THE REFLEX RESPONSE OF HUMAN NECK MUSCLES TO WHIPLASH-LIKE PERTURBATIONS

by

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B.A.Sc. (Hons), University of British Columbia, 1986

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF
THE REQUIREMENTS FOR THE DEGREE OF

DOCTOR OF PHILOSOPHY

in

THE FACULTY OF GRADUATE STUDIES

(School of Human Kinetics)

We accept this thesis as conforming
to the required standard

THE UNIVERSITY OF BRITISH COLUMBIA

June, 2001

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Date August 14, 2001

DE-6 (2/88)

ABSTRACT

Most whiplash injuries are sustained in isolated rear-end collisions which occur without warning. Most studies of whiplash injury, however, have used multiple tests of subjects aware of the imminent perturbation. This thesis examined how multiple exposures and subject awareness of the presence, timing and amplitude of a whiplash-like perturbation affected the activation and amplitude of the neck muscle response and the peak kinematic response of the head and torso. In Experiment 1, the malleability of neck muscle reflexes was examined in 20 subjects (9F, 11M) who performed ballistic flexion and rotation head movements in a warned, simple reaction-time protocol. When a loud startling sound (124 dB) replaced the 'go' tone (76 dB), a hypermetric version of the reaction-time movement was evoked at the startle reflex onset latency. This result indicated that a reflex neck muscle response could be altered by mental preparation of a movement. In Experiment 2, 66 seated subjects (35F, 31M) underwent multiple perturbations with a peak forward acceleration of 1.5g. To their first perturbation, subjects who were deceived and unexpectedly perturbed responded differently than subjects given either exact or inexact information regarding perturbation timing. Advance warning of the perturbation appeared to produce anticipatory facilitation of the sensorimotor system mediating the reflex response. Subjects exposed to ten more perturbations exhibited a rapid habituation of their muscle response and complex changes in their kinematic response. Thirty-six of the 66 subjects (20F, 16M) then underwent 72 more perturbations interspersed with high (2.2g) or low (0.8g) acceleration perturbations. Response differences were not observed between warned and unwarned presentation of these different perturbations, which suggested that advance knowledge of acceleration amplitude did not affect subject responses. The remaining 30 subjects (15F, 15M) were exposed to seven different perturbations which showed that neck muscle and kinematic responses were graded to both perturbation acceleration and velocity. These experiments demonstrated that subject awareness of an imminent perturbation and habituation of the muscle response to multiple perturbations produced complex changes in the kinematic response, and suggested that neck muscle and kinematic responses of unprepared occupants in real whiplash collisions were different than human subject responses observed in most whiplash injury studies.

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CONTRIBUTION OF THE AUTHOR

This thesis contains two experiments that have been undertaken by the candidate, Gunter P. Siegmund, under the supervision of David J. Sanderson (Associate Professor, School of Human Kinetics). The conduct, analysis and documentation of each experiment was primarily the work of the candidate.

The above statement was written by Gunter P. Siegmund and agreed upon by the undersigned.

David J. Sanderson, Ph.D.

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PREFACE

This thesis consists of two experiments. The first experiment was designed to address two related questions and was written up as a single study (Chapter 2). The second experiment was designed to address four separate questions and was therefore written up as four separate studies (Chapters 3 through 6). To assist in the future publication of these studies, each chapter contains a complete introduction, methods, results and discussion relevant to that chapter. Chapter 2 was written for and has been accepted by the Journal of Physiology. Chapters 3 and 4 have been written for journals focused on the biomechanics of whiplash injury, and Chapters 5 and 6 have been written for neurophysiology journals. A consequence of this format was some repetition of material, particularly of the Instrumentation and Data Reduction sections within the Methods of each chapter, and a minor loss of fluidity between chapters. To smooth the transition between chapters, bridging summaries have been added to the end of Chapters 2 through 5. The final Discussion in Chapter 7 was then focused on an overall interpretation of the results and how the combined results of these different experiments affected the study of whiplash injury and our understanding of the aetiology of whiplash injury.

ACKNOWLEDGEMENTS

I would like to thank a number of individuals and organizations for their support of my graduate studentship and my thesis research.

Considerable thanks are due to my thesis advisor, Dr. David J. Sanderson. He deftly provided the right balance of questions and answers to create an optimal learning environment. In addition to many other things, he has taught me that I still have much to learn. I also wish to thank Dr. J. Timothy Inglis for encouraging me to understand the neurophysiological implications of my work and Dr. Barry S. Myers for ensuring that my work remained soundly rooted in engineering mechanics. It is hard to imagine a better thesis committee than one composed of these three individuals.

My doctoral experience would not have been possible without the financial support and encouragement of my fellow partners at MacInnis Engineering Associates. Thank you for allowing me the time and flexibility to pursue a dream.

Thank you to Mr. Jeff Nickel and Mr. Mircea Oala-Florescu of MacInnis Engineering Associates for designing and constructing the sled used for these experiments. Thank you also to Dr. J. Timothy Inglis for the use of his EMG recording system and to Dr. Romeo Chua for the use of his Optotrak system.

Volunteer fees for Experiment 1 were paid from a grant to Dr. J. Timothy Inglis by the Natural Sciences and Engineering Research Council of Canada (NSERC). Partial funding for Experiments 2 through 5 was provided by a Woodbridge Grant awarded by the Physical Medicine Research Foundation, Vancouver, BC. I would also like to acknowledge the Post-Graduate Scholarship (PGS) program offered by (NSERC) and the Graduate Research Engineering and Technology (GREAT) scholarship program offered by the Science Council of British Columbia (SCBC) for their support throughout my tenure as a doctoral student.

I would like to dedicate this thesis to my wife Louise and our children Emma and Hannah. Without their steadfast support and generous patience, this selfish endeavor would not have been possible. Thank you.

CHAPTER 1 INTRODUCTION

Little is known about the aetiology of whiplash injury. Biomechanical investigations into whiplash injury have used various models, including human subjects, cadavers, physical models and mathematical models, to attempt to better understand the dynamic response and ultimately the distribution of tissue loading in whiplash-injured individuals. Each model has its strengths and weaknesses. Human subject data have included a muscle response not present in cadavers, and both physical and mathematical models have relied on human subject data for validation. Human subject data, however, also have limitations. In addition to necessarily remaining below a severity which causes lasting injury, the external validity of the laboratory response to a simulated rear-end collision remains a question. External validity is the extent to which the results of a study can be applied or generalized to the actual population under study (Campbell and Stanley, 1963). A real whiplash-producing collision consists of a single perturbation, often with little or no warning. Laboratory experiments, on the other hand, have often consisted of multiple perturbations and have used subjects who were aware, to varying degrees, of the timing, amplitude and indeed the presence of an imminent whiplash-like perturbation. For the purposes of this thesis, these three different levels of awareness have been called temporal awareness, amplitude awareness and event awareness respectively. In this context, the overall goal of this thesis was to improve our understanding of the role that multiple exposures and information regarding the timing, amplitude and presence of a whiplash-like perturbation have on the response of human subjects in a laboratory setting.

1.1 An Overview of Whiplash Injury and Biomechanics

Whiplash injury presents with a palette of symptoms which include neck and back pain, headaches, dizziness, blurred vision, paraesthesias, and cognitive difficulties (Bogduk, 1986; Evans, 1992). The injured tissues responsible for chronic whiplash symptoms have been the subject of much speculation and, to date, only one controlled clinical study designed to anatomically isolate the injured tissue has been carried out. Using a double blind, placebo-controlled study, Lord et al. (1996) showed that the cervical facet joints were the source of neck pain in 60 percent of a population with chronic pain secondary to whiplash injury. Transient symptoms lasting less than one week have been attributed to muscle injury (Bogduk, 1986; Brault et al., 2000).

Most whiplash injuries have occurred in rear-end collisions in which the injured party's vehicle was struck at the rear and accelerated forward (Jakobsson et al., 2000). In keeping with this observation, the whiplash-like perturbations used in this thesis consisted of horizontal forward accelerations applied to subjects seated in an automobile seat. Numerous human subject studies have

shown that during a low-speed rear-end impact, the pelvis of a forward-facing, normally-seated occupant is accelerated forward first. Because of seat back compliance and occupant posture, acceleration of the upper torso lags acceleration of the pelvis. As the forward translation of the torso relative to the initially stationary head increases, the lower vertebrae of the cervical spine extend and the upper vertebrae flex. Shear forces developed at the top of the cervical spine ultimately accelerate the base of the skull forward and set up a rearward rotation (extension) of the head. In the presence of a head restraint, both the head extension and the horizontal translation between the torso and head (called retraction) are arrested by the combination of an external force applied to the head by the head restraint and internal forces developed in the ligamentous cervical spine and cervical muscles. If no head restraint is present, larger extension and retraction motions occur before the head motion is arrested by internal forces alone.

1.2 Cervical Muscle Response

The neck muscles have been shown to activate early in the dynamic response of subjects exposed to whiplash-like perturbations (Table 1.1). Aside from one study, the mean activation times of the sternocleidomastoid muscle (SCM) were less than previously-reported voluntary activation times to a somatosensory stimulus (117 ± 16 ms) and an acoustic stimulus (107 ± 21 ms) (Mazzini and Schieppati, 1992). The amplitude of the muscle response evoked by a whiplash-like perturbation has also been shown to increase with perturbation intensity (Brault et al., 2000). Taken together, the pre-voluntary activation times and the graded nature of the neck muscle response to perturbation intensity are indicative of a reflexive response (Gordon and Ghez, 1991).

Though consistent activation times have been observed, activation itself has not been consistently reported in all studies of whiplash biomechanics. Two studies listed in Table 1.1 reported low or no cervical paraspinal muscle (PARA) response (Gutierrez, 1978; Ono et al., 1997). In both studies, subjects were exposed to multiple trials, and as a result of the study design, subjects in both studies knew when impact would occur. In contrast to these findings, Brault et al. (2000) observed a clear SCM and PARA muscle response in novice subjects exposed to only two unexpected perturbations spaced a week or more apart. These differing results suggested that multiple sequential exposures or information regarding the timing of a perturbation (temporal awareness), or a combination of both, may have affected the reflex response of the posterior neck muscles in the earlier two studies. These explanations were consistent with well-documented phenomena related to reflex muscle responses: habituation and central set.

Habituation has been defined as a centrally generated decrement in response magnitude to repetitive stimulation (Harris, 1943). It typically consists of a rapid initial decrement which becomes

progressively slower with time and exposure repetitions. Rapid habituation has been observed in postural reflexes (Nashner, 1976; Hansen et al., 1988; Woollacott et al., 1988; Allum et al., 1992; Bisdorff et al., 1994; Timmann and Horak, 1997) and startle reflexes in humans (Davis, 1984). In free-fall experiments of supine subjects, the magnitude of the electromyographic (EMG) signals was reduced by 30 to 50 percent by the third exposure (Bisdorff et al., 1994). Although habituation produces a decrement in response, not all components of a response are necessarily reduced by the same amount. Habituation of the startle reflex has been shown to occur more readily in the extensor muscles, leaving primarily a flexor muscle response after habituation (Davis, 1984). Moreover, complete elimination of reflex activation in antagonist muscles has been observed during repeated postural perturbations (Woollacott et al., 1988). This extreme form of habituation may be consistent with the absence of an extensor muscle response observed in subjects exposed to multiple whiplash-like perturbations (Gutierrez, 1978; Ono et al., 1997). In recognition of habituation, many studies of reflex responses have used practice trials to achieve a stable response in their subjects before beginning data collection.

Table 1.1. Summary of data from previous whiplash studies in which muscle activity was measured. Mean (S.D.) given for muscle activation times. Δv, velocity change; SCM, sternocleidomastoid muscle; PARA, cervical paraspinal muscles; M, male; F, female; NR, not reported.

Study	Subjects		Perturbation properties		Muscle activation times	
	Gender (M/F)	Age (yrs)	Δv (km/h)	Duration (ms)	SCM (ms)	PARA (ms)
Gutierrez (1978)	3/0	22-29	6.6	80-87	68 (12)[1]	NR
Ono et al. (1997)	3/0	24[2]	~6[3]	~140[4]	79 (9)	NR
Szabo and Welcher (1996)	4/1	22-54	7.5-10.0	80-90[4]	118 (11)	117 (11)
Meyer et al. (1998)	2/0	~30	5.5-9.0	75-160	72[5]	72[5]
Castro et al. (1997)	14/5	26-47	8.7-14.2	105-169[6]	NR	61[5]
Pope et al. (1998)	10/0	18-25	NR	NR	~100	~100
Magnusson et al. (1999)	8/0	24-56	~2[7]	~250-300	73 (15)	175 (67)[8]
Brault et al. (2000)	21/21	20-40	4	138 (4)	91 (9)	96 (11)
Brault et al. (2000)	20/19	20-40	8	135 (2)	81 (8)	84 (9)

1. From onset of sled acceleration as estimated from graphs of all data in appendix.
2. Mean age of all 12 subjects in entire experiment.
3. Impact speed, rebound not known and therefore speed change not known.
4. Estimated from sample data in figures.
5. Relative to onset of vehicle acceleration.
6. From partial results reported by Meyer et al. (1998).
7. Estimated from sample acceleration pulse; acceleration pulse was biphasic.
8. Onset for splenius capitus from wire electrodes.

Another well-documented feature of postural perturbations is the development of a preparatory state called central set (Nashner, 1976; Horak et al., 1989). Central set is an *a priori* selection of the appropriate pattern and magnitude of a muscle response to a stimulus based on prior experience and the expectation that the next stimulus will be of a similar magnitude. Whereas habituation refers to a non-specific decrement in response magnitude, central set refers to a tuning process that may increase or decrease the response of a muscle based on prior experience (Timmann and Horak, 1997). Central set has been observed to develop under conditions where muscles were reflexively activated before complete information regarding the intensity of the stimulus was available; it has not been observed when the stimulus information needed to grade the muscle response was available prior to activation (Horak et al., 1989). Central set is therefore a predictive phenomenon. Since muscle activation has been shown to precede the end of the perturbation in some whiplash exposures (Brault et al., 2000), it is possible that awareness of the perturbation amplitude, i.e., amplitude awareness, has been used by subjects in some experiments to tune their muscle response during sequential exposures to similar intensity perturbations. Though perturbation amplitude has been varied in many previous whiplash experiments using human subjects, the effect of amplitude awareness on the response of subjects to whiplash-like perturbations has not been previously studied.

Both habituation and central set require multiple exposures to a stimulus to develop. Collisions which result in whiplash injuries, however, are frequently one-time events which occur without warning (Sturzenegger et al., 1994). Therefore, it is the first, unhabituated response of a surprised individual to a whiplash-like perturbation that should be studied to understand a large proportion of whiplash injuries. Based on this criterion, none of the studies listed in Table 1 actually replicated real rear-end collisions in which occupants are surprised by the collision. Even if not aware of the exact timing of a perturbation, i.e., not temporally aware, these subjects still knew that some form of perturbation would occur, i.e., they were event aware. Based on epidemiological data, this difference in event awareness between laboratory tests of whiplash biomechanics and real collisions may be important. Unprepared occupants have reported a higher frequency of multiple symptoms and more severe headaches than prepared occupants (Sturzenegger et al., 1994). Moreover, individuals reporting multiple initial signs and symptoms after a whiplash-producing collision were more prone to develop a chronic injury (Suissa, 2001). Sturzenegger et al. (1994) hypothesized that the muscle response was likely the primary variable which differed between surprised and prepared subjects, but did not propose a mechanism by which awareness of the impending collision might reduce the frequency and intensity of symptoms.

A difference in muscle response was also a distinguishing feature between the results of

whiplash experiments conducted on novice subjects who were not temporally aware (Brault et al., 2000) and whiplash experiments conducted on experienced subjects who were temporally aware (Ono et al., 1997). This difference in the muscle response observed under two different experimental protocols raises questions regarding both the role of awareness in the response of human subjects exposed to whiplash-like perturbations and the role that multiple exposures play in producing habituated responses that might not be representative of responses in real collisions. To date, there have been no systematic biomechanical investigations of the effect of either subject awareness or habituation on the muscle and kinematic responses produced during whiplash-like perturbations. An understanding of how these phenomena affect both the cervical muscle response and the induced kinematics during whiplash-like perturbations will help to define how to better study whiplash injury in the laboratory, will assist in interpreting the external validity of previous whiplash experiments using human subjects, and may provide insight into the aetiology of whiplash injury.

1.3 Overall Goal

The overall goal of this thesis was to answer the following question: Do awareness and habituation alter the muscle and kinematic responses of human subjects exposed to whiplash-like perturbations? Three elements of awareness were considered: i) temporal awareness, defined as information regarding the timing of a stimulus; ii) amplitude awareness, defined as information regarding the intensity of the stimulus; and iii) event awareness, defined as information regarding the presence of the stimulus.

1.4 Development of the Experiments

1.4.1 Experiment 1 – Malleability of a Neck Muscle Reflex

The research question posed above assumes that a degree of malleability exists in the reflex response of the human cervical muscles. It further assumes that a subject aware of an imminent perturbation could exploit this malleability, either consciously or unconsciously, to alter their reflexive neck muscle response. If such readiness is able to alter the muscle activation, then information regarding the magnitude of that alteration and how it changes over multiple sequential trials is important. If, on the other hand, such readiness is unable to alter the muscle activation, then questions regarding the role of awareness might, at best, require a different framework or, at worst, be moot.

The first part of this investigation was therefore to quantify the degree of malleability available in the reflex response of the cervical muscles. Valls-Solé et al. (1997, 1999) recently examined how the motor readiness present immediately before a ballistic reaction time movement

altered the startle reflex in distal upper and lower limb muscles. To examine this phenomenon, the "go" stimulus they used during a forewarned, simple reaction-time task was occasionally replaced with a loud sound capable of producing an acoustic startle reflex. Their data showed that the movement prepared for the reaction-time trial was preserved during the startled trials, but that the muscle onset latency was shortened to that of the acoustic startle reflex (Valls-Solé et al., 1999). Although a quantitative analysis of the muscle response magnitude was not performed, their qualitative analysis indicated that there was no difference in magnitude between the startle-induced motor response and the voluntary motor response. Therefore, the normal startle response in the distal limb muscles appeared to be extinguished by a subject's readiness to execute a movement and replaced by the muscle response prepared for the reaction-time task.

An important component of the study conducted by Valls-Solé et al. (1999) was the level of readiness that existed in subjects between the warning and go stimuli. Although a similarly heightened state of readiness may not be present in subjects about to undergo a whiplash-like perturbation, the heightened level of readiness present in advance of a ballistic reaction-time task provides an opportunity to study the maximal effect that readiness might have on the cervical muscle response. The results reported by Valls-Solé et al. (1999) implied that mental preparation of a motor response in advance of a whiplash-like perturbation could have a profound effect on the muscle response evoked by the perturbation if subjects used their awareness of the perturbation timing to ready such a response.

Unfortunately, the results reported by Valls-Solé et al. (1999) in distal limb muscles cannot be directly extrapolated to the cervical muscles. The cervical muscles, particularly the SCM muscle, have been shown to respond more strongly to an acoustic startle stimulus than do limb muscles (Brown et al., 1991a; Vidailhet et al., 1992) and it was not clear how this stronger connection would interact with subject readiness (Hypothesis 1A). It was also not known whether the startle-triggered movement reported by Valls-Solé et al (1999) was the result of readiness to perform the movement or whether the practice acquired during the numerous reaction-time trials preceding the startled trials also played a role. If a normal reflex response could only be altered by a previously-practiced movement, then the first few responses of a subject exposed to a whiplash-like perturbation might be representative of real exposures for a limited number of initial perturbations (Hypothesis 1B). Based on these unresolved issues, Experiment 1 was designed to test whether motor readiness could alter the reflex response of the neck muscles and whether this phenomenon was present in the first trial prior to practicing the movement. To answer this question, a whiplash perturbation was a less suitable stimulus than the acoustic startle used by Valls-Solé et al. (1999) because it superimposed a relatively large perturbation-induced movement onto the movement generated by the muscle response. Isolating

the movement related to the muscle response from the overall movement would be difficult. To eliminate this problem, an acoustic stimulus rather than a whiplash-like perturbation was used to evoke the neck muscle reflex for Experiment 1.

