TACTILE GATING IN A REACHING AND GRASPING TASK REVEALS SENSORY AND MOTOR INTERACTIONS AFFECTING TACTILE SENSITIVITY OF THE MOVING UPPER LIMB

by

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Abstract

A multitude of events bombard our sensory systems at every moment of our lives. Thus, it is important for the sensory cortex to gate unimportant events. Tactile suppression is a well-known phenomenon defined as a reduced ability to detect tactile events on the skin before and during movement. Previous experiments (e.g., Chapman et al., 1987) found detection rates decrease just prior to and during finger abduction, and decrease according to the proximity of the tactile event to the moving effector. However, there is no consensus regarding the cause of tactile gating. Previous work debated between centrally- vs. peripherally generated mechanisms causing tactile gating under different circumstances resulting in a reasonably good understanding of contributing neural networks. The present dissertation examined how tactile detection changes during a reach to grasp task. In a series of experiments, participants were asked to reach and grasp a cylinder. Tactors were attached to the index finger, the fifth digit and the forearm of both the right and left arm and vibrated at various epochs relative to an imperative “go” tone. These stimulation times were then renormalized post-hoc relative to movement onset. Movement performance was recorded using an infrared camera system tracking infrared emitting diodes. Results showed that detection rates at the forearm decreased before movement onset; whereas at the right index finger, right fifth digit and at the left index finger, left fifth digit and forearm sites no such reduction was observed (participants only moved the right upper limb). These results indicate that the task affects gating dynamics in a temporally- and contextually-dependent manner and implies that feedforward motor planning processes can modify sensory signals shifting the response criterion so that the likeliest response is negative.
Preface

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List of Symbols, Abbreviations or Other

% -- percent

aIPS – anterior intraparietal sulcus

ANOVA – analysis of variance

ANOVA<sub>RM</sub> – repeated measures analysis of variance

BOLD – Blood-oxygen level dependent

BP – Bereitschaftspotential

C – response bias (criterion measure)

cm – centimeter

CNS – central nervous system

cTBS – continuous theta burst stimulation

d’ – sensitivity

DLPFC – dorsolateral prefrontal cortex

DRG – dorsal root ganglion

EMG – electromyography

etc – et cetera

F – F statistic

FA – fast-adapting

FDI – first dorsal interosseus muscle

g – gram

GTO – Golgi tendon organ

H-reflex – Hoffmann reflex

Hz – Hertz, oscillation frequency, cycles/second

IRED – infrared-emitting diode
LCD – liquid crystal diode
LSD – least significant difference
M1 – primary motor cortex
mg/mL – milligrams per milliliter
ms – millisecond
MT – movement time
MRCP – movement-related cortical potential
N – sensory noise distribution
p – significance level
PA – peak acceleration
PD – peak deceleration
PFC – prefrontal cortex
PGA – peak grip aperture
PIP – proximal interphalangeal joint
PV – peak velocity
PVC – polyvinyl chloride
RA – rapidly adapting
rCBF – regional cerebral blood flow
RT – reaction time
S+N – sensory signal and noise distribution
S1 – primary somatosensory cortex
S2 – secondary somatosensory cortex
SA – slow-adapting
SD – standard deviation

SEM – standard error of the mean

SEP – sensory evoked potential

SMA – supplementary motor area

TENS – transcutaneous electrical nerve stimulation

TPA – time to peak acceleration

TPD – time to peak deceleration

TPGA – time to peak grip aperture

TPV – time to peak velocity

TRP – transient receptor potential

TRPA – transient receptor potential A

TRPM – transient receptor potential M

TRPV – transient receptor potential V

TRT – total response time

VPL – ventral posterolateral nucleus of the thalamus

$Z_{fa}$ – $z$-score of false alarm rate

$Z_h$ – $z$-score of hit rate

$\alpha$ – Type I error rate

$\Delta$ – change in a measure

$\eta$ – partial eta squared, effect size measure
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1 Chapter: Introduction

Every second, our sensory systems are flooded by a multitude of stimuli that our central nervous systems must rapidly and accurately interpret. Every moment of every day of our lives we are tasked with detecting, categorizing, interpreting and, often, generating a response to a given stimulus. For example, an actor reaches out to a coffee cup in the morning, but in the process of pouring the coffee some of it spills onto the counter. Despite the large number of sensory and motor events in this picture, let us focus our attention on the grasping movement for the coffee cup that is slippery. Tactile information from the hands becomes particularly important to successfully grasp. Logically, it is advantageous for the central nervous system to facilitate the processing of signals that convey touch information from the slippery cup of coffee. That is, the actor perceives touch information from the coffee cup. By contrast, touch information that does not directly provide insight into the coffee cup, for example, from the touch of a shirt sleeve on the forearm, is irrelevant to the task demands and should be ignored. Therefore, when sensory information is task-irrelevant then afferent information will be ignored/removed and will not be perceived by the actor – this is sensory gating. But, what determines which sensory information is important for later processing and how does that effect manifest? How can physiological gating be differentiated from concurrent attentional processes?

In the above example the coffee cup surface depressed the surface of the skin and activated the underlying sensory receptors. These sensory receptors are stimulated by mechanical deformations of the skin. Hence they are collectively called mechanoreceptors. There are several different types of receptors that convey slightly different sensations.
However, it must be recognized first that these mechanoreceptors are a part of the larger somatosensory system.

The somatosensory system is a complex network of peripheral and central networks that work in concert providing a complete perception of the body in space and any objects a person contacts. Hence the somatosensory system is capable of providing the central nervous system with two general types of sensory perception: exteroception and interoception. The former refers to a sensory perception of the external environment. For example, the coffee cup, in our example above, deforms the mechanoreceptors in the skin and the mechanoreceptors convey a volley of action potentials along the axons that eventually reach the primary somatosensory cortex of the cerebral cortex\(^1\) (where these signals are consciously perceived). The latter term, interoception, refers to sensory perception of the state of the organism’s internal environment. There are many examples of interoception, such as visceral sensation, that are beyond the scope of this document. The present dissertation focuses on somatic sensation. In the above example of the coffee cup, proprioception is a form of interoception and a subtype modality of somatic sensation. Of course, proprioception signals muscle length and, in general, conveys real-time sensations of body posture and position of limb segments (Kandel et al., 2013). Proprioception is an important sensory modality as demonstrated by individuals in which it is abolished (see below).

In the following section, I will discuss the anatomy and physiology of the somatosensory system, pointing out, in particular, how touch is perceived and affected by peripheral and central mechanisms. Also, this introduction will review the processing of visual information, with particular attention to the processes required to physically interact

\(^1\) I will provide a more complete treatment of the anatomy and physiology of somatosensation in a subsequent section.
with the external environment. I will then discuss the proposed mechanisms of gating and their importance to processing tactile inputs, explaining pain gating and saccadic suppression to demonstrate that gating is a generalizable physiological strategy of the central nervous system.

1.1 The Somatosensory System

1.1.1 Submodalities of the Somatosensory System other than Discriminative Touch

Before I provide an overview of the anatomy of the somatosensory system, it is important to recognize that there are several different submodalities of somatosensation that reach the cerebral cortex via different anatomical pathways. These pathways are responsible for conducting different forms of sensory information (i.e., proprioception [muscle spindles and Golgi tendon organs], nociception and thermoception). Also, there are mechanisms by which lower spinal segments can be affected by higher centers through the same anatomical pathways (e.g., primary somatosensory cortex, S1). It is by these latter mechanisms that tactile gating should manifest.

Proprioception is principally transduced by a specialized receptor called the muscle spindle (sub-divided into type Ia and II muscle spindle endings) that detects change in muscle length as well as static length of a muscle. The muscle spindle itself is a small collection of intrafusal muscle fibers surrounded by a capsule. Muscles spindles also receive efferent signals from gamma motor neurons and activation of gamma motor neurons causes the intrafusal fibers to contract, increasing the likelihood that the Ia (large diameter) and II (medium diameter) sensory endings will fire action potentials in response to muscle stretch. Sensory endings coil around the intrafusal fibers that detect change in muscle length by...

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2 Gamma motor neuron activation does not add force to muscle contraction in a significant manner (Kandel et al., 2013).
mechanically-gated ion channels that dot the surface membrane of the sensory endings. Importantly, there are several types of muscle spindle: dynamic nuclear bag fiber, static nuclear bag fiber and nuclear chain fibers. All three types of muscle spindles are innervated by Ia sensory axons and type II sensory axons innervate the nuclear chain fibers and static nuclear bag fibers. A key observation from Brown & Matthews (1966) demonstrates that recording sensory afferents from a muscle spindle during stretch alone shows an initial increase in action potentials (termed dynamic response) that quickly reduces to an elevated level of action potential firing (steady state response). Brown and Matthews also stimulated static gamma motor neurons while stretching a muscle and observed a markedly prolonged steady state response with the initial dynamic response. Lastly, when the dynamic gamma motor neuron was stimulated, Brown and Matthews observed enhanced dynamic response and the steady state response gradually returned to baseline. These findings suggest that muscle spindles transduce several different muscle actions to the spinal cord and to the somatosensory cortex.

Whereas muscle spindles transduce change in muscle length, Golgi tendon organs (GTO) are sensitive to change in muscle tension. GTOs are small structures surrounded by a capsule that are about 1 mm long and 0.1 mm in diameter, located at the myotendon junction. Internally, collagen fibers are paired and each pair has a braided, double-helix arrangement, with each fiber connected to a group of muscle fibers in series. A Ib afferent axon innervates one GTO and that axon branches into many fine nerve endings that are intertwined amongst the collagen fibers. When a muscle is activated, the tension generated causes the GTO to stretch and, therefore, straightens the collagen fibers, which in turn, compresses the nerve endings causing action potentials to travel along the sensory axon to the spinal cord. Crago,
Houk & Rymer (1982) observed that muscular force linearly correlated to GTO discharge rate of the soleus muscle of the cat, demonstrating that GTO inputs encode muscular tension.

In addition to proprioceptors, receptors exist that are selectively sensitive to stimuli that can damage healthy tissue. Nociceptors respond directly to mechanical and thermal stimuli and tissue damage releases chemicals that indirectly stimulate the same receptors. Nociceptors convey action potentials along Aδ fibers that convey sharp and pricking sensations. Most Aδ fibers are mechanically stimulated by penetrating, pricking or squeezing the skin, but can also be stimulated by noxious thermal stimuli that damage the skin. Nociceptors are also innervated by small-diameter, unmyelinated C fibers that convey the sensation of dull, burning pain. The nerve endings that are responsible for the dull, burning pain tend to respond to various noxious mechanical, thermal and chemical stimuli. Electrical stimulation of the unmyelinated C fibers elicits prolonged sensation of burning pain (Kandel, Schwartz, Jessel, Siegelbaum & Hudspeth, 2013).

Thermal receptors sense the temperature of objects that are touched. Thermal perception is a uniquely somatosensory modality as object temperature is not usually apparent with other sensory modalities (thermal radiation is not seen). Thermal sensations arise from the difference in temperature between the touched object and the temperature of the skin (approximately 32°C). There are various types of thermal skin receptors, and each type is sensitive to a specific temperature range. Specifically, there are six types of thermal receptors that work in concert to elicit the thermal sensations that humans feel. These are low-threshold and high-threshold cold receptors, warm receptors, and two classes of heat nociceptors (there are six transient receptor potential [TRP] channels).
Humans are not sensitive to temperature changes within 31°C to 36°C, but when temperatures drop below 31°C the sensation changes from cool to cold. Once temperatures drop below 15°C, the sensation progresses to pain. Temperatures above 36°C elicit warm sensations, and temperatures above 45°C evoke sensations of pain. Both myelinated Aδ fibers and C fibers convey nociception but there are several important differences between cold and warm receptors. Warm receptors behave like simple thermometers so that firing rates increase with increasing skin temperature (up to the pain threshold) and they are located at the terminals of C fibers. But, warm receptors are less responsive to rapid changes in skin temperature; cold receptors are more responsive to rapid changes in skin temperature. Cold receptors respond in two stages to temperature decreases. First, low-threshold receptors fire action potentials that evoke a cool sensation. Continued cooling of the skin then progresses sensation from cool to cold and progressively becomes more painful (Kandel et al., 2013).

How are warm and cool sensations transduced into signals that the central nervous system can interpret? When a neuron depolarizes voltage-gated channels open when the potential difference between the neuron cytosol and extracellular environment reaches threshold. TRP channels behave like voltage-gated channels and are named based on the genetic subfamily. For example, the TRPV-1 channel is an abbreviation for vallinoid-1, TRPM-8 is an abbreviation for TRP melastatin-8 and TRPA-1 is an abbreviation for TRP ankyrin-1. Two types respond to cold temperatures and are inactivated by warm temperatures. The TRPM-8 subclass responds to temperatures below 25°C and evokes sensations of cool or cold. TRPA-1 receptors respond to lower temperatures (17°C) and the sensation is described as cold or frigid. Only TRPM-8 receptors are expressed in low-
threshold sensory endings, while TRPA-1 and TRPM-8 receptors are expressed in high-threshold sensory endings.

There are four TRP receptors that respond to warm temperatures and do not function in cooler temperatures. Nerve endings that express TRPV-3 receptors respond to temperatures that exceed 35°C. TRPV-3 receptors underlie the sensation of warm and hot. Burning pain sensations are the result of the activation of TRPV-1 and TRPV-2 receptors that respond to temperatures exceeding 45°C. Finally, TRPV-4 receptors activate under temperatures that exceed 27°C and respond to normal skin temperature.

In summary, there are multiple submodalities of somatosensation that each contributes to sensation of the internal and external environments. Critically, each contributes to the survival of humans. For example, the central nervous system receives sensory information from proprioceptors regarding muscle length and tension that informs the motor planning networks in the cortex. These networks plan appropriate movements to goals. One famous example is that of Ian Waterman who, at the age of nineteen, became paralyzed. To his physicians’ surprise, he did not lose the ability to activate his muscles but lost proprioception and touch perception below his neck. He retains thermal perception and nociception. The precise pathology of his condition is not known, however, physicians speculate that his Aα, large-diameter, fibers were destroyed due to some form of viral neuropathy. Therefore, Mr. Waterman could not move his limbs because his motor system did not have peripheral feedback from his muscle spindles, GTOs and joint receptors (Kandel et al., 2013). Despite the lost proprioception, Mr. Waterman regained his ability to move; he is able to walk, reach and grasp objects. This incredible feat was achieved with rigorous training using visual feedback to guide his actions.
The loss of thermal perception and nociception is not any less devastating. Those who lose the ability to feel thermal stimuli or pain are at higher risk of sustaining injuries from burns or lacerations that may become septic. Brown-Séquard syndrome is an example of thermoanaesthesia and loss of pain sensation and was first described by French physiologist Charles Edouard Brown-Séquard in 1850. Hemisection of the spinal cord generates Brown-Séquard syndrome commonly caused by trauma. Brown-Séquard syndrome is characterized by three distinct pathological presentations (1) paralysis of muscles ipsilaterally and at the level of the lesion, (2) loss of touch and proprioception ipsilaterally below the lesion and (3) loss of pain and thermal sensation contralaterally and below the lesion. Anatomically, the dorsal column-medial lemniscus pathway conveys discriminated touch information from the limbs to the cortex decussates at the internal arcuate fasciculus via the ventral posterolateral nucleus of the thalamus. Therefore, touch sensation is lost ipsilateral to the lesion. However, pain and thermal sensation are lost contralaterally to the lesion because pain and thermal information decussates at the same level of the spinal cord as they enter from the periphery and travel to the cortex via the anterolateral system in the spinal cord. Brown-Séquard syndrome is often caused either by a spinal cord tumor or trauma (e.g., bullet). The probability of recovery often depends on the nature of the lesion. The above neural pathologies offer striking demonstrations of neural structure and function that offer important clues regarding how sensory pathways are organized. The following section discusses the anatomy and function of the somatosensory system.
1.2 Anatomy of the Somatosensory System

1.2.1 Receptors underlying discriminative touch

It is important to have a basic understanding how the nervous system transduces mechanical stimuli and, therefore, I begin the discussion with an outline of the receptors responsible for sensory transduction. Four receptor specializations underlie touch perception: Meissner corpuscle, Merkel disk receptor, Pacinian corpuscle and Ruffini endings. All the above touch receptors feed into either A\(\alpha\) axons (large diameter, fast conduction velocity, 72 – 120 m/s) or A\(\beta\) axons (medium diameter, moderate conduction velocity, 36 – 72 m/s). The above receptor types are classified as mechanoreceptors. Mechanoreceptors respond to physical deformation of the cell that, in turn, allows ions to pass through the nerve ending causing a depolarizing receptor potential and subsequent action potential to be propagated along the nerve fiber. Mechanical deformation of the skin opens ion channels that increase sodium (Na\(^+\)) and calcium (Ca\(^{2+}\)) flux across the membrane (Kandel et al., 2013).

The above receptors feed sensory information to specific types of axons that demonstrate functional organization at the most peripheral portions of the nervous system. Skin mechanoreceptors are innervated either by slow-adapting (SA) or fast-adapting (FA) axons and their behaviour is readily observed in electrical recordings. SA fibers generate near constant frequency action potentials from stimulus onset to offset but they eventually adapt. FA fibers generate an initial burst of action potentials that signal stimulus onset and quickly stop firing (as soon as the stimulus is stationary). Also, the receptors are further divided according to their location in the skin. Therefore, skin mechanoreceptors can be classified both by electrophysiological behaviour and anatomical location in the skin. Each type forms a functional unit that consists of an afferent axon, the axon’s distal branches and the specific receptor morphology that surround each nerve branch. Each receptor type can be
found either in superficial or deep layers of skin (i.e., type 1 or 2). The result is a simple 2 x 2 matrix that describes all mechanoreceptor species. The following sections describe the specific types of mechanoreceptors, their behaviours and their functional connections to the spinal cord.

Generally, type 1 fibers end in clusters of small receptors in the superficial layers of skin and tend to be 6 – 12 um in diameter with varying conduction velocities between 35 – 70 m/s (see Tables 23-1 in Kandel et al., 2013). The most common receptor type is the rapidly-adapting type 1 (RA1) mechanoreceptor, also known as Meissner corpuscle, and at a density of 2/mm². Each RA1 ending is a fluid-filled oval structure with several layers called lamellae originating from the myelin sheath. RA1 receptors are tightly anchored to the skin by collagen fibers, thus contributing to their fine mechanical sensitivity detecting object texture (e.g., different grain patterns in wood, etc.). Each RA1 fiber innervates 10 – 20 Meissner corpuscles and they are located in the dermal papillae (i.e., skin ridges). Furthermore, two to five RA1 fibers innervate one Meissner corpuscle. Lateral motion of the finger optimally stimulates Meissner corpuscles that respond with action potentials firing between 1 Hz – 300 Hz (best frequency: 50 Hz). Like all type 1 fiber receptors, Meissner corpuscles exhibit highly localized fields of which the pattern on the skin reflects axon branching. Receptive fields of RA1 fibers are most dense at the fingertips, with average receptive field size of 25 mm². Receptive fields become progressively larger on the proximal aspects of the digits and the palm. RA1 receptors are designed to detect low-amplitude and low-frequency stimuli making them especially suited to detect objects when the hand is

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³ For the sake of simplicity, this document will refer to skin mechanoreceptors according to the innervating fiber (i.e., RA1, RA2, SA1, & SA2), as it describes the entire functional unit of a particular mechanoreceptor subsystem.
passing over them and are capable of detecting surface textures as small as 10 µm. For example, Braille readers are reliant on proper Meissner corpuscle function deciphering dot patterns on a piece of paper (Kandel et al., 2013).

In contrast, slowly-adapting type 1 fibers (SA1) innervate wide areas of the skin particularly in the glabrous skin of the fingertips. Merkel disk receptors are the transducers for SA1 units which are located adjacent to sweat glands in the epidermis. Each SA1 fiber branches into many Merkel disk receptors allowing the advantage of receiving many inputs from a small area. The Merkel disk receptor surrounds the nerve ending and consists of epithelial cells. When the skin is deformed the epithelial cells transfer the mechanical energy to the nerve ending depolarizing the nerve ending. Interestingly, synapse-like junctions exist between Merkel epithelial cells and the adjacent axon suggesting that transduction of any given mechanical stimulus rests in the Merkel cells themselves rather than in the axon. Functionally, Merkel cells detect skin deformation and lateral stretch of the skin. Like Meissner corpuscles, SA1 fibers exhibit highly localized receptive fields that are most dense at the fingertips with average receptor area of 11 mm². Functionally, SA1 fibers detect pressure and object form and, therefore, encode object shape (see below). Paradoxically, slowly-adapting fibers fire more action potentials per unit time if a stimulus has higher curvature, despite stimulating less receptors. In other words, an object with a smaller surface area indenting the skin exerts higher pressure (assuming force is held constant) and the receptors respond to pressure exerted by an object on the skin. Since pressure is a function of force over area, \(( P = F/A )\) object size and peak firing rates of slowly-adapting fibers are negatively correlated. Therefore, a large object elicits lower firing rates than a smaller object due to the inverse relationship between force and area. Likewise, introducing a second small
and sharp object adjacent to another small, sharp object already pressing on the skin evokes fewer action potentials per unit time because the collective pressure elicited by the sharp objects is exerted over a wider area thus decreasing pressure. Behaviorally, humans tend to grasp objects surfaces that are flat and, therefore, exert the least amount of pressure on the skin whereas grasping the edge exerts higher pressure.

Type 2 fibers have wide receptive fields on the skin and sparsely populate the dermis of the skin or in subcutaneous tissue. Type 2 receptors are physically larger than type 1 receptors, innervate only one receptor and their depth allows them to detect stimuli that may occur at some distance from the receptor itself. This physical arrangement has several important consequences such as larger receptive fields that preclude the ability to detect motion and vibration. There are two subtypes of type 2 fibers. RA2 fibers conduct action potentials at a velocity between 35 – 70 m/s and have axon diameters ranging from 6 – 12 \( \mu \text{m} \). RA2 fibers are best stimulated by vibration frequency range between 5 – 1,000 Hz. The receptor mediating sensory transduction for RA2 fibers is the Pacinian corpuscle and fires action potentials when the skin is indented as little as 0.01 \( \mu \text{m} \). These characteristics make the Pacinian corpuscle the most sensitive receptor in the somatosensory system. RA2 fibers do not branch into multiple sensory receptors (as is the case with Merkel disk receptors) and are larger and sparsely populate the skin. Pacinian corpuscles have a lamellar structure, like that of Meissner corpuscles, often described as “onion-like” and have thin layers of fluid that separate each lamella. The lamellae collectively act as a high-frequency amplifier, an important function for tool use. Also, the Pacinian corpuscle population in the hand changes as an individual ages ranging from 2,400 corpuscles in young children and dwindles to 300 in old adults (Kandel et al., 2013). In response to tuning fork stimulus, Pacinian corpuscles
synchronously fire action potentials eliciting a buzzing sensation on the skin. Tuning forks provide a sensitive method to test RA2 integrity and can localize peripheral nerve damage.

Slowly-adapting type 2 (SA2) fibers innervate the Ruffini endings. These receptors are concentrated at finger and wrist joints and in the skin folds in the palm. SA2 fibers conduct action potentials at velocities ranging from 35 – 70 m/s and have axon diameters ranging from 6 – 12 µm. SA2 fibers sustain a train of action potentials that slowly adapts and are best stimulated by indentations of 40 µm. The Ruffini endings are long, fusiform structures with collagen fibers that extend from the receptor and attach to the skin folds at the joints. The nerve endings intertwine among the collagen fibers (like Golgi tendon organs) and activate in response to stretching of the Ruffini ending along the longitudinal axis. Functionally, SA2 fibers are especially suited to encode the three-dimensional properties of objects that are grasped with the entire hand because Ruffini endings respond to skin stretch. SA2 fibers play a secondary role in proprioception providing position information of the fingers when the hand is empty. Ruffini endings exhibit tuning curves in which a small range of stretch orientations maximally activates that particular receptor (Kandel et al., 2013).

Thus far I have provided a basic anatomical and functional outline of the mechanoreceptors that transduce mechanical stimuli into signals that the nervous system interprets. The following section outlines the various roles tactile receptors play providing afferent signals when contacting objects.

1.3 Tactile Receptors are Critical for Grip Control

Each afferent fiber type plays a role in skillful grasping of objects. Johansson (1996) recorded neural activity from different fiber types using microneurography of the median
nerve. Participants performed a grasp and lift task during which activity from the different mechanoreceptor fibers were recorded during contacting, lifting, holding, lowering and resting. Johnasson observed that each receptor type played its own role during the task. At object contact, RA1, SA1 and RA2 and SA2 fibers fire action potentials. According to the firing patterns, SA1 fibers encoded finger pressure applied on the object, while RA1 fibers encoded how quickly force was applied. When the object lifts from the surface RA2 fibers exhibit a burst of signals while SA1 and RA1 fibers conduct a steady train of action potentials that provide tonic signals when the object is in contact with the skin. Specifically, SA1 fibers provide information of object contact while SA2 fibers convey skin stretch information indicating hand position. Coincidentally, RA1 and RA2 fibers cease action potentials once grip is established. Upon table contact, RA2 fibers conduct a short volley of action potentials in response to the mechanical shockwave caused by contacting object with the table surface. Finally, Johansson (1996) observed a volley of action potentials conducted by RA1 fibers upon object release again indicating rate of force.

Grip force control is a highly controlled behavior that relies on sensory feedback and feedforward control⁴. Manual manipulation of objects involves a series of evolving phases. Each phase is marked by the completion and initiation of specific stages. Johansson (1996) offers a framework within which to categorize these stages. When the fingers make contact with the object, it is the called pre-load phase and is signaled by FA1 and SA1 afferent fibers. The following stage, or load phase, sees a sharp increase in grip force and ceases at movement start (signaled by FA2 afferents) and is followed by a static phase in which an object is held stationary by the fingers. SA1 and SA2 afferent fibers convey object contact

⁴ For the purpose of the present discussion, I will focus on the feedback aspect of grip control then a discussion of feedforward mechanisms follows.
information to the somatosensory cortex. When an object is lowered and makes contact SA2 afferents fire a volley of action potentials that signal table contact. Once an object makes contact with the table there is a slight delay between load force reduction and grip force reduction finishing with progressively decreasing grip force, in the unload phase, until the fingers release the object.

Feedforward mechanisms are important for successful grasping because predicting future sensory and motor events compensates for sensorimotor delays. Feedback delay is problematic because signals traveling from the receptor to the cerebral cortex take time. With the time delay sensory feedback does not reflect the current state of the sensorimotor system and the external environment. Feedforward mechanisms predict future events removing the time delay associated with sensory feedback. Predicting future states is a successful strategy that the central nervous system uses that will be described. However, using only feedforward control has is disadvantages. If any movement errors occur, as a result of planning or movement, they will not be corrected and compound over time. Complex effector systems such as the arm make it very difficult to predict sensory and motor consequences.

Combining characteristics of the feedback and feedforward control provides an optimal strategy monitoring the state of the body and the environment. When participants know the weight of an object before picking it up the force applied on the object overcomes gravity and inertia. Specifically, grip force increases proportionately with object weight reflecting a preplanned action since the load force and grip force have identical profile shapes (see Johansson et al., 1991). When object weight is larger than expected, the object initially slips but force is quickly increased to compensate for the higher weight.
Afferent signals are important for the control of grip (Edin et al., 1992). FA1 behavior changes as a function of the surface material (Johansson & Westling, 1987). Johnasson & Westling (1987) found that FA1 afferents respond strongly to surface materials that have a lower coefficient of friction relative to surface materials with higher coefficients of friction. This behavior accounts for the increase in grip force in response to a more slippery surface. Or, when participants grasp an object that was heavier than expected grip force adjustment occurred very rapidly. This adjustment, a response delay, is often very short – approximately 70 ms (Johansson & Westling, 1987). The ‘grip slips’ are encoded by FA1, FA2, and SA1 afferents that trigger an increase in grip force to prevent object slip. Increase in grip force responds quickly enough that slippage is not perceived. Local anaesthesia disrupts friction adjustment so subjects employ stronger grip forces that would be otherwise unnecessary (Johansson & Westling, 1984). The observed mean time for grip adjustment in response to object slip is so quick that it precludes any possibility of cortical involvement. Despite this, tactile gating is likely not mediated by the same processes as grip adjustment. Previous work suggests that tactile gating originates from higher centers (Colino et al., 2014; Bolton & Staines, 2011, 2014; Yamaguchi & Knight, 1990). Therefore, I will explore tactile processing at higher centers in the cortex and briefly discuss the various spinal pathways that mediate somatosensation.

1.4 Anatomical Pathways to Higher Somatosensory Centers

1.4.1 Dorsal Column-Medial Lemniscus Pathway

Afferent fibers carrying somatosensory information (i.e., touch, pressure, vibration, proprioception and position) to the primary somatosensory cortex travel along the dorsal column-medial lemniscal pathway. Primary sensory axons reach the primary sensory neurons in the dorsal root ganglion (DRG) outside of the CNS. Assuming the primary
sensory neurons in the DRG receive an action potential they will send an action potential to higher centers along either the *fasciculus gracilis* (lumbar afferents) or *fasciculus cuneatus* (cervical afferents). The secondary sensory neurons are housed either in the *nucleus gracilis* or *nucleus cuneatus*. Therefore, the dorsal column-medial lemniscal axis is topographically organized with the lower body represented medially and the upper body represented laterally. Secondary sensory neurons project axons that decussate in the lower medulla, ascend in the medial lemniscus and form synaptic connections to tertiary sensory neurons in the thalamic ventral posterolateral nucleus. Finally, thalamic neurons project information to the postcentral gyrus, otherwise known as the primary somatosensory cortex (S1), via the posterior limb of the internal capsule (Felten & Shetty, 2010).

1.4.2 **Spinocerebellar Pathway**

The Spinocerebellar pathway conveys proprioceptive primary somatosensory axons from joints, tendons, and muscle spindle afferents (1a afferents) and from ligaments (1b afferents). Like the dorsal column-medial lemniscal pathway, the spinocerebellar pathway is topographically organized; 1a afferents below T6 enter the spinal cord via the dorsal root ganglion, in which primary sensory neurons lie, ascend the dorsal spinocerebellar tract (DSCT) and enter the cerebellum via the inferior cerebellar peduncle. 1a afferents above T6 travel to the cerebellum via the fasciculus cuneatus and the lateral cuneate nucleus. 1b afferents (e.g., Golgi tendon organs) below T6 enter the CNS and immediately decussate through the anterior white commissure. They ascend the ventral spinocerebellar tract (VSCT) and enter the contralateral cerebellum, via the superior cerebellar peduncle, and immediately cross the midline again. 1b afferents entering the CNS above T6 do not decussate at the spinal cord level but they ascend the rostral spinocerebellar tract (RSCT) and
enter the cerebellum via the inferior cerebellar peduncle (Felten & Shetty, 2010; Kandel et al., 2013).

1.4.3 Spinothalamic and Spinoreticular Pathways

The following section explores pain and temperature sensations and their distinct ascending spinal pathways. The spinothalamic and spinoreticular pathways convey pain and temperature sensation to higher centers. The former projects information to S1 and S2 while the latter bilaterally conveys information to the pontine, medullary and midbrain reticular formation. Pain and temperature afferents from each spinal segment enter the CNS via a DRG and immediately decussate across the anterior white commissure and ascend to higher centers via the spinothalamic/spinoreticular pathway. Afferents that form part of the spinothalamic pathway ascend to the thalamic ventral posterolateral nucleus projecting to S1 and S2. Afferents that form the spinoreticular pathway ascend the spinal cord and target several structures such as the lateral reticular formation of the lower medulla, deep layers of superior colliculus, periaqueductal gray matter, and non-specific thalamic nuclei. Finally, spinoreticular fibers travel to various cortical targets such as S1 and S2, cingulate, insular and prefrontal cortices. The cortical targets process and interpret nociceptive pain and temperature information (Felten & Shetty, 2010).

In summary, there are several existing anatomical pathways that project receptor afferent information that detect different sensory stimuli such as touch and vibration, muscle length and tension, and pain and temperature. The anatomical pathways reveal topographical and functional organization – a basic principle of the central nervous system. The dorsal column-medial lemniscus pathway conveys touch and proprioception to the thalamus and, subsequently, S1. The dorsal column-medial lemniscus pathway is critical for surface texture perception, provides sensory feedback for grip control, and carries information from muscle
spindles sensing limb position and from Golgi tendon organs sensing muscle tension. The spinocerebellar pathway projects proprioceptive information from 1a and 1b afferents to the cerebellum to contribute to the coordination of limb movements. The spinothalamic/spinoreticular pathway projects pain and temperature information from thermal receptors and nociceptors. This pathway is necessary for pain and thermal perception that can be disrupted by lesions in the anterolateral pathway affecting resulting in analgesia (Felten & Shetty, 2010; Kandel et al., 2013).

