properties closely match the attributes of peripheral stimuli (Mountcastle 1957), but nonetheless is the generally accepted paradigm for somesthesis in much of clinical neurology and neurosurgery. Notwithstanding the clinical primacy of this simple conception of somesthesis, a large body of evidence now exists in favour

of functional feedback loops between cortex and the various somatosensory relays, feedback loops which likely serve to control transmission gain in the relays. According to Towe (1973), serious interest in the study of centrifugal influences on ascending somatosensory systems began about mid-century (e.g. Hagbarth and Kerr 1954), marking the beginning of a greater awareness that the cortex is not simply a passive recipient of sensory information, but rather exerts strong controls over sensory transmission. The role of inhibition in sensory systems is often considered to be the enhancement of specific signals at the expense of others that are "later, lesser or more lateral" (Von Békésy 1967). The simplification or optimisation of the afferent signal would thus serve to highlight that information which is most important to the organism. In this section, a description of reductions in somatosensory transmission during movement will first be presented. Possible centrally and peripherally controlled mechanisms underlying these reductions in transmission will then be discussed.

Experimental evidence for the existence of movement-related reductions in transmission at the somatosensory relay nuclei of the tactile system comes from many sources. Studies examining somatosensory evoked potentials (SEPs) during movement have found that sensory transmission through the DC/ML pathway is decreased (e.g. Chapman et al. 1988) at the level of the dorsal column nuclei (DCN). Accumulated experimental evidence clearly demonstrates that transmission is reduced by 10-40% up to 200 ms *before* the onset of movement (Chapman et al. 1988; Coulter 1974; Dyhre-Poulsen 1978; Ghez and Pisa 1972;

Ghez and Lenzi 1971). The timing of these reductions is consistent with the hypothesis that the preparation and execution of movement modulates somatosensory system responsiveness. Consistent with this hypothesis, there is much evidence that transmission through the DCN can be modulated by corticofugal signals. Plausible sources of a pre-movement gating signal are the primary motor cortex , as demonstrated by both transcranial magnetic stimulation (Cohen et al. 1991) and direct intracortical microstimulation (Jiang et al. 1990) and/or the supplementary motor area, as demonstrated using a mental movement simulation paradigm (Cheron and Borenstein 1992). Control via an efference copy mechanism may be mediated via direct cortico-bulbar projections from primary motor and sensory cortices (Bentivoglio and Rustioni 1986; Cheema et al. 1985; Jones and Wise 1977; Kuypers 1958, 1960; Martinez et al. 1995). Physiological support for the hypothesis that motor cortical activity during movement can directly inhibit transmission in the DC/ML system comes from studies in the cat, which demonstrated that direct stimulation of the motor or somatosensory cortex can modify the responses of single cells at the level of the DCN to cutaneous stimulation (Jabbur and Towe 1960, Andersen et al 1963b, 1963c). In the rat, responses of DCN neurones to peripheral stimuli were increased when the receptive field corresponded to the location of joint about which movement occurred and reduced when the receptive field was adjacent to this area. Units with a more distant receptive field showed no change (Giuffrida et al. 1985). These results suggest that the cortical control over transmission in the DCN is somatotopically organised. In SI of monkey (Jiang et al. 1990), a proximo-distal gradient was observed: weak conditioning stimulation of motor cortex decreased SI responses to stimulation of sites over, or distal to the (potentially) activated muscle, but no modulation was observed for SI

recording sites receiving skin input from areas located proximal to the potentially activated muscle group, demonstrating a striking pattern of functional organisation for the primate motor cortical controls over DCN transmission.

Parallel to descending controls which exist at every relay of the somatosensory system, peripheral inputs themselves also exert strong inhibitory influences on transmission of somatosensory afference at the level of the DCN. These actions can be direct or indirect, via feedback loops through SI. It has long been known that a centripetal volley originating in the dorsal columns will suppress transmission in the DCN (Marshall 1941). Later, Ghez & Pisa (1972) showed that peripheral stimulation, through a PAD mechanism, could also inhibit DCN responsiveness. However, when they examined DCN transmission during passive movement, no significant decrease was found, a result which was later confirmed by Chapman et al. (1988). Thus, the role for peripheral input in mediating movement-related reductions in DCN responsiveness remains unclear. In summary, early decreases in DCN responsiveness that occur before movement may be controlled by motor cortex via its projections to the DCN, while later modulation could be a combination of primary motor and sensory cortical inputs. Though peripheral inputs could also play a role in mediating decreases after movement onset, present evidence from passive movement studies does not confirm a role for peripheral input at the level of the DCN during movement.

At the thalamic level, Chapman et al. (1988) demonstrated further reductions in the transmission of potentials evoked by ML stimulation and recorded in cortex, additive to the effects already seen when recording in the ML. The added reduction in transmission at the thalamic level was, however, observed only after the onset of movement, active or passive. The timing of reductions at the thalamic level, as well as their presence during passive movement, favour the interpretation that the reductions are generated by peripheral movement-related reafference. However, during active movement, concomitant centrally modulated reductions in transmission mediated by pyramidal tract neurones cannot be excluded (Tsumoto et al. 1975). Potential anatomical substrates for cortical control of transmission through the thalamic relay have been identified. Motor cortex has been shown to project to VL and VPLo relay nuclei as well as the thalamic reticular complex, although not directly to VPLc thalamus (Jones 1975). The motor cortex also projects to several of the cytoarchitectonic divisions of the somatosensory cortex (Jones et al. 1978). The latter in turn projects to VPLc thalamus as well as the thalamic reticular nucleus (Jones and Wise 1977). In addition to central controls at the thalamic level, peripheral afference also controls thalamic transmission. Centripetal volleys originating in the dorsal column suppress transmission in the ventrobasal thalamus (Andersen et al. 1964b) via both pre- and post-synaptic mechanisms. To summarise, at the thalamic level, both central and peripheral signals can modulate transmission of tactile afference, but reductions in transmission are only seen after the onset of movement,

favouring the interpretation that peripherally originating signals play a key role in inhibiting transmission at the thalamic relay during movement.

The amplitude of cortical primary SEPs have also been observed to decrease during movement (Cohen and Starr 1987, Chapman et al 1988), with the observed decrease being larger than that observed at either of the lower level relay nuclei (Chapman et al 1988). The time-course of cortical SEP attenuation during active digit movement in humans was estimated by Cohen and Starr (1987). They reported that the early cortical components of the SEP (pre-central P22 and post-central P27) in response to median nerve stimulation were significantly diminished beginning roughly 50-100 ms before the onset of movement-related electromyographic (EMG) activity, which itself preceded the onset of movement, and that attenuation was no longer apparent approximately 400 ms after EMG activity had ended. A more precise determination of the time-course of the movement-related attenuation of the short-latency responses to electrical percutaneous stimulation or air-puff stimulation was obtained in the monkey using an intracortical recording electrode (Chapman et al. 1988). Again and regardless of the nature of the stimulation, active movement was found to significantly reduce the amplitude of short latency SEPs beginning as early as 60 ms before the onset of movement-related EMG. The demonstration of reductions in the amplitude of SEPs which preceded the onset of peripheral movement-related EMG activity must be considered extremely strong evidence in favour of central control of somatosensory system responsiveness in the awake behaving primate.

Whether reductions in cortical primary SEPs are the reflection of sub-cortical SEP reductions or represent additional reductions in transmission at the cortical level was addressed by Chapman et al. (1988). By stimulating VPLc thalamus and recording cortical EPs, they demonstrated that cortical mechanisms produced reductions in evoked responses independently of sub-cortical modulation, but that these supplementary decreases occurred only after movement onset. Thus, decreases in cortical SEPs represent the sum of sub-cortical signal modulation combined with added cortical modulation of the primary SEP.

Evoked potential studies in humans have also demonstrated that active isotonic or isometric movement (Jones et al. 1989; Papakostopoulos et al. 1975; Rushton et al. 1981), externally generated (passive) movement (Huttunen and Homberg 1991; Jones et al. 1989; Rushton et al. 1981), or even mental movement simulation (Cheron and Borenstein 1992) tasks are all capable of reducing the amplitude of post-central SEPs. Interestingly, when the time course of reductions in SEPs was examined (Chapman et al. 1988), passive movements, which do not involve activation of motor cortex before movement onset, produced a decrease in the SEP only after the onset of movement, demonstrating that peripheral movement-related afference can also affect transmission through the somatosensory system, but with a time course which is different from that seen with voluntary movement.

The relation between stimulus relevance and gating of transmission has also been addressed experimentally. Single cell recordings in areas 3b and 1 of SI have demonstrated that when the stimulus is not behaviourally relevant, about 9 cells out of 10 show decreases in short latency discharges evoked by peripheral stimulation when these stimuli are delivered during movement (Jiang et al. 1991). This modulation was occasionally seen up to 50 ms before EMG onset in an active movement task, although on average the decrease in responsiveness more or less paralleled the onset of EMG. In contrast to these findings, responses to behaviourally relevant stimuli may in fact show some evidence of "sparing" from the ubiquitous gating actions described above. Chapin and Woodward (1982) demonstrated in rats that single cells could respond variably to peripheral stimulation during movement, depending on the importance of peripheral feedback for the successful execution of the motor task. During regular, rhythmic locomotion movements, cells responded only weakly to footfall, whereas during "irregular", feedback-controlled locomotion, the same cells could discharge strongly in response to footfall. Further evidence for centrally mediated modulation of SI cell responses to peripheral vibratory stimuli comes from the work of Lebedev et al. (1994). In their experimental paradigm, the stimulus was behaviourally relevant as it also served as a movement cue. A detailed analysis of somatosensory cortex cell responses to vibratory stimuli during the pre-movement motor-preparation period demonstrated that pre-movement-related activity could either increase or decrease the firing rate of cells. However, when firing rates were increased, synchronisation between the frequency of the vibratory stimulus

and the neuronal discharge decreased. Thus, increases in discharge may not have reflected "facilitation" of transmission. Rather, they perhaps represented the addition of a second asynchronous pre-movement-related signal, which disrupted the synchronised stimulus-related discharge pattern. In summary, transmission of behaviourally irrelevant stimuli appears to be relatively non-specifically gated during movement, whereas behaviourally relevant stimuli are subject to more complex modulation before and during movement.

Decreases in SEPs during movement have mostly been studied using upper limb movement and stimulation paradigms. Similar decreases are also observed during lower limb movement and stimulation (Brooke et al. 1997; Staines et al. 1997a; Staines et al. 1997b). Decreases in early cortical SEPs are observed up to 60-100 ms before EMG onset during active movement (Morita et al. 1998; Staines et al. 1997b), but coincide with the onset of passive movement. These timing values are compatible with those found during movement-related gating of SEPs from the upper limb, and again demonstrate the ability of both central and peripheral movement-related signals to reduce somatosensory transmission.

1.3.3 Gating of cortical processing.

All of the results presented in the previous section dealt with movement-related modifications in the earliest potentials evoked by tactile stimuli. However, it is also possible that later deflections could also be affected by movement. Due to their less obvious relationship to the peripheral stimulus, longer latency evoked responses have received less study that short latency SEPs. However, there is evidence that conscious perception of tactile stimuli is related more to the amplitude of the longer latency evoked responses which follow the initial sensory evoked potential to peripheral stimuli than to the amplitude of the shorter latency responses (Gomes 1998, Kulics et al. 1977; Libet et al. 1964), corresponding to those examined in most studies of movement-related gating. A few researchers have examined some later components of the SEP, with variable results (Brooke et al. 1996; Rushton et al. 1981), but did not correlate SEP variation with reported variations in perception. A recent report from Brosch et al. (1998) has reported selective suppression in monkey auditory cortex of longer latency responses to auditory stimuli by another auditory stimulus delivered up to 140-180 ms after the onset of the test stimulus. This observation indicates that longer latency responses to peripheral stimuli are potential targets for movement-related reductions in amplitude. Whether or not such reductions of longer latency responses occur during movement, and whether they correlate with perceptual reductions, remains to be determined.

1.3.4 Masking of tactile stimuli.

"*The important thing in a communication network is not the output level attained but the signal to noise ratio, for it is this ratio that determines our ability to recognise the signal as distinct from the noise. In the nervous system it was found that noise is always present...and a sensory effect has to be identified in the presence of this background*".

(Von Békésy, 1967)

Considerable experimental evidence exists to support the notion that peripheral movement-related tactile afference plays a key role in the generation of movement-related gating of tactile transmission and perception (see above). This suggestion receives additional support from a large body of psychophysical experiments studying "masking". Masking refers to the phenomenon by which the perception of a "test" stimulus is reduced in the presence of another stimulus, known as the "masker". This overview concentrates on results obtained using tactile stimuli as both the test and masker stimuli. The goal of the psychophysical experiments interested in masking has been to determine how information processing, measured in terms of perception, is modified in the presence of competing stimuli, with the ultimate aim of designing efficient communication systems for the different sensory modalities (Weisenberger 1994). To this end the effect of various types of masking stimuli on subject performance in both

detection and pattern recognition tasks has been investigated. This has usually been accomplished by presenting human subjects with both a tactile test stimulus which they must detect or identify, and a tactile masking stimulus which can be made to precede, coincide with or follow the test stimulus. These types of studies offer easier control of several important variables when compared with studies involving movement. These include: masking stimulus intensity, masking stimulus duration and interstimulus interval. Most of the results of these studies are presented in dB of attenuation using scales where 0 dB absolute represents perceptual threshold, in part to facilitate comparisons with similar studies using auditory stimuli. The basic result of the early masking studies was that the ability to detect a tactile test stimulus was impeded when a tactile masking stimulus was applied (Scherrick 1964; Schmid 1961; Weisenberger 1994). These and later studies also determined that the effectiveness of the masking stimulus depended on the following factors:

Distance between the site of the test stimulus and the site of the masking stimulus. Tactile masking stimuli are most effective when they are applied to the same site as the test stimulus. Increasing the distance between the two stimulation sites reduces the impact that the masking stimulus has on perception of the test stimulus, all other things being equal. For example, Scherrick (1964) found that when the test stimulus was applied to the right index finger, a masking stimulus applied to the lip area produced a maximum of only 10 dB of attenuation, as compared to a maximum of 23 dB attenuation when the masking stimulus was

applied to the right index finger. Masking is observed even when the distance between masking stimulus and test stimulus is great enough to preclude direct mechanical interactions between the masking stimulus and the tactile receptors activated by the test stimulus. The observed relationship between inter-stimulus distance and masking ability parallels that seen in tactile gating studies for the site-of-movement-to-stimulation-site distance.

Intensity of the masking stimulus. More intense masking stimuli produce more pronounced masking effects. For example, (Schmid 1961) was able to double the effectiveness of a masking stimulus by increasing its intensity.

Interstimulus interval. The interval of time between the end of the masking stimulus (masker) and the beginning of the test stimulus is critical in determining the magnitude of the masking effect. Test stimuli that follow the masker can be affected (forward masking), as can test stimuli that *precede* the masker (backward masking). Maximal masking always occurs when the interstimulus interval is minimised (e.g. Schmid 1961; Weisenberger 1994). However, when detection performance is tested by delivering the test stimulus immediately before the masking stimulus (forward masking), a greater effect on test-stimulus perception, over a longer time-course, is seen compared to when detection performance is tested by delivering test stimuli immediately after the masking stimulus (backward masking) (Schmid 1961). Interestingly, when a tactile recognition task is substituted for the detection task, this asymmetry in the forward and backward

masking functions is reversed: greater amounts of masking over longer periods of time are observed when the test stimulus follows the masker (Kirman 1984).

The existence of backward masking, which can extend for interstimulus intervals of up to 100 ms, provides insight into the central processing of tactile information. Backward masking especially is thought to be the result of the masker influencing high level cortical processing of the test stimulus (e.g. (Scheerer 1973; Schultz and Eriksen 1977)). Two processes have been proposed to account for backward masking. In the *integration* hypothesis, the masking stimulus is proposed to interact with the test stimulus during processing to produce a composite result that does not allow the perception of the test stimulus as a separate event. This hypothesis is equivalent to postulating a decrease in the signal to noise (S/N) ratio. In the *interruption* hypothesis, the arrival of the masking stimulus stops the processing of the test stimulus, generating an incomplete perception of long duration stimuli and completely obliterating perception of short duration stimuli. Electrophysiological evidence in favour of the interruption hypothesis exists for both short latency neuronal responses to a tactile test stimulus (Laskin and Spencer 1979) and longer latency neuronal responses evoked in response to an auditory stimulus (Brosch et al. 1998).

Duration of the masking stimulus. Weisenberger (1994) tested masking stimulus durations from 50-300 ms. All other factors being equal, masking stimuli of longer duration generally produced larger masking effects. For

example, at an interstimulus interval of 100 ms, a 300 ms masker of 25 dB intensity was sufficient to reduce the intensity of the test stimulus from 20 dB to threshold, while a 50 ms masker needed to be of 37 dB intensity in order to reduce the perceived intensity of the test stimulus by the same amount.

Number of masking stimuli. Weisenberger (1994) also examined the effect of two vibrotactile masking stimuli on the perception of test stimuli. While strict additive effects were not always found, multiple maskers always increased the amount of masking produced. Two forward maskers produced masking effects close to what would be predicted by simple addition of their individual masking effects; Two backward maskers, identical to the forward maskers, produced masking effects that were greater than predicted by the simple addition of their individual masking effects. She interpreted this as evidence that the mechanisms underlying forward and backward masking are not the same. From this work it can be reasonably assumed that multiple, relatively weak stimuli such as those produced by movement should be quite effective at masking a tactile stimulus.

Subject age. Gescheider et al. (1992) specifically looked for age-related differences in the effectiveness of masking stimuli. He found that, depending on the interstimulus interval tested (from 20 to 500 ms), an identical masking stimulus produced threshold shifts of 4-19 dB in a group of 7 subjects with a mean age of 65 years compared to 2-12 dB in a group of 7 subjects with a mean

age of 19 years. They concluded that these differences where the result of greater adaptation during presentation of the masking stimulus in older subjects.

In summary, tactile masking has been well-characterised with regards to most of the important stimulus-related variables, and demonstrates convincingly that peripheral signals can profoundly affect detection of tactile stimuli. The neuronal mechanisms underlying masking remain unclear, although several interesting studies have begun to provide some insight into this question (Brosch et al. 1998; Laskin & Spencer 1979; see above). Although masking may be the result of peripheral stimuli influencing tactile transmission, as explored in relation to movement-related gating in section 1.3.3 above, it does not necessarily imply active inhibition of transmission in the somatosensory system. One can also hypothesise that masking results from an increase in the noise level from which stimuli must be distinguished, and so a decrease in the S/N ratio which renders the stimuli to be detected more difficult to perceive. As such, masking would appear to be an unavoidable consequence of the way our sensory systems are designed. Current psychophysical masking results are compatible with the psychophysical characterisation of movement-related gating of tactile detection. Therefore, tactile masking is potentially mediated by the same mechanisms as movement-related gating of tactile perception. Whether or not this apparent compatibility between masking and movement-related gating holds up to closer scrutiny can only be evaluated by performing further gating experiments in which more of the critical factors listed in the psychophysical section on movement-related gating are

explicitly evaluated, as well as more physiological experiments in order to precisely determine through what mechanisms stimuli interact in tactile masking. This will make it possible to clearly define which movement-related reductions in tactile perception are mediated by the same mechanisms as tactile masking, and which are not.

1.4 *Summary and statement of purpose.*

Much of the gating literature attempts to argue that movement-related gating serves a functional purpose. This "active" gating hypothesis could be summarised by the following statement: gating is the result of active control of the transmission and subsequent processing of the neural activity representing the gated stimulus by efferent and/or afferent signals. It serves to control the amount of afferent information which must be processed by the central nervous system during movement, thus preserving resources for the processing of more relevant information. On the other hand, a review of the masking literature shows it to be dominated by a "S/N" hypothesis, which could be summarised by stating that attenuation of one signal by another is the result of a decrease in the signal to noise ratio of the somatosensory system. If this hypothesis applies to movement-related gating of tactile perception, gating would thus reflect the inherent perceptual limitations of the somatosensory system, and would not necessarily confer a functional advantage to the organism. It is important to note that these two hypotheses are not mutually exclusive: actively modulated controls may

selectively inhibit the transmission and processing of precisely the stimuli which would in any case be masked (and thus be rendered useless to the organism) by movement-related reafference.

The three series of experiments which form the body of this thesis were designed to explore the issues raised by the masking and gating literature. Test stimuli can be described in terms of their timing, localisation, intensity, duration, and modality. The effect of systematically modifying the first three items on this list were explored in the first two papers. Since tactile masking exhibits characteristic spatio-temporal parameters, clearly defining the spatio-temporal characteristics of movement-related gating of tactile perception is important if comparisons between the two are to be made. Similarities in the spatio-temporal parameters of the two phenomena would argue in favour of interpreting movement-related gating in terms of the "S/N" or combined "S/N" and "active" hypotheses. Differences in the spatio-temporal characteristics of tactile masking and movement-related gating would argue in favour of the "active" interpretation of movement-related gating.

The third series of experiments represents an attempt to assess the relative importance of the various centrally and peripherally originating movement-related signals for the gating phenomenon, by comparing both the time-course and the magnitude of movement-related gating obtained during various motor tasks. The results of these experiments could also serve to differentiate between "active" and

"S/N" mediated gating. If differences in movement-related gating are found by selectively eliminating potential control signals, this would argue in favour of "active" gating.

CHAPTER 2

TIME-COURSE AND MAGNITUDE OF MOVEMENT-RELATED GATING OF TACTILE DETECTION IN HUMANS.

I IMPORTANCE OF STIMULUS LOCALISATION

Time Course and Magnitude of Movement-Related Gating of Tactile Detection in Humans. I. Importance of Stimulus Location

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Williams, Stephan R., Jafar Shenasa, and C. Elaine Chapman. Time course and magnitude of movement-related gating of tactile detection in humans. I. Importance of stimulus location. *J. Neurophysiol.* 79: 947–963, 1998. The time course and spatial extent of movement-related suppression of the detection of weak electrical stimuli (intensity, 90% detected at rest) was determined in 118 experiments carried out in 47 human subjects. Subjects were trained to perform a rapid abduction of the right index finger (D2) in response to a visual cue. Stimulus timing was calculated relative to the onset of movement and the onset of electromyographic (EMG) activity. Electrical stimulation was delivered to 10 different sites on the body, including sites on the limb performing the movement (D2, D5, hand, forearm and arm) as well as several distant sites (contralateral arm, ipsilateral leg). Detection of stimuli applied to the moving digit diminished significantly and in a time-dependent manner, with the first significant decrease occurring 120 ms before movement onset and 70 ms before the onset of EMG activity. Movement-related and time-dependent effects were obtained at all stimulation sites on the homolateral arm as well as the adjacent trunk. A pronounced spatiotemporal gradient was observed: the magnitude of the movement-related decrease in detectability was greatest and earliest at sites closest to the moving finger and progressively weaker and later at more proximal sites. When stimuli were applied to the distant sites, only a small (~10%), non-time–dependent decrease was observed during movement trials. A simple model of perceptual performance adequately described the results, providing insight into the distribution of movement-related inhibitory controls within the CNS.

INTRODUCTION

The CNS has a variety of mechanisms at its disposal that modulate the quantity and quality of the sensory information that it processes. One such mechanism, gain control, enhances or diminishes sensory feedback. Several forms of gain control are found in the somatosensory system, including direct controls over receptor sensitivity (fusimotor control of muscle spindle sensitivity) and controls over the transmission of somatosensory signals within the CNS. An example of the latter is the suppression or "gating" of the transmission of cutaneous signals seen in association with voluntary movement. Single-unit and evoked-potential studies have demonstrated that the transmission of cutaneous signals through the dorsal column-medial lemniscal pathway to primary somatosensory cortex is decreased during movement (e.g., Chapman et al. 1988; Ghez and Lenzi 1971; Rushton et al. 1981; see Chapman 1994 for a recent review). Movement-related gating also exerts powerful influences on perception. Thus the detection of near-threshold cutaneous stimuli is decreased during movement (Coquery et al. 1971; Dyhre-Poulsen 1978), and detection threshold is elevated correspondingly (Chapman et al. 1987; Post et al. 1994). Magnitude estimates of clearly supra threshold innocuous stimuli, including vibrotactile stimuli, also are diminished during movement, although relative differences are preserved and so discrimination thresholds are unchanged (Chapman et al. 1987; Milne et al. 1988; Post et al. 1994).

Knowledge of the *timing* of movement-related decreases in transmission and perception provides insight into the source of the gating influences. Modulation that precedes the onset of movement and electromyographic (EMG) activity generally is interpreted as evidence that central signals, related to the preparation and execution of the movement, play a role in the phenomenon (Chapman et al. 1988; Coulter 1974; Dyhre-Poulsen 1978; Ghez and Lenzi 1971; Jiang et al. 1990b). Peripheral feedback, generated during movement execution, also plays an important role in the modulation that follows movement onset (e.g., Chapman et al. 1988; Huttunen and Hömberg 1991; Jones et al. 1988). Estimates of the time of onset of gating influences from cortical somatosensory evoked-potential (SEP) studies and psychophysical studies vary widely. Some studies observed modulation 60–100 ms before the onset of EMG activity and so well before movement onset, whereas other studies reported modulation only after the onset of EMG and movement (Chapman et al. 1988; Cohen and Starr 1987; Coquery et al. 1971; Dyhre-Poulsen 1978; Jiang et al. 1990b). These different results may arise from a number of factors, including differences in the intensity of the stimulus, differences in the spatial relation between the stimulus and the movement, and differences in the motor tasks, including the movement studied (digit vs. elbow) as well as the associated movement kinematics (Chapman et al. 1988; Ghez and Pisa 1972; Rauch et al. 1985).

Insight into the mechanisms underlying movement-related decreases in transmission and perception also comes from studies of the modulation's *spatial extent,* which defines the distribution of the gating influences. There is limited evidence that proximity is an important factor in determining the magnitude of the gating influence on cortical SEPs in humans (Rushton et al. 1981; Tapia et al. 1987). In monkeys, on the other hand, the gating effects have been described as widespread and nonspecific (Jiang et al. 1990b). The latter contrasts with the topographically organized gating influences elicited by intracortical microstimulation ap-

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plied to motor cortex (area 4), a potential source of central gating signals: Jiang et al. (1990a) reported that these influences show a proximal to distal gradient whereby the modulation is directed toward cutaneous inputs from the skin either overlying or distal to the motor output. Several psychophysical studies have examined the spatial extent of the gating influences on perception (Coquery et al. 1971; Post et al. 1994; Schmidt et al. 1990), and all reported that proximity between the site of stimulation and the movement was an important factor with sites closer to the movement showing a greater modulation. In contrast to the results of Jiang et al. (1990a), however, Coquery et al. (1971) reported that magnitude estimates were decreased at sites located both distal and also proximal to the site of movement. The latter results were confounded potentially by differences in the associated movements because subjects were required to make a variety of movements (digits, wrist, elbow, and shoulder). Furthermore, potential differences in the time course of the gating influences at the different sites may have contributed to the results.

Differences in stimulus intensity, stimulus location, and the motor task probably account for much of the variation seen in the literature. Clearly further experiments are warranted to define more precisely the influence of each of these variables. The purpose of this study was to examine in detail the importance of location on the time course and spatial extent of movement-related decreases in the ability to perceive weak, near-threshold cutaneous stimuli. In this study, the time course was established both in relation to the onset of movement and the onset of EMG activity for a discrete and relatively invariant motor task, abduction of the index finger (D2). A single clearly defined stimulus intensity was used to test perceptual performance at several stimulation sites both close and distant to the body part in motion. The results presented here form part of a larger investigation aimed at quantifying the influence of the various factors that may account for previously reported variations in the distribution, timing, and amplitude of movement-related decreases in tactile perception. A preliminary account of these results has been published (Williams and Chapman 1996).

METHODS

Subjects

A total of 47 naive, paid volunteers (25 males and 22 females, ages 15–30 yr) participated in this study. Four subjects were left handed for writing and 43 were right handed. The experimental protocol was approved by the institutional ethics committee, and all subjects or their legal guardian gave their informed consent before participating in the study. Data from each subject were gathered in one or two sessions lasting 1–3 h each. At the beginning of each session, subjects received verbal instructions about the motor and perceptual tasks that they were to perform. This was followed by a small block of practice trials. In brief, the motor task consisted of an active abduction of the right index finger (D2), whereas the perceptual task consisted of determining whether or not a weak near-threshold electrical stimulus had been delivered to the skin. One stimulation site was investigated in each experiment, and two to three different sites were tested in each experimental session. Altogether, 118 experiments were performed, using 10 different stimulation sites mainly located on the right arm.

Motor task

A simple reaction time (RT) task was employed, whereby subjects made a rapid abduction of the right D2 as soon as possible after the appearance of a visual "GO" cue (illumination of a 3 × 3 array of LEDs placed at eye level, 1 m in front of the subject). As shown in Fig. 1*A*, subjects were seated in a chair beside a small table on which the right arm rested. The index finger rested on a small pivoting plate (1.8 × 10 cm) the base of which was aligned with the axis of rotation of the D2 metacarpophalangeal joint (Fig. 1*B*). The subjects were asked to relax with D2 in a neutral position. An oscilloscope was used to display D2 position (Fig. 1*A*) along with two reference lines corresponding to the starting (neutral) position and to the minimal required amplitude of movement (15°). No maximal movement amplitude was specified, but subjects were instructed to avoid hitting the stop situated 45° from the start position. Subjects also were requested to limit muscle contraction to the relevant agonist muscle (1st dorsal interosseous, 1st DI) during the initial phase of the movement to the extent that this was possible.

Perceptual task

Subjects were asked to report whether or not they detected the occurrence of weak electrical stimuli delivered to a site on the skin (see further) under two experimental conditions: at rest and while performing the motor task. No information regarding the proportion of trials with and without a stimulus was given to the subjects. No feedback was given with regard to the accuracy of the subject's perceptual judgements. The stimulus consisted of a single 2-ms square wave pulse generated by a Grass S88 stimulator and delivered via a SIU7 constant current photoelectric stimulation isolation unit. The stimulus was applied to the skin via surface electrodes (7 mm diam) spaced 30 mm apart, and in all cases only a localized sensation was produced. Before applying the electrodes, skin resistance was minimized and electrode adhesion maximized by vigorous cleansing of the area immediately beneath the electrodes with 70% alcohol. For experiments involving stimulation of a digit, the electrodes were not in contact with the supporting surface but were recessed in a specially designed cavity in the foam padding. At the beginning of each experiment (subject at rest), a modified sequential tracking procedure using six observations per stimulus intensity tested (as described by Wetherhill and Levitt 1965) was used to find the stimulus intensity level at which ~90% of the stimuli were detected. This intensity (range 0.18–1.32 mA) then was used throughout the experiment.

Stimulation sites

Ten stimulation sites were employed (Fig. 2). Six sites were situated on the arm ipsilateral to the moving digit: the glabrous skin of the middle and distal phalanges of digits 2 (iD2) and 5 (iD5), the dorsum of the hand (iHA), the mid-forearm (iFA), the lower arm (iA), and the shoulder (iSH). The remaining sites were one on the ipsilateral pectoral girdle just below the sternocleidomastoid joint (iPG), two sites on the contralateral arm (cSH and cD2), and one on the ipsilateral thigh (iTH). Three subjects were tested at 8 stimulation sites, 6 at 7 sites, 1 at 6 sites, 5 at 3 sites, and 31 at 1 site (iD2). Those subjects tested at three or fewer sites participated in other experiments, not reported in this paper, which will form the basis of other publications. The order of testing for the different stimulation sites in each subject was randomly determined. The exact number of subjects tested at each site is given in Fig. 2.