1.4.2 Experiment 2 – Perturbation Studies

Establishing that motor readiness could alter the neck muscle reflex without prior practice did not mean that subjects actually readied a motor response in advance of whiplash-like perturbations. Magnusson et al. (1999) observed no difference in the onset, duration or amplitude of the cervical muscle response between seated subjects who were given a countdown to a perturbation (alerted) and subjects who underwent randomly-spaced perturbations (unalerted). Kumar et al. (2000), on the other hand, reported that the peak head acceleration of subjects aware of the time and intensity of an impending horizontal perturbation was about half as large as the peak head acceleration of subjects who were not aware of the exact time or intensity of the perturbation. In a recently published abstract containing muscle response data from the same experiment, Kumar et al. (2001) reported that awareness of the perturbation significantly affected the onset and amplitude of the EMG signal of various neck muscles. Unfortunately, these authors did not describe the nature of the relationship between awareness and the muscle response. Based on their previously reported kinematic response, however, it was presumed that awareness of the time and intensity of the perturbation reduced the muscle response.

Although the apparently contradictory findings of the two research groups cited above warrant additional investigation, further work was also needed to eliminate the potentially confounding effects of habituation and to study a more realistic level of awareness. Both research groups used repeated exposures to study the effect of awareness and did not report whether their subjects were habituated to the perturbation prior to acquiring their data. Including both habituated and unhabituated responses may have confounded their analyses of awareness. In addition, both Magnusson et al. (1999) and Kumar et al. (2000) only studied the effect of temporal awareness on the response of their subjects to whiplash-like perturbations. Neither group studied the effect of event awareness. Neither the alerted nor unalerted conditions studied by these two groups of researchers may have been representative of many real whiplash exposures – even during their subjects' first exposures. The intensity of the first reflexive muscle response under surprised conditions may be different from the first reflexive muscle response elicited in subjects aware of the presence of an imminent perturbation, whether or not they know its exact timing (Hypothesis 2A). To examine the effect of event awareness on the response to whiplash-like perturbations, deception was used in the current experiments to create a pre-perturbation awareness level closer to the surprised condition

present during most real collisions.

Most whiplash experiments employing human subject have used multiple exposures to similar stimuli. If the free-fall experiments of Bisdorff et al. (1994) are an indication, rapid habituation of the muscle response likely occurred as a result of these multiple exposures. Although motor readiness, similar to that being studied in Experiment 1, has been shown to reduce habituation (Valls-Solé et al., 1997), the degree of habituation to multiple, seated, horizontal perturbations and the degree to which habituation is affected by temporal awareness has not yet been quantified (Hypothesis 2B). Since the deception used to generate the surprised response in the first trial could not be repeated, habituation could only be studied in the alerted and unalerted subjects. Information regarding the habituation pattern will help to identify which human subject experiments should be used to validate future whiplash injury models and which might be less suitable. Furthermore, a better understanding of habituation to whiplash-like perturbations could provide a basis to limit the number of exposures per subject in future human subject testing.

The first two hypotheses of Experiment 2 were focused on event and temporal awareness, however, awareness of the perturbation amplitude might also affect a subject's response. Based on the experimental design used by Kumar et al. (2000), it was not possible to discern whether their reported awareness effect was due to temporal awareness or amplitude awareness. Therefore, it remains unknown whether subjects use information regarding the intensity of a perturbation to alter their muscle response (Hypothesis 2C). Unlike temporal awareness, amplitude awareness could not be studied in tandem with event awareness. Subjects could be misled regarding the amplitude of a perturbation, however, the information conveyed in the act of this deception eliminated the surprise needed to simultaneously study event awareness. Moreover, deception regarding the perturbation amplitude assumed that subjects had a baseline reference against which to compare their next perturbation. This baseline does not exist in subjects not previously exposed to a perturbation. To overcome these problems, the effect of amplitude awareness was only studied in subjects who had habituated to a baseline perturbation condition.

All of the preceding hypotheses have focused on whether subjects, based on their level of advance knowledge, could potentially alter the amplitude of their neck muscle reflex response and thereby alter the amplitude of the induced kinematic response. To put these kinematic changes into context, the variation in these same responses with stimulus magnitude was also examined. Some information regarding the stimulus-response relationship would be generated by the different levels of perturbation intensity used in the experiment to test Hypothesis 2C. More than one component of the perturbation kinematics, however, may affect the response. Recent evidence has shown that the amplitude of the induced kinematics during whiplash-like perturbations varies with both the

acceleration and velocity change of the perturbation (Boström et al., 2000, Siegmund and Heinrichs, 2001). These latter experiments were conducted using a BioRID II crash test dummy specifically designed for whiplash testing and therefore provided no information regarding the effect of acceleration and velocity change on the reflex muscle response. To date, the question of whether the neck muscle reflex response is graded to perturbation velocity or acceleration has not been quantified (Hypothesis 2D).

1.5 Testable Hypotheses

The proposed research seeks to determine how awareness and habituation affect the muscle and kinematic responses evoked by whiplash-like perturbations. Based on the rationale presented above, two experiments consisting of six hypotheses were performed. The hypotheses which were tested in each experiment are outlined below:

1.5.1 Experiment 1

1A) The cervical muscle response evoked by an acoustically startling stimulus is different in relaxed subjects than in subjects ready to execute a rapid head movement.

1B) The cervical muscle response evoked by an acoustically startling stimulus can be altered by readiness to perform a rapid head movement without that movement being practiced.

1.5.2 Experiment 2

2A) The neck muscle and peak kinematic responses of subjects exposed to a whiplash-like perturbation are different in alerted, unalerted, and surprised conditions.

2B) The neck muscle response of alerted and unalerted subjects habituates to multiple sequential whiplash-like perturbations.

2C) Prior knowledge of the intensity of a perturbation affects the neck muscle response to that perturbation.

2D) The neck muscle response is graded to both the acceleration and velocity change of a whiplash-like perturbation.

Both hypotheses of Experiment 1 were combined and are reported in Chapter 2. Hypotheses 2A through 2D were treated as separate experiments and are reported in Chapters 3 through 6 respectively.

1.6 Statement of Ethics

All of the experiments documented in this thesis were conducted in accordance with the ethical guidelines of the University of British Columbia.

CHAPTER 2 MALLEABILITY OF NECK MUSCLE REFLEXES

2.1 Introduction

Loud acoustic stimuli produce an involuntary muscle response known as the startle reflex (Landis and Hunt, 1939). Startling stimuli can generate a whole-body reflex response, however the response rapidly habituates in distal muscles and is often reduced to only an eye blink after relatively few stimuli (Landis and Hunt, 1939; Davis, 1984). Interestingly, recent studies have shown that readiness to execute a voluntary movement facilitates the startle reflex and reduces habituation in the muscles used for the voluntary movement (Valls-Solé et al., 1995; Valls-Solé et al., 1997).

This phenomenon of reduced habituation in the presence of motor readiness was recently used to study ballistic movements in the upper and lower limbs (Valls-Solé et al., 1999). These authors reported that an acoustic startle-inducing stimulus superimposed on a visual GO stimulus produced the same muscle response pattern observed in reaction-time (RT) trials, but with an onset of electromyographic (EMG) activity advanced to that of the startle reflex response. These researchers reported that the muscle response observed during startled movement trials was not simply the summation of a normal startle response and a temporally-normal voluntary reaction-time muscle response. Valls-Solé et al. (1999) also reported no extra EMG activity in the distal limb muscles during startled movement trials - an observation that suggested the muscle response during these trials was also not the sum of a normal startle and a temporally-advanced reaction-time response. Based on these findings, Valls-Solé et al. (1999) proposed that the startling stimulus had released a pre-programmed movement stored in subcortical structures.

This proposal of a pre-programmed movement triggered by a startle did not, however, explain what became of the descending startle volley. Since the startle-only responses in the distal limb muscles studied by Valls-Solé et al. (1999) were relatively small, it was possible that the addition of a normal startle response to an accelerated voluntary muscle response was too small to detect. The primary goal of the present experiment was to study further the potential summation of a startle response and a temporally advanced voluntary muscle response using the larger and more robust startle response of neck muscles (Brown et al., 1991a; Vidailhet et al., 1992). Ballistic, self-terminated head movements in flexion and axial rotation were used to examine two combinations of muscle synergies between the sternocleidomastoid (SCM) and cervical paraspinal (PARA) muscles. It was hypothesized that the amplitude of the neck muscle response would be larger during startled movement trials and that the relationship between the startled movement responses and the reaction-time responses would provide the information needed to determine whether the startle-induced

response consisted of only the triggered voluntary movement or whether it was some combination of a startle reflex and a temporally-advanced movement. A preliminary report of this study has been previously published in abstract form (Siegmund et al., 2000a).

2.2 Methods

2.2.1 Subjects

Twenty healthy subjects (9 F, 11 M) between 18 and 35 years old participated in the experiment. All subjects gave their informed consent and were paid a nominal amount for their participation. The use of human subjects for this experiment was approved by the university's Ethics Review Board and the study conformed with the Declaration of Helsinki.

2.2.2 Instrumentation

Electromyographic (EMG) activity in the orbicularis oculi (OO), masseter (MAS), sternocleidomastoid (SCM) and cervical paraspinal (PARA) muscles was recorded bilaterally using 10 mm pre-gelled surface electrodes (H59P, Kendall-LTP, Huntington Beach, CA) and an Octopus AMT-8 amplifier (Bortec, Calgary, AB). Two uniaxial accelerometers (Kistler 8302B20S1; ±20g, Amherst, NY) and a single uniaxial angular rate sensor (ATA Sensors ARS-04E; ±100 rad/s, Albuquerque, NM) were positioned at the subject's forehead. The sensitive axis of one accelerometer was oriented vertically to measure head acceleration during flexion movements and the sensitive axis of the other accelerometer was oriented mediolaterally to measure head acceleration during axial rotation movements. The angular rate sensor was reoriented appropriately between the blocked movement trials to capture both flexion and axial rotation movements. High gains (overall sensitivity of 2.9V/g) were used for the accelerometers to improve detection of movement onset (Corcos et al., 1993). These gains resulted in peak acceleration data being clipped, however, these data were used only for onset detection. A force transducer (Artech S-Beam SS20210, ±2 kN, Riverside, CA) was used to measure reaction loads during normalizing contractions of the SCM and PARA muscles. EMG signals were bandpass filtered at 10 Hz to 1 kHz and transducer signals were low-pass filtered at 1 kHz before being simultaneously sampled at 2 kHz and stored for subsequent analysis. Auditory signal magnitude was measured using a Cirrus Research CR252 sound level meter (Hunmanby, North Yorkshire, UK) at a location which coincided with the midpoint of the subjects' ears.

2.2.3 Test Procedures

Seated subjects underwent two blocks of 20 trials in which they were instructed to react as rapidly as possible to an auditory GO stimulus (76 dB, 1000 Hz, 40 ms duration) by performing a

ballistic head movement. In one block of trials, subjects flexed their head and neck forward from a neutral head position; in the other block of trials, subjects axially rotated their head to the right from a neutral head position. Half the subjects underwent flexion trials first; the other half underwent rotation trials first. The GO stimulus was preceded by an identical warning tone at randomly-varying foreperiods uniformly-distributed between 1.5 and 3.5 seconds. The time between trials was 15 seconds and a rest period of about 3 minutes was used between blocks. Subjects received qualitative verbal feedback and enthusiastic encouragement between trials.

Subjects were not permitted to practice either motion prior to the experiment. Immediately preceding a block of trials, the experimenter described and demonstrated the desired movement to the subject and then passively moved the subject's head from the neutral position to an approximate endpoint and back to the neutral position. Subjects were then instructed to visualize and practice the movement mentally without actually moving. Targets were provided to assist the subjects with moving through about 45 degrees of head rotation, although subjects were instructed to focus on rapidly initiating and executing the prescribed movement rather than on endpoint repeatability. On trials 1, 4, 8, 11, 12, 15 and 20 of each block, the GO stimulus was replaced by a startle-inducing stimulus (124dB, 1000 Hz, 40 ms). The warning tone was unaltered. Trials in which the subject received the GO stimulus were designated reaction time trials (RT trials) and trials in which the startling stimulus replaced the GO stimulus were designated startled movement trials (ST trials). In addition to the two blocks of twenty trials for each movement, three startle-only control trials (CT trials) were administered: one before, one between and one after the two blocks of movement trials. For the startle-only control trials, subjects were relaxed, i.e., not ready to move, and the startling stimuli were presented without warning stimuli.

After completion of the above protocol, seated subjects performed sub-maximal isometric contractions in flexion and extension to generate normalizing data for the SCM and PARA muscles respectively. A strap attached to the load cell was placed around a subject's head and its length adjusted to ensure the subject's head was neutrally positioned. The strap was located immediately above the glabella for flexion contractions and at the height of the external occipital protuberance for extension contractions. Subjects were instructed to generate a force of 25 N with visual feedback, first in flexion and then in extension. EMG and load cell data were acquired for 5 s during each contraction.

2.2.4 Data Reduction

The onset of head movement was determined directly from the accelerometer data using the algorithm developed in Appendix A. Peak angular velocity (ω_{max}) of the movement was determined

directly from the angular rate sensor data after the raw data had been digitally compensated to reduce the sensor's high-pass frequency to 0.002 Hz (Laughlin, 1998). Angular acceleration was computed by finite differences (5 ms window) from the compensated angular velocity data and its peak (α_{max}) was determined. Total head angular displacement (θ_{max}) was computed by integrating the compensated angular velocity. The time at which each of the three angular kinematic parameters reached a maximum was also determined and the relative timing between these three maxima was used to evaluate whether the responses in the ST trials and RT trials were temporally similar. The acceleration interval was defined as the time between acceleration onset and peak angular velocity (ω_{max}). The time between peak angular velocity and peak angle (θ_{max}) was used to represent the deceleration interval because some subjects continued to negatively accelerate for a considerable period after reaching their peak angular displacement.

EMG onset times were determined using a double-threshold detector (Bonato et al., 1998) and then confirmed visually. For each muscle, the root mean squared (RMS) amplitude of the EMG was calculated over the acceleration interval for movement trials. The kinematics could not be used to define a comparable interval for control trials because little or no movement occurred. Therefore, the average duration of the acceleration interval for all movement trials was used to compute the RMS amplitude of the EMG for the first control trial of each subject. The SCM and PARA muscle EMG amplitudes were normalized by the RMS amplitude obtained during the 5 s sub-maximal contraction for the corresponding muscle. Entire trials were rejected if movement preceded the stimulus or if movement did not occur within 200 ms of stimulus onset. Data from individual muscles within an accepted trial were rejected if the muscle was active within 20 ms of stimulus onset, if onset was absent, or if onset was ambiguous.

Ratios and arithmetic differences were then computed from the EMG amplitude and onset latency data obtained from the left and right neck muscles under the different stimuli and movement conditions. From the EMG amplitude data, ST/RT ratios were computed by dividing the EMG amplitude observed in the ST trials by the EMG amplitude observed in the RT trials. For each subject, a separate ST/RT ratio was calculated for each of the four neck muscles in each of the two movement conditions (eight ratios per subject). Eight matching RT-ST differences were computed by subtracting the EMG amplitude of the ST trials from the EMG amplitude of the RT trials. A comparison between the ST/RT ratios and RT-ST differences in the different neck muscles and movement conditions was then used to evaluate whether the EMG amplitude observed during ST trials was a scaled or biased version of the EMG amplitude observed during RT trials. If the EMG amplitude observed during ST trials was a scaled version of that observed during RT trials, then similar ST/RT ratios would be expected in all muscles and movement conditions. If instead the EMG amplitude observed during ST

trials was biased up or down relative to that observed during RT trials, then similar ST-RT differences would be expected in all muscles and movement conditions.

The expected bilateral asymmetry in neck muscle activity during rotation trials provided an opportunity to compare left and right muscle activity using the same technique. For these comparisons, left/right (L/R) ratios of EMG amplitude in the left and right muscles of each functional neck muscle pair were computed for each stimulus condition and each movement direction (eight ratios per subject). Eight matching L-R differences in the EMG amplitude were also computed. As before, a comparison between these L/R ratios and L-R differences was used to evaluate whether bilateral differences in the EMG amplitude observed during ST trials were scaled or biased versions of the EMG amplitude observed during RT trials.

In addition to EMG amplitude, ST/RT ratios, ST-RT differences, L/R ratios and L-R differences was also computed from the onset latency data. A comparison between the ST and RT ratios and differences was used to evaluate whether the shortened onset latency observed in the ST trials was scaled forward in time or biased forward in time relative to the onset latency observed in RT trials. A comparison between the left and right ratios and differences was used to evaluate whether bilateral differences in onset latencies observed during ST trials were scaled or biased versions of the bilateral differences observed during RT trials.

2.2.5 Statistical Analysis

Prior to statistical comparisons, separate within-subject means were calculated for the dependent variables in the reaction-time trials and startled movement trials. For each kinematic variable, a two-way repeated-measures analysis of variance (ANOVA) was used to assess differences related to stimulus type (RT, ST) and movement direction (flexion, rotation). For EMG onset times and amplitudes, a three-way repeated-measures ANOVA for stimulus type, movement direction and muscle side (left, right) was used. Prior to statistical analysis, the reaction-time data were checked to ensure they were normally distributed using a Kolmogorov-Smirnov one-sample test. Separate three-way ANOVAs were used for the SCM and PARA muscles. Differences in the onset latencies of both neck muscles and the onset of head acceleration between the RT, ST and CT trials were compared with a one-way repeated-measures ANOVA. For these latter analyses, post-hoc comparisons were performed using a Scheffé test.

Each of the ratios and differences computed from the onset latencies and EMG amplitudes were analyzed separately for each movement direction. For each ST/RT ratio or difference, a two-way, repeated-measures ANOVA for muscle (SCM, PARA) and muscle side (left, right) was used. For each left/right ratio or difference, a two-way, repeated-measures ANOVA for muscle (SCM,

PARA) and stimulus type (ST, RT) was used. A qualitative comparison between the results of the analyses of all ratios and differences was then made to interpret the overall relationship of the ST muscle response to the RT muscle response. A three-way repeated-measures ANOVA was also used to compare the EMG amplitude observed in the control trials to the difference in EMG amplitude observed between the ST and RT trials. The three factors in this analysis were muscle (SCM, PARA), side (left, right) and movement direction (flexion, rotation and control). All statistical tests were performed using Statistica (v.5.1, Statsoft Inc., Tulsa, OK) and a significance level of α=0.05.

2.3 Results

Muscle activity was observed in the first control trial of all subjects (Figure 2.1A). Responses to the latter two control stimuli were typically diminished and in about 10 percent of these latter trials only the OO response remained intact (Figure 2.1B). Within the flexion and rotation blocks, rejected trials reduced the average number of ST trials per subject from 7 to 6.75 ± 0.26 per block and the average number of RT trials per subject from 13 to 9.1 ± 1.5 per block. All of the ST trial rejections and a small number of RT trial rejections were due to pre-stimulus movement; the remaining RT trial rejections were due to prolonged (>200 ms) response times. Within accepted trials, the SCM muscles were individually rejected once and the PARA muscles were individually rejected eight times in 800 trials. Each individual rejection was due to an ambiguous onset time.