1.5 Sensory Information Processing at S1 is Convergent in Nature

The following section will discuss somatosensory processing in the cerebral cortex and will focus primarily on the processing of touch and proprioception, as both are crucial for tactile gating to occur. As described in the previous section somatosensory information enters the postcentral gyrus of the cerebral cortex via the ventrolateral posterior nucleus of the thalamus. Importantly, each cortical neuron receives afferent information from a specific body segment and all the receptors that innervate the cortical neuron form the receptive field of that cortical neuron. For example, if a feather is brushed on the surface of the index finger a neuron in S1 will activate. Electrical stimulation of the same cortical neurons can replicate the same percept of the feather or other sensory experience (e.g., Penfield & Boldrey, 1937). Furthermore, body segments having a high tactile receptor density are disproportionately represented in the cerebral cortex (i.e., there is more cortical area devoted to processing tactile inputs from the fingers and face than there are neurons representing the thigh). This observation underlies the principle of cortical magnification. The same is true for the visual system in which the fovea of each eye has disproportionately larger cortical area devoted to processing stimuli that fall on that area of the retina (Felten & Shetty, 2010; Haines, 2012; Kandel et al., 2013).
Four cytoarchitectural (Brodmann’s) areas comprise S1 and are rostrocaudally arranged: areas 3a, 3b, 1 and 2 (see Figure 1.1). Neurons in areas 3a and 2 receive inputs from proprioceptors in muscles, joints and skin whereas area 3b and area 1 neurons receive inputs from receptors in the skin. All somatosensory information, including touch and proprioception, are processed in parallel and sent to higher centers. However, tactile information itself is serially processed in which information from the VPL nucleus projects information to area 3b and, in turn, sends information to area 1. Coincidentally, areas 3b and 1 project tactile information to S2. Furthermore, specific S1 areas receive inputs from specific receptor types. Area 3a receives inputs from muscle spindles, area 3b receives SA1 and RA1 inputs, and area 1 receives RA1 and RA2 inputs.

Tactile processing is convergent in nature. For example, a cortical sensory neuron in area 3b representing the right index finger (300 – 400 sensory fibers) has a larger receptive field than a DRG neuron that represents information from a single receptor that lies in a small spot of the skin. Therefore, receptive field size increases in a peripheral-central fashion. The cortical areas devoted to particular body segments have a stereotyped arrangement with lower body segments such as the toes, foot, leg, hip and trunk represented dorsomedially and into the central fissure. The head, neck, shoulder, arm, forearm, wrist and hand are represented more laterally and the fingers, facial structures, tongue, pharynx and viscera represented most laterally. This arrangement is called the sensory homunculus. The sensory homunculus is not a permanent arrangement; it changes with experience and/or injury. Representations in secondary somatosensory cortex (S2) are more complex; S2 neurons represent symmetric skin segments on the contralateral and ipsilateral sides of the body (Felten & Shetty, 2010; Haines, 2012; Kandel et al., 2013).
Figure 1.1 Anatomical schematic representing major motor and sensory areas and information flow

Once information from the periphery reaches the primary somatosensory cortex, processing occurs in a gradually convergent fashion with more abstract representations at secondary somatosensory cortex and superior parietal lobe. Also, note that motor activity influences sensory processing. There are heavy reciprocal connections between motor and sensory cortices. S1 is subdivided into four Brodmann’s areas, 3a, 3b, 1 and 2 (area 3 is condensed for the sake of clarity).

Tactile receptive fields have an excitatory zone surrounded by an inhibitory area.

When a stimulus falls in the excitatory zone of the receptive field of a neuron then the neuron has a higher activation probability. The contrary is true when a stimulus falls in the inhibitory zone of a neuron. Sripati et al. (2006) observed that primate S1 neurons in areas
3b and 1 exhibit one of three modes of activity: exclusively excitatory, excitation replaced by inhibition or excitatory center-inhibitory surround. Sripati and colleagues performed cortical S1 neuron recordings from monkeys (Macaca mulatta). A microelectrode array was inserted into the cortex and cortical mapping proceeded until areas 3b and 1 were identified by functional stimulation of the contralateral fingers and palm. Once data preprocessing was completed, Sripati and colleagues analyzed 138 neurons and, subsequently, mapped each neuron’s receptive field (RF) by gently indenting the glabrous skin of the contralateral hand. The authors found that 56 out of 58 neurons in area 3b had receptive fields restricted to one fingerpad, while the remaining two neuron’s receptive fields spanned multiple finger pads. In area 1 27 out of 80 neurons had receptive fields covering one finger pad, 33 out of 80 neurons had receptive fields covering multiple fingerpads and the remaining 20 neurons had RF spanning multiple digits. The experimenter adhered a 1 cm², 20 x 20 tactile probe array stimulating the distal fingerpads of digits two to four. Each probe was independently controlled and randomly indented the skin. The experimenters randomized indentation amplitude between 0 and 500 μm. Sripati and colleagues (2006) found three broad classes of cortical neurons based on electrical response. The first type responded with excitation and little inhibition. The second type of neurons responded with initial excitation that was replaced with inhibition. The third type of neuron responded with a large inhibitory area surrounding an excitatory center replaced with inhibition 45 ms after stimulation. Area 1 had proportionately more neurons with surrounding inhibition than did area 3b (52% compared to 64%, respectively). Area 1 had less neurons with replacing inhibition (25% compared to 42%, respectively). Area 1 had more purely excitatory neurons than area 3b (11% compared to 6%, respectively).
Furthermore, Sripati and colleagues confirmed that cortical neurons exhibit larger receptive field area compared to peripheral inputs. A receptive field is defined as the skin surface area within which an adequate stimulus causes a cortical neuron to activate. Area 3b and 1 neurons exhibited larger mean receptive field area (30 mm$^2$) than RA and SA peripheral receptive field areas (25 mm$^2$ and 18.7 mm$^2$, respectively). Total receptive field (inhibitory plus excitatory) area changes with respect to time, area 3b and area 1 neurons achieve peak field area 15 ms after stimulus and linearly decrease to zero 100 ms post stimulus. However, when excitatory and inhibitory volume were examined separately, Sripati and colleagues found that excitatory volumes achieved peak area at 15 ms and inhibitory area achieved peak area 40 – 45 ms post stimulus.

The question arises: what is the cause of the inhibitory surround observed in S1? The authors suggest that the inhibition arises from SA1 afferents. Three arguments support the hypothesis: (1) the latency suggests the presence of interneuron connections; (2) there is more surround inhibition observed in the cortex and it is not symmetrically arranged relative to the center; and (3) the inhibitory to excitatory ratio in the cortex exceeds 2 to 4 times that observed from peripheral inputs. It is likely that somatosensory areas use intracortical feedback loops mediating inhibitory mechanisms as in the visual cortex. Also, area 3b had larger inhibitory areas that decayed rapidly, whereas those of area 1 did not. These observations suggest that area 3b and area 1 perform slightly different functions. For example, Sripati and colleagues observed smaller volume of replacing inhibition in area 1 suggesting that it detects spatial differences in tactile inputs whereas area 3b is sensitive to velocity.
1.5.1 Somatosensory Cortex Neurons are arranged in Functional Columns

The functional connectivity of somatosensory neurons provides clues to observed behaviour. The cerebral cortex is a thin layer of tissue containing six laminae\footnote{I use the terms “laminae” and “layers” interchangeably, as well as their singular forms.} and has a total surface area of roughly 2,600 cm\(^2\) and is 3 – 4 mm thick (Mountcastle, 1997). The cortex has approximately 28 x 10\(^9\) neurons and roughly the same number of glial cells. The number of synapses, estimated at 10\(^{12}\) connections, is staggering. Despite the sheer vastness of complexity that the cerebral cortex exhibits there are regular connective organization patterns between neurons. The basic organizational unit is the minicolumn and consists of a narrow neuron chain extending through laminae II through VI of the cortex. Each minicolumn contains roughly 80 – 100 neurons but this number can change according to sensory modality and functional complexity. For example, minicolumns of the primary visual cortex contain upwards of 250 neurons per minicolumn.

Afferent inflow (or lack thereof), in part, maintains columnar organization. Afferent inflow is necessary for any given column to maintain its function otherwise a column adopts another input such as in cases of deafferentation via amputation. Another defining feature that determines columnar organization is the peripheral receptive field. Cortical columns form the next level of organization in the cortex consisting of many minicolumns connected by short horizontal connections and all neurons within a column share common functional properties. For example, each primary visual area column is sensitive to a specific edge orientation and expresses a characteristic tuning curve; one column responds strongly to a bar of light at an angle of 45\(^\circ\) while a neighboring column responds strongly to a bar of light at an angle of 15\(^\circ\). Cortical columns, therefore, receive specific inputs from a specific spatial...
location and modality. Input specificity holds true for somatosensory columns in the cortex as well. In the somatosensory cortex, the modality of any given column matches a particular set of primary afferent fibers; a cortical column sensitive to SA afferents exclusively processes SA afferents and no other type of afferent input. Also, a column represents the collective afferent inputs from a particular region of the body. Rapidly adapting cortical neurons in S1 with receptive fields on the glabrous skin are driven by Meissner afferents; slowly-adapting cortical neurons from epidermis receive inputs from Merkel receptors; Pacinian cortical neurons receive inputs from Pacinian corpuscles; Ruffini endings have a mixed touch and proprioceptive function (discussed below) and, therefore, project to neurons in area 3a and 3b. Generally, cortical columns receive a number of inputs and have a number of outputs. Efferent neurons with different targets reside in laminae II, III, V and VI. Neurons residing in laminae II and III project efferents to other cortical targets and to adjacent columns via horizontal connections that allow information sharing when neighboring columns are simultaneously activated by the same stimulus. Also, laminae II and III neurons project to other cortical targets, on the same side of the brain and to mirror locations in the opposite hemisphere. It is for this reason that neurons in laminae II and III have large receptive fields. Laminae V and VI receive inputs from layer II and III of the same column or from adjacent columns and project efferents to subcortical structures. Layer IV receives thalamic inputs and projects to laminae II and III. These differences suggest that processing within the different laminae proceeds in different ways (Mountcastle, 1997). Layer I conveys feedback signals from higher cortical centers such as other somatosensory areas, frontal motor areas, limbic regions, middle temporal lobe involved in memory formation and storage, and sensory integration areas (posterior parietal cortex). These
connections may function to select relevant sensory information for cognitive processing (attention) and may be involved in sensory gating during motor activity (Kandel et al., 2013; Mountcastle, 1997).

In summary, there are four separate somatotopic maps of the body in the postcentral gyrus, one each in areas 3a, 3b, 1 and 2. The somatotopic organization roughly corresponds to the spinal dermatomes. Sacral segments are represented medially, lumbar and thoracic segments centrally and upper limb and trigeminal inputs from the face represented laterally.

1.5.2 Functional Mapping of S1

The following section outlines experimental evidence showing somatosensory organization from functional recordings. Functionally mapping the primate somatosensory cortex was performed with optical imaging in response to air puffs applied to each finger and was strongest in narrow bands across areas 3b and 1 (Shoham & Grinvald, 2001). Shoham & Grinvald (2001) performed optical imaging of monkey (Macaca mulatta) somatosensory cortex. As expected, the stimulation activated, according to stimulated finger, a narrow band of cortical neurons. In other words, there was clear somatic progression of activation from one finger representation to the next. The authors calculated differential optical maps subtracting each digit activation map from those of the other digits. Surprisingly, the cortical maps representing the fingers significantly overlap especially for digits 3 through 5. There was no overlap observed for the thumb (digit 1) and the index finger (digit 2); consistent with the fact that the finger and thumb often operate independently. Digit 1 had the widest functional territory followed by digit 2 and digits 3, 4 and 5 each had the narrowest territories. This observation underlies the importance the finger and thumb have in hand function. Furthermore, activation maps do not shift location upon repeated activations. In other words, cortical activation maps are spatially stable.
Subsequent electrophysiological recordings confirmed the optical mappings (Shoham & Grinvald, 2001). The experimenter inserted microelectrodes into locations of strongest activation detected optically and made multiunit recordings from four microelectrode penetrations at different locations of the somatosensory cortex. The first electrode penetration occurred at the strongest activation of digit 1 neurons and found that they were stimulus-locked when digit 1 was stimulated. The authors did not find activation of other digit representations confirming the relative isolation of digit 1 representation. The experimenter inserted the second electrode at the border of digit 2 and digit 3 representations. The results showed that digits 2 and 3 representations activated when each digit received an air puff, and there was little response from stimulation of other digits. The experimenter inserted the fourth electrode into the center of the 5th digit representation and found that stimulation of neurons representing the 5th digit readily evoked a neuronal response, but the same neurons activated in response to stimulation of the 4th digit as well. Finally, the experimenter inserted an electrode into an area that activated all five digits in response to stimulation. This “common” area of cortex was located posteromedially to the digit 5 representation. This common area strongly activated by digit 1 or digit 2 stimulation but less strongly by stimulating other digits. Additionally, touching the wrist, hand or forearm activated this common area of cortex. In addition to the monkey study, Shoham & Grinvald (2001) performed intraoperative recording of human patients. Most procedures required the central region of the cerebral cortex be exposed. However, microvascular noise made it impossible to measure and localize cortical activation. Therefore, the experimenters recorded electrical activity from cortical neurons using a 4 x 5 microelectrode array. Stimulating one finger evoked activation of a small area of cortex recorded by one electrode
confirming that somatosensory representations in human cortex are specific to certain skin regions.

Different regions of S1 mediate different submodalities of somatosensation. Areas 3b and 1 process tactile information from the cutaneous receptors such as the Meissner corpuscles, Merkel disks, Pacinian corpuscles and Ruffini endings. Furthermore, somatosensory areas process surface textures and project to secondary somatosensory cortex (S2). Area 2 is responsible for processing the object size and shape, and processes proprioception inputs projecting to area 5 located in the superior parietal lobule. Indeed, the common activation area found by Shoham & Grinvald (2001) is perhaps area 2 of S1 because neurons in that area detect abstract stimulus features such as stimulus movement across the fingers or object shape. Area 5 processes somatosensory inputs related to active touch and projects to the dorsal stream (see Milner & Goodale, 1992) that ultimately reaches frontal motor areas via the superior longitudinal fasciculus.

1.5.3 Processing in S2

Information from S1 is processed and projected mainly to S2. S2 neurons receive inputs from S1 but S2 is strongly influenced by top-down inputs from other centers that carry higher order signals such as that related to attention. In humans and non-human primates, S2 is located on the upper bank of the Sylvian fissure adjacent the parietal operculum. S2 receives the bulk of its inputs from areas 3b and S1. S2 has a role in object recognition. S2 has four separate cytoarchitectural subregions like S1. For example, any one person, barring sensory deficits, is able to distinguish a sphere placed in the left hand from a cylinder placed in the right hand. Additionally, S2 neurons do not, strictly speaking, respond to stimulus features but, rather, respond to combinations of features. The same sphere in the above example can have texture and local surface features that identify the sphere as a baseball.
Indeed, almost any individual identifies a sphere with surface textures consistent with a baseball – so long as an individual has prior experience regarding what stimulus features makes up a baseball. S2 neurons fire at different rates for different stimulus patterns and the neuron responses also depend on context and motivation. How S2 achieves this is discussed below.

S1 and S2 form a complementary network to represent and process tactile stimuli. S1 processes somatosensory information that is detected by the various receptors located in the skin. Tactile receptors are sensitive to different types of tactile stimuli and pass that information via spinal pathways up to S1. S1 is organized into anatomical columns that receive specific afferent information; each column receives sensory information from specific peripheral receptors such that one column receives inputs from exclusively Pacinian afferents, etc. The majority of S1 outputs project to S2. S2 is located on the upper bank of the Sylvian fissure directly caudal to S1. S2 neurons encode complex tactile inputs and, therefore, often respond to tactile inputs. For example, a particular neuron may only activate in response to a specific shape of a tactile stimulus and another neuron may be sensitive to the direction of a stimulus traveling across the skin. Importantly, other cortical centers influence activity in S2. Indeed, Romo and colleagues (2002) found that S2 neurons change their activity depending on context of past stimuli if that past stimulus is behaviourally relevant.

Romo, Hernández, Zainos, Lemus & Brody (2002) measured neural correlates of decision making in S2 during a two-alternate forced choice task. The authors sought to determine where in the primate brain the comparison between a novel tactile stimulus and a reference stimulus take place. Romo et al. reasoned that S2 is the likeliest candidate for this
function because it: (1) maintains connections to many cortical areas and, therefore, can facilitate sensory-driven and memory-driven processes; (2) has activity that are affected by attentional processes. Indeed, it has been suggested that S2 plays a similar functional role in the tactile domain that the inferior temporal (IT) cortex performs in the visual system (Mishkin, 1979; Murray & Mishkin, 1984). IT neurons integrate afferent visual information and short-term memory signals (see Miller, Li & Desimone, 1993). Requiring primates to choose whether a probe stimulus was higher frequency or lower frequency than a reference stimulus stored in short-term memory allowed Romo and colleagues to correlate tactile integration and the decision that their subjects made.

Romo and colleagues lowered a small mechanical probe on the glabrous skin of one finger and the monkey indicated this event by pressing its free hand on an immovable key. After a variable delay, the probe oscillated at a set frequency (f1) followed by a 3-second delay and a second stimulus was delivered at another frequency (f2). The monkey was trained to remove its free hand from the immovable key and indicate whether f2 oscillated at a higher or lower frequency by pressing one of two buttons. The authors trained four monkeys (Macaca mulatta) to perform the task and recorded S2 neuron activity while each monkey performed the task. There were 517 neurons (from all monkeys) that responded differently from pre-trial firing rates (500 ms period before the probe indented the skin). Approximately 60% of the neurons increased or decreased their firing rate in response to f2, but their firing rates did not respond to stimulus frequency. By constrast, 40% of the neurons did respond to stimulus frequency. The authors analyzed trials in which the monkey made correct discriminations between f1 and f2. They also ensured that the stimuli were well
above discrimination threshold to decrease the likelihood that attention effects differ between trials.

The smaller proportion of neurons responded in an interesting manner: these neurons responded to f2 differently depending on it related to f1 confirming that the monkeys held f1 in short-term memory (f1 occurred 3 seconds before f2). Furthermore, this effect evolved over the course of f2; in the first 200 ms of f2 S2 neuron firing rates were not sensitive to f2 frequency. However, in the last 200 ms of f2, neurons exhibited higher firing rates when f2 > f1 and they exhibited lower firing rates when f2 < f1. These responses corresponded to the two choices the monkeys made in the discrimination task such that the neural response correlated with choice. Hence, it appears that f1 memory is stored in memory and that same information is fed back to S2. The recalled f1 information influences S2 neuron activity such that it increases or decreases firing rate depending on whether f1 was higher or lower in frequency than f2.

1.6 Tactile Gating

Tactile stimuli are gated from perceptual awareness when that information is not behaviourally relevant. Thus, tactile afferents are blocked from conscious awareness at some level in the central nervous system. This reduction in perceiving tactile stimuli during movement is known as ‘tactile gating’ (see Chapman et al., 1987; Rushton et al., 1981). Indeed, this behaviour is well-known in the somatosensory literature as “pain gating.” The perception of pain is reciprocally regulated by nociceptive and non-nociceptive inputs. Most neurons in the dorsal horn of the spinal cord receive noxious and non-noxious inputs. Melzak & Wall (1965) proposed that a relative balance between non-noxious and noxious inputs exists at the level of the spinal cord and the inputs affect the pain signal transmission to higher centers. Specifically, they proposed that non-nociceptive inputs close a “gate”
preventing transmission of nociceptive inputs whereas nociceptive inputs attempt to open the
gate. The original pain gate theory (Melzak & Wall, 1965) proposed that non-nociceptive
fibers, such as Aβ fibers, and nociceptive fibers, such as C fibers, form separate synapses
onto a projection neuron in the spinal cord dorsal horn. An inhibitory interneuron receives
collateral inputs from the Aβ fiber (excitatory) and C fibers (inhibitory) and sends an
inhibitory synapse to the projection neuron. Therefore, when a C fiber sends a signal to the
projection neuron the projection neuron relays pain information to higher centers and,
subsequently, pain perception occurs. In other words, excitation of a C fiber inhibits the
interneuron, thus increasing the activity of the projection neuron, whereas excitation of an
Aβ fiber excites the interneuron, thus decreasing the activity of the projection neuron. For
example, if a child accidentally burns herself on a stove element, there is an initial sharp
pain, transmitted by Aδ fibers, followed by a slow, burning pain, transmitted by C fibers.
According to pain gate theory (Melzak & Wall, 1965) activating non-nociceptive inputs
surrounding the burn injury should produce mild analgesia and, indeed, it does. Another
example is shaking the hand after striking a finger with a hammer. Viewed most broadly, the
convergence of high- and low-threshold inputs at spinal and higher sites produces the
observed behaviour in the above examples. The idea of convergence contributed to novel
analgesic methods such as transcutaneous electrical nerve stimulation (TENS) and dorsal
column stimulation. In TENS, electrodes are placed in locations surrounding an injury that
activate large diameter afferent fibers (Aα and Aβ fibers). The region of analgesia maps to
the same segments of the spinal cord in which non-nociceptive and nociceptive afferents end.

In vision, a similar phenomenon known as saccadic suppression prevents consciously
perceiving visual information during a saccade. Saccades are fast, ballistic eye movements
repositioning gaze that humans perform three times per second (Ross et al., 2001). In other words, simple extrapolation would predict that humans perform over 250,000 saccades every day! These movements can be performed voluntarily but many are automatic and are unnoticed. A person conversing with a close friend makes hundreds even thousands of saccades without noticing that these saccades were made. Yet, despite the large number of these movements we perceive the visual world to be stable. In other words, the visual world does not “smear” when a saccade is performed; visual information is gated during a saccade.

Indeed, Helmholtz recognized that the movement of the visual world is “sensed but not perceived” (Helmholtz, 1866/1963). Later, Sperry (1950) and Von Holst & Mittelstaedt (1954) formalized Helmholtz’s ideas in two closely related constructs. The corollary discharge (Sperry, 1950) or the efference copy (Von Holst & Mittelstaedt, 1954) is dictated and transmitted with the motor command to cancel out image motion due to saccades. Indeed, this idea was popular but subsequent research found that saccadic suppression is not simply a sensory cudgel but rather akin to a scalpel. For example, during saccades stimuli defined by high spatial frequencies appear to “grey out” while stimuli defined by low spatial frequencies are detectable and sometimes become more conspicuous (Burr & Ross, 1982).

Regardless, tactile suppression, like saccadic suppression, involves a reduction in the ability to detect stimuli. Indeed, this does not discount the possibility that a stimulus would be detected if it exhibited a specific feature or achieved high amplitude. Rather, the key idea is that there is a reduction in the ability to detect a stimulus. Like saccadic suppression, tactile suppression is a reduction in the ability to perceive tactile stimuli as opposed to visual stimuli. Despite the anatomical and sensory differences there is still reduction in the ability to perceive tactile stimuli, while performing a primary task (e.g., a goal-directed movement,
see Buckingham et al., 2010). This reduction is often measured as the percentage of correct reports of a probe stimulus relative to a fixed time point (e.g., movement onset). Several studies (Buckingham et al., 2010; Chapman et al., 1987; Milne et al., 1988; Rushton et al., 1981; Voss et al., 2008; Williams et al., 1998) reported suppression of tactile sensations just before and during movement. This is determined by presenting probe stimuli to a participant at randomly varied timed points. The participant's task is to perform the primary task (e.g., grasping a cylinder) satisfactorily and report whether or not s/he perceived the probe stimulus by a two-alternate forced choice (yes/no). All yes/no responses are compiled and separated by time relative to the “go” cue and normalized relative to movement onset. Probe stimulus detection is visualized by plotting percent correctly detected over time.

Indeed, data suggest that before any movement is performed (< -50 ms), detection is near 100% (Buckingham et al., 2010). But, just before movement onset (> -50 ms) detection rates sharply decrease. In other words, just before an actor grasps a coffee cup, detection threshold increases such that tactile events are difficult to detect. However, prior studies (e.g., Chapman et al., 1987; Milne et al., 1988; Williams et al., 1998) examined sensory suppression in relatively simple tasks, such as abducting the index finger and, thus, only contracting the first dorsal interosseus muscle. It is the core tenet of this thesis that tasks such as these do not reveal the entire picture. Indeed, ecologically valid tasks need to be employed, particularly when haptic feedback is likely to provide useful information to complete the task. In the context of ecologically valid task, tactile gating has been examined in pointing (Buckingham et al., 2010), juggling (Juravle & Spence, 2011), grasping (Juravle et al., 2011), and during normal gait (Duysens et al., 1995; Morita et al., 1998; Staines et al., 1998). These previous experiments agree that detection of tactile events decreases during
movement. However, there is debate regarding the cause of gating – is it of central (i.e., cortical) origin or simply due to sensory reafference? It is another core tenet of this thesis that tactile gating is central in origin and that it arises from predictive mechanisms (Bays et al., 2006; Buckingham et al., 2010; Voss et al., 2008).

1.6.1 Mechanisms underlying Tactile Gating

Recent data indicate a predictive mechanism that has the motor plan as its input and adjusts the afferent input to reduce the amplitude of possibly irrelevant information. This may be an analogue of predictive sensory cancellation as was observed in the well-studied electrosensory organ of the electric fish (Bell, 2001). Bell (2001) observed that expected sensory input was subtracted out given they do not provide any new information. Electrosensory neurons are sensitive to afferent activity and efferent signals. Bell paired excitatory and inhibitory stimuli at different intervals and recorded neuron activity in electrosensory lobes of fish and found excitatory and inhibitory activity exhibit inverse discharge patterns (Bell, 1986). What are the neural mechanisms that account for this tactile suppression? During any motor task the motor command is sent to the effector muscle groups via descending motor tracts, namely the internal capsule to the lateral corticospinal tract and finally terminating at the alpha motor neurons. Concurrently, an efference copy of the same motor command is sent to the primary somatosensory area, via short intracortical axon bundles, and this mechanism is predictive in nature (Bays et al., 2006). It is this efference copy that elicits suppression of afferent somatic perception (Chapman et al., 2006; Voss et al., 2006, 2008). Indeed, tactile suppression has been observed even in the absence of movement, that is participants reported lower tactile detection and this reduction occurred before movement onset (Buckingham et al., 2010; Voss et al., 2006). Tactile suppression in the absence of movement is a striking result given tactile gating was thought to be the result
of sensory reafference generated from the movement itself. Suppression occurs at the level of the spinal cord in non-human primates (e.g., Seki et al., 2003) and more centrally in the cortex (Morita et al., 1998; Nakata et al., 2005; Shimazu et al., 1999; Voss et al., 2006, 2008; Wasaka et al., 2005).

Indeed, reafference does not appear to be the principal cause of tactile suppression. In a study by Bays and coworkers (2006), participants discriminated the strength of a tap on the left index finger. Two groups of participants were recruited for different protocols, group A and group B. Group A participants rested their left and right index fingers in a molded support attached to a torque motor that was occluded from view. The right index finger was held above the left index finger so that the right index finger was constrained to a single-joint flexion movement. On contact trials participants used the right index (or, active) finger pressing the force sensor that, in turn, activated a torque motor moving a lever contacting the left (or, passive) finger. On contact trials with delay, participants tapped with the right index finger on a force sensor and two taps were delivered to the left index finger by the torque motor (each tap was separated by 700 – 1,200 ms). Participants were asked to discriminate which tap was stronger by pressing one of two response buttons. On no-contact trials the top force sensor was moved without the participant’s knowledge before the go signal. Participants made finger flexion movements with their right index finger but it did not contact the torque motor to elicit a tap on the left index finger. Group B participants experienced an identical procedure as Group A participants but no contact was made with the force sensor on all trials. Results showed that Group A perceptual discrimination between perceived tap magnitude was near equality but contact trials show impaired perceptual discriminability. Interestingly, the no-contact condition shows the same pattern of perceptual
attenuation as the contact trials. In Group B, no differences between delay and contact trials were observed. Given that the participant right finger did not make contact with the force transducer to elicit a tap on the left finger, there was less role for sensory reafference to cause the suppression. Taken together, the results from Bays and coworkers suggests that tactile perception depends upon a forward model that is sensitive to expected sensory information. If a movement occurs like tapping fingers together, it is expected that a tap will occur.

Introducing an unexpected failure to make contact still resulted in gating. The experiment presented by Bays and coworkers demonstrates that sensory feedback is not necessary for this to occur.

Conversely, during movement, sensory reafference accounts for suppression as passive movement of a limb (Chapman & Beauchamp, 2006; also see Staines et al., 1998 for sensory suppression in the lower limb in humans) elicits tactile gating. The above observations support the idea that tactile suppression during movement is the result of feedforward mechanisms rather than sensory inflow. It is still unclear from these observations the purpose of tactile gating. Is it to remove irrelevant and unnecessary afferent information? In other words, does tactile gating serve to remove as much sensory noise as possible (see Section 6 below). Alternatively, tactile gating may serve to optimize sensory flow in which case particular afferent information is allowed to “pass the gate” and be processed. Both questions result in very similar experimental outcomes and it requires subtle experimental design to parse the difference. Therefore, Experiment Five attempts to answer these questions (Chapter 6).

What about the influence of other sensory modalities – vision, in particular. It is critical to grasp the role vision plays in tactile gating because most reaching and grasping
movements are made with vision of the target object and it is known that vision modulates tactile sensitivity (Tipper, Lloyd, Shorland, Dancer, Howard & McGlone, 1998; Tipper, Phillips, Dancer, Lloyd, Howard & McGlone, 2001). We commonly use vision and tactile perception in conjunction when using tools, grasping or manipulating objects. Vision and touch ascend the sensory axons and ultimately integrate in cortical multisensory areas. When participants are asked to respond to tactile stimulation, reaction times decrease when participants see the stimulated limb, even when the tactile stimuli were not seen (Tipper, Lloyd, Shorland, Dancer, Howard & McGlone, 1998; Tipper, Phillips, Dancer, Lloyd, Howard & McGlone, 2001). Furthermore, two-point discrimination thresholds decrease when participants received non-informative vision of their stimulated arm relative to a condition when a neutral object was presented in the same spatial location as tactile stimuli (Kennett, Taylor-Clarke & Haggard, 2001). Furthermore, Taylor-Clarke, Kennett & Haggard (2002) observed enhanced N80 component of the sensory-evoked potential when participants had vision of the limb. These observations demonstrate visual enhancement of touch and it is critical that vision effects are examined. Experiment Three attempts to examine the role of vision in tactile gating (Chapter 4).

1.6.2 The Role of Cognitive Mechanisms

It is possible that cognitive mechanisms (e.g., attention shift away from tactile stimuli) account for the observed reduction in detected tactile stimuli in the context of goal-directed movements. It is a reasonable hypothesis that during a goal-directed movement attention should be directed to the moving limb. Therefore, one should predict higher detection rates at the moving limb relative to a stationary limb.

Contrary to this hypothesis, ample evidence shows that attention plays no role in tactile suppression. Bays et al., (2006) also concluded that attention couldn’t account for
tactile suppression. Group A participants had to attend to two different tasks – motor generation and sensory judgement and so did group B. If attention played a role the perceptual judgement data between groups A and B would be identical. This was not observed and the authors concluded that tactile suppression is due to some other physiological mechanism rather than the task’s attentional demands.

Similarly, Juravle et al., (2011) made the same conclusion. Participants performed a dual-task whereby they were asked to make speeded reach and grasp movements followed by a speeded detection of a vibration on the distal phalanx of the index finger. The vibration was delivered with equal likelihood to either hand or with a higher probability to one hand or the other and was delivered before, during, or after movement. Participants grasped the object with a power palmar grasp. When participants detected a tactile pulse they pressed down a pedal with their foot while still executing the movement. Attention was manipulated with higher probabilities of tactile stimulation at either the moving hand or the resting hand. The results showed faster responses to tactile stimulation when there was higher probability of tactile stimulation to either the moving hand or the resting hand. This effect may be due to an attentional shift to the hand more likely to be stimulated with a vibration. However, participants’ reaction times slowed when participants were stimulated with a vibration during the motor preparation phase before movement onset thus indicating a possible dual-task interference (i.e., psychological refractory period, see Welford, 1952). Furthermore, vibration detection thresholds were not different between the preparation and execution phases irrespective of the probability of tactile stimulation. This indicates that preparing to move an effector does not elicit a shift in attention to that effector but that the effect is likely due to an inhibitory mechanism due to the movement itself that begins before movement.
onset (Juravle et al., 2011). Therefore, sensory suppression due to attention shift cannot explain the observed tactile suppression.