The distance between the various stimulation sites and the body

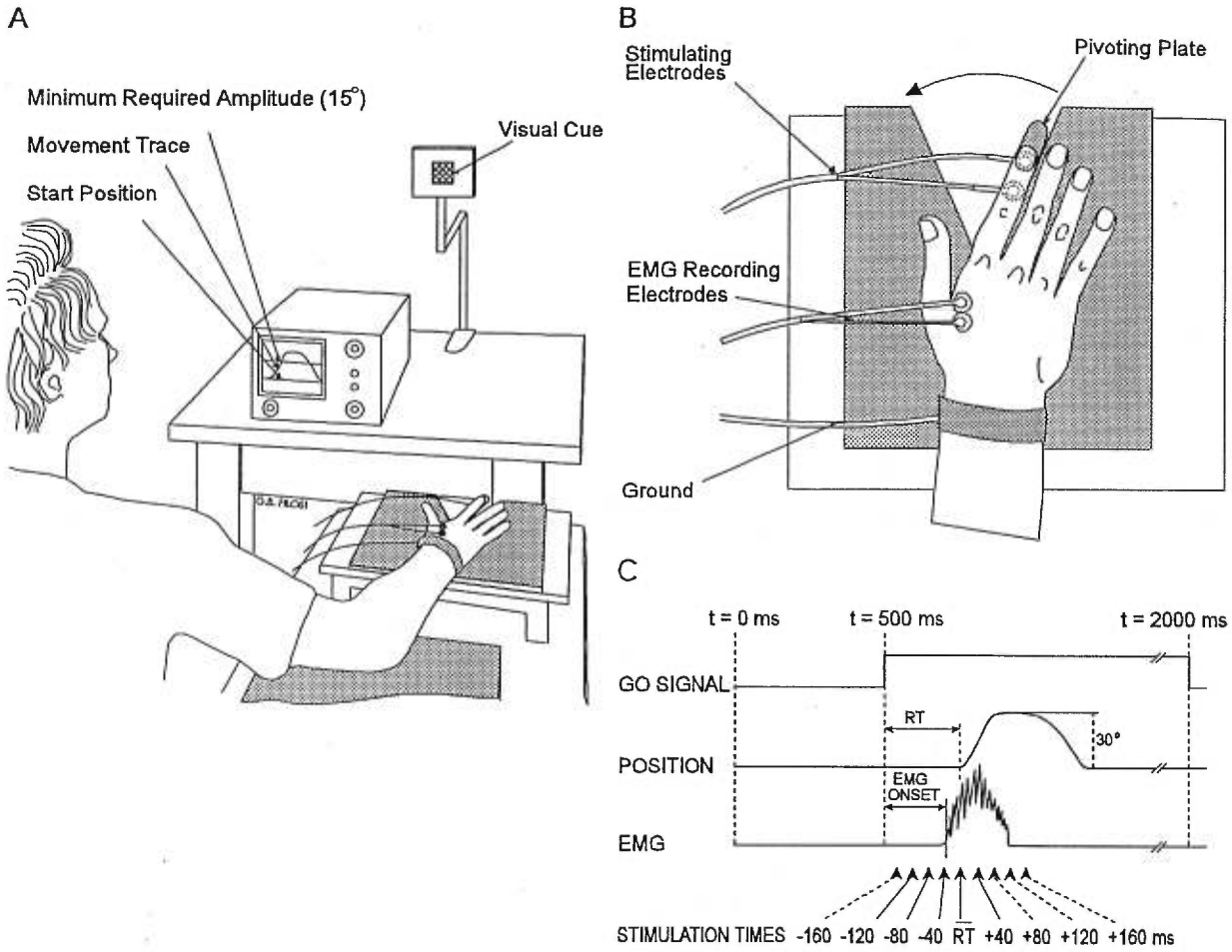


FIG. 1. *A*: experimental set-up showing the subject's position facing the visual cue and the oscilloscope that displayed movement-related information. *B*: close-up of hand showing the pivoting plate on which the right index finger rested (here in initial rest position). Shown also are the positions of the stimulating electrodes applied to the index finger and the electromyographic (EMG) recording electrodes over the 1st dorsal interosseous muscle. *C*: time course of events in movement + stimulation trials. Stimuli were applied at one of 5 (solid lines) or 9 delays, encompassing the time of EMG onset and the reaction time (RT).

part in motion (iD2) also is reported in Fig. 2. Distances are shown as a proportion of subject height and were estimated to the nearest 0.025 using standard anthropometric tables (Contini 1972; NASA 1978). Distances were estimated from the metacarpophalangeal joint of iD2; iD2 itself was assigned a distance of 0, being the body part in motion.

Data collection

The data acquisition and task were under the control of a PDP 11/73 minicomputer. Each trial was initiated by the experimenter and lasted for 2 s. Three types of trials were presented. In the majority of trials (70%), subjects were invited to move and a stimulus was presented (movement + stimulation); these trials were used to gather data on the effects of movement on the perception of the test stimulus. In 20% of the trials, subjects were asked not to move during the trial, and the stimulus was applied at rest (rest + stimulation); these trials monitored the stability of perceptual performance at rest. Finally, catch trials (no stimulus) represented 10% of the total trials and were divided between the movement and no-movement trials; these trials evaluated each subject's rate of false positive responses. Subjects were not aware of the proportion of trials in which a stimulus was presented. Subjects were instructed verbally before the onset of each trial whether to move or not; no information was given as to whether or not there would be a stimulus, and the experimenter could not be seen by the subjects. Figure 1*C* schematizes the events occurring during a movement + stimulation trial. Data collection was initiated at *time 0*. The light signaling the subject to move was illuminated 500 ms after the beginning of the trial, allowing a period during which any spontaneous movement or EMG activity could be observed. The stimulus was given at one of five or nine delays relative to the onset of the GO cue (see further). At the end of each trial, subjects indicated verbally whether or not they had perceived a stimulus, and their response was entered into the computer by the experimenter and stored with the trial. The intertrial interval varied from 1 to 10 s. Subjects were observed carefully by the experimenter

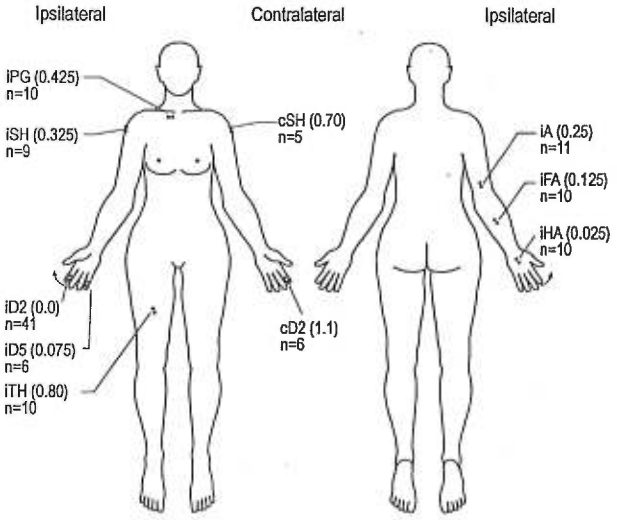


FIG. 2. Stimulation sites tested relative to the moving digit (iD2). Distance between each stimulation site and iD2 as a proportion of subject height is shown in parentheses. Number of subjects tested at each site also is indicated. A, arm; c, contralateral; D2, digit 2; D5, digit 5; HA, hand; i, ipsilateral; PG, pectoral girdle; SH, shoulder; TH, thigh.

during each trial. Any trial during which the subject produced movements unrelated to the motor task, as well as any trial after which the subjects reported inattention, discomfort, or lack of readiness was rejected and repeated later in the session.

To concentrate sampling around the onset of movement, each subject's mean RT, i.e., the mean difference between the time at which the GO signal was presented and the time of movement onset, was estimated from a series of practice trials performed at the beginning of the session. Using this value, five delays relative to the GO cue were calculated: RT − 120 ms, RT − 80 ms, RT − 40 ms, RT, and RT + 40 ms. Four additional delays (RT − 160 ms, RT + 80 ms, RT + 120 ms, and RT + 160 ms) were used in 10 experiments at the iD2 site. Natural trial-to-trial variations in RT, combined with the use of multiple stimulus presentation delays, resulted in the collection of an adequate sample of perceptual ability during a 200-ms time interval, which included EMG onset and movement onset. Because of variation in RT, trial-by-trial delays between the onset of peripheral movement-related activity and stimulus delivery were not known to either the subject or the experimenter at the time of the experiment. Data were collected in blocks of ≥22 trials, one stimulus delay being tested in each block, with the order of delay testing being varied between experiments. Within a block, ≥15 movement + stimulation trials, 5 rest + stimulation trials, and 2 catch trials were performed. The different types of trials were intermixed randomly, the order of presentation varying from block to block. Thus at each stimulation site, a minimum of 110 trials were recorded.

Angular displacement of D2 was measured using a potentiometer integrated into the plate that supported the finger. The EMG activity of 1st DI was recorded via 4-mm-diam surface electrodes placed 15 mm apart (center to center) on the skin overlying the muscle. In a few subjects, the EMG activity of forearm extensors ($n = 3$), elbow extensors (triceps brachii, $n = 6$), and the muscles of the hypothenar eminence ($n = 1$) also was recorded to evaluate the extent of coactivation present during the motor task. EMG activity was amplified and filtered with a band-pass width of 100–3,000 Hz, then full wave rectified and integrated during 5 ms. Both D2 position and EMG activity were digitized at 200 Hz and stored for later off-line analysis.

Data analysis

For each movement trial, the onset of EMG was determined visually. Several timing values were calculated off-line automatically, including RT (determined by an algorithm that found the first of 5 consecutive position samples that changed in the same direction), the lead time between 1st DI EMG onset and movement onset, and movement duration. Kinematic parameters also were calculated, including movement amplitude, peak velocity (computed by 3-point numerical differentiation and appropriate digital filtering of the displacement trace), and peak acceleration. Approximately 1% of the trials were discarded at this stage, usually because EMG activity was observed in the 500-ms interval before the GO signal. The presence of a causal relationship between EMG and movement was assessed by performing linear regression analyses between EMG onset and movement onset. The rationale for this test is that EMG activity responsible for the movement should not only precede the onset of movement but also show a high degree of correlation with RT with a slope approaching 1 (see e.g., Chapman et al. 1986). The possibility of significant intersite differences in the temporal and kinematic parameters was evaluated by calculating individual subject averages for each parameter and then comparing across the stimulation sites using one-way analyses of variance (ANOVA, level of significance $P < 0.05$).

Data from different sites were gathered from different, but not completely independent, groups of subjects. To determine whether the data from different sites could be treated as independent, the effects of intersubject differences in performance on the interpretation of the data were quantified. For this, trials from a given subject and site were grouped into 40-ms bins relative to either movement or EMG onset, and the proportion of stimuli perceived was calculated for each bin. Analysis of covariance (ANCOVA) examined the relative importance of site, timing, and subject on perceptual performance. This analysis showed that <5% of the total variation in the data could be explained by intersubject differences in performance, whether or not the significant effects of both timing and site were taken into account. As such, the data from different sites were treated as independent in the ensuing analyses.

All trials from a given stimulation site were pooled for further analyses. The proportion of stimuli that were perceived while performing each trial type was calculated for each stimulation site. ANOVAs were used to examine whether or not the stimulation site influenced perceptual performance for each type of trial. A Fischer one-tailed exact probability test for a 2 × 2 contingency table (level of significance, $P < 0.01$) was used to compare the average proportion of stimuli perceived at rest to the average proportion of stimuli perceived during trials involving movement. The latter test was used in all subsequent proportion comparisons.

Because performance during movement was to be compared with performance at rest, it was important that perceptual performance during immobile trials be constant throughout the experimental sessions. To verify this, the first and last 20% of immobile trials performed at each stimulation site were grouped, and performance was compared.

To study the time course of any variation in perception during movement, trials occurring within 300 ms of movement or EMG onset were grouped into 20-ms bins relative to either movement or EMG onset at each stimulation site. The proportion of stimuli perceived was calculated for each bin along with the 95% confidence interval. These proportions then were compared with the proportion of stimuli perceived during the corresponding immobile trials. To provide an adequate description of the temporal evolution of perceptual abilities relative to movement and EMG onset, linear

and logistic functions (APPENDIX A) were fitted to the pooled data from each stimulation site (Matlab, version 4.2, The Mathworks). The fitting algorithm attempted to minimize total squared error between the descriptive function and the data points. The goodness of fit of the linear and logistic descriptions was evaluated by comparing the total squared error between the model and actual data to the appropriate χ^2 distribution [df = number of points that defined perceptual performance at the site (number of parameters in the fitting equation + 1)]. The best fitting model (linear or logistic) was retained if it provided an adequate description of the data, i.e., if the probability of obtaining the observed amount of total squared error was >0.05. If a linear model was retained, the presence of a slope significantly different from zero was evaluated using a *t*-test. If a logistic descriptor was retained, four parameters then were determined: the maximum predicted perceptual performance, the minimum predicted perceptual performance, the peak slope (measure of the peak rate of decrease in perceptual performance), and the timing of the peak slope (the time at which perceptual performance decreased most rapidly). The effects of distance on these parameters were evaluated using adjusted correlation coefficients and *F* tests.

Bias

Our experimental approach was designed to minimize the duration of each experiment, so minimizing drift in performance and fatigue (recognizing that there was a motor component to the task), and to maximize the collection of data by presenting a relatively high proportion of trials with a stimulus. Positive bias was monitored continuously throughout each experiment using catch trials (Green and Swets 1988). The existence of systematic and significant positive or negative biases induced by the method or the experimenter was further evaluated in experiments that compared detection performance obtained with the main experimental method to detection performance obtained using a bias-free two alternative forced-choice (2AFC) procedure (3 subjects: 2 females, 1 male; 2 of 3 right handed). Stimuli were delivered to the iD2 site. Both experimental methods were tested in a single session, ensuring that stimulating conditions were similar. The main experimental method was as described above. For the 2AFC procedure, in each "trial," the GO cue was presented twice, i.e., there were two observation periods per trial, separated by a delay of ~1 s. Observation period duration, stimulus presentation delays, etc., remained the same. The stimulus was assigned randomly to one of the GO cue presentations in each trial: 50% of the trials contained the stimulus in the first interval and 50% in the second interval. At the end of each trial, the subject was asked to report in which of two intervals a stimulus was presented. Immobile trials were again interspersed between the movement trials to provide for a continuous monitoring of performance at rest.

The results obtained using both procedures were analyzed as described above, and are shown in Fig. 3. With both methods, detection performance declined rapidly ~40–60 ms before movement onset or 10–20 ms before EMG onset. During movement, performance was close to chance using the 2AFC method (0.52–0.53) and complete nondetection using the main method (0–0.01). To compare the results, the detection performance data obtained using the main experimental procedure were transformed in the following manner: $p'(i) = [p(i) + 1]/2$, where $p(i)$ = the proportion of stimuli perceived in a given time interval, and a logistic function was fitted to the resulting data. No significant differences were observed when this logistic function was compared with that describing the 2AFC data ($P > 0.05$, Kolmogorov-Smirnov statistic), making it unlikely that experimenter- or procedure-induced biases played a significant role in the results obtained with the main experimental procedure.

RESULTS

Performance of the movement task

A total of 118 experiments were performed in 47 subjects (Fig. 2). Figure 4 shows an example of the movement traces and EMG records from one subject. First dorsal interosseus EMG activity, the principal agonist, consistently preceded movement onset by an average of 27 ms in this example (Fig. 4, *A* and *B*) and showed a strong linear correlation with movement onset (Fig. 4*C*). Cocontraction was evident in abductor digiti minimi (Fig. 4, *A*, *B*, and *D*), but the activity was weaker (note the change in scale), more irregular, and always followed the onset of 1st DI EMG activity (average delay, 10 ms). At the forearm extensor site, EMG activity was weak, frequently absent, and followed movement onset. In 118 experiments, the mean correlation coefficient between 1st DI and movement onset was 0.96 ± 0.02 (mean ± SE) and the mean slope of the linear regression line was 0.95 ± 0.04. First DI EMG activity led movement onset by 50 ± 14 ms (Table 1).

Before examining site-related differences in perceptual performance, the possibility that the movements themselves might have varied as a function of the site of stimulation was evaluated using one-way ANOVAs applied to the various temporal and kinematic parameters (Table 1). All but one measure, mean peak amplitude, showed no significant change across the 10 stimulation sites. With regard to movement amplitude, post hoc Scheffé analyses showed that only a few intersite comparisons were significantly different [iD2 and iD5 (smallest movement amplitudes) vs. cD2, cSH, and iSH (largest movement amplitudes)]. The effect of this difference on the results was minimal because the sampling period did not include the time at which movement reached its peak amplitude.

Finally, regression analyses showed that stimulus delay had no effect on RT. The average correlation coefficient between stimulus delay and RT at all sites was close to zero (0.005).

Performance of the perceptual task

Table 2 summarizes the global performance of all subjects in the three trial types. Overall, pooled data showed that subjects perceived 94% of the stimuli presented at rest. A one-way ANOVA failed to discern any significant difference in pooled perceptual performance at rest across the experimental test sites, thus providing a constant baseline against which performance during movement could be evaluated. Of 1,479 catch trials (pooled data), only 12 false positive responses were noted (0.8%), indicating that subjects used a very conservative response strategy throughout the series of experiments. As detailed in the legend for Table 2, no stimulation site was associated with significantly more false positives than any other. Pooled data showed that subjects perceived 58% of the stimuli presented during movement + stimulation trials, and a one-way ANOVA (Table 2) showed that there was significant intersite variability in pooled perceptual performance for these trials. Inspection of Table 2 reveals that sites closer to the body part in motion, iD2, showed greater reductions in the proportion of stimuli perceived during movement

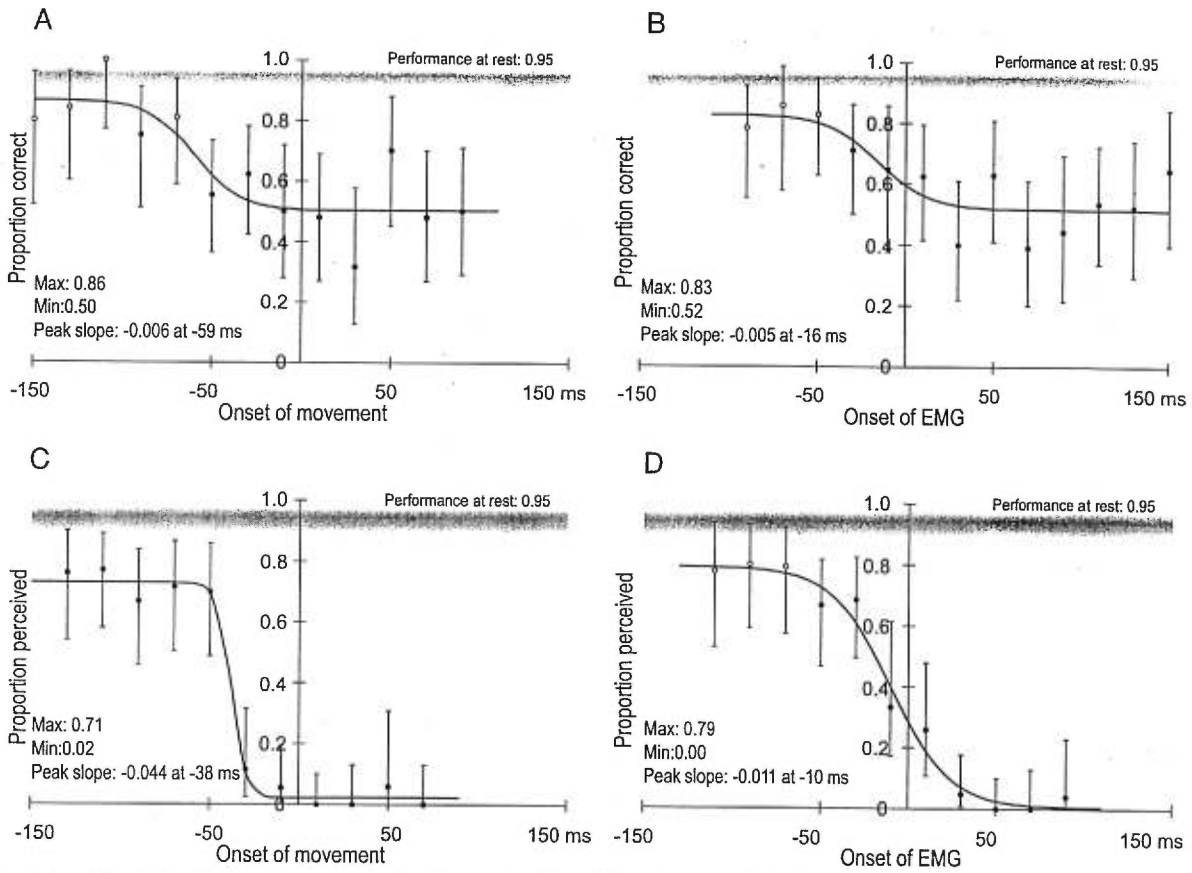


FIG. 3. *A–D*: effects of iD2 abduction on the detection of stimuli applied to the moving digit in 3 subjects using a 2 alternative forced-choice (2AFC) version of the experiment (*A* and *B*) and using the main experimental procedure (*C* and *D*). Perceptual performance over time is plotted relative to movement onset (*A* and *C*) and the onset of 1st DI EMG (*B* and *D*). Error bars represent the 95% confidence intervals. The shaded area shows the 95% confidence interval for perceptual performance at rest. For all bins (20 ms), ●, perceptual performance during movement + stimulation trials was significantly lower than that observed at rest ($P < 0.01$); ○, no change.

than did those that were more distant from the movement site. Comparisons between performance in the rest + stimulation trials and the movement + stimulation trials showed that significantly fewer stimuli were perceived during movement at all 10 stimulation sites ($P < 0.001$, Fischer exact tests for a 2 × 2 contingency table).

Variation over time of perceptual performance at rest

For each stimulation site, perceptual performance in the first 20% of immobile trials was compared with perceptual performance in the last 20% of immobile trials. No significant differences were observed at any of the sites ($P > 0.01$, Fischer exact probability tests). For data from all sites pooled together, the proportion of stimuli perceived in the first 20% of immobile trials was 0.95, whereas performance in the last 20% was 0.93.

Time-dependent change in the detection of stimuli applied to the moving digit (iD2)

Figure 5 shows that the effects of iD2 movement on the ability of 41 subjects to detect stimuli applied to the moving digit were not uniform over time. Although modest but significant decreases in the proportion of stimuli perceived were observed ≤200 ms before the onset of movement (Fig. 5*A*), performance declined precipitously beginning 60–80 ms before movement onset so that practically no stimuli were perceived after the onset of movement. A logistic function was found to best fit the data. A number of parameters were calculated from the logistic function (see METHODS), and these are detailed in Fig. 5*A*. In particular, the time of the peak decrease in perceptual performance preceded movement onset by 50 ms.

When the same data were replotted in relation to 1st DI EMG onset (Fig. 5*B*), a similar pattern was observed, but the peak decrease in perceptual performance now occurred 8 ms before EMG onset, in effect coinciding with the onset of agonist EMG given the temporal resolution of the analysis (20-ms bins). The other parameters of the logistic function were essentially the same. Thus the major change was an ~40- to 50-ms shift in the data points, corresponding to the lead time between EMG onset and movement onset. Given the similarity in the results, perceptual performance is only

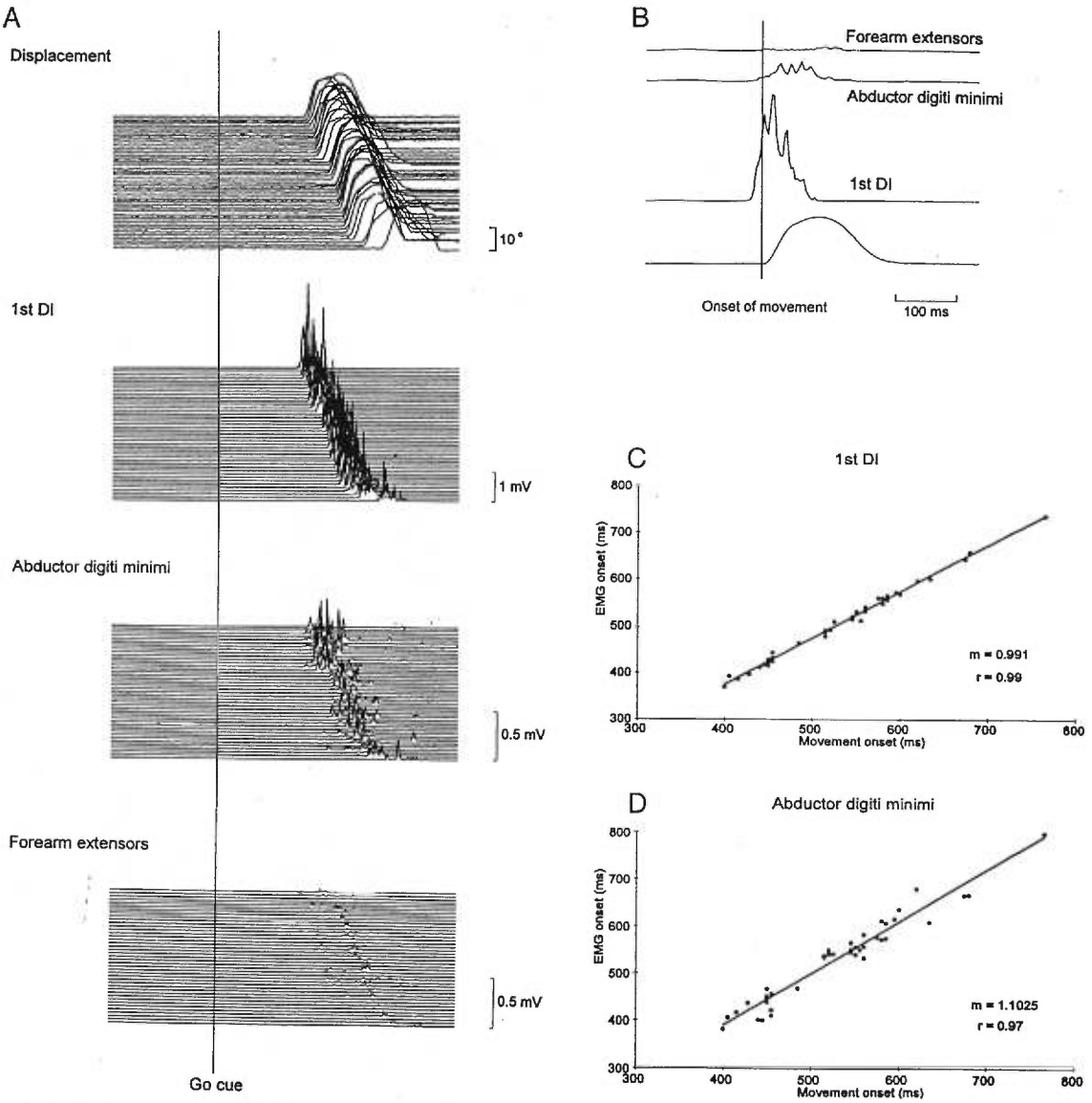


FIG. 4. *A*: sample movement and EMG traces for 1st dorsal interosseous (1st DI), abductor digiti minimi, and the forearm extensors aligned on the onset of the GO cue. *B*: averaged movement and EMG traces for the same muscles, aligned on the onset of movement (same scales as *A*). *C* and *D*: scatter plots of EMG onset as a function of RT for 1st DI (SE of the residual, 1.0 ms; regression constants: $m = 0.99$, $b = -21$ ms) (*C*) and abductor digiti minimi (SE of the residual, 3.5 ms; regression constants: $m = 1.10$, $b = -51$ ms; *D*). Linear regression lines are shown along with the slope (m) and the correlation coefficient (r).

plotted against the onset of 1st DI EMG for the nine other stimulation sites tested in this study.

Effects of changing the site of stimulation on the movement-related decrease in tactile detection

Figure 6 summarizes the results obtained at the six other stimulation sites located on, or adjacent to, the ipsilateral arm, in increasing order of distance from the moving digit (Fig. 6, *A*–*F*). All six sites showed time-dependent modifications in perceptual performance that were best described by logistic functions. Two major observations were made. First, the depth of modulation of tactile detection, reflected in logistic function minima, lessened as the distance between the stimulation site and the site of movement increased. Second, the timing of the peak decrease in perceptual performance shifted as distance increased, from just around EMG onset for the sites immediately adjacent to iD2 (Fig. 6, *A*–*C*) to well after EMG onset for the sites located on the proximal arm and adjacent trunk (Fig. 6, *D*–*F*).

TABLE 1. *Temporal and kinematic parameters describing the performance of the motor task, iD2 abduction and the results of ANOVAs comparing values across 10 stimulation sites*

Movement-Related Parameter	Mean Response	ANOVA*	
		F	*P*
Temporal			
RT	255 ± 45 ms	0.85	0.57
1st DI EMG onset	205 ± 43 ms	0.97	0.47
EMG lead time	50 ± 14 ms	0.80	0.62
Movement duration	210 ± 75 ms	0.48	0.89
Kinematic			
Peak amplitude	30 ± 8°	2.12	0.03
Peak velocity	360 ± 100°/s	0.60	0.79
Peak acceleration	6,500 ± 1,500°/s²	0.76	0.65

Results for 118 experiments and 47 subjects. iD2, ipsilateral digit two; ANOVA, analysis of variance; RT, reaction time; 1st DI, first dorsal interosseous; EMG, electromyographic. * df = 9,108.

To quantify the effect of distance, logistic function parameters from the seven stimulation sites closest to the body part in motion were plotted as a function of distance from the body part in motion (Fig. 7, *A–D*). Site-dependent trends were described using the best-fit linear or logistic functions and evaluated using adjusted correlation coefficients (see legend, Fig. 7). A strong positive correlation between the timing of the peak decrease in perceptual performance and distance from the site of movement was observed (Fig. 7*A*). A strong positive correlation between the minimum predicted perceptual performance and distance also was obtained (Fig. 7*B*). In contrast, the peak slope and maximum predicted perceptual performance values did not show significant trends with increasing distance (Fig. 7, *C* and *D*).