2.3.1 Kinematic Response

The timing and amplitude of the head kinematics varied with both stimulus type and movement direction (Table 2.1). Head acceleration onset and peak angular head acceleration (α_{max}), velocity (ω_{max}) and displacement (θ_{max}) all occurred earlier during ST than RT trials. The peak magnitudes of all three measures of angular head kinematics were also larger during ST than RT trials. Consistent with these differences in kinematics, subjects qualitatively described their movements during ST trials as being assisted by something in addition to their own will.

Overall, the duration of the head acceleration interval was longer during ST than RT trials, however a similar stimulus effect was not observed in the duration of the deceleration interval (Table 2.1). When the acceleration interval was examined more closely, however, a different pattern emerged. For flexion movements only, the time between acceleration onset and α_{max} increased from 88 ± 27 ms for RT trials to 122 ± 15 ms for ST trials (post-hoc $p<0.0001$) and the time between α_{max} and ω_{max} decreased from 78 ± 15 ms for RT trials to 66 ± 21 ms for ST trials ($p<0.01$). No stimulus effect was observed in the sub-components of the acceleration interval for rotation movements.

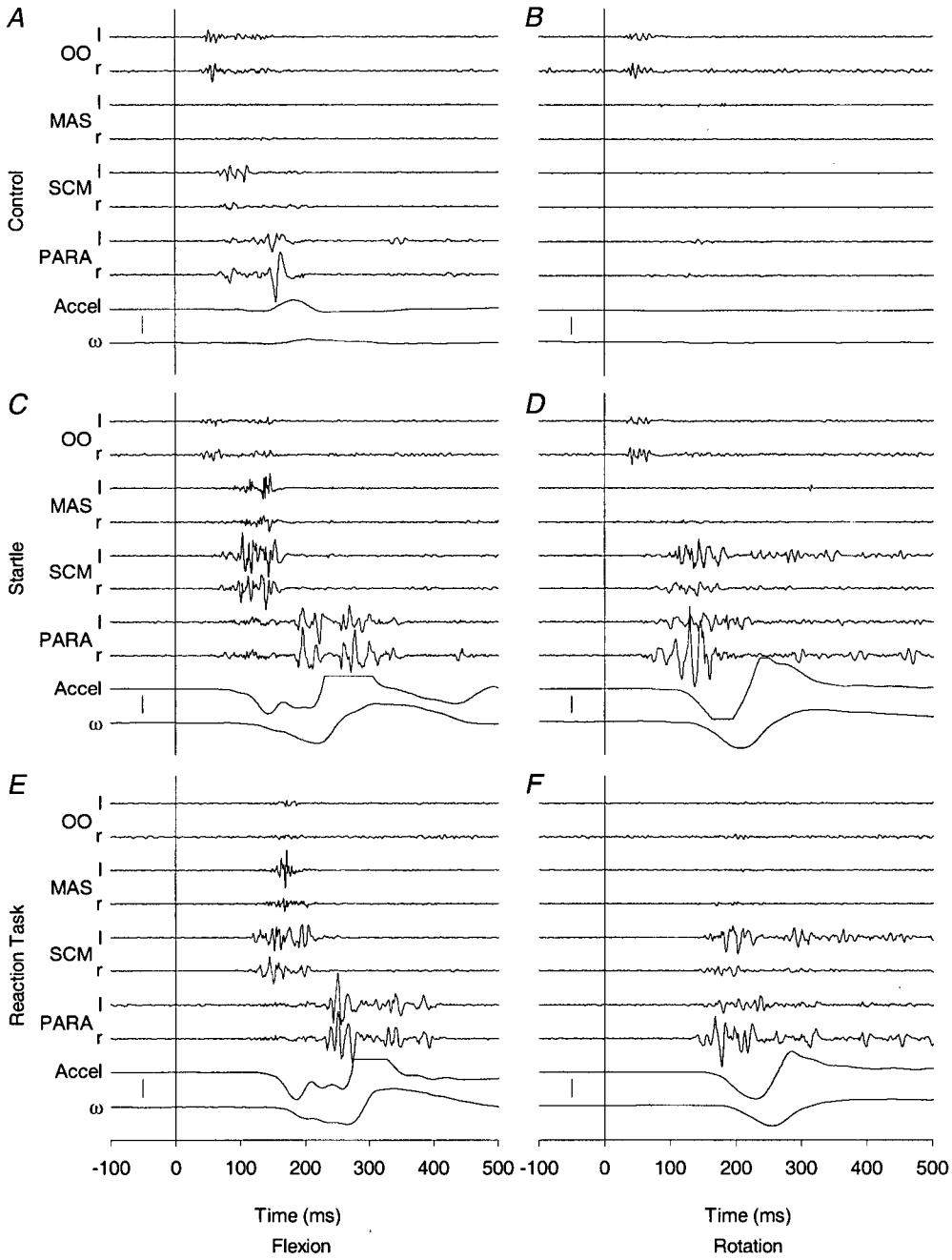


Figure 2.1 Sample EMG recordings from the control, startle and reaction time trials of a single subject. *A*, EMG recordings from the first control trial. *B*, EMG recordings from the second control trial, administered between the flexion and rotation blocks. *C*, EMG recordings from a startle trial in which the subject was ready to perform a ballistic flexion movement. *D*, EMG recordings from a startle trial in which the subject was ready to perform a ballistic axial rotation movement. *E*, EMG recordings from a reaction time trial for a flexion movement. *F*, EMG recordings from a reaction time trial for an axial rotation movement. The vertical bar between the Accel and ω traces is equivalent to 1g and 5 rad/s. OO, orbicularis oculi; MAS, masseter; SCM, sternocleidomastoid; PARA, cervical paraspinal muscles, l, left; r, right; Accel, linear head acceleration at the forehead; ω, angular velocity of the head. The vertical line through all traces of a single trial indicates the onset of either the GO or startling tone.

Table 2.1 Mean (S.D.) of head kinematics as a function of stimulus and motion direction. Upper portion of table summarizes data as a function of motion direction (control trials, flexion trials, rotation trials) and stimulus intensity (startle tone, reaction time tone). Lower portion of table summarizes the results (F-statistics, df=1,19) of seven separate 2-way repeated-measures ANOVAs using motion direction and stimulus intensity as independent variables. Control data were not used in these analyses. Accel, acceleration; Decel, deceleration; α, head angular acceleration; ω, head angular velocity; θ, head angle; max, maximum; CT, control trial; ST, startle trial; RT, reaction time trial.

Description		Time (ms)				Duration (ms)		Magnitude		
		Accel. onset	α_{max}	ω_{max}	θ_{max}	Accel.	Decel.	α_{max} (rad/s^2)	ω_{max} (rad/s)	θ_{max} (deg)
Control	CT	58 (12)								
Flexion trials	ST	55 (10)	177 (18)	243 (21)	364 (56)	187 (17)	122 (53)	106 (33)	6.8 (1.6)	43 (12)
	RT	127 (25)	215 (32)	292 (29)	409 (43)	166 (23)	117 (44)	86 (25)	5.7 (1.6)	38 (11)
Rotation trials	ST	64 (7)	162 (17)	219 (20)	332 (46)	155 (20)	113 (39)	169 (59)	9.0 (2.5)	54 (12)
	RT	129 (27)	217 (31)	273 (34)	391 (54)	144 (26)	118 (39)	140 (54)	7.8 (2.0)	48 (10)
ANOVA F-statistics										
stimulus (ST/RT)		188****	97.8****	123****	46.5****	10.8**		30.7****	30.3****	14.2**
motion (flex/rot)		5.64*		16.6***	4.91*	46.0****		33.9****	44.8****	42.8****
stimulus x motion			8.58**							

* p<0.05, ** p<0.01, *** p<0.001, ****p<0.0001

When the head acceleration onset times during the three different trial conditions with startle tones (CT, flexion ST and rotation ST) were compared, a significant difference was present ($F_{2,36}$=6.4, p=0.004) (Table 2.1). Post-hoc analysis showed that the onset of head acceleration occurred earlier during flexion ST trials than rotation ST trials; differences between the other two combinations of conditions were not significant.

2.3.2 EMG Timing

The temporal pattern of neck muscle EMG in individual ST trials was visibly advanced compared to RT trials (Figure 2.1,C-F). For both movements, the onset latencies of the SCM and PARA muscles during ST trials were significantly shorter and exhibited less variation (Table 2.2, Figure 2.2A, B). Mean onset latencies during ST trials were between 42 and 51 percent of their respective onset latencies during RT trials (Figure 2.2C). The shorter onset latencies during ST trials were neither uniformly scaled in time nor uniformly biased forward in time relative to the onset latencies during RT trials. Within flexion movements, the SCM and PARA muscles were advanced by significantly different proportions ($F_{1,19}$=9.7, p=0.006, Figure 2.2C). The arithmetic difference in onset latencies between the ST and RT trials was also significantly different for the SCM and PARA muscles during flexion movements ($F_{1,19}$=29.7, p<0.0001) (Figure 2.2D).

SCM activation during RT trials occurred earlier in flexion than rotation ($F_{1,19}$=6.9, p=0.017), whereas PARA activation occurred later in flexion than rotation ($F_{1,19}$=8.2, p=0.010) (Figure 2.2A, B). These movement-related differences in activation times were not present during ST trials. For each muscle, the onset latencies for flexion and rotation movements during ST trials were not significantly different from each other or the CT trials.

A small but significant bilateral asymmetry was present in the neck muscle activation sequence during rotation trials (Table 2.2, Figure 2.2A,B). The right SCM and right PARA muscles were active 10 ± 14 percent earlier than their left counterparts ($F_{1,19}$=25.5, p<0.0001) and this relative timing was not significantly different between the ST and RT conditions (Figure 2.2E). The arithmetic difference between the activation time of the right and left muscles between the ST and RT conditions, however, was significantly different ($F_{1,19}$=11.4, p=0.003, Figure 2.2F).

2.3.3 EMG Amplitude

During the acceleration portion of the head motion, the RMS amplitude of the normalized EMG was larger during ST than RT trials for both muscles during both types of movements (Table 2.2, Figure 2.3A, B). EMG amplitude was bilaterally symmetrical for all flexion trials, but bilaterally asymmetrical for all rotation trials. For both ST and RT trials during rotation, the EMG

amplitude

Table 2.2 Mean (S.D.) of muscle activation time and normalized EMG amplitude for the sternocleidomastoid and cervical paraspinal muscles. Upper portion of table summarizes data as a function of muscle (SCM, PARA), side (left, right), motion direction (control trials, flexion trials, rotation trials) and stimulus intensity (startle tone, reaction time tone). Lower portion of table summarizes the results (F-statistics, df=1,19) of four separate 3-way repeated-measures ANOVAs using muscle side, motion direction and stimulus intensity as independent variables. Control data were not used in these analyses. Each statistical result is centered below its source data. SCM, sternocleidomastoid muscles; PARA, cervical paraspinal muscles; L, left; R, right; CT, control trial, ST, startle trial; RT, reaction time trial.

		Muscle activation time (ms)				Normalized EMG amplitude			
		SCM		PARA		SCM		PARA	
Description		L	R	L	R	L	R	L	R
Control	CT	56 (13)	55 (13)	66 (23)	64 (23)	2.8 (2.1)	2.6 (2.1)	4.2 (3.4)	4.3 (3.5)
Flexion trials	ST	52 (12)	52 (12)	59 (11)	60 (14)	4.3 (2.1)	3.8 (1.4)	2.9 (1.9)	3.1 (2.3)
	RT	107 (28)	107 (25)	141 (31)	140 (30)	2.9 (1.7)	2.5 (1.3)	1.7 (0.7)	1.8 (0.7)
Rotation trials	ST	52 (8)	49 (7)	58 (11)	55 (9)	4.4 (2.1)	2.4 (1.5)	3.5 (1.7)	7.3 (3.1)
	RT	123 (32)	116 (28)	131 (29)	120 (28)	3.4 (1.9)	1.2 (0.9)	2.3 (1.1)	5.9 (2.4)
ANOVA F-statistics									
side (L/R)		8.66**				19.7***		64.2****	
motion (flex/rot)		5.16*		11.0**		7.23*		64.9****	
stimulus (ST/RT)		145****		267****		103****		25.2****	
side × motion		20.7***		10.3**		73.8****		60.4****	
side × stimulus									
motion × stimulus		7.07*		4.98*					
side × motion × stim									

* p<0.05, ** p<0.01, *** p<0.001, ****p<0.0001

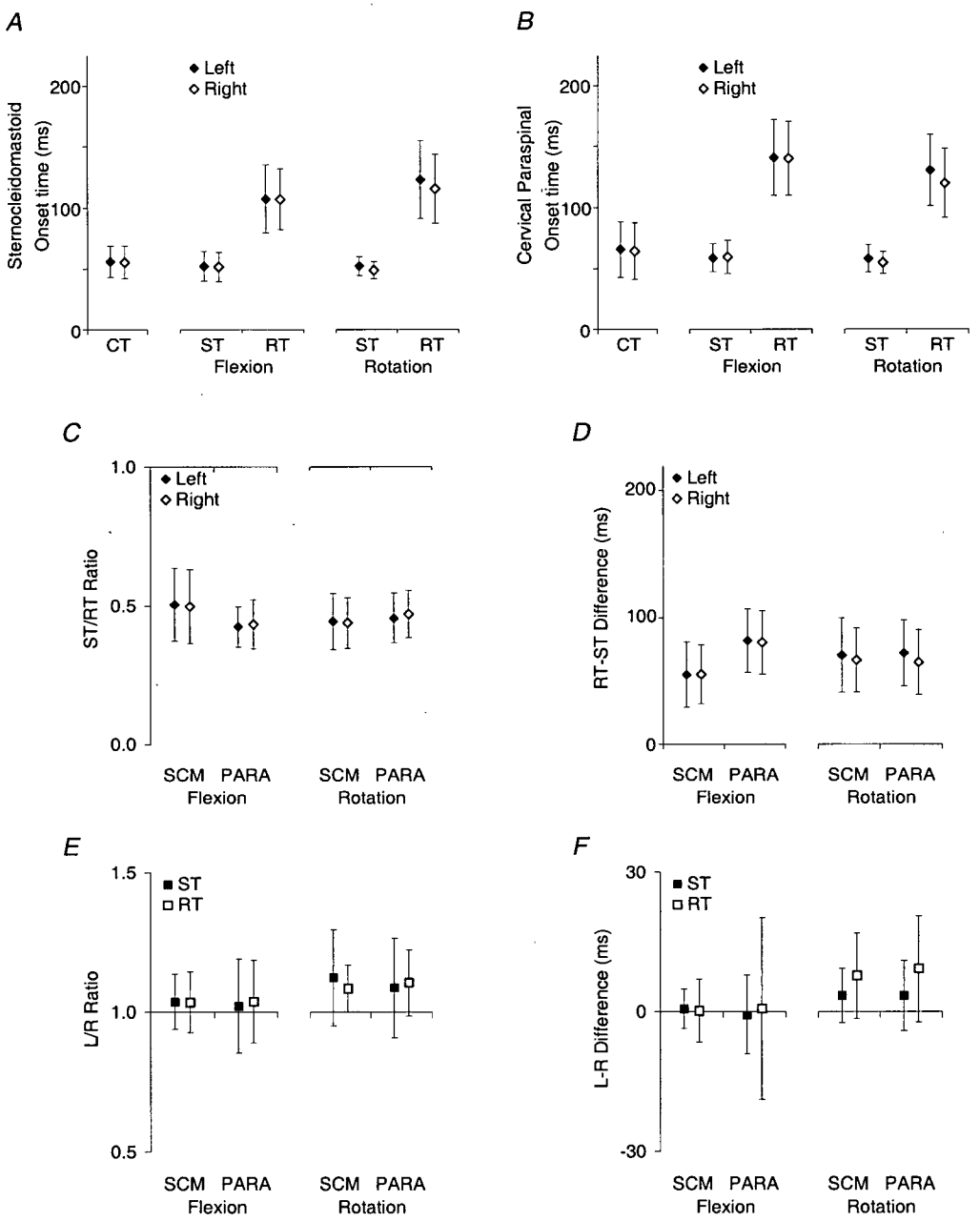


Figure 2.2 Muscle activation times, ratios and differences for the neck muscles of all subjects. *A*, Mean onset times ± 1 S.D. for the left and right sternocleidomastoid muscles during control, flexion and rotation trials. Note that onset times during control and startle trials were significantly faster than onset times for reaction-time trials. *B*, Similar to previous panel except for the cervical paraspinal muscles. *C*, mean ratio ± 1 S.D. of the ST onset time to the RT onset time (ST/RT) for each muscle as a function of muscle side (L, R) and movement type (flexion, rotation). *D*, mean arithmetic difference ± 1 S.D. of the ST and RT onset times (RT-ST) for each muscle as a function of muscle side and movement type. *E*, mean ratio ± 1 S.D. of the left to right onset latency (L/R) for each functional muscle pair as a function of stimulus (ST, RT) and movement type. *F*, mean arithmetic difference ± 1 S.D. of the left and right onset times (L-R) for each functional muscle pair as a function of stimulus and movement type.