1.7 Global Question and Hypotheses

The overall question guiding this thesis is how does the central nervous system manage and process the high volume of somatosensory information arising from the planning, initiation and execution of goal-directed movements. This question can be subdivided into three overarching themes: i) how does tactile gating function in a complex, goal-directed movement; ii) how does tactile gating behave in response to different task demands; and iii) how does tactile gating interact with other sensory modalities? The present dissertation contains five experiments to address hypotheses arising from questions above.

Experiment One addresses the hypothesis that tactile gating will manifest before movement onset as it is primarily governed by a predictive mechanism and will only occur at the limb performing the movement. Experiment Two seeks to test the hypothesis that tactile gating will diminish during movement, returning tactile sensitivity to baseline later in execution. Baseline return is expected because tactile information from the hand is relevant to successful task completion and movement-related sensory noise diminishes as object contact is approached. Experiment three seeks to test the hypothesis that tactile sensitivity will decrease when vision is available compared to no vision as visual information will dominate sensory perception following the visual dominance effect (Colavita, 1974; Hartcher-O’Brien, Gallace, Krings, Koppen & Spence, 2008). Experiment four tests the hypothesis that changing the limb segment that contacts the target should change tactile gating location such that it will not be observed at the segment contacting the target object. Specifically, grasping the target object with fifth digit and thumb should result in improved tactile sensitivity at the fifth digit. Experiment five attempts to test the hypothesis that tactile
gating acts to optimize task-relevant sensory input. Specifically, pantomime grasping will reduce tactile sensitivity at the limb segments that would contact the target object during normal grasping. If tactile gating acts to optimize sensory input, tactile sensitivity will be diminished at the index finger in pantomimed grasping and unaffected when grasping.
2 Chapter: Tactile Gating in a Reaching and Grasping Task (Experiment One)

2.1 Thesis Context for Experiment One

Experiment One attempts to show how tactile gating functions in a goal-directed upper limb movement and establish a viable experimental paradigm from which subsequent experiments shall follow. Experiment One presents a behavioural paradigm that has a participant make reaching and grasping movements to a target and asking a participant to make perceptual judgments regarding the presence of a tactile tap at one of six body locations: i) left index finger, ii) left fifth digit, iii) left forearm, iv) right index finger, v) right fifth digit and vi) right forearm. Experiment One differs from subsequent experiments in that it tests more body locations. The astute reader will note that other experiments do not include the fifth digits in the design (as in the case of Experiments Two and Five) while another (Experiment Two and Five) excludes particular left limb locations. Likewise, subsequent experiments also have different numbers of stimulation epochs for the sake of simplifying experimental protocol. Experiment One presents data that follows previous work (e.g., Buckingham et al., 2010) showing how goal-directed reaching and grasping elicits tactile gating in a pattern not previously observed. Specifically, tactile gating manifests strongly at the right forearm, somewhat at the right fifth digit and no tactile gating at the right index finger, left index finger, left fifth digit and the left forearm. These data presented in Experiment One show that tactile gating has a central origin given that it occurs well before movement onset.

2.2 Background for Experiment One

In the context of motor output, the sensory system detects, identifies and recognizes sensory patterns to guide an appropriate response. For example, an actor pours coffee into a
mug, but some coffee spills on the side of the mug. Immediately, the actor gets a dish towel and grasps the mug to wipe it clean. Tactile information from the hands becomes particularly important for successfully grasping and cleaning the mug. Logically, it is advantageous for the central nervous system to facilitate processing of signals that convey touch information from the coffee on the mug because decreased friction exists between the surface of the skin and the mug. That is, tactile signals from the fingertips should be readily perceived by the actor, whereas tactile signals that are not relevant to the goal of a movement will be ignored.

This reduction in the ability to perceive tactile stimuli during movement is known as ‘tactile gating’ (see Chapman et al., 1987; Rushton et al., 1981). This gating is often measured as the percentage of correct reports of a probe stimulus or by calculating sensitivity ($d'$). Several studies (Buckingham et al., 2010; Chapman et al., 1987; Milne et al., 1988; Rushton et al., 1981; Voss et al., 2008; Williams et al., 1998) reported suppression of tactile sensations just before and during movement. The majority of these studies (e.g., Chapman et al., 1987; Milne et al., 1988; Williams et al., 1998) have examined sensory suppression in simple motor tasks, such as abducting the index finger. More recently, tactile gating has been examined in a wider variety of visuo-motor tasks, such as pointing (Buckingham et al., 2010), juggling (Juravle & Spence, 2011), grasping (Juravle et al., 2011), and during normal gait (Duysens et al., 1995; Morita et al., 1998; Staines et al., 1998). None of these studies, however, examined how tactile gating manifests at task-relevant vs. task-irrelevant locations on the moving limb (cf. Williams & Chapman, 2002).

Furthermore, these previous experiments have failed to resolve the debate regarding whether the suppression is caused by central or peripheral sources. Indeed, there is evidence that cortical networks involving the prefrontal cortex drive somatosensory gating (Bolton et
Bolton and colleagues (2011) observed higher P100 event-related potential amplitude (ERP), sensitive to the direction of spatial attention (see Hillyard, Vogel & Luck, 1998), when tactile stimuli were attended. However, factors such as task difficulty, task type, attentional manipulation and the characteristics of the stimulus itself can influence how tactile gating manifests. The present study directly examined these issues and provides evidence that tactile gating is central in origin, arises from predictive mechanisms (Bays et al., 2006; Voss et al., 2008), and is restricted to task-specific parts of a moving limb – all within the context of goal-directed grasping movements.

2.3 Methods

2.3.1 Participants

Participants (8 females, 6 males) were recruited from the local graduate and undergraduate population (mean age = 24 years; SD = 3.99). They were all self-reported right-handed individuals, had normal or corrected-to-normal visual acuity, and reported no previous neurological conditions. Participants gave written informed consent, and all procedures were approved by the local research ethics board.

2.3.2 Apparatus

An Optotrak Certus (Northern Digital, Inc., Waterloo, ON) tracked at 250Hz the three-dimensional position of three infrared-emitting diodes (IREDs) affixed to the index finger, thumb and wrist of each participant’s right hand. Six custom-built tactile micromotor vibrators (tactors) were taped to the dorsal surface of the proximal phalanx of the left and right index fingers, the dorsal surface of the proximal phalanx of the fifth finger of both hands and dorsal surface of the mid-forearm of both arms. Micromotor vibration stimuli consisted of a single 7.5 ms long vibration burst which caused a 1mm deformation of the skin resulting in the perception a readily detectable tap at rest (17 mm long, 7 mm diameter,
weigh 1 g). Participants were seated in an upright padded chair with the left arm resting on a flat grasping surface that was at the level of the upper abdomen. The right arm always began at the home position that was 35 cm to the right of each participant’s midline. The elbow was flexed at 90°.

2.3.3 Task

On each trial, participants performed speeded reaching and grasping movements with the right hand to a target object cylinder concluding with a simple lift off the reaching surface. Once the grasping movement was complete, participants made a detection judgment whether a vibration was felt (i.e., yes/no) and where the vibration was felt (e.g., left mid forearm, proximal phalanx of the left index finger, proximal phalanx of the left fifth digit etc).

Data collection took place inside a small sound-isolated room. Participants sat in front of the horizontal reaching surface, wearing liquid crystal display goggles to occlude vision during the period between trials. All trials began with the right hand 30 cm to the right of the midline and 15 cm in front of the torso and the left hand in the mirror symmetric location. A computer generated tone (2000 Hz, 300 ms duration) warned participants that a trial was imminent and 1 second later the goggles opened. After a subsequent variable foreperiod (1000-1500 ms) the imperative cue consisting of a piezoelectric auditory buzzer (50 ms duration) was presented and participants reached out and grasped the 2 cm diameter and 5 cm high polyvinyl chloride (PVC) cylinder with the index finger and thumb of the right hand. They were required to initiate the movement within 400 ms after the buzzer and complete it in 800 ms or less. Movement initiation was defined as sustained velocity of 50 mm/s for 50 ms. On each trial, the cylinder was located at one of two possible target locations that the experimenter changed randomly during the inter-trial period. The locations
were 5cm to the left or right of a position 25 cm directly anterior to the home location for the right hand to prevent participants from predicting target location.

The micromotor vibrations occurred during one of several epochs relative to the imperative cue from 0 ms (at the same time as the imperative cue) to 360 ms (after the imperative cue) in 60 ms bins (i.e., 0, 60, 120, 180, 240, 300 and 360 ms). Once a trial was successfully completed, the LCD goggles closed and participants made a yes/no choice (Y/N) regarding the occurrence of a vibration. In addition, if a vibration was detected, the participant verbally indicated where on the body the vibration was felt (e.g., “left index finger”). There were ten trials per epoch per vibrator motor (i.e., 10 trials with a delay of 0 ms, 10 trials with a delay of 60 ms, etc). In addition there were 10 catch trials per motor in which no vibration was delivered, to assess participants’ false alarm rate. Each experimental session was, thus, comprised of 420 trials and lasted between 100 – 120 minutes. Participants were given opportunities to rest throughout the procedure.

2.3.4 Data Analysis

All trial data were segmented into 60-ms time bins to achieve temporal accuracy because a reaction time of any given trial may widely differ. To capture the time at which the stimulus was delivered relative to movement onset we subtracted each participant’s reaction time (for each trial) from the time relative to the imperative cue (e.g., 60 ms – 300 ms = -240 ms). Nine time bins were created such that they collectively spanned 359 ms before movement onset through 180 ms after movement onset. The time bins were organized as follows: -359 to -300 ms, -299 to -240 ms, -239 to -180 ms, -179 to -120 ms, -119 to -60 ms, -59 to 0 ms, 1 to 60 ms, 61 to 120 ms, and 121 to 180 ms. Every participant’s set of trial data were organized into the stimulation epochs relative to the imperative cue (see above).
However, too few cases were included into the first and last time bin (i.e., -359 to -300 ms and 121 to 180 ms, respectively) and, therefore, excluded from further analysis.

Sensitivity ($d'$) and criterion ($C$) were calculated for every condition within each participant (Gescheider, 1997). Sensitivity was calculated by subtracting the false alarm $z$-score ($Z_{fa}$) from the hits $z$-score ($Z_{h}$; see Gescheider, 1997, p. 119). False alarm rates were pooled together across all conditions and were used to calculate $d'$. Half the sum of $Z_{h}$ and $Z_{fa}$ resulted in $C$. Negative $C$ values reflect bias toward frequent “yes” responses, whereas positive values of $C$ reflect bias toward frequent “no” responses (Gesheider, 1997). $C$ was chosen because the range of $C$ does not depend on $d'$ (Gescheider, 1997).

In addition to the detection variables, several different movement performance variables were also monitored. These included reaction time, movement time, peak velocity, peak acceleration, and peak grip aperture. All detection and movement performance variables were submitted to a 6 (vibration location: left and right index finger, left and right fifth digit, left and right forearm) x 7 vibration (-299 to -240 ms, -239 to -180 ms, -179 to -120 ms, -119 to -60 ms, -59 to 0 ms, 1 to 60 ms, 61 to 120 ms) epoch repeated measures analysis of variance (ANOVA$_{RM}$). All statistically significant effects and interactions were subjected to paired samples t-tests for all possible pairwise comparisons with no correction for multiple comparisons. Statistical significance was set to $p < .05$. Gating was defined as a significant reduction in detection and $d'$ relative to the first time bin. Error trials were removed from analysis and the trial was re-run later in the experimental session. An error consisted of dropped target, occluded IRED marker, reaction time longer than 400 ms, or movement time longer than 850 ms. The average error trials rate across all participants was 7%.
2.3.5 Determining Tactile Gating Onset

In addition to the group average detection data, detection rates over time were calculated and fit with a 4-parameter sigmoid regression curve (SigmaPlot, SYSTAT Systems). This was done to determine when gating occurred relative to movement onset. First, participant data was screened to determine if gating was observed. In the present sample \( n = 14 \), two participants did not show gating. As the purpose of the current work was to examine tactile gating, we focused our analysis on the subset of the sample that did experience tactile gating. In the subset that showed gating, gating onset was determined by calculating the point in time of the greatest slope in the sigmoid regression curve. We examined gating onset relative to reaction time using a group-wise one-sample t-test \((\alpha = .05)\). We also correlated gating onset with reaction time using Pearson’s correlation (see Buckingham et al., 2010).

2.3.6 Baseline Detection at Rest

To control for any potential differences in tactile sensitivity across the stimulation sites, eight \( n = 4 \) female of the original 14 participants completed a follow-up baseline condition. In this condition, participants were tested with the same vibrator motors adhered to the same testing sites (i.e., left and right fifth digits, left and right index fingers, left and right forearms) after giving informed consent. The protocol consisted of randomly vibrating one site per trial at one of seven vibrator activation intervals (1, 2, 3, 4, 5, 6, or 7 ms) while both arms remained stationary. The longest vibration duration was similar to that used in the main experiment (i.e., 7.5 ms). Eight repetitions were completed for each combination of stimulation site and duration resulting in 336 stimulation trials. In addition, the same number of trials without stimulation were randomly interspersed throughout the protocol, resulting in a total of 672 trials. At the end of each trial, participants were required to indicate whether
they felt the stimulation and, if so, at what site. The statistical design was similar to that performed in the main experiment.

2.4 Results

2.4.1 Baseline Detection at Rest

Baseline results demonstrate differences between stimulation sites only at the shortest stimulation time (i.e., 1-ms stimulation). The interaction between vibration site and vibration duration achieved significance [$F(30,150) = 2.45, p = .0001$]. The main effect of vibration site achieved significance [$F(5,35) = 4.69, p = .002$], and the main effect of vibration duration achieved significance [$F(6,42) = 32.9, p = .0001$]. Subsequent simple main effects analyses were conducted targeting the difference in detection rates across sites for each vibration duration (e.g., detection rates of all vibration sites at the 1-ms duration, and so forth). At the 1-ms duration, there was a main effect of vibration site [$F(5,35) = 3.39, p = .013$]. Least significant difference (LSD) post-hoc comparisons revealed significantly lower detection rates at the left forearm compared to the left index finger and fifth digit ($p = .015$ and $p = .012$, respectively). On the right side, post-hoc comparisons only revealed a significant difference in detection rate between the right forearm and the right index finger ($p = .042$). Hence, on the right side, the lowest detection rate was observed at the right forearm. Importantly, there were no differences between the index fingers, fifth digits or forearms (all $ps > .10$), indicating equivalent detection rate performance between left and right sites.

There were no significant effects at any of the other durations (all $ps > .15$), with the interesting exception at the 6-ms duration that achieved a main effect of vibration site [$F(5,35) = 3.08, p = .021$]. However, LSD post-hoc comparison did not reveal any statistically significant differences in detection rate (all $ps > .05$) at the 6-ms duration.

2.4.2 Sensory Detection
For summaries of the detection rate and movement data see Tables 2.1 and 2.2. The omnibus repeated measures ANOVA of $d'$ revealed significant main effects of vibration location, $F(5,65) = 24.177, p < .001$, and vibration epoch $[F(6,78) = 11.370, p < .001]$. There was also a significant two-way interaction between vibration location and vibration epoch $[F(20,390) = 6.495, p < .001]$.

### Table 2.1 Detection Rate Summary.

Average proportion of correctly detected stimuli across all participants. The first column shows the time bins in which the detection data were calculated. Comparisons were made with respect to the first time point (i.e., -299 to -240 ms). *$p < .05$. **$p < .01$. ***$p < .001$. Collective false alarm rate was 0.02% across all participants.

<table>
<thead>
<tr>
<th>Stimulation Times</th>
<th>Left Fifth Finger</th>
<th>Left Index Finger</th>
<th>Left Forearm</th>
<th>Right Fifth Finger</th>
<th>Right Index Finger</th>
<th>Right Forearm</th>
</tr>
</thead>
<tbody>
<tr>
<td>-299 to -240 ms</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
<td>0.89</td>
<td>0.99</td>
<td>0.93</td>
</tr>
<tr>
<td>-239 to -180 ms</td>
<td>1.0</td>
<td>1.0</td>
<td>0.98</td>
<td>0.63**</td>
<td>0.91</td>
<td>0.52**</td>
</tr>
<tr>
<td>-179 to -120 ms</td>
<td>1.0</td>
<td>0.98</td>
<td>0.98</td>
<td>0.77*</td>
<td>0.97</td>
<td>0.54**</td>
</tr>
<tr>
<td>-119 to -60 ms</td>
<td>1.0</td>
<td>0.99</td>
<td>0.98</td>
<td>0.87</td>
<td>0.97</td>
<td>0.50***</td>
</tr>
<tr>
<td>-59 to 0 ms</td>
<td>1.0</td>
<td>1.0</td>
<td>0.97</td>
<td>0.93</td>
<td>0.96</td>
<td>0.48***</td>
</tr>
<tr>
<td>1 to 60 ms</td>
<td>1.0</td>
<td>1.0</td>
<td>0.97</td>
<td>0.84</td>
<td>0.96</td>
<td>0.53***</td>
</tr>
<tr>
<td>61 to 120 ms</td>
<td>0.98</td>
<td>1.0</td>
<td>0.96</td>
<td>0.87</td>
<td>0.94</td>
<td>0.59***</td>
</tr>
</tbody>
</table>
Table 2.1 Movement performance summaries.
Mean movement performance and kinematic data from all participants, across all vibration conditions. Values are reported mean followed by standard deviation (SD) in parentheses. There were no changes observed to movement performance and kinematics.

<table>
<thead>
<tr>
<th>Movement Parameter</th>
<th>Left Fifth Digit</th>
<th>Left Index Finger</th>
<th>Left Forearm</th>
<th>Right Fifth Digit</th>
<th>Right Index Finger</th>
<th>Right Forearm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reaction time, ms</td>
<td>272 (29)</td>
<td>274 (31)</td>
<td>271 (30)</td>
<td>273 (31)</td>
<td>272 (28)</td>
<td>272 (30)</td>
</tr>
<tr>
<td>Movement time, ms</td>
<td>594 (154)</td>
<td>593 (168)</td>
<td>596 (172)</td>
<td>598 (177)</td>
<td>599 (170)</td>
<td>595 (169)</td>
</tr>
<tr>
<td>Peak velocity, mm/s</td>
<td>1225 (102)</td>
<td>1230 (110)</td>
<td>1235 (117)</td>
<td>1222 (104)</td>
<td>1226 (104)</td>
<td>1224 (107)</td>
</tr>
<tr>
<td>Peak acceleration, mm/s²</td>
<td>9066 (1481)</td>
<td>9091 (1499)</td>
<td>9161 (1547)</td>
<td>9028 (1467)</td>
<td>9110 (1535)</td>
<td>9142 (1470)</td>
</tr>
<tr>
<td>Peak Grip aperture, mm</td>
<td>64.9 (0.4)</td>
<td>65.3 (0.4)</td>
<td>64.9 (0.3)</td>
<td>64.5 (0.5)</td>
<td>64.8 (0.2)</td>
<td>64.7 (0.3)</td>
</tr>
</tbody>
</table>

Subsequent one-way repeated measure ANOVAs were performed to break down the significant two-way interaction. Post-hoc comparisons for vibration location confirmed that the right limb displayed lower sensitivity (left & right fifth digits, $p = .004$; left and right second digits, $p = .018$; left and right forearms, $p < .0001$). A significant effect of vibration epoch was found only at the right fifth digit [$F(6,78) = 6.840, p < .0001$] and at the right forearm [$F(6,78) = 11.571, p < .0001$]. There were no significant effects at all other vibration locations ($ps > .09$). Post-hoc comparisons of the right fifth digit revealed that the -
239 to -180 ms time bin decreased the most in sensitivity and it was significantly different from all other time bins \((ps < .05)\), with the largest difference between that and the preceding time bin \((\Delta = 1.22, p < .0001)\). Post-hoc comparisons of the right forearm confirmed that largest decrease occurred between the -299 to -240 ms and the -239 to -180 time bin \((\Delta = 1.7, p < .0001)\). There were no significant differences between all other time bins \((ps > .25; \text{See Figure 2.1}).\)

Figure 2.1 Sensitivity \((d')\) at all vibrator motor locations. Sensitivity \((d')\) calculated from hits and false alarms when the right or left arm was stimulated with vibration at various times relative to movement onset. Right forearm, A. Right fifth digit, B. Right second digit, C. Left forearm, D. Left fifth digit, E. Left second digit, F. For the right arm, \(d'\) was reduced considerably over the second and third stimulation times and remained diminished; was transiently decreased then returned to baseline at the fifth digit; and remained unchanged at the second digit. For the left arm, \(d'\) remained constant at all stimulation sites and time. Error bars denote standard deviation. *\(p < .05\). **\(p < .01\).
However, $d'$ must be measured in light of a criterion measure as it is known that changes in $d'$ can simply be due to confounding changes in criterion as opposed to a change in the sensitivity of sensory receptors (Gescheider, 1997). Therefore, $C$ was calculated for all conditions for every participant (Figure 2.2). An omnibus repeated-measures ANOVA was performed to analyze $C$ and found the main effects of vibration location [$F(5,65) = 22.533, p < .0001$] and vibration epoch [$F(6,78) = 9.041, p < .0001$]. A significant two-way interaction between vibration location and vibration epoch was found [$F(30,390) = 7.068, p < .0001$].

Post-hoc comparisons of vibration location confirmed that $C$ was higher at the right limb (left vs. right fifth digits, $p = .006$; left vs. right second digits, $p = .019$; left vs. right forearms, $p < .0001$). Subsequent one-way repeated measures ANOVAs were conducted to investigate the vibration epoch revealed significant effects at the right fifth digit [$F(6,78) = 7.342, p < .0001$] and right forearm [$F(6,78) = 8.733, p < .0001$]. Post-hoc comparisons of the right fifth digit showed the -239 to -280 ms time bin was significantly increased relative to all other time bins ($ps < .05$; see Figure 2). Comparisons of the right forearm revealed there was a significant increase in $C$ after the -299 to -240 ms time bin ($\Delta = -0.662, p = .001$). This increase persisted throughout all time bins (all other $ps < .002$).
Figure 2.2 Response Bias (C)

The top panel depicts criterion (C) calculated from hits and false alarms when the left and right arms were stimulated with vibration. Right forearm, A. Right fifth digit, B. Right second digit, C. Left forearm, D. Left fifth digit, E. Left second digit, F. C plotted on the y-axis. Time relative to movement onset is plotted on the x-axis. Error bars denote standard deviation. *p < .05. **p < .01.

2.4.2.1 Determining Tactile Gating Onset

In addition to the group averaged detection data, individual detection rates from the left and right forearm of each individual participant were fit with a 4-parameter sigmoid regression curve (Figure 2.3). The index finger and fifth digit stimulation sites were not considered as no gating was observed to occur at these sites. These curves highlight the
sharp drop in detection rates observed in most participants. This pattern is similar to that observed during single-joint movements (e.g., Chapman et al., 1987).

Figure 2.3 Individual participant detection rate profiles
The above table depicts individual participant detection rate profiles showing that detection rates decrease for 12/14 participants. Also, the drops occur before movement onset. Two participants did not exhibit any gating behavior and 1/14 participants showed tactile gating after movement onset.

The time point of the steepest slope (i.e., the highest rate of change) of each curve was calculated and offers a measure of tactile suppression onset. Tactile suppression occurred before movement onset in the vast majority of the sample (see Table 3). A one-sample t-test was conducted and found that tactile suppression occurred, on average, 177 ms before movement onset [$t(11) = 6.443, p < .0001$]. Additionally, we noted a significant Pearson’s correlation between individual reaction times and suppression onsets [$r(11) = .648, p < .05$].
The present correlation supports the central thesis of the present study that sensory gating is unlikely to be a result of sensory reafference.

Table 2.2 Individual Participant Mean movement onset and tactile gating onset relative to movement onset.

Tactile gating was not observed in participants 4 and 12. Therefore, they did not contribute to the calculation of the mean values at the bottom of the table. Timing values are rounded up to the nearest millisecond. Millisecond, ms; standard deviation, SD.

<table>
<thead>
<tr>
<th>Participant</th>
<th>Mean reaction time, ms (SD)</th>
<th>Gating onset, relative to reaction time; Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>235 (9)</td>
<td>160 ms before</td>
</tr>
<tr>
<td>2</td>
<td>263 (10)</td>
<td>152 ms before</td>
</tr>
<tr>
<td>3</td>
<td>275 (14)</td>
<td>208 ms before</td>
</tr>
<tr>
<td>4</td>
<td>244 (6)</td>
<td>No gating</td>
</tr>
<tr>
<td>5</td>
<td>281 (9)</td>
<td>213 ms before</td>
</tr>
<tr>
<td>6</td>
<td>299 (10)</td>
<td>163 ms before</td>
</tr>
<tr>
<td>7</td>
<td>321 (14)</td>
<td>212 ms before</td>
</tr>
<tr>
<td>8</td>
<td>296 (14)</td>
<td>183 ms before</td>
</tr>
<tr>
<td>9</td>
<td>321 (14)</td>
<td>224 ms before</td>
</tr>
<tr>
<td>10</td>
<td>258 (7)</td>
<td>159 ms before</td>
</tr>
<tr>
<td>11</td>
<td>242 (10)</td>
<td>173 ms before</td>
</tr>
<tr>
<td>12</td>
<td>235 (8)</td>
<td>No gating</td>
</tr>
<tr>
<td>13</td>
<td>251 (10)</td>
<td>53 ms before</td>
</tr>
<tr>
<td>14</td>
<td>295 (12)</td>
<td>222 ms before</td>
</tr>
<tr>
<td>Mean</td>
<td>273 (32)</td>
<td>177 (45) ms before</td>
</tr>
</tbody>
</table>

2.5 Discussion

Previous studies have demonstrated that tactile gating occurs before and during a movement (e.g., Chapman et al., 1987; Milne et al., 1988; Williams et al., 1998). However, they have only examined tactile gating in the context of simple movements in which there
would be no reasonable expectation for task-relevant tactile information. By studying tactile gating within the context of goal-directed movement we can deconstruct the influence of task on tactile gating. The present study aimed to examine the above issues and hypothesized that tactile gating is central in origin, arises from predictive mechanisms (Bays et al., 2006; Voss et al., 2008), and is restricted to task-specific parts of a moving limb. Indeed, the present data support the hypothesis that tactile information is attenuated prior to and at the start of a reach and grasping movement. Interestingly, gating was observed at the right forearm (i.e., at the limb that made the reaching and grasping movement). Also, no gating effect was observed at the digits.

2.5.1 Baseline Detection at Rest

Baseline data clearly show that there was no reduction in detection rates across most stimulus durations, except for the 1-ms stimulus duration. Also, there were no detection rate differences between stimulation sites at most stimulus durations (except at the 1-ms duration). The stimulus duration used in the main grasping experiment corresponded to the longest stimulus duration in the baseline study. More importantly, there were no differences across appendages (i.e., no difference between index fingers, fifth digits and forearms). In light of this baseline data, it is unlikely that detection results from the grasping study are the result of solely baseline detection differences between stimulation sites.

2.5.2 Detection Data

The detection data demonstrate that the tactile gating pattern is affected by task demands. That is, in the right arm substantial suppression occurred in the forearm; whereas it was only transiently present in the fifth digit and did not occur at all in the index finger. By contrast, there was no suppression across all three stimulation locations in the stationary left limb (see Figure 1).
These data are the first to provide evidence for the existence of a relationship between tactile suppression within the moving limb and the task that limb must perform. In particular, we have demonstrated that the presence of tactile suppression depends on the limb being moved, the segment that contacts an object, and when it makes contact. Traditionally, tactile suppression at a moved limb has been observed to be strongest at the limb segment that moved. For example, Chapman et al. (1998) observed tactile suppression to be maximal at the movement effector (in that case the index finger during finger abduction) and systematically reduced the farther away the segment was from the effector. However, in finger abduction there is no expectation for tactile information to be relevant for successful goal completion and, therefore, no need for it to remain effective. By contrast, subcutaneous afferents in the index finger play a crucial role in detecting excessive or insufficient fingertip forces (Johansson & Flanagan, 2009) when performing the reach to grasp movements examined in the current work. Thus, the lack of suppression that we observed for the right index finger reflects the context-dependent requirement for the afferents to maintain their sensitivity.

The findings from the current experiment clearly demonstrate that tactile gating is more complex than had previously been reported. Sensitivity (d') calculations show a reduced sensitivity in the moved limb that are confined to the forearm of the moving limb but not the index finger nor the stationary limb. Importantly, d' was found to be reduced throughout the course of the movement for the forearm of the moving limb only.

In addition to this contextually specific nature of the suppression, there was also evidence of temporal specificity. In particular, the majority of participants experienced tactile suppression before movement onset. This pre-movement gating coupled with an increased
criterion suggests that the suppression stems from a centrally generated predictive sensorimotor planning mechanism. This is further supported by the fact that there was a clear relationship between individual reaction times and suppression onsets (Buckingham et al., 2010). Taken together, this evidence implies that tactile suppression is a consequence of movement preparation – an event that clearly works centrally and before movement takes place.

2.5.3 Importance of Task

In contrast to previous research on tactile gating, the current work has shown that task demands play a significant role in the modulation of tactile gating, with increasingly lower levels of tactile gating at areas closer to the limb segment that contacts the target surface. For the first time, we have shown that tactile suppression is not observed at points of contact with an object in a task requiring tactile feedback. The observation of tactile gating before movement onset suggests that central mechanisms preemptively change the ability to detect tactile events according to the likelihood that a specific limb segment will receive tactile information during the course of a movement. This mechanism is consistent with a feed-forward mechanism that specifies the expected sensory dynamics throughout a movement.

Despite these striking data, other alternative explanations need to be considered before making a conclusion. The present results are difficult to reconcile with the well-documented effect of tactile gating while passively moving a limb (Williams & Chapman, 2002). In the Williams and Chapman (2002) study, a predictive mechanism is unlikely to account for the gating effect since tactile gating occurred in the absence of active movement. In other words, there could not be a central motor command in this context and, by extension, no predictive sensorimotor planning signal. In the case of gating without a motor command, a “postdictive” explanation would suggest gating occurs as a result of sensory inflow in the
presence of other sensory events. Present accounts of the pain gating mechanism agree with the postdictive explanation, the best-known example being the inhibitory inputs from large (Aβ) fibres to the dorsal horn of the spinal cord (Melzack & Wall, 1965). However, the present data demonstrate the occurrence of gating before movement opposes the postdictive view. Grasping is a complex movement that requires relevant sources of tactile information. However, in simple single joint movements, the central nervous system would not predict that tactile information would be used later in the movement and, therefore would be more likely to gate that information. By contrast, tactile gating would not occur at the specific effectors in a grasp (i.e., the fingers and thumb) because tactile information will be a relevant source of information.

2.5.4 Mechanisms Underlying Tactile Gating

Functional magnetic resonance imaging (fMRI) has shed some light on the neural mechanisms underlying somatosensory gating effect. Indeed, decreased blood-oxygen level dependent (BOLD) signal relative to baseline was observed at the parietal operculum when tactile gating was induced (Jackson, Parkinson, Pears & Nam, 2011) and this reduction was only observed during movement preparation. However, Jackson et al. (2011) raise important questions regarding the mechanisms of somatosensory gating.

There are two seemingly complementary arguments. One side argues that somatosensation should be enhanced if an effector will be in close proximity to a target, as predicted by the premotor theory of attention (Rizzolatti, Rigio, Dascola & Umilta, 1987). This idea underlies observations of enhanced somatosensation at an effector when that effector is in close proximity to the target (Huttunen, Wikström, Korvenoja, Seppäläinen, Aronen & Ilmoniemi, 1996). This idea lends credence to claims of “active” touch during exploratory hand movements.
Conversely, feed-forward model accounts argue that self-produced sensory events convey little novel information and should be, therefore, attenuated. This is critical because external events carry important information that may be crucial to an organism’s survival (Von Holst & Mittelstaedt, 1954). Forward models are proposed to generate estimates of sensory consequences of movements and cancel those afferent signals that match the signals predicted by the forward model. Therefore, resources may be dedicated to the preferential processing of externally generated events (Blakemore, Wolpert & Frith, 1998; Frith, Blakemore & Wolpert, 2000; Voss et al., 2006). A recent study (Parkinson, Plukaard, Pears, Newport, Dijkerman & Jackson, 2011) reports data that is consistent with our contention that movement planning attenuates tactile perception. Parkinson et al. (2011) had participants make reaching movements in response to a visual cue and provided tactile stimuli at various points before or after movement onset. The authors predicted, and demonstrated, that a tactile stimulus would need to be delivered to the moving limb at an earlier point in time for participants to judge movement onset and tactile stimulus delivery occurring simultaneously (Parkinson et al. 2011); this finding suggests motor planning leads to tactile gating at the limb that is about to move. Furthermore, these authors also demonstrated that the parietal operculum (secondary somatosensory cortex, S2) was found to express less blood oxygen level dependent (BOLD) response when tactile stimulation occurred at the moving compared to stationary arm.