A different pattern of perceptual modulation was observed at the three more distant sites, cD2, cSH, and iTH (Fig. 8, *A–C*). Even though overall performace during movement was decreased significantly with regard to performance at

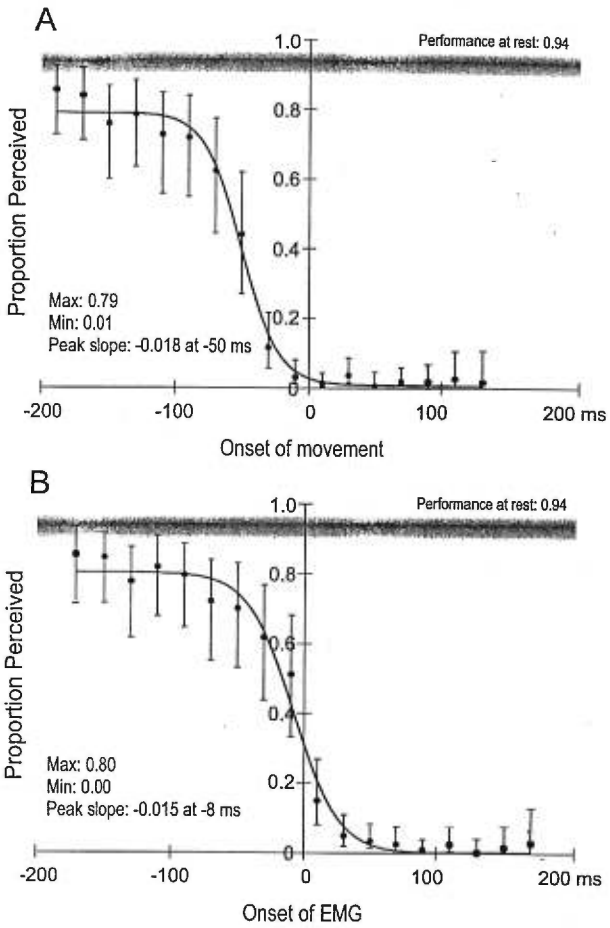
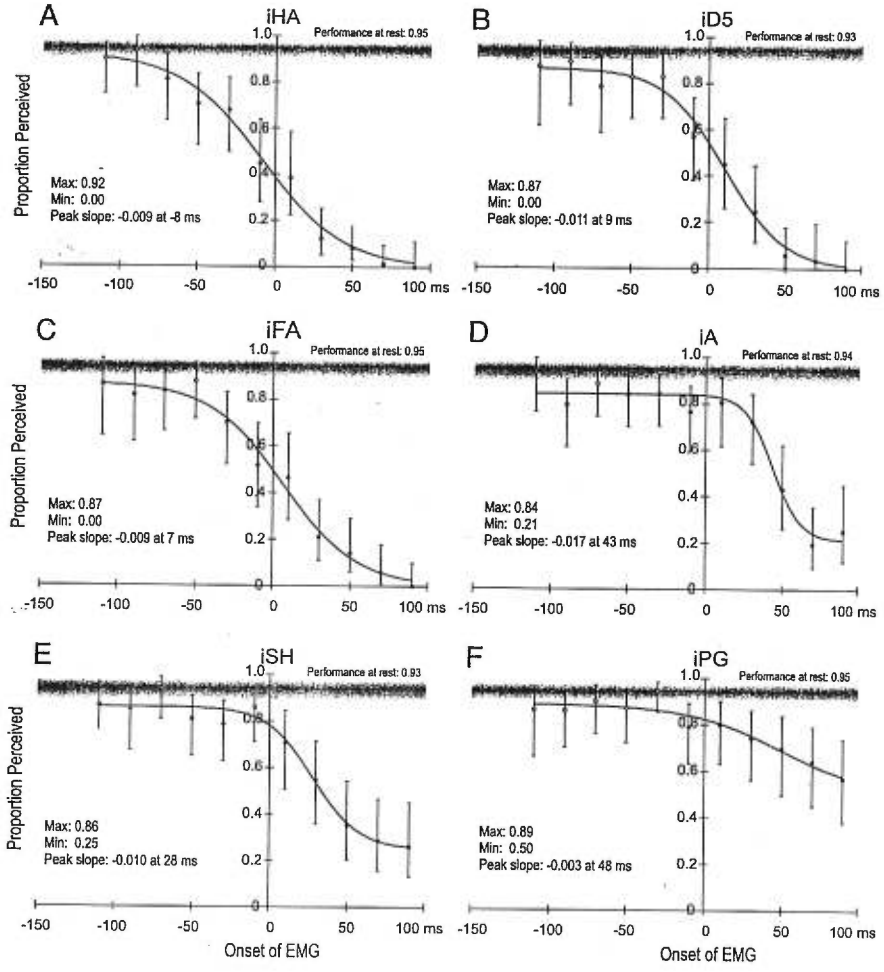


FIG. 5. *A* and *B*: effects of iD2 abduction on the detection of stimuli applied to the moving digit in 41 subjects. Perceptual performance over time is plotted relative to movement onset (*A*) and the onset of 1st DI EMG (*B*). Plotted as in Fig. 3.

TABLE 2. *Proportion of stimuli detected in the three trial types as a function of the site of stimulation*

Stimulation Site	Proportion of Positive Responses		
	Rest + Stimulation*	Movement + Stimulation*	Catch Trials*
i Digit 2	0.94 (0.93, 0.95)	0.40 (0.38, 0.41)†	0.00 (0.00, 0.03)
i Hand	0.95 (0.91, 0.97)	0.39 (0.35, 0.42)†	0.00 (0.00, 0.04)
i Digit 5	0.93 (0.87, 0.96)	0.45 (0.41, 0.50)†	0.00 (0.00, 0.07)
i Forearm	0.95 (0.92, 0.97)	0.44 (0.40, 0.47)†	0.01 (0.00, 0.07)
i Arm	0.94 (0.90, 0.96)	0.64 (0.60, 0.67)†	0.00 (0.00, 0.04)
i Shoulder	0.93 (0.89, 0.96)	0.60 (0.57, 0.64)†	0.00 (0.00, 0.04)
i Pectoral Girdle	0.93 (0.89, 0.96)	0.78 (0.74, 0.81)†	0.02 (0.00, 0.08)
i Thigh	0.96 (0.92, 0.97)	0.88 (0.85, 0.90)†	0.01 (0.00, 0.04)
c Shoulder	0.96 (0.89, 0.98)	0.83 (0.78, 0.86)†	0.00 (0.00, 0.08)
c Digit 2	0.95 (0.89, 0.97)	0.84 (0.80, 0.87)†	0.00 (0.00, 0.06)
Overall	0.94 (0.93, 0.95)	0.58 (0.57, 0.59)†	0.01 (0.00, 0.02)

Data for 118 experiments and 47 subjects (95% confidence intervals in parentheses). c, contralateral; i, ipsilateral. * One-way ANOVAs (df = 9, 108) showed that there was no intersite variation in perceptual performance during Rest + Stimulation trials ($F = 0.54$, $P = 0.84$) or Catch trials ($F = 0.89$, $P = 0.53$), but significant intersite variation was seen for Movement + Stimulation trials ($F = 37.9$, $P < 0.0001$). † Performance during Movement + Stimulation trials was significantly lower than performance during Rest + Stimulation trials at each stimulation site and overall (Fischer exact tests for a 2 × 2 contingency table, $P < 0.001$).



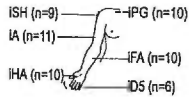


FIG. 6. *A–F*: effects of iD2 abduction on perceptual performance over time for the 6 ipsilateral limb stimulation sites. Data plotted relative to EMG onset as in Fig. 5*B*. At each stimulation site, there was a significant, time-dependent reduction in the proportion of stimuli perceived during movement trials (●: $P < 0.01$) as compared with at rest. As distance increased, peak reductions occurred later and the minimum proportion of stimuli perceived increased.

rest (Table 2) at each of these sites, no sustained decrease in perceptual performance was observed at any of these sites when the data were placed into 20-ms bins. Moreover, linear equations with slopes not significantly different from zero ($P > 0.05$) provided the best description of the data, indicating that there was no time-dependent change in performance. An ANCOVA showed that the site variable did not explain a significant portion of the variation present in the data from these sites (F test, $P = 0.30$). These data therefore were combined into a single "Distant" data set (Fig. 8*D*, 21 subjects). A slight but significant and sustained decrease in perceptual performance, consistent with the small overall reduction in perceptual performance at these sites (Table 2), now was observed. A linear equation with a slope of 0 and a y intercept of 0.85 (as compared with 0.96 at rest) provided the best description of the Distant data set. These findings suggested that there was a relatively small and non-time–dependent decrease in perceptual performance at these sites.

The non-time–dependent decrease in perceptual performance observed at the Distant sites also may have been present at the sites closer to the moving digit. ANCOVAs performed on the data from each site showed that the effect of timing on performance only became significant ($P < 0.05$) when data from bins later than −70 ms were included in the analyses. The effect of site on performance only became significant ($P < 0.05$) when data from bins later than −50 ms were included in the analyses. The data from the three earliest bins at each site were pooled, and significant differences with perceptual performance at rest were seen ($P < 0.05$) at all sites but iSH ($P = 0.09$). Performance observed over the initial three bins (60 ms) for the seven locations closest to iD2 was not significantly different from perceptual performance during the movement + stimulation trials at the Distant sites ($P > 0.05$, Fischer exact probability test). All of these results suggest that site- and timing-independent modulation was present at all sites, along with superimposed time- and location-dependent modulation at the seven closest sites. To provide a precise estimate of the time of onset of the time- and location-dependent effect at each of the seven locations closest to iD2, performance in each time bin at each of these sites was compared with perceptual performance in the same time bin for the Distant data set ($P < 0.05$). The results are summarized in Fig. 9. The

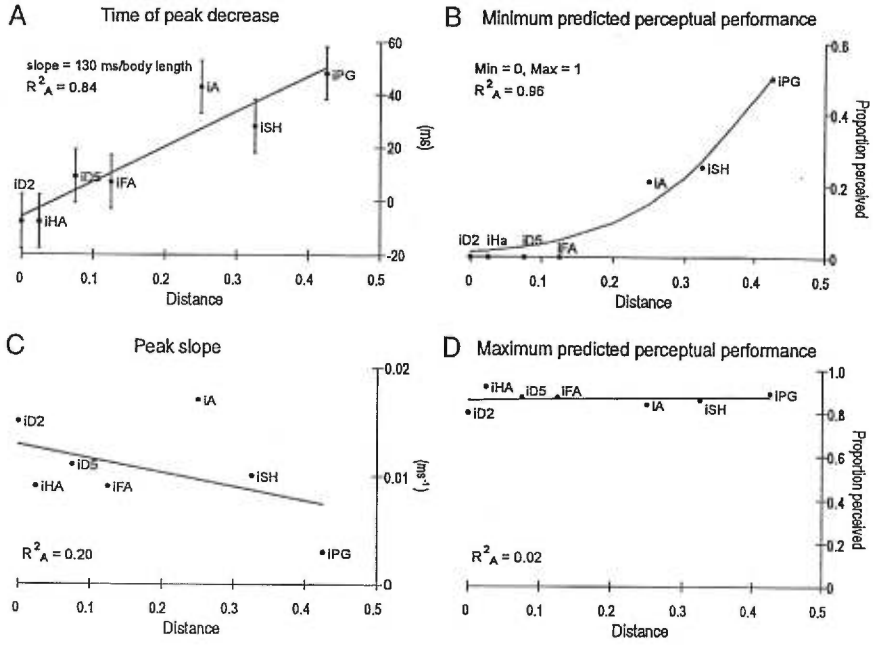
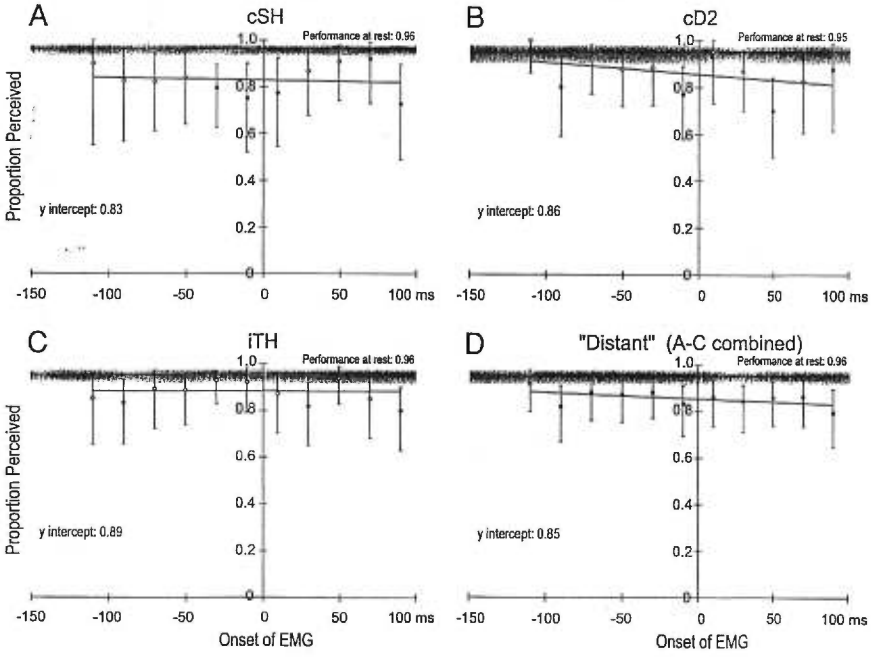


FIG. 7. A–D: logistic equation parameters plotted as a function of distance (proportion of body height) from the site of movement, iD2, for the 7 closest stimulation sites, all of which showed a time-dependent decease in perceptual performance. Both the timing of peak decreases in perceptual performance (A, ±10 ms) and the minimum predicted perceptual performance (B) showed strong positive correlations with distance. Maximum predicted perceptual performance varied very little with distance (D) and peak slope varied irregularly (C). R^2_A, coefficient of determination adjusted for the number of parameters in the fitted equation.

earliest sustained decrease (>3 consecutive bins) was at iD2 where performance was significantly decreased beginning in the bin centered 70 ms before EMG onset. The latest decrease was seen at iPG, 50 ms after EMG onset. These data were best described by a linear function with a positive slope significantly different from zero ($P < 0.05$), indicating that the time of onset of perceptual suppression increased significantly as the distance between the stimulation site and the moving digit (iD2) increased.

On the basis of the results of the preceding analyses, a model of perceptual performance during the motor task that incorporated the importance of stimulus timing and distance



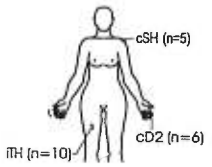


FIG. 8. A–C: effects of iD2 abduction on perceptual performance over time for the 3 most distant stimulation sites. At each site, performance was best described by a linear function with a slope not significantly different from zero ($P > 0.05$). D: data were pooled to generate a single "Distant" data set. Plotted as in Fig. 5B.

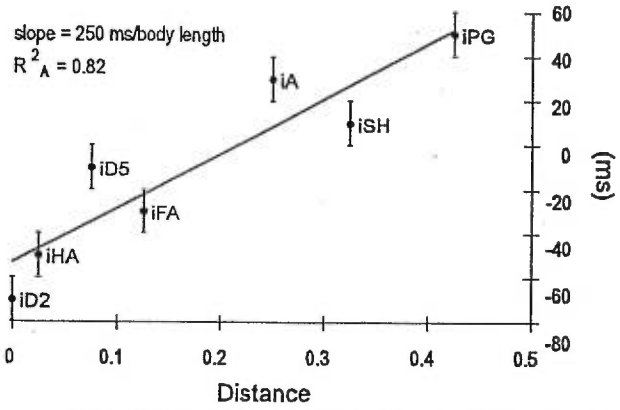


FIG. 9. Time of onset of the 1st significant time-dependent decrease in perceptual performance during movement trials (20-ms bins), measured relative to 1st DI EMG onset, as a function of the distance (proportion of body height) from the site of movement, iD2. See text for further description.

between the stimulation site and the body part in motion was created (Fig. 10, APPENDIX B). Data from the Distant sites were assigned a distance that corresponded to the average of the distances for each of the three sites (0.875). The perceptual data during the movement + stimulation trials then were used to define a surface, presented in Fig. 10*A*. This representation permitted a visualization of modifications in perceptual performance at all locations at any given time. A model surface (*Eq.* B5, APPENDIX B) then was fitted to the surface created by the perceptual data (Fig. 10*B*). The total squared error between the surface described by the perceptual data and the surface described by the model was 0.31, and the peak squared error was <0.03 (Fig. 10*C*). The maximum predicted perceptual performance for the model surface was 0.86. The minimum predicted perceptual performance was 0, although it increased sharply as distance increased; performance was constant at 0.86 at the Distant site. The timing of the peak decrease in performance varied with distance, increasing >85 ms from the iD2 stimulation site to the iPG site.

Relationship between movement parameters and perceptual performance

It has been reported that movement-related gating is a function of the kinematics of the movement, with faster movements producing larger gating effects (Angel and Malenka 1982; Chapman et al. 1988, 1996; Rauch et al. 1985). With this in mind, for each of the seven stimulation sites that showed a time-dependent decrease in the proportion of stimuli perceived, movement + stimulation trials were examined to see if modifications in perceptual performance could be related to variations in kinematic parameters (peak amplitude, peak velocity, peak acceleration). Two different analyses were performed. First, the entire data set was divided into two groups: trials in which stimulation was applied before the onset of EMG and trials in which stimulation was applied after the onset of EMG. For each group, the kinematic parameters were compared across trials in which the stimulus was, or was not, perceived (*t*-tests). No significant differences were found. Second, the analysis was repeated, this time using only trials in which a stimuli was delivered within 30 ms of the time of the peak decrease in perceptual performance. In effect, this served to minimize the confounding effect of the time-dependent variation in detection previously described by concentrating on the time period where changes in detection were occurring. As shown in Fig. 11, significant differences were now obtained (filled symbols), especially at the stimulation sites closest to the moving digit, iD2. At these sites, the kinematic parameters were consistently, and usually significantly, smaller when the stimulus was perceived as compared with when the stimulus was not perceived. Sites further away showed more variability in their kinematic relationships.

DISCUSSION

The results of the present study have shown that the movement-related decrease in detection of near-threshold cutaneous stimuli can precede movement onset and the onset of EMG activity. In addition, both the time course and amplitude of the modulation were dependent on the distance between the stimulation site and the body part in motion.

Methodological considerations

Electrical stimulation was used in this study for the following reasons. It had been shown previously that detection thresholds for electrical stimuli the parameters of which were identical to those used in this study are increased during movement (Chapman et al. 1987). In addition, electrical stimulation represented a reliable means of providing stimuli of identical duration and detectability to different body parts. The stimulating electrodes were placed at sites where stimulation affected mainly cutaneous afferents, although the electrodes on the finger sites also may have stimulated articular afferents traveling in the digital nerves. Subjects reported that the electrical stimuli did not feel particularly unnatural, just "extremely weak" at all stimulation sites. By calibrating stimulus intensity relative to a given detection level (~90% detected), baseline detection performance at rest was identical from site to site and subject to subject. This considerably facilitated intersite comparisons and permitted the pooling of data from multiple subjects. Finally, the inclusion of two contralateral sites identical to the ipsilateral sites made it possible to ensure that the observed distance-related effects could be distinguished from possible differences in the psychometric function at each stimulation site.

The movement performed in this study, abduction of the index finger, was chosen because it has only one major agonist (1st DI). Other intrinsic and extrinsic hand muscles showed relatively little co-contraction, and their activity followed 1st DI contraction (Fig. 4, *A* and *B*). First DI EMG activity thus represented the earliest peripheral response to the motor command, simplifying both data analysis and interpretation of the results.

Practice and fatigue effects did not appear to play a significant role on perceptual performance in these experiments. Perceptual performance at rest did not change between the beginning and the end of each experiment.

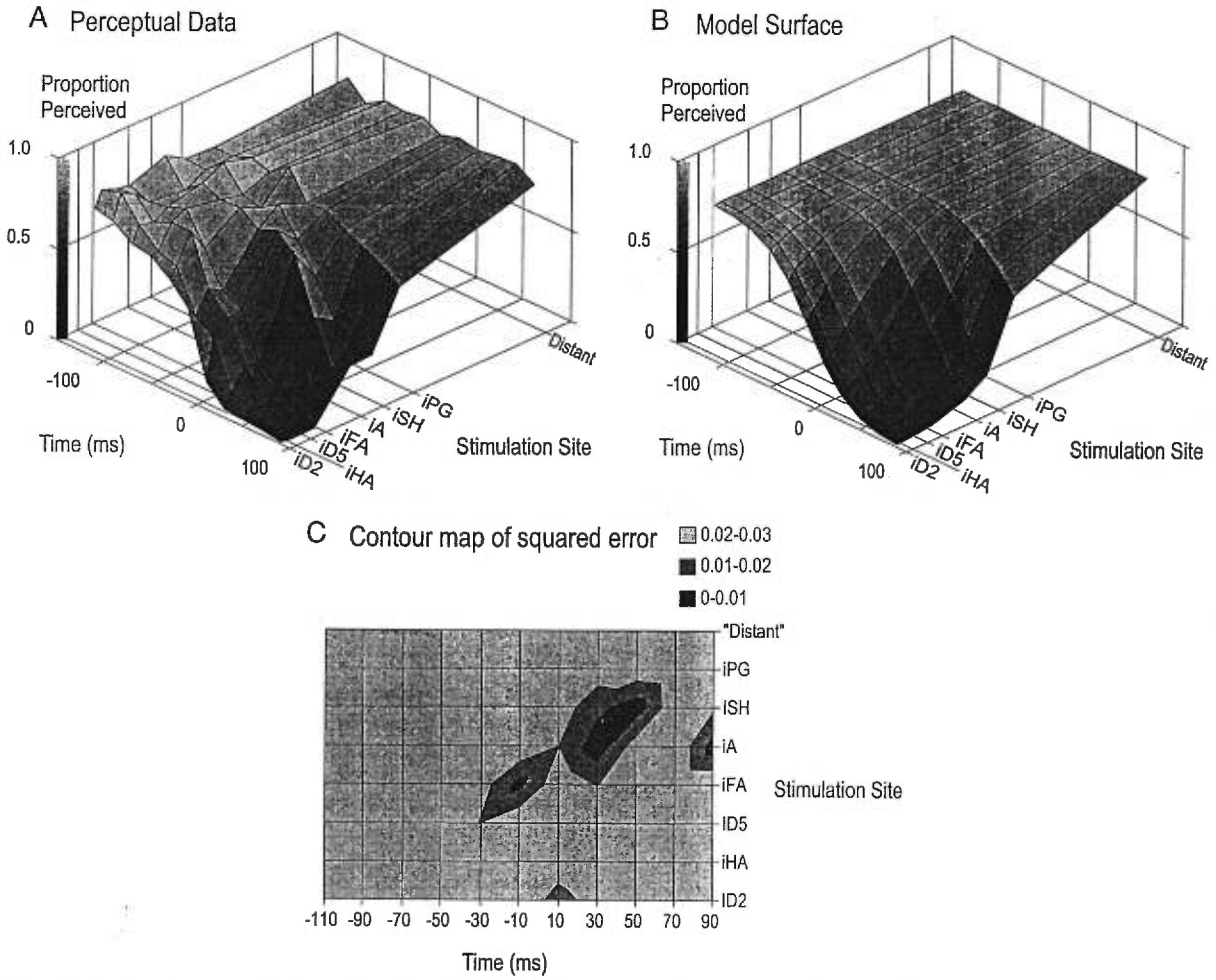


FIG. 10. *A–C*: surfaces showing time and location dependence of perceptual performance. *A*: pooled performance data during movement trials (z axis) plotted as a function of time (x axis), and distance (as a proportion of body height) between the moving digit and the site of stimulation (y axis). *B*: best-fit model surface for the data shown in *A*. Model surface was not significantly different from the actual performance data ($P > 0.05$). Model parameters are described in the text. *C*: contour map of the squared error between the surfaces plotted in *A* and *B*.

Experimenter- and method-induced biases do not appear to have played an important role in the results obtained in this study. In test subjects, the same results were obtained using a bias-free experimental strategy (Fig. 3). Moreover, the experimental design minimized experimenter-subject interaction and did not favor particular response strategies; also no feedback was given to the subjects on their performance. Subject strategy in the absence of feedback was almost optimal in terms of maximizing signal detection while minimizing false positives. The rate of false positive responses was always extremely low, a good indication that positive bias was not a significant factor in the experimental results. Negative bias at rest was also extremely low. With 94% of stimuli perceived at rest and a false positive rate of 1%, the area under a receiver operating characteristic curve, which includes this point, a simple measure of the detectability of the stimuli (Green and Swets 1988), was ≥ 0.965 and at most 0.995. Maximum negative bias at rest was therefore between 0.025 and 0.055.

During movement + stimulation trials, estimates of maximum perceptual performance at the closer stimulation sites (Figs. 7*D* and 10) as well as performance at the distant sites (Fig. 8) were 8–10% lower than performance at rest. This observation may reflect an increase in negative bias during movement trials, although other factors may also have contributed (see further). The time-dependent decreases in performance are unlikely to be the result of an overconservative response strategy that varied over time because subjects showed the same time-dependent decrease when a bias-free 2AFC procedure was used. The results are consistently explained only by a time- and distance-dependent decrease in the detectability of the stimuli, an explanation that is entirely

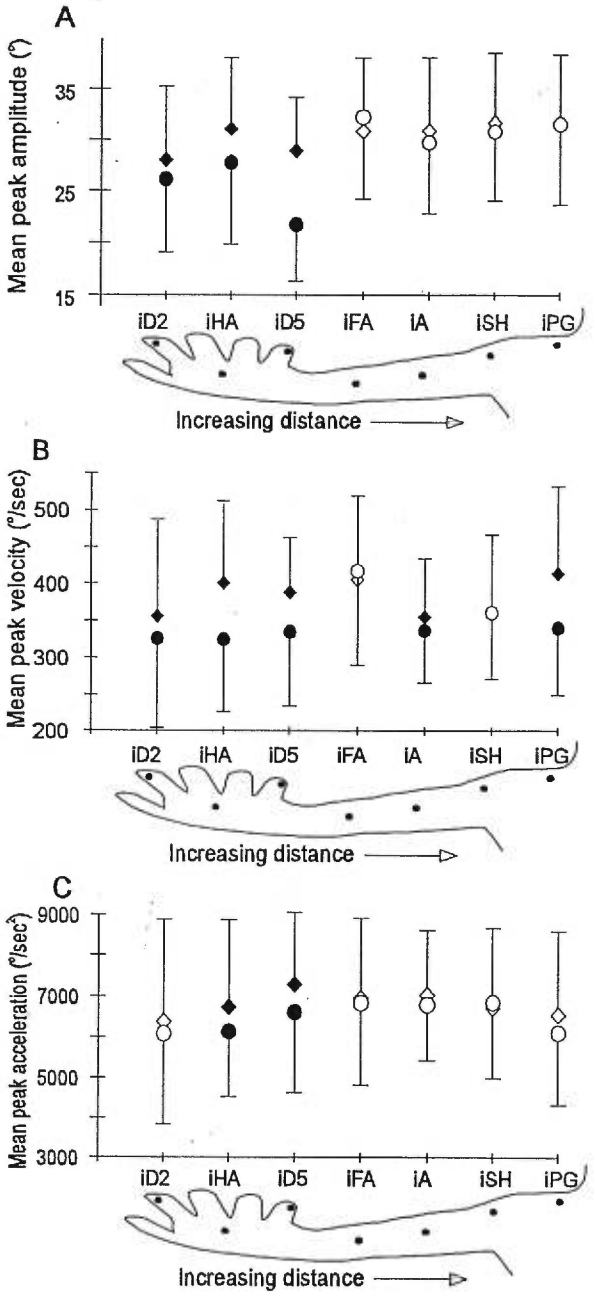


FIG. 11. *A–C*: comparison of kinematic parameters during movement + stimulation trials at the 7 sites showing a time-dependent decrease in detection, as a function of whether the stimulus was perceived (circles) or not perceived (diamonds). Analysis was restricted to those trials in which the stimulus was delivered within 30 ms of the time of peak decrease in perceptual performance. Significant *t*-test results ($P < 0.05$) are shown as filled symbols (nonsignificant, open symbols). Kinematic parameters were often significantly smaller in those trials where the stimulus was perceived than in those trials where the stimulus was not perceived. Peak velocity (*B*) was more frequently a significant factor (5 of 7 sites) than peak amplitude (*A*) or peak acceleration (*C*).

compatible with previous results from this laboratory and others.

Distribution of non-time–dependent decreases in tactile detection

The three distant sites showed no time-dependent decrease in perceptual performance, but rather a constant, ~10%, decrease. Chapman et al. (1987) had seen small elevations in detection threshold for sites contralateral to the movement, but previous experiments using suprathreshold stimuli showed no significant change in either perception (Chapman et al. 1987; Milne et al. 1988) or cortical SEPs (Cohen and Starr 1987; Rushton et al. 1981) when the stimuli were delivered to a limb other than the one producing the movement. Analyses indicated that the non-time–dependent decrease was present at all stimulation sites and that the time-dependent decreases were superimposed on this. As explained above, an increase in negative bias could have produced this sort of result; the low false positive rate at rest makes it impossible to exclude this hypothesis. On the other hand, similar reductions were observed in test experiments using a bias-free experimental procedure. Previous studies have shown that spatial and cross-modal shifts in attention can modify tactile detection (Butter et al. 1989; Meyer et al. 1963; Post and Chapman 1991). Given that the subjects had to divide their attention between the motor and perceptual tasks, it is suggested that attentional influences might have been responsible for the non-time–dependent decrease in performance.

Spatiotemporal characteristics of decreases in tactile detection

All sites on the moving limb (including the adjacent trunk, iPG) showed time-dependent reductions in the proportion of stimuli perceived. At the four sites closest to the moving digit, these reductions began before EMG onset (Fig. 9). The timing of these reductions is in agreement with the earliest modulation reported in previous psychophysical investigations (Coquery et al. 1971; Dyhre-Poulsen 1978), as well as the earliest modulation reported in evoked-potential and single-unit recording studies (Chapman et al. 1988; Cohen and Starr 1987; Jiang et al. 1991). On the other hand, the timing of the *peak* decrease in perceptual performance at the four distal sites coincided with the onset of agonist EMG (Fig. 7*B*). The possible significance of this with regard to the role of peripheral feedback in the reduction of detection during movement is discussed in the text following.

During movement, there was almost complete suppression of detection at the four sites closest to the moving digit (iD2, iHA, iD5, and iFA). At these sites, movement kinematic parameters likely play a role in determining the maximal amplitude of the observed suppression (see further). As the distance between the site of stimulation and the site of movement increased, the magnitude of the reduction declined. In addition as distance increased there was a 60-ms shift in the time of the peak decrease and a 120-ms shift in the first significant decrease. These results are compatible with the

findings of previous studies that showed that detection thresholds are increased during movement (e.g., Chapman et al. 1987; Duysens et al. 1995; Post et al. 1994), with the largest change occurring at sites closest to the moving segment.

Sources and mechanisms of the movement-related decrease in tactile detection

The spatiotemporal characteristics of the movement-related decrease in detectability, discussed earlier, help to define the necessary attributes of the gating mechanism(s). Both centrally mediated inhibition, originating from central and/or peripheral sources, and also physical factors in the periphery may have contributed to generating the observed spatiotemporal gradient.