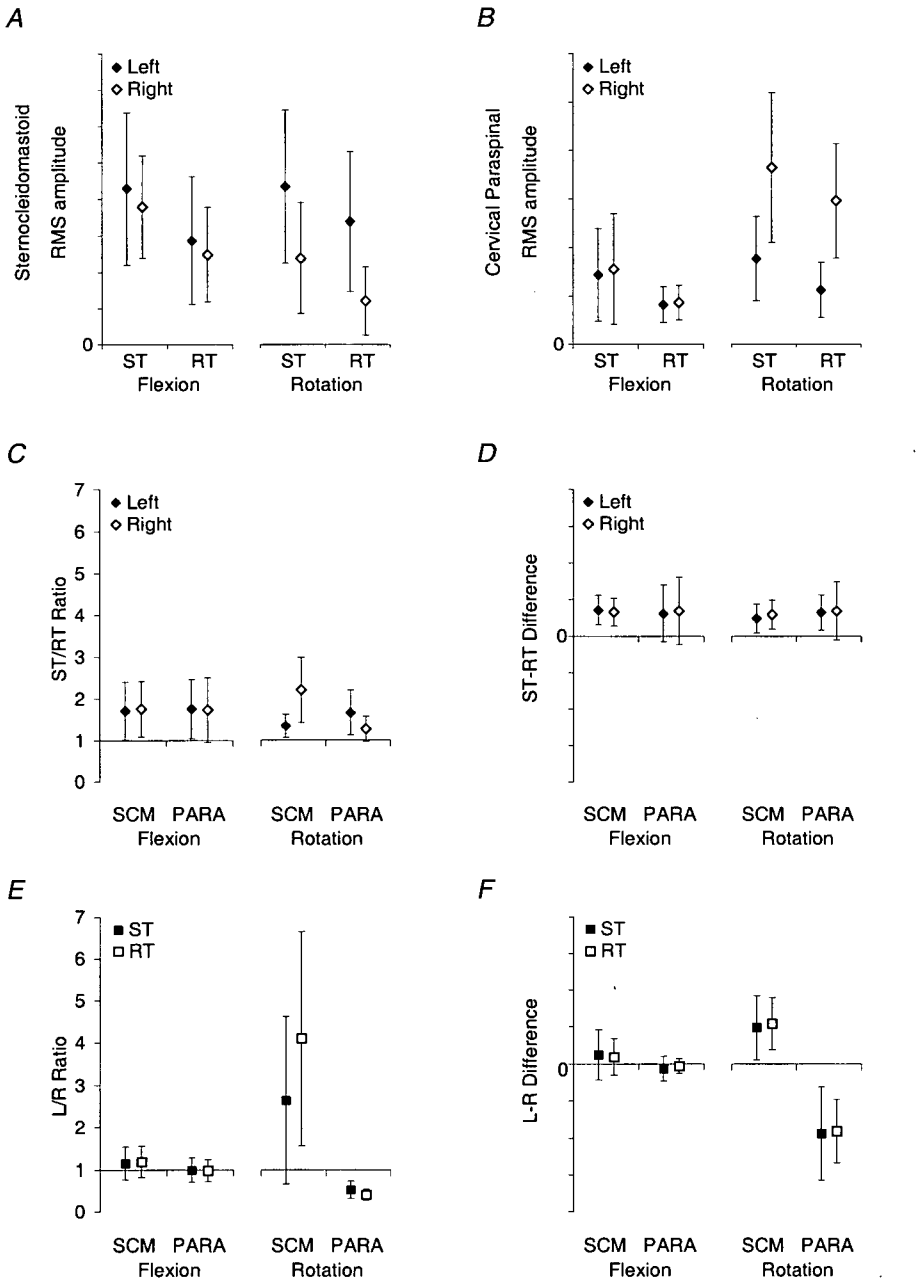


Figure 2.3 EMG amplitudes, ratios and differences for the neck muscles of all subjects. *A*, Mean normalized RMS EMG amplitude ± 1 S.D. of the left and right sternocleidomastoid muscles as a function of stimulus (ST, RT) and movement type (flexion, rotation). *B*, Similar to previous panel except for the cervical paraspinal muscles. Note the bilateral symmetry during flexion movements and bilateral asymmetry during rotation movements. *C*, mean ratio ± 1 S.D. of the ST amplitude to the RT amplitude (ST/RT) for each muscle as a function of muscle side (L, R) and movement type. *D*, mean arithmetic difference ± 1 S.D. of the ST and RT amplitudes (ST-RT) for each muscle as a function of muscle side and movement type. Note the consistent upward bias present in the startle trials. *E*, mean ratio ± 1 S.D. of the left to right amplitudes (L/R) for each functional muscle pair as a function of stimulus and movement type. *F*, mean arithmetic difference ± 1 S.D. of the left and right amplitudes (L-R) for each functional muscle pair as a function of stimulus and movement type.

was larger for the left SCM muscle than the right SCM muscle, whereas for the PARA muscles this pattern was reversed. The proportional increase in EMG amplitude between the RT and ST trials varied between muscles and side of muscle during rotation movements (Figure 2.3C), whereas the bias in EMG varied with neither parameter during either flexion or rotation movements (Figure 2.3D). The EMG amplitude of the left and right muscles appeared to be bias upward by a similar amount in both movements. This uniform upward bias implied that the difference in EMG amplitude between the left and right muscles would also be similar between stimulus conditions, and a comparison of the ratios and differences of the left and right EMG amplitudes confirmed that the RT movement appeared to be preserved atop the upward bias in EMG amplitude present in the ST trials (Figure 2.3E, F). A comparison between the control trial EMG amplitude and the amount of the upward bias between the RT and ST trials for each pair of neck muscles revealed that they were significantly different ($F_{2,38}$=16.0, $p<0.0001$) (Table 2.2). The amplitude of the control trials varied between 10 and 900 percent of the upward bias between the ST and RT trials.

2.3.4 Habituation

Neither muscle activation time, EMG amplitude nor peak angular head kinematics changed significantly with repeated exposure to startle in the movement trials (Figure 2.4). This absence of habituation was observed in both blocks of trials, and therefore normalized data from the first and second blocks were pooled for Figure 2.4. Despite the absence of habituation in startled movement trials, large and in some cases complete habituation of the neck muscle response was observed in the startle-only control trials between and after the movement blocks (Figure 2.1A, B).

2.4 Discussion

A loud acoustic stimulus capable of producing a startle reflex shortens the time to muscle activation in subjects ready to execute a simple reaction-time task. Using this technique, Valls-Solé et al. (1999) observed that the EMG amplitude of a startle-induced muscle response in distal limb muscles was not different from the EMG amplitude of the reaction-time muscle response. Based on this finding, these authors discounted a summation of the startle reflex and pre-programmed movement, and instead proposed that the startling stimulus triggered the release of a pre-programmed movement stored in subcortical structures. This proposal did not, however, explain what became of the descending startle volley.

In the current study, this same technique was used to study the startle-induced response of neck muscles ready to execute ballistic head movements. Neck muscles were selected because they have a larger startle response (Brown et al., 1991a; Vidailhet et al., 1992) and might therefore be

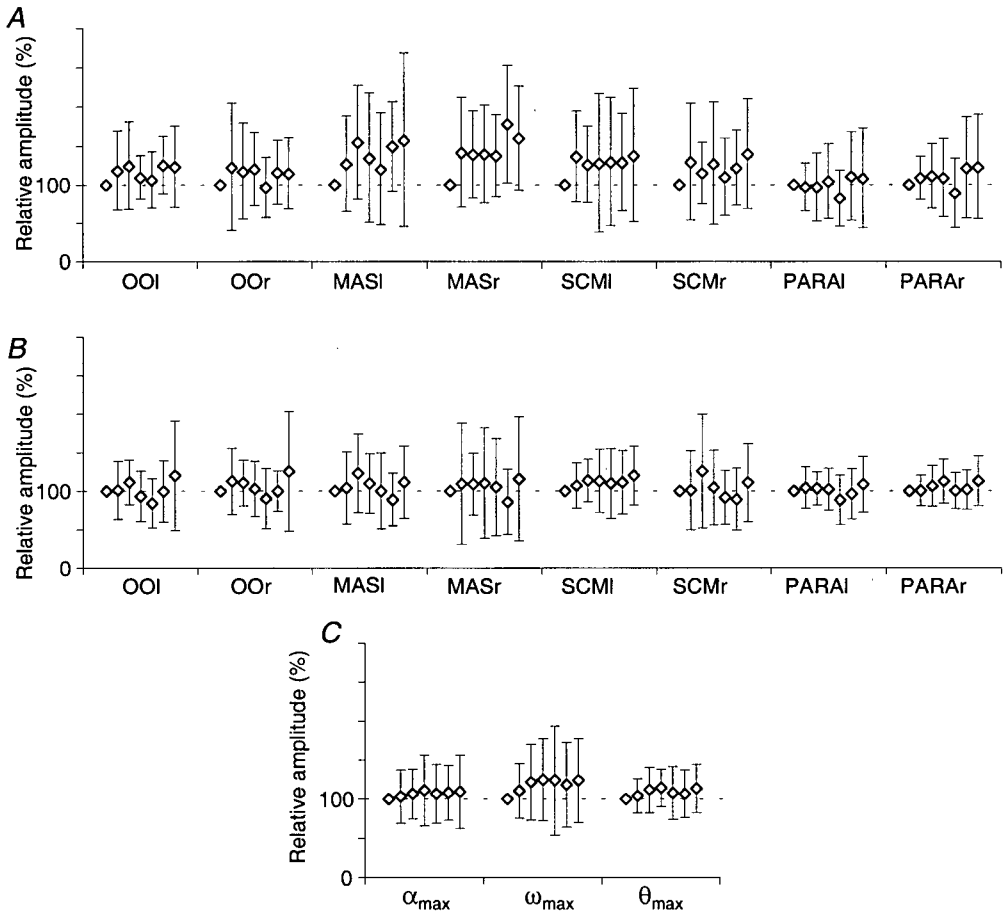


Figure 2.4 Absence of habituation to startle during sequential trials. *A*, Mean EMG amplitude ± 1 S.D. of all muscles over the seven sequential startle trials during the flexion block. The EMG amplitude of each one of the subject's muscles was first expressed as a percentage of amplitude observed in that muscle during the first trial and then the mean was calculated. Note the absence of habituation between the first startle trial (the first trial of a block) and the seventh startle trial (the 20th trial within a block). *B*, Similar to previous panel, but for rotation movements. *C*, Mean amplitude ± 1 S.D. of similarly normalized angular head kinematics. OO, orbicularis oculi; MAS, masseter; SCM, sternocleidomastoid; PARA, cervical paraspinal muscles, l, left; r, right; α, head angular acceleration; ω, head angular velocity; θ, head angle.

better candidates with which to study the potential summation of startle and reaction-time muscle responses. Two head movements, flexion and axial rotation, were used so that the within-muscle effects of startle could be examined in different muscle synergies during otherwise similar states of readiness. It was thought that a comparison of the muscle response between these two movements would provide additional information with which to evaluate whether the muscle response produced by the startling stimulus was a temporally-advanced, but otherwise unaltered, version of the reaction-time muscle response, or the summation of a startle response and a temporally-advanced reaction-time muscle response.

2.4.1 Muscle Response

The onset of neck muscle EMG activity in the current study occurred earlier in startled movement trials than in reaction-time trials. Compared to RT trials, the onset of the response in the different neck muscles during ST trials was neither proportionally scaled forward in time nor biased forward in time (Figure 2.2C, D). Instead, activation of the SCM and PARA muscles during ST trials appeared to be aligned with activation of these muscles during the startle-only control trials. Therefore, the onset of EMG activity in the neck muscles during the ST trials was indistinguishable from and consistent with the leading edge of the descending startle volley.

The amplitude of the neck muscle response in the current study was larger in ST trials than in RT trials (Figure 2.1 and Figure 2.3A, B). This increased amplitude was inconsistent with the acoustic startle reflex only releasing a pre-programmed movement resident in the brainstem and suggested that some type of interaction between the startle reflex and prepared movement had occurred. One possible interaction was a summing of the startle reflex and the movement; another possible interaction was a scaling of the movement with the intensity of the acoustic stimulus. A comparison between the ratios and arithmetic differences of the EMG amplitude from the ST and RT trials indicated that the larger muscle response during ST trials was due to an upward bias in the EMG amplitude rather than a proportional upward scaling of the EMG amplitude (Figure 2.3C, D). This bilaterally-symmetrical and movement-independent increase in EMG amplitude for both neck muscle groups suggested that the muscle response during ST trials was not just a prepared movement released by the acoustic startle reflex, but rather the summation of a temporally-advanced movement and a generalized neck muscle activation due to the startle reflex.

The apparent summation of a startle reflex and a pre-programmed reaction-time movement was also examined by comparing the EMG amplitude in the control trials to the magnitude of the upward bias observed between the reaction-time and startled movement trials. This analysis revealed that the upward bias was unrelated to the magnitude of the muscle response in the startle-only control

trials. The results of such a comparison, however, must be considered cautiously because the level of baseline readiness in the unwarned startle-only control trials was not the same as the level of readiness in the forewarned startled movement trials. In contrast, the level of motor readiness in the startled movement trials of the flexion and rotation movement blocks was likely similar and therefore a comparison between the startle-induced increase in EMG amplitude between the two different movements was preferred. Though needing cautious interpretation, the comparison between the EMG amplitude of the control trials and upward bias between RT and ST trials did demonstrate that the startle reflex could generate sufficient EMG amplitude to account for the upward bias observed in the startled movement trials.

Increased EMG amplitude was not reported by Valls-Solé et al. (1999) in the distal limb muscles they studied. One possible explanation for the difference between studies is the more variable and less robust startle response in distal limb muscles than in neck muscles (Brown et al., 1991a; Chokroverty et al., 1992; Vidailhet et al., 1992). The superposition of a small startle-related bias on a comparatively large movement-related distal muscle response may not have been qualitatively detectable by Valls-Solé et al. (1999). Another potential explanation for the difference between studies is the variable foreperiod used in the present study and the fixed foreperiod used by Valls-Solé et al. (1999). This protocol difference may have produced differing levels of preparatory activity in the cortex, brainstem and spinal cord and the specific state of this preparatory activity may have affected the startle-induced muscle response. A third possible explanation for the difference between studies lies in the brainstem circuits mediating the acoustic startle reflex and is developed more fully below.

A number of different pathways for the mammalian acoustic startle reflex have been proposed (see summary in Yeomans and Frankland, 1996). All of the proposed pathways include an initial synapse in the cochlear nucleus, which then either monosynaptically or disynaptically, via neurons in or near the lateral lemniscus, terminate in midbrain reticular nuclei. The axons of the reticular nuclei then synapse either directly, or indirectly via spinal interneurones, onto spinal motoneurones. Giant neurons in the nucleus reticularis pontis caudalis (nRPC) are thought to be the sensorimotor interface of the startle reflex (Wu et al., 1988; Lingenhöhl and Friauf, 1994; Koch, 1999). Large-diameter descending axons from these giant neurons have both sufficiently diffuse and multi-segmental spinal connections (Lingenhöhl and Friauf, 1992; Lingenhöhl and Friauf, 1994) and sufficiently high conduction velocities (Wu et al., 1988; Lingenhöhl and Friauf, 1994) to be strong candidates for carrying a descending startle volley. Corticoreticular fibres from the primary motor cortex and pre-motor area also terminate in the vicinity of the reticular nuclei and may provide the reticular nuclei with sufficient information of the impending movement for the reticulospinal fibres to

modulate reflex actions and to coordinate posture and movement (Matsuyama and Drew, 1997; Kably and Drew, 1998).

Based on their observation of an accelerated motor programme without increased EMG or movement amplitude, Valls-Solé et al. (1999) proposed that sufficient detail of the planned movement might be stored in the brainstem and spinal cord so that the movement could be triggered by the same reticular structures responsible for the startle reflex. Moreover, these authors suggested that the reticulospinal system might be an important response channel for ballistic reaction-time tasks. Both proposals are consistent with the startle pathways described above. In the present study, however, EMG amplitude was larger in ST trials than in RT trials, and the increase in EMG amplitude consisted of an upward bias that was seemingly independent of the EMG amplitude present during the voluntary movement. This bias was difficult to reconcile with a single descending pathway and suggested that parallel pathways might be responsible.

Pellet (1990) has shown that the head and neck startle may be mediated slightly differently than startle in the limbs. Pellet (1990) observed that another reticular structure, the nucleus reticularis gigantocellularis (nRG), has monosynaptic connections with the neck muscle motoneurones and may be excited independently from the nRPC during startle. Moreover, axonal branches from acoustically-driven neurons in the nRPC terminate on neurons in the nRG (Lingenhöhl and Friauf, 1994). Pellet (1990) proposed that parallel pathways between the cochlear nuclei and the neck muscle motoneurones via the nRPC and nRG might mediate different components of the startle reflex in the head and neck. Such a parallel arrangement might explain a muscle response which simultaneously consists of a bilaterally-uniform increase in neck EMG amplitude, perhaps mediated through one of the reticular nuclei, and a temporally advanced version of the reaction-time movement, perhaps mediated by pre-movement facilitation or inhibition through the other reticular nucleus. Therefore, differences in the neuroanatomical pathways for the startle reflex of the neck and limb muscles may explain why increased EMG amplitude was observed in the present study using neck muscles but not observed previously in distal limb muscles (Valls-Solé et al., 1999).

2.4.2 Kinematic Response

Like the muscle response, the peak head kinematics occurred earlier and were of greater magnitude in ST trials than in RT trials (Table 2.1). Once initiated, however, the temporal aspects of the movements observed in the ST and RT trials were remarkably similar. No differences in the relative timing of acceleration onset and peak angular head kinematics were observed between the ST and RT trials involving the rotation movement. For the flexion movement, differences between the ST and RT trials were present only during the acceleration interval. Within this acceleration interval,

two contrary effects were observed. The sub-interval between acceleration onset and peak angular acceleration was longer in flexion ST trials than in flexion RT trials, and the sub-interval between peak angular acceleration and peak angular velocity was shorter in flexion ST trials than in flexion RT trials. The reason for this pattern and why it appeared only in the flexion movement is not known, but it may be related to a flexor bias observed by some in the startle reflex (Landis and Hunt, 1939; Davis, 1984).

Although the analysis of EMG amplitude suggested that the RT movement was preserved atop the startle-induced bias in ST trials, the movement kinematics were larger in ST trials than in RT trials. These kinematic differences indicated that the forces generated by the muscles were larger during ST trials than during RT trials, even though the difference in the amplitude of the EMG measured from these muscles remained the same (Figure 2.3F). The reason for this apparent discrepancy between the muscle and kinematic responses is not known, however, factors which might have contributed to this phenomenon are temporal summation due to possible differences in the rate of muscle activation, or the recruitment of different or additional motor units during ST trials.

2.4.3 Habituation

An unexpected finding in the present study was the absence of habituation in all four muscles during startled movement trials over the 15-minute interval required for both blocks of trials (Figure 2.4). This finding contrasted sharply with the clear habituation observed over the three control trials placed before, between and after the two movement blocks (Figure 2.1). This difference in habituation suggested that readiness to move facilitated the startle reflex. Moreover, since the first startled movement trial within each movement block was only preceded by mental preparation for that movement, practice was not needed for this readiness to facilitate the startle-induced muscle response.

Reduced habituation to startle has previously been reported in both MAS and SCM using acoustic startle superimposed on a visual GO stimulus in an upper limb reaction-time task (Valls-Solé et al., 1997; Valldeoriola et al., 1998). The difference in habituation rates, namely the absence of habituation in the present study compared to the reduced habituation in the previous studies, might be explained by differences in subject readiness. Readiness to perform a voluntary RT task has been modeled using separate facilitated motor and sensory systems (Silverstein et al., 1981; Brunia, 1993). The motor preparation aspects of the current reaction-time task were similar to previous studies (Valls-Solé et al., 1997), although the involvement of the SCM and MAS muscles was different. SCM was a prime mover in the current study and MAS may have helped stabilize the jaw during the rapid head movements. These muscles were likely not involved in the limb movements used by Valls-

Solé et al., (1997). Sensory facilitation in the current study, however, was likely quite different from these previous studies. In the current study, the warning, GO and startling stimuli were in the same modality and therefore a facilitated auditory system may have generated a large afferent signal. In contrast, previous studies (Valls-Solé et al., 1997; Valldeoriola et al., 1998) had subjects focus on a visual GO stimulus – a task that would have facilitated the visual system and may have inhibited the auditory system against an acoustic startle. A sensory-mediated difference in habituation rates between studies was consistent with previous reports of larger eye-blink EMG amplitudes during acoustic startle when subjects attended to acoustic rather than visual stimuli (Schicatano and Blumenthal, 1998; Lipp et al., 2000). Whatever the explanation of the short-term elimination of habituation observed here, an experimental protocol that eliminates habituation to startle allows increased use of acoustic startle as both a clinical and research tool to study the central nervous system.

A small asynchrony in the activation of the left and right SCM and PARA muscle during startled rotation movements suggested that subtle temporal aspects of the reaction-time movement were preserved even when the movement was temporally advanced by the startling stimulus. If pre-activation of the right SCM muscle in a movement dominated by the left SCM muscle is accepted as evidence of an anticipatory postural adjustment (APA), then the preservation, and indeed the scaling, of this activation asynchrony may be evidence that APAs and focal movements are coupled at or below the level of the brainstem. Although it was unclear whether this asynchrony represented an APA, startle may be a potentially novel method of studying the coupling of the focal and postural components of movements.

In summary, the results of the current neck muscle study showed that the acoustic startle reflex was facilitated by readiness to execute a reaction-time task and that the reflexive muscle response evoked by startle could be sculpted by this same readiness. The similar onset latencies of the pure startle reflex and the startle-induced movements, combined with the consistent increase in EMG amplitude and movement kinematics from the reaction-time trials to the startled movement trials, provided compelling evidence that startle-induced movements in the neck muscles were the summation of a startle response and a temporally advanced pre-programmed movement. Parallel neural pathways unique to the neck muscle motoneurones might explain why startle increased EMG amplitudes in the current study, but not in previous studies employing distal limb muscles.