It is possible that the prefrontal cortex (PFC) provided the inhibitory input to somatosensory cortex in the present study. Indeed, PFC is known to exercise inhibitory control over incoming somatosensory input (Yamaguchi & Knight, 1990). Yamaguchi & Knight (1990) observed enhanced early sensory-evoked potentials (SEP) in patients with
PFC damage compared to control participants. However, it remains unclear how PFC functions when tactile input is relevant to the goal of a task. Chapman (1994) argues that tactile gating is largely central in origin for two reasons. First, gating often occurs before EMG activity in the limb that will move; second, peripheral reaффerence does not have any effect on evoked potentials due to peripheral stimulation. Likewise, the present experiment shows that tactile gating occurs before movement onset. Therefore, it is unlikely that peripheral reaффerence plays a role in tactile gating. However, the present experiment does not show the time course of tactile gating throughout the grasping movement.

It is, however, possible that participants might have experienced prolonged tactile gating after movement onset. Indeed, previous studies observed reduced H-reflex amplitude during passive lower limb movements (Brooke, 2004) and reduced SEP amplitude for passive upper limb movements (Jones, 1981; Rushton et al., 1981). SEP responses from passive limb movements may reflect sensory reaффerence to spinal cord sensory neurons via inhibitory interneurons and, subsequent presynaptic inhibition of the same sensory neurons in the spinal cord. Thus, any ascending sensory volleys are effectively prevented from reaching higher centers (Brooke, 2004). However, there are also central signals from the brain that show sensory attenuation in response to motor commands (Shimazu et al., 1999; Ogata et al., 2009) and these signals modulate sensory input based on the task relevance (Staines et al., 2000). Indeed, somatosensory evoked potentials are attenuated in response to descending motor commands.

It is also well-known that somatosensory evoked potentials are reduced before and during movement (Rushton et al., 1981; Staines et al., 1997a,b). SEPs provide clues regarding the mechanisms underlying sensory gating and have shed light on the influence of
movement itself and highlight the importance of task demands. In a study by Shimazu and coworkers (1999), gating was induced in response to a simple finger and wrist extension and frontal N30, parietal P30 and central N60 SEPs were reduced relative to when movements were not performed. Importantly, Shimazu and colleagues observed that the P14 subcortical potential, the N20 from the primary sensory cortex, and the frontal P22 generated from motor cortex were unchanged. The authors concluded that SEP gating was unaffected by muscle afferent signals. However, it is unclear whether tactile gating occurs in response to the motor command itself. Ogata et al., (2009) provide a clue to this possibility by asking participants to make self-initiated movements. Most studies (the present study included) elicit motor output in response to an imperative cue. By contrast, having participants generate self-initiated movements has the advantage of allowing an investigator to record movement-related cortical potentials (MRCP) such as the Bereitschaftspotential (BP) that precedes movement onset. Ogata and colleagues (2009) demonstrated that the P27 potential recorded at C3’ (2 cm posterior to C3) was found to be different from the resting baseline during the 1500 ms pre-movement time epoch. Other sensory potentials progressively became reduced as movement initiation approached, with most potentials significantly reduced approximately 500 ms before movement initiation. The SEP reduction time course closely resembles the BP time course and these processes may be correlated (Ogata et al., 2009). Furthermore, evidence indicates that BPs are generated in the supplementary motor area (Neshige et al., 1988).

Given this context, we suggest that the present results reflect efference copy signals originating from the motor cortices affecting neuronal activity in the primary sensory cortex, thus, gating task-irrelevant somatosensory signals (see Brown et al., 2015). Unfortunately,
Ogata and colleagues did not measure the correlation between SEP gating and BP generation. Therefore, future research should investigate the possibility of this correlation -- solidifying the link between motor planning and sensory function. Indeed, sensory gating is certainly influenced by task and the expectation of receiving sensory feedback (see above). Staines and colleagues (2000) tested whether SEPs are influenced by task. They chose to stimulate either the tibial nerve or the sural nerve, testing proprioceptive and cutaneous inflow, respectively. They also presented cutaneous stimuli in the absence of movement and tested proprioceptive function by asking participants to match the passively moved left foot with the right foot. Their principal finding was that SEP gating was modulated by the task demands. Specifically, SEPs generated during the passive movement and cutaneous conditions were suppressed when the sural nerve was stimulated, leaving the SEP generated during the position matching condition relatively unmodulated. Conversely, when the tibial nerve was stimulated SEPs generated in the cutaneous condition were reduced with passive movement and position matching. Staines and colleagues support the position that sensory input can be affected at early stages of processing and that sensory gating is sensitive to task demands. The present results support this position, as tactile stimuli delivered to the right forearm were gated but tactile inputs from the right index finger and fifth digit were unaffected. However, again, the task employed by Staines & colleagues was a passive movement of the left foot. Therefore, there would not have been a motor plan generated to elicit gating. This difference in task makes comparison with the present study somewhat troublesome because gating in the present study and gating observed by Staines and colleagues must have been generated by sensory discharges associated with the movement.
In the present experiment, we observed sensory attenuation (or, gating) at specific regions of the moving effector. That observation lends support to the feed-forward model argument that irrelevant sensory events become attenuated if those sensory events do not convey any novel information. However, no sensory attenuation was observed at the location of the right second digit (i.e., index finger), giving the impression that information from the index finger would provide useful information for the purpose of grasping an object. Future studies will be directed at further disentangling the present observation and answering whether the central mechanism attenuates afferent signals deemed irrelevant or optimize inflow by facilitation of afferents from regions that come into contact with objects.

2.6 Experiment 1 Summary

The current data provide new insights into how the largely understudied tactile gating phenomenon occurs in the context of movement planning. Based on the fact that gating was observed before movement onset, our results are consistent with the fact that tactile gating is a centrally-driven effect. Furthermore, tactile gating was not observed to be a global effect across both limbs. Rather, it appears to be specific to the to-be moved effector and specific to segments of skin in that moved effector. Central mechanisms are able to modulate tactile gating depending on the predicted relevance of tactile information. This observation shows that feed-forward mechanisms are modulated in sensorimotor networks, likely optimizing sensory input.
3 Chapter: Time Course of Tactile Gating in a Reaching and Grasping Task (Experiment Two)

3.1 Thesis Context for Experiment Two

Experiment One presents data that establishes a base effect from which subsequent experiments can be compared and from which subsequent experiments modify particular aspects of experimental design for the sake of answering a particular experimental question. Experiment Two seeks to answer how long does the tactile gating effect endure past movement onset. This is a logical step given Experiment One probes tactile sensitivity only 120 ms after movement onset and reaching and grasping movements tend to last several hundred milliseconds. Tactile sensitivity was probed up to 800 ms after movement onset. The reader will observe that Experiment Two replicates results from Experiment One closely but will notice that the left forearm was included in the design removing the left index finger and fifth digit. This change was made for two reasons: i) reduce trial number and ii) allow tactile sensitivity comparison observed in the left and right forearms given prior observations. Experiment Two presents data demonstrating that tactile sensitivity at the right forearm decreases prior to movement onset but recovers prior to movement end suggesting that when the motor plan is sent to the effectors its effect on tactile sensitivity diminishes as the movement progresses.

3.2 Background for Experiment 2

In the context of motor output, the sensory system detects, identifies and recognizes sensory patterns to guide an appropriate response. In the context of goal-directed action tactile information is critical to successfully grasp an object. Microneurographic recording of the median nerve revealed that tactile afferents respond to specific events of a grasp (Johansson, 1996). Therefore, it is advantageous that these signals are processed when the
task goal is to grasp an object. However, observations of tactile gating complicate tactile afferent processing (Buckingham et al., 2010; Chapman et al., 1987; Colino et al., 2014; Milne et al., 1988; Rushton et al., 1981; Voss et al., 2008; Williams et al., 1988; Williams & Chapman, 2002). Previous work found that detection thresholds increase before movement (e.g., Bays et al., 2006; Buckingham et al., 2010; Colino et al., 2014) supposing a feed-forward mechanism arising from motor planning processes that triggers tactile gating (Chapman & Beauchamp, 2006; Milne et al., 1988; Williams et al., 1998). Indeed, cortical networks associated with motor planning drive tactile gating (Shimazu, Kaji, Murase, Kohara, Ikeda, Shibasaki, Kimura & Rothwell, 1999; Yamaguchi & Knight, 1990). Sensory-evoked potential data show enhanced P100 that is sensitive to the direction of spatial attention when tactile stimuli were attended (Bolton & Staines, 2011; also see Hillyard, Vogel & Luck, 1998). In non-human primates imminent voluntary movement presynaptically inhibited sensory input to spinal cord (Seki, Perlmutter & Fetz, 2013). Alternatively, peripheral sources can attenuate tactile signals. Indeed, studies using passive movements to probe tactile gating observed decreased sensory evoked potentials in response to the movement (e.g., Staines, Brooke & McIlroy, 2000). Typically, tactile gating is observed in passive limb movement, from which there is no possibility of a defined motor plan. Therefore, tactile gating is thought to arise from sensory reaference. Sensory reaference masks tactile signals because other afferents, such as spindle, Golgi tendon organ (among others) integrate at the level of spinal cord and interfere with tactile signal transmission. Likewise, Staines and colleagues (2000) found that passive movement of the lower limb is sufficient to attenuate sensory-evoked potentials from the tibial and sural nerves.
Previous work from our laboratory found that tactile gating occurs before movement onset supporting the notion that tactile gating has an arguably cortical origin (see Colino et al., 2014). The present study attempts to answer this problem by examining how tactile sensitivity evolves throughout the course of a goal-directed movement. If gating is central then it follows that tactile detection thresholds should be increased well into the movement at specific sites, as previously found (Colino et al., 2014). But, if peripheral sources play a role, then tactile detection thresholds will improve later in the movement such that participants will detect tactile stimuli readily, compared to pre-movement stimulation.

3.3 Methods

3.3.1 Participants

Participants (7 females, 7 males) were recruited from the local undergraduate and graduate population (median age = 22.5 years; SD = 3.8 years). All participants had normal or corrected-to-normal vision and were self-reported right handers. None reported previous neurological conditions. Participants provided written informed consent and the local research ethics board approved the procedure of the present study.

3.3.2 Apparatus

Micromotors delivered tactile stimuli that consisted of a single 6 ms long vibration burst causing a 1 mm skin deformation and, as a result, participants readily perceived a single tap at rest (micromotor parameters: 17 mm long, 7 mm diameter, weight: 1 g). Six custom-built micromotors were taped to the dorsal surfaces of the second and fifth digit’s proximal phalanx and the right and left forearm’s dorsal surface. Each participant were seated in an upright position in a padded chair with the left arm resting on the reaching surface at the level of the upper abdomen. The right arm always began every trial with the elbow flexed at approximately 80° such that the right hand began at the home position 35 cm to the right of
the midline. An Optotrak Certus infrared camera (Northern Digital, Inc., Waterloo, ON) tracked three infrared-emitting diodes (IREDs) affixed to the index finger, thumb and second metacarpophalangeal joint of the right hand. The target object was a 5 cm high and 2 cm diameter polyvinyl chloride (PVC) cylinder.

3.3.3 Task

On each trial, participants performed speeded reach-to-grasp movements to a target object cylinder concluding with a lift off the reaching surface. When participants completed the required movement, they made a single detection judgment whether a tactile tap was felt (i.e., yes or no) and where the tap was felt (e.g., left forearm, right second digit, right fifth digit, or right forearm). Data collection was performed in a small sound-isolated room. The participant was given liquid crystal goggles to occlude vision during inter-trial interval and was seated such that the reaching surface was before them. Each trial began with the right hand located at the home position and a computer-generated tone (2000 Hz, 300 ms duration) warned participants a trial was imminent. The goggles opened one second later. The imperative cue delivered by a piezoelectric buzzer signaled participants to reach the target object and grasp it with the index finger and thumb of the right hand. All participants were required to initiate their movements within 400 ms of the imperative cue and complete the entire movement within 800 ms. Movement initiation was defined as sustained velocity of 50 mm/s for 50 ms. The target cylinder location was randomly changed between two possible locations during the inter-trial interval. The locations were 5 cm to the left or right of a position directly anterior to the home location preventing participants from predicting target location.

Micromotor taps occurred during one of several epochs relative to the imperative cue from 0 ms to 1,100 ms afterwards in 100 ms increments (i.e., 0, 100, 200, 300, 400, 500, 600,
700, 800, 900, 1000, 1100 ms). If a participant detected a tap, s/he verbally reported where on the body the tap was felt (e.g., “right index finger”). Ten trials per epoch per stimulation site (i.e., 10 trials per site with a delay of 100 ms, 200 ms, etc.) were presented to each participant. Additionally, there were 10 trials in which no tap was delivered, to assess participants’ false alarm rate. Therefore, each experimental session consisted of 480 trials and lasted between 120 – 140 minutes. The experimenter provided each participant several opportunities to rest throughout the protocol.

3.3.4 Data Analysis

All trial data were renormalized with respect to movement onset. This was done to compensate for variable reaction times between trials and captured the time at which the tap was delivered relative to movement onset, for every trial, from the time relative to the imperative cue (e.g., 100 ms – 350 ms = -250 ms). Thirteen time bins spanned from 400 ms before movement onset through 900 ms after movement onset. The time bins were organized as follows: -399 to -300 ms, -299 to 200 ms, -199 to 100 ms, -99 to 0 ms, 1 to 100 ms, 101 to 200 ms, 201 to 300 ms, 301 to 400 ms, 401 to 500 ms, 501 to 600 ms, 601 to 700 ms, 701 to 800 ms, and 801 to 900 ms. However, too few cases were included in the final time bin and, therefore, it was excluded from analysis. Additionally, sensitivity (d') and criterion (C) were calculated for every condition for each participant (Gescheider, 1997). Sensitivity is the difference between the false alarm z-score (Zfa) and hit z-score (Zh); see Gescheider, 1997, p. 119). False alarm rates were pooled across all conditions and used to calculate d’. Lower d’ values indicate worse sensitivity to stimuli. Calculating C is half the sum of Zn and Zta and negative C values reflect bias toward frequent “yes” responses, conversely, positive C values reflect bias toward frequent “no” responses (Gescheider, 1997). Movement performance
variables recorded included reaction time, movement time, total response time, peak velocity, time to peak velocity, peak acceleration, time to peak acceleration, peak grip aperture and time to peak grip aperture.

Detection and movement variables were each analyzed using a 4 (vibration location: left and right forearm, right second digit and right fifth digit) x 13 vibration epoch (-399 to -300 ms, -299 to 200 ms, -199 to 100 ms, -99 to 0 ms, 1 to 100 ms, 101 to 200 ms, 201 to 300 ms, 301 to 400 ms, 401 to 500 ms, 501 to 600 ms, 601 to 700 ms, 701 to 800 ms, and 801 to 900 ms) repeated measures analysis of variance. All statistically significant effects and interactions were subjected to polynomial contrasts. Statistical significance was set to \( p < 0.05 \). Error trials were removed from analysis and the trial was re-run later in the experimental session. An error consisted of dropped target, occluded IRED marker, reaction time longer than 400 ms, or movement time longer than 850 ms. The average error trials rate across all participants was 15%.
3.4 Results

None of the movement performance measures revealed statistically significant effects or interactions (see Table 3.1).

Table 3.1 Mean movement performance and kinematic data from all participants, across all vibration conditions.

Reported values are mean followed by standard error of the mean in parentheses. No changes observed.

<table>
<thead>
<tr>
<th>Movement Parameter</th>
<th>Left Forearm</th>
<th>Right Index Finger</th>
<th>Right Fifth Digit</th>
<th>Right Forearm</th>
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<tbody>
<tr>
<td>RT, ms</td>
<td>295 (9)</td>
<td>298 (8)</td>
<td>298 (7)</td>
<td>297 (8)</td>
</tr>
<tr>
<td>MT, ms</td>
<td>704 (23)</td>
<td>711 (23)</td>
<td>704 (21)</td>
<td>709 (20)</td>
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<tr>
<td>TRT, ms</td>
<td>1000 (25)</td>
<td>1009 (24)</td>
<td>1003 (20)</td>
<td>1006 (20)</td>
</tr>
<tr>
<td>PV, mm/s</td>
<td>1072 (58)</td>
<td>1087 (55)</td>
<td>1082 (53)</td>
<td>1078 (53)</td>
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<tr>
<td>TPV, ms</td>
<td>255 (8)</td>
<td>256 (6)</td>
<td>256 (7)</td>
<td>256 (7)</td>
</tr>
<tr>
<td>PA, mm/s^2</td>
<td>7190 (528)</td>
<td>7306 (510)</td>
<td>7260 (502)</td>
<td>7226 (509)</td>
</tr>
<tr>
<td>TPA, ms</td>
<td>120 (5)</td>
<td>120 (4)</td>
<td>120 (4)</td>
<td>121 (5)</td>
</tr>
<tr>
<td>PD, mm/s^2</td>
<td>6479 (583)</td>
<td>6505 (572)</td>
<td>6468 (557)</td>
<td>6455 (561)</td>
</tr>
<tr>
<td>TPD, ms</td>
<td>391 (18)</td>
<td>389 (16)</td>
<td>387 (16)</td>
<td>391 (16)</td>
</tr>
<tr>
<td>PGA, mm</td>
<td>69 (2)</td>
<td>69 (2)</td>
<td>69 (2)</td>
<td>68 (2)</td>
</tr>
</tbody>
</table>
3.4.1 Sensory Detection

A repeated measures ANOVA analyzing detection rate (Figure 2.4) revealed statistically significant effects of stimulus location \( (F_{3,39} = 15.93, p < .0001, \text{partial } \eta^2 = .55) \), stimulus time \( (F_{11,143} = 15.65, p < .0001, \text{partial } \eta^2 = .54) \) and a two-way interaction between these factors \( (F_{33,429} = 7.58, p < .0001, \text{partial } \eta^2 = .36) \). Subsequent trend analysis revealed a statistically significant linear relationship for stimulus epoch \( (F_{1,13} = 34.36, p < .0001, \text{partial } \eta^2 = .72) \) indicating that detection rate improved across stimulus epochs. Simple main effects analysis was conducted distilling the two-way interaction. Analyses revealed statistically significant effects at the right index finger \( (F_{11,143} = 3.56, p < .0001, \text{partial } \eta^2 = .21) \) with a significant linear trend \( (F_{1,13} = 6.96, p = .02, \text{partial } \eta^2 = .34) \) showing progressively improving detection rates over stimulus epochs. Similarly, a statistically significant effect was found at the fifth finger of the right hand \( (F_{11,143} = 10.18, p < .0001, \text{partial } \eta^2 = .43) \) with a significant linear trend \( (F_{1,13} = 32.54, p < .0001, \text{partial } \eta^2 = .71) \). The present study observed the strongest detection rate decrease at the right forearm \( (F_{11,143} = 16.31, p < .0001, \text{partial } \eta^2 = .55) \) with a strong quadratic trend to decreased detection rate at early stimulus epochs and progressive recovery across later epochs \( (F_{1,13} = 22.75, p < .0001, \text{partial } \eta^2 = .63) \). There was no statistically significant effect at the left forearm \( (F_{11,143} = 0.92, p = .51, \text{partial } \eta^2 = .06) \).
Figure 3.1 Detection rate expressed as a function of hits when the right or left arm was stimulated with vibration at various times relative to movement onset.

Green arrows indicate movement onset and red arrows indicate movement end. Right forearm, A. Right fifth digit, B. Right index finger, C. Left forearm, D. For the right arm, $d'$ was reduced considerably over the second and third stimulation times and remained diminished; was transiently decreased then returned to baseline at the fifth digit; and decreased modestly right index finger. For the left arm, $d'$ remained constant at all stimulation sites and time. Error bars denote standard deviation. *$p < .05$. **$p < .01$. ***$p < .001$. 
Sensitivity was calculated from hit rate and false alarm rate (Figure 3.2) and reflected similar pattern as detection rate.

**Figure 3.2 Sensitivity ($d'$) plotted over tactile stimulus presentation latency relative to movement onset.**

Arrangement same as in Figure 2.4. Result pattern mirrors that of detection rate and $d'$ provides a bias-free estimate of sensitivity and shows sensitivity decreases prior to movement start. Error bars denote standard error of the mean. *$p < .05$. **$p < .01$. ***$p < .001$.**
Omnibus repeated measures ANOVA analyzing sensitivity ($d'$) revealed statistically significant main effects of stimulus location ($F_{3,39} = 17.12, p < .0001, \text{partial } \eta^2 = .56$), stimulus epoch ($F_{11,143} = 17.34, p < .0001, \text{partial } \eta^2 = .57$) and a significant interaction ($F_{33,429} = 6.72, p < .0001, \text{partial } \eta^2 = .34$) between stimulus location and stimulus epoch.

Subsequent main effect analyses revealed statistically significant effects at the right index finger ($F_{11,143} = 4.59, p = .003, \text{partial } \eta^2 = .26$), the right fifth digit ($F_{11,143} = 12.43, p < .0001, \text{partial } \eta^2 = .48$) and the right forearm ($F_{11,143} = 16.71, p < .0001, \text{partial } \eta^2 = .56$) but not at the left forearm ($F_{11,143} = .74, p = .69, \text{partial } \eta^2 = .05$). Statistically significant positive linear trends were found at the right index finger ($F_{1,13} = 10.39, p = .007, \text{partial } \eta^2 = .44$) and at the right fifth finger ($F_{1,13} = 48.76, p < .0001, \text{partial } \eta^2 = .79$) confirming that sensitivity improved at all upper right limb stimulus locations across stimulus epochs (Figure 3.2). A statistically significant quadratic trend was observed at the right forearm ($F_{1,13} = 28.48, p < .0001, \text{partial } \eta^2 = .68$) showing that sensitivity initially decreased and progressively increased across stimulus epochs, thus, reflecting the same trend observed from detection rate.

Repeated measures ANOVA analysis of criterion revealed statistically significant effects of stimulus location ($F_{3,39} = 17.08, p < .0001, \text{partial } \eta^2 = .56$), stimulus epoch ($F_{11,143} = 17.31, p < .0001, \text{partial } \eta^2 = .57$), and a statistically significant two-way interaction between the ANOVA terms ($F_{33,429} = 6.73, p < .0001, \text{partial } \eta^2 = .34$). Subsequent main effects analysis (Figure 3.2) revealed statistically significant effects at all locations of the right upper limb but not at the left upper limb.
Figure 3.3 Response bias (C) plotted over tactile stimulus presentation latency relative to movement onset.

Results mirror those of Figure 3.2. Positive C indicates bias toward frequent “no” responses and negative C values indicate bias toward frequent “yes” responses. Notice that C in the present study mirrors that observed in d’ indicating that sensitivity decrease accompanies response bias shift. The range of C does not depend on d’ (Gescheider, 1997). *p < .05. **p < .01. ***p < .001.

Specifically, statistically significant main effects were found at the right fifth digit \( (F_{11,143} = 12.43, p < .0001, \text{partial } \eta^2 = .48) \), right index finger \( (F_{11,143} = 4.60, p = .003, \text{partial } \eta^2 = .26) \) and right forearm \( (F_{11,143} = 16.67, p < .0001, \text{partial } \eta^2 = .56) \). Statistically significant
negative linear trends were found at the right fifth digit ($F_{1,13} = 47.16, p < .0001$, partial $\eta^2 = .78$) and the right index finger ($F_{1,13} = 10.34, p = .007$, partial $\eta^2 = .44$) indicating that $C$ biased to more frequent “yes” responses as the movement progressed to its end. A statistically significant quadratic trend was found at the right forearm ($F_{1,13} = 28.34, p < .0001$, partial $\eta^2 = .68$) showing that participants collectively had slight bias to “yes” responses then frequent “no” responses and returning to slight bias to “yes” responses.

3.5 Discussion

The present study examined tactile gating time course throughout a goal-directed movement and recovers before movement end. Before movement onset, detection rate and sensitivity were decreased and progressively improved as movement end approached. Previous studies examining tactile gating observed that it occurs before and during movement (e.g., Buckingham et al., 2010; Chapman et al., 1987; Colino et al., 2014; Milne et al., 1988; Williams et al., 1998). Indeed, Williams and Chapman (2002) examined the time course of tactile gating under various conditions and found that detection rate decreased at EMG onset with finger abduction and decreased when the finger was abducted (passively or actively). However, studying tactile gating within the context of goal-directed action elucidates the influence of task. The present results demonstrate that tactile gating has a central origin arising from predictive mechanisms (Bays et al., 2006; Voss et al., 2008). Furthermore, the present results replicate our previous findings (Colino et al., 2014) observing that tactile gating is restricted to specific parts of the moving limb. Tactile gating was observed to occur at the upper right limb forearm and fifth digit. Furthermore, the present study shows that tactile sensitivity recovers approaching movement end opening further research questions regarding the purpose of tactile gating.
The present detection data support the notion that the pattern of tactile gating is affected by task demands. In particular, tactile gating was substantial at the right forearm, significantly muted at the fifth and second digit of the right hand, and completely absent at the left limb. At the right forearm substantial magnitude of tactile gating occurred, somewhat at the fifth digit, index finger but none at the left limb. The present study demonstrates that the locus of tactile gating depends on which limb is moved, the limb segment that contacts the target, and when it makes contact (Figure 2.5). Chapman and coworkers (1998) observed that tactile gating is maximal at the movement effector and systematically reduced the farther away the stimulus location is from the effector. However, in finger abduction there should not be any expectation for relevant tactile information for successful task completion. However, reaching-to-grasp demands tactile information to be processed by the central nervous system as cutaneous afferents play a critical role regulating grip forces (Johansson & Flanagan, 2009) and processing edge orientation (Pruszynski & Johansson, 2014). Despite the statistically significant $d'$ reduction at the index finger, sensitivity decreased by 21% translating to 83% detection rate. Clearly, the present study participants are very much able to detect tactile events at the index finger and sensitivity recovered between 301 to 400 ms after movement onset, well before movement end (mean movement time = 707 (19) ms). Therefore, participants readily perceived tactile stimuli before they contacted the target object. Sensitivity at the other right limb sites saw tactile gating to a larger magnitude than the index finger. At the fifth digit, sensitivity maximally reduced by 48% relative to the first stimulus epoch translating to a 50% detection rate from 199 to 100 ms. In other words, participants were only able to detect tactile stimulus half of the time but recovered to 90% detection rate at 201 to 300 ms post movement onset.
Likewise, $d'$ decreased 56% relative to the first time epoch corresponding to a 53% detection rate reduction. Sensitivity shows reduced sensitivity, to varying degrees, at the right limb but not the stationary limb. Also, present data indicate that tactile gating shows temporal specificity. Particularly, all participants experienced tactile gating before movement onset coupled with shifting $C$ suggests that gating stems from a central predictive mechanisms associated with movement planning. Indeed, the clear relationship between individual reaction times and gating onsets suggests that this may be the case (Buckingham et al., 2010; Colino et al., 2014).

However, $d'$ and $C$ at all right upper limb sites returned to levels observed at the first stimulus epoch. The present $d'$ and $C$ patterns collectively suggest the action of both predictive mechanisms and postdictive mechanisms. The observed pattern observed well into movement suggests that central contributions to tactile gating subside and what are left are peripheral gating sources. Perhaps peripheral sources come to play at muscle activity (EMG) onset and movement start. Certainly, peripheral gating sources can be quite effective increasing cutaneous detection threshold. Indeed, Williams & Chapman (2002) observed EMG onset and tactile gating are correlated observing largest decreases in detection rate at approximately the time when EMG onset occurs. Angel & Malenka (1982) instructed participants to make oscillatory movements with the second metacarpophalangeal joint and correlated detection threshold with movement frequency and found that detection threshold positively correlated with oscillation frequency. In other words faster movement velocities yielded higher detection thresholds. Likewise, Williams & Chapman (2002) found that tactile threshold, while passively moving the index finger, did not change relative to rest performance, citing a backward masking explanation from peripheral reafference. Likewise,
Rushton et al., (1981) observed sensory-evoked potential (SEP) changes as a function of movement velocity with late SEP components being attenuated at higher movement velocities. In the case of gating without a motor command, a “postdictive” explanation would suggest gating occurs as a result of sensory inflow in the presence of other sensory events. Pain gating theory (Melzack & Wall, 1965) posits sensory reafference from large (Aβ) fibres masks pain signals carried by Aδ and C fibres. Removing tactile stimulation unmasks pain signals that subsequently become perceived. Chapman and colleagues (1987) observed increased detection threshold when the stimulated arm was both actively and passively moved relative to no movement. Conversely, when the stimulated arm remained stationary and the contralateral arm was moved there were no observed differences between movement and no-movement conditions. Peripherally, reduced H-reflex amplitude was observed during passive lower limb movements (Brooke, 2004) and decreased SEP amplitude during passive upper limb movements (Jones, 1981; Rushton, Rothwell & Craggs, 1981). It is likely that reduced H-reflex is a consequence of descending inhibitory influence on primary sensory neurons (see Seki et al., 2013). These SEP responses may reflect sensory reafference masking tactile signals using inhibitory circuits in the spinal cord preventing afferent volleys reaching higher centres (Brooke, 2004). However, most of the above examples study tactile gating in the context of oscillatory movements rather than discrete movements; these are two fundamentally different classes of movement. Most importantly, there is no reasonable expectation to use tactile information during a single-joint, oscillatory movement. A complete assessment requires studying tactile gating within a goal-directed, complex movement (or at least any movement in which tactile information during which tactile information is useful). Staines, Brooke & McIlroy (2000) tested whether task
influences SEPs by stimulating the tibial nerve or sural nerve, probing proprioceptive and cutaneous inflow, respectively. Participants were presented with cutaneous stimuli in the absence of movement and proprioceptive function was probed, by matching the passively moved left foot with the right foot. Staines and colleagues observed SEP amplitude reduction during passive movement. Likewise, cutaneous SEPs were reduced during sural nerve stimulation. Tibial nerve stimulation during passive movement produced SEP amplitude reduction. Taken together, Staines and colleagues’ observations support the position that sensory processing can be strongly influenced by peripheral sources. The present observation that tactile sensitivity increased when movement end approached may reflect decreased afferent flow from the right forearm, right fifth digit and right index finger. If that is the case, it follows that $d'$ improves as participants approached movement end. However, Staines and colleagues used a passive movement to study tactile gating, making comparison with the present study somewhat troublesome because gating in the present study occurred before movement onset implicating central gating sources.

The present observations of pre-movement tactile gating reconcile with central, predictive explanations. Predictive mechanisms, such as feed-forward models, argue that self-produced events convey little novel information and are, therefore, attenuated. Jackson, Parkinson, Pears & Nam (2011) observed reduced blood-oxygen level dependent (BOLD) signal relative to baseline at the parietal operculum that was only observed during movement preparation. However, it is difficult to reconcile the present results with accounts of active touch (e.g., Chapman, 1994). Tactile detection thresholds should be reduced if an effector will contact a target (Rizzolatti, Rigio, Dascola & Umilta, 1987; also see Huttunen, Wikstrom, Korvenoja, Seppäläinen, Aronen & Ilmoniemi, 1996). Alternatively, forward
accounts argue neural resources become dedicated to processing external sensory events (Blakemore, Wolpert & Frith, 1998; Frith, Blakemore & Wolpert, 2000; Voss, Ingram, Haggard, & Wolpert, 2006; Voss, Ingram, Wolpert & Haggard, 2008). Indeed, movement planning increased detection threshold of tactile events (Parkinson, Plukaard, Pears, Newport, Dijkerman & Jackson, 2011) consistent with the present observations. Parkinson and colleagues (2011) replicated the observation of reduced BOLD signal at S2 when tactile stimulation was delivered to the moving limb compared to when the limb was stationary. An outstanding question is: what is the source of reduced activity at somatosensory cortex?