CENTRAL SOURCES. One finding in favor of a central source for the gating signals is the observation that at the sites closest to the moving digit, detection began to decline 120 ms before movement onset and 70 ms before the onset of EMG, i.e., well before any peripheral feedback could have been generated. The earliest time-dependent decrease may be related to the preparation and execution of the movement, as has been suggested by others (Chapman et al. 1988; Coulter 1974; Ghez and Lenzi 1971; Jiang et al. 1990a,b, 1991). Although the timing is consistent with the precentral motor areas playing a role, the spatial distribution observed here is not consistent with the pattern of modulation of cutaneous transmission elicited by intracortical microstimulation of primary motor cortex in nonhuman primates (Jiang et al. 1989, 1990a). In the latter studies, only sites on the same segment or distal to the activated muscle showed evidence of sensory gating, and microstimulation did not modulate transmission from the glabrous skin of the hand (although inputs from the hairy dorsum of the digit and hand were gated). In contrast, the present findings showed that sensory modulation extends to sites located proximal to the moving segment and to the glabrous skin of the digits. Providing the mechanisms are similar in humans and monkeys, this suggests that area 4 is unlikely to be the only source for the widely distributed gating effects reported here. Other areas (e.g., premotor cortical regions) also must be involved if the present findings are to be explained by a centrally originating signal.

PERIPHERAL SOURCES. Other spatial and temporal characteristics of the gating argue in favor of a role for peripheral sources in mediating the movement-related gating. The timing of the peak decrease at the stimulation sites closest to the moving digit coincided with the onset of EMG activity. It is reasonable to assume that peripheral feedback begins at this moment, e.g., muscle spindle discharge elicited by alpha-gamma coactivation, and that this feedback contributed to the sharp decrease in detection performance at this time. In support of this, passive movements can be as effective as active movements in generating movement-related decreases in tactile inputs (e.g., Chapman et al. 1987, 1988; Huttunen and Hömberg 1990), although in a study of cortical SEPs elicited by suprathreshold cutaneous stimuli, Chapman et al. (1988) found that the time course for modulation was delayed considerably for passive as compared with active movements, i.e., the modulation followed movement onset instead of preceding it. A variety of mechanoreceptors are activated by passive movements, and evidence suggests that muscle spindle feedback contributes to the decrease in cortical SEPs during active and passive movement (Brooke et al. 1997). Cutaneous feedback also diminishes the amplitude of cortical SEPs (Jones et al. 1988).

CENTRAL MECHANISMS. Whatever the source of the gating signal(s), any explanation for the results needs to explain the widespread spatial distribution of the time-dependent decrease in detectability and the pronounced temporal gradient across the homolateral limb. Clearly a single inhibitory signal distributed simultaneously across a given somatosensory relay would not produce the complex spatiotemporal gradient observed here. Several, not necessarily exclusive, explanations can be advanced to explain the large changes in time and magnitude at the more proximal sites on the arm. The spatial distribution may be the result of lateral inhibition generated by the gating signals. The timing of the modulation would reflect the time necessary for spread of the lateral inhibition, whereas the amplitude of the observed decrease in detectability would reflect the strength of the inhibitory signal. The distant sites that showed no obvious time-dependent modulation would either be outside the zone of effective inhibition or the inhibition might have occurred at delays >100 ms after movement onset (the longest delay tested here). Given that synaptic delay is only 0.5 ms and that conduction distances within any candidate relay (dorsal column nuclei (DCN), ventrobasal thalamus, primary somatosensory cortex) are short, then this suggestion requires an extremely slow rate of spread through multiple synapses to explain delays of the order of 60 ms. This makes it more likely that multiple loops within the CNS may be involved in generating this spatiotemporal gradient, possibly via descending cortical projections to somatosensory relays, including cortico-thalamic, cortico-DCN, cortico-reticulo-DCN, and corticospinal projections. If such is the case, then it may well be that a combination of signals interact to produce the observed spatiotemporal gradient, with only a subset of these mechanisms being effective at the more distant sites. Such a suggestion is supported by our observation that only the sites closest to the moving digit showed evidence of a relationship between perceptual performance and movement kinematics.

The preceding suggestions are independent of the potential source of the gating signal, peripheral or central. It needs to be stressed, however, that there is evidence that a strong peripheral signal can modify the perception of weaker and earlier peripheral stimuli (backward masking). Evidence for this comes from studies in humans that have shown that detection of cutaneous stimuli is decreased if the stimulus is followed by a second "masking" stimulus (Laskin and Spencer 1979; Scherrick 1964; Schmid 1961; Weisenberger 1994). Although the spatial pattern is similar to that seen here, no temporal shift over distance has been reported in masking studies, leaving the importance of these observations unclear with regard to the present findings.

CONTRIBUTION OF PERIPHERAL FACTORS. Although central mechanisms can explain the results, the potential contribution of physical factors (distance, nerve conduction velocity)

to the spatiotemporal gradient also should be considered. The shift in timing of the peak decrease over distance might be explained by the decrease in distance (and therefore travel time) between the more proximal stimulation sites and the CNS. Assuming that conduction velocity was similar at all stimulation sites, that low-intensity electrical stimuli preferentially activated large diameter (A beta) cutaneous fibers with a conduction velocity of ≥50 m/s (Diabetes Control and Complications Trial Research Group 1995) and that the average distance between the proximal and distal sites was ~70 cm, then at most a 10-ms shift in the timing of the modulation would be explained by differences in travel times. This suggests that differences in path length did not contribute significantly to the results.

Second, we considered the possibility that the gating signal originated from faster conducting afferents (group I muscle afferents) than those activated by our near-threshold stimulus. Considering the iD2 stimulation site and assuming that spindle feedback begins at EMG onset and that cutaneous and muscle afferent conduction velocites are ~50 and 60 m/s, respectively, then a test stimulus given at EMG onset would arrive ~3 ms later at the spinal cord as compared with the EMG-related feedback in an average subject. The earliest modulation at the iD2 site, on the other hand, preceded EMG onset by 70 ms, which again could only be explained by differences in conduction velocity if test stimulus conduction was much slower than it presumably was. The latter is not supported by studies in humans that have shown no significant difference in the conduction velocities of the fastest muscle and cutaneous afferents (Macefield et al. 1989). In summary, physical factors likely made a small contribution to the results and it appears more likely that central mechanisms need to be invoked.

Functional significance

We recently suggested that simple reductions in the signal-to-noise ratio, produced by an increase in background noise, could not explain all of the changes in perception that accompany movement (Chapman et al. 1996; Post et al. 1994). Although such a mechanism explains well the decreases in tactile detection with unchanged discrimination thresholds, it also predicts reduced magnitude estimates at low, but not high, intensities of stimulation. Using spatially distributed vibrotactile stimuli, however, we found that magnitude estimates were decreased at high, but not low, intensities (Post et al. 1994). Consideration of other models, linear and nonlinear inhibitory surrounds, led us to suggest that the latter provided the closest approximation to the experimental data (Chapman et al. 1996). The model developed here, based largely on logistic functions, thus provides some support for our suggestion that the underlying inhibitory processes may have a nonlinear distribution. Relative distances between body parts were used in the model. This permits the generation of simple, testable predictions for the amount of gating that will be observed at a given distance on the body surface from the site of motion. However, because the gating effects are exerted within the CNS where the representation reflects peripheral innervation density and not absolute size, it would be interesting to incorporate the relative distance between body parts at each of the somatosensory relays within the CNS into the model. Unfortunately, such data for humans are not currently available. In summary, the model represents an initial step toward developing a complete description of the effects of inhibitory mechanisms associated with movement-related gating on detection performance and provides a framework for future physiological studies aimed at characterizing the exact sources and sites of action of these mechanisms.

These movement-related inhibitory controls diminish the amount of afferent input that must be processed within the CNS during movement. As suggested by Coulter (1974), movement-related gating controls may suppress redundant inputs that can be predicted from the motor command so that the detection of other unexpected or novel stimuli is enhanced. The spatial distribution and large temporal shift in the gating actions (hand vs. shoulder) may reflect the spread of nonspecific lateral inhibition originating from the body part in motion. Alternatively, the spatiotemporal gradient could be the result of "hard wired" gating, which reflects common patterns of use for the upper limb, thus providing an automatic reduction in afferent input during natural goal-directed movements.

APPENDIX A

Logistic equation used to fit perceptual data for each stimulation site, where P represents the proportion of stimuli perceived, t represents time (ms), *mpk* represents the peak slope, *tpk* represents the time of the peak slope, max represents the maximum proportion of stimuli perceived, and min represents the minimum proportion of stimuli perceived.

$$P = [(\max - \min)(1/1 + e^{-4mpk(t-tpk)})] + \min \qquad (A1)$$

APPENDIX B

The equation for the model surface was constructed using the following reasoning. Because logistic functions provided a good description of the effects of time on perceptual performance (Figs. 5 and 6) and distance on function minima (Fig. 7*B*), this type of function again was used to model these effects. A linear function was used to model distance-dependent modifications in the timing of peak decreases in performance based on the finding that a linear model provided an adequate description of the effect of distance (Fig. 7*A*). Because maximum performance was invariant between sites, it was modeled with a constant. The resultant model can be described as follows, where Fdist is a function representing the proportion of stimuli detected, i represents time (ms), j represents the distance between the stimulation site and iD2 (proportion of subject height), and max represents the maximum proportion of stimuli detected.

Distance-dependent component (affects mostly minimum predicted perceptual performance)

$$\text{Distlogistic}_{(j)} = 1/[1 + e^{(-96j+41)}] \qquad (B1)$$

Distance-dependent shift in the timing of the peak decrease in perceptual performance

$$\text{Shift}_{(j)} = -9.4j \qquad (B2)$$

Time-dependent component (modifies perceptual performance over time)

$$\text{Timelogistic}_{(i)} = 1/[1 + e^{(0.046t + (0.36 + \text{Shift}_{(j)}))}] \quad (B3)$$

Maximum predicted perceptual performance

$$\max = 0.86 \quad (B4)$$

Global equation

$$F\text{dist}_{(i,j)} = \max\,[\text{Distlogist} + \text{Timelogist}(1 - \text{Distlogist})] \quad (B5)$$

The model surface was fitted first to the actual data using a least mean squares method, producing a mean square error value (MSE1). The peak difference between the model surface and the data points was calculated using this model surface (PEAK1). The final fitting minimized the value of an error variable calculated in the following manner

$$\text{Error variable} = \text{MSE} + (\text{MSE1}/\text{PEAK1})\text{PEAK} \quad (B6)$$

For the model surface presented in this paper, this produced a 25% decrease in peak error with only a 5% increase in MSE.

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CHAPTER 3

TIME-COURSE AND MAGNITUDE OF MOVEMENT-RELATED GATING OF TACTILE DETECTION IN HUMANS.

II EFFECTS OF STIMULUS INTENSITY ON DETECTION AND SCALING OF TACTILE STIMULI.

ABSTRACT

This study examined the effect of systematically varying stimulus intensity on the time course and magnitude of movement-related gating of tactile detection and scaling in 17 human subjects trained to perform a rapid abduction of the right index finger (D2) in response to a visual cue. Electrical stimulation was delivered to D2 at five different intensities. At the lowest intensity, approximately 90% of stimuli were detected at rest (1 x P_{90}); four multiples of this intensity were also tested (1.25, 1.5, 1.75 and 2.0 x P_{90}). At all intensities of stimulation, detection of stimuli applied to the moving digit was diminished significantly and in a time-dependent manner, with peak decreases occurring within ± 12 ms of the onset of electromyographic activity in the first dorsal interosseous (25-45 ms before movement onset). Reductions in the proportion of stimuli detected were greatest at the lowest stimulus intensity and progressively smaller at higher intensities. No shift in the timing of the decreases in performance was seen with increasing intensity. Once the weakest intensity at which most stimuli were perceived during movement had been established (2 x P_{90}), magnitude estimation experiments were performed using two stimulus intensities, 2 x P_{90} (5 subjects) and 3 x P_{90} (3 subjects). Significant movement-related decreases in estimated stimulus magnitude were observed at both intensities, the time course of which was similar to the time course of reductions in detection performance. As stimulus intensity increased, the magnitude of the movement-related decrease in scaling diminished. A model of detection performance that accurately described the effect

of stimulus intensity and timing on movement-related reductions in detection was created. This model was then combined with a previous model which described the effects of stimulus localisation and timing in order to predict detection performance at a given stimulation site, intensity and time during movement. Movement-related gating of tactile perception represents the end result of movement-related effects on the transmission and subsequent processing of the stimulus. The combined model clearly defines many of the requirements that proposed physiological mechanisms of movement-related gating will have to fulfil.

INTRODUCTION

Transmission of somatosensory stimuli is modulated during movement. Studies in rat, cat, monkey and human, examining both somatosensory evoked potentials (SEPs) (Brooke et al. 1997; Coquery et al. 1972; Jiang et al. 1990; Morita et al. 1998; Starr and Cohen 1985) or single unit responses (Chapin and Woodward 1982; Jiang et al. 1991; Shin et al. 1994), have documented movement-related decreases in somatosensory transmission to the primary somatosensory cortex (SI) during movement. Psychophysical studies in humans have confirmed the existence of concomitant decreases in the detection of near-threshold stimuli during movement (Chapman et al. 1987; Coquery et al. 1971; Pertovaara et al. 1992; Post et al. 1994; Williams et al. 1998). However, wide variations have been reported with regards to the timing of the onset of movement-related gating, ranging from 200 ms before the onset of movement (Coulter 1974; Chapman et al 1988; Ghez and Lenzi 1971) to 30 ms after movement onset (Kristeva-Feige et al. 1996), and in the magnitude of the effects, with observed reductions ranging from 7.5% (Coulter 1974) to over 90% (Kristeva-Feige et al. 1996). The reasons for these important variations in the parameters defining movement-related gating remain unclear.

We recently demonstrated that the time course for movement-related suppression of detection of near-threshold tactile stimuli in humans is similar to that observed for the decreases in somatosensory transmission to SI in monkeys

(Williams et al. 1998). Our results indicated that peak decreases in detection can occur as early as 50 ms before the onset of movement, at about the same time movement-related electromyographic (EMG) activity begins. We also demonstrated the importance of stimulus location in determining the timing and magnitude of movement-related suppression. Reductions in detection were greatest and occurred earliest when the stimulus was delivered near the moving body part, the index finger (D2), and time-dependent movement-related decreases were restricted to the homolateral upper limb. Time-dependent reductions in detection were smaller and occurred up to 60 ms later as the distance between D2 and the stimulation site was increased. A modest (~10%) non time-dependent decrease in detection was also observed at all stimulation sites (including distant ones such as the contralateral D2), possibly related to the attentional demands placed on the subjects when they had to simultaneously perform D2 abduction and attempt to detect stimuli.

While our previous results contribute to explaining some of the wide variations in the time course and magnitude of gating effects, another potentially important variable is the intensity of the test stimulus employed to evaluate sensory responsiveness. Most of the above studies used relatively strong test stimuli (several times threshold). In contrast, we used a single, near-threshold intensity (90% detected at rest, P_{90}), and did not examine the effect of higher stimulus intensities. The present study addressed this issue by determining the influence of systematically increasing the intensity of the test stimulus on the time

course and magnitude of movement-related decreases in tactile perception. In order to facilitate comparisons with our previous results, we used the same motor task, D2 abduction. The effects of movement on stimulus perception were quantified using a detection task for weak stimuli and a stimulus scaling task for suprathreshold stimuli. A model describing the effect of stimulus intensity and timing on detection performance was created, and combined with a previous model describing the effect of stimulus location and timing (Williams et al. 1998) to clarify the relative importance of each of these factors in determining detection performance. A preliminary account of these results has been published (Williams and Chapman 1996)

METHODS

Subjects. A total of 17 naive, paid volunteers (9 males and 8 females, ages 16 to 28 years) participated in the study. All subjects but one 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. Nine subjects participated in the stimulus detection tasks. Eight other subjects participated in the stimulus scaling tasks. Data from each subject were gathered in one or two sessions lasting one to three hours each. At the beginning of each session, subjects received verbal instructions about the motor and perceptual task that they were to perform. This was followed

by a series 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 below.

Motor task. Subjects were asked to actively abduct the right index finger (D2) as soon as possible after the illumination of a visual Go cue within a discrete 2 second "trial" period (see below and Fig. 1C in Williams et al. 1998). Subjects initiated the movement from a neutral position and produced an abduction of at least 15 degrees and no more than 45 degrees (Fig. 1A, Williams et al. 1998).

Perceptual tasks. The *detection* task was identical to the one described in Williams et al. 1998. The stimulus consisted of a single, 2-ms, constant-current pulse applied via surface electrodes to the glabrous skin of the middle and distal phalanges of D2. Subjects were asked to report whether or not they detected the occurrence of a stimulus within a trial period; 90% of trials contained a stimulus, while 10% did not (catch trials). No information regarding the proportion of trials with or without a stimulus was given to the subjects, and no feedback was given with regard to the accuracy of subject's perceptual judgements. Although the experimental design was not strictly bias-independent, we have shown that the same results are obtained using a bias-independent two alternative forced choice version of the task (Williams et al. 1998). The shorter and simpler trials reduced the effects of memory and fatigue on results. Five different electrical stimulus intensities were tested in separate blocks of trials. The lowest stimulus intensity

produced detection at rest of approximately 90% of stimuli (P_{90}) (current range 0.55 mA to 1.12 mA); this was estimated as in the previous paper by using the method of Wetherhill and Levitt (1965). Four multiples of this current intensity were also examined: 1.25 x P_{90}, 1.5 x P_{90}, 1.75 x P_{90} and 2 x P_{90}. All nine subjects that participated in the detection experiments were tested at every intensity. The order of testing for different intensities was counterbalanced between subjects. Data gathered at 1 x P_{90} is a subset of a previously published 41-subject data set gathered at this intensity (Williams et al. 1998). Perceptual performance in the subset was not significantly different than perceptual performance in the complete data-set ($p>0.05$, Kolmogorov-Smirnov test).

In the *scaling* task, subjects were asked to rate the intensity of a single 2 ms constant current electrical stimulus delivered to the glabrous skin of the middle and distal phalanges of D2 on a continuous numeric scale. Two stimulus intensities were tested: 2 x P_{90} (5 subjects, current range 1.4 to 1.76 mA) and 3 x P_{90} (3 subjects, current range 1.98 to 2.22 mA). The 2 x P_{90} stimulus intensity was chosen because data from the detection experiments showed that this was the lowest intensity at which the vast majority of stimuli were perceived during performance of the movement task. The higher stimulus intensity was included to examine the effect of increasing intensity on scaling during movement. Each subject was tested using only one stimulus intensity. A stimulus of identical intensity was delivered in every trial. Subjects were told that "in each trial, a stimulus may or may not be delivered, and that the intensity of the stimulus may

or may not vary from trial to trial". Subjects were completely free to choose the rating values they desired, including fractions and decimals if they preferred, and were told to use an open scale, i.e. no fixed maximum or minimum. Subjects established their scale in a series of practice trials before data collection began. No scaling performance feedback was given at any time during the practice trials or the experiments.

Experimental design. Data were collected in discrete trials lasting two seconds each. The position of D2, as well as electromyographic (EMG) activity from first dorsal interosseous (1^{st} DI), were recorded during each trial. Before the onset of each trial, subjects were instructed whether or not to move. Approximately 75% of trials involved movement. In the remaining 25% the subject remained immobile, providing a running estimate of perceptual performance at rest for both the detection and scaling tasks. Three trial types were used in the detection experiments: movement + stimulation trials, rest + stimulation trials, and catch trials (no stimulus, movement or rest). Two trial types were used in the scaling experiments: movement + stimulation trials and rest + stimulation trials. Trials were initiated by the experimenter, and consisted of an initial 500 ms period to monitor the presence of spontaneous movement or EMG activity without the subject being aware that the trial had begun (Fig. 1C, Williams et al. 1998), followed by the illumination of an array of light-emitting diodes (Go cue) for 1500 ms. The Go cue served two purposes: 1) a signal for subjects to perform the appropriate perceptual task and 2) a signal to initiate a movement in the

movement trials. After the Go cue was turned off (2000 ms), subjects were asked in the detection experiments to report verbally whether or not they had detected a stimulus. In the scaling experiments, the subjects were asked to verbally rate the intensity of the stimulus. Responses were stored along with the rest of the trial data. A random time interval ranging from 1 to 10 seconds separated the end of one trial from the beginning of the next.

To sample variations in perceptual performance *over time* for each perceptual task, nine different stimulus presentation delays were used at each stimulus intensity. All trials at a given delay were performed before another delay was tested; the order of testing for the various stimulation delays was randomly determined and varied from intensity to intensity and subject to subject. To determine the stimulus presentation delays, each subject's average reaction time (RT, time from the Go cue to movement onset) was estimated before data collection began. Stimuli were presented at RT - 160 ms, RT - 120 ms, RT - 80 ms, RT - 40 ms, RT, and RT + 40 ms, RT + 80 ms, RT + 120 ms and RT + 160 ms. For the detection task, at least 15 movement + stimulation trials, 5 rest + stimulation trials, and 2 catch trials (trials with no stimulation, with or without movement) were performed at each delay (minimum of 22 trials/intensity). Catch trials were omitted for scaling experiments (minimum of 20 trials/intensity). Thus for each detection experiment, a minimum of 198 trials were recorded, and for each scaling experiment, a minimum of 180 trials were recorded. Neither the subject nor the experimenter knew the actual trial-by-trial delay between the onset

of peripheral movement-related activity and stimulus delivery at the time of the data acquisition.

Data Analysis. For both detection and scaling experiments, movement timing (EMG onset, movement onset, movement duration) and kinematic (amplitude, peak velocity, peak acceleration) parameters were determined for each movement trial as described in Williams et al. (1998). Trials in which spontaneous EMG activity or movement was seen in the initial 500 ms were eliminated from further analysis. For each subject and stimulus intensity, the mean value for each of these parameters was calculated. For each stimulus intensity, the overall means using combined data from all subjects were also calculated. The existence of significant inter-intensity differences in these parameters, which could affect perceptual performance (Angel and Malenka 1982; Chapman et al. 1996; Williams et al. 1998), were evaluated using two-way analysis of variance (ANOVA; level of significance, $p<0.05$) for the detection experiments and t-tests (level of significance, $p<0.05$) for the scaling experiments.

To provide an overview of the effect of movement and stimulus intensity on performance in the *detection* experiments, the overall proportion of stimuli perceived for each of the three trial types (movement + stimulation, rest + stimulation, catch trials) was calculated for each stimulus intensity using combined data from all subjects. Significant changes in detection performance as stimulus intensity was varied were evaluated for movement + stimulation, rest +

stimulation and catch trials using two-way ANOVAs. If an ANOVA was significant, an analysis of covariance (ANCOVA) was performed to examine whether or not there was a significant correlation between stimulus intensity and perceptual performance. For each stimulus intensity, Fisher one-tailed exact probability tests for a 2x2 contingency table (level of significance, $p<0.01$) were used to evaluate whether the proportion of stimuli detected in movement + stimulation trials was significantly different from the proportion of stimuli detected during rest + stimulation trials. This same test was also used in all subsequent proportion comparisons.

To analyse the effect of movement and stimulus intensity on *scaling* performance, only trials in which a stimulus was perceived were retained (see Results). For each subject, the magnitude estimate in each trial was normalised by dividing the response by the mean magnitude estimate of the rest + stimulation trials. This approach allowed data from different subjects (using different absolute scales) to easily be compared and pooled while preserving the relative changes in reported stimulus magnitude over time and stimulus intensity. Data from all subjects at each stimulus intensity were pooled, and the mean proportion of stimuli perceived along with the standard deviation in movement + stimulation and rest + stimulation trials was calculated. At each of two intensities tested, magnitude estimates for movement + stimulation and rest + stimulation trials were compared using t-tests. T-tests were also used to evaluate significant inter-intensity differences in scaling of movement + stimulation trials.

To evaluate the relative importance of performance differences between subjects, stimulus intensity and stimulus timing, trials from a given subject, intensity and perceptual task were then grouped into 40 ms bins relative to either movement or EMG onset. The proportion of stimuli detected along with the 95% confidence interval or the average magnitude estimate along with the standard error (SEM) was calculated for each bin. ANCOVA was performed on the resulting performance data. Since inter-subject variation in detection performance explained little of the total variation in detection performance (see Results), all trials from all subjects for a given stimulus intensity and perceptual task were pooled for further analyses. This allowed trials to be grouped into 20 ms bins relative to EMG onset for a more precise analysis of the time course. The proportion of stimuli detected along with the 95% confidence interval (detection task) or the average magnitude estimate along with the SEM (scaling task) was again calculated for each bin. Performance in each bin was then compared to performance in the rest + stimulation trials using the appropriate statistical test (see above). In order to provide an adequate description of the temporal evolution of perceptual abilities relative to EMG onset, linear and modified logistic functions were fitted as described in Williams et al. (1998) to the pooled data from each stimulus intensity and perceptual task. Linear functions were tested as in general they provided the best fit to data where no time-dependent reductions were observed, while logistic functions were tested as they provided better descriptions of time-dependent reductions in detection. The best fitting model (linear or logistic) was retained if it provided an adequate description of the data,

i.e. if the probability of obtaining the observed amount of total squared error was greater than 0.05. If a linear model was retained, the presence of a slope significantly different from zero was evaluated using a t-test. If a logistic descriptor was retained, four parameters were determined: the maximum predicted perceptual performance, the minimum predicted perceptual performance, the peak slope (measure of the peak rate of decrease in perceptual performance), and the timing of the peak slope (the time at which perceptual performance decreased most rapidly). Since movements were of varying duration (defined in this study as the time needed to reach peak amplitude), the shortest movement duration was determined for each stimulus intensity and only data up to the end of the shortest movement were fitted.

For the data gathered in the detection task experiments, the effects of intensity on the logistic parameters described above were evaluated using correlation coefficients (adjusted for the number of parameters used to fit the data) and F-tests to determine the best fitting between a linear and a logistic model for parameters constrained to the interval [0,1]. Polynomial functions were also considered for variables not limited to this interval.

RESULTS

Performance of the motor task.

A total of 45 detection and 8 scaling experiments were performed in 17 subjects. Before examining intensity-related differences in perceptual performance, the possibility that the movements themselves might have varied when stimulus intensity was changed was evaluated. For detection experiments, all movement-related kinematic and timing parameters (Table 1) but one, mean peak amplitude, showed no significant change across the five stimulus intensities. Peak amplitude varied significantly ($p=0.01$) but very little in absolute terms (32 to 36°), and the amplitude did not covary with stimulus intensity (ANCOVA). For the scaling experiments, comparisons between the two stimulus intensities for the movement kinematic and timing parameters were also made. No significant difference was seen except for peak velocity, which was significantly higher at intensity 2 x P_{90} as compared to intensity 3 x P_{90} (t-test, $p<0.05$).

First DI EMG activity preceded movement onset by 38 ms on average, and as was seen previously (Williams et al. 1998) was highly correlated with movement onset ($r=0.98$). The fact that 1st DI EMG preceded and was highly correlated with the onset of movement is consistent with 1st DI being the major agonist of the abduction movement studied here.

Performance of the detection task.

Table 2 summarises the global detection performance of all subjects in the rest + stimulation, movement + stimulation, and catch trials. At intensity 1 x P_{90}, subjects detected 94% of stimuli delivered at rest, a result similar to the detection performance reported previously at this stimulus intensity (Williams et al. 1998). At all other stimulus intensities, detection performance was higher (99-100%). As a result of this difference, a two-way ANOVA found a significant inter-intensity difference in the proportion of stimuli perceived at rest, and an ANCOVA showed a significant correlation between stimulus intensity and the proportion of stimuli perceived at rest (see legend for details).

To determine whether practice or fatigue significantly altered detection performance over the course of the experiments, perceptual performance in the first 10% of immobile trials was compared to perceptual performance in the last 10% of immobile trials for each stimulus intensity. No significant differences were observed at any of the stimulus intensities ($p>0.01$, Fisher exact probability test).

Of 747 catch trials (all detection experiments combined), only 4 false positive responses were noted (0.5%), indicating that subjects used a very conservative response strategy throughout the series of experiments. As detailed in the legend

for Table 2, no stimulus intensity was associated with significantly more false positives than any other.

Significantly fewer stimuli were detected during movement for all five stimulus intensities, as compared to detection performance at rest ($p<0.001$, Fisher exact tests). Subjects perceived 62% of the stimuli presented during movement + stimulation trials. A two-way ANOVA (Table 2) showed that there was significant inter-intensity variability in perceptual performance for movement + stimulation trials, while an ANCOVA demonstrated the existence of a positive correlation between stimulus intensity and detection performance during movement trials.

Time and intensity-dependent changes in the detection of stimuli.

ANCOVA was performed on detection performance data, and demonstrated that both timing and stimulus intensity explained a significant portion of the variance present in the data ($p<0.001$), while inter-subject differences explained less than 0.5% of the variation accounted for by the ANCOVA ($p>0.01$). Since the contribution of inter-subject differences to performance was small, all trials from all subjects for a given stimulus intensity were pooled for further analyses.

Using the pooled data, detection performance was plotted relative to the time of EMG onset and movement onset for all five stimulus intensities. Given the

high correlation between EMG onset and movement onset (above), the movement onset curves were almost identical to the EMG onset curves, except for a 40 ms shift in the timing values which corresponded to the lead time between EMG onset and movement onset. Consequently, only curves plotted relative to EMG onset are presented. Figure 1 shows the effects of D2 movement on the ability to detect stimuli applied to the moving digit over time at each of the five intensities tested. Logistic functions were retained to describe detection performance over time at all five stimulus intensities, and are also shown on Fig. 1. At the lowest intensity (1x P_{90}, Fig. 1A) only, maximum performance in the three earliest bins (-110 to -70 ms) was approximately 10% lower on average than performance at rest, with two of the three bins having detection performance significantly different from that at rest (filled dots). The maximum detection performance estimated by the logistic function which best described the 1 x P_{90} data was only 0.82. We previously argued that this early decrease reflected a non time-dependent reduction in detection performance (Williams et al. 1998). When stimulus intensity increased modestly to 1.25 x P_{90} (Fig. 1B), detection performance in the earliest bins (-110 to -70 ms) was close to that seen at rest (open circles in all three bins, 0.98 estimated maximum detection performance in movement + stimulation trials), suggesting that the non time-dependent reduction in detection was no longer present. Similar results were obtained at all other intensities (Fig. 1C-E). A time-dependent decrease in detection, on the other hand, was observed at all stimulus intensities. At all but the highest stimulus intensity, detection performance was seen to decline precipitously around the time of EMG

onset, with the timing of the peak decrease occurring within the bin immediately preceding EMG onset at 1 x P_{90} and the bin immediately following EMG onset at all other intensities tested. At the highest intensity (2 x P_{90}), a modest decrease in detection performance could still be observed at approximately the time of EMG onset (Fig. 1E), with a small but significant reduction in detection being observed in the initial 80 ms after EMG onset (filled dots).

A partial rebound in detection performance appeared to occur for stimuli delivered more than 100 ms after EMG onset, especially at the higher stimulus intensities. When detection performance was compared across the first and second 100 ms after EMG onset (Table 3), significantly more stimuli were perceived in the later interval at the three highest stimulus intensities. Rebounds in detection performance appeared to coincide with the time at which peak movement velocity was attained (92 ± 11 ms).

Modelling the contribution of stimulus timing and intensity on detection performance during movement.