2.5 Bridging Summary

For this thesis, the first hypothesis of this experiment was that the cervical muscle response evoked by an auditory startle stimulus was different in relaxed subjects than in subjects ready to

execute a rapid neck movement. The results of this experiment clearly showed that the reflex response of the neck muscles was altered by consciously preparing to execute a ballistic reaction time task, and therefore the first hypothesis was accepted. The second hypothesis, that this alteration in the reflex response could occur in the first trial without practicing the movement, was also accepted. Together, these two findings indicated that the reflex response of the neck muscles was malleable and provided a mechanism by which event awareness, and in particular temporal awareness, of a stimulus could affect the reflex muscle response generated by that stimulus. The ability to alter the reflex response of the neck muscles, however, did not mean that subjects necessarily make use of this phenomenon in non-reaction-time settings. The remainder of the experiments conducted for this thesis focused on whether subjects exploited this malleability during exposures to whiplash-like perturbations.

CHAPTER 3 RESPONSE TO THE FIRST PERTURBATION

3.1 Introduction

The biomechanical factors that affect the risk of whiplash injury remain poorly understood. Compared to other crash directions, rear-end collisions, where an occupant's vehicle is struck from behind and accelerated forward, have been associated with both an increased risk of whiplash injury (Nygren et al., 1985; Otte and Rether, 1985; Olney and Marsden, 1986; Deans et al., 1987; van Koch et al., 1995; Jakobsson et al., 2000) and a higher frequency of multiple symptoms (Sturzenegger et al., 1994). Female gender has also been associated with increased risk of whiplash injury (O'Neill et al., 1972; Balla, 1980; Kahane, 1982; Lövsund et al., 1988; Otremski et al., 1989; Jakobsson et al., 2000). Few other risk factors for whiplash injury have been established, although a recent cohort study of whiplash-injured individuals has shown that being unprepared for a collision was related to both a higher frequency of multiple symptoms and more severe headaches in the first weeks following the collision (Sturzenegger et al., 1994). Being unprepared, in combination with an "inclined" head position, was also associated with an increased likelihood of symptoms lasting at least one year (Sturzenegger et al., 1995). Both of these recent studies suggested that some component of being unprepared for a collision increased the potential for whiplash injury.

Physical preparation, such as pre-tensing the neck muscles or altering pre-impact posture, in advance of a whiplash-like perturbation has been shown to reduce the peak magnitude of an occupant's kinematic response to a perturbation (Ono et al., 1997; Pope et al., 1998). This reduced response may be responsible for the reduced injury potential observed by Sturzenegger et al. (1994, 1995). A second component of being unprepared for the collision – being unaware of the imminent perturbation – may also be important, particularly in the study of whiplash injury biomechanics in the laboratory. Awareness of an event, independent of external physical preparations, creates anticipation. Using a reaction-time protocol, Frank (1986) has shown that this anticipation results in facilitation of spinal reflexes related to a preparatory set in advance of the event. Siegmund et al. (in press) have also shown that anticipation, in the form of mental preparation of a movement, prior to a reflex-evoking stimulus can alter the reflex muscle response and the resulting kinematics.

In all human subject experiments investigating whiplash injury biomechanics, subjects have been aware that a perturbation was imminent (Severy et al., 1955; Mertz and Patrick, 1967; Gutierrez, 1978; McConnell et al., 1993; Geigl et al., 1994; Matsushita et al., 1994; Szabo et al., 1994; McConnell et al., 1995; Ono and Kanno, 1996; Szabo and Welcher, 1996; Castro et al., 1997; Ono et al., 1997; Siegmund et al., 1997; Davidsson et al., 1998; Pope et al., 1998; van den Kroonenberg et

al., 1998). Though varying levels of temporal awareness, i.e., awareness regarding the exact timing of the perturbation, have been used in these studies, all of the subjects were nonetheless aware that a perturbation would occur. As a result, the pre-perturbation condition of these experimental subjects was not identical to that of unprepared occupants in most real collisions. Given that awareness has been associated with measures of both injury severity and duration (Sturzenegger et al., 1994), this difference between laboratory conditions and actual injury-producing collisions may be important.

The aim of this study was to examine how awareness of the presence and timing of a whiplash-like perturbation affected the muscle and kinematic responses of human subjects. A unique aspect of this experiment was the use of deception to create a surprised condition that better represented the unprepared state of vehicle occupants in most real collisions. This surprised condition was compared to two other levels of awareness that encompassed those used in previous human subject studies of whiplash biomechanics. An alerted condition, in which subjects received a countdown to the perturbation, represented an extreme condition in which the timing of the perturbation was precisely known. This condition might represent sled tests in which subjects were initially moving rearward and could potentially predict when they would be abruptly halted (Mertz and Patrick, 1967; Ono et al., 1997), or vehicle-to-vehicle collisions in which instrumentation noise, i.e., camera windup, or limited regard to controlling visual and auditory cues might have allowed subjects to predict the time of impact (Severy et al., 1955; McConnell et al., 1993). An unalerted condition represented most other studies in which subjects knew an impact would occur, but could not predict its precise timing.

Based on the increased risk of injury identified in unprepared vehicle occupants (Sturzenegger et al., 1994), it was hypothesized that the neck muscle and kinematic responses of subjects at different levels of awareness regarding the presence and timing of a whiplash-like perturbation would be different. To account for the previously-identified risk factors of collision direction and gender, a simulated rear-end collision and similar numbers of male and female subjects were used to examine potential gender-specific differences. Awareness-related difference or gender-related differences in an awareness effect, if present, might identify biomechanical variables important to the aetiology of whiplash injury.

3.2 Methods

3.2.1 Subjects

Sixty-six subjects participated in the experiment. Physical characteristics for the subjects are given in Table 3.1. The subjects had no history of whiplash injury, medical conditions that impair

sensory or motor function, or prolonged neck or back pain in the previous three years. All subjects gave their informed consent and were paid a nominal amount for their participation. The use of human subjects for this experiment was approved by the university's Ethics Review Board and the study conformed with the Declaration of Helsinki.

Table 3.1 Mean (S.D.) of subject age and physical characteristics.

	Female	Male
n	35	31
Age, yrs	24 (5)	27 (7)
Height, cm	166 (7)	176 (8)
Mass, kg	60 (10)	77 (15)

3.2.2 Instrumentation

Electromyographic (EMG) activity in the orbicularis oculi (OO), masseter (MAS), sternocleidomastoid (SCM) and cervical paraspinal (PARA) muscles was recorded bilaterally using 10 mm pre-gelled surface electrodes (H69P, Kendall-LTP, Huntington Beach, CA) and an Octopus AMT-8 amplifier (Bortec, Calgary, AB). Two uniaxial accelerometers (Kistler 8302B20S1; ±20g, Amherst, NY) and a single uniaxial angular rate sensor (ATA Sensors ARS-04E; ±100 rad/s, Albuquerque, NM) were strapped tightly to the midline of a subject's forehead, immediately above the glabella. The sensitive axes of the accelerometers were mutually orthogonal and oriented to measure horizontal (x') and vertical (z') acceleration in the mid-sagittal plane (Figure 3.1). The rotational axis of the angular rate sensor was oriented mediolaterally to measure flexion and extension motion in the sagittal plane. In some subjects, torso acceleration was measured using an uniaxial accelerometer (Kistler 8302B20S1; ±20g, Amherst, NY) applied with adhesive over the manubrium about 2 cm below its superior margin. Sled acceleration was measured using an uniaxial accelerometer (Sensotec JTF3629-05; ±10g, Columbus, OH) oriented horizontally along the axis of motion. Displacement was measured using an Optotrak motion analysis system (Northern Digital, Waterloo, ON) with markers placed over the left mastoid process and midline at the subject's forehead, vertex of the head, manubrium and C7 spinous process (Figure 3.1). A sixth marker was placed on the left seat hinge. A force transducer (Artech S-Beam SS20210, ±2 kN, Riverside, CA) was used to measure reaction loads during normalizing contractions of the SCM and PARA muscles. EMG signals were bandpass filtered at 10 Hz to 1 kHz and transducer signals were low-pass filtered at

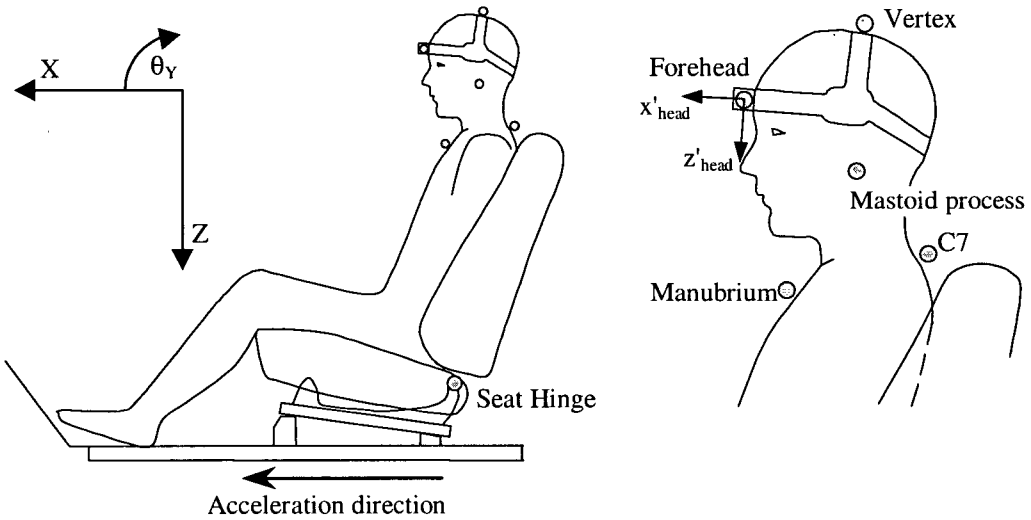
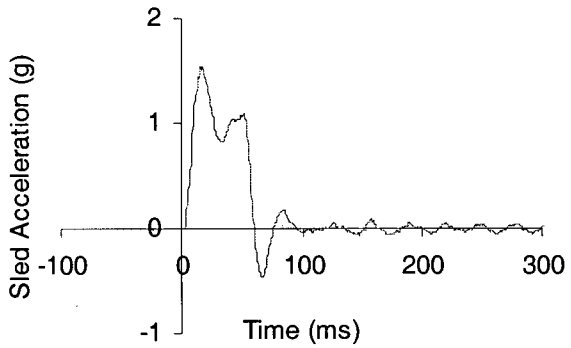


Figure 3.1 Schematic of the test configuration showing the locations of the Optotrak markers (shaded circles), the lab reference frame (X, Z) and the head reference frame (x', z'). The initial orientation of the forehead reference frame was determined by how the head band fit the subject and varied between 12 ± 4 deg relative to the lab reference frame.



Property	Mean (S.D.)
Peak acceleration (g)	1.52 (0.04)
Time to peak acceleration (ms)	16.3 (1.2)
Pulse duration (ms)	59.9 (0.3)
Speed change (m/s)	0.497 (0.006)
Average acceleration (g)	0.897 (0.004)

Figure 3.2 Sample acceleration pulse and descriptive statistics of selected pulse properties.

1 kHz before being simultaneously sampled at 2 kHz and stored for subsequent analysis. Optotrak data were acquired at 200 Hz per marker.

3.2.3 Test Procedures

Subjects were seated in an automobile seat obtained from the front passenger location of a 1991 Honda Accord. The head restraint was removed from the top of the seat back to eliminate the potential for an externally applied load to the head during the perturbation. The seat was mounted on a custom fabricated sled powered by a feedback-controlled linear induction motor (Kollmorgen IC55-100A7, Kommack, NY). The sled generated no pre-perturbation signals, either audible or mechanical, which subjects could use to predict onset of the perturbation.

To install and connect some of the instrumentation, subjects were seated on the sled about 15 minutes before their perturbation. This allowed the subjects sufficient time to adopt a comfortable seated posture. Prior to testing, subjects were instructed to sit normally, face forward, rest their forearms on their lap and to relax their face and neck muscles. Each subject was exposed to a single perturbation which accelerated the subject horizontally forward (Figure 3.2). Subjects did not receive a practice or demonstration perturbation prior to testing.

Subjects were randomly assigned to one of three groups: alerted, unalerted or surprised. Subjects in the alerted group received a countdown consisting of two auditory tones (1000 Hz, 80 dB, 40 ms duration) spaced 1 s apart. The perturbation then occurred 1 s after the second tone, coincident with the expected time of a third tone had one been presented. Subjects in the unalerted group were told to expect a perturbation some time in the next 60 seconds and then received a perturbation about 15 seconds later. In order to elicit the surprised response, subjects in the third group were deceived. Once ready for their first perturbation, subjects in the surprised group were told that a baseline measurement of their relaxed muscle activity was needed before their perturbation. For this baseline test, these subjects received the same instructions regarding their seated posture and muscle activity level as subjects in the other two groups. During this fictitious baseline measurement, these subjects received their perturbation. Immediately after their perturbation, subjects in the surprised group were informed of the true nature of the study.

Prior to the above protocol, seated subjects performed sub-maximal isometric contractions in flexion and extension to generate normalizing data for the SCM and PARA muscles respectively. A strap attached to the load cell was placed around a subject's head and its length adjusted to ensure the subject's head was neutrally positioned. The strap was located immediately above the glabella for flexion contractions and at the height of the external occipital protuberance for extension contractions. Subjects were instructed to generate a force of 50 N with visual feedback, first in flexion

and then in extension. EMG and load cell data were acquired for 5 s during each contraction.

3.2.4 Data Reduction

A subject's initial position was determined from the Optotrak data immediately preceding their perturbation. Initial head position was defined in the laboratory reference frame using the horizontal (X-axis; positive forward) and vertical (Z-axis; positive downward) positions of the mastoid process relative to the seat hinge (Figure 3.1). Initial head angle in the sagittal plane was determined from the forehead and vertex markers and reported relative to the positive X-axis ($+\theta_y$ rotation corresponded to extension). Initial torso position was defined using the horizontal and vertical positions of the midpoint between the manubrium and C7 spinous process markers relative to the seat hinge, and torso angle relative to the horizontal plane was determined using the same two markers. The RMS accuracy of the position measurements from the Optotrak system was less than 0.1 mm, and based on marker separation, the RMS accuracy of the calculated angles was less than 0.1 degrees.

The onset of head and torso movement, peak head accelerations and the time of the peak head accelerations were determined directly from the accelerometer data. The algorithm used to determine onset is developed in Appendix A. Forehead acceleration data were not resolved into the lab reference frame. Peak retraction, defined as the maximum horizontal translation of the top of the cervical spine rearward relative to the bottom of the cervical spine, was estimated using the maximum relative horizontal displacement in the lab reference frame between the Optotrak markers on the mastoid and manubrium. Peak angular velocity of the head was determined from the angular rate sensor data after it had been digitally compensated to reduce the sensor's high-pass frequency to 0.002 Hz (Laughlin, 1998). Angular acceleration was then computed by finite differences (5 ms window) from the compensated angular velocity data. Total head angular displacement was determined from the Optotrak markers located at the forehead and vertex. Horizontal and vertical accelerations at the mastoid process were computed by double differentiation of the Optotrak data. This technique produced results within 0.1g of the values computed using a rigid body transformation of the linear and angular accelerations measured at the forehead and was preferred because it relied on data from only one Optotrak marker rather than data from two accelerometers, an angular rate sensor and two Optotrak markers needed for the rigid body calculation. Mastoid accelerations were reported in the lab reference frame.

Pre-stimulus noise in each channel of the EMG data was quantified using the root mean squared (RMS) amplitude of the EMG signal over the 100 ms preceding the perturbation. EMG onset was defined as the time at which the RMS amplitude, computed from the raw EMG data using a

moving 20 ms window, reached 10 percent of it maximum value and was then confirmed visually. For each muscle, the RMS amplitude of the EMG signal was calculated for the interval between EMG onset and peak head extension angle. The corresponding pre-stimulus noise for each muscle was then subtracted from this quantity. The SCM and PARA muscle EMG amplitudes were normalized by the RMS amplitude obtained during the 5 s sub-maximal contraction for the corresponding muscle.

Angular kinematic data from one subject were lost due to a grounding problem and Optotrak data were lost in another subject due to synchronization problems. A mastoid marker was not present for the first subject, and other mastoid data were deleted due to marker dropout, excessive vibration or the absence of an initial upward acceleration component. EMG onset could not be reliably determined for the OO and MAS muscles in one subject and for the PARA muscles in three different subjects due to excessive sled-induced noise. This precluded calculating the PARA RMS magnitude for these same subjects.

3.2.5 Statistical Analysis

For each kinematic dependent variable, a two-way analysis of variance (ANOVA) was used to assess differences related to level of awareness (surprised, unalerted, alerted) and gender (female, male). Electromyographic variables were first assessed using a three-way ANOVA with muscle side (left, right) added as a repeated measures variable to the awareness and gender factors. After confirming that there were no statistical effects related to muscle side, the mean of the left and right sided data was computed and a two-way ANOVA for awareness and gender only was performed. Dependent variables which contained within-cell values that were not normally distributed (Shapiro-Wilks test, $p<0.05$) were reanalyzed using a rank-based non-parametric ANOVA (Zimmerman and Zumbo, 1993). If the results of the parametric and non-parametric ANOVAs were similar, then it was assumed that the parametric analysis was valid (Conover, 1999). All statistical tests were performed using Statistica (v.5.1, Statsoft Inc., Tulsa, OK) and a significance level of $\alpha=0.05$. Post-hoc comparisons between the three levels of awareness were first performed using a Scheffé test, but then relaxed to a Tukey's honest significant difference test.

3.3 Results

3.3.1 Initial Position

An absence of awareness-related differences in the initial position and angle of the head and torso indicated that subjects did not use the pre-perturbation warning to adjust their seated posture (Table 3.2). Gender-related differences, however, were present in the initial head and torso positions and were consistent with male subjects being larger than female subjects. The mastoid process of the

Table 3.2 Mean (S.D.) of the initial position and angle of the head and torso. Upper portion of table summarizes data as a function of awareness (surprised, unalerted, alerted) and gender (female, male). Positions are relative to an origin at the seat hinge; angles are relative to the horizontal. Lower portion of table summarizes the results (F-statistics) of separate 2-way ANOVAs using awareness and gender as independent variables. X, horizontal position, +ve forward; Z, vertical position, +ve downward; N, number of subjects in analysis.

		Mastoid process		Head	Manubrium-C7 midpoint		Torso
		X (mm)	Z (mm)	angle (deg)	X (mm)	Z (mm)	angle (deg)
Female	Surprised	15 (21)	-649 (43)	6.9 (4.6)	23 (16)	-560 (37)	-16.3 (4.8)
	Unalerted	11 (16)	-642 (23)	7.1 (1.9)	21 (14)	-552 (19)	-15.9 (4.1)
	Alerted	7 (19)	-641 (35)	7.9 (3.4)	21 (10)	-555 (29)	-16.7 (4.7)
Male	Surprised	20 (20)	-681 (33)	8.2 (6.6)	33 (16)	-583 (30)	-16.5 (5.0)
	Unalerted	29 (12)	-664 (36)	9.3 (3.8)	38 (6)	-573 (33)	-15.7 (3.4)
	Alerted	21 (18)	-672 (25)	9.6 (4.8)	28 (14)	-582 (25)	-17.2 (5.0)
N		64	64	65	62	62	62
ANOVA F-statistics							
Aware							
Gender		6.82*	11.2**		10.5**	9.64**	
Aware x Gender							

* $p<0.05$, ** $p<0.01$

average male subject was located 12 mm forward ($F_{1,58}$=6.82, $p<0.05$) and 28 mm above ($F_{1,58}$=11.2, $p<0.01$) the mastoid process of the average female subject. Similarly, the midpoint between the manubrium and C7 markers of the average male subject was 11 mm forward ($F_{1,56}$=10.5, $p<0.01$) and 23 mm above ($F_{1,56}$=9.64, $p<0.01$) the same point on the average female subject. Despite these position differences, there were no gender differences in either the initial head or torso angles.