One possible candidate is the prefrontal cortex (PFC). Yamaguchi & Knight (1990) measured SEPs in patients with PFC damage and observed enhanced SEP amplitude when the patients were about to move. Indeed, motor planning arises from prefrontal areas and, therefore, tactile gating may arise from the motor plan itself. Ogata, Okamoto, Yamasaki, Shigeto & Tobimatsu (2009) instructed participants to perform self-initiated movements and measured movement-related cortical potentials such as the Bereitschaftspotential (BP) and showed that an early positive potential, namely the P27, changed compared to resting baseline during the 1500 ms pre-movement interval. Furthermore, as movement initiation approached, as indicated by the BP, other cortical sensory-related potentials reduced amplitude. Most SEP amplitudes reduced significantly approximately 500 ms before movement onset. Ogata and colleagues (2009) provide data demonstrating that movement preparation, indicated by BP, influences SEP amplitude reduction. BP is thought to be generated in the supplementary motor area (Neshige, Luders & Shibasaki, 1988). If this is true, then motor planning signals from higher-order motor centres influence sensory areas (see Huttunen et al., 1996; Parkinson et al., 2011). In self-initiated movements positron
emission tomography (PET) shows the left primary somatosensory cortex, supplementary motor area bilaterally, lateral premotor cortex bilaterally and right dorsolateral prefrontal cortex activate (Jahanshahi, Jenkins, Brown, Marsden, Passingham & Brooks, 1995). Of course, it is difficult to interpolate the neural activation time course leading to overt behaviour from PET. However, it is clear that movement planning is associated with tactile gating and further research must be conducted to elucidate this association.

SEPs may elucidate the neural activation time course of cortical structures. These central signals modulate in response to motor planning (Shimazu, Kaji, Murase, Kohara, Ikeda, Shibasaki et al., 1999; Ogata et al., 2009) and in response to task demands (Staines, Brooke & McIlroy, 2000). SEP gating was induced in response to finger and wrist extension reducing frontal N30, parietal P30 and central N60 SEPs. Additionally, P14 subcortical potential, frontal P22 generated at motor cortex were unchanged; only SEPs modulated in response to motor planning and not affected by muscle afferent signals. Indeed, Ogata and colleagues demonstrated that tactile gating likely arises from motor planning processes (see above). The time course of SEP reduction closely follows BP time course.

3.6 Experiment Two Summary
The present study observed tactile gating at specific regions of the moving effector before movement time supporting the notion that central sources are responsible for tactile gating. However, peripheral sources certainly play a role, as $d'$ improved approaching movement end. The strongest attenuation effect occurred at the right forearm and somewhat at the right index finger and right fifth digit, whereas no attenuation occurred at the left limb. The right index finger only saw approximately 20% sensitivity reduction, corresponding to 80% detection rate, leaving participants very much able to detect tactile events. This observation supports the notion that tactile information from the index finger is useful in the
present task. The current data provide insights into the tactile gating phenomenon indicating that central and peripheral gating sources are at work, likely complementing each other reflecting the differences observed in the literature. Tactile gating occurred before movement onset supporting that tactile gating is a centrally-driven effect but $d'$ improved perhaps reflecting the fact that there was little peripheral afferent drive to mask tactile inputs leaving those inputs unmolested. As feed-forward and sensory feedback control mechanisms are both used in motor planning and execution, so too is the sensory system reliant on predictive and postdictive mechanisms to control tactile afferents, likely acting to optimize sensory inflow. Future studies will be directed to investigate interactions with other sensory modalities and determine whether tactile gating acts to optimize inflow or attenuate sensory noise.
Chapter: The Availability of Vision and Tactile Gating in a Reaching and Grasping Task (Experiment Three)

4.1 Thesis Context for Experiment Three

Experiment Three is somewhat a departure from the previous experiments, certainly, relative to subsequent experiments and requires explanation for the sake of logical progression of this thesis. It would be prudent and worthwhile to examine any possible multisensory effects. Indeed, work conducted by Tipper and colleagues (1998; 2001) show that mere visual observation of the stimulated limb enhances tactile sensitivity. The candidate hopes that the reader can now understand the underlying reason for conducting Experiment three. However, Experiment Three will make this explicitly clear. It is valuable to know if mere vision of the limb, even in the periphery, is enough to elicit visually-mediated modulation of tactile sensitivity (i.e., visual enhancement of touch effect). Previous work (e.g., Tipper et al., 1998; 2001) suggests that it does and, therefore, it may be that the effects observed in the Experiments One and Two are the result of visuo-tactile interactions simply enhancing tactile sensitivity. Experiment Three shows that vision enhances tactile sensitivity and replicates base findings from Experiments One and Two. Importantly, Experiment Three shows only a main effect of vision and no interactions were observed suggesting that vision changes the general excitability of the somatosensory system and doesn’t differentially affect tactile sensitivity of different skin locations. With that observation in hand, it allows examining the purpose of tactile gating (Experiments Four and Five).

4.2 Background

The somatosensory system detects, identifies and recognizes sensory patterns to guide appropriate outputs. Tactile information from the fingertips is especially crucial to
successfully grasp an object. Each tactile receptor encodes unique grasp events such as contact, lift, hold, lowering and release. Indeed, Johansson (1996) used microneurography to record from the median nerve and observed these signals in response to specific events of a grasp and specific tactile receptors signaled each unique event. Therefore, it is advantageous for the central nervous system to facilitate sensory signal processing that carry tactile information from the hands when grasping an object is the goal. However, tactile gating complicates this obvious necessity. What are the mechanisms responsible for tactile gating? Do these mechanisms occur at different levels of the CNS. Before any motor task the motor plan is sent to the effector muscle groups via descending motor tracts and terminate at the alpha motor neurons. Concurrently, an efference copy of the same motor plan is sent to the primary somatosensory area of the cortex and this mechanism is predictive in nature (Bays et al., 2006). The efference copy itself triggers the events required for tactile gating reducing afferent magnitude (Chapman et al., 2006; Voss et al., 2005, 2008). Previous work in our laboratory (Colino et al., 2014) and others (Buckingham et al., 2010; Chapman et al., 1987; Milne et al., 1988; Rushton et al., 1981; Voss et al., 2008; Williams et al., 1998) reported tactile gating before and during movement. Furthermore, most studies (e.g., Chapman et al., 1987; Milne et al., 1988; Williams et al., 1998) observed tactile gating in simple tasks, such as index finger abduction. But, tactile gating has been examined in a variety of tasks such as pointing (Buckingham et al., 2010), grasping (Colino et al., 2014; Juravle et al., 2011), juggling (Juravle & Spence, 2011), and in gait (Duysens et al., 1995; Morita et al., 1998; Staines et al., 1998). Previous studies commonly found that participants fail to perceive tactile events just before and during movement. Previous studies in this thesis did not examine the effect of vision on tactile gating. Previous studies (see below) found that vision
has a robust enhancing influence on tactile sensitivity in perceptual judgment and motor tasks. However, this study attempted to examine how the presence of vision modulates tactile gating.

Indeed, humans often grasp objects when those objects are visible. We commonly use vision and tactile perception in conjunction when using tools, grasping or manipulating objects. Vision and touch ascend the sensory axons and ultimately integrate in cortical multisensory areas. When participants are asked to respond to tactile stimulation, reaction times decrease when participants see the stimulated limb, even when the tactile stimuli were not seen (Tipper, Lloyd, Shorland, Dancer, Howard & McGlone, 1998; Tipper, Phillips, Dancer, Lloyd, Howard & McGlone, 2001). Furthermore, Kennett, Taylor-Clarke & Haggard (2001) observed two-point discrimination thresholds decrease when participants received non-informative vision of their stimulated arm relative to a condition when a neutral object was presented in the same spatial location as tactile stimuli. Furthermore, Taylor-Clarke, Kennett & Haggard (2002) observed enhanced N80 component of the sensory-evoked potential when participants had vision of the limb. These observations demonstrate the visual enhancement of touch. The present study examines whether vision modulates tactile gating measuring tactile stimulus detection rate and calculating sensitivity.

4.3 Methods
4.3.1 Participants

Fourteen participants (7 females) were recruited from the local graduate and undergraduate population (median age = 22.5 years; SD = 3.8 years). They were all self-reported right-handed individuals, had normal or corrected-to-normal visual acuity, and reported no previous neurological conditions. Participants gave written informed consent, and the local research ethics board approved experimental procedures.
4.3.2 Apparatus

An Optotrak Certus (Northern Digital, Inc., Waterloo, ON) tracked at 250Hz the three-dimensional position of three infrared-emitting diodes (IREDs) affixed to the index finger, thumb and wrist of each participant’s right hand. Six custom-built tactile micromotor vibrators (tactors) were taped to the dorsal surface of the proximal phalanx of the left and right index fingers, the dorsal surface of the proximal phalanx of the fifth finger of both hands and dorsal surface of the mid-forearm of both arms. Micromotor vibration stimuli consisted of a single 7.5 ms long vibration burst causing 1mm deformation of the skin resulting in a readily detectable tap at rest (17 mm long, 7 mm diameter, weighs 1 g).

Participants were seated in an upright padded chair with the left arm resting on a flat grasping surface that was at the level of the upper abdomen. The right arm always began at the home position that was 35 cm to the right of each participant midline. The elbow was flexed at 90°.

4.3.3 Task

On each trial, participants performed speeded reaching and grasping movements to a target object cylinder concluding with a simple lift off the reaching surface. Once the grasping movement was complete, participants made a detection judgment whether a vibration was felt (i.e., yes/no) and where the vibration was felt (e.g., left mid forearm, proximal phalanx of the left index finger, proximal phalanx of the left fifth digit etc). The participant was given either full vision or no vision in one of two experimental sessions. Full vision refers to complete view of the target and reaching surface before and after the imperative cue. No vision refers to complete view of the target and reaching surface before the imperative cue but was removed coincidentally with the imperative cue.

Data collection took place inside a small sound-isolated room. Participants sat in front of the horizontal reaching surface, wearing liquid crystal display goggles to occlude
vision during the period between trials. All trials began with the right hand 30 cm to the right of the midline and 15 cm in front of the torso and the left hand in the mirror symmetric location. A computer generated tone (2000 Hz, 300 ms duration) warned participants that a trial was imminent and 1 second later the goggles opened. After a subsequent variable foreperiod (1000-1500 ms) the imperative cue consisting of a piezoelectric auditory buzzer (50 ms duration) was presented and participants reached out and grasped the 2 cm diameter and 5 cm high polyvinyl chloride (PVC) cylinder with the index finger and thumb of the right hand. They were required to initiate the movement within 400 ms after the buzzer and complete it in 800 ms or less. Movement initiation was defined as sustained velocity of 50 mm/s for 50 ms. The cylinder was located at one of two possible target locations that the experimenter changed randomly during the inter-trial period. The locations were 5 cm to the left or right of a position 25 cm directly anterior to the home location for the right hand. This spatial uncertainty prevented the participant from predicting target location.

The micromotor vibrations occurred during one of several epochs relative to the imperative cue from 0 ms (at the same time as the imperative cue) to 1100 ms (after the imperative cue) in 100 ms bins (i.e., 0, 100, 200…1100 ms). Once a trial was successfully completed, the LCD goggles closed and participants made a yes/no choice (Y/N) regarding the occurrence of a vibration. In addition, if a vibration was detected, the participant verbally indicated where on the body the vibration was felt (e.g., “left index finger”). There were eight trials per epoch per stimulation site (i.e., 8 trials with a delay of 0 ms, 8 trials with a delay of 100 ms, etc). In addition there were 40 catch trials throughout the experiment in which no vibration was delivered to assess participants’ false alarm rate. Each experimental
session was, thus, comprised of 420 trials and lasted between 100 – 120 minutes.

Participants were given opportunities to rest throughout the procedure.

4.3.4 Data Analysis

All trial data were segmented into 60-ms time bins to achieve temporal accuracy because a reaction time of any given trial may widely differ. To capture the time at which the stimulus was delivered relative to movement onset we subtracted participant reaction time (for each trial) from the time relative to the imperative cue (e.g., 60 ms – 300 ms = -240 ms). Nine time bins were created such that they collectively spanned 359 ms before movement onset through 180 ms after movement onset. The time bins were organized as follows: -399 to -300 ms, -299 to -200 ms, -199 to -100 ms, -99 to -0 ms, 1 to 100 ms, 101 to 200 ms, 201 to 300 ms, 301 to 400 ms and 401 to 500 ms. Participant trial data were organized into the stimulation epochs relative to the imperative cue (see above). However, too few cases were included into the first and last time bins (i.e., -399 to -300 ms and 401 to 500 ms, respectively) and, therefore, these time bins were excluded from further analysis.

Sensitivity (d’) and criterion (C) were calculated for every condition within each participant (Gescheider, 1997). Sensitivity was calculated by subtracting the false alarm z-score (Zfa) from the hits z-score (Zh; see Gescheider, 1997, p. 119). False alarm rates were pooled together across all conditions and were used to calculate d’. Half the sum of Zh and Zfa resulted in C. Negative C values reflect bias toward frequent “yes” responses, whereas positive values of C reflect bias toward frequent “no” responses (Gesheider, 1997). C was chosen because the range of C does not depend on d’ (Gescheider, 1997).

In addition to the detection variables, several different movement performance variables were also monitored. These included reaction time, movement time, peak velocity,
peak acceleration, and peak grip aperture. Sensitivity and movement data were submitted to a 2 (vision, no vision) x 4 (vibration location: right index finger, right fifth digit, right forearm & left forearm) x 7 vibration time (-299 to -200 ms, -199 to -100 ms, -99 to -0 ms, 1 to 100 ms, 101 to 200 ms, 201 to 300 ms, 301 to 400 ms) repeated measures analysis of variance (ANOVA$_{rm}$). All statistically significant effects and interactions were subjected to paired samples t-tests for all possible pairwise comparisons with no correction for multiple comparisons. Statistical significance was set to $p < .05$. Gating was defined as a significant reduction in detection and $d'$ relative to the first time bin. Error trials were removed from analysis and the trial was re-run later in the experimental session. An error consisted of dropped target, occluded IRED marker, reaction time longer than 400 ms, or movement time longer than 850 ms. The average error trials rate across all participants was 6%.

4.4 Results

4.4.1 Sensory Detection

Omnibus repeated measures ANOVA revealed a main effect of Vision [$F(1,9) = 6.031, p = .036$], Stimulus location [$F(3,27) = 14.076, p = .002$] and Stimulus time [$F(6,54) = 6.199, p = .004$]. The main effect of vision revealed that full vision enhanced $d'$ (Figure 2.7).

Also, there was a statistically significant interaction between Stimulus location and Stimulus time [$F(18,162) = 6.266, p < .0001$] but there was no statistically significant interactions between Vision and Stimulus location [$F(3,27) = 1.407, p = .262$] and Vision and Stimulus time [$F(6,54) = 1.225, p = .308$].
The three-way interaction between Vision, Stimulus location and Stimulus time failed to achieve statistical significance \(F(18,162) = 1.096, p = .361\). Subsequent simple main effects analysis was performed on the statistically significant two-way interaction between Stimulus location and Stimulus time. Each stimulus location was analyzed separately determining whether sensitivity changed across stimulation times (Figure 4.2).
Figure 4.2 Tactile sensitivity ($d'$) across time relative to movement onset (msec).
The upper left panel depicts sensitivity at the right arm and the upper right panel depicts the same at the right fifth digit. The lower left panel depicts sensitivity at the right index finger and the lower right panel depicts sensitivity at the left arm. Sensitivity between Vision and No Vision is illustrated. However, the three-way interaction was not statistically significant. *$p < .05$; **$p < .01$; ***$p < .001$.

Simple main effects analysis of the left arm revealed a trend toward statistical significance ($p = .061$) but sensitivity did not change across time at the left arm replicating
previous findings in our laboratory (Colino et al., 2014). Likewise, there were no changes in 
d’ at the right index finger ($p = .241$). However, sensitivity changed across stimulus times at 
the right fifth digit and the right forearm. There was a main effect of stimulus time at the 
right fifth digit [$F(6,54) = 6.381, p = .003$]. Pairwise comparisons between the first time 
point and all others revealed that sensitivity decreased by 1.7 $d’$ units from the first stimulus 
time (mean $d’ = 3.85$) and $d’$ rose steadily toward baseline. These units simply refer to the 
distance, in $z$-score units, between the means of the hit and false alarm distributions. There 
was a main effect at the right forearm [$F(6,54) = 9.765, p = .0001$]. Pairwise comparisons 
revealed that $d’$ decreasing nearly two $d’$ units from the first stimulus time bin (i.e., -299 to 
-200 ms; mean $d’ = 3.73$) to the fourth stimulus bin (i.e., 1 to 100 ms; mean $d’ = 1.77$). Figure 
2.7 depicts $d’$ observations from all four stimulus locations.

In summary, the present study observed right fifth digit and right forearm reduced 
sensitivity whereas no reduction was observed at the left forearm and the right index finger. 
Also, $d’$ reduction occurred before movement start and there was enhancement of sensitivity 
when participants had full vision of the object and reaching surface relative to no vision. 
There were no effects of vision on movement data.

4.5 Discussion

4.5.1 Effect of Vision

Humans often use vision to guide their interactions with objects and the hands and 
arms are often in view. Tactile stimulation reaction time decreases when subjects see their 
hand, even when the tactile stimuli were not seen (Tipper, Lloyd, Shorland, Dancer, Howard 
visual effect may be an example of visual enhancement of touch (Serino & Haggard, 2010).
Visual enhancement of touch is not thought to be an effect of spatial attention (although spatial attention may function in concert with visual enhancement of touch). Visual enhancement of touch persists when attention is experimentally controlled. Indeed, ample evidence shows that attention plays no role in tactile suppression of externally generated tactile events. Bays and coworkers (2006) also concluded that attention cannot account for tactile suppression concluding that it is due to some other physiological mechanism rather than task attentional demands. Juravle and coworkers (2011) arrived at the same conclusion. Like Bays and coworkers (2006) each participant performed a dual-task whereby they were asked to make speeded reach and grasp movements followed by a speeded detection of a vibration on the distal phalanx of the index finger. Attention was manipulated with higher probabilities of tactile stimulation at either the moving hand or the resting hand. The results show faster responses when there was higher probability of tactile stimulation to either the moving hand or the resting hand. This effect may be due to an attentional shift to the hand more likely to be stimulated with a vibration. However, participant reaction times slowed when stimulated with a vibration during the motor preparation phase before movement onset thus indicating a possible dual-task interference (i.e., psychological refractory period, see Welford, 1952). Furthermore, vibration detection thresholds were not different between the preparation and execution phases irrespective of the probability of tactile stimulation. No difference in tactile thresholds indicates that preparing to move an effector does not elicit a shift in tactile perception to that effector but that the effect is likely due to an inhibitory mechanism due to the movement itself that begins before movement onset (Juravle et al., 2011). Therefore, sensory suppression due to attention shift cannot explain the observed tactile suppression. However, the study by
Juravle and colleagues (2011) does not directly manipulate vision nor does it provide direct evidence supporting that visual enhancement of touch is a perceptual context effect.

Kennett, Taylor-Clarke & Haggard (2001) found that two-point discrimination thresholds decreased when participants received non-informative vision of their stimulated arm relative to a condition in which a neutral object was presented in the same spatial location as the stimulation. This observation precludes the possibility that spatial attention has a role in visual enhancement of touch. Furthermore, Taylor-Clarke, Kennett & Haggard (2002) recorded cortical potentials and observed an enhanced N80 component of the somatosensory evoked potential. The authors argue that all the above results support descending feedback from parietal areas affecting processing at primary somatosensory cortex. In a more recent study, Press, Taylor-Clarke, Kennett & Haggard (2004) studied the conditions in which visual enhancement of touch occurs. They conducted four separate experiments that differed in the spatial distribution and difficulty of the tactile stimuli. Press and colleagues (2004) measured participants’ two-point discrimination using a staircase procedure distance on the right forearm and ensured that visual information did not carry any informative cues regarding stimulus activation. Press and colleagues observed that when spatial discrimination difficulty is high, vision of the arm enhanced spatial discrimination as reflected in speeded reaction times. However, participants did not commit any more errors when the arm was viewed compared to viewing an object at the same spatial location precluding the possibility that visual enhancement of touch is not the result of a spatial attention effect.

The present experiment found evidence of visual touch enhancement. Specifically, higher $d'$ scores were observed when participants viewed the reaching surface during the
task. The present results reconcile with data from Press and colleagues (2004) because they observed visual touch enhancement with suprathreshold stimuli – as is presently the case. Interestingly, the present experiment demonstrated visual touch enhancement in a dual-task context and did so measuring simple detection. Evidence suggests that disrupting primary somatosensory activity decreases tactile acuity while viewing the arm (Fiorio & Haggard, 2005). Participants in this study had single-pulse transcranial magnetic stimulation (TMS) applied over primary somatosensory cortex before making tactile gating judgments when the hand was viewed. However, no accuracy reduction was observed when TMS disrupted S1 while a neutral object was viewed. Additionally, no accuracy drop was observed when secondary somatosensory cortex was disrupted and vision could not offer any information regarding tactile stimuli as stimuli were delivered in dark conditions. The Fiorio and Haggard (2005) study offers important clues as to the mechanisms of visual touch enhancement and a strong explanation of the present observation. A recent study identifies anterior intraparietal sulcus (aIPS) contributing to the visual enhancement of touch effect via the integration of vision and somatosensation (Konen & Haggard, 2014). Konen and Haggard (2014) found that disrupting aIPS with TMS abolished the visual enhancement of touch effect. However, applying TMS to other areas of cortex did not have any effect. Taken together, this implies that the visual enhancement of touch may require a feedback loop between aIPS and primary somatosensory cortex (Konen & Haggard, 2014).

4.5.2 Tactile Sensitivity

The current study examined the visual availability effect on sensitivity and observed enhanced sensitivity under full vision relative to no vision. The present study replicated previous findings from our laboratory (Colino et al., 2014) and extends findings from others (e.g., Buckingham et al., 2010; Chapman, 1994; Chapman & Beauchamp, 2006; Williams &
Chapman, 1998; Morita et al., 1998; Staines et al., 1998; Voss et al., 2008). Indeed, previous investigations studied tactile gating within the context of simple finger abduction movements e.g., Chapman et al., 1987; Milne et al., 1988; Williams et al., 1998). Indeed, task demands affect tactile gating demonstrated by the present observations where sensitivity decreased at the right forearm and right fifth digit but sensitivity did not change at the left forearm and the right index finger. Also, sensitivity changed across time with decreasing sensitivity approaching movement onset. Specifically, participants experience the largest sensitivity reduction at the right forearm approaching movement onset. Observing tactile gating before movement onset strongly suggests the presence of predictive sensorimotor processes generated by frontal motor areas and passed to sensory areas (perhaps as an efference copy). Therefore, tactile gating appears to function in a predictive manner; it is the result of movement planning – an event that clearly occurs centrally and propagates peripherally.

Task clearly has an effect on tactile gating; the present observation did not yield tactile gating at the right index finger – the limb segment that contacted the target object in the present protocol. It appears that central mechanisms associated with tactile gating have the ability to gate tactile inputs at a specific limb segment according to the likelihood that a limb segment receives tactile information. Hence, this action is consistent with a feed-forward system that specifies the expected sensory utility throughout a movement suggesting many inputs converge into sensory areas.

However, previous studies (e.g., Williams & Chapman, 2002) observed tactile gating occurred without overt movement making it difficult to reconcile with present observations. Williams & Chapman (2002) passively moved the limb of each subject and observed that without overt, voluntary movement planning could not elicit tactile gating. In other words,
there could not be a central motor command in this context and, by extension, no predictive sensorimotor planning signal. In the case of gating without a motor command, a “postdictive” explanation would suggest gating occurs as a result of sensory inflow in the presence of other sensory events. Indeed, Williams and Chapman proposed a backward masking effect where sensory information from the movement masks or blocks tactile information from processing and, therefore, prevents tactile events reaching conscious perception. Present accounts of the pain gating mechanism agree with the postdictive explanation, the well-known example being the inhibitory inputs from large (Aβ) fibres to the dorsal horn of the spinal cord (Melzack & Wall, 1965). However, the present data disagree with the postdictive account of tactile gating. Namely, tactile gating occurred before movement onset highlighting the strong possibility that tactile gating is largely the result of central motor planning processes. Simple movements, such as finger abduction, do not necessarily demand tactile information to be a useful information source. On the other hand, grasping is a complex movement that requires relevant sources of tactile information. However, in simple single joint movements, the central nervous system would not predict that tactile information would be used later in the movement and, therefore would be more likely to gate that information. By contrast, tactile gating would not occur at the specific effectors in a grasp (i.e., the fingers and thumb) because tactile information will be a relevant source of information.

4.6 Summary

The current experiment observed reduced sensitivity before movement onset and during the first moments of movement. Also, visual enhancement of touch may occur when participants are not explicitly instructed to view the limb, rather they view the goal object. It may be that mere vision of the limb may induce visual enhancement of touch as observed in
the present study underlying the putative role of parietal sulcus to integrate motor and visual signals, process such signals and subsequently send signals to primary somatosensory cortex. The present study, along with multisensory literature, underlines the interactions between somatosensation, motor processes and vision.
5 Chapter: Altering Grasp Configuration does not Affect Tactile Gating (Experiment Four)

5.1 Thesis Context for Experiment Four

Experiment Four presents the first attempt to examine the purpose of tactile gating and to see whether the locus of tactile gating can be changed if the task constraints are modified (i.e., normal grasp configuration with index finger and thumb compared to grasping with the fifth digit and thumb). As the reader will note, changing grasp configuration has no effect on tactile sensitivity. Specifically, comparing the two grasp configurations did not reveal any effect. It could be that the manipulation was too subtle to affect tactile gating and perhaps a more drastic manipulation is required. Experiment Five compares the effect of grasping and pantomimed grasping on tactile sensitivity. That being said, Experiment Four was unfortunately inconclusive and offered no new insight but did replicate the base results observed in the previous experiments. This observation bolstered confidence in the present tactile gating effect in that it is certainly robust and replicable.

5.2 Background

Tactile gating is a diminished ability to detect events on the surface of the skin while performing a movement and in anticipation of a movement that subsequently does not occur. In the context of goal-directed movements, tactile gating was observed to be restricted to the moving limb (Buckingham et al., 2010; Colino et al., 2014; Williams & Chapman, 2002). Critically, previous studies observed tactile gating to manifest before movement onset (Buckingham et al., 2010; Chapman, 1994; Chapman & Beauchamp, 2006; Colino et al., 2014; Jiang et al., 1990; Shimazu et al., 1999) and in the absence of movement (Voss et al., 2006, 2008), in other movements (Juravle & Spence, 2011; Juravle, Deubel & Spence, 2011). However, other studies suggest that tactile gating arises from sensory reafference due to the
movement itself (Chapman et al., 1987; Milne et al., 1988; Duysens et al., 1995).

Historically, most studies that examined tactile gating did so using single-joint movements such as finger abduction or isometric muscle contractions. Such simple movements preclude the expectation of using tactile information. That is, when an actor performs a finger abduction task, gating was observed to be maximal at the moving joint and gating strength diminished as a function of the distance from the primary effector.

However, finger abduction and isometric contractions do not require the central nervous system to expect useful tactile information. That is, there is no expectation to interact with a goal object – behaviour that requires tactile information. Therefore, it is advantageous for the central nervous system to preemptively facilitate tactile information from effectors that will interact with a target object (see Colino et al., 2014). Colino et al. (2014) had participants perform perceptual judgments of tactile taps from tactors adhered to the index fingers, fifth digit and forearm of both upper limbs whilst reaching and grasping a goal object. The left limb remained stationary throughout the experimental protocol and each participant moved her/his right limb. Participants detected all tactile events that occurred on the left (stationary) limb, but were less able to detect tactile events at the right forearm. Interestingly, participants preserved their ability to detect vibrations only at the right index finger. The present study replicates the finding that tactile gating manifests at specific segments of the moving limb while leaving tactile inputs from the stationary limb unmolested. However, previous studies failed to manipulate how individuals grasp a target. Therefore, the present study attempts to manipulate task-related gating by having participants grasp a target with a finger-and-thumb grasp and a fifth-digit-and-thumb grasp. If tactile gating is sensitive to changes in the present task, no gating will be observed at the index
finger when grasping is performed with the index finger and thumb and, by extension, no gating will be observed at the fifth digit when participants grasp with the first and fifth digit but tactile inputs from the second digit will be gated.

5.3 Methods
5.3.1 Participants
Twelve participants (9 females; median age 22 years) were recruited from the local undergraduate and graduate student population to take part in the present experiment. All participants had normal to corrected-to-normal vision and reported no history of neurological impairments. The local ethics review board approved all procedures in the present experiment.

5.3.2 Task
Two experimental blocks were conducted in which a participant grasped the target object either with the first digit and the second digit (normal grasp) or with the first digit and the fifth digit (altered grasp). Block order was counterbalanced across participants. On each trial participants performed speeded reaching and grasping movements with the first and second digits to a two-centimeter target object cylinder concluding with a simple lift off the reaching surface. Once the grasping movement was complete, participants made a detection judgment whether a vibration was felt (i.e., yes/no) and where the vibration was felt (e.g., left mid forearm, proximal phalanx of the left index finger, proximal phalanx of the left fifth digit etc). A second condition was identical and given in a separate block of trial in which participants were asked to grasp the target object with the first and fifth digits.

In both conditions participants sat in front of the reaching surface, wearing LCD goggles to occlude vision of the target and surface between trial completion and initiation of the following trial. All trials began at a home position that was 20 cm to the right of the
midline of the reaching surface. There were two possible target locations, medial and lateral, that the experimenter changed randomly during the experiment. Both possible locations were 25 cm from the home location ahead of the participant. Before the goggles opened, a computer generated auditory tone (2000 Hz, 300 ms duration) warned participants that a trial was imminent and one second later the goggles opened. A trial began 1000 ms to 1500 ms later and the imperative cue was a sound emitted by a piezoelectric auditory buzzer (80 ms duration). Participants were instructed to reach and grasp the target as quickly and accurately as possible with particular emphasis on speed. Participants were required to initiate a movement within 400 ms after the buzzer and complete the reach and grasp movement in 800 ms or less. These instructions forced participants to fully concentrate on the reach and grasp task to successfully complete each trial.

The vibrations occurred during one of several epochs relative to the imperative cue from 0 ms (at the same time as the imperative cue) to 360 ms (after the imperative cue) in 60 ms bins and elicited a 1 mm deformation of the skin (i.e., 0, 60, 120, 180, 240, 300 and 360 ms). A pilot experiment conducted prior to this study found that this vibration pulse is readily detectable at rest. Once a trial was successfully completed, the LCD goggles closed and participants made a yes/no choice (Y/N) regarding the occurrence of a vibration at one of the six vibrator motor locations. If a vibration was detected, the participant verbally indicated where on the body the vibration was felt (e.g., index finger). There were ten trials per epoch per site (i.e., 10 trials with a delay of 0 ms, 10 trials with a delay of 60 ms, etc). In addition there were 10 catch trials per site in which no vibration was delivered, to assess participants’ false alarm rate. Each experimental session of 420 trials lasted between 100 – 120 minutes. Participants were given opportunities to rest throughout the procedure.
5.3.3 Data Analysis

All trial data were segmented into 60-ms time bins to achieve temporal accuracy because a reaction time of any given trial may widely differ. To capture the time at which the stimulus was delivered relative to movement onset we subtracted participant reaction time (for each trial) from the time relative to the imperative cue (e.g., 60 ms – 300 ms = -240 ms). Nine time bins were created such that they collectively spanned 359 ms before movement onset through 180 ms after movement onset. The time bins were organized as follows: -359 to -300 ms, -299 to -240 ms, -239 to -180 ms, -179 to -120 ms, -119 to -60 ms, -59 to 0 ms, 1 to 60 ms, 61 to 120 ms, and 121 to 180 ms. Participant trial data were organized into the stimulation epochs relative to the imperative cue (see above). However, too few cases were included into the last time bin (i.e., 121 to 180 ms) and, therefore, excluded from further analysis.

Sensitivity (d’) and criterion (C) were calculated for every condition within each participant (Gescheider, 1997). Sensitivity was calculated by subtracting the false alarm z-score (Zfa) from the hits z-score (Zh; see Gescheider, 1997, p. 119). Half the sum of Zh and Zfa resulted in C. Negative C values reflect bias toward frequent “yes” responses, whereas positive values of C reflect bias toward frequent “no” responses (Gesheider, 1997). C was chosen because the range of C does not depend on d’ (Gescheider, 1997). All detection and movement performance dependent variables were submitted to a 2 (grasp mode: normal grasp, augmented grasp) x 6 (vibration location: left and right second digits, left and right fifth digits, left and right mid-forearms) x 8 vibration epoch (-359 to -300 ms, -299 to -240 ms, -239 to -180 ms, -179 to -120 ms, -119 to -60 ms, -59 to 0 ms, 1 to 60 ms, and 61 to 120 ms) repeated measures analysis of variance (ANOVA_{RM}). In the event the main effect of
grasp mode was found statistically not significant, data were collapsed across grasp mode. All statistically significant effects and interactions were subjected to Bonferroni-adjusted paired samples t-tests for all possible pairwise comparisons. Statistical significance was set to \( p < .05 \) for \( \text{ANOVA}_{\text{RM}} \) tests and post-hoc t-test comparisons significance was set at \( p < .05 \). Error trials were removed from analysis and the trial was re-run later in the experimental session. An error consisted of dropped target, occluded IRED marker, reaction time longer than 400 ms, or movement time longer than 850 ms. The average error trials rate across all participants was 7%.