In order to describe the effects of stimulus intensity on detection performance in a manner similar to that used to describe the effect of stimulus location in the previous paper (Williams et al. 1998), the logistic function parameters detailed in Fig. 1 are plotted as a function of stimulus intensity in Fig. 2. Intensity-dependent

trends were described using the best fitting of a variety of models (see Methods). The timing of peak decreases in detection performance (Fig. 2A) was accurately modelled by a second order polynomial ($p<0.01$, not shown), but this function was not monotonic over the range of intensities studied, and peak decreases were all within ± 12 ms of EMG onset, i.e. within the 20 ms temporal resolution of the analysis. A logistic function provided the best fit for minimum estimated detection performance over a wide range of detection performance values (Fig. 2B). Variation in the peak slope could not be modelled successfully (Fig. 2C). In the case of maximum estimated detection performance (Fig. 2D), the fitted logistic function was essentially a straight line with a slight inflection at the lowest stimulus intensity.

Based on the results of the above analyses, a model of detection performance during movement + stimulation trials that incorporated the importance of stimulus timing and stimulus intensity was created (Fig. 3). The detection data during the movement trials were used to define a surface, presented in Fig. 3A. This representation permitted a visualisation of modifications in detection performance for all intensities at any given time. A model surface (Fig. 3B) was then fitted to the surface created by the detection data. The equation for the model surface was constructed using the following reasoning, as in Williams et al. (1998). Since logistic functions provided a good description of the effects of time on the proportion of stimuli detected (Fig. 1) and of intensity on both maximum and minimum detection performance (Fig. 2B,D), this type of function was again used

to model these effects (equations 1 to 3 below). Several different models were tested for the timing of peak decreases, including fixing the timing of peak decreases at EMG onset, fitting the timing of peak decreases to a constant value different from EMG onset, and varying the timing of peak decreases linearly with stimulus intensity. Even when allowed to vary, the timing of peak decreases remained within ± 1.5 ms of EMG onset at all intensities. The very small changes in the timing of peak decreases led to a very small reduction in total error (< 5%) which did not justify the added variables necessary to implement them. The model used to generate Fig. 3B is detailed in equation (4), where *Fint* represents the time- and intensity-dependent proportion of stimuli perceived, *i* represents time (ms), and *k* represents stimulus intensity.

Intensity-dependent change in maximum predicted detection performance:

$$IntmaxLogistic_{(k)} = 1/[1 + e^{(-68.5k + 67)}] \quad \textbf{(1)}$$

Intensity-dependent change in minimum predicted detection performance:

$$IntminLogistic_{(k)} = 1/[1 + e^{(-5.75k + 10)}] \quad \textbf{(2)}$$

Time-dependent component (modifies detection performance over time):

$$InttimeLogistic_{(i)} = 1/[1 + e^{(0.085i)}] \quad \textbf{(3)}$$

Global equation:

$$Fint_{(i,k)} = IntmaxLogistic_{(k)}[IntminLogistic_{(k)} + Inttimelogistic_{(i)}(1-IntminLogistic_{(k)})] \quad \textbf{(4)}$$

The model surface was not significantly different from the surface generated by the actual data ($p>0.05$). The total squared error between the surface described by the detection data and the surface described by the model was 0.34, and the peak squared error was less than 0.035 (Fig. 3C). The maximum estimated detection performance over the model surface was 1.0, i.e. 100% detected. The minimum estimated detection performance over the model surface was 0.01, though it increased sharply as intensity increased, reaching 0.8 at the highest intensity over the time period considered by the model, therefore adequately describing the effect of stimulus intensity on minimum detection performance as detailed in Figures 1 and 2D. The model was a concise and accurate description of the experimental results, which could be combined with a previous model (Williams et al. 1998) describing the effect of stimulus location and timing on detection performance to generate quantitative predictions of detection performance during movement (see Discussion).

Time and intensity-dependent changes in scaling.

Subjects perceived and scaled 94% of stimuli delivered during movement + stimulation trials at intensity 2 x P_{90} and all of the stimuli delivered at intensity 3 x P_{90}. For both stimulus intensities, the mean magnitude estimates in the first and last 10% of the rest + stimulation trials were not significantly different.

Normalised magnitude estimates during movement + stimulation trials were significantly lower than magnitude estimates during immobile trials at both stimulus intensities (t-test, $p<0.001$), averaging 0.64 ± 0.01 at $2 \times P_{90}$ and 0.75 ± 0.01 at $3 \times P_{90}$. A significantly greater relative decrease in magnitude estimates was seen at intensity $2 \times P_{90}$ than at intensity $3 \times P_{90}$ (t-test, $p<0.001$).

Magnitude estimates from the movement + stimulation trials were pooled and plotted relative to EMG onset, and logistic functions were fitted (Fig. 4A and B). The relative intensity of the stimuli delivered earliest relative to EMG onset was more affected at $2 \times P_{90}$ (maximum rating estimated by the logistic function of only 0.81) than at $3 \times P_{90}$ (estimated maximum rating: 0.97). Time-dependent decreases in stimulus scaling were observed at both $2 \times P_{90}$ and $3 \times P_{90}$. Stimuli presented after EMG onset were rated lower than those perceived before EMG onset at both intensities (t-test, $p<0.001$). At both intensities, peak decreases in scaling values occurred within 10 ms of EMG onset.

DISCUSSION

The results of the present study demonstrated that time-dependent and movement-related decreases in tactile detection occurred during a simple abduction movement for stimuli in an intensity range spanning from 1 to $2 \times P_{90}$. Increasing the stimulus intensity decreased the magnitude of the movement-

related decrease in detection, but had relatively little effect on the timing of the decrease, with peak decreases in detection always occurring within ± 12 ms of EMG (25-45 ms before movement onset). The subjective intensity of stimuli perceived during movement was also decreased, and this in a time and intensity-dependent manner that was similar to that observed for changes in detection.

Methodological considerations.

As discussed in Williams et al. (1998), the electrical stimuli used in this series of experiments represented a reliable, reproducible, easily calibrated source of perceptual afference, identical in all respects but intensity to those used to gather data in a previous series of experiments (Williams et al. 1998). Using these stimuli, subjects consistently detected almost all stimuli delivered during rest trials, while producing a negligible false positive rate, thus essentially behaving as "perfect receivers" and making analysis and comparisons of detection performance during the motor task relatively simple. Consistent with previous observations (Williams et al. 1998), the onset of 1st DI EMG always preceded movement onset and was highly correlated with movement onset. 1st DI EMG represented the earliest easily observable response to the motor command, and thus an appropriate reference point for evaluating the timing of decreases in perceptual performance during movement. Practice and fatigue effects did not appear to play a significant role on perceptual performance at rest in either the

detection or the scaling experiments: neither detection nor scaling performance varied significantly from the beginning to the end of each experiment. 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; Rauch et al. 1985; Williams et al. 1998); this must be considered a potential confounding factor when comparing detection or scaling performance. In the case of the data presented in this study, kinematic parameters varied little over the different intensities tested, and the differences in perception across different stimulus intensities could not be explained by any systematic change in movement kinematics.

Stimulus intensity and detection performance.

The results of this study confirm our previous observation that stimulus timing relative to EMG or movement onset is a key variable in determining detection performance. They extend these observations by showing that the magnitude of the decrease in detection performance after EMG onset is graded as a function of stimulus intensity. At the lowest intensity (1 x P_{90}), only 5% of stimuli were detected in the initial 100 ms after EMG onset; as intensity was increased, performance gradually improved so that 90% of stimuli were detected at the highest intensity tested (2 x P_{90}). The intensity-dependent increase in stimulus detection after EMG onset is consistent with previous observations that

detection threshold is elevated during motor activity (Chapman et al. 1987; Post et al. 1994). The results also showed that the timing of the peak decrease in detection was relatively invariant over the range of intensities tested, always falling within ± 20 ms of EMG onset, on average about 40 ms before movement onset. These timing values are in agreement with the apparent timing of *peak* decreases observed using single stimulus intensities in studies that employed psychophysical (Coquery et al. 1971), evoked potential (Chapman et al. 1988; Morita et al. 1998) or single cell methodologies (Jiang et al. 1991). The relative invariance in the timing of peak decreases across a range of stimulus intensities is an interesting contrast with experiments in which stimuli of identical intensity were delivered to sites at different distances from the moving digit (Williams et al. 1998); in those experiments the timing of the peak decrease shifted by as much as 60 ms as distance increased (D2 to pectoral girdle). This difference may reflect differences in the mechanisms by which reductions in movement-related gating influences lessen as intensity or distance increases.

The present results demonstrate that differences in stimulus intensity strongly affect the amplitude of movement-related gating, and so resolve some of the differences in the amplitude of reductions reported in the literature. However, changes in stimulus intensity did not affect the timing of these reductions. Several factors probably explain differences in the values reported in the literature with regards to the timing of the onset of movement-related gating of tactile afference (see Introduction). The first factor is the physical relationship between the body

part in motion and the site of stimulation, which affects the timing of movement-related decreases in detection as detailed in Williams et al. (1998). Most studies applied stimuli to the moving body part, and we have shown that gating of perception is strongest and earliest in this case. Although our results were restricted to movements of the digit, we have made similar observations for movements about the elbow, both when stimuli were applied to the forearm (see companion paper) and when stimuli were applied to D2 (unpublished observations, Williams, Shenasa and Chapman). The second factor is the variable temporal resolution of previous studies, with some reported time courses based on bin widths as wide as 100 ms (e.g. Cohen and Starr 1987). The third factor is variation in the stimulus intensity used in the different studies, experimental paradigms using near-threshold stimulus intensities being more affected by non time-dependent effects (see below) and thus generating much earlier estimates of the onset of movement-related gating. The fourth, and related, factor is methodological. In the present study, we concentrated on timing the more robust peak decreases in detection, rather than the first reduction in detection or response amplitude, as in most previous studies (e.g. Chapman et al 1988, Cohen and Starr 1987). This approach was based on the assumption that the detection function over the interval being modelled is monotonic. Concentrating on the timing of peak reductions in perceptual performance had the advantage of being insensitive to the non time-dependent decrease seen at the lowest stimulus intensity, and avoided the inherent difficulty of attempting to define the first and therefore smallest significant change in detection. Our timing values were based on the

largest, and therefore the easiest to measure/identify, change in perceptual performance. Finally, many previous studies have reported the onset of gating relative to movement onset. As shown in the companion paper, measuring the onset of gating relative to the onset of EMG activity rather than movement onset minimises differences in timing when comparing results across different motor tasks.

A partial rebound in detection performance began after peak movement velocity was attained (>100 ms after the onset of EMG), but the time course of this recovery was not determined because subject behaviour was not specified after reaching the criterion amplitude: some subjects maintained D2 abduction, while others actively returned to the start position, and others relaxed and so resumed the neutral position.. The relatively rapid rebound in detection performance seen with this experimental paradigm is earlier that that observed in previous studies. Recovery in somatosensory evoked potentials has been reported approximately 400-500 ms after the end of movement-related EMG (Angel et al. 1986; Cohen and Starr 1987). Detection and scaling performance has also been seen to recover in the seconds following the end of a finger movement (Schmidt et al. 1990) and 900 ms after the onset of isometric jaw contractions (Kemppainen et al. 1993). The more rapid rebound in detection performance seen in this study may be a function of stimulus intensity and/or motor-task parameters. It could be hypothesised that this rebound in detection coincides with decreases in peripheral movement-related reafference as the movement slows and/or the end of the motor

command, since many motor cortical units show a burst that ends at peak velocity in a similar motor task (e.g. Lamarre et al. 1980).

In summary, the present results show that tactile detection is decreased but not abolished during movement, with the minimum proportion of stimuli perceived during movement-related gating increasing as a function of stimulus intensity. The time course of the movement-related gating of detection is invariant across a range of stimulus intensities.

Stimulus intensity and scaling.

The perceived magnitude of suprathreshold stimuli was found to be reduced in the movement + stimulation trials. This is in agreement with some previous investigations (Coquery et al. 1971; Milne et al. 1988; Post et al. 1994) using both electrical and vibrotactile stimuli, but not with others (Chapman et al. 1987). Post et al. (1994) suggested that the amount of cutaneous feedback generated during the motor task is a key factor for observing movement-related decreases in magnitude estimates. The present findings suggest that the intensity of the test stimulus was also a critical factor. We found that stimuli of higher intensity showed less relative reduction in magnitude than stimuli of lesser intensity. Consistent with this, a previous study (Chapman et al. 1987) that did not observe decreases in perceived magnitude employed intensities at far greater multiples of threshold than the stimuli used here (up to 10 X detection threshold).

The timing of the peak decrease in subjective intensity was similar to the timing of the reduction in detection performance for stimuli of lower intensity, with the peak decrease always occurring within ± one bin (20 ms) of EMG onset. This argues in favour of the reduction in magnitude for suprathreshold stimuli being mediated by similar mechanisms as reductions in detection for stimuli of lower intensity. Contrary to the results with the detection task, no recovery in scaling was observed in the later bins. This may reflect a modest difference in sampling: the time of peak velocity was later in the scaling experiments (110 ms

vs. 92 ms after EMG onset in the detection experiments), and data in the scaling task were not available for as long after EMG onset (160 ms after EMG onset vs. 200 ms for detection tasks). Alternatively, scaling performance may simply return to normal more slowly after the end of movement. A differently designed motor task, as well as later sampling, would be needed to distinguish between these two possibilities.

Intensity and non time-dependent decreases in perceptual performance.

It has been previously argued that the earliest reduction seen at the lowest stimulus intensity tested (1 x P_{90}) represents a non time-dependent decrease in detection performance, possibly related to attentional influences (Williams et al. 1998). In this study, no evidence of a non time-dependent decrease in detection was observed with stimuli greater than 1 x P_{90}. At all intensities but the lowest one tested, detection performance in the earliest bins during movement + stimulation trials was not significantly different from performance at rest. However, at the lowest intensity tested in the scaling task (2 x P_{90}), the perceived *magnitude* of suprathreshold stimuli delivered quite early (up to 120 ms before EMG onset) was significantly lower than the perceived intensity of stimuli delivered at rest. This result suggests that the same non time-dependent effect which affected detection at 1 x P_{90} may still modulate perceived magnitude at higher stimulus intensities, a hypothesis which could be confirmed by performing

scaling experiments at more distant sites where it is expected that only a non time-dependent reduction would be observed.

Predicting the effect of movement on the detection of stimuli.

In a previous paper, data were gathered at several stimulation sites using stimuli of intensity 1 x P_{90} in subjects performing a motor task identical to the one used in this study. A model was then created which accurately described the effect of distance, expressed in standardised proportions of total body lengths (National Aeronautics and Space Administration 1978; Contini 1972), between the moving digit and the site of stimulation on detection performance (Williams et al. 1998). In equation 5 below, the simple model developed in this study to describe the effects of intensity on detection performance (Fig. 3) is combined with the previous model describing the effects of distance on stimulus detection in order to predict detection performance at combinations of stimulation sites and stimulus intensities that have not been tested experimentally. In this combined model, *P(i,j,k)* represents the proportion of stimuli perceived at time *i*, distance *j* and intensity *k*, *Fint(i,k)* is the time- and intensity-dependent model described in this paper (equation 4); *IntmaxLogistic(k)* corresponds to equation 1; *Fdist(i,j)* (time- and distance- dependent model) and *Max* correspond respectively to equations B5 and B4 in Williams et al. (1998). In essence, the intensity-dependent model defines maximum and minimum detection performance for a stimulation site that

corresponds to D2 stimulation (j=0). As the distance between the site of stimulation and the body part in motion increases (j>0), the proportion of stimuli that remain undetected decreases as a function of the distance-dependent model. To give an idea of the relationship between proportional distances and actual sites on the body, 0 distance corresponds to D2, 0.125 corresponds to the middle of the forearm, 0.25 distance corresponds to the lower third of the upper arm, and 0.425 corresponds to the ipsilateral sterno-clavicular joint; all distances beyond this point correspond to sites that cannot be considered functionally as part of the upper limb.

$$P(i, j, k) = Fint_{(i,\,k)} + \left(IntmaxLogistic(k) - Fint_{(i,\,k)}\right) x \ \frac{Fdist_{(i,\,j)} - Fdist_{(i,0)}}{Max - Fdist_{(i,0)}} \qquad \textbf{(5)}$$

This model makes it possible to predict detection performance at a given time, stimulus intensity, and distance between the moving digit and the site of stimulation. Figure 5 gives examples of the predictions generated by equation (5). In Fig. 5A detection performance over time when stimuli are of intensity 1.5 x P_{90} is predicted at distances from the site of stimulation ranging from 0 to 0.5 body lengths. In Fig. 5B detection performance 50 ms after EMG onset is predicted for intensities ranging from 1 to 2 x P_{90} and distances from the site of stimulation ranging from 0 to 0.5 body lengths. Detection functions such as those in Fig. 5A, or "psychophysical functions" similar to those in Fig. 5B, can be generated at any distance, intensity, and timing value, producing 2, 3 or 4 dimensional prediction arrays.

An interesting situation which arises when the two models are combined is the fact that the effect of time on detection performance is modelled twice, once in the intensity model where peak decreases always occur at t = 0, and once in the distance model where the timing of peak decreases occurs later as distance increases. If this accurately reflects the reality of the distance and intensity effects, two time-dependent equations are appropriate. However, it is also possible that the timing of intensity-dependent decreases varies with distance. If this is the case and shifts in timing over distance are similar in the distance and intensity functions, it may be possible to model the effect of time on detection performance using one equation rather than two. Data from more distant locations at higher stimulus intensities would resolve this question.

For detection performance after EMG onset, the range of stimulus intensities and locations tested was sufficient to sample detection performance over a wide range of values from those approaching zero to those approaching 1 (Fig. 2B). However, for detection performance before EMG onset, distance had no effect while only one stimulus intensity produced a maximum value significantly less than 1 (1 x P_{90}, Fig. 2D). Therefore, for time values < 0, the current model's predictions with regards to detection performance as intensity decreases to values less than 1 x P_{90} is based on very little information, and could very well need to be adjusted when data from stimulus intensities lower than those tested in our experiments become available. Nonetheless, the basic

behaviour of the model as intensity decreases to values lower than 1 x P_{90} is sound: detection performance remains near 0 at time values > 0, and also rapidly approaches 0 at time values < 0.

In conclusion, the modifications in the timing and/or magnitude of movement-related gating of tactile detection and suprathreshold magnitude estimation described in this paper and the preceding one (Williams et al. 1998) represent the "end result" of all movement-related effects on the transmission and subsequent processing of the stimulus. The combined model provides an accurate description of the timing and intensity of movement-related decreases in tactile detection, makes it possible to clearly define the perceptual consequences of these effects, and therefore defines the requirements that physiological mechanisms brought forward to explain these decreases will have to fulfil.

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TABLES

Table 1: Temporal and kinematic parameters describing the performance of the motor task (D2 abduction) in the detection experiments, and the results of ANOVAs comparing values across the 5 stimulation intensities.

Movement-related parameter		Mean (± SEM)	ANOVA *	
			F	P
Temporal	RT	239 ± 43 ms	1.32	0.28
	1st DI EMG onset	200 ± 45 ms	1.50	0.22
	EMG lead time	38 ± 7 ms	0.51	0.73
	Movement duration	179 ± 45 ms	0.45	0.77
Kinematic	Peak amplitude	34 ± 5^{0}	4.15	0.01[a]
	Peak velocity	$440 \pm 100\ ^{0}/sec$	1.93	0.13
	Peak acceleration	$6900 \pm 1400\ ^{0}/sec^{2}$	0.36	0.83

Results for 45 experiments and 9 subjects. 1st DI, first dorsal interosseous; ANOVA, analysis of variance; D2, digit two; EMG, electromyographic; RT, reaction time.

* df=8, 4, 44.

[a] significant intersite difference, range 32° (P_{90}) to 36° (1.5 X P_{90}). ANCOVA showed no significant intensity-dependent trend for peak amplitude (df=1, p=0.79).

Table 2. Detection experiments: perceptual performance in the three trial types as a function of the stimulus intensity.

Stimulation intensity (x P_{90})	Proportion of positive responses		
	Rest + stimulation[a]	Movement + stimulation[a]	Catch trials
1.0	0.94 (0.88,0.97)	0.39 (0.36,0.42)[b]	0.00 (0.00,0.03)
1.25	1.00 (0.99,1.00)	0.46 (0.43,0.49)[b]	0.01 (0.00,0.05)
1.5	0.99 (0.98,1.00)	0.65 (0.62,0.68)[b]	0.01 (0.00,0.05)
1.75	0.99 (0.97,1.00)	0.69 (0.66,0.72)[b]	0.00 (0.00,0.03)
2.0	1.00 (0.98,1.00)	0.94 (0.93,0.95)[b]	0.01 (0.00,0.05)
Overall	0.98 (0.97,0.99)	0.62 (0.61,0.63)[b]	0.01 (0.00,0.02)

Results from 45 experiments, 9 subjects (95% confidence intervals in parentheses).

[a] Two-way ANOVAs showed that there was significant inter-intensity variation in perceptual performance during rest + stimulation trials (df=8, 4, 44; F=12.35; $p<0.0001$) and movement + stimulation trials (F=49; $p<0.0001$) but not catch trials (F=0.51; p=0.73). ANCOVA showed that perceptual performance increased with intensity for both the rest + stimulation trials (F=13, p=0.001) and the movement + stimulation trials (F=152, $p<0.0001$).

[b] Performance during movement+stimulation trials was significantly lower than performance during trials at rest for each stimulation intensity ($p<0.001$, Fischer exact test for a 2X2 contingency table).

Table 3. Detection performance after EMG onset

Stimulation intensity (x P_{90})	Proportion detected	
	t=1-100 ms	t=101-200 ms
1.0	0.05 (0.03,0.08)	0.03 (0.01,0.05)
1.25	0.11 (0.07,0.15)	0.14 (0.10,0.20)
1.5	0.26 (0.21,0.31)	0.40 (0.34,0.47)[a]
1.75	0.44 (0.38,0.49)	0.55 (0.48,0.62)[a]
2.0	0.90 (0.86,0.94)	0.96 (0.93,0.98)[a]
Overall	0.34 (0.32,0.37)	0.42 (0.39,0.45)[a]

Results from 45 experiments, 9 subjects (95% confidence intervals in parentheses). [a] Significantly more stimuli were detected in the second 100 ms interval after EMG onset as compared to the first 100 ms after EMG onset ($p<0.01$, Fischer exact test for a 2X2 contingency table).

FIGURE LEGENDS.

Fig. 1A-E Effects of D2 abduction on the detection of stimuli applied to the moving digit in nine subjects using five different stimulus intensities. Detection performance over time is plotted relative to the onset of 1st DI EMG (20 ms precision). Error bars represent the 95% confidence intervals. The shaded area shows the 95% confidence interval for perceptual performance in the rest + stimulation trials. Filled symbols indicate that perceptual performance during movement+stimulation trials was significantly lower than that observed at rest ($p<0.01$); open symbols, no change. The solid lines represent logistic functions fitted to the data points up to + 90 ms. Logistic equation parameters are also shown. Data plotted in A are a subset of data plotted in Williams et al. (1998).

Fig. 2A-D Logistic equation parameters from Fig. 1 plotted as a function of stimulus intensity. The timing of peak decreases (A) did not vary by more than ± 20 ms relative to EMG. Both the minimum (B) and maximum (D) estimated detection performance showed strong positive correlations with intensity, and were best described by logistic functions. Peak slope (C) could not be modelled successfully. R^2_A, coefficient of determination adjusted for the number of parameters in the fitted equation.

Fig. 3A-C Surfaces showing time- and intensity-dependence of perceptual performance. **A:** Pooled performance data during movement trials (z axis) plotted

as a function of time (x axis), and stimulus intensity (y axis). **B:** Best-fit model surface for the data shown in A. The timing of peak decreases in performances was modelled as constant (at t=0) for this surface. The model surface was not significantly different from the actual performance data ($p>0.05$). **C:** Contour map of the squared error between the surfaces plotted in A and B, showing areas of poorest fit.

Fig. 4A,B Effects of D2 abduction on mean perceived intensity (± SEM) of suprathreshold stimuli applied to the moving digit in five subjects (A, 2 x P_{90}) and three subjects at (B, 3 x P_{90}). Data plotted as in Fig. 1.The shaded area shows the mean estimate of detection performance at rest (± SEM). In each panel, data were normalised by dividing the response by the mean magnitude estimate in the rest + stimulation trials.

Fig. 5A,B Predicted detection performance generated by the combined model (equation 5) that described the effects of the intensity, timing and localisation of the stimulus on the detection of tactile stimuli during movement. stimulus timing-intensity-localisation model. A: at intensity 1.5 x P_{90}, predicted detection performance at different distances (expressed as standardised proportions of total body length, and varying from 0 to 0.5) from D2 are shown. B: at t=50 ms, predicted "psychophysical functions" over stimulus intensities 1 to 2 x P_{90} are shown for different distances from D2.

FIGURE 1

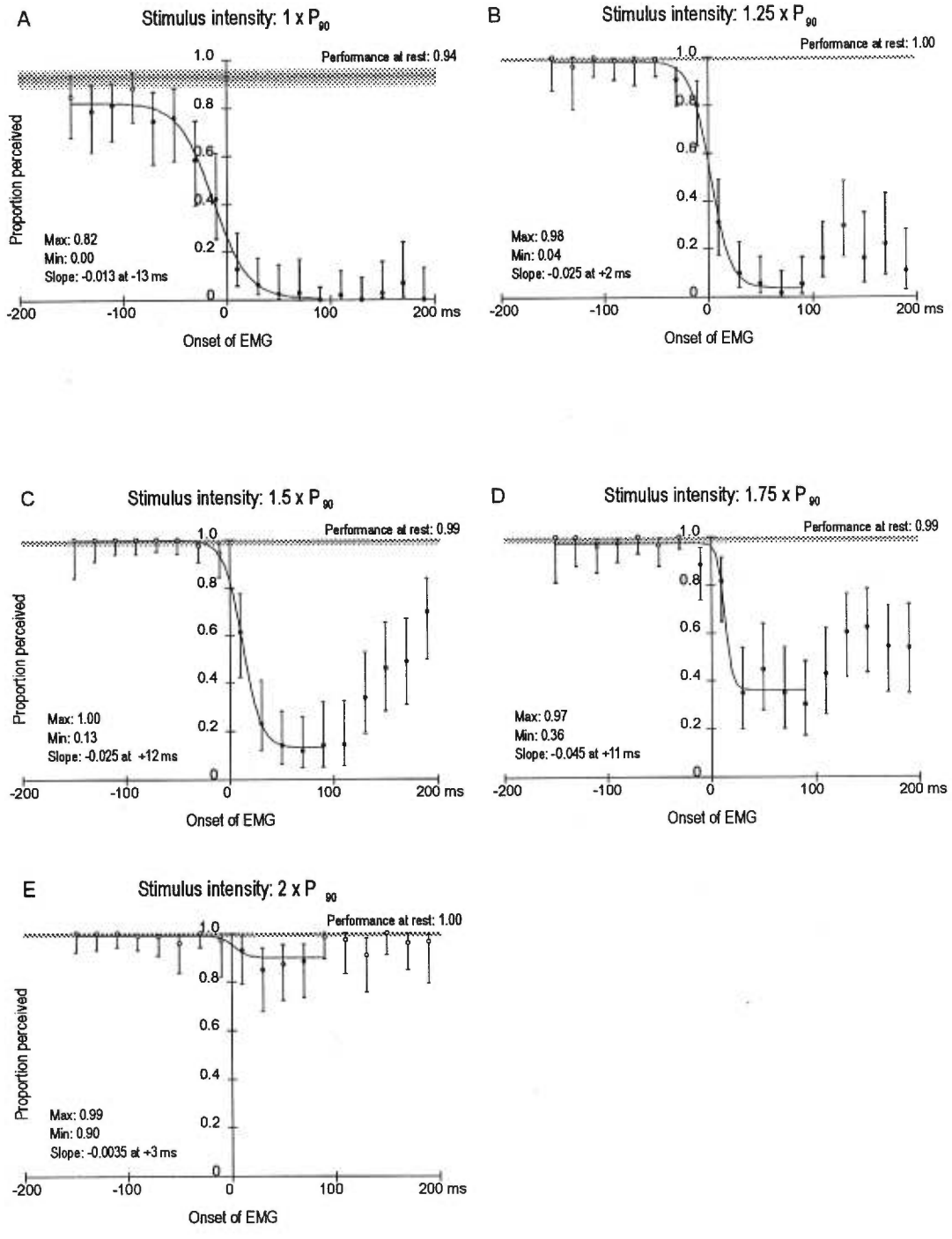

A
Stimulus intensity: 1 x P90
Performance at rest: 0.94
Proportion perceived
Max: 0.82
Min: 0.00
Slope: -0.013 at -13 ms
Onset of EMG
B
Stimulus intensity: 1.25 x P90
Performance at rest: 1.00
Max: 0.98
Min: 0.04
Slope: -0.025 at +2 ms
Onset of EMG
C
Stimulus intensity: 1.5 x P90
Performance at rest: 0.99
Max: 1.00
Min: 0.13
Slope: -0.025 at +12 ms
Onset of EMG
D
Stimulus intensity: 1.75 x P90
Performance at rest: 0.99
Max: 0.97
Min: 0.36
Slope: -0.045 at +11 ms
Onset of EMG
E
Stimulus intensity: 2 x P90
Performance at rest: 1.00
Max: 0.99
Min: 0.90
Slope: -0.0035 at +3 ms
Onset of EMG