3.3.2 Kinematic Response

The perturbations produced a stereotypical response in all subjects (Figure 3.3). Neither the onset of torso acceleration nor the time of peak torso acceleration were related to awareness or gender. Averaged across all conditions, the onset of torso acceleration occurred at 20 ± 2 ms after perturbation onset and peak torso acceleration occurred at 78 ± 9 ms after perturbation onset. Evidence of head acceleration appeared first in the vertically mounted accelerometer 29 ± 5 ms after perturbation onset and was similarly unrelated to awareness or gender.

Amongst the peak kinematic parameters analyzed (labeled with hollow circles in Figure 3.3), only peak retraction (r_x) varied with awareness ($F_{2,57}$=4.00, $p<0.05$, Table 3.3). This main effect was tempered by an awareness × gender interaction ($F_{2,57}$=3.23, $p<0.05$), which indicated that awareness affected male and female subjects differently. Two other kinematic parameters also yielded significant interaction effects: peak horizontal acceleration of the mastoid process (x_2 - $F_{2,57}$=4.36, $p<0.05$) and peak angular head acceleration in flexion (α_2 - $F_{2,59}$=4.70, $p<0.05$). Post-hoc testing of these parameters revealed that the surprised female subjects had a 30 percent lower mastoid acceleration than unalerted female subjects ($p=0.020$) and surprised female subjects had a 29 percent larger retraction than all other awareness-gender combinations ($p<0.031$) except that of unalerted male subjects. Surprised males, on the other hand, had a larger head angular acceleration in flexion (α_2) than alerted male subjects ($p=0.021$). One surprised male subject with a large angular acceleration (-410 rad/s²) increased the mean presented in Table 3.3 (mean=-146 ± 28 rad/s² without this data point) and may have skewed the ANOVA. A rank-based non-parametric ANOVA run on the same data also yielded a significant awareness × gender interaction and supported the same post-hoc result ($p=0.022$). In addition to these awareness effects, the peak amplitude of two other parameters varied with gender. Compared to male subjects, female subjects had a 15 percent lower peak horizontal forehead acceleration (x_1 – $F_{1,60}$=7.61, $p<0.01$) and a 15 percent larger head extension angle (θ - $F_{1,59}$=5.51, $p<0.05$).

Despite awareness-related effects in the amplitude of the kinematic peaks, timing of these kinematic peaks in relation to the perturbation did not vary with subject awareness (Table 3.4). Gender, on the other hand, was related to the timing of seven of the eleven kinematic peaks

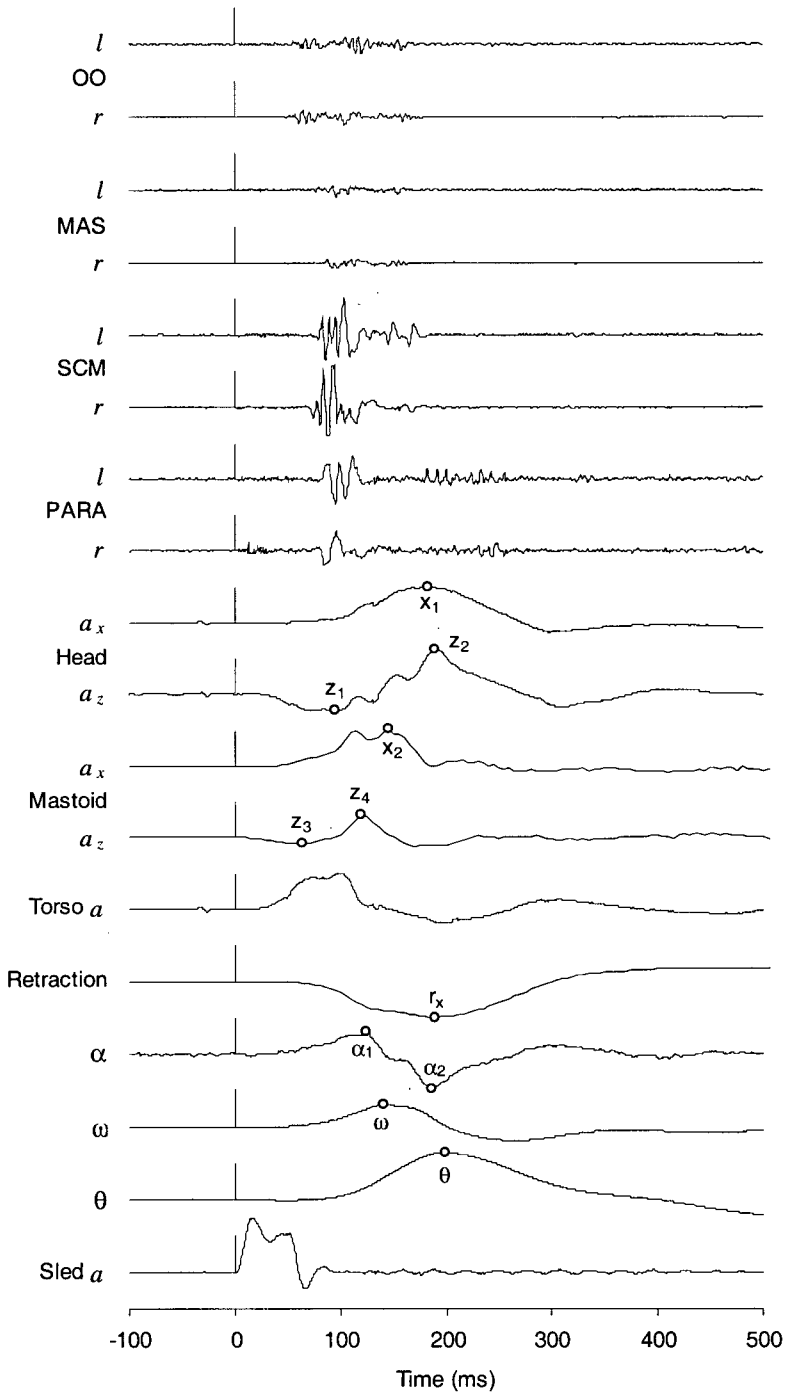


Figure 3.3 Sample EMG and kinematic data from a single subject. Labeled hollow circles indicate kinematic peaks used in the analysis. The vertical scale bars are aligned with the onset of the perturbation and are equivalent to 1g, 25 mm, 100 rad/s², 5 rad/s and 10 deg. OO, orbicularis oculi; MAS, masseter; SCM, sternocleidomastoid; PARA, cervical paraspinal muscles, l, left; r, right; *a*, linear acceleration, subscript *x* refers to the x-direction; subscript *z* refers to the z-direction; α, head angular acceleration; ω, head angular velocity; θ, head angle.

Table 3.3 Mean (S.D.) of the peak amplitude of selected kinematic data. Upper portion of table summarizes data as a function of awareness (surprised, unalerted, alerted) and gender (female, male). Lower portion of table summarizes the results (F-statistics) of separate ANOVAs for the effect of awareness and gender on each of the peak responses. Individual peaks are labeled with hollow circles in Figure 3.3. N, number of subjects in analysis.

		Linear kinematics							Angular kinematics			
		Forehead			Mastoid process			Retraction	Head			
		x_1 (g)	z_1 (g)	z_2 (g)	x_2 (g)	z_3 (g)	z_4 (g)	r_x (mm)	α_1 (rad/s²)	α_2 (rad/s²)	ω (rad/s)	θ (deg)
Female	Surprised	0.96 (0.22)	-0.64 (0.15)	1.52 (0.53)	0.95 (0.27)	-0.23 (0.08)	0.51 (0.18)	-27.0 (4.8)	67.9 (17.4)	-124 (55)	3.3 (0.8)	13.4 (2.2)
	Unalerted	1.14 (0.23)	-0.59 (0.12)	1.65 (0.57)	1.38 (0.37)	-0.20 (0.08)	0.65 (0.34)	-20.9 (3.5)	75.0 (21.9)	-146 (52)	3.2 (1.1)	11.6 (4.3)
	Alerted	1.11 (0.17)	-0.64 (0.21)	1.51 (0.41)	1.30 (0.40)	-0.19 (0.06)	0.51 (0.19)	-21.5 (3.1)	79.3 (30.5)	-133 (45)	3.5 (0.9)	12.4 (2.3)
Male	Surprised	1.36 (0.32)	-0.75 (0.21)	2.13 (0.93)	1.16 (0.19)	-0.16 (0.06)	0.52 (0.22)	-21.3 (3.6)	74.0 (21.7)	-175 (92)	3.3 (1.0)	11.0 (3.0)
	Unalerted	1.29 (0.35)	-0.66 (0.22)	1.62 (0.26)	1.11 (0.28)	-0.21 (0.06)	0.53 (0.21)	-21.7 (5.7)	70.1 (25.8)	-112 (22)	3.1 (0.5)	10.7 (2.5)
	Alerted	1.12 (0.32)	-0.72 (0.14)	1.48 (0.37)	1.07 (0.18)	-0.17 (0.07)	0.37 (0.11)	-20.0 (3.8)	64.3 (13.5)	-96 (29)	3.0 (0.5)	10.8 (2.1)
N		66	66	66	63	62	61	63	65	65	65	65
ANOVA F-statistics												
Aware								4.00*				
Gender		7.61**						4.18*				5.51*
Aware x Gender					4.36*			3.23*		4.70*		

* $p<0.05$, ** $p<0.01$

Table 3.4 Mean (S.D.) of the time of peak amplitude of selected kinematic data. Upper portion of table summarizes data as a function of awareness (surprised, unalerted, alerted) and gender (female, male). Lower portion of table summarizes the results (F-statistics) of separate ANOVAs for the effect of awareness and gender on each of the peak responses. Individual peaks are labeled with hollow circles in Figure 3.3. N, number of subjects in analysis.

		Linear kinematics							Angular kinematics			
		Forehead			Mastoid process			Retraction	Head			
		x_1 (ms)	z_1 (ms)	z_2 (ms)	x_2 (ms)	z_3 (ms)	z_4 (ms)	r_x (ms)	α_1 (ms)	α_2 (ms)	ω (ms)	θ (ms)
Female	Surprised	178 (12)	89 (17)	166 (15)	116 (15)	62 (5)	132 (11)	177 (29)	103 (7)	175 (16)	134 (10)	195 (26)
	Unalerted	170 (12)	85 (10)	169 (7)	116 (11)	60 (8)	127 (14)	155 (22)	112 (16)	167 (5)	134 (13)	179 (13)
	Alerted	171 (13)	93 (23)	169 (16)	112 (15)	62 (4)	125 (9)	160 (22)	108 (16)	173 (10)	136 (13)	184 (13)
Male	Surprised	163 (10)	97 (13)	160 (9)	114 (15)	64 (10)	133 (14)	159 (25)	105 (11)	161 (10)	127 (8)	172 (25)
	Unalerted	168 (9)	90 (11)	158 (14)	116 (13)	63 (6)	137 (18)	167 (26)	104 (8)	161 (12)	130 (6)	176 (13)
	Alerted	167 (14)	93 (10)	159 (15)	112 (9)	70 (10)	150 (27)	167 (15)	100 (8)	157 (15)	128 (12)	180 (18)
N		66	66	66	63	62	61	63	65	65	65	65
ANOVA F-statistics												
Aware												
Gender		5.58*		7.39**		5.80*	7.61**			16.95***	5.35*	4.88*
Aware x Gender												

* $p<0.05$, ** $p<0.01$, *** $p<0.001$

considered. Though statistically significant, differences in timing between male and female subjects were relatively small – less than 15 ms for all seven of the parameters in Table 3.4. All of the peak angular head kinematics occurred earlier in male subjects than in female subjects. This same gender-related pattern was present in the forehead kinematics, which were partially influenced by the angular head kinematics. At the mastoid process, however, peak kinematics occurred later in male subjects than in female subjects.

3.3.3 Muscle Response

The perturbation evoked a muscle response in all four muscle groups, particularly the SCM muscle (Figure 3.3). There were no significant differences between the onset latencies or RMS amplitudes of the left and right muscles and therefore the average response of the left and right muscles was used for the remainder of the analysis.

The onset latencies of both neck muscles varied significantly with awareness (SCM: $F_{2,60}=11.1$, $p<0.0001$; PARA: $F_{2,57}=3.21$, $p<0.05$) (Table 3.5). Post-hoc analyses indicated that SCM activation occurred an average of 7 ms later in surprised subjects than in the alerted ($p=0.0035$) and unalerted subjects ($p=0.0002$), and PARA activation occurred 5 ms later in surprised subjects than in the alerted subjects ($p=0.036$). The time of maximal RMS amplitude in the SCM muscle varied with awareness ($F_{2,60}=3.54$, $p<0.05$), and although these data contained outliers, a rank-based non-parametric ANOVA produced a similar awareness effect. Post-hoc analysis revealed that surprised subjects reached maximal EMG amplitude later than alerted subjects ($p=0.048$), but not significantly later than unalerted subjects ($p=0.058$).

When averaged across awareness conditions, SCM activation occurred 5 ms later in male subjects than female subjects ($F_{1,60}=13.9$, $p<0.001$) and PARA activation occurred 3 ms later in male subjects than female subjects ($F_{1,57}=4.61$, $p<0.05$) (Table 3.5). The delayed activation in male subjects relative to female subjects was further explored using a linear regression which included independent variables for gender, awareness level, height and mass. Both height and mass were eliminated from the regression in favour of the two discrete variables, a result that suggested the observed gender effect was not related to differences in size between the male and female subjects.

The normalized EMG amplitude of the SCM muscles was significantly larger in male subjects than female subjects ($F_{1,59}=20.9$, $p<0.0001$). In the PARA muscles, however, significant main effects for both awareness and gender were qualified by an awareness × gender interaction ($F_{2,57}=3.68$, $p<0.05$). Post-hoc analysis showed that surprised males had a significantly larger EMG amplitude than alerted males ($p=0.018$), surprised females ($p=0.006$) and alerted females ($p=0.012$).

Table 3.5 Mean (S.D.) of EMG onset time, maximum RMS time, and normalized RMS magnitude. Upper portion of table summarizes data as a function of awareness (surprised, unalerted, alerted) and gender (female, male). Lower portion of table summarizes the results (F-statistics) of separate 2-way ANOVAs using awareness and gender as independent variables. RMS, root-mean square; OO, orbicularis oculi; MAS, masseter; SCM, sternocleidomastoid; PARA, cervical paraspinal; N, number of subjects in analysis.

		Onset (ms)				Max RMS (ms)	RMS Magnitude	
		OO	MAS	SCM	PARA	SCM	SCM	PARA
Female	Surprised	70 (13)	80 (8)	77 (5)	80 (4)	119 (18)	0.15 (0.07)	0.10 (0.07)
	Unalerted	65 (7)	71 (8)	68 (6)	77 (7)	106 (7)	0.21 (0.08)	0.16 (0.08)
	Alerted	69 (14)	79 (12)	69 (5)	76 (7)	104 (11)	0.17 (0.05)	0.11 (0.07)
Male	Surprised	74 (10)	82 (11)	81 (7)	83 (4)	112 (10)	0.42 (0.20)	0.29 (0.19)
	Unalerted	64 (9)	81 (9)	74 (5)	82 (7)	109 (13)	0.31 (0.14)	0.22 (0.15)
	Alerted	68 (8)	75 (8)	76 (6)	78 (6)	111 (10)	0.35 (0.31)	0.11 (0.07)
N		65	65	66	63	66	66	63
ANOVA F-statistics								
Aware				11.1****	3.21*	3.54*		3.22*
Gender				13.9***	4.61*		20.9****	7.82**
Aware x Gender								3.68*

* p<0.05, ** p<0.01, *** p<0.001, ****p<0.0001

3.4 Discussion

The goal of this study was to examine how event awareness and temporal awareness affected the neck muscle and kinematic responses of seated subjects exposed only once to a whiplash-like perturbation. Event awareness was studied by comparing the response of a group of subjects who were deceived into thinking they would not be perturbed to the response of a group of subjects who were aware they would be perturbed. Within this latter group, some subjects knew exactly when they would be perturbed, whereas others knew only that a perturbation was imminent. This division of the event-aware subjects allowed the effect of temporal awareness to be simultaneously studied.

The results of the present study indicated that event awareness affected the temporal development of the neck muscle response evoked by a whiplash-like perturbation. In both male and female subjects, activation of the SCM and PARA muscles occurred later in surprised subjects compared to both unalerted subjects, who were event aware but not temporally aware, and alerted subjects, who were both event and temporally aware. Since there was no difference in either the perturbation, the initial position or the onset times of the torso or head acceleration between the different awareness groups, this delayed neck muscle activation appeared to be directly related to event awareness. Similarly shortened onset latencies to reflex-evoking stimuli have been observed in warned reaction-time experiments (Silverstein et al., 1981) and have been attributed to changes in the membrane potential of sensory and motor neurons due to selective attention and motor preparation respectively (Brunia, 1992). The current results, therefore, suggested that the sensorimotor system of the alerted and unalerted subjects was facilitated by their awareness of an imminent perturbation and that this facilitation resulted in earlier activation of the neck muscles. This facilitation appeared to be similar in both the alerted and unalerted conditions, and suggested that knowledge of an imminent perturbation (event awareness) was a more important factor in producing a facilitated state than knowledge of the perturbation's precise timing (temporal awareness).

Although awareness-related differences in muscle activation were present in both genders, awareness-related differences in the kinematic responses were gender specific. Peak horizontal mastoid acceleration (x_2) was lower and peak retraction (r_x) was higher in surprised female subjects than in other subject groups. In contrast to the lower linear acceleration observed in surprised female subjects, a larger angular acceleration (α_2) was observed in surprised male subjects than in alerted male subjects. All three of these kinematic peaks occurred after neck muscle activation, a temporal pattern that suggested the observed differences in muscle activity were responsible for these kinematic differences. It was not clear from the data, however, how a similarly delayed SCM and PARA activation in surprised subjects of both genders produced kinematic differences that were gender specific. The lower forward mastoid acceleration (x_2) and larger retraction (r_x) in surprised

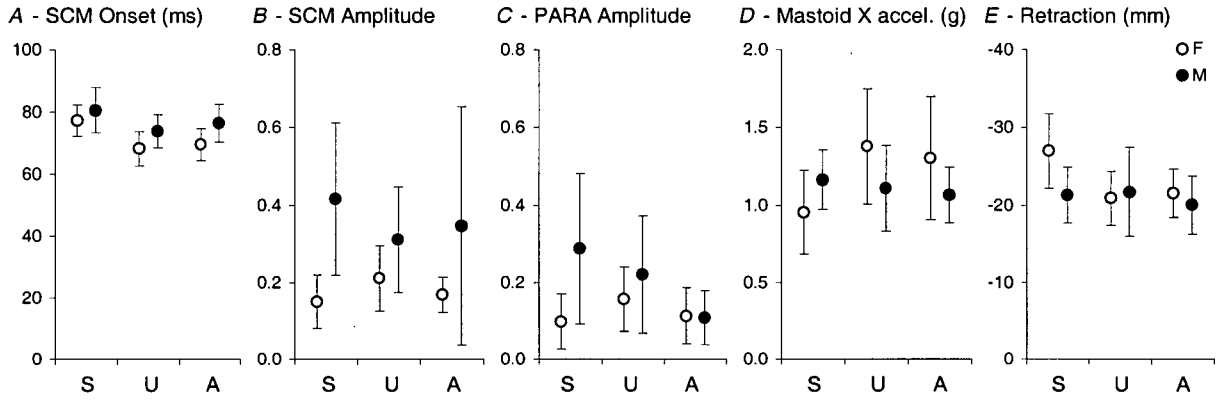


Figure 3.4 Summary of awareness-related differences (mean ± SD) that, when combined, suggested a mechanism explaining why female subjects responded differently in surprised conditions. See text for a complete description. SCM, sternocleidomastoid muscles; PARA, cervical paraspinal muscles; RMS, root mean square; F, female; M, male; S, surprised; U, unalerted; A, alerted.

female subjects suggested a weaker muscle contraction in surprised female subjects than in the other female subjects. Conversely, the larger angular head acceleration in flexion in surprised male subjects suggested a stronger muscle contraction in surprised male subjects than in the other male subjects. Despite these expectations, the analysis of EMG amplitude for the SCM muscles revealed only a strong gender difference, and the analysis of EMG amplitude for the PARA muscles showed that the response of surprised male subjects was larger than in alerted male subjects, but not larger than in unalerted male subjects (Table 3.5). Further analysis of the SCM amplitude data, however, showed that the observed gender difference was most pronounced in the surprised condition and that the large variability in the amplitude data, particularly in the male subjects, may have obscured a possible interaction effect similar to that observed in the PARA muscles (Table 3.5, Figure 3.4B, C). Anthropometry differences between subjects in the three awareness groups was another possible explanation for the gender-specific kinematic response, however, no significant within-gender height or mass differences were present between the awareness groups.