5.3.4 Movement Performance and Kinematics

Movement performance and kinematics were compared between vibration and no-vibration trials using Bonferroni-corrected t-tests. Also, vibration conditions were compared with each other to examine any potential differences between vibration locations with Bonferroni-corrected t-tests (\( \alpha = .05 \)). Due to technical difficulties associated with collecting grip aperture data from fifth digit-thumb grasp configuration, grip aperture analysis was excluded from further analysis. No effects were found between either grasping configuration. Therefore, each movement performance variable (i.e., reaction time, movement time, peak velocity, peak acceleration, time to peak velocity and time to peak acceleration) were submitted to a 2 (limb: right, left) x 3 (stimulus location: fifth digit, index finger, forearm) x 8 stimulus epoch (-359 to -300 ms, -299 to -240 ms, -239 to -180 ms, -179 to -120 ms, -119 to -60 ms, -59 to 0 ms, 1 to 60 ms, and 61 to 120 ms). Investigation of simple main effects was conducted in the case of significant interactions. Where appropriate, polynomial contrasts were examined for significant trends and post-hoc t-tests were conducted with Bonferroni-adjusted correction for multiple comparisons.
5.4 Results

5.4.1 Sensory Detection

Analysis of $d'$ revealed no statistically significant effects of grasp mode ($p = .55$). Therefore, $d'$ and $C$ data were collapsed across grasp mode and the data were analyzed with three terms, 2 limb (left and right) x 3 vibration location (second digit, fifth digit and forearm) x 8 vibration time (-359 to -300 ms, -299 to -240 ms, -239 to -180 ms, -179 to -120 ms, -119 to -60 ms, -59 to 0 ms, 1 to 60 ms, and 61 to 120 ms) in a repeated measures ANOVA. Results from $d'$ shall be presented first followed by $C$.

Analysis of $d'$ revealed statistically significant main effects of limb [$F(1,11) = 66.5, p < .001$], vibration location [$F(2,22) = 51.6, p < .001$], and vibration time [$F(6,66) = p < .001$]. Also, statistically significant two-way interactions were found between all three terms, limb x vibration location [$F(2,22) = 55.8, p < .001$], limb x vibration time [$F(6,66) = 14.4, p < .001$], and vibration location x vibration time [$F(12,132) = 8.7, p < .001$]. Importantly, analysis revealed a statistically significant three-way interaction that superseded all other main effects and interactions [$F(12,132) = 8.8, p < .001$]. Therefore, two separate two-way repeated measures ANOVAs were conducted to investigate the limb main effect, one two-way ANOVA for the right limb and another for the left limb. Analysis of the right limb revealed a statistically significant main effects of vibration location [$F(2,22) = 55, p < .001$] and vibration time [$F(6,66) = 17, p < .001$]. Also, a statistically significant two-way interaction between vibration location and vibration time at the right limb was revealed [$F(12,132) = 2.4, p < .001$]. However, analyzing vibration location and vibration time at the left limb revealed a different picture: the main effect of vibration location trended to significance [$F(2,22) = 3.3, p = .058$] and the main effect of vibration time was not statistically significant [$F(6,66) = 1.9, p = .14$]. The interaction between vibration location and vibration time at the
left limb was not statistically significant \( (p = .9) \). The above results suggest that the right limb drove the effects in the omnibus analysis (Figure 2.9). Post-hoc \( t \)-tests (Bonferroni adjustment for multiple comparisons) of vibration locations at the right limb revealed that the right limb was significantly less sensitive than either the right second digit \( (p < .001) \) or the fifth digit \( (p < .001) \) and the right fifth digit was less sensitive than the right second digit \( (p = .002) \).

To examine the changes in \( d' \) over across time at each vibration location at the right limb three separate one-way repeated measures ANOVAs were performed. Results from the right forearm are presented first, followed by results from the right fifth digit and results from the right second digit (see Figure 5.1). Results from the one-way ANOVAs revealed a main effect of vibration time at the right forearm \( [F(6, 66) = 19.4, p < .001] \) and fifth digit \( [F(6,66) = 5.6, p < .001] \). Post-hoc \( t \)-tests revealed that \( d' \) at the right forearm decreased from the first vibration time to the second vibration time \( (p = .003) \) and \( d' \) remained decreased at all instances the right forearm was vibrated. At the right fifth digit, \( d' \) decreased from the first vibration time to the second vibration time \( (p = .011) \) but \( d' \) increased again by the fourth vibration time that was no longer significantly different from the first vibration time \( (p = .54) \). Interestingly, vibration time at the right second digit did not achieve statistical significance \( [F(6,66) = 2.8, p = .1] \). Post-hoc \( t \)-tests (Bonferroni adjustment for multiple comparisons) revealed that \( d' \) did not change as a result of vibration before or after movement.
Figure 5.1 Sensitivity ($d'$) as a function of time of stimulus relative to reaction time (RT).

The top panel depicts sensitivity ($d'$), calculated from hits and false alarms, observed at the left upper limb and the bottom row depicts the same from the upper right limb. Sensitivity graphs have the same shape and pattern as observed detection rate. For the right arm, $d'$ was reduced considerably over the second and third stimulation times and remained diminished; was transiently decreased then returned to baseline at the fifth digit; and remained unchanged at the second digit. For the left arm, $d'$ remained constant at all stimulation sites and time. Error bars denote error of the mean. *$p < .05$. **$p < .01$. ***$p < .001$.

Analysis of criterion ($C$) revealed statistically significant main effects of limb [$F(1,11) = 62.1, p < .001$], vibration location [$F(2,22) = 52.5, p < .001$], and vibration time [$F(6,66) = 17.1, p < .001$]. Additionally, statistically significant two-way interactions were found between limb and vibration location [$F(2,22) = 55.6, p < .001$], limb and vibration...
time \( F(6,66) = 14.9, p < .001 \), and vibration location and vibration time \( F(12,132) = 9.9, p < .001 \). Importantly, a statistically significant three-way interaction between all factors was found \( F(12,132) = 10.3, p < .001 \). Post-hoc \( t \)-tests revealed the right limb was different from the left limb \( (p < .001) \). Therefore, two separate two-way ANOVAs with vibration location and vibration time as factors were conducted to break down the statistically significant effect of limb. Analysis of the right limb found statistically significant main effects of vibration location \( F(2,22) = 54.6, p < .001 \) and vibration time \( F(6,66) = 16.8, p < .001 \). Importantly, analysis revealed a significant two-way interaction between vibration location and vibration time \( F(12,132) = 10.9, p < .001 \). Post-hoc \( t \)-tests with Bonferroni adjustment revealed statistically significant differences between the right second digit, right fifth digit and the right forearm. Namely, \( C \) at the right forearm was significantly different from the right fifth digit \( (p < .001) \) and the right second digit \( (p < .001) \). Also, \( C \) at the right second digit and the right fifth digit was significantly different \( (p = .005) \). In contrast, at the left limb, the main effect of vibration location saw a trend to statistical significance \( (p = .067) \), while vibration time did not achieve statistical significance \( (p = .14) \). See Figure 2.10 for response bias observation.

Three separate one-way repeated measures ANOVA were conducted to break down the significant vibration location \( \times \) vibration time interaction of the right second digit, right fifth digit and the right forearm at the right limb and to see if any changes in \( C \) occurred across time. Results from the right limb will be presented first, followed by the right fifth digit and the right second digit. One-way repeated measures ANOVA of the right forearm revealed a statistically significant effect of vibration time \( F(6,66) = 18.9, p < .001 \). Post-hoc comparisons show a decrease of 0.7 units from the first vibration time to the second time.
that was statistically significant \((p = .003)\) and remained decreased throughout all vibration times (i.e., there was no statistically significant difference between all other vibration times, \(ps > .1\)).

One-way repeated measures ANOVA of the right fifth digit revealed a statistically significant effect of vibration time \([F(6,66) = 5.242, p < .001]\). Post-hoc comparisons show a decrease of 0.46 units from the first vibration time to the second vibration time that was statistically significant \((p = .01)\) and remained decreased throughout all subsequent vibration times (i.e., there was no statistically significant difference between all other vibration times, \(ps > .7)\). Finally, there was no effect of vibration time at the right second digit.

### 5.4.2 Movement Performance

Analysis of reaction time revealed a main effect of stimulus epoch \((F_{7,77} = 73.99, p < .0001)\) with a statistically significant cubic relationship trending to slower reaction time when vibration was delivered at the earliest vibration epoch (i.e., \(-359\) to \(-300\) ms) and fastest reaction time at the latest vibration epoch (i.e., \(61\) to \(120\) ms). Analyses of peak velocity and peak acceleration did not achieve statistical significance. Time to peak velocity analysis revealed a main effect of limb \((F_{1,11} = 8.27, p = .015)\) with participants achieving peak velocity 3 ms faster when the left limb was stimulated with vibration relative to stimulating the right (moved) limb. Peak acceleration analysis similarly revealed a main effect of limb \((F_{1,11} = 5.404, p = .04)\) with participants achieving peak acceleration 1 ms faster when stimulating the left limb relative to the right limb.
Figure 5.2 Movement time decreases linearly between stimulation epochs.
The leftmost graph depicts movement time as a function of stimulation epoch from the right index finger. The middle and rightmost graphs depict the same from the right fifth digit and right forearm, respectively. Post-hoc $t$-tests using Bonferroni correction for multiple comparisons reveal a statistically significant difference between the first epoch and last epoch ($p = .009$) at the right index finger.

Analysis of movement time revealed a significant main effect of stimulus epoch ($F_{7,77} = 3.093, p = .036$). This effect was not observed in prior studies and perhaps may be an artifact. Additionally, analysis of movement time revealed a significant three way interaction ($F_{14,154} = 2.712, p = .001$). Simple main effects analysis revealed the interaction was driven by a significant negative linear trend ($F_{1,11} = 32.709, p < .0001$) with slower movement times at the earliest stimulus epoch and fastest movement times at the latest stimulus epoch (Figure 5.2). Post-hoc pairwise comparisons revealed a statistically significant difference between the first stimulus time and the last stimulus time (-359 to -300 ms v. 61 to 120 ms: $p = .009$), a 73 ms difference. This movement time effect may reflect sensory afferent processes affecting motor planning processes such that initial impulses become affected when the imperative cue, tactile probe temporally coincide. All other simple main effects did not achieve statistical significance.
5.5 Discussion

5.5.1 Movement performance

Before discussion of the detection data, aspects of movement performance must be addressed. When the right limb received tactile stimulation at the first stimulus epoch (-359 to -300 ms) reaction time was slowest. This particular observation agrees with a previous report (Buckingham et al., 2014) where reaction time was longer at the earlier stimulus epoch and faster at later epochs. When tactile stimuli were presented closer to movement onset, reaction times were 59 ms faster on average than earliest stimulus epoch. Shortened reaction time is perhaps due to multi-modal excitation when seeing the target and feeling the vibration (Nickerson, 1974). However, one important methodological difference between the present study and that of Buckingham and colleagues (2014) is that the visual target onset was the imperative cue whereas in the present study the visual target was visible to each participant well before movement onset and the imperative cue was an auditory buzzer. Therefore, it cannot be confirmed that movement onsets stabilized to pre-movement levels (e.g., Buckingham et al., 2010). The present study observed significant effects of limb on time to peak velocity (TPV) and time to peak acceleration (TPA). Stimulating the right limb led to slightly longer (1.4 ms longer) TPV compared with left limb stimulation. Likewise, stimulating the right arm resulted in slightly longer (a 0.84 ms difference) TPA compared with left limb stimulation. These two effects are likely related to multi-sensory acceleration effect as observed with reaction time (Nickerson, 1974).

The significant interaction of stimulus presentation on movement time is novel. Simple main effects analysis revealed that stimulation at the right index finger movement time led to a linear decrease in movement time across tactile stimulation epochs. This is perhaps related to the multi-modal excitation of receiving tactile stimulation and an auditory
stimulus achieving fastest movement time after movement onset. Interestingly, movement time does not change across stimulation epochs when the left limb receives tactile stimulation nor does movement time change when the right fifth digit or the right forearm is stimulated by vibration. These observations suggest that tactile information not immediately processed by the somatosensory cortex has no effect on movement planning or execution. Movement time decreases only when the right index finger receives tactile stimulus near, coincident and soon after movement onset. This observation suggests an interaction between motor planning processes and somatosensory perception (Jackson, Parkinson, Pears & Nam, 2011). This observation may reflect sensory afferent processes affecting motor planning processes when the imperative cue and tactile probe temporally coincide. However, since this effect was not observed in prior studies it is very likely not a real effect.

5.5.2 Sensory Detection

It is now well established that tactile gating occurs before and during movements (e.g., Buckingham et al., 2010; Chapman et al., 1987; Milne et al., 1988; Williams et al., 1998) and is central in origin (Bays et al., 2006; Voss et al., 2008). However, there is debate regarding the processes underlying tactile gating, particularly with respect to how it manifests. There are two arguments: one side argues that somatosenation should be enhanced if an effector is in close proximity to the target, as predicted by premotor theory of attention (Rizzolatti, Rigio, Dascola & Umilta, 1987). This theory is supported by observations of enhanced tactile perception in the presence of a target (Huttunen, Wikström, Korvenoja, Seppäläinen, Aronen & Ilmoniemi, 1996). Conversely, feedforward models affirm that self-produced sensory events are “canceled out” because those self-produced signals do not convey any novel information and are, therefore, attenuated to prevent unnecessary processing of irrelevant sensory information and free neural resources to process
externally-generated information. Cancellation of self-produced tickle sensation is the prototypical observation of the action of internal models to attenuate internally-generated sensory events (Blakemore, Wolpert & Frith, 1998). Indeed, extending the ideas from observations of efference copy in oculomotor networks (Bridgeman, 1995) and electrosensory perception of fish (Bell, 2001), we can begin to understand the interactions between motor commands and afferents at play in the context of tactile gating.

The present study aimed to manipulate task demands by instructing participants to reach and grasp objects with the index finger and the thumb or the pinky finger and thumb. This manipulation was performed with the expectation that sensory attenuation will be modulated based on proximity effector used to the target object. In other words, if tactile gating is sensitive to grasp modes (i.e., grasp with index finger and thumb or pinky finger and thumb), then the effector level locus of tactile gating should shift to the specific digit that does not contact the target object.

However, the present manipulation did not elicit any changes in the behavioural manifestation of tactile gating. Switching from one grasp mode to the other did not elicit a change in the locus of tactile gating. It may be that the randomized design did not allow internal models time to adapt to new movement pattern and account for that change across time. Therefore, a blocked design might manifest a change in gating locus depending on the grasp mode. It has been observed, using positron emission tomography, that attention directed to an expected vibratory stimulus can cause increase in regional cerebral blood flow (rCBF) (Meyer, Ferguson, Zatorre, Alivisatos, Marret, Evans & Hakim, 1991). Importantly, other laboratories observed that rCBF decreased at primary somatosensory cortex contralateral to stimuli that were not expected (Drevets, Burton, Videen, Snyder, Simpson &
Raichle, 1995). These previous data suggest that attention is the primary mechanism by which sensory information is facilitated or attenuated arguing for the convergence of multiple sensory systems (see Rizzolatti et al., 1987). However, the present data do not support this view given the fact that no shift in tactile gating was observed. Namely, tactile gating was not observed at the stationary limb; participants were able to detect tactile stimuli at the stationary limb nearly all the time. Attention models would predict tactile gating to occur at the stationary limb as the majority of processing resources shift to the moving limb but this did not occur. Lack of gating at the stationary limb poses a large problem for theories based on the action of attention and, indeed, an alternative must be sought. Therefore, it is unlikely that attention played a primary role in the present study. Other possibilities exist to explain the pattern of results at present.

Despite no effect of grasp mode, we replicated previous findings in our laboratory (Colino et al., 2014) demonstrating that tactile gating occurred at the forearm of the moving limb, to a lesser degree at the pinky finger and not at all at the index finger (i.e., the limb segment that contacted the target object). We suggest that predictive mechanisms (i.e., internal models) are the primary sources of tactile gating (also see Buckingham et al., 2010; Colino et al.; Voss et al., 2006, 2008). Predictive mechanisms are clearly at work as $d'$ and $C$ changed as a function of time and location on the moving limb. Participants became less sensitive to tactile stimuli at the forearm of the moving limb. Also, $d'$ decreased (2.8 $d'$ units) before movement onset, consistent with our previous report. Also, Criterion ($C$) decreased at the forearm of the moving limb. As a reminder, negative values of $C$ reflect a responder bias toward “yes” responses. In the present experiment, participants’ criteria shifted to “yes” responses. Despite the shift in $C$ that might make detection more likely,
participants still could not detect tactile stimulation at the moving forearm. These pair of observations may indicate that any stimulation that was not gated would have been easily detected. Or, interpreted differently, the shift in $C$ reflects the fact that tactile stimulation at the forearm is noisy and, therefore, detecting stimuli above the background noise became difficult. A criterion shift to “yes” responses is consistent with this interpretation.

There are alternative explanations for the current observations that must be considered. Overall, the present results do not support postdictive explanations for tactile gating; postdictive mechanisms stem from the observations of passive limb movement causing gating (Williams & Chapman, 2002). In the Williams and Chapman (2002) study, a predictive mechanism is unlikely to account for the gating effect since tactile gating occurred in the absence of active movement. In other words, there could not be a central motor command in this context and, by extension, no predictive sensorimotor planning signal. In the case of gating without a motor command, a “postdictive” explanation would suggest gating occurs as a result of sensory inflow in the presence of other sensory events. Present accounts of the pain gating mechanism agree with the postdictive explanation, the best known example being the inhibitory inputs from large (Aβ) fibres to the dorsal horn of the spinal cord (Melzak & Wall, 1965).

However, the present study replicates previous findings in our laboratory that gating onset occurs before movement onset. This means that there would not be any afferent information from the movement that needs to be attenuated at that time. It could be the somatosensory system is gating tactile information that is predicted to be noisy in order to optimize processing of sensory input from the index finger and thumb (i.e., the same effectors closest to the target object). But, despite replicating the previous results, our
present hypothesis of shifting of gating locus from the pinky finger to the index finger, and vice-versa, was not supported. This null effect may be due to limits in the experimental design. As mentioned previously, the randomized trial design employed in the present study did not allow enough time for the grasp mode effect to materialize. Rather, a blocked design might allow the grasp mode effect to manifest. Subsequent studies will test this hypothesis.

It may be that neural resources become dedicated to processing externally generated events and ignore internally generated ones such as movement related sensory noise (Blakemore, Wolpert & Frith, 1998; Frith, Blakemore & Wolpert, 2000; Voss et al., 2006). Furthermore, the present data is consistent with previous reports that movement planning attenuates tactile perception and that somatosensory areas undergo reduced blood oxygen level dependent signal when a limb moves and not when it remains stationary (Parkinson, Plukaard, Pears, Newport, Dijkerman & Jackson, 2011). What causes this decrease? Sensory-evoked potential (SEP) data may provide an insight. Yamaguchi & Knight (1990) observed enhanced SEP in patients with PFC damage compared to controls. It may be that pre-frontal areas, where motor planning areas are located, provide the signal to attenuate somatosensory information. However, the mechanism of this observation remains unclear. Chapman (1994) argues that tactile gating is centrally-generated for two reasons. First, gating often occurs before muscle activity onset and, second, peripheral reafference cannot influence afferent processes without overt movement. The present experiment cannot confirm this as electromyography was not recorded.

On the other hand, it is possible for sensory reafferent processes to predominate tactile gating after movement onset. For example, Brooke (2004) observed reduced H-reflex amplitude while passively moving the lower limb in humans. Likewise SEP amplitude is
reduced during passive upper limb movements (Jones, 1981; Rushton, Rothwell & Craggs, 1981). These peripheral sources of tactile gating may reflect synaptic inhibition at the level of spinal cord like that proposed for pain perception (Melzack & Wall, 1965). Despite these findings, central mechanisms also play a critical role modulating afferents (Shimazu, Kaji, Murase, Kohara, Ikeda, Shibasaki, Kimura, & Rothwell, 1999; Ogata, Okamoto, Yamasaki, Shigeto & Tobimatsu, 2009) based on task relevance (Staines, Brooke, Angerilli & McIlroy, 1998; Staines, Brooke & McIlroy, 2000). SEPs provide clues regarding attenuation mechanisms. For example, Shimazu & colleagues (1999) observed reduced frontal N30, parietal P30 and central N60 components when participants performed finger and wrist extension without any changes to subcortical, primary sensory cortex and frontal motor potentials hinting that muscle afferents do not affect SEP gating. Also, self-generated movements elicit progressively reduced sensory potentials 500 ms before movement onset closely corresponding to the time course of movement-related potentials such as the Bereitschaftspotential (Ogata et al., 2009) generated in the supplementary motor area (Neshige, Luders & Shibasaki, 1988). Indeed, motor intention does modulate sensory processes (Parkinson, Plukaard, Pears, Newport, Dijkerman & Jackson, 2011). Peripheral and central mechanisms play roles modulating tactile sensitivity.

5.6 Summary

The present data replicates previous findings from our laboratory. In particular, we have confirmed gating occurs at the forearm of the moving limb before movement onset, indicating that predictive mechanisms, independent of attention, play a primary role. However, we could not observe any changes in the locus of gating within the moving hand and we attribute this to experimental design that could be corrected by using a blocked design. Despite the limitation of the current experiment, the fact that gating was observed
before movement onset is consistent with this phenomenon being a centrally-driven mechanism. Furthermore, tactile gating was not observed to be a global effect across both limbs. Rather, it appears to be specific to the to-be moved effector and specific to segments of the moved effector.
6  Chapter: Tactile Gating Optimizes Sensory Inflow (Experiment Five)

6.1  Thesis Context for Experiment Five

Experiment Five attempts to answer what role does tactile gating play? What purpose does tactile gating serve; does it act to remove sensory noise or optimize sensory input or contribute to both ends? Performing pantomimed grasping results in reduced tactile sensitivity at the index finger compared with tactile sensitivity at the index finger while grasping. If tactile gating acts to optimize sensory input then tactile sensitivity will diminish at the index finger in pantomimed grasping but remain unaffected when grasping. Experiment Five found that tactile sensitivity is mildly reduced at the index finger in a pantomimed grasp and remains intact when grasping. However, Experiment Four replicated reduced tactile sensitivity at the forearm irrespective of task goal. It appears that tactile gating optimizes sensory input because tactile sensitivity decreased at the index finger only when participants performed pantomiming grasping. This finding indicates that gating is sensitive to task goal, thereby adjusting sensory input. However, tactile gating at the forearm was replicated from previous experiments. Therefore, tactile gating also appears to attenuate sensory noise. Whether these effects are mutually exclusive or attributable to a single mechanism is unclear.

6.2  Background

Tactile signals from the fingertips are crucial for successful grasping behaviour. Indeed, the various receptor types are sensitive to different types of tactile stimuli and encode different phases of object contact to grip (Johansson, 1996). Grip force control is a highly controlled behaviour that relies heavily on sensory feedback and feedforward control. Grip control involves several stages in which different tactile afferents signal unique events as the fingers make initial contact through grip establishment. In the context of reach and grasp
movements, the time course of grip control precludes the possibility of cortical involvement, as grip force control occurs too quickly (e.g., 70 ms observed by Johansson & Westling, 1987) for long-loop circuits. However, tactile gating has high cortical involvement as evidenced by a robust predictive effect before movement onset (Buckingham et al., 2010; Chapman et al., 1987; Colino et al., 2014; Juravle et al., 2011; Milne et al., 1988; Rushton et al., 1981; Voss et al., 2008; Williams et al., 1998).

Tactile gating, therefore, appears to gate particular inputs from the skin when that information is not immediately relevant to a task. That is, tactile gating reduces tactile stimulus intensity so that the individual is not perceptually aware of stimulus presentation. Additionally, this phenomenon occurs before movement onset (e.g., Buckingham et al., 2010; Colino et al., 2014; Voss et al., 2008). However, this does not discount the possibility that a tactile stimulus could be detected despite tactile gating if that stimulus is of high amplitude. Alternatively, as observed previously (Colino et al., 2014), tactile stimuli can be readily detected depending on where and when they are presented in an effector- and movement-dependent fashion. As in the case of grasping, tactile inputs from the glabrous skin of the digits is essential for fine force adjustment while removing sensory information that does not contribute to the task goal (i.e., skin inputs from the forearm). Removal of unimportant sensory data leaves more resources to process sensory information for the sake of adequate grip control. Previous work shows that just before movement onset tactile stimuli become difficult to detect. This effect has been observed in various contexts such as pointing (Buckingham et al., 2010), juggling (Juravle & Spence, 2011), grasping (Juravle et al., 2011), and during normal gait (Duysens et al., 1995; Morita et al., 1998; Staines et al., 1998). Indeed, data from previous work indicate that tactile gating occurs as a result of the
motor plan. In other words, a predictive mechanism appears to have the motor plan as input and adjusts tactile afferent inputs reducing the amplitude of possibly non-useful information. It has been suggested that this occurs as a result of an efference copy of the motor plan being forwarded to somatosensory areas of the cortex (Bays et al., 2006). This efference copy, therefore, elicits the tactile gating effect (Chapman & Beauchamp, 2006; Voss et al., 2008). Indeed, previous workers observed tactile gating occurred before movement onset (Buckingham et al., 2010; Colino et al., 2014) and in the absence of movement (Voss et al., 2006).

In the present study, participants were asked to perform a reach-to-grasp task in which they grasped the cylindrical target or were asked to pantomime a reach-to-grasp movement. The present manipulation sought to assess whether changing task instruction affects motor planning such that it changes the prediction output. Based on results from previous work (Colino et al., 2014) it was expected that grasping the target would produce tactile gating at the right forearm while preserving tactile sensitivity at the right index finger. That observation suggests that tactile gating acts to remove task-irrelevant sensory noise in response to the presence of a motor plan. Oppositely, pantomiming grasping should produce a different tactile gating pattern: gating at the index finger along with the forearm since no tactile information is expected given the motor plan (i.e., no target contact with the index finger). Therefore, measuring tactile sensitivity at right index finger, fifth finger and forearm should elucidate the potential effect of task instruction on tactile gating. Specifically, tactile inputs delivered on the right index finger should be less readily perceived when participants are instructed to perform pantomime grasping as opposed to true grasping. Globally, the
The purpose of the present paper is to determine whether tactile gating serves to reduce sensory noise or optimize sensory inflow.

6.3 Methods

6.3.1 Participants

Ten participants (5 females) were recruited from the local graduate and undergraduate population (median age = 22 years; SD = 2.6 years). Due to technical problems, data from one participant was excluded from further analysis. They were all self-reported right-handed individuals, had normal or corrected-to-normal visual acuity, and reported no previous neurological conditions. Participants gave written informed consent, and the local research ethics board approved experimental procedures.

6.3.2 Apparatus

An Optotrak Certus (Northern Digital, Inc., Waterloo, ON) tracked at 250Hz the three-dimensional position of three infrared-emitting diodes (IREDs) affixed to the index finger, thumb and wrist of each participant right hand. Four custom-built tactile micromotor vibrators (tactors) were taped to the dorsal surface of the proximal phalanx of the left and right index fingers and the dorsal surface of the mid-forearm of both arms. Micromotor vibration stimuli consisted of a single 6.5 ms long vibration burst causing 1-mm deformation of the skin resulting in a readily detectable tap at rest (17 mm long, 7 mm diameter, weighs 1 g). Participants were seated in an upright padded chair with the left arm resting on a flat grasping surface that was at the level of the upper abdomen. The right arm always began at the home position that was 35 cm to the right of each participant’s midline. The elbow was flexed at 90°.

6.3.3 Task
On each trial, participants performed speeded reaching and grasping movements to a target object cylinder concluding with a simple lift off the reaching surface. Once the grasping movement was complete, participants made a detection judgment whether a vibration was felt (i.e., yes/no) and where the vibration was felt (e.g., left mid forearm, proximal phalanx of the left index finger, proximal phalanx of the left fifth digit etc).

Pantomime grasping was performed in a separate experimental session and the protocol was identical to the grasping session except that the participant was instructed to pantomime reaching and grasping. Specifically, the participants were asked to reach out to the target and grasp at the target if it was in reach.

Data collection took place inside a small sound-isolated room. Participants sat in front of the horizontal reaching surface, wearing liquid crystal display goggles to occlude vision during the period between trials. All trials began with the right hand 30 cm to the right of the midline and 15 cm in front of the torso and the left hand in the mirror symmetric location. A computer generated tone (2000 Hz, 300 ms duration) warned participants that a trial was imminent and 1 second later the goggles opened. After a subsequent variable foreperiod (1000-1500 ms) the imperative cue consisting of a piezoelectric auditory buzzer (50 ms duration) was presented and participants reached out and grasped the 2 cm diameter and 5 cm high polyvinyl chloride (PVC) cylinder with the index finger and thumb of the right hand. They were required to initiate the movement within 400 ms after the buzzer and complete it in 800 ms or less. Movement initiation was defined as sustained velocity of 50 mm/s for 50 ms. The cylinder was located at one of two possible target locations that the experimenter changed randomly during the inter-trial period. The locations were 5 cm to the
left or right of a position 25 cm directly anterior to the home location for the right hand. This spatial uncertainty prevents participants from predicting target location.

The micromotor vibrations occurred during one of several epochs relative to the imperative cue from 0 ms (at the same time as the imperative cue) to 300 ms (after the imperative cue) in 50 ms bins (i.e., 0, 50, 100...300 ms). Once a trial was successfully completed, the LCD goggles closed and participants made a yes/no choice (Y/N) regarding the occurrence of a vibration. In addition, if a vibration was detected, the participant verbally indicated where on the body the vibration was felt (e.g., “left index finger”). There were eight trials per epoch per vibrator motor (i.e., 8 trials with a delay of 0 ms, 8 trials with a delay of 100 ms, etc). In addition there were 40 catch trials throughout the experiment in which no vibration was delivered to assess participants’ false alarm rate. Each experimental session was, thus, comprised of 420 trials and lasted between 100 – 120 minutes. Participants were given opportunities to rest throughout the procedure. Participants began with grasping or pantomime grasping and alternated across blocks. Block order was counterbalanced between participants to control for order effects.

6.3.4 Data Analysis

All trial data were segmented into 50-ms time bins to achieve temporal accuracy because a reaction time of any given trial may widely differ. To capture the time at which the stimulus was delivered relative to movement onset we subtracted participant reaction time (for each trial) from the time relative to the imperative cue (e.g., 50 ms – 300 ms = -250 ms). Eleven time bins were defined such that they collectively spanned 399 ms before movement onset through 250 ms after movement onset. The time bins were organized as follows: -449 to 400 ms, -399 to -350 ms, -349 to -300 ms, -299 to -250 ms, -249 to -200 ms, -199 to -150 ms, -
Participant trial data were organized into the stimulation epochs relative to the imperative cue (see above). However, too few cases were included into the first two and last time bins (i.e., -449 to -400 ms, -399 to -350 ms, and 51 to 100, ms respectively) and, therefore, excluded from further analysis.

Sensitivity ($d'$) and criterion ($C$) were calculated for every condition within each participant (Gescheider, 1997). Sensitivity was calculated by subtracting the false alarm $z$-score ($Z_{fa}$) from the hits $z$-score ($Z_h$, see Gescheider, 1997, p. 119). False alarm rates were pooled together across all conditions and were used to calculate $d'$. Half the sum of $Z_h$ and $Z_{fa}$ resulted in $C$. Negative $C$ values reflect bias toward frequent “yes” responses, whereas positive values of $C$ reflect bias toward frequent “no” responses (Gesheider, 1997). $C$ was chosen because the range of $C$ does not depend on $d'$ (Gescheider, 1997).