FIGURE 2

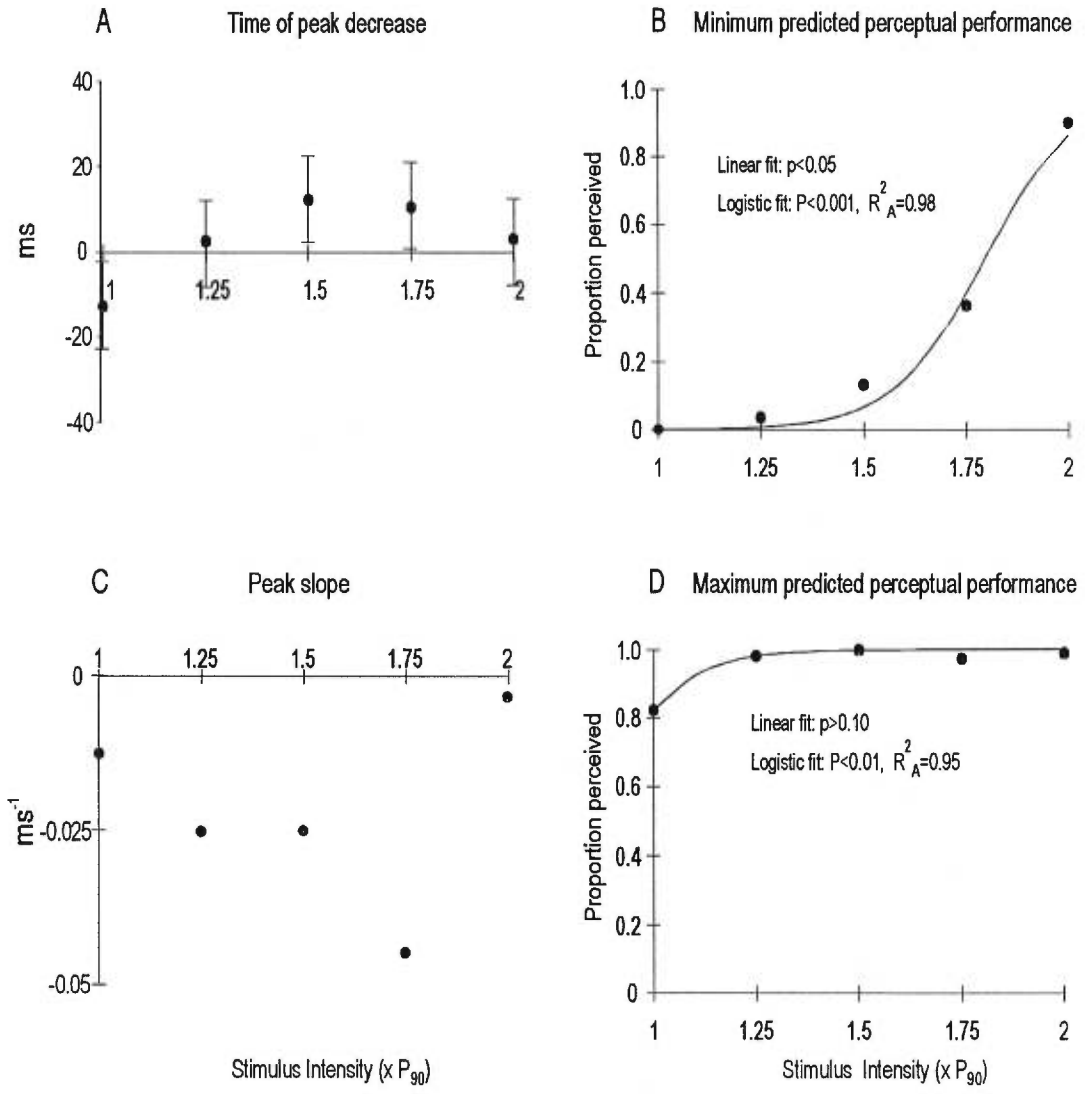

A
Time of peak decrease
ms
40
20
0
-20
-40
1
1.25
1.5
1.75
2
B
Minimum predicted perceptual performance
Proportion perceived
1.0
0.8
0.6
0.4
0.2
0
Linear fit: p<0.05
Logistic fit: P<0.001, R²A=0.98
C
Peak slope
ms⁻¹
0
-0.025
-0.05
Stimulus Intensity (x P90)
D
Maximum predicted perceptual performance
Linear fit: p>0.10
Logistic fit: P<0.01, R²A=0.95
Stimulus Intensity (x P90)

FIGURE 3

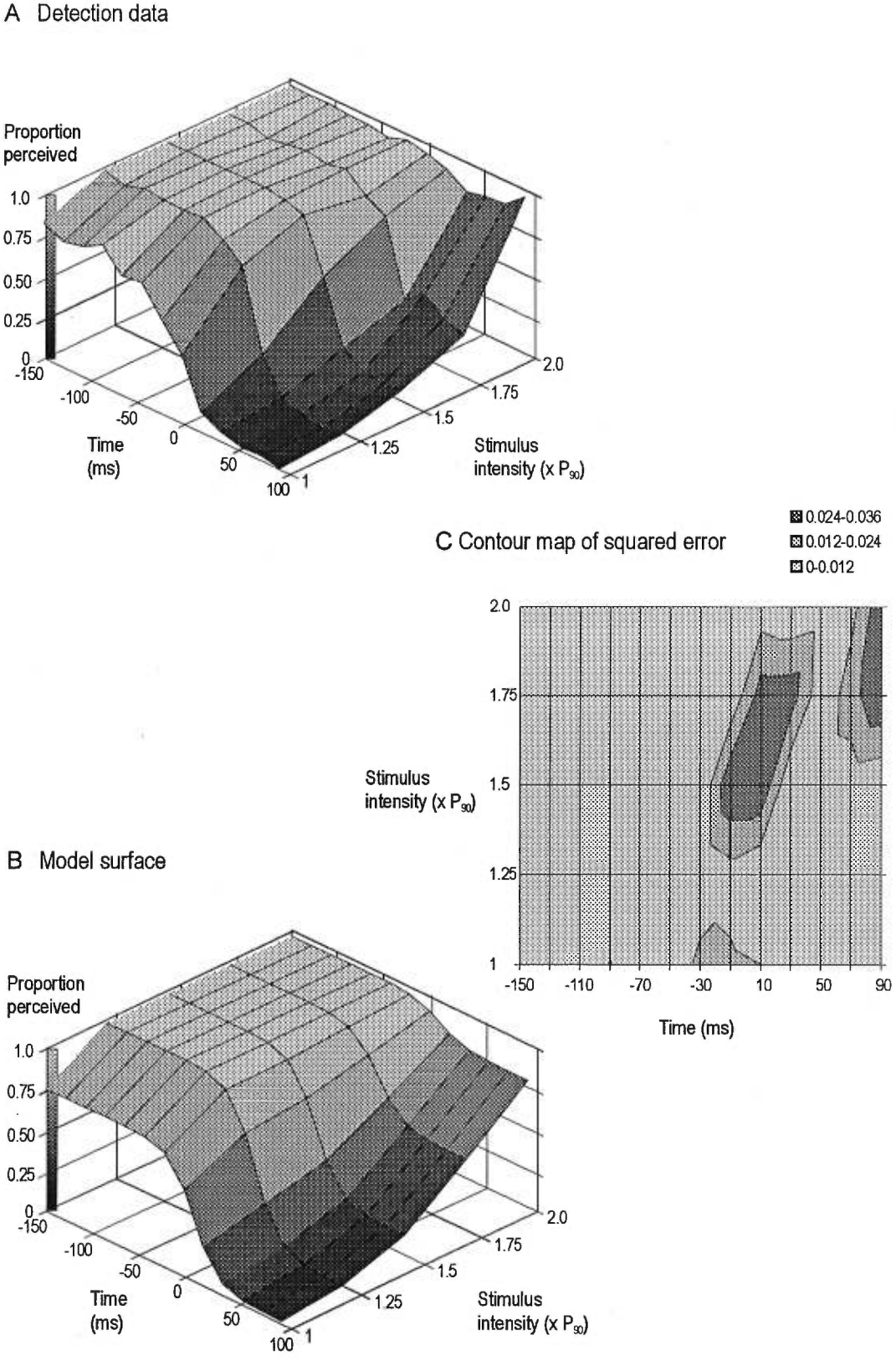

A Detection data
Proportion perceived
1.0
0.75
0.50
0.25
0
-150
-100
-50
0
50
100
Time (ms)
1
1.25
1.5
1.75
2.0
Stimulus intensity (x P90)
C Contour map of squared error
0.024-0.036
0.012-0.024
0-0.012
Stimulus intensity (x P90)
2.0
1.75
1.5
1.25
1
-150
-110
-70
-30
10
50
90
Time (ms)
B Model surface
Proportion perceived
1.0
0.75
0.50
0.25
0
-150
-100
-50
0
50
100
Time (ms)
1
1.25
1.5
1.75
2.0
Stimulus intensity (x P90)

FIGURE 4

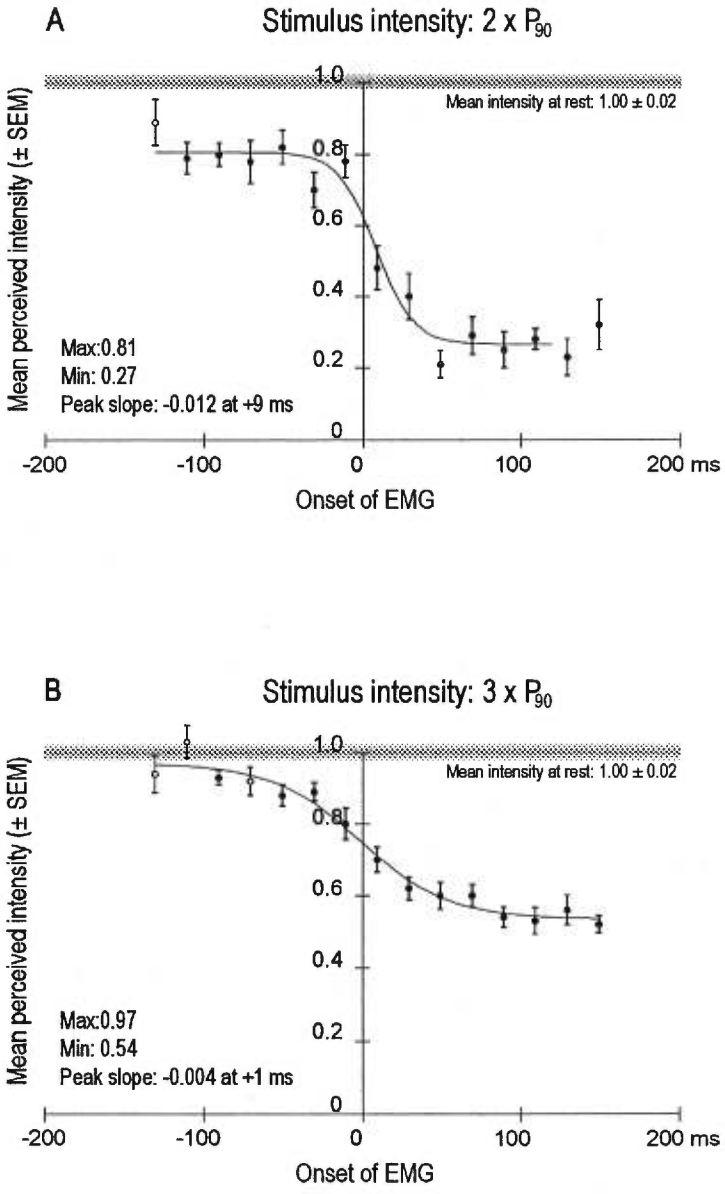
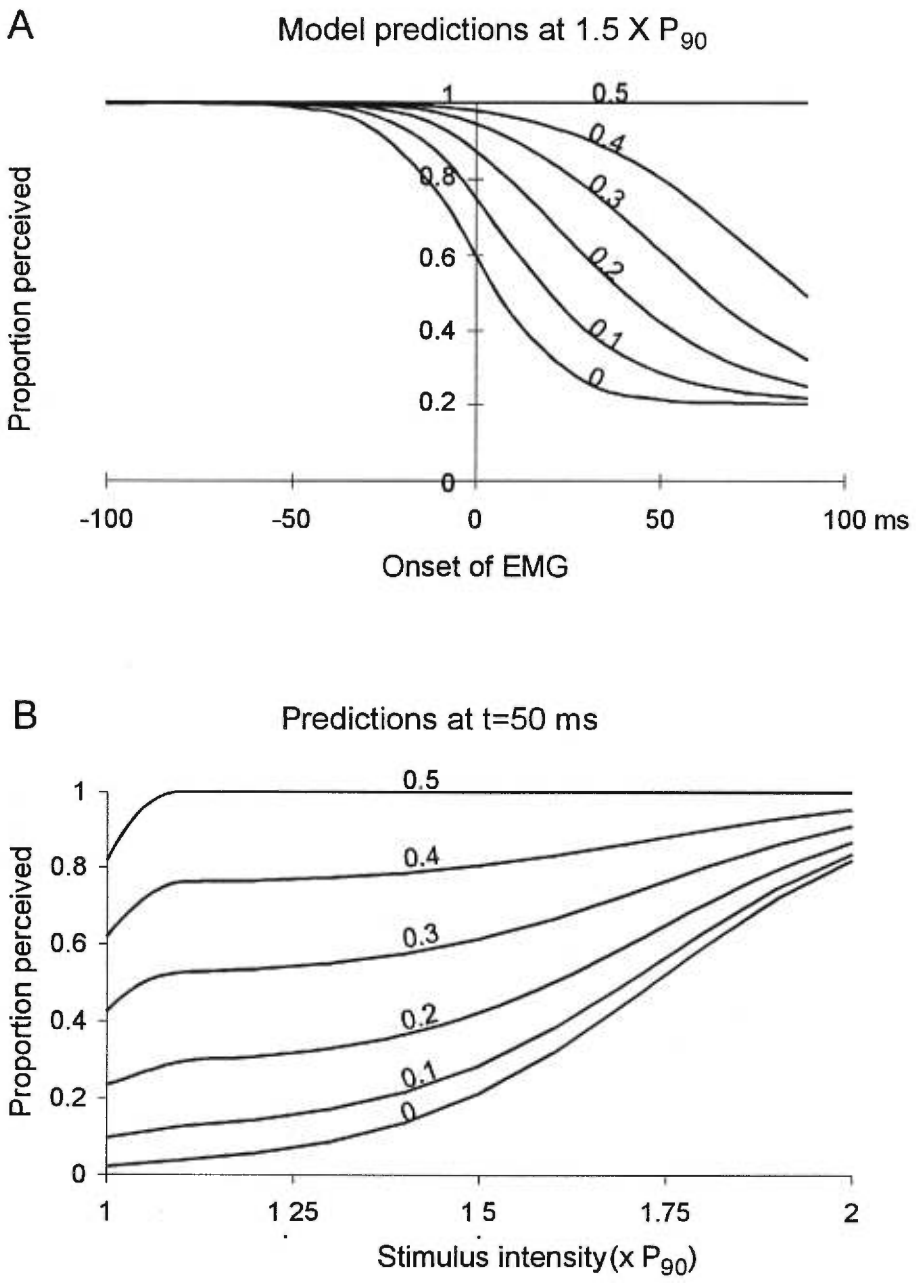

A
Stimulus intensity: 2 x P_{90}
Mean intensity at rest: 1.00 ± 0.02
Mean perceived intensity (± SEM)
Max:0.81
Min: 0.27
Peak slope: -0.012 at +9 ms
1.0
0.8
0.6
0.4
0.2
0
-200
-100
0
100
200 ms
Onset of EMG
B
Stimulus intensity: 3 x P_{90}
Mean intensity at rest: 1.00 ± 0.02
Mean perceived intensity (± SEM)
Max:0.97
Min: 0.54
Peak slope: -0.004 at +1 ms
1.0
0.8
0.6
0.4
0.2
0
-200
-100
0
100
200 ms
Onset of EMG

FIGURE 5


A
Model predictions at 1.5 X P_{90}
Proportion perceived
1
0.5
0.4
0.8
0.3
0.6
0.2
0.4
0.1
0.2
0
0
-100
-50
0
50
100 ms
Onset of EMG
B
Predictions at t=50 ms
Proportion perceived
0.5
1
0.8
0.4
0.6
0.3
0.4
0.2
0.2
0.1
0
0
1
1 25
1 5
1.75
2
Stimulus intensity (x P_{90})

CHAPTER 4

TIME-COURSE AND MAGNITUDE OF MOVEMENT-RELATED GATING OF TACTILE DETECTION IN HUMANS.

III EFFECT OF MOTOR TASKS.

ABSTRACT.

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 active and passive movement (right digit 2 (D2) abduction and right elbow extension) and during isotonic and isometric contractions (D2 only). Weak electrical stimuli (2 ms pulse; intensity, 90% detected at rest) were applied to the right D2 (all D2 abduction tasks), the dorsal forearm (elbow extension tasks) or the right shoulder (isotonic vs. isometric D2 abduction tasks). Significant time-dependent movement-related decreases in detection were obtained with all of the motor task/stimulation site combinations. When the results obtained during active isotonic movement tasks were compared to those obtained during passive movement or isometric tasks, no significant differences in the functions describing detection performance over time were seen. In the passive motor tasks, peak decreases in detection clearly preceded movement onset (by 36 ms with D2 abduction and 97 ms with elbow extension), despite the lack of a motor command or peripheral feedback associated with muscular contraction. The results obtained using isometric motor tasks 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. The results obtained using passive movement tasks are best explained by invoking backward masking of the test stimuli by movement-related afference, and demonstrate that movement-related afference is sufficient to produce decreases in detection with a time course and of an 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 1999) on the reduction in perception during movement.

The relation between factors related to the performance of the motor task and reductions in tactile detection is still unclear. Centrally originating 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 favour 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 which 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 or surrounding reticular formation (Bentivoglio and Rustioni 1986; Cheema et al. 1985; Jones and Wise 1977; Kuypers 1958, 1960; Martinez et al. 1995).

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 which 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 which is different from that seen for active movements since 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, 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 up to 120 ms (Williams et al. 1998), favours 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 eliminating peripheral feedback are likewise equivocal. Schmidt et al. (1990b) found that local anaesthetic 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 21 naive, paid volunteers (10 males and 11 females, ages 17 to 27 years) participated in the study. All subjects were right handed for writing. The experimental protocol was approved by the institutional ethics committee, 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 one to three hours each. At the beginning of each session, subjects received verbal instructions about the motor task and perceptual 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 are included below.

Motor tasks. Six different motor tasks were tested, all on the right side. One series of four tasks involved abduction of the index finger (D2). The other series (2 tasks) involved extension of the elbow. All tasks were reaction time tasks, i.e. subjects or their helpers (passive movements) were instructed to initiate their motor response as rapidly as possible after the illumination of a visual Go cue. The order of testing for the different motor tasks was randomly determined in each subject, and all trials with a given motor task were performed before another motor task was tested.

In the first series of experiments, active isotonic D2 abduction served as the reference motor task (9 subjects); this is described in the companion paper (Williams and Chapman 1999). Results obtained using the reference motor task were compared to results obtained in three test tasks: active, isometric abduction of D2 (n=7), passive abduction of D2 (n=9), and "freehand" abduction of D2 (active abduction of D2 without the position sensing apparatus; n=9). In the first test task, isometric D2 abduction, the subject's D2 was maintained in a maximally abducted position by a rubber hockey puck (Fig. 1A) in order to permit first dorsal interosseous (1st DI) activation without D2 movement or the generation of supplementary cutaneous afference during muscle activation. The subject attempted to abduct D2 in the same way as in the active movement task, producing 1st DI activation; task performance was monitored by inspecting the electromyographic (EMG) activity of 1st DI during the experiment. The second test task consisted of passive abduction of D2 in a manner similar to active movement, force for the movement being generated by a helper's D2 and transmitted by a connecting rod to the position detecting apparatus which in turn entrained the subject's D2 (Fig 1B). 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" motor task, subjects actively abducted D2 while their right arm hung unsupported by their side. D2 was in contact only with the stimulating electrodes. This motor

task aimed to quantify the effects on perceptual performance of tactile afference generated by entrainement 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 above for the isometric task.

In the second series of experiments (3 subjects), the reference task was active elbow extension, while the test task was passive elbow extension. The basic design of the motor task was preserved as in Fig. 1 of Williams et al (1998), but the subjects' arms rested on two horizontal independent manipulanda that permitted elbow flexion/extension. A potentiometer in the elbow hinge recorded forearm position. Subjects were instructed to adopt a rest position of 90 degrees flexion at the elbow (displayed on an oscilloscope), and at the Go cue to rapidly extend the elbow at least 45 degrees while attempting to minimise contraction of muscles not directly involved in the extension movement. In the test task, passive elbow extension, a helper (not visible to the subject) generated the elbow extension by means of a connecting rod attached to the subject's right manipulandum. The instructions to the subject and helper were as for the passive D2 abduction task described above.

Perceptual task. The detection task was identical to the one described in the companion paper. Stimuli consisted of single 2 ms square wave electrical pulses applied via surface electrodes at an intensity where approximately 90% were

detected at rest (1 x P_{90}, current range 0.4 to 0.88 mA). The three stimulation sites used in this study match the corresponding locations in Fig. 2 of Williams et al. (1998): right D2 (9 subjects), right dorsal forearm (3 subjects), right shoulder (5 of 7 subjects tested with isometric D2 abduction; data compared to previously published data from 9 subjects performing isotonic D2 abduction, Williams et al. 1998). All stimuli were ipsilateral to the body segment involved in the motor task.

Experimental design. The experimental design is described in the companion paper. For the D2 abduction task, D2 position and EMG activity of 1st DI were recorded, while for the elbow extension tasks, the joint angle of the elbow and EMG activity from triceps brachii were recorded. Different combinations of motor tasks and perceptual tasks were studied. In the first series of experiments, stimuli were applied to D2 while subjects performed active isotonic, active isometric, passive and "no apparatus" abduction of D2. Isotonic and isometric abduction of D2 were also studied using stimuli delivered at the right shoulder. In the second series of experiments, the ability of subjects to detect stimuli applied to the right forearm was studied during active and passive elbow extension.

The order of testing for the different motor tasks was randomly determined. All trials with a given motor task were performed before another motor task was tested. Movement + stimulation, rest + stimulation, and catch (no stimulation) trials were again used, the organisation of stimulus presentation delays and the number of trials being as described in the companion paper.

Data Analysis. Movement timing (EMG onset, 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 movement trial, as described in Williams et al. (1998). Trials in which spontaneous activity was seen in the 500 ms monitoring period 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.

Detection performance data were analysed as in the previous and companion papers. To examine absolute differences in detection performance between a test task (passive movement, movement without the position detecting apparatus, or isometric contractions) and its reference task (isotonic movement), the proportion of stimuli detected during rest trials and during movement trials was compared between tasks. Since all motor task/stimulation site combinations showed time-dependent decreases in detection performance, this first comparison, although usually useful, was potentially confounded by sampling differences (differences in the average timing of the stimuli relative to the motor response). To minimise differences due to sampling, data were grouped into 20 ms bins relative to EMG or movement onset, and logistic functions fitted to the resulting data points. A difference function was then generated by subtracting the logistic function describing perceptual performance over time for the test task from that

describing the reference task. The difference function was then compared to the appropriate value of the Kolmogorov-Smirnov statistic to see whether or not it reached significance ($p<0.05$). All comparisons were made using the same group of subjects for both the test task and reference task, except for D2 abduction with shoulder stimulation in which different groups of subjects performed the test task and the reference task.

In addition to time dependent reductions, a non-time dependent reduction in the proportion of stimuli detected during active movement trials has been postulated (Williams et al. 1998). To verify whether or not this non-time dependent reduction in the proportion of stimuli detected was also present in other types of motor tasks, detection performance from the three earliest bins in all of the test tasks was compared to detection performance at rest as well as to detection performance in the three earliest bins from the reference task.

RESULTS

A total of 61 experiments (n = 21 subjects) using the 8 different motor task/stimulation site combinations were analysed. Subjects reported having detected a stimulus in 93.5% of rest + stimulation trials, 44.3% of movement + stimulation trials and 0.25% of catch trials. 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 task/stimulation site combinations, significantly fewer stimuli were detected during movement trials than in rest trials ($p<0.001$). The results of time course analyses of detection performance as well as comparisons between test tasks and reference tasks are given below.

Isotonic versus isometric D2 abduction.

The ability to detect near threshold stimuli applied to D2 was measured in seven subjects during isotonic D2 abduction and an isometric abduction attempt with D2 already maximally abducted. EMG onset correlated well with the onset of movement for the isotonic task ($r=0.94$). As detailed in Table 1, the timing 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 in the isotonic task (0.55 vs. 0.43), but 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. 2A,B), both tasks were found to produce similar reductions in detection performance. For both tasks, peak decreases occurred around the time

of EMG onset (+2 ms, -4 ms), and the estimated minimum proportion of stimuli detected after EMG onset approached 0. The peak values of the difference between the logistic functions describing detection performance over time in each of the motor tasks did not reach statistical significance (Fig. 2C).

To gain an appreciation of the extent of body surface over which detection performance was affected during the isometric motor task, the ability of five of the nine subjects to detect stimuli delivered to a more remote site (the ipsilateral shoulder) while performing the isometric D2 motor task was evaluated. For this comparison only, the same subjects did not perform the reference motor task; instead the results were compared to data previously collected and published using the same stimulation site and the reference isotonic active D2 abduction motor task (Williams et al. 1998). There was no difference in the proportion of stimuli detected during rest + stimulation trials and movement + stimulation trials across the isotonic and isometric motor tasks (Table 2). The timing of EMG onset relative to the Go cue was also not significantly different. For the isotonic task, movement onset and EMG onset correlated well ($r=0.97$). As reported previously, isotonic D2 abduction produces a weaker and later time-dependent reduction in the detection of stimuli applied to the shoulder as compared to the moving digit (Figs 2A, 3A). Results for both the timing and magnitude of reductions in detection for the isometric task were similar (Fig. 3B), and the difference function (Fig. 3C) showed that there was no significant difference in detection performance over time when the isotonic and isometric results were compared.

These results suggest that the spatio-temporal gradient of reductions in detection described previously for isotonic D2 abduction (Williams et al. 1998) also holds for isometric D2 abduction attempts.

In summary, isometric D2 abduction attempts produced reductions in detection performance, the magnitude and timing of which was 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. In addition, these results suggest that the spatio-temporal gradient of reductions in detection described previously for isotonic D2 abduction (Williams et al. 1998) may be generalisable to isometric D2 abduction attempts.

Active versus passive D2 abduction.

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 was compared to the results obtained during active movements (9 subjects). Table 3 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 was almost halved. Overall detection performance at rest and during movement trials was not significantly different for active and passive movement. Analyses of the time course of observed reductions in the proportion of stimuli detected during movement + stimulation trials are shown in Fig. 4A,B, in this case plotted relative to movement onset as there was no EMG activity in the passive movement trials. The magnitude of the decrease in detection was virtually identical for active and passive movements (predicted minima of 0.01 and 0.00, respectively). Surprisingly, the time course was also similar, with peak decreases preceding movement onset in the active (-44 ms) and passive (-36 ms) conditions. The difference between the logistic functions describing detection performance in the active and passive tasks (Fig. 4C) was not significant. The results of these comparisons indicate that the peripheral afference generated by passive D2 abduction can produce reductions in detection not significantly different from those produced by active D2 abduction, i.e. that central motor preparation and commands are not necessary to explain observed movement-related decreases in tactile detection.

Effect of position-detection apparatus on detection.

In the interest of determining the effect of extraneous cutaneous feedback generated by the digit resting on the position-detection apparatus on detection performance, the subjects that performed the D2 passive task were re-tested in the D2 active task with D2 resting unsupported by the subjects' side. Detection performance was compared to results obtained with active D2 abduction using the position-detection apparatus. D2 stimulation was used in both conditions. Although most movement parameters were not available for the test task, the timing of EMG onset was not significantly different between the two tasks (Table 3). Overall detection performance in both the rest and the movement trials was not significantly different between conditions. Both Fig. 5A (with apparatus) and Fig. 5B (without apparatus) show similar reductions in detection performance and in the timing and detection performance parameters of the logistic functions that describe them. The difference between the two functions (Fig. 5C) never attained the level of significance. 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.

Active versus passive elbow extension.

In order to determine if the results of the active/passive comparison could be extended to movements about other joints, three subjects were tested during active and passive elbow extension. We chose to study elbow movements because previous experiments in monkeys had determined the time course of reductions in somatosensory transmission during active and passive elbow movements (Chapman et al. 1988). As in the latter study, stimuli were applied to the dorsal forearm. Both active and passive movements significantly decreased the proportion of stimuli detected as compared to detection at rest (Table 4, $p<0.01$). Active extension at the elbow produced a time-dependent reduction in detection performance, with the peak decrease in detection occurring approximately 100 ms before movement onset (Fig. 6A) or 20 ms before EMG onset (Fig. 6B). When movement timing and kinematic values as well as detection performance were compared between the active and passive tasks (Table 4), no significant differences were observed. Figure 6C shows detection performance over time during passive extension at the elbow. Passive extension also produced a time-dependent reduction in detection performance. As seen with D2 abduction, both the time course (peak decrease at –97 ms) and magnitude of the time dependent decrease in detection were similar to those observed with active movement. Again, no significant differences between the functions describing detection during active and passive elbow extension were observed (Fig. 6D).

Did changing the movement from D2 abduction about the metacarpophalangeal joint to forearm extension about the elbow joint significantly change the detection function over time during movement? Figure 7 partially answers this question. The difference function in Fig. 7A revealed significant differences ($p<0.01$), reflecting large differences in the timing of reductions in detection between elbow extension (110 ms before movement onset) and D2 abduction (44 ms before movement onset) when these were plotted *relative to movement onset*. This result appeared to indicate that changes in the movement being performed significantly changed the timing of reductions in detection. However, Fig. 7B shows the difference function for detection performance over time plotted *relative to EMG onset*. When this realignment of the data was performed, differences in the timing of peak decreases (23 ms before EMG onset for elbow extension vs. 2 ms after EMG onset for D2 abduction) were considerably less, and a significant difference in detection performance was no longer observed. This result suggests that, provided detection performance is plotted relative to EMG onset, the spatio-temporal gradient of reductions in detection described previously for isotonic D2 abduction (Williams et al. 1998) may be generalisable to movements about other joints.

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, the timing and amplitude of movement-related decreases were similar for movements of different body parts (digit versus arm), when data were aligned relative to EMG onset.

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 was 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. This was explained by the experimental set-up in which two test apparatuses were yoked, the mass to be displaced in the passive task by the helper's abduction movement therefore being approximately doubled. In both tasks, performance during movement fell to 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 because identical results were also obtained in the other active vs. passive comparison (elbow extension), in which case the kinematics were not significantly different.

Subtle differences in detection performance in the different experimental conditions may have been obscured by the choice of stimulus intensity and the magnitude of the reduction in detection. As shown in the companion paper, however, stimulus intensity did not affect the timing of peak reductions in detection for active isotonic D2 abduction. In addition, shifting the site of stimulation from D2 to the shoulder, where a weaker and later reduction in detection was observed with isotonic D2 abduction, failed to reveal a difference between the isometric and isotonic motor tasks. These supplementary findings give an indication that the absence of difference across the various comparisons was most likely a robust observation.

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 afference). 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 reticular nucleus (Jones and Wise 1977) and motor cortical projections to the dorsal column nuclei (DCN) and surrounding reticular formation (Bentivoglio and Rustioni 1986; Cheema et al. 1985; 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 neurones 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 primary somatosensory 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 1971; 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, whereas movement-related peripheral afference only plays a role in reducing the responsiveness of the somatosensory system after the onset of peripheral feedback.

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 the active motor tasks, and a shift in the timing of the peak decrease to after the onset of movement in the passive tasks. Instead, we found that the peak decrease in detection preceded the onset of passive movement by 36 ms for D2 abduction. This result was confirmed by results obtained during passive elbow extension, where the peak decrease in detection occurred 97 ms before movement onset. In neither case was there a difference when comparing active and passive movement. The results thus suggest that central motor commands are not necessary for reductions in perception before the onset of movement. The results also suggest that there is not a one to one link between changes in the amplitude

of short latency SEPs in sensory thalamus and primary somatosensory cortex and changes in perception: thalamic and cortical SEPs show no change prior to passive movement, but our results indicate that tactile detection is decreased prior to movement onset.

How then to explain the decrease in detection before the onset of passive movement? One possibility is that the Go cue (light), which informed the subjects of the beginning of the trial, may have triggered central gating even in the passive condition. This seems unlikely for two reasons. First, we previously showed that there is no movement-related gating of detection for stimuli applied to distant sites (e.g. contralateral arm) even though the light cue was presented in each trial (Williams et al. 1998). Secondly, such a mechanism would imply a constant temporal relationship between the light (Go cue) and the peak changes in detection. Although the timing of the peak decrease for the D2 abduction tasks was similar for the active and passive versions (respectively 208 and 195 ms after the light), there was a large difference for the elbow task, with peak decreases occurring 244 ms after the light in the passive condition as compared to 196 ms in the active. This contrasted with virtually identical latencies (-44 and –36 ms respectively) when measured relative to movement onset. Together, these observations make it unlikely that a central gating signal could explain the results obtained in the passive task.