Without a clear link between the awareness-related, but gender-neutral, differences in the muscle response and the awareness-related, but gender-specific, differences in the kinematic response, the current data could only be used to postulate a mechanism by which surprised subjects respond differently than alerted or unalerted subjects. Event aware subjects, even though relaxed, prepared for the perturbation by facilitating the sensorimotor systems responsible for the reflexive neck muscle contraction. The sensorimotor systems of the unaware subjects, however, were not similarly facilitated and therefore neck muscle activation in these subjects was delayed (Figure 3.4A).

It was postulated that the surprised male subjects accommodated this delayed activation by increasing the magnitude of their SCM and PARA contractions (Figure 3.4B, C). This compensatory response produced levels of mastoid acceleration (Figure 3.4D) and head retraction (Figure 3.4E) that were similar, but levels of angular head acceleration that were higher, than those observed in aware male subjects. Surprised female subjects, on the other hand, did not compensate for the delayed onset with a larger muscle contraction and this resulted in both a lower forward mastoid acceleration and a larger head retraction movement. The current data, though providing the foundation for this proposed explanation, lacked the statistical power to determine post-hoc whether the postulated gender × awareness interactions were actually present in both the SCM and PARA activation levels. Future work based on the mechanism proposed here could explicitly hypothesize these effects and their directions, and could therefore use planned comparisons rather than post-hoc testing to establish this link. Such planned comparisons run on the current data showed that the postulated effect was present, however, could not be relied upon because the effect was not specifically hypothesized.

Though unexpected, the observation of a 25 percent larger peak retraction in only surprised female subjects was consistent with two aspects of the whiplash literature: first, females have a higher risk of whiplash injury than males (O'Neill et al., 1972; Balla, 1980; Kahane, 1982; Lövsund et al., 1988; Otremski et al., 1989; Jakobsson et al., 2000); and second, unprepared subjects have both more symptoms and more severe symptoms than prepared subjects (Sturzenegger et al., 1994; Sturzenegger et al., 1995). A larger retraction would cause larger strains in the ligamentous tissues of the cervical spine. If the increased retraction observed here were distributed uniformly over the cervical spine, about 1 mm of additional translation would occur between each pair of cervical vertebra. Based on data presented by Siegmund et al. (2000b), this additional motion would increase the maximum principal strain in the facet joint capsular ligaments by about 0.03, which was about 4 to 27 percent of the strains reported by these authors to cause sub-catastrophic failures in these ligaments. The cervical facet joints have also been isolated as the source of pain in about half of a population with chronic whiplash pain (Lord et al., 1996). Though in need of additional investigation, the results of the current study provide a possible biomechanical explanation for why unprepared female occupants have an increased risk of whiplash injury.

Although the current data provided incomplete evidence of why kinematic differences were observed between subjects aware and unaware of the perturbation, it clearly showed that unprepared subjects responded differently to a whiplash-like perturbation than did subjects whose level of awareness was similar to that present in most, if not all, previous whiplash experiments using human subject. This finding calls into question the applicability of the muscle, kinematic and, perhaps most importantly, clinical response data of all previously-conducted whiplash experiments using human

subjects to study real whiplash collisions. Based on the differences observed here, it may be possible to interpret the muscle and kinematic data of previous studies to determine how their observed responses might have been different had their subjects been truly surprised. Post-hoc interpretation of the transient clinical symptoms experienced by subjects in these previous experiments will likely be more difficult. Given the larger retraction observed in surprised female subjects, previous reports of clinical symptoms generated in whiplash experiments may underestimate the risk of whiplash injury in real collisions.

3.4.1 Comparison to Previous Studies

None of the EMG or kinematic variables examined here exhibited a significant post-hoc difference between the alerted and unalerted conditions. This finding was at odds with results reported by Kumar et al. (2000), who observed a significant difference in anterior head acceleration between subjects who knew both the timing and intensity of a whiplash-like perturbation and subjects who were deprived of both visual and aural information, but nonetheless knew a perturbation would occur. A direct comparison between the previous and current data may, however, be invalid. Despite a similar peak sled acceleration, peak torso acceleration in the previous study was reached 238 to 263 ms after perturbation onset (compared to 78 ± 9 ms in the current study) and peak horizontal forehead acceleration in the previous study was reached 284 to 376 ms after perturbation onset (compared to 170 ± 12 ms in the current study). The reason for the delayed kinematics reported by Kumar et al. (2000) was not clear, but could be related to differences between the molded plastic seat used by these researchers and the automobile seat used in the current study.

The current data were consistent with results reported by Magnusson et al. (1999), who observed no difference in the muscle activation times between subjects aware and not aware of the exact timing of a perturbation. In addition, the onset latencies of torso acceleration (20 ± 2 ms), head acceleration (29 ± 5 ms) and SCM activation (74 ± 7 ms) observed in the current data were similar to the 17, 36 and 74 ms latencies, respectively, reported by Magnusson et al. (1999). As in the current study, Magnusson et al. (1999) also used an automotive seat. These similarities in the initial kinematic and muscle responses between studies were remarkable given the large difference in perturbation pulse characteristics used in the two studies. In the current study, a peak acceleration of 1.52 g was achieved in 16.3 ms, and produced a velocity change of about 0.5 m/s over a duration of about 60 ms (average acceleration was about 0.9 g) (Figure 3.2). In the study conducted by Magnusson et al. (1999), a peak acceleration of about 0.4 g was achieved in about 150 ms, and produced a velocity change of about 0.6 m/s over a duration of about 250 ms (average acceleration=0.24 g). The similar SCM activation times between studies suggested that large

differences in the early components of the perturbation were equally effective in triggering a reflexive muscle response.

Gender differences observed in the muscle and kinematic responses of male and female subjects in the current study were consistent with previous studies. Brault et al. (2000) also observed an earlier neck muscle activation in female subjects than male subjects in staged rear-end collisions, as did Foust et al. (1973) using a direct tug on the head rather than a whiplash perturbation. Gender-related kinematic differences were also similar, though not identical, to those observed previously at higher perturbation intensities using the same seat (Siegmund et al., 1997). Kinematic differences between studies appeared to be due to the absence of a head restraint in the present study and contact with the head restraint present in previous work.

A potential limitation of the current study was the relatively low perturbation magnitude used here compared to that used in most other whiplash experiments. Due to the unknown effect of the deceived condition, however, a low intensity pulse which was capable of evoking a neck muscle response without inducing excessive and potentially injurious loads was required. A previously-observed gradation of both the muscle response (Brault et al., 2000) and head and torso kinematics (Siegmund et al., 1997) with pulse intensity suggested that the effects observed here would also be present at greater perturbation intensities. Another potential limitation of this study was its inability to truly reproduce the unprepared state of individuals in a relaxed vehicle environment. The results observed here indicated that the surprised condition was different from the two aware conditions, however, it remains unknown how effectively the surprised condition used here mimicked the state of an unprepared individual driving an automobile. Future work may wish to quantify motoneurone excitability in both a relaxed vehicle environment and a pre-perturbation environment to quantify this effect.

In summary, the data presented in this study showed that surprised subjects, and in particular surprised female subjects, responded differently to a whiplash-like perturbation than did alerted and unalerted subjects. A mechanism by which event awareness altered reflex excitability, which then led to delayed muscle activation and ultimately to different kinematic responses in surprised and aware subjects was proposed. Additional research is needed, however, to determine whether the postulated difference in neck muscle amplitude between female and male subjects actually exists under surprised conditions. The increased retraction observed in surprised female subjects may explain previous findings in the literature regarding increased risk of whiplash injury in both females and in individuals unprepared for a rear-end collision.

3.5 Bridging Summary

The results of the current study showed that a difference in the neck muscle response between alerted, unalerted, and surprised subjects exposed to whiplash-like perturbations existed. The hypothesis underlying this experiment was therefore accepted. The results showed differences in the muscle and kinematic responses related to event awareness, but not to temporal awareness; a finding which suggested that depriving subjects in whiplash experiments of information regarding the precise timing of a perturbation may not be an effective method of simulating the unprepared state of vehicle occupants in advance of real whiplash collisions.

Having established that awareness plays a potentially important role in altering the response to a whiplash-like perturbation, the next question addressed in this thesis was whether and how much the response adapted over multiple exposures. For the next chapter, the aware subjects from the current experiment (both alerted and unalerted subjects) underwent ten additional perturbations of the same intensity to quantify how habituation affected the magnitude of the muscle and kinematic responses. Subjects from the deceived group were not included in the habituation experiment because the deception used in their first perturbation could not be replicated over the next ten perturbations. The deceived subjects nonetheless underwent the same habituation process so that they could be used in subsequent experiments.

CHAPTER 4 RESPONSE HABITUATION TO MULTIPLE PERTURBATIONS

4.1 Introduction

A rapid attenuation of the reflex response to sequentially-presented stimuli has been observed in postural reflexes (Nashner, 1976; Keshner et al., 1987; Hansen et al., 1988; Woollacott et al., 1988; Allum et al., 1992; Bisdorff et al., 1994; Timmann and Horak, 1997) and startle reflexes in humans (Landis and Hunt, 1939; Davis, 1984, Brown et al., 1991b). This attenuation, called habituation, is a centrally generated process that results in a generalized decrement in response magnitude to repetitive stimulation (Harris, 1943). It typically consists of a rapid initial decrement which becomes progressively slower with the number of stimulus exposures. Habituation to platform perturbations in the study of standing posture have been shown to occur in one to three exposures (Woollacott et al., 1988; Allum et al., 1992; Timmann and Horak, 1997). In both acoustic startle and free-fall experiments, 30 to 50 percent reductions in the magnitude of electromyographic (EMG) activity in facial, neck and shoulder muscles have been observed by the second or third exposure (Bisdorff et al., 1994; Valls-Solé et al., 1997). In recognition of the rapid transient changes in muscle and kinematic responses as a result of habituation, many studies of reflexes have used practice trials or discarded initial trials to achieve a stabilized response in subjects (Horak et al., 1989; Allum et al., 1992; Timmann and Horak, 1997; Chong et al., 1999). If, however, the initial response is of primary interest, then habituation is a potential source of experimental error and must be accommodated by limiting the number of exposures to those before significant habituation occurs.

In the study of whiplash injury biomechanics using human subjects, many studies have used either repeated exposures or practice trials prior to acquiring their data (Severy et al., 1955; Mertz and Patrick, 1967; Gutierrez, 1978; McConnell et al., 1993; Geigl et al., 1994; Szabo et al., 1994; McConnell et al., 1995; Ono and Kanno, 1996; Szabo and Welcher, 1996; Ono et al., 1997; Siegmund et al., 1997; Davidsson et al., 1998; Pope et al., 1998; van den Kroonenberg et al., 1998; Kaneoka et al., 1999; Magnusson et al., 1999). Habituation has only been considered in one study – and then only tangentially. Severy et al. (1955) attributed a lower peak head acceleration in the second exposure of one subject to a "conditioned muscle reflex...acquired from his initial experience." Most other studies have not explicitly reported whether practice trials were conducted, and some studies have not reported the number of repeated exposures to each subject. The muscle and kinematic data reported in these studies, therefore, might not be representative of individuals in real collisions where only one exposure occurred. The minimal or absent cervical paraspinal muscle response observed in experienced subjects exposed to whiplash-like perturbations (Gutierrez, 1978; Ono et al., 1997) and

the clear presence of this response in novice subjects at similar perturbation intensities (Brault et al., 2000) suggests that habituation may have contaminated some of the published human subject data.

In addition to the possible presence of habituation in some of the published whiplash-biomechanics data, different levels of warning regarding the exact time of the perturbation have been used. Information regarding the timing of a low-intensity whiplash-like perturbation has been shown to both affect and not affect the muscle and kinematic responses of repeatedly-exposed subjects (Magnusson et al., 1999; Kumar et al., 2000; Kumar et al., 2001). The question of whether precise knowledge of perturbation timing affects the magnitude of the response after habituation or the habituation process itself remains unanswered.

The goal of this study was to quantify the effect of habituation on the muscle and kinematic responses during multiple, sequential, whiplash-like perturbations and the degree to which the process of habituation was affected by awareness regarding the exact timing of the perturbation. Two levels of awareness were used: alerted subjects, who knew the exact time of the perturbation, and unalerted subjects, who knew that a perturbation would occur, but not its exact time. Based on previous studies in which the habituation of reflexive responses has been observed, it was hypothesized that the muscle responses of subjects exposed to sequential whiplash-like perturbations would habituate, and that changes in the muscle response would result in changes in the kinematic response. Previous studies have also shown a gender difference in the amplitude of the muscle and kinematic response to whiplash perturbations (Siegmund et al., 1997; Brault et al., 2000). To also assess whether gender-specific changes related to habituation occur, a similar number of female and male subjects were tested. In addition to identifying the number of perturbations that a subject can undergo before significant response changes occur, the current data might identify features of the initial response that improve our understanding of whiplash biomechanics and injury. A preliminary report of this study has been previously published in abstract form (Siegmund et al., 2001a).

4.2 Methods

4.2.1 Subjects

Forty-four subjects participated in the experiment. Physical characteristics for the subjects are given in Table 4.1. The subjects had no history of whiplash injury, medical conditions that impair sensory or motor function, or prolonged neck or back pain in the previous three years. All subjects gave their informed consent and were paid a nominal amount for their participation. The use of human subjects for this experiment was approved by the university's Ethics Review Board and the study conformed with the Declaration of Helsinki.

Table 4.1 Mean (S.D.) of subject age and physical characteristics.

	Female	Male
n	23	21
Age, yrs	23 (4)	28 (6)
Height, cm	165 (6)	174 (8)
Mass, kg	60 (9)	78 (18)

4.2.2 Instrumentation

Electromyographic (EMG) activity in the orbicularis oculi (OO), masseter (MAS), sternocleidomastoid (SCM) and cervical paraspinal (PARA) muscles was recorded bilaterally using 10 mm pre-gelled surface electrodes (H69P, Kendall-LTP, Huntington Beach, CA) and an Octopus AMT-8 amplifier (Bortec, Calgary, AB). Two uniaxial accelerometers (Kistler 8302B20S1; ±20g, Amherst, NY) and a single uniaxial angular rate sensor (ATA Sensors ARS-04E; ±100 rad/s, Albuquerque, NM) were strapped tightly to the midline of a subject's forehead, immediately above the glabella. The sensitive axes of the accelerometers were mutually orthogonal and oriented to measure horizontal (x') and vertical (z') acceleration in the mid-sagittal plane (Figure 4.1). The rotational axis of the angular rate sensor was oriented mediolaterally to measure flexion and extension motion in the sagittal plane. In some subjects, torso acceleration was measured using an uniaxial accelerometer (Kistler 8302B20S1; ±20g, Amherst, NY) applied with adhesive over the manubrium about 2 cm below its superior margin. Sled acceleration was measured using an uniaxial accelerometer (Sensotec JTF3629-05; ±10g, Columbus, OH) oriented horizontally along the axis of motion. Displacement was measured using an Optotrak motion analysis system (Northern Digital, Waterloo, ON) with markers placed over the left mastoid process and midline at the subject's forehead, vertex of the head, manubrium and C7 spinous process (Figure 4.1). A sixth marker was placed on the left seat hinge. A force transducer (Artech S-Beam SS20210, ±2 kN, Riverside, CA) was used to measure reaction loads during normalizing contractions of the SCM and PARA muscles. EMG signals were bandpass filtered at 10 Hz to 1 kHz and transducer signals were low-pass filtered at 1 kHz before being simultaneously sampled at 2 kHz and stored for subsequent analysis. Optotrak data were acquired at 200 Hz per marker.

4.2.3 Test Procedures

Subjects were seated in an automobile seat obtained from the front passenger location of a 1991 Honda Accord. The head restraint was removed from the top of the seat back to eliminate the

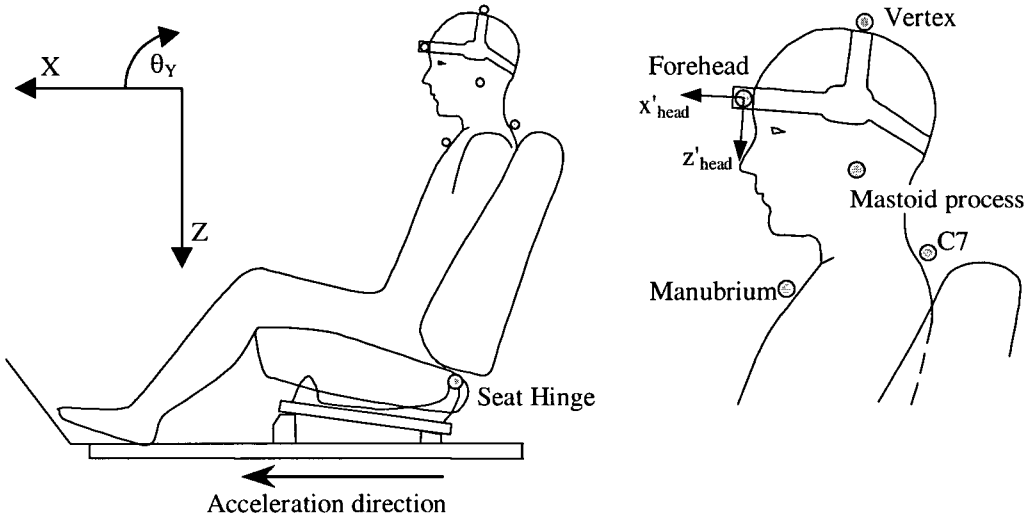
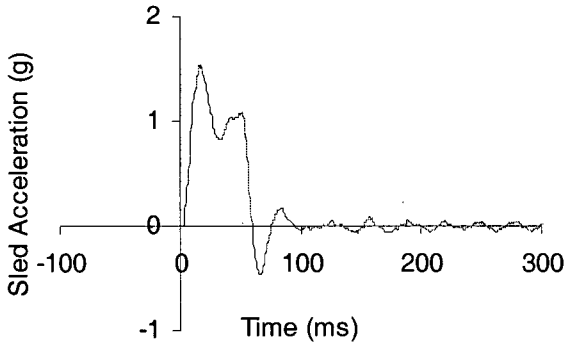


Figure 4.1 Schematic of the test configuration showing the locations of the Optotrak markers (shaded circles), the lab reference frame (X, Z) and the head reference frame (x', z'). The initial orientation of the head reference frame was determined by how the head band fit the subject and varied between 12 ± 4 deg relative to the lab reference frame.