In addition to the detection variables, several different movement performance variables were also monitored. These included reaction time, movement time, peak velocity, peak acceleration, peak grip aperture and time to peak grip aperture. All detection and movement performance variables were submitted to a 2 (task: grasp or pantomime) x 4 (vibration location: left and right index finger, left and right forearm) x 8 vibration epoch (-349 to -300 ms, -299 to -250 ms, -249 to -200 ms, -199 to -150 ms, -149 to -100 ms, -99 to -50ms, -49 to 0 ms, 1 to 50 ms) repeated measures analysis of variance (ANOVA$_{RM}$). All statistically significant effects and interactions were subjected to Tukey’s least significant difference pairwise comparisons. Statistical significance was set to $p < .05$. Gating was defined as a significant reduction in detection and $d'$ relative to the first time bin. Error trials were removed from analysis and the trial was re-run later in the experimental session. An
error consisted of dropped target, occluded IRED marker, reaction time longer than 400 ms, or movement time longer than 850 ms. The average error trials rate across all participants was 11%.

6.4 Results
6.4.1 Sensory Detection

Omnibus repeated measures ANOVA analyzing $d'$ revealed statistically significant main effects of Stimulus location ($F_{3,24} = 15.774, p = .004$) and Stimulus time ($F_{7,56} = 8.728, p = .002$). Main effect of Task did not achieve statistical significance ($F_{1,8} = 2.843, p = .13$). Additionally, a statistically significant two-way interaction between Stimulus location and Stimulus time ($F_{21,168} = 1.641, p = .001$) was observed but there was no significant interactions between Task and Stimulus location ($F_{3,24} = 1.606, p = .21$) or between Task and Stimulus time ($F_{7,56} = 1.204, p = .31$). Importantly, a statistically significant three-way interaction between Task, Stimulus location and Stimulus time was revealed ($F_{21,168} = 1.641, p = .045$). Subsequent simple main effects analysis was performed on the statistically significant three-way interaction between Task, Stimulus location and Stimulus time. Each stimulus location was analyzed separately determining whether sensitivity changed across stimulation times and task and determine if task influenced sensitivity.

Simple main effect analyses of the left forearm while grasping revealed a main effect of stimulus time ($F_{7,56} = 2.281, p = .041$). However, pairwise comparisons (Tukey’s LSD correction for multiple comparisons) did not reveal any statistically significant differences between stimulation times for sensitivity ($d'$). In contrast, there was no statistically significant effect found at the left arm while pantomiming ($p = 1.0$). Likewise, no effects were found at the left index finger under grasping ($p = 1.0$) or pantomime ($p = .33$).
However, sensitivity decreased across stimulation times at the right forearm (see Figure 6.1). Under grasping conditions, a statistically significant main effect was found \((F_{7,56} = 5.352, p = .007)\).

**Figure 6.1 Sensitivity \((d')\) as a function of vibratory stimulus time relative to reaction time (RT).**  
Leftmost graphs depict \(d'\) at the forearms (top, right forearm; bottom, left forearm) and rightmost graphs depict \(d'\) at the index fingers (top, right index finger; bottom, left index finger). At the right forearm, \(d'\) decreased approaching movement start. At the right index finger, there was a momentary \(d'\) decrease when participants pantomimed grasping. Likewise there was a greater decrease in sensitivity observed at the right forearm when participants pantomimed. There were no observed changes at the left arm or the left index finger.
Pairwise comparisons revealed statistically significant sensitivity decrease by 1.1 $d'$ units at the seventh time bin (i.e., 1 to 50 ms) and decrease another 1.13 $d'$ units at the eighth time bin (i.e., 51 to 100 ms). The fourth time bin (-299 to -250 ms) approached statistical significance ($p = .057$), as did the fifth time bin ($p = .06$) and sixth time bin ($p = .079$). The present results at the right forearm replicate those from a previous study conducted in our laboratory (Colino et al., 2014). Additionally, a statistically significant main effect at the right forearm was found when pantomime grasping ($F_{7,56} = 8.454, p < .0001$). Pairwise comparisons revealed statistically significant sensitivity decrease of 0.346 $d'$ units at the fourth time bin (-299 to -250 ms) and continuing to steadily decrease (all $p < .05$).

Furthermore, there was no main effect of stimulation time at the right index finger when participants grasped the object ($F_{7,56} = 1.0, p = .44$). Interestingly, there was a main effect of stimulation time at the right index finger when participants performed pantomime grasping ($F_{7,56} = 2.27, p = .041$), suggesting tactile inputs are gated if they do not carry task-relevant sensory information. However, subsequent pairwise comparisons revealed a trend toward statistical significance ($p = .092$) at the fourth time interval. This may indicate that the present study is underpowered with only nine participants.

Additional post-hoc paired samples t-tests were conducted to assess any differences between grasping and pantomiming within each time bin. There were no statistically significant differences between grasping and pantomiming at the left arm or the left index finger (all $p > 0.1$). However, differences approaching statistical significance were found at the -49 to 0 ms time bin at the right arm ($p = 0.06$) and at the -199 to -150 time bin at the right index finger ($p = 0.09$).
Figure 6.2 Response bias (C) as a function of time of stimulus relative to reaction time (RT) indicating shift in response criterion.

Leftmost graphs depict C at the forearms (top, right forearm; bottom, left forearm) and rightmost graphs depict C at the index fingers (top, right index finger; bottom, left index finger). At the right forearm, C increased approaching movement start. At the right index finger, there was a momentary C increase when participants pantomimed grasping that approached statistical significance (p = .057). Likewise there was a greater increase in C observed at the right forearm when participants pantomimed (p = .043). There were no observed changes at the left arm or the left index finger.

6.4.1.1 Response Bias

Omnibus repeated measure ANOVA of response bias (C) revealed stimulus location ($F_{3,24} = 13.90, p = .006$, partial $\eta = .63$) and stimulus time ($F_{7,56} = 7.63, p = .003$, partial...
There was no main effect of pantomime grasping ($p = .21$). There was a statistically significant two-way interaction between stimulus location and stimulus time ($F_{21,168} = 8.99, p = .001$, partial $\eta^2 = .53$). Also, there was statistically significant three-way interaction between all terms ($F_{21,168} = 1.63, p = .048$, partial $\eta^2 = .17$). Subsequent simple main effects analysis found statistically significant two-way interactions between stimulus location and stimulus time for grasping ($F_{21,168} = 4.97, p = .011$, partial $\eta^2 = .38$) and pantomiming ($F_{21,168} = 6.75, p = .003$, partial $\eta^2 = .49$). Response bias increased at the right forearm when grasping ($F_{7,56} = 5.16, p = .0001$, partial $\eta^2 = .39$) and pantomiming ($F_{7,56} = 6.78, p = .0001$, partial $\eta^2 = .46$). Response bias momentarily increased 0.193 $z$-score units ($F_{7,56} = 2.52, p = .025$, partial $\eta^2 = .24$) at the fourth time bin (-199 to -150 ms) indicating participants expressed bias toward negative responses. Conversely, there was no effect at the right index finger when participants grasped the target ($F_{7,56} = 1.00, p = .44$, partial $\eta^2 = .11$). This increase indicates that the response criterion shifts so that more sensory events are judged to have not occurred (note that positive criterion values reflect a stringent criterion and negative criterion values reflect a liberal criterion; see Figure 6.2). Subsequent paired samples t-tests were conducted comparing grasping and pantomime trials found statistically significant difference at the right forearm ($t_7 = 2.46, p = .043$) and a trend toward statistical significance at the right index finger ($t_7 = 1.81, p = .057$). There were no response bias effects at any other stimulus location ($p > .10$).

There were no statistically significant effects found in the movement performance variables.
6.5 Discussion

We have demonstrated that tactile gating indeed occurs before movement (Buckingham et al., 2010; Chapman et al., 1987; Colino et al., 2014; Milne et al., 1988; Williams et al., 1998). Additionally, there is some evidence to suggest that tactile gating may be sensitive to task instruction, although the present results are challenged by a small sample size.

The present study aimed to differentiate two probable goals of tactile gating, either to remove excess sensory noise from inputs or optimize sensory inflow. In a previous experiment (i.e., Colino et al., 2014), tactile sensitivity decreased during reach and grasp movements to a target made with the right limb. This reduction was observed to be specific to the right forearm and occurred before movement onset while leaving sensitivity at other locations unchanged. Clearly, reaching and grasping influences tactile gating behavior such that specific segments of a moving limb gate tactile inputs while others, namely the index finger and the stationary limb, do not. These observations likely reflect differential processing of tactile inputs from different areas of the skin. It could be that index finger tactile afferents are enhanced and forearm tactile inputs are gated leaving more resources available to process index finger inputs. The previous study however could not differentiate the function of tactile gating (i.e., noise reduction or optimizing afferent flow). Therefore, the goal of the present study is to determine whether tactile gating serves to reduce noise or optimize sensory inflow. If tactile gating serves to reduce noise, tactile gating would be observed only at the right forearm as most sensory noise would be generated at the beginning of movement and leave inputs from the index finger unmolested while grasping. When pantomiming there is no expectation to contact the target and tactile gating should be observed at all sites at the to-be-moved limb. Conversely, optimizing sensory inflow predicts...
that tactile inputs at the right index finger should be readily detected while grasping and the reverse should be observed while pantomiming.

The present results are consistent with both predictions. Generally, grasping results in no sensitivity change at the right index finger and the left limb vibration sites. Indeed, Williams, Shenasa & Chapman (1998) observed that tactile gating is maximal at the effector and the gating effect reduces the farther away the segment is from the effector. However, in the Chapman study, participants performed index finger abduction and there was no expectation for tactile information to be relevant for successful goal completion. Likewise, there should be no expectation to use tactile information while pantomiming and there is a modest decrease in index finger sensitivity when pantomiming (although this effect was not statistically significant). This effect is also observed at the right arm. In contrast, tactile sensitivity is higher at all sites on the right limb when participants grasped relative to pantomime grasping. Given the literature reporting the importance of tactile sensory feedback for movements, it is not entirely surprising that grasping elicited enhanced tactile sensitivity compared to pantomime grasping.

The present results are difficult to reconcile with accounts that observed more gating at locations closer to the effector. However, in such cases (e.g., Williams et al., 1998), simple movements were used in which there was no expectation to utilize tactile information (also, see Williams & Chapman, 2002). In such contexts, there is no anticipation of functionally relevant skin contact within the context of this movement and, therefore, no need to inhibit sensory gating. Rather, gating might be the result of a postdictive mechanism where tactile gating arises from sensory reafference that masks other sensory events. Present accounts of pain gating agree with the postdictive explanation. However, gating occurred
before movement onset opposing the postdictive hypothesis. A reach-to-grasp task was chosen because it is a complex behavior requiring many sources of sensory information. While reaching and grasping, the central nervous system should predict that afferent sources would be used. It is valuable to define sensory usage differences in an active behaviour (Edin, 2001). Therefore, in the context of reaching-to-grasp, tactile gating at the fingertips would not occur because tactile information is a relevant source of information and may indicate higher-order control from PFC (see Yamaguchi & Knight, 1990).

Indeed, there is evidence that certain aspects of tactile afferents function for proprioception. In one study Edin & Johansson (1995) anaesthetized the distal index finger such that participants could not perceive any tactile stimuli at or distal of the proximal interphalangeal joint (PIP; achieved with 5 mg·ml⁻¹ bupivicain). Participants were asked to perform a matching task with the unanaesthetized finger while an experimenter manipulated the anaesthetized finger through its normal range of motion. When the experimenter flexed or extended the PIP joint, all subjects correctly matched the movements with the unanaesthetized finger. Interestingly, when the experimenter induced distal stretch of the dorsal surface and compression of the palmar surface, participants responded by flexing the unanaesthetized finger. Likewise, when the experimenter induced dorsal compression and palmar stretch, each participant responded with extension. When the experimenter restricted skin stretch by applying a metal ring around the finger, participants failed to perceive movements.

Furthermore, lateral cutaneous femoral nerve microneurographical recordings indicate that slowly-adapting cutaneous afferents provide kinaesthetic feedback of knee movement. Edin (2001) found three types of slow-adapting afferents. As expected, slowly-
adapting group I afferents (SA I) and slowly-adapting group II afferents (SA II) were found. Unexpectedly, a third group that exhibited shared characteristics of both SA I and SA II afferents. These so-called SA III afferents appear to increase firing rate in response to change in knee joint angle. Cutaneous afferents located on the ventral surface of the thigh exhibit the strongest responses to knee flexion. Most tactile receptors were found either on the posterior or anterior surface of the thigh that would place them at optimal locations to detect skin stretch. Taken together, it appears that stretch receptors in the skin can provide proprioceptive cues for movement control.

6.5.1 Mechanisms Underlying Tactile Gating

There are well-known examples in the literature in which tactile perception changes in the presence of various circumstances. Melzak & Wall (1965) proposed that nociceptive and non-nociceptive inputs form separate synapses on the same projection neuron in the spinal cord dorsal horn. Specifically, they proposed that non-nociceptive inputs close a “gate” preventing nociceptive signal transmission, while nociceptive inputs attempt to open the gate. For example, if a child burns herself on a stove element, there is an initial “sharp” pain (communicated by Aδ fibers) followed by a slow, burning sensation (communicated by C fibers). Activating tactile afferents by stroking the skin surrounding the burn produces mild analgesia. Likewise, shaking a hand after accidentally striking it with a hammer produces the same effect. Broadly speaking, convergence of high- and low-threshold inputs centrally produces the above behaviour (see Johansson & Flanagan [2009] for a review).

In the visual world, saccadic suppression prevents an individual from perceiving the visual world during a saccade. Saccades are fast, ballistic eye movements redirecting gaze on a visual object. Saccades are largely performed voluntarily but many are automatic and are not perceived. Yet, humans perceive a stable visual world despite these movements.
Helmholtz (1866/1963) recognized this and, later, Sperry (1950) and Von Holst & Mittelstaedt (1954) formalized Helmholtz’s ideas in two closely related constructs. The corollary discharge (Sperry, 1950), or the efference copy (Von Holst & Mittelstaedt, 1954), is transmitted with the motor command to cancel out image motion caused by a saccade, although low spatial frequencies are detectable and may become more conspicuous (Burr & Ross, 1982).

Data indicate that a predictive mechanism adjusts the tactile afferent amplitude, presumably removing irrelevant and/or optimizing afferent flow. It appears tactile gating is an analogue of predictive sensory cancellation akin to that observed from the electrosensory organ of the electric fish (Bell, 2001). It is likely that tactile behaviour observed in the present study arise from an efference copy sent to the somatosensory cortex (Bays, et al., 2006). Functional magnetic resonance imaging shed some light on this phenomenon. Reduced BOLD signal relative to baseline was found at the parietal operculum when tactile gating was behaviorally observed and during movement preparation (Jackson, Parkinson, Pears & Nam, 2011; Parkinson, Plukaard, Pears, Newport, Dijkerman & Jackson, 2001). A feed-forward mechanism may account for the above observation as feed-forward accounts argue that self-produced sensory events convey no novel information and are, therefore, attenuated. Forward models generate estimates of sensory consequences of movements and attenuate those signals that match signals predicted by the forward model.

Moreover, the prefrontal cortex (PFC) provides inhibitory input to somatosensory cortex (Yamaguchi & Knight, 1990). Patients with PFC damage demonstrated enhanced SEP compared to healthy controls but it is unclear what functions PFC plays when tactile input is relevant to the task goal. The PFC may project excitatory inputs to S1 activating
inhibitory connections within S1, but there is no evidence to confirm this speculation. However, this observation lends credence to the idea that tactile gating has a central origin for two reasons (Chapman, 1994). First, tactile gating occurs before EMG activity; second, peripheral reafference does not have any effect on evoked potentials due to peripheral stimulation. Since gating occurred before movement onset, it is most likely central, predictive mechanisms play a role in tactile gating.

Somatosensory-evoked potentials (SEP) change before and during movement. SEPs offer clues regarding mechanisms underlying observed behaviour. For example, finger and wrist extension induces gating while frontal N30, parietal P30 and central N60 SEPs had reduced amplitude but the same SEPs did not change when no movement occurred (Shimazu et al., 1999). Also, frontal P22 component generated from motor cortex was unchanged but it remains unclear whether tactile gating occurs in response to the motor command. When participants make self-initiated movements, movement-related cortical potentials, such as the Bereitschaftspotential (BP), precede movement onset. BP is thought to reflect movement preparation processes generated in the supplementary motor area (Neshige et al., 1988). Ogata and colleagues (2009) found that sensory potentials was progressively reduced as movement initiation approached, with most potentials significantly reduced approximately 500 ms before movement onset. SEP reduction time course appears coupled to the BP time course. The present results may reflect efference copy signals from motor cortices affecting the sensory areas. Furthermore, there is some evidence that tactile gating may serve two purposes: remove sensory noise and optimize afferent inflow. The present study provides an indication that tactile gating is sensitive to task by adjusting tactile sensitivity at appropriate
segments of a moving limb. Indeed, sensory gating, in general, is influenced by task (Staines et al., 2000).

Presently, tactile gating was observed at specific areas of the moving effector lending support to the feed-forward argument that irrelevant sensory events attenuate (also see Colino et al., 2014). Additionally, tactile gating is sensitive to task instruction, albeit the effect did not achieve statistical significance. When participants grasped the object no gating was observed at the index finger. But, when participants performed pantomimed grasping, slight attenuation was observed at the index finger. Low power and small sample size likely contributed to the null effect. However, with a larger sample (N = 50), it is conceivable that the null task instruction effect may become statistically significant. Future studies will be attempt to demonstrate whether the task effect is robust (or not).

6.6 Summary

Present results are in line with centrally-driven gating accounts replicating previous findings (e.g., Colino et al., 2014) that tactile gating occurs before movement onset. Also, tactile gating was found to be specific to the moved effector and tactile gating is sensitive to task instruction. Central mechanisms modulate tactile gating depending on the predicted relevance of tactile information. This observation shows that feed-forward mechanisms are modulated in sensorimotor networks, likely optimizing sensory input.
Chapter: Conclusion

7.1 General Remarks

The global question this dissertation attempts to answer can be grouped into three related areas: (1) to characterize how tactile gating behaves in the context of goal directed movements, (2) how the availability of vision interacts with tactile gating within a goal-directed context and (3) how tactile gating responds to different task demands. In the example offered at the beginning of this dissertation and focusing attention on the grasping movement for the coffee cup, tactile information from the hand and fingertips is important to successfully complete the task. It is advantageous for the central nervous system to process tactile information that conveys information from the coffee cup leading to a conscious perception of touching the coffee cup. If tactile information is deemed irrelevant to the task then sensory information will be gated and, therefore, go unperceived. The five studies conducted in the present dissertation attempted to answer the above research question.

Experiments one and two characterized tactile gating within the context of goal-directed action. Specifically, experiment one established when tactile gating manifests and where it manifests, whereas experiment two characterized the time course tactile gating throughout the reach-to-grasp movement. Experiment three broadened the lens examining the influence of other sensory modalities on tactile gating – namely the availability of visual information. Experiments four and five sought to determine how task demands affect tactile gating manifestation and what purpose tactile gating serves. There are two possible answers: tactile gating may serve to attenuate sensory noise or optimize sensory input\(^6\). Collectively, the five studies included in this dissertation demonstrated that tactile gating arises from a centrally

\(^6\) However, it is not suggested that these two sources of gating are mutually exclusive. Rather, they likely function in concert.
generated efference copy signal borne out of movement planning processes in higher order motor centers and is elicited at specific segments of a planned-to-move effector. This concluding chapter will integrate the results from all five experiments addressing the hypotheses posed within each experiment to establish a comprehensive theory explaining the manifestation of tactile gating in movement when sensory information is relevant to task goal.

7.2 Summary of Experiments Conducted

7.2.1 Tactile Gating in a Reaching and Grasping Task

Experiment one found that tactile gating manifests in a reach-to-grasp task flowing from previous work (Buckingham et al., 2010; Chapman et al., 1987; Chapman & Beauchamp, 2006; Williams & Chapman, 2002; also see Colino et al., 2014). Experiment one characterized tactile gating within the context of a reach-to-grasp movement and hypothesized that tactile gating occurs before movement onset. A substantial decrease in sensitivity was observed at the right forearm prior to movement initiation, transiently at the right fifth digit and not at all at the right index finger. There was no observed sensitivity decrease at any tested location on the left limb. However, if a control task was designed that does not require tactile feedback from the index finger (i.e., a ballistic “punch” to a spatial target) it is expected that tactile gating should manifest at the index finger. Collectively, experiment one observations demonstrate that the location of tactile gating depends on whether that limb is moved and whether a particular limb segment contacts the target object at the end of the movement. Tactile gating was observed to be strongest at the limb segment that moves (Chapman et al., 1998). Chapman and colleagues (1998) observed that tactile gating occurred at the second metacarpophalangeal joint when participants abducted the index finger. Experiment one observed maximal gating at the forearm. There appears to be
considerable overlap between the results of experiment one and those of Chapman and colleagues (1998). In both cases participants experienced decreased sensitivity at the primary joint of action indicating that tactile gating may play a role attenuating sensory noise associated with movement. However, there is no expectation for tactile information to be relevant for successful goal completion and, therefore, no need for it to remain effective. In goal-directed reach-to-grasp, cutaneous afferents in the index finger play an important role in signaling phases of object contact and lift and providing sensory feedback of fingertip forces (Johansson & Flanagan, 2009). Clearly, how tactile gating manifests depends on the task context. Sensitivity ($d'$) calculations show reduced sensitivity was confined to the forearm of the moving limb but not at the index finger or any segment of the stationary limb.

What about the sites of vibratory stimulation? Is there a difference in sensitivity between glabrous skin and hairy skin? If there is a difference between glabrous and hairy skin sensitivities and how would that difference change the present observations? Hamalainen, Warren & Gardner (1985) measured sensitivity difference between hairy skin and glabrous skin with air puff stimuli. Stimuli were delivered to the hairy dorsal surface of the thumb and the glabrous thenar eminence. Hamalainen and coworkers observed that reaction time to stimuli applied to hairy skin was shorter than the same stimulus applied to glabrous skin. However, glabrous skin sensitivity was superior to hairy skin when stimuli were concentrated at a single point than when applied over wider skin area. Moreover, when the hair was removed by chemical depilation, hairy skin sensitivity matched that of glabrous skin. Therefore, if vibration stimuli were presented on glabrous skin it is expected that sensitivity would decrease. In other words, if the present experiments compared hairy and
glabrous skin sensitivities, there would be a main effect of skin type; sensitivity at glabrous skin would be detrimentally affected compared to sensitivity of hairy skin.

Tactile gating appears to have temporal specificity. The majority of participants experienced reduced tactile sensitivity before movement onset coupled with an increased detection criterion, indicating bias toward frequent “no” responses, likely indicating that gating originates from a centrally generated mechanism related to movement planning. Indeed, previous work found that reaction time and tactile gating onsets are correlated (Buckingham et al., 2010). Parkinson and colleagues (2011) asked participants to make reaching movements in response to a visual cue and providing tactile stimuli at various time points before or after movement onset and demonstrated that tactile stimuli need to be delivered to the moving limb at early time points for participants to judge movement onset and tactile stimulus delivery occurring simultaneously. This may indicate that motor planning processes elicit tactile gating at the limb about to be moved. The same authors demonstrated that the parietal operculum (secondary somatosensory cortex) expressed decreased BOLD signal when tactile stimulation occurred at the moving arm compared to that when the arm was stationary. Collectively, it appears that movement planning processes attenuate tactile perception.

The prefrontal cortex may be the source of tactile gating to the extent that it provides inhibitory input to somatosensory cortex (Yamaguchi & Knight, 1990). Yamaguchi & Knight (1990) observed enhanced SEPs in patients with PFC damage compared to control participants and exhibit reduced gating. In other words, it appears that the prefrontal cortex exerts inhibitory influence upon the somatosensory cortex resulting in reduced tactile sensitivity at skin locations proximal to primary movement effector (Buckingham et al.,
Experiment One revealed that tactile gating occurs before movement onset indicating a central origin of gating. The results of Yamaguchi & Knight (1990) support the interpretation that tactile gating occurs centrally. Chapman (1994) argues that tactile gating has a central origin for two reasons: first, gating often occurs before EMG activity and second, there are no peripheral stimuli to elicit sensory masking of tactile afferents.

Indeed, Brown and coworkers (2015) show evidence for PFC involvement using continuous theta burst stimulation (cTBS) on DLPFC. Their study attempted to determine if task-relevant gating effect is present in the arm and hypothesized that the N19-P25 SEP amplitude will be depressed with movement. This particular SEP occurs when information arrives at S1 elicited by median nerve stimulation relative to passive movement. Brown and coworkers predicted that when proprioceptive information is relevant, SEP amplitude would be higher. They also predicted that DLPFC is responsible for relevancy-based facilitation. Likewise, a prior study conducted by Bolton and Staines (2011) demonstrated that the P100 cortical potential is typically suppressed when stimuli are task-relevant but cTBS over DLPFC removes this inhibition. P100 suppression typically occurs when attention is directed elsewhere therefore the stimuli are irrelevant for the task at hand. Brown and coworkers had the participant make wrist flexion and extension movements without cTBS, cTBS over DLPFC and cTBS over S1. Their baseline results show that task-irrelevant median nerve stimulation reduces N19 – P25 amplitude relative to baseline and task-relevant nerve stimulation was also reduced with cTBS during passive movement. This gating is partially reduced when the information is task-relevant (i.e., used to guide an active movement). Consistent with the Brown and coworkers hypotheses, cTBS over DLPFC
abolished the task-related enhancement of movement-related gating state, while stimulating S1 resembled that observed under cTBS stimulation. This study shows that DLPFC is, in part, responsible for tactile gating observed in this thesis. It is clear that DLPFC and S1 have important roles facilitating or inhibiting afferent information by modulating afferent information based on task-relevancy (DLPFC) and overall excitability (S1).

However, experiment one does not offer any evidence as to what occurs as a movement progresses toward completion. It is equally likely that tactile sensitivity remains impaired relative to baseline throughout the movement and that tactile sensitivity recovers as a movement approaches endpoint. Reduced H-reflex amplitude was observed during passive lower limb movement (Brooke, 2004) and upper limb movements (Jones et al., 1981; Rushton et al., 1981). Attenuated tactile sensitivity in response to passive limb movement reflects sensory reafference masking tactile events. If tactile sensitivity recovers prior to movement endpoint then this observation reflects diminishing central influence and reliance on peripheral gating sources. In other words, as any movement approaches its end, deceleration of the limb would progressively remove any impetus to gate tactile inputs. Experiment two addressed this question directly by probing tactile sensitivity at intervals throughout the entire movement beyond endpoint.

7.2.2 Time Course of Tactile Gating

Experiment two examined the time course of tactile gating in a goal-directed movement. Experiment 2 replicated experiment one’s finding that tactile sensitivity decreased prior to movement onset at the right forearm before movement onset. However, experiment two found that tactile sensitivity recovers as the movement progresses towards its end indicating that central gating sources have little effect on tactile perception. Tactile sensitivity pattern observed in experiment two reflect that tactile gating is sensitive to task
demands. Indeed, Chapman et al., (1998) found that gating is maximal at the movement effector and progressively weakens at skin locations distal to primary effector. Reaching and grasping demands processing tactile information for effective goal completion (Johansson & Flanagan, 2009) and processing edge orientation (Pruszynski & Johansson, 2014). In experiment two, tactile sensitivity recovered 301 to 400 ms after movement onset – well before movement end (mean movement end = 707 ± 19 ms). It is certainly possible that peripheral gating sources come into play when EMG activity and peripheral sources can be quite effective at reducing tactile sensitivity (Williams & Chapman, 2002). Williams & Chapman (2002) observed largest tactile sensitivity decreases at EMG onset. Moreover, Angel & Malenka (1982) found that the detection threshold correlated positively with oscillation frequency of a reciprocal movement. In experiment two, tactile sensitivity progressively recovered indicating that there was no peripheral source of tactile gating influencing sensitivity. Rather, it appears that once a movement command sequence is sent to the effector muscles, central gating sources have little influence and are no longer effective in eliciting gating. Prior experiments normally found tactile gating in response to oscillatory passive limb movements and asked participants to detect the presence of a threshold stimulus. Hence, there was a continuing presence of peripheral sensory input during oscillatory movement. However, with discrete reaching and grasping movements, as in experiment two, there is little peripheral reafference at later movement stages.

Therefore, central gating sources strongly influence tactile sensitivity observations in experiment two, at least during motor planning and the initial few moments of the movement. Tactile gating occurred before movement onset implicating central mechanisms predicting sensory consequences arising from a planned goal-directed movement. Feed-forward
accounts argue that self-produced events do not convey novel information and are, therefore, attenuated. Buckingham et al., (2010) found their participants experienced reduced tactile sensitivity approximately 50 ms before movement onset. Likewise, Colino and colleagues (2014) found tactile gating occurs on average 75 ms before movement onset. These pre-movement gating observations are difficult to reconcile with studies showing tactile gating during passive movements, in which there is no movement command (see for example Williams & Chapman, 2002). Such studies often cite a masking or postdictive cancellation mechanism caused by temporally coincident stimuli, rather than by predictive mechanisms arising from efferent sources (see Bays et al., 2006 and Voss et al., 2008).

Interestingly, Bays and colleagues (2006) demonstrated tactile gating arising from predictive mechanisms in the context of an amplitude discrimination task. The task goal was to differentiate which one of two tactile taps on the left index finger was stronger by pressing one of two response buttons. Participant’s right index finger was held above the left index finger such that participants could only make flexion-extension movements and touch to the left index finger was delivered via force-transducer and motor system such that there was an 11-ms delay. Participants were split into two groups. The first group experienced predominantly trials in which the right finger elicited left finger contact. The force transducer was moved without the participant knowledge on no-contact trials before the imperative cue but a tap on the left finger was given when the right index finger passed the flexion angle required to contact the force transducer. The second group experienced the same protocol as the first group except that no contact was made on any trial. The authors found the perception of the test tap was reduced compared to the comparison tap when participants expected to contact the force transducer. Similarly, participants experienced
similar reduction on no-contact trials. By contrast, the second participant group did not experience this reduction. In other words, when contact is expected, sensation in the passive finger was attenuated and occurred regardless of whether contact actually occurred. What accounts for the predictive nature of tactile gating? Jackson, Parkinson, Pears & Nam (2011) observed decreased BOLD signal relative to baseline at the parietal operculum (S2) during movement preparation (also see Parkinson, Plukaard, Pears, Newport, Dijkerman & Jackson, 2011). Parkinson and colleagues found similar results when tactile stimulation was delivered to the moving compared to the stationary limb. Likewise, Yamaguchi & Knight (1990) observed reduced somatosensory evoked potentials (SEP) over individuals with intact prefrontal cortex compared to patients with prefrontal cortex damage. Presumably, processes associated with motor planning elicit tactile gating as a usual response to efference copy. Taken together, these results indicate that tactile gating is a response in anticipation of movement and, likely, highly associated with the movement plan.

7.2.3 Availability of Vision and Tactile Gating in Reaching and Grasping Task

With respect to visual touch enhancement, experiment three examined what effect vision may have on tactile gating. Humans often grasp objects that are visible and utilize vision and tactile perception when using tools and/or manipulating objects. Indeed, vision of a limb decreases response time to tactile stimuli on that limb compared to when the same limb is not visible (Tipper, Lloyd, Shorland, Dancer, Howard & McGlone, 1998; Tipper, Phillips, Dancer, Lloyd, Howard & McGlone, 2001). Additionally, two-point discrimination thresholds reduce when participants received non-informative vision of the stimulated arm relative to when neutral objects were presented at the same spatial locations as the tactile stimuli. Furthermore, Taylor-Clark and colleagues (2002) observed enhanced SEP when participants had vision of the limb when they responded to tactile stimuli, suggesting that
vision of the limb enhances tactile sensitivity of that skin location. The results from Experiment Three demonstrate that when participants had vision of the target tactile sensitivity was enhanced relative to no vision. There was also a significant interaction between stimulus location and stimulus time replicating previous results already presented in this dissertation such that the largest tactile sensitivity decrease was observed at the right forearm and the smallest decrease observed at the right index finger.

Experiment Three demonstrates tactile gating before movement onset suggesting the predictive mechanisms at work. Indeed, Bays and colleagues (2006) demonstrated that participants judge a probe tactile stimulus to be weaker relative to a comparison tactile stimulus when participants expected to receive tactile stimulus. The mere expectation to receive tactile stimulus elicits reduced tactile sensitivity. Likewise, and closely related to the results presented in the present dissertation, Voss and colleagues (2008) observed that the mere expectation to move a limb elicits tactile gating, regardless of whether that limb moved or not.