It seems more likely that the reductions in detection observed during passive movement are generated by movement-related peripheral reafference. In order to reconcile the results with observations of no change in the earliest component of the cortical SEP before movement onset (Chapman et al 1988), it is suggested that gating influenced the response to the test stimulus at some point *after* the stimulus had traversed the relay nuclei but *before* conscious perception of the stimulus was established. This temporal sequence of events has previously been proposed in studies examining "masking" in the somatosensory system, whereby the perception 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 ~ 100 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 (up to 140-180 ms after the onset of the test stimulus). Further to this, there are suggestions in the literature to the effect that conscious perception 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 perception 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 perception 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 which 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 both this study and in Williams et al (1998) is not apparent in masking studies. Instead, maximum decreases in perceptual 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 during a passive movement task temporal shifts in the timing of peak decreases as distance increases are still observed, or alternatively if the timing of decreases remains relatively constant.

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.

Sources of afference that are present in active isotonic movement but almost completely eliminated in active isometric contractions include movement-related cutaneous afference and antagonist tendon organ or spindle discharge. An important advantage inherent to the design of our isometric task, which placed the agonist in a shortened position, was the elimination of added cutaneous discharge generated when a body part exerts force against an immovable object (this being an important confounding factor in previous studies of isometric movement). 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 anaesthesia on movement-related reductions in tactile perception (Schmidt et al. 1990b). Furthermore, during isometric movement agonist tendon organ and spindle discharge 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 afference in the passive movement task. These two mechanisms may or may not coexist.

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 afference 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.

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 which 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 afference is expected, that is before movement onset at the *expected* time of agonist EMG onset (Figs. 1A and 4A,B for D2 abduction; 6A-C for elbow extension). 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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TABLES

Table 1: Temporal parameters, kinematic parameters and detection performance for isotonic and isometric D2 abduction tasks, D2 stimulation (7 subjects).

	Isotonic	Isometric	p value
Temporal			
Movement onset	239 ± 30 ms	--	--
1st DI EMG onset	191 ± 25 ms	194 ± 29 ms	0.85
Movement duration	213 ± 88 ms	--	--
Kinematic			
Peak Amplitude	32 ± 6.5 ^{0}	--	--
Peak Velocity	375 ± 86 ^{0}/sec	--	--
Peak Acceleration	6900 ± 1300 ^{0}/sec^2	--	--
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.

Table 2: Temporal parameters, kinematic parameters and detection performance for isotonic (9 subjects) and isometric (5 different subjects) D2 abduction tasks, shoulder stimulation .

	Isotonic	Isometric	p value
Temporal			
Movement onset	241 ± 30 ms	--	--
1st DI EMG onset	184 ± 17 ms	216 ± 44 ms	0.08
Movement duration	238 ± 123 ms	--	--
Kinematic			
Peak Amplitude	22 ± 6^{0}	--	--
Peak Velocity	$272 \pm 72\ ^{0}$/sec	--	--
Peak Acceleration	5700 ± 1550 ^{0}/sec^{2}	--	--
Detection performance			
Rest	0.93 (0.90, 0.96)	0.94 (0.89, 0.97)	0.51
Motor task	0.60 (0.57, 0.64)	0.61 (0.56, 0.66)	0.40

Table 3: Temporal parameters, kinematic parameters and detection performance for active D2 abduction, passive D2 abduction and active D2 abduction without the position detection apparatus (9 subjects), D2 stimulation.

	Active	Passive	Act. Vs Pas. p value	No apparatus	Act. vs No app p value
Temporal					
Movement onset	252 ± 45 ms	235 ± 23 ms	0.27	--	--
1st DI EMG onset	205 ± 50 ms	--	--	175 ± 120 ms	0.33
Movement duration	170 ± 70 ms	218 ± 50 ms	0.14	--	--
Kinematic				--	--
Peak Amplitude	29 ± 5^{0}	32 ± 4^{0}	0.12		
Peak Velocity	365 ± 90^{0}/sec	270 ± 50^{0}/sec	0.02	--	--
Peak Acceleration	$6200 \pm 1650^{0}/sec^{2}$	$3300 \pm 750^{0}/sec^{2}$	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

Table 4: Temporal parameters, kinematic parameters and detection performance for active and passive elbow extension tasks (3 subjects), forearm stimulation.

	Active	Passive	p value
Temporal			
Movement onset	306 ± 30 ms	341 ± 23 ms	0.37
Triceps EMG onset	186 ± 10 ms	--	--
Movement duration	553 ± 138 ms	532 ± 53 ms	0.87
Kinematic			
Peak Amplitude	89 ± 20 ^{0}	69 ± 19 ^{0}	0.06
Peak Velocity	244 ± 42 ^{0}/sec	193 ± 52 ^{0}/sec	0.20
Peak Acceleration	1540 ± 320 ^{0}/sec^2	1150 ± 250 ^{0}/sec^2	0.30
Detection performance			
Rest	0.94 (0.88, 0.97)	0.94 (0.89, 0.98)	0.49
Motor task	0.25 (0.21, 0.30)	0.30 (0.26, 0.35)	0.06

FIGURE LEGENDS

Fig. 1A,B. A: Experimental position for isometric D2 abduction. A rubber puck was placed between D2 and 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.

Fig. 2A-C. Comparison of the effects of active, isotonic D2 abduction and active, isometric D2 abduction on the detection of stimuli applied to the moving digit in seven subjects. A,B: detection performance over time is plotted relative to the onset of EMG (20 ms precision). Error bars represent the 95% confidence intervals. The shaded area shows the 95% confidence interval for perceptual performance in the rest + stimulation trials. Filled symbols indicate that perceptual performance during movement+stimulation trials was significantly lower than that observed at rest ($p<0.01$); open symbols, no change. The solid lines represent logistic functions fitted to the data points. Logistic equation parameters are also shown. C: function representing the difference between the logistic equations shown in A and B. The dashed lines represent the critical value ($p<0.05$) for the Kolmogorov-Smirnov test statistic. Data using active, isotonic D2 abduction are a sub-set of data previously published in Williams et al. (1998, Fig. 5).

Fig. 3A-C. Comparison of the effects of active, isotonic D2 abduction in nine subjects and active, isometric D2 abduction in five different subjects on the

detection of stimuli applied to shoulder of the limb ipsilateral to the moving digit. Data using active, isotonic D2 abduction were previously published in Williams et al. (1998, Fig. 6E). Plotted as in Fig. 2.

Fig 4A-C. Comparison of the effects of active and passive D2 abduction on the detection of stimuli applied to the moving digit in nine subjects. Detection performance over time is plotted relative to the onset of movement. Data using active, isotonic D2 abduction are a sub-set of data previously published in Williams et al. (1998, Fig. 5).

Fig. 5A-C. Comparison of the effects of active D2 abduction with (data from figure 4A) 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. 2.

Fig. 6A-D. Comparison of the effects of active and passive elbow extension on the detection of stimuli applied to the dorsal forearm in three subjects. Detection performance is plotted relative to movement onset (A,C,D) or EMG onset (B).

Fig. 7A,B. Difference functions comparing detection performance during active, isotonic D2 abduction and elbow extension relative to movement onset (A) or EMG onset (B).

FIGURE 1

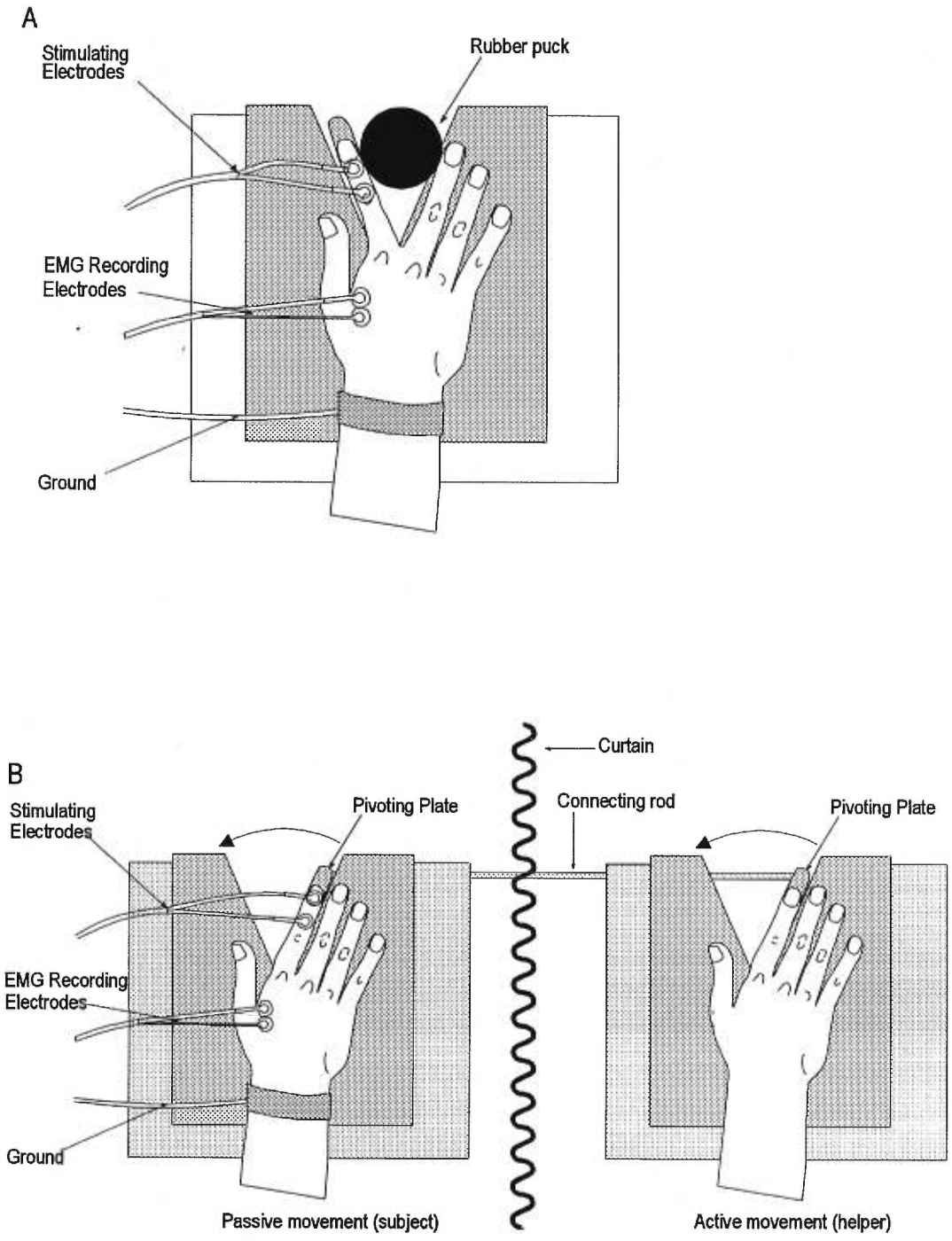

A
Stimulating Electrodes
Rubber puck
EMG Recording Electrodes
Ground
B
Curtain
Stimulating Electrodes
Pivoting Plate
Connecting rod
Pivoting Plate
EMG Recording Electrodes
Ground
Passive movement (subject)
Active movement (helper)

FIGURE 2

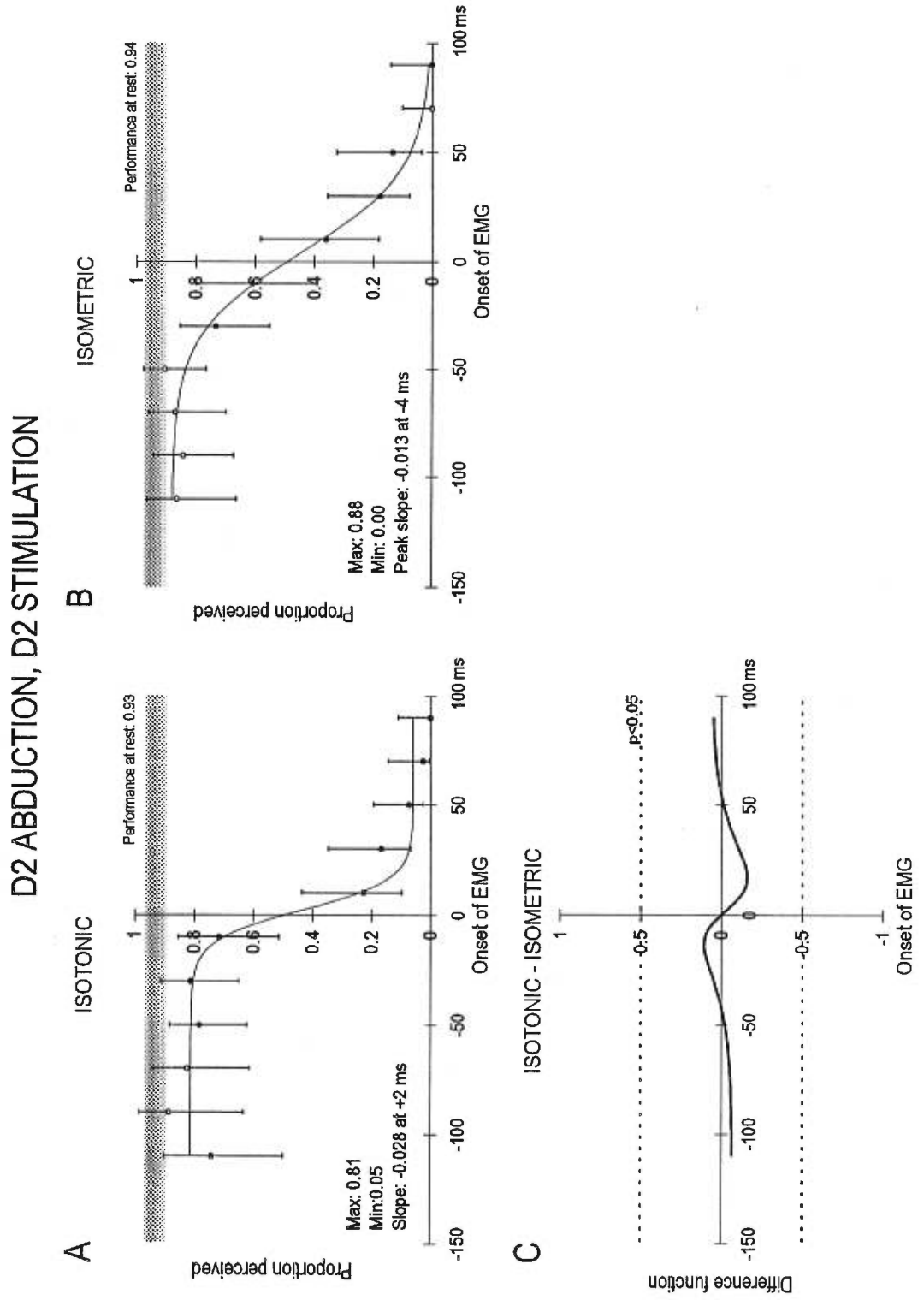

D2 ABDUCTION, D2 STIMULATION
A
ISOTONIC
Performance at rest: 0.93
Proportion perceived
Max: 0.81
Min:0.05
Slope: -0.028 at +2 ms
Onset of EMG
B
ISOMETRIC
Performance at rest: 0.94
Proportion perceived
Max: 0.88
Min: 0.00
Peak slope: -0.013 at -4 ms
Onset of EMG
C
ISOTONIC - ISOMETRIC
p<0.05
Difference function
Onset of EMG

FIGURE 3

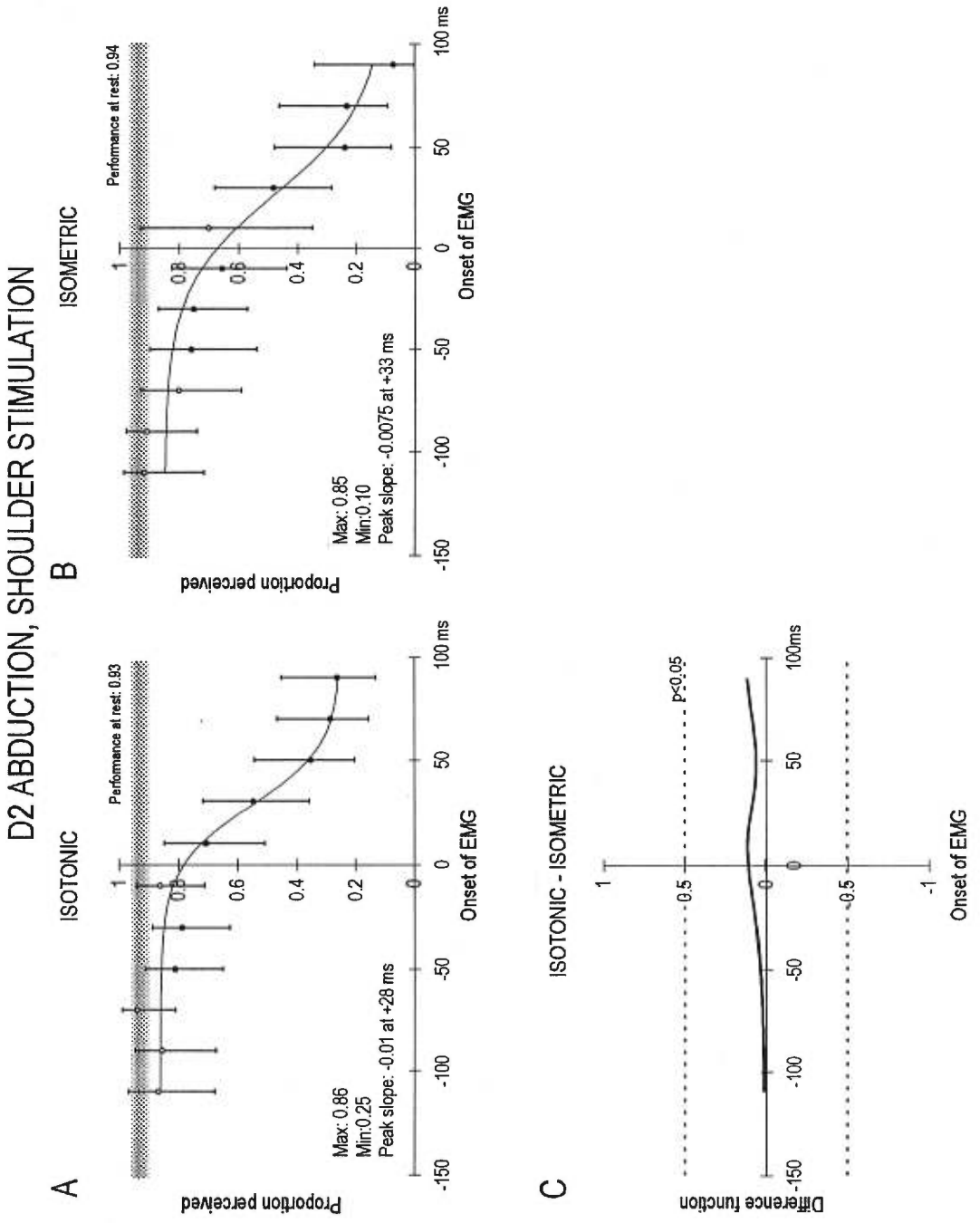

D2 ABDUCTION, SHOULDER STIMULATION
A
ISOTONIC
Performance at rest: 0.93
Proportion perceived
Max: 0.86
Min:0.25
Peak slope: -0.01 at +28 ms
Onset of EMG
B
ISOMETRIC
Performance at rest: 0.94
Proportion perceived
Max: 0.85
Min:0.10
Peak slope: -0.0075 at +33 ms
Onset of EMG
C
ISOTONIC - ISOMETRIC
p<0.05
Difference function
Onset of EMG

FIGURE 4

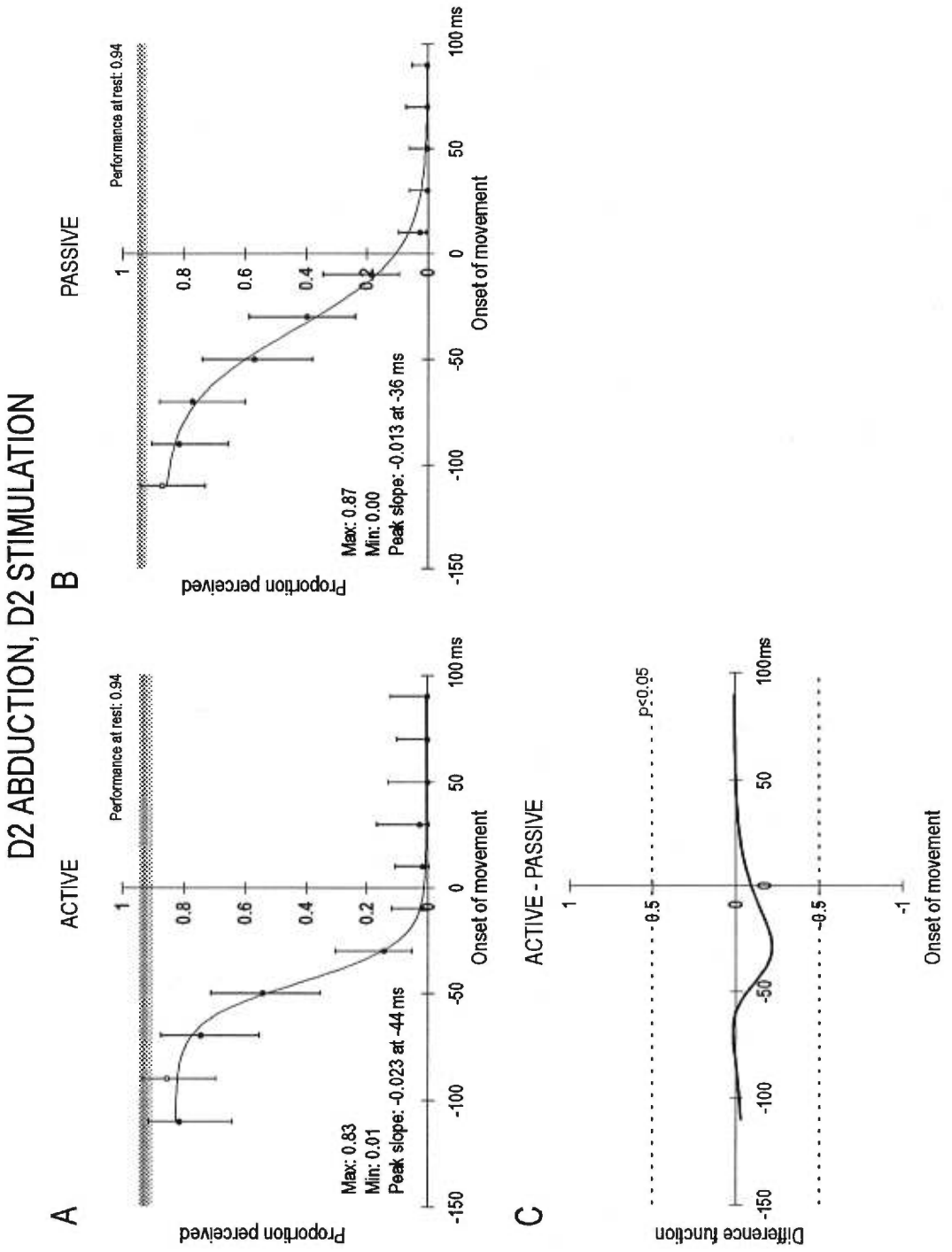

D2 ABDUCTION, D2 STIMULATION
A
ACTIVE
Performance at rest: 0.94
Proportion perceived
Max: 0.83
Min: 0.01
Peak slope: -0.023 at -44 ms
Onset of movement
100 ms
B
PASSIVE
Performance at rest: 0.94
Proportion perceived
Max: 0.87
Min: 0.00
Peak slope: -0.013 at -36 ms
Onset of movement
100 ms
C
ACTIVE - PASSIVE
p<0.05
Difference function
Onset of movement
100ms

FIGURE 5

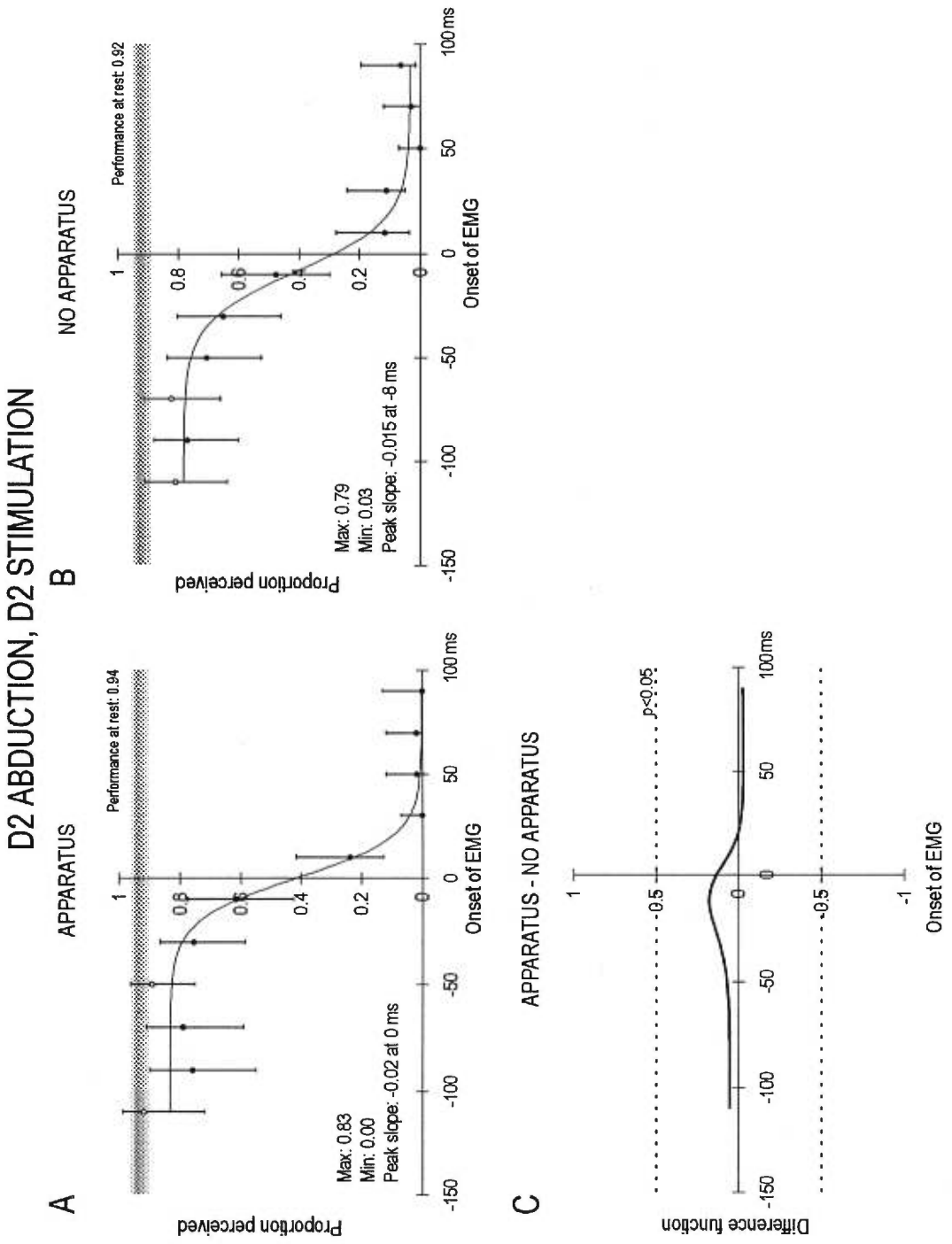

D2 ABDUCTION, D2 STIMULATION
A
APPARATUS
Performance at rest: 0.94
Proportion perceived
Max: 0.83
Min: 0.00
Peak slope: -0.02 at 0 ms
Onset of EMG
B
NO APPARATUS
Performance at rest: 0.92
Proportion perceived
Max: 0.79
Min: 0.03
Peak slope: -0.015 at -8 ms
Onset of EMG
C
APPARATUS - NO APPARATUS
p<0.05
Difference function
Onset of EMG