7.2.4 Task Parameters Affect Tactile Gating

Experiments Four and Five address the question how task parameters influence tactile gating behaviour. Experiment Four attempted to address this question by exposing participants to two distinct conditions: a normal grasping configuration (i.e., index finger and thumb) and an altered grasping configuration (i.e., fifth and second digits). Despite the grasp configuration manipulation, there was no statistically significant effect of grasp mode. It could be that if a to-be-moving limb is known in advance of execution, grasp mode does not affect the motor plan if that particular movement pattern is not well practiced. It is more likely that planned movement impulses sent to finger flexors and extensors were either not large enough to effect gating or the predicted movement goal deemed tactile input from both
the digits relevant to successful task completion and, therefore, are not attenuated. Despite the null result of Experiment Four with respect to the grasping configuration manipulation, task does, indeed, affect tactile gating. For example, when participants made bimanual pointing movements perceived intensity reduction occurred at the fingers (see Buckingham et al., 2010), whereas grasping elicited a different spatial gating pattern with tactile gating strongest at the skin area surrounding the forearm (Colino et al., 2014; Juravle et al., 2011). Additionally, tactile gating has been examined in a wider variety of visuo-motor tasks, such as juggling (Juravle & Spence, 2011) and during normal gait (Morita et al., 1998).

Juravle and Spence (2011) hypothesized that response bias shift while performing juggling would result in decreased tactile sensitivity. A participant was asked to detect a short gap in an otherwise continuous vibratory stimulus delivered to the wrist of the participant. Participants were less likely to detect the vibratory gap as a result of a more conservative response criterion making it less likely that a participant would say that a gap was present. Juravle and Spence (2011) demonstrate that there is a decisional component to tactile gating indicating that it may be triggered somewhere in the cortical network and, the authors suggest, before the motor command is sent to the effectors.

Morita and coworkers (1998) examined somatosensory evoked potentials (SEP) when stimulating the tibial nerve and the sural nerve. Potentials were recorded before and during voluntary plantarflexion, dorsiflexion and co-contraction of both ankle joints. Gating of the SEP began 60 ms before muscle activity onset and was maximal 50 – 100 ms after muscle activity onset. Gating was observed from activation of muscle afferents and cutaneous afferents. Also, when cutaneous and muscle afferents were blocked via transient ischemia SEP amplitude was less affected by gating. The tibial nerve SEP was depressed during tonic
muscle contraction, suggesting peripheral components driving gating. Morita and colleagues (1998) suggest that lower limb gating is mediated by both central and peripheral components. Specifically, Morita and coworkers suggest that SEP gating before muscle activity has central origin since there was no way for sensory feedback to affect the SEP. Likewise, ischemic block of afferents precludes the possibility that those afferents would affect the SEP. SEP gating during movement is likely the result of a combination of central and peripheral mechanisms. Gating during tonic muscle contraction is the result of peripheral sources. Taken together, Morita and coworkers suggest caution when interpreting results from gating experiments and care should be taken designing experimental protocols.

In an effort to address this issue in more detail, Experiment Five explored how the task goal would affect tactile gating. In other words, does tactile gating arise simply from specification of a movement plan or is tactile gating sensitive to movement end state? Globally speaking, however, experiment five served to identify which purpose tactile gating serves: remove sensory noise or optimize sensory inflow. Reaching and grasping, indeed, affect tactile gating behaviour such that specific segments of a moving limb attenuate tactile inputs while other segments do not. To that end, participants were asked to perform reach-to-grasp movements with the dominant upper limb in which a cylindrical target was presented. In another condition, participants reached without grasping the target effectively pantomiming an actual grasp. Results revealed reduced tactile sensitivity at the right index finger in the pantomime grasp condition. Experiment Five demonstrated that tactile gating serves to both reduce sensory noise by virtue of muted tactile sensitivity observed at the right forearm before movement onset and optimize sensory inflow because tactile sensitivity decreased at the index finger in the pantomime condition. Therefore, it is appropriate to
repeat that logically there is no expectation to use tactile feedback from the reaching hand when there is no object with which to interact. If that is the case then a sensory inflow optimization account predicts that tactile gating will be observed at the hand while pantomiming. Experiment Five results reconcile with both predictions. As observed in previous experiments, grasping generally elicits tactile gating at the forearm leaving the index finger unchanged (see experiments one through three for results pertaining to the fifth finger). Thus, within the context of a goal-directed action, specifically reaching-to-grasping, fingertip tactile information is a relevant sensory source.

It appears that tactile gating is a necessary consequence of movement planning and appears in a wide variety of visuo-motor tasks such as grasping (Colino et al., 2014; Juravle et al., 2011), juggling (Juravle & Spence, 2011) and during normal gait (Duysens et al., 1995; Morita et al., 1998; Staines et al., 1998) and is sensitive to task (experiment five; also see Williams & Chapman, 2002) and availability of visual input during movement (experiment three of present dissertation). In all cases, tactile gating acts to attenuate sensory noise that would interfere with processing useful sensory information. Using psychophysics may explain how the somatosensory system behaves while tactile gating. Humans are not ideal detectors such that a response profile for a continuum of stimulus intensity does not resemble a step function. In contrast, an ideal detector behaves in such a way that all subthreshold stimuli are not detected while threshold and suprathreshold stimuli always result in positive stimulus detection and that detector has a perfect ability to distinguish between subthreshold and suprathreshold stimuli. In such a circumstance, any stimulus intensities too weak to achieve threshold are undetectable. Once stimulus intensity reaches absolute threshold, and above, stimuli are detectable. However, humans are not ideal detectors and sensory systems
are laden with noise. Signal detection theory (SDT) posits a statistical method accounting for
behaviour in detection situations (Green & Swets, 1989). SDT posits to account for
cognitive factors influencing decision-making that determine whether a signal is deemed
worthy of an affirmative response. SDT posits two probability distributions as a function of
stimulus magnitude: those stimuli caused by noise (N) and those caused by signal and noise
(S+N). The predominant noise source is neuron baseline random activity taking the form of
a normal distribution. In the presence of a signal, the activity associated with the signal adds
to the N distribution. This additive effect means that the S+N distribution will always lie to
right of the N distribution. This consequentially poses a problem since the N and S+N
distributions may occupy very close spaces in the stimulus magnitude dimension. In other
words, noise magnitude may be very high relative to signal magnitude and noise may be
mistaken for signal or noise may be too weak and signal mistaken for noise (Swets, 1996).
Throughout the present dissertation the experimenter asked participants to judge whether
they felt a tactile tap. In other words, did participants perceive a signal added to baseline
noise or was there noise alone? However, one cannot assume that detection rates do not
change given task circumstance and it is important to determine how participants decide
whether any given stimulus is signal or merely noise. Also, what role would expectation
play in the present experiments?

Each participant sets a criterion value in the sensation magnitude dimension from
which any stimulus is judged. If a stimulus magnitude is higher than the criterion then that
stimulus will be judged as a signal or judged as noise if it falls below criterion. If one
expresses this in terms of the N distribution, any stimulus above the criterion is deemed a
false alarm and anything below the criterion is deemed a correct rejection. Otherwise, if a
signal is present (S+N distribution) in a given trial, any stimulus below criterion is a miss and any stimulus above criterion is a hit. Within an experimental session, one can predict false alarm, correct rejection, hit and miss rates if the criterion is known and assuming normal N and S+N distributions. These measures can be predicted by calculating the area under the N distribution below criterion resulting in a predicted correct rejection rate and above criterion gives predicted false alarm rate. Likewise, anything below criterion in the S+N distribution gives predicted miss rate and above give predicted hit rate. The present experiments consistently observed reduced hit rate (expressed as “detection rate” in the experiments) at specific sites on the moved limb, whereas detection rates did not change at other sites (i.e., stationary limb and right index finger). However, the present experiments delivered stimuli on the majority of trials and that expectation certainly shifted criterion. If stimuli are presented in the majority of trials then one expects adoption of a liberal criterion biasing to more frequent “yes” responses. This criterion shift could explain why detection rates were very high. Alternatively, stimulus intensities across all five studies may explain the observed high detection rates and low false alarm rates. Synthesizing the two predictions may compound and result in higher detection rates since N and S+N distribution means are very different. In other words, frequent stimulus presentation sets a liberal criterion while presenting supra-threshold stimuli results in large disparity between N and S+N distributions.

The distance between the N and S+N distribution means is expressed as the difference between the N distribution z-score and the S+N distribution z-score. Classical psychophysical methods, such as establishing S-shaped psychometric functions, are susceptible to criterion shifts (Gescheider, 1997). That is, observed sensitivity changes can be due to nonsensory factors such as expectation and vigilance. For example, two separate
observers may have vastly different detection thresholds yet express identical sensitivity – one observer adopts a liberal criterion while the other observer adopts a stringent criterion. In other words, the threshold in the current example is a biased sensitivity estimate because it is contaminated by criterion shift. Therefore, criterion shifts introduce an important potential confound in any psychophysical experiment for which it must be accounted. Estimating sensitivity requires an unbiased measure that is not affected by criterion shift. The primary dependent variable analyzed in the present dissertation is $d'$. Since $d'$ reflects the relative difference between the N distribution and S+N distribution $z$-scores $d'$ is an index of stimulus detectability uncontaminated by criterion. Sensitivity observed at the unmoved limb shows $d'$ does not change remaining at near-perfect detection rates. This implies that the N and S+N distributions are very far apart reflecting the fact that stimulus intensity is quite large and, hence, easily detectable but $d'$ measured at the moving limb shows that N and S+N distributions overlap more completely implying that detecting stimuli at the right limb are more likely to be dismissed as baseline noise. Furthermore, all experiments observed, regardless of individual experimental manipulations, reduced $d'$ at the moving limb before movement onset.

Hence, there are three factors that contribute to observed results: noise level, signal strength and response bias. Which of these three factors influence the observed data in movement-related tactile gating? If increased noise level were the sole cause of tactile gating, then higher false alarm rates would have been observed without any change in detection rate. However, the present results do not reconcile with the noise level prediction; during movement trials there was no change in false alarm rate with decreased tactile detection. Increased noise level cannot be the primary factor causing tactile gating since that
rationale does not explain current results (also see Williams & Chapman, 2000). Signal strength is the second factor. In a cortical recording study conducted in macaque monkeys it was demonstrated that somatosensory evoked activity evoked by air puff was markedly reduced in 89% of recorded cells when monkeys performed elbow flexion and extension and attenuation was present prior to movement onset (Jiang, Chapman & Lamarre, 1991). This pattern of results suggests tactile inputs are attenuated in a nonspecific way. However, like in finger abduction, there is no reasonable expectation to utilize tactile information. The key point is that movement modulates cortical neuronal activity. The third factor is response bias. Response bias is the tendency to prefer one response over another, determined by factors other than signal intensity. In SDT, response bias, expressed as \( C \) or criterion, is defined as half the sum of the \( z \)-score of \( N \) distribution and the \( z \)-score of \( S+N \) distribution. The present studies used \( C \) as a response bias measure because its range does not depend on \( d' \) (Banks, 1970; Ingham, 1970; MacMillan & Creelman, 1991) and it provides an index of the distance, in \( z \)-score units, from the crossing point of \( N \) and \( S+N \) distributions. In other words, when sensitivity and criterion change as a function of experimental condition, \( C \) is the only response bias measure that can be interpreted without knowing \( d' \) (MacMillan & Creelman, 1990; 1991).

Having this knowledge of \( C \) in mind, the present results can be clearly interpreted. In all cases, detection rate and sensitivity decreased primarily at the right forearm, the spatial occurrence of the tactile gating effect agrees with that observed in monkeys performing elbow flexion and extension (Jiang et al., 1991). However, prior studies did not explicitly measure response bias. Negative \( C \) values indicate bias to frequent affirmative responses whereas positive \( C \) values indicate bias to frequent negative responses (Gescheider, 1997).
Since C is expressed in z-score units, it represents the number of z-score units that the criterion is above or below the point at which the N and S+N distributions cross. It is also advantageous that $d'$ and $C$ are expressed in the same units streamlining interpretation. Thus, when $d'$ decreases as tactile gating occurs, $C$ values increase indicating that response bias shifts to frequent no responses. Thus, decreasing $d'$ coupled with increasing $C$ indicates that tactile gating acts to decrease tactile stimuli detection rate due to non-sensory factors since stimulus intensity was not changed throughout all experiments. Furthermore, tactile gating does not increase noise level since false alarm rates did not increase (Williams et al., 1998; Colino et al., 2014). Such non-sensory factors, which the present candidate posits, must be one of two general mechanisms: a predictive mechanism or a postdictive mechanism.

### 7.3 Mechanisms Responsible for Tactile Gating

The sensory gating observed across all studies in the present dissertation not only reflect a purely masking process, as implied by a cursory signal detection interpretation, but also movement planning processes occurring at higher levels of the nervous system namely the cerebral cortex. Further, these processes are conceived as affecting somatosensory cortex such that tactile stimuli are attenuated and unprocessed because such stimuli fall into the background neural noise due to their being irrelevant to the task goal. However, tactile gating also serves to optimize sensory input as shown in experiment five. In experiment five, it was hypothesized that if tactile gating occurred at the index finger while pantomiming a grasping movement then this result would reflect that tactile gating also serves to optimize input, along with observations from experiments one through four, that grasping a target spares the index finger from tactile gating. It appears that movement planning is sufficient to elicit tactile gating. Indeed, without prefrontal cortical involvement, the area responsible for the movement planning processes, there is no signal attenuating somatosensory stimulus
processing (Yamaguchi & Knight, 1990). However, it cannot be said that movement planning is a sufficient cause of tactile gating. Staines & colleagues (2000) tested the influence of task on sensory-evoked potentials (SEP) by stimulating either the tibial nerve or the sural nerve such that they probed proprioceptive and cutaneous afferents, respectively. Additionally, they presented cutaneous stimuli without movement (i.e., dorsi/plantar flexion) and asked participants match the passively moved foot with the contralateral foot thereby probing proprioceptive function. Staines and colleagues (2000) found that tactile gating occurred with passive movement of the foot and was sensitive to task. When the sural nerve was stimulated SEPs generated during passive movement and cutaneous stimulation were reduced, while SEPs during the position matching condition did not change. Perhaps tactile gating arises from sensory expectation as opposed to motor planning? Such a hypothesis requires further testing. However, when the tibial nerve was stimulated SEPs generated by cutaneous stimulation reduced with passive movement and position matching tasks. Staines and colleagues (2000) support the notion that movement does indeed attenuate sensory signals in a variety of contexts. This idea lends credence to the present results since tactile stimuli delivered to the right forearm were gated but tactile inputs from other locations were unaffected. Furthermore, tactile gating responds to differences in visual information and task constraints. Vision serves to enhance tactile sensitivity (Tipper, Lloyd, Shorland et al., 1998; Tipper, Phillips, Dancer et al., 2001) as previous studies show that vision of a stimulated limb enhances tactile acuity performance. However, Staines and colleagues (2000) used a passive foot movement and, thus, there would not be any movement plan generated to cause gating. Such a task difference is difficult to reconcile with the present results since tactile gating was generated by different sources.
7.3.1 Predictive and Postdictive Mechanisms Responsible for Tactile Gating

There are generally two complementary views regarding tactile gating mechanism: the postdictive view and the predictive view. The postdictive hypothesis posits that tactile gating arises from peripherally driven sensory processes that generally invoke a backward masking explanation for tactile gating action. In a series of experiments conducted by Chapman (Williams, Shenasa & Chapman, 1998; Williams and Chapman, 2000; 2002) participants were exposed to various conditions examining the effects of stimulus location, stimulus intensity and motor tasks on tactile gating. In all experiments, participants were asked to perform index finger abduction activating first dorsal interosseus (FDI) muscle of the hand. Such a task was chosen since finger abduction solely activates the FDI as long as the proximal limb segments are restrained. Such an arrangement is advantageous because the FDI is a fusiform muscle, its electrical activity can be easily recorded and the FDI is easily subjected to experimental manipulations. Therefore, the authors sought to explore the various factors affecting tactile gating. Williams, Shenasa & Chapman (1998) asked participants to perform finger abduction in response to an imperative cue and probed tactile sensitivity at various intervals surrounding reaction time at different locations of the body. Specifically, tactile stimuli were delivered to the ipsilateral second and fifth digits, dorsal hand, forearm, upper arm, shoulder, pectoral girdle and thigh, along with contralateral second digit and thigh. Skin locations most proximal to the first metacarpophalangeal joint had decreased sensitivity during movement while skin locations distal to the movement had a weaker gating effect with higher proportions of detected stimuli at these locations. In other words, there was a strong positive correlation between increasing distance from movement site and the proportion of stimuli that were detected successfully. The authors suggest that a nonlinear distribution accounted for tactile gating behaviour and was influenced by both
central and peripheral factors. Furthermore, stimulus intensity also plays a major role. Williams and Chapman (2000) compared detection rates in five different stimulus intensities. The lowest stimulus intensity was that observed at rest in which 90% of stimuli were detected and four multiples of stimulus intensity were examined. As expected, stronger stimulus intensities elicited higher detection rates with strongest stimulus intensity (i.e., twice the intensity of baseline) resulting in highest detection rates. Williams & Chapman (2002) examined the motor task effect on tactile gating and extensively studied potential causes.

Using the same finger abduction task as in previous studies, the authors manipulated how participants performed that task. The authors asked participants to perform one of four tasks: an isotonic FDI muscle contraction or an isometric contraction of the same muscle, an active movement in which participants abducted their index fingers or passive abduction of the index finger. In every case, Williams & Chapman (2002) found that the peak slope of detection curves occurred before electromyographic (EMG) activity, although no differences were found between task conditions. The authors concluded that during the active movement, there was no effect of central influence when movement was expected, therefore, invoking a backward masking effect gating sensory information from the skin near to the effector. Backward masking refers to the decrease in perceived stimulus intensity when a second sensory input occurs quickly after a first stimulus. It could be that when the second stimulus arrives at cortex interfering the first stimulus’ processing and, therefore, the first stimulus is less intense\(^7\). However, Williams & Chapman (2002) posit that central mechanisms may play a role in movement-related tactile gating effect (see below). The

\(^7\) It should be noted that backward masking effects typically occur when the target stimulus and the mask are presented within 50 ms of each other. The present studies all show gating well before (> 100 ms in most cases) movement onset, discounting the backward masking explanation.
authors observed that movement-related detection rates decreased in active and passive movement before movement onset and posit that this must be due to peripherally-mediated backward masking. This explanation seems likely since the authors did not observe any surface EMG activity from FDI. In the case of passive movement, tactile gating must influence signal strength at some point in the transmission from receptor to somatosensory cortex after stimulus delivery and before movement onset. This interpretation seems likely considering near-simultaneous delivery of a stronger stimulus masks the test stimulus during finger abduction (Gescheider, 1989). Therefore, it is likely that peripheral gating mechanisms play a significant role in movement and cannot be ignored. Indeed, gating was observed during passive movement when participants were instructed to relax and allow the index finger to be moved by an external source. Such generalized effects across a variety of tasks may reflect a functional limit of the somatosensory system to simultaneously handle noise and signal (Williams & Chapman, 2002).

Predictive mechanisms, on the other hand, have been observed across a variety of sensory systems and across species (Bell, 2001; Poulet & Hedwig, 2003; Roy & Cullen, 2004; Cullen, 2004). Indeed, self-generated sensory stimuli are perceived to be weaker than externally generated stimuli (Blakemore, Frith & Wolpert, 1999; Bays, Wolpert & Flanagan, 2005) and this difference is posited to be the result of a predictive mechanism. This hypothesis generally states that prior to a movement an efference copy, generated from movement planning processes, predicts the sensory consequences of the movement (Sperry, 1950; Von Holst & Mittelstaedt, 1950; Von Holst, 1954). Indeed, the present data clearly demonstrate that movement-related tactile gating occurs before movement onset when there is a clear expectation to utilize sensory feedback at the fingers. Hence, it was observed that
tactile gating occurs at the forearm, distal to the elbow (also observed in three participants in Williams & Chapman, 2002).

A recent study (Parkinson, Plukaard, Pears, Newport, Dijkerman & Jackson, 2011) reports data that is consistent with our contention that movement planning attenuates tactile perception. Parkinson et al. (2011) had participants make reaching movements in response to a visual cue and provided tactile stimuli at various points before or after movement onset. The authors predicted, and demonstrated, that a tactile stimulus would need to be delivered to the moving limb at an earlier point in time for participants to judge movement onset and tactile stimulus delivery occurring simultaneously (Parkinson et al. 2011); this finding suggests motor planning leads to tactile gating at the limb that is about to move. Furthermore, these authors also demonstrated that the parietal operculum (secondary somatosensory cortex, S2) was found to express less blood oxygen level dependent (BOLD) response when tactile stimulation occurred at the moving arm compared to the BOLD when the limb was stationary.

Therefore, it is likely that movement planning processes housed in the prefrontal cortex play a significant role in tactile gating. Demonstrably, Yamaguchi & Knight (1990) compared two participant groups: patients with prefrontal lesions and healthy controls. Yamaguchi & Knight (1990) observed enhanced early sensory-evoked potentials (SEP) in the patients with PFC damage compared to control participants. What function does PFC have when tactile input is relevant? Brown and colleagues (2015) demonstrate that the N19-P25 SEP amplitude increases when cutaneous input is relevant and argue that PFC performs decisions to allow certain inputs to pass the gate. Blakemore and coworkers (1999) demonstrate that the cerebellum activity correlated with activity in S1 and S2 only when
stimuli were self-generated not when stimuli were externally generated. This observation suggests that the cerebellum identified sensory noise generated by task-irrelevant sources and actively generated an efference copy effectively canceling out the self-generated activity. Therefore, vibratory stimuli generated by the micromotors (e.g., the right forearm) would not be perceived since any neural activity from affected skin regions are cancelled out by the efference copy. This clearly points to central mechanisms working in concert and Chapman (1994) agrees arguing that tactile gating is largely central in origin for two reasons. First, gating often occurs before EMG activity in the limb that will move; second, peripheral reafference does not have any effect on evoked potentials due to peripheral stimulation. Likewise, the present experiment shows tactile gating to occur before movement onset. Therefore, it is unlikely that peripheral reafference plays a role in tactile gating – at least in the realm of motor planning. Experiment two clearly shows that $d'$ and $C$ gradually return to pre-movement levels before movement end. This reflects that either signal levels increase as movement end draws near since $d'$ values increase (reflecting increased distance between the $N$ and $S+N$ distribution means) and criterion point shifts closer to the crossing point between the two distributions (decreasing $C$ values). Ogata and colleagues (2009) offer evidence suggesting that motor planning plays a large role in tactile gating by measuring changes in tactile detection and correlating those changes to the Bereitschaftspotential that is generated at the supplementary motor area (Neshige et al., 1988). Participants generated self-initiated movements (i.e., no imperative cue was given). Ogata and colleagues (2009) demonstrated that the P27 potential recorded at C3’ (2 cm posterior to C3) was found to be different from the resting baseline during the 1500 ms pre-movement time epoch. Other sensory potentials progressively became reduced as movement initiation approached, with most potentials
significantly reduced approximately 500 ms before movement initiation. The SEP reduction time course closely resembles the BP time course and these processes may be correlated (Ogata et al., 2009).

How do these neurophysiological observations correlate to the present findings? Movement-related cortical potentials (MRCPs) are recorded from the scalp with EEG. MRCPs consist mainly of Bereitschaftspotential (BP) followed by a negative slope change. The BP is typically recorded from 1,500 ms before movement onset, while the negative slope change is recorded 500 ms before movement onset. During this pre-movement time period, SEP amplitudes decrease and may, therefore, be correlated (Ogata et al., 2009). The supplementary motor area (SMA), a cortical area responsible for movement planning, generates the BP (Neshige et al., 1988). In the present experiments, sensitivity decreased markedly after the first time bin (~300 ms) before movement onset and likely is correlated to the first appearance of the negative slope change (often observed 500 ms before movement onset). BP and negative slope change is often recorded in self-paced movement tasks, in which there is no imperative cue to begin movement (Neshige et al., 1988). In other words, participants initiated movement at their own time. The present experiments had participants begin movement in response to an imperative cue that perhaps decreased the time interval between negative slope onset and movement onset. Clearly, based on the expectation that MRCPs are related to movement planning and tactile gating occurs in the presence of a motor plan, then it is likely that tactile gating must be related to the onset of the negative slope.

7.4 Concluding Remarks

The current data provide new insights into how tactile gating occurs in the context of movement planning. Based on the fact that it was observed before movement onset, our
results are consistent with the fact that tactile gating is a centrally driven effect. Furthermore, tactile gating was not observed to be a global effect across both limbs. Rather, it appears to be specific to the to-be moved effector and specific to segments of skin in that moved effector. Central mechanisms are able to modulate tactile gating depending on the predicted relevance of tactile information. The present observations show that feed-forward mechanisms are modulated in sensorimotor networks, optimizing sensory input. Likewise, peripheral mechanisms play a large role, as well. Given both are at work before and during movement, it is critical to understand what is the functional limit of the somatosensory system; how much information can the somatosensory system process (Williams & Chapman, 2002)? This general question will drive future research in a host of applications in a variety of contexts. For example, certain car manufacturers employ tactile feedback systems installed in the driver’s seat informing the driver that another car is in her blindspot or, very simply, providing vibratory feedback to video game players by game controllers with installed vibrator motors. Furthermore, complete understanding of the somatosensory system will pave the road to designing and implementing prostheses that are fully integrated into the nervous system. Such a prosthetic should provide a complete sensory experience along with skilled motor performance.

Therefore, it is critical that we understand precisely how the somatosensory system functions so that effective applications are provided to society. Improving our understanding of the somatosensory system will offer more options and better strategies for industrial and commercial applications. Beyond these applications, complete and accurate knowledge of how the brain controls and adjusts sensory inflow can improve the lives of individuals.
7.5 Limitations of the present research

Despite insightful views the present dissertation revealed, there are, unavoidably, limitations that must be admitted. All of the present studies used psychophysical measurements which can be susceptible to biases, which is the reason $C$ was calculated to measure response bias change. Additionally, $d'$ is known to change between experimental conditions for reasons unrelated to sensory processing but $d'$ changes simply due to non-sensory factors such as changing response criterion (Gescheider, 1997). Also, all the present studies examined tactile gating using suprathreshold stimuli in which all stimuli were detectable at rest. This introduces a ceiling effect into sensitivity data. In future studies, a standard near-threshold stimulus intensity (i.e., 90% of stimuli detected) is better suited to demonstrate a decrease in detection rate. This may provide a better control than using the stationary limb detection rate as a control. However, this is not the case as previous behavioural and neurophysiological studies assert that tactile gating can occur in the absence of overt movement and assumes that tactile gating only occurs in the presence of overt movement (see Bays et al., 2006; Voss et al., 2008). Indeed, past investigations that studied tactile gating in the context of goal-directed action did not include a traditional baseline detection measure because such studies used dual-task paradigms. Additionally, the present studies did not intersperse equal numbers of no movement trials assessing detection rate with movement trials. Hence, the present results must be interpreted with caution. Most recent studies in our laboratory included this condition into the experimental design and found that participants are able to detect nearly 100% of tactile stimuli while not moving. Also, vibrator motor placement may have changed slightly across experimental sessions but this is an issue of statistical power and differences in vibrator motor attachment would contribute to the error term. Participants who did perform baseline detection at rest exhibited the same detection behavior at all stimulus intensities.
used. Finally, future studies recruiting more participants (especially in experiment 5) will reveal interactions more effectively increasing observed power.

7.6 Global Interpretation of the Present Research

Tactile gating is the result of motor planning processes and sensory feedback from peripheral sources evidence by reduction of tactile gating before movement onset and tactile sensitivity’s recovery later in the movement. Konen & Haggard (2014) posit that tactile gating results from sensorimotor interactions inhibiting specific somatosensory areas under certain conditions. Principally, combined results across all five studies are consistent with the fact that tactile gating is a centrally-driven effect. Furthermore, tactile gating was not observed to be a global effect across both limbs. Rather, it appears to be specific to the to-be moved effector and specific to segments of skin in that moved effector. Central mechanisms are able to modulate tactile gating depending on the predicted relevance of tactile information. This observation shows that feed-forward mechanisms are modulated in sensorimotor networks, optimizing both sensory input and attenuating movement-related sensory noise.
References


Appendix

Appendix A Consent Form

THE UNIVERSITY OF BRITISH COLUMBIA

CONSENT FORM
Utilization of visual information for the preparation and control of a goal-directed reaching movement.

Principal Investigator: Gord Binsted, PhD
Dept of Human Kinetics, UBC-O
(250) 807-9642

Co-Investigators:

Sponsor:
Natural Sciences and Engineering Research Council of Canada (Binsted)

INVITATION TO PARTICIPATE
You are invited to participate in this study because you are a healthy adult, between the ages of 19 and 50 years of age, and you have either normal vision or wear corrected lenses. Please read this form carefully, and feel free to ask any questions you might have.

PURPOSE
Over the last few years, we have conducted a number of studies designed to examine characteristics of manual control. Our interests have been the factors that affect the interactions between perception and action. As part of our continuing research program, we are conducting further investigations to extend work on the processes underlying the coordination of perception and movement planning and control. In this study, you will be asked to reach to an experimenter specified target in response to a computer-generated signal (tone or target presentation). Your goal will be to move your hand(s) as quickly and accurately as possible to the target. The experimenter may also request for you to fixate (i.e. look at) specific locations prior to, during, or upon completion of your limb movement.

STUDY PROCEDURES
In this experiment, you will be asked to make either key-presses or reaching movements in response to visual targets. Your primary goal will be to respond as quickly and as accurately as possible. A computer will monitor your eye and/or hand movements as well as your overall
Your limb movements will be recorded by the combination computer input (i.e. mouse/keyboard) and the monitoring of an infra-red light placed on your hand (similar to the light used by a T.V. remote control). A set of cameras tracks the movement of this light. No viewable record is retained; only a file containing the position of this marker is saved for later analysis. Eye movements are recorded using similar infra-red light based cameras. This system, worn like a construction helmet, films your eye movement and records a trace of the position of your eyes.

A computer will coordinate the equipment to monitor your eye and hand movements. A computerized movement analysis system will be used to study these movements in detail. This information will help us understand specific features of your visual-motor performance.

Following your receipt of this consent form, the researcher will schedule a brief meeting (approx. 10-15 min) to answer any questions you have regarding this study. If you choose to participate, the experiment will require approximately 2 additional hours to complete; a date and time for this will be set at your convenience.

POTENTIAL RISKS
The risks involved in participating in this experiment will be minimal. That is, the risks to you are no greater than the risks experienced in everyday life. All the movements/tasks you will be asked to perform are intended to simulate activities of daily living and should therefore present no appreciable threat to your welfare. You might experience slight fatigue, as you will be asked to maintain focused attention and concentration throughout the experiment.

POTENTIAL BENEFITS:
The basic interest of this research is to understand the neural (brain) processes associated with everyday movement. Please note, however, that there will be no direct benefit to you by participating in this investigation.

CONFIDENTIALITY
Any information resulting from this research study will be kept confidential. All documents will be identified only by a code number and kept in a locked filing cabinet in the principal investigators research office. You will not be identified by name in any reports or scientific publications of the completed study. All backup computer files will be stored on password-protected media in a locked filing cabinet, and any data files that reside on the data analysis computer in the Coordinating Perception-Action Lab (University of British Columbia, Okanagan). Only Dr. Binsteed and his research team will have password access to these files. The data will be used
for publication purposes only, and will be retained for a minimum of five years post-publication or completion of the study, after which time raw data will be destroyed.

Your confidentiality will be respected. No information that discloses your identity will be released or published. Research records identifying you may be inspected in the presence of the Investigator by representatives of the Natural Sciences and Engineering Research Council of Canada, or the UBC Research Ethics Board for the purpose of monitoring the research. However, no records that identify you by name or initials will be allowed to leave the Investigators' offices.

**COMPENSATION**

You will be reimbursed at a rate of $10.00/hr upon completion of your participation.

**CONTACT**

If you have any questions or desire further information with respect to this study, you should contact Dr. Gord Binsted at his phone number (250 807 9642), which operates 24 hours a day, seven days a week.
PARTICIPANT CONSENT

If you have any concerns about your treatment or rights as a research subject, you may contact the Research Subject Information Line in the UBC Office of Research Services at 604-822-8598 or if long distance e-mail to RSIL@ors.ubc.ca.

Consent:
Your participation in this study is entirely voluntary and you may refuse to participate or withdraw from the study at any time without jeopardy to your [for example, employment, class standing, access to further services from the community centre, day care, etc.].

Your signature below indicates that you have received a copy of this consent form for your own records.

Your signature indicates that you consent to participate in this study.

______________________________________________
Subject’s Name (Print)

______________________________________________
Subject’s Signature Date