FIGURE 6

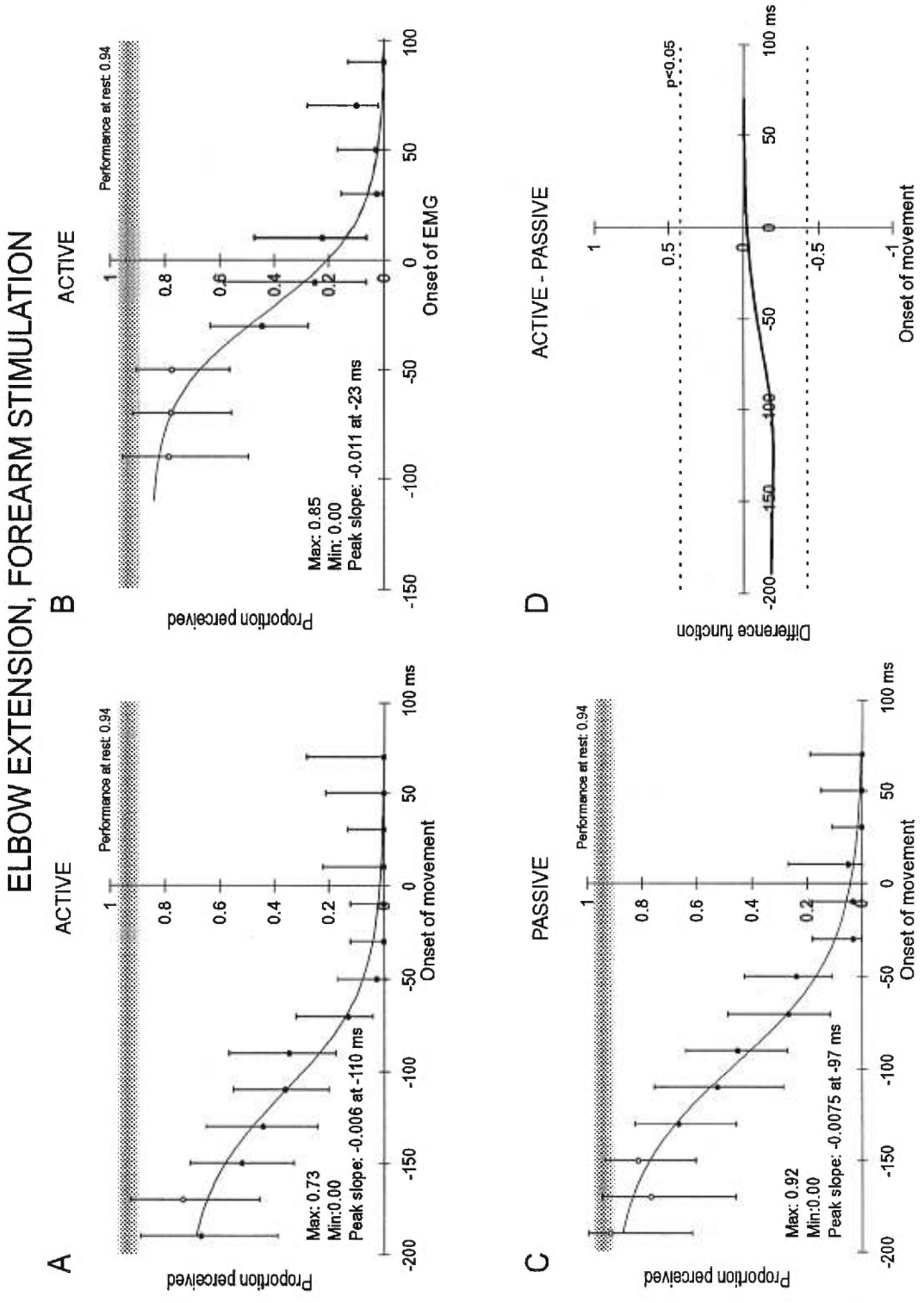
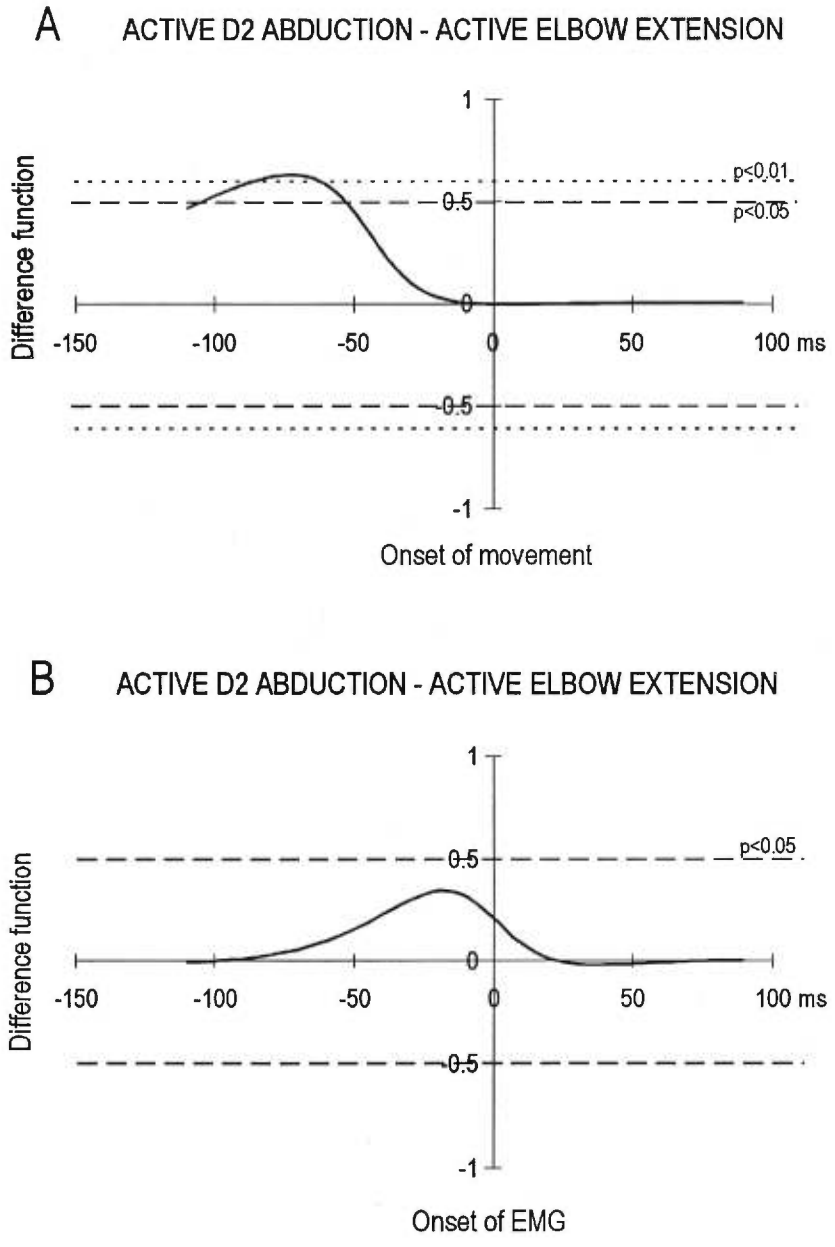

ELBOW EXTENSION, FOREARM STIMULATION
A
ACTIVE
Performance at rest: 0.94
Proportion perceived
Max: 0.73
Min:0.00
Peak slope: -0.006 at -110 ms
Onset of movement
B
ACTIVE
Performance at rest: 0.94
Proportion perceived
Max: 0.85
Min: 0.00
Peak slope: -0.011 at -23 ms
Onset of EMG
C
PASSIVE
Performance at rest: 0.94
Proportion perceived
Max: 0.92
Min:0.00
Peak slope: -0.0075 at -97 ms
Onset of movement
D
ACTIVE - PASSIVE
p<0.05
Difference function
Onset of movement

FIGURE 7


A
ACTIVE D2 ABDUCTION - ACTIVE ELBOW EXTENSION
Difference function
1
0.5
0
-0.5
-1
p<0.01
p<0.05
-150
-100
-50
0
50
100 ms
Onset of movement
B
ACTIVE D2 ABDUCTION - ACTIVE ELBOW EXTENSION
Difference function
1
0.5
0
-0.5
-1
p<0.05
-150
-100
-50
0
50
100 ms
Onset of EMG

CHAPTER 5: GENERAL DISCUSSION

In this thesis, the influence of stimulus location, intensity, timing relative to the onset of EMG or movement, and the influence of the motor task itself were all quantified with regards to their effect on the detection of tactile stimuli during the performance of a motor task. The influence of stimulus-related parameters on detection performance during movement were described using modified logistic functions, and these functions were combined into a more general model which in turn provides a quantitative description of the relative importance of each of these factors on detection performance. Stimulus location was found to affect both the timing and the magnitude of movement-related reductions in tactile detection. Stimulus intensity was found to affect only the magnitude of movement-related reductions in tactile detection. In the studies that examined each of these factors, stimulus timing relative to EMG onset determined to a great extent the probability that a stimulus would be detected or not, with stimuli delivered after EMG onset being much more affected by reductions in detection than stimuli which preceded EMG onset. When movements of different body segments were studied (D2 abduction vs. elbow extension), the timing of reductions in detection varied significantly in relation to movement onset, but not in relation to EMG onset, suggesting that reductions in detection are coupled to the motor command and/or muscular activity, and not to the actual movement. However, for movement about a given body segment, active isotonic, isometric, or even passive versions of the movement/motor task all produced reductions in detection with a similar time course when measured relative to the same peripheral movement-related event.

The similarity in the time courses of reductions in detection during the isometric motor task and its isotonic equivalent indicates that movement itself is not necessary to produce observed levels of motor-task-related reductions in tactile detection. The time course of reductions during passive movements indicates that movement alone is, however, sufficient to produce the reductions in detection observed during active movement[1]. Taken together, these results seem to confirm that movement-related reductions in tactile detection are mediated by several different mechanisms, that putative "gating signals" can be both central and peripheral in origin, and that whatever the origin of the gating signal, a functional convergence serves to produce similar movement-related gating effects.

5.1 *Methodological considerations*

5.1.1 Psychophysical Methods.

In this thesis, a tactile detection task was used to measure movement-related decreases in the perception of test stimuli. Signal detection theory (Green and Swets 1988) provides a ready-built framework for the analysis of the processes involved in any signal detection task. It divides the factors influencing subject performance during this type of task into two categories. The first category attempts to model the ability of the detection apparatus (the somatosensory system in the case of our studies) to distinguish a test signal

[1] Unless "mental rehearsal" of movements is affecting results (discussed in chapter 4, control experiments forthcoming).

(stimulus) from the background noise. The second category models the more "cognitive" components of the task (attention, motivation, guessing strategy, memory), which can independently affect perceptual judgements, as a "bias" signal. In any signal detection task, within a given trial, either a signal is presented or no signal is presented. Therefore, a signal detection trial can have one of four outcomes: identification of a signal (hit), signal miss, correct rejection, or false alarm. In signal detection theory, a popular representation of the importance of each set of factors is the ROC (receiver operating characteristic) curve, a two-dimensional graph with the proportion of false alarms on the abscissa and the proportion of hits on the ordinate. When ROC curves are applied to perceptual judgements, different curves represent different signal receivers or different signal intensities, whereas different points along the same ROC curve represent cognitive variations such as modifications in the level of attention, the strategy of the subject, etc.

It follows from this description that the proportion of stimuli detected in any psychophysical task depends on many factors, such as the physical characteristics of the receiver, the physical characteristics of the signal, and the nature of the bias signal. Any study of detection should control or at least measure not only those factors that are under study, but also all other factors which determine detection performance, in order for modifications in those factors which are of interest to be interpretable. Our study examined the effect of stimulus parameters on the time course and the magnitude of movement-related

reductions in tactile detection. In order to effectively accomplish this task, the ensemble of factors determining subject "bias" needed to be kept constant throughout the experimental session. To express our goal in terms of ROC curves, the goal of the tactile detection task was to measure points on different ROC curves rather than points along a given ROC curve. One way of ensuring bias-independent results is to use the gold standard for bias-independent measures of detection performance: the 2-alternative-forced-choice (2AFC) trial. Most of the advantages and disadvantages of 2AFC trials, the advantages and disadvantages of the type of detection trial used in these experiments, and the equivalence of perceptual measures obtained using the two methodologies, were described in the Methods of the first paper. An additional difference between the type of trial used in this thesis and 2AFC trials is the ability of the latter to evaluate both conscious and non-conscious detection of stimuli (for example (Libet et al. 1991), for details see section 5.5 below). In practice, the experimental methods used in this thesis produced extremely steep ROC curves with minimal positive or negative bias. Thus, using signal-detection theory terminology, subjects behaved very close to "perfect receivers" at rest. One this had been ascertained, is was then possible to evaluate the data without the need of analysis techniques developed from signal detection theory.

A magnitude estimation task was used to measure the perceived intensity of suprathreshold stimuli during the performance of a movement task. The ability of human subjects to relate physical attributes of a stimulus to number

scales is well established (Stevens 1975). To prevent "boundary effects", an infinite number scale was used in these experiments. The results from the magnitude estimation experiments were presented as proportional ratings with the average magnitude at rest being normalised to 1. This was done to facilitate inter-intensity and inter-subject comparisons of the effect of movement on scaling performance. Power functions were not fitted to the magnitude estimates provided by the subjects at different stimulation intensities since different subjects were used at different intensities, each with their own set of values for perceptual ratings, and also because only 2 stimulus intensities were tested.

5.1.2 Electrical stimulation

One question which much be asked when dealing with electrical stimuli is: what type(s) of afferents are activated by the electrical pulse? In the case of this series of experiments, the type of electrical stimulation (weak, single DC pulses delivered through surface electrodes) limits the possible structures activated by the pulse. In general, electrical currents selectively activate the largest diameter and closest nerve fibres. For stimuli delivered to the fingers, the most common large diameter afferent fibres are cutaneous mechanoreceptive afferents innervating Merkel and Meissner type mechanoreceptors , although afferents innervating Pacinian, Ruffini, joint, and Golgi tendon organs may also have been activated (reviewed in Darian-Smith 1984). When stimuli were delivered to other

parts of the body covered by hairy rather than glabrous skin, the stimulus may have activated hair follicle afferents as well, in lieu of Meissner afferents. This difference in receptor types was not reflected by differences in the subjective report of the quality of sensation produced by electrical stimulation when different areas were stimulated, most subjects reporting the sensation as either an extremely weak "shock" sensation or as a rather undefinable extremely short-lasting "next thing to nothing" sensation, somewhat like being lightly brushed against by an insect. Another difference for the hairy skin stimulation sites was the presence of underlying muscle. In no case was muscular contraction produced by the electrical stimulation in these experiments, and muscular afferents, being situated deeper beneath the skin than cutaneous receptors, were much less likely to have been activated by the near-threshold electrical stimulation.

5.1.3 Motor task

The major motor task, D2 abduction, was chosen for several reasons. Firstly, it produced a relatively discrete activation of intrinsic hand muscles. Secondly, it could be easily adapted to passive and isometric versions. Thirdly, the experimental goal of evaluating the spatial gradient of the gating actions was simplified by choosing a motor task at a very distal site on the arm. The companion motor task of elbow extension motor task was primarily chosen because this movement has been extensively evaluated in previous gating studies

(Chapman et al. 1987, 1988; Jiang et al. 1991; Jiang et al. 1990; Feine et al. 1990), facilitating the linking of the psychophysical results of this thesis to previous psychophysical and electrophysiological work. It provided a contrast to D2 abduction since the movement involved a more proximal joint, the elbow, and so allowed evaluation of sites distal to the body part in motion (see below).

5.1.4 Statistical methods.

Most of the statistical methods used in this thesis were relatively standard. One exception was the test used to evaluate whether the curves modelling perceptual performance over time in different motor tasks were significantly different or not. The Kolmogorov-Smirnov (K-S) statistic was chosen for several reasons. The first is simplicity. The second is that the method is quite sensitive to large average differences, and just as sensitive to large peak differences such as would occur if there was an important difference in the timing of peak decreases in detection. It was recognised that the K-S statistic is relatively insensitive to moderate differences occurring over many points, but this generally was not the case for our data.

5.2 *Extending the model of detection performance.*

The complete model of detection performance presented in equation 5 of Chapter 4 provides a precise description of the effects of stimulus timing, intensity and location on detection performance during the performance of an active isotonic abduction of D2. Logistic functions were chosen to model detection performance as they represent the most basic equation which describe boundary limited growth and/or decreases. The model was designed in a manner that the influence of individual parameters on overall detection performance can be easily re-optimised, giving the basic structure of the model a great deal of flexibility in the event it is reused to model detection performance in subjects performing different motor and perceptual tasks. Obvious parameters which could be experimentally investigated in terms of their effect on tactile detection during movement, then modelled and integrated into the overall model structure, include, for example, stimulus pulse number and train duration, and the surface area over which stimuli are delivered. Whether or not the model is generalisable to other movements and stimulus modalities awaits the generation of similar quantities of data with other motor and perceptual tasks. However, little difference in detection performance was observed when active D2 abduction, upon which the model was based, was replaced by isometric or passive equivalents, or even a completely different motor task (elbow extension, see Chapter 4). This suggests that a description of detection performance similar to the present model may adequately

describe actual detection performance during isometric and passive D2 abduction as well as active and passive elbow extension.

One difficulty which may be encountered when generalising the model to other types of motor tasks is defining distance for points at different locations along the body segment in motion. For D2 abduction, zero was defined as the "body part in motion", i.e. the entire D2 was at distance zero. In an elbow extension task, however, the segment put in motion (the forearm) is much larger, and it is not certain that reductions in detection are equivalent throughout the segment (e.g. Post et al 1994). One obvious alternative is that zero is the joint at which the movement takes place. Unfortunately, elbow extension is not an ideal task to study this question, because although the segment in motion is much longer than a finger, it is still only 0.2 body lengths on average. Movements about the shoulder would be preferable for this purpose, as a longer segment would be put into motion. However, preliminary evidence indicates that distance-dependent shifts in timing do not occur distal to the joint producing the movement. Data from 12 subjects were gathered during elbow extension, using stimuli delivered to the dorsal forearm or to D2. As can be observed in Figure 1, reductions in the detection of stimuli applied to D2 were similar to reductions observed when stimuli were applied to the mid-dorsal forearm. In particular, the timing of the observed reductions was within a few milliseconds of EMG onset for both tasks, and when the two conditions were compared quantitatively (Fig. 1C), no significant difference was observed.

FIGURE 1

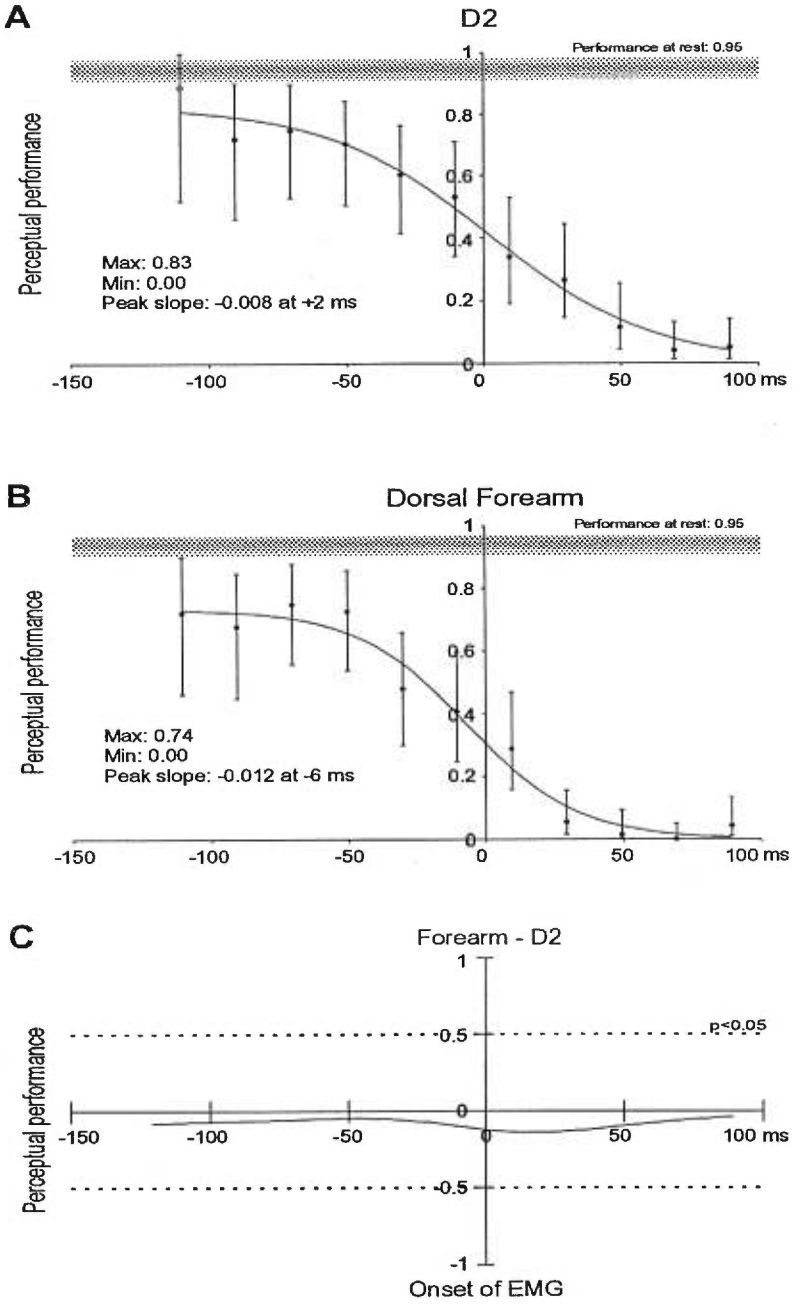


FIGURE 1: Detection performance over time during elbow extension, when the test stimulus was applied to D2 (A) or the dorsum of the forearm (B). A similar reduction in detection was observed at both sites, and the difference function (C) did not reach significance over the range tested.

Reconciling the psychophysical model presented in this thesis with anatomical and electrophysiological data is an attractive but difficult endeavour at this time. For example at the anatomical level, it would be intriguing to correlate the distance-dependence of tactile detection during movement with the actual distances between the central representations of the various parts of the arm at one or several of the somatosensory relays, to see for example whether the effect of distance on detection performance correlates with physical distances between representations of the respective body parts in the relay nuclei or SI, or the terminal branching patterns of descending control pathways. Unfortunately, maps with sufficient precision to perform this type of correlation analysis do not exist at this time for humans (R.W. Dykes, personal communication). Physiologically, it would be equally interesting to see whether the psychophysical results presented in this thesis correlate well with movement-related modifications in longer latency components of the SEPs, and also to analyse the locus of these longer latency components using either electroencephalographic or magnetoencephalographic techniques. Similarly, studies of longer latency neuronal responses could also be correlated with the psychophysical data presented in this thesis.

5.3 *Parallels with movement-related gating in the visual system.*

In the third article of this thesis (Chapter 4), we reported that passive movement produced reductions in detection which preceded the onset of movement, with a time course similar to that seen during active movement. A fascinating parallel with the findings of this thesis exist in the literature exploring the gating of visual stimuli during eye movement. In the early 1960s, movement-related reductions in the detection of visual stimuli were reported (Volkmann 1962). Later, these reductions were further investigated and characterised with regards to their time course relative to the onset of saccadic eye movements (Volkmann et al. 1968). Reductions in the detection of visual stimuli were found to begin 40-60 milliseconds before the onset of the saccade, this at first being interpreted as evidence that corollary discharge related to the motor command inhibits sensory transmission in the visual system (much as similar findings have often been interpreted in studies of movement-related gating of tactile perception). In 1970, the results of an important control experiment in which subjects attempted to detect a visual test flash while the retinal image was displaced *externally* and the eye remained immobile were published (MacKay 1970). A strong parallel between this type of experiment and those involving passive movement in this thesis can be made. In both cases, voluntary movement was eliminated, to be replaced by an externally imposed displacement, of the visual field in one case and the body segment in the other. Remarkably, reductions in visual detection abilities still preceded the onset of external visual

field displacement by about 40 ms, clearly demonstrating that an efference copy is not necessary to produce reductions in visual detection before the onset of visual field displacement. Although their results did not exclude a role for efference copy during active saccades, they did underline the fact that active movement is not necessary to explain even the earliest reductions in visual detection. These conclusions are strikingly similar to those produced in this thesis after analysis of the time course of reductions in tactile detection during passive movement tasks. Therefore, similar mechanisms may underlie movement-related gating seen in different sensory modalities. Whether movement-related gating extends to other sensory modalities than touch and vision (e.g. hearing) awaits experimental investigation.

5.4 *Active vs. passive touch revisited.*

The somatosensory system provides information about objects that we actively explore with our tactile senses (active touch), but also objects that touch us when we are not engaged in tactile exploration (passive touch). The fact that the transmission of tactile information is reduced during movement, leading to a concomitant increase in detection threshold, could lead to the speculation that active touch is "inferior" in its perceptual capabilities to passive touch. In fact, most studies which have compared perceptual abilities using either active or passive touch have found little difference between the two. For example, active

touch is *equivalent* to passive touch for tactile detection (for example Johansson and LaMotte (1983) vs. LaMotte and Whitehouse (1986)). Also, discrimination of complex stimuli is similar with active or passive touch. For example, Vega-Bermudez et al. (1991) found no difference between active and passive touch in the ability of human subjects to recognise embossed letters, and Schwartz et al. (1975) found no difference for 2-D pattern discrimination. Scaling of roughness also does not seem to depend on whether the tactile stimuli are acquired actively or passively Lederman (1981). A few studies have even found that active touch delivered superior performance to passive touch (For example Gibson 1962; Heller 1986). Unfortunately, significant flaws in the methodology of the latter studies allowed greater amounts of information to be acquired by the subjects in the active touch task, thus biasing the results in favour of active touch. In conclusion, the experimental evidence does not support the notion that passive touch is superior to active touch for the detection, discrimination, and scaling of tactile stimuli.

In this thesis, the tactile detection task should be considered as a form of "passive touch" because the test stimulus was generated by an external agent, and not by the subject's own movements. Passive touch is designed to warn the organism that an outside physical body is interacting with it. It is not the result of movement, but of an external agent acting upon the skin, independently of whether or not the organism is producing a movement at the time. The implications of the interaction between stimulus and organism during passive

touch are often highly relevant to the primary goals of an organism (for example survival), and the interaction can occur when noise in the sensory system is at a minimum (i.e. at rest) or when noise in the sensory system is high (i.e. during movement). Equivalent performance in active and passive touch may be achieved using different tactile information processing strategies, depending on context. For example, during passive touch while the organism is at rest, tactile sensitivity to extremely weak stimuli can be maximal because the background noise level is low. During active touch or passive touch during movement, the sensitivity of the somatosensory system is not likely to be maximal because movement-related afference is generated during active exploration, thus increasing the background noise level from which relevant stimuli must be distinguished. During active touch, small decreases in sensitivity can be compensated for by optimisation of exploratory movement strategies, for example by decreasing scan speed to minimise movement-related gating (Vega-Bermudez et al. 1991), by orienting the exploratory segment so that the most sensitive regions contact the object, etc. Such strategies may be largely unconscious, and would involve minimising the irrelevant movement-related feedback, minimising movement-related gating, and optimising stimulus-receptor interactions. On the other hand, for passive touch during active movement, this process of optimisation does not occur. One can conclude from this that the sensitivity of the somatosensory system to passively acquired stimuli should vary according to the background noise level, whereas the sensitivity of the somatosensory system to actively acquired stimuli should vary according to the background noise level and to the level of optimisation of the

active exploratory movement. By this reasoning, since our experimental task is as an evaluation of "passive touch", the level of background noise would be the key peripheral variable determining detection performance in our task.

5.5 *Movement-related gating and the timing of conscious experience.*

The reductions in tactile detection observed before the onset of passive movement allow an unexpected link to be made between the body of work that forms this thesis and the study of consciousness. At first glance, it seems difficult to accept the notion of movement affecting the detection of stimuli presented before the occurrence of the movement. Nonetheless, masking, visual gating, and now tactile gating studies all demonstrate that this can occur. One reasonable interpretation of the fact that stimuli can be rendered undetectable by a movement which occurs several tens of milliseconds after the stimulus was delivered to the skin is that the movement must be affecting the stimulus after it has reached cortex (since a time interval greater than the transmission delay to cortex has occurred), but before the subject has become *conscious* of it. The delay between cortical activation and conscious perception has been studied by Benjamin Libet. By directly stimulating somatosensory cortex in human subjects undergoing neurosurgery, he found that to be consciously perceived, stimuli needed to be delivered for a certain period of time, which was found to depend on stimulus intensity and frequency (Libet et al. 1964). With what Libet considered optimal

frequency and minimal stimulus intensity, the minimum duration of cortical stimulation that produced conscious awareness was approximately 500 ms. Libet interpreted this as the minimum delay for conscious awareness of the stimulation to occur. Several other interpretations have been put forward (Gomes 1998); one of the more plausible is that only at the end of the 500 ms (rather than at the beginning) does the stimulus become adequate for conscious perception. An unmeasured delay then may occur between the time at which the stimulus becomes adequate for conscious perception to occur and the time at which subjects become conscious of it, and this unmeasured delay would represent the true delay between the beginning of potentially consciously perceivable cortical activity and consciousness. This interpretation is compatible with the fact that peripheral stimuli delivered at the end of the 500 ms cortical stimulation were perceived to occur at the same time as the cortical stimulus, whereas peripheral stimuli delivered at the onset of the 500 ms cortical stimulus were perceived to occur 500 ms before the cortical stimulus (Libet et al. 1979). How then to measure the delay between the onset of an adequate stimulus and the time at which subjects become conscious of the stimulus?

As discussed previously, there is a strong parallel to be made between the reduction in tactile detection before passive movement onset and backward masking. Backward masking studies, in turn, are regarded by some as the strongest evidence of the non-instantaneous nature of conscious perception (Gomes 1998), i.e. for the existence of a delay between the onset of adequate

stimulation and conscious perception of the adequate stimulus. The hypothesis is that backward masking of a stimulus is possible during the delay between the beginning of potentially consciously perceivable spatio-temporal patterns of neuronal activity generated by an adequate stimulus and the putative generation of cortical activity which leads to conscious perception of the stimulus no matter what masker is applied. In other words, the masking stimulus prevents the test stimulus from "reaching" consciousness if it is applied within the critical period after cortical responses to the test stimulus begin but before the onset of cortical activity that inevitably leads to conscious perception. The time course of backward masking would represent for any test stimulus a "minimum time to consciousness", which could nicely explain why it is observed to occur with a similar time course in all of the sensory modalities capable of fine temporal resolutions, including vision, hearing (e.g. Viemeister and Plack 1993), and touch (e.g. the time course of the reduction in detection seen with passive movement in this thesis).

Raising the problem of consciousness also leads to another possible interpretation of the experimental results in this thesis. Surprisingly, experimental evidence exists that electrical stimulation of the somatosensory system which cannot be consciously perceived can still be detected with greater than chance accuracy using 2AFC experimental paradigms (Libet et al. 1991). This phenomenon could be termed "blind tactile detection", as it appears phenomenologically analogous to the "blind sight" of subjects who have suffered

lesions to the visual cortex but can still detect, locate, and identify spatial characteristics of visual stimuli without being consciously aware of them (Weiskrantz et al. 1974). It could be argued that our results may represent the effect of movement on "conscious" detection performance rather than "conscious + unconscious" detection performance. One experimental condition (active isotonic abduction of D2 with D2 stimulation) was replicated using 2AFC methodology (paper 1, figure 3), and no difference was seen between the two experimental methods, suggesting that this is not the case. Nonetheless, a replication of this series of experiments using a 2AFC paradigm would complement the present findings, and a comparison of results using the two experimental paradigms would provide a definitive answer to the question of consciousness versus detection within the framework of movement-related reductions in tactile detection.

5.6 *Conclusions.*

Starting from discrepancies in the literature with regards to the timing and magnitude of movement-related decreases in tactile perception, as well as discrepancies between reductions in tactile perception and electrophysiological measures of somatosensory transmission during movement, the present study examined to what extent these discrepancies could be reconciled by differences in stimulation parameters (timing, intensity, location) and differences in the nature

of the motor task. In order to adequately describe the influence of these parameters on detection, a mathematical description of the detection performance resulting from the interaction of these parameters was created. The principal conclusions of this body of work are that:

1) The influence of stimulus timing, intensity and location on detection performance during movement can be adequately described using modified logistic functions. These functions quantify the effects of movement on detection performance in terms of maximum detection performance and minimum detection performance, and the timing and slope of peak reductions in detection. These functions describe the time dependence of movement-related reductions in tactile detection. They adequately describe the finding that stimuli of P_{90} intensity delivered before a critical period, that at the earliest approximately coincided with EMG onset in active movement, were generally detected, while stimuli delivered after this period were not. The effect of stimulus location is also described with a modified logistic function, with stimuli proximal to the site of movement showing distance-dependent gradients in the minimum proportion of stimuli perceived and the *timing* of decreases in detection. Finally, the effect of stimulus intensity is described, with an intensity-dependent gradient in the minimum proportion of stimuli detected being observed.
2) The timing and magnitude of movement-related decreases in tactile detection are not modified when an isometric motor task is substituted for the standard

isotonic motor task, demonstrating that movement itself is not *necessary* to produce motor-task related reductions in tactile detection.

3) The timing and magnitude of movement-related decreases in tactile detection are not modified when a passive motor task is substituted for the standard active motor task, demonstrating that movement alone is *sufficient* to produce motor-task-related reductions in tactile detection even before the onset of the motor task. This finding indicates that movement affects not only the transmission of tactile information to the cortex, but also the non-conscious neural processing of this information and/or the passage of the information into consciousness

It seems likely that discussions of gating in the somatosensory system that attempt to provide a single simple unifying explanation of all available experimental results are doomed to failure, because "gating" is in fact a catch-all term for many different modulatory processes, all occurring concurrently in the tactile system. Rather, it seems more appropriate to consider gating as the sum total of several different types of modulation, which exert inhibitory effects at all levels of the central nervous system and all stages of stimulus processing.

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