Properties	Mean (S.D.)
Peak acceleration (g)	1.51 (0.02)
Time to peak acceleration (ms)	16.5 (0.9)
Pulse duration (ms)	59.9 (0.2)
Speed change (m/s)	0.498 (0.003)
Average acceleration (g)	0.898 (0.004)

Figure 4.2 Sample acceleration pulse and descriptive statistics of selected pulse properties.

potential for an externally applied load to the head during the perturbation. The seat was mounted on a custom fabricated sled powered by a feedback-controlled linear induction motor (Kollmorgen IC55-100A7, Kommack, NY). The sled generated no pre-perturbation signals, either audible or mechanical, which subjects could use to predict onset of the perturbation. To install and connect some of the instrumentation, subjects were seated on the sled about 15 minutes before their first perturbation. This period of time allowed the subjects sufficient time to adopt a comfortable seated posture. Prior to testing, subjects were instructed to sit normally, face forward, rest their forearms on their lap and to relax their face and neck muscles. Each subject was exposed to eleven perturbations which accelerated the subject horizontally forward (Figure 4.2).

Subjects were randomly assigned to one of two groups: alerted or unalerted. Prior to each perturbation, subjects in the alerted group received a countdown consisting of two auditory tones (1000 Hz, 80 dB, 40 ms duration) spaced 1 s apart. The perturbation then occurred 1 s after the second tone, coincident with the expected time of a third tone had one been presented. Subjects in the unalerted group were told to expect their first perturbation within 60 seconds of the start of the experiment and then received a perturbation about 15 seconds later. The median time between the first and second perturbations of all subjects was 2.2 minutes (range 1.2 to 9.7). The time between the remaining ten perturbations varied randomly (uniform distribution) between 20 and 30 seconds. Subjects did not receive a practice or demonstration perturbation prior to their first perturbation.

Prior to the above protocol, seated subjects performed sub-maximal isometric contractions in flexion and extension to generate normalizing data for the SCM and PARA muscles respectively. A strap attached to the load cell was placed around a subject's head and its length adjusted to ensure the subject's head was neutrally positioned. The strap was located immediately above the glabella for flexion contractions and at the height of the external occipital protuberance for extension contractions. Subjects were instructed to generate a force of 50 N with visual feedback, first in flexion and then in extension. EMG and load cell data were acquired for 5 s during each contraction.

4.2.4 Data Reduction

A subject's initial position was determined from the Optotrak data immediately preceding the perturbation. Initial head position was defined in the laboratory reference frame using the horizontal (X-axis; positive forward) and vertical (Z-axis; positive downward) positions of the mastoid process relative to the seat hinge (Figure 4.1). Initial head angle in the sagittal plane was determined from the forehead and vertex markers and reported relative to the positive X-axis ($+\theta_y$ rotation corresponded to extension). Initial torso position was defined using the horizontal and vertical positions of the midpoint between the manubrium and C7 spinous process markers relative to the seat hinge, and torso

angle relative to the horizontal plane was determined using the same two markers. The RMS accuracy of the position measurements from the Optotrak system was less than 0.1 mm, and based on marker separation, the RMS accuracy of the calculated angles was less than 0.1 degrees.

The onset of head and torso movement, amplitude of peak head accelerations and the time of the peak head and torso accelerations were determined directly from the accelerometer data. The algorithm used to determine onset is developed in Appendix A. Forehead acceleration data were not resolved into the lab reference frame. Peak retraction, defined as the maximum horizontal translation of the top of the cervical spine rearward relative to the bottom of the cervical spine, was estimated using the maximum relative horizontal displacement in the lab reference frame between the Optotrak markers on the mastoid and manubrium. Peak angular velocity of the head was determined from the angular rate sensor data after it had been digitally compensated to reduce the sensor's high-pass frequency to 0.002 Hz (Laughlin, 1998). Angular acceleration was then computed by finite differences (5 ms window) from the compensated angular velocity data. Total head angular displacement was determined from the Optotrak markers located at the forehead and vertex. Horizontal and vertical accelerations at the mastoid process were computed by double differentiating the Optotrak data. This technique produced results within 0.1g of the values computed using a rigid body transformation of the linear and angular accelerations measured at the forehead and was preferred because it relied on data from only one Optotrak marker rather than data from two accelerometers, an angular rate sensor and two Optotrak markers needed for the rigid body calculation. Mastoid accelerations were computed in the lab reference frame.

Pre-stimulus noise in each channel of the EMG data was quantified using the root mean squared (RMS) amplitude of the EMG signal over the 100 ms preceding the perturbation. EMG onset was defined as the time at which the RMS amplitude, computed from the raw EMG data using a moving 20 ms window, reached 10 percent of its maximum value and was then confirmed visually. For each muscle, the RMS amplitude of the EMG signal was calculated for the interval between EMG onset and peak head extension angle. The corresponding pre-stimulus noise for each muscle was then subtracted from this quantity. The SCM and PARA muscle EMG amplitudes were normalized by the RMS amplitude obtained during the 5 s sub-maximal contraction for the corresponding muscle.

4.2.5 Statistical Analysis

For each kinematic dependent variable, a three-way analysis of variance (ANOVA) was used to assess differences related to level of awareness (alerted, unalerted), gender (female, male) and number of trials (one through eleven) as the repeated measure. Electromyographic variables were first assessed using a four-way ANOVA with muscle side (left, right) included as a second repeated

measures variable. After confirming that there were no statistically significant effects related to muscle side, the mean of the left and right muscle data were computed and analyzed using a three-way ANOVA for awareness, gender and trial only. All statistical tests were performed using Statistica (v.5.1, Statsoft Inc., Tulsa, OK) and a significance level of α=0.05. Post-hoc comparisons amongst the eleven trial levels were performed using a Tukey's honest significant difference test. A Scheffé test was not used because once habituation was identified, an overly conservative test might mask habituation and produce artificial confidence in trials where significant habituation had occurred. Post-hoc testing of interaction terms that included the repeated-measures trial variable was performed by running a separate two-way ANOVA on each trial to ascertain which trials were responsible for the interaction effect.

Instrumentation problems resulted in incomplete data for some trials. Because of the repeated-measures nature of the analysis, a lost trial potentially eliminated that subject's entire data set from the analysis. To minimize the effect of lost data, subject data were deleted on a variable-by-variable basis if lost. Optotrak data were lost in one subject due to synchronization problems. A mastoid marker was not present for the first subject, and other mastoid data were occasionally deleted due to marker dropout, excessive vibration or, in the case of vertical mastoid acceleration, the absence of an initial upward component to the acceleration. A grounding problem contaminated the EMG data of one subject and onset could not be reliably determined for OO, MAS and PARA muscles of three subjects due to excessive sled-induced noise.

4.3 Results

Changes in many of the dependent variables were observed over the eleven trials. In all cases, post-hoc testing showed no significant variation over the last 5 trials and therefore the descriptive data reported in the tables were limited to the first trial and the average of the last five trials. Descriptive data for each variable and each trial are included in Appendix B.

4.3.1 Initial Position

Significant changes were observed over the eleven trials in all six variables used to define a subject's initial position (Table 4.2). Averaged across all subjects, the initial vertical position of the mastoid process shifted 3 mm downward ($F_{10,380}$=23.7, $p<0.0001$) over the course of eleven perturbations. Post-hoc analysis revealed that this vertical displacement occurred entirely between the first and third trials. The initial horizontal position of the mastoid process was significantly different between trials ($F_{10,380}$=4.00, $p<0.0001$), however it did not shift uniformly in one direction. The average horizontal position of the mastoid moved forward over the first three trials and then rearward

Table 4.2 Mean (S.D.) of the initial position and angle of the head and torso. Upper portion of table summarizes data as a function of awareness (unalerted, alerted), gender (female, male) and trial number (1 to 11). Trial data are summarized here for the first trial and the average of the last five trials only. Positions are relative to an origin at the seat hinge; angles are relative to the horizontal. Lower portion of table summarizes the results (F-statistics) of separate 3-way ANOVAs using awareness, gender and trial number as independent variables. The eleven trials were considered separately in the ANOVA. X, horizontal position, +ve forward; Z, vertical position, +ve downward; N, number of subjects in analysis; A, awareness; G, gender; T, trial.

		Mastoid process		Head	Manubrium-C7 midpoint		Torso
		X (mm)	Z (mm)	angle (deg)	X (mm)	Z (mm)	Angle (deg)
First Trial							
Female	Unalerted	11 (16)	-642 (23)	7.1 (1.9)	21 (14)	-552 (19)	-15.9 (4.1)
	Alerted	7 (19)	-641 (35)	7.9 (3.4)	21 (11)	-559 (28)	-17.6 (4.0)
Male	Unalerted	28 (12)	-661 (36)	9.4 (4.0)	38 (6)	-572 (35)	-15.3 (3.3)
	Alerted	21 (18)	-672 (25)	9.6 (4.8)	28 (15)	-584 (25)	-17.2 (5.3)
Last 5 trials							
Female	Unalerted	9 (14)	-639 (22)	8.7 (4.3)	17 (11)	-549 (17)	-15.4 (5.3)
	Alerted	6 (18)	-637 (36)	9.1 (3.6)	16 (10)	-557 (29)	-16.3 (4.4)
Male	Unalerted	26 (20)	-658 (36)	8.9 (7.3)	37 (12)	-570 (35)	-15.2 (3.7)
	Alerted	17 (16)	-668 (26)	10.7 (5.6)	25 (16)	-582 (24)	-16.3 (4.9)
N		42	42	43	38	38	38
ANOVA F-statistics							
Aware							
Gender		7.76**	6.90*		13.5***	6.66*	
Trial		4.00****	23.7****	2.08*	7.57****	8.35****	5.12****
Aware × Gender							
Aware × Trial							
Gender × Trial							
A × G × T							

* p<0.05, ** p<0.01, *** p<0.001, ****p<0.0001

over the remaining trials. As a result, the post-hoc analysis revealed that no one trial was significantly different from the first trial, however the horizontal position of the mastoid position in the third trial was significantly further forward than in each of the last four trials ($p<0.05$). Despite achieving statistical significance, the average initial head angle deceased (was flexed further forward) by less than one degree over the eleven trials ($F_{10,390}=2.08$, $p=0.025$). No one trial was significantly different from the first trial.

The average position of the midpoint between the manubrium and the C7 spinous process shifted 3 mm rearward ($F_{10,340}=7.57$, $p<0.0001$) and 2 mm downward ($F_{10,340}=8.35$, $p<0.0001$) over the eleven perturbations. The horizontal position of the manubrium-C7 midpoint was not significantly different from the first trial until the sixth trial, whereas the vertical displacement occurred entirely between the first and second trials. Like head angle, the initial manubrium-C7 angle was flexed further forward by less than one degree over the eleven perturbations ($F_{10,340}=5.12$, $p<0.0001$), although no one trial was significantly different from the first trial.

A gender difference consistent with the male subjects being larger than the female subjects was observed in the four variables describing initial position, but not in the two variables describing initial head and torso orientation. The mastoid process of the average male subject was located 15 mm forward ($F_{1,38}=7.76$, $p=0.008$) and 25 mm above ($F_{1,38}=6.90$, $p=0.012$) the mastoid process of the average female subject. The midpoint between the manubrium and C7 markers of the average male subject was 12 mm forward ($F_{1,34}=13.5$, $p=0.0008$) and 22 mm above ($F_{1,34}=6.66$, $p=0.014$) the same point on the average female subject.

4.3.2 Kinematic Response

The perturbation produced a stereotypical response in all subjects (Figure 4.3). The torso was accelerated forward first, followed by the head, which was accelerated linearly upward and forward, and angularly into extension. During their first perturbation, all subjects responded by rapidly restoring their upright head position. During later trials, some subjects altered their response and did not attempt to restore their upright head position until much later. In these subjects, head angle and retraction remained large for a considerable period of time after the perturbation (see middle panel of Figure 4.3). In most subjects who responded in this manner, a clear peak was still visible in both the retraction and head angle data (as seen in Figure 4.3), however, in six subjects, no clearly defined peak was present in one or more of their later trials (Figure 4.4). Since their response represented an extreme adaptation to the stimulus, these six subjects were removed from the data set and the remaining 38 subjects were used for the rest of the analysis.

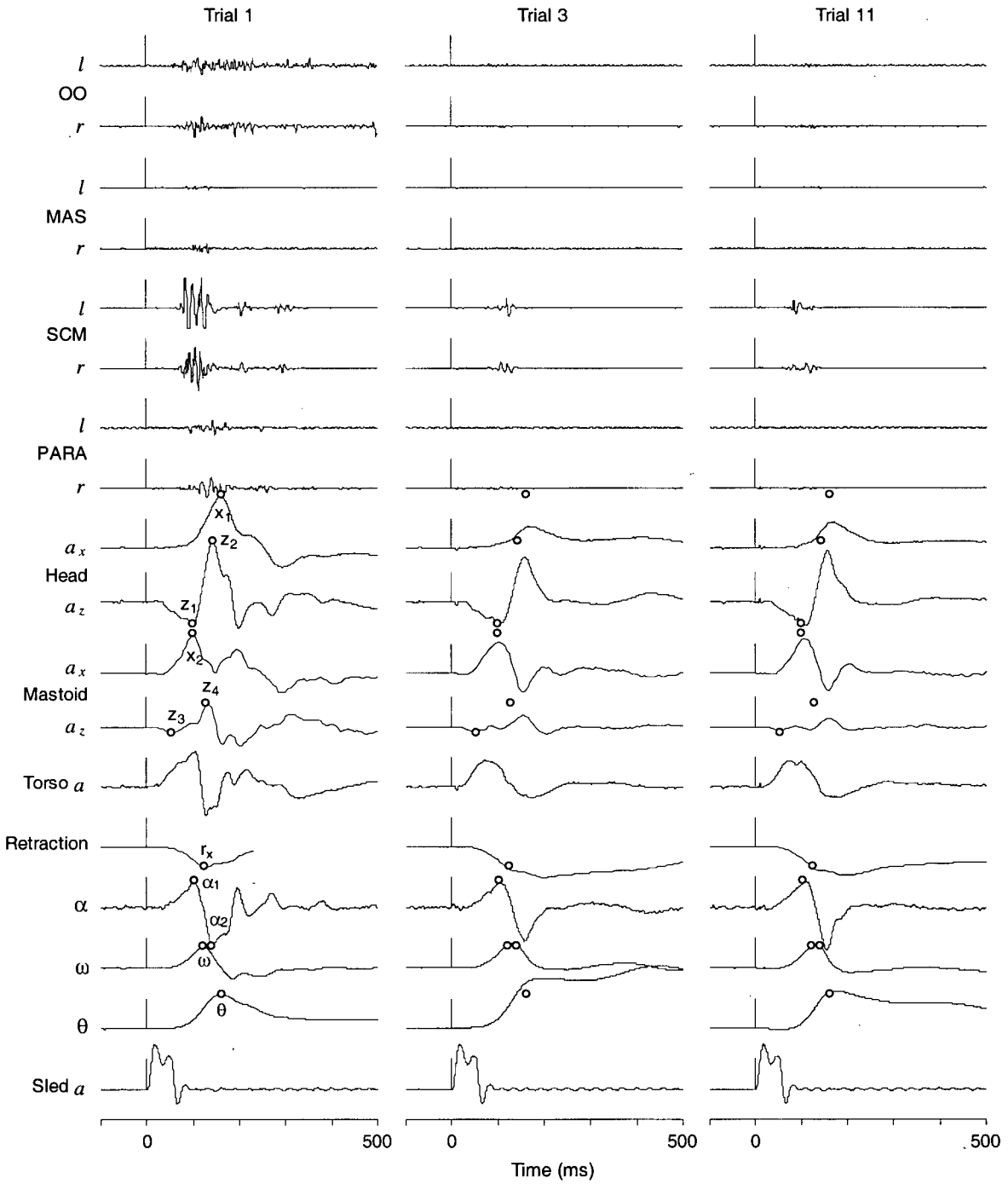


Figure 4.3 Sample data from the first, third and eleventh trial of a single subject who exhibited habituation of their muscle response and corresponding changes in their kinematic response. Labeled hollow circles in the first trial represent kinematic peaks used for subsequent analysis. Similarly-located circles in the other trials highlight changes due to habituation. The vertical scale bars are aligned with perturbation onset and are equal to 1g, 25 mm, 100 rad/s², 5 rad/s and 10 deg. OO, orbicularis oculi; MAS, masseter; SCM, sternocleidomastoid; PARA, cervical paraspinal, l, left; r, right; *a*, linear acceleration, subscript *x* and *z* refers to the x- and z-directions; α, head angular acceleration; ω, head angular velocity; θ, head angle.

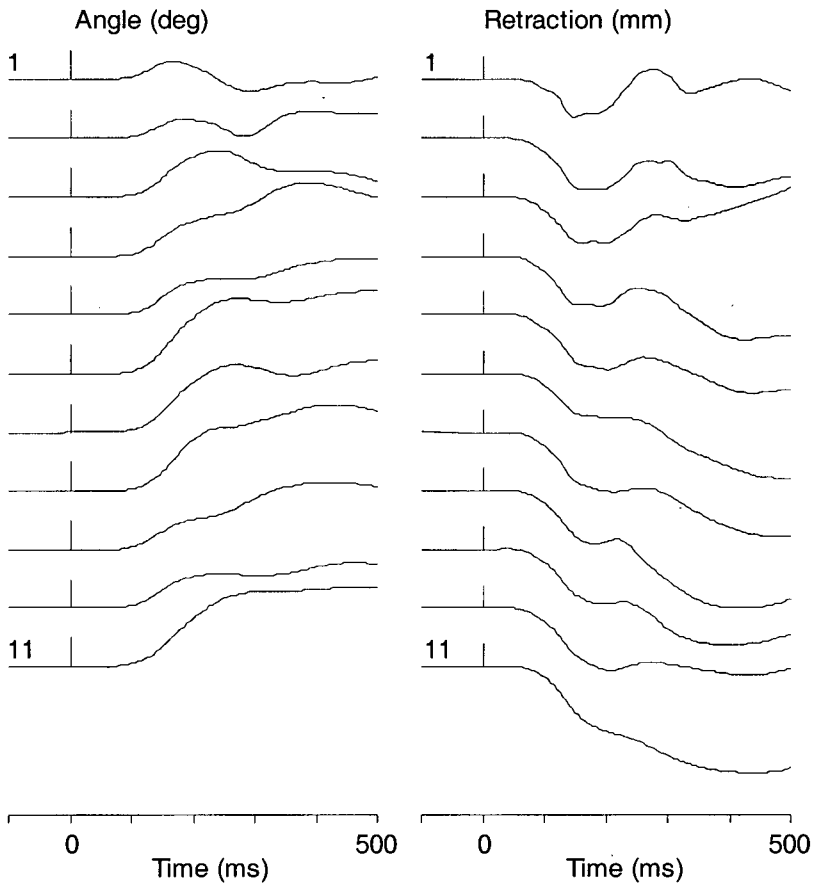


Figure 4.4 Head angle (left) and retraction (right) data for trials 1 (top) through 11 (bottom) of a subject who adopted a different response strategy. The data indicated that the subject did not attempt to immediately re-establish an upright head position after the perturbation. The vertical scale bars are aligned with perturbation onset and are equal to 10 deg and 10 mm.

Neither the onset of torso acceleration nor the time of peak torso acceleration varied with awareness, gender or number of trials. Averaged across all conditions and trials, the onset of torso acceleration occurred 20 ± 2 ms after the onset of the perturbation and peak torso acceleration occurred 77 ± 11 ms after the onset of the perturbation. Evidence of head acceleration appeared first in the vertically-mounted accelerometer at the forehead and the analysis of these onset times showed a significant awareness × trial interaction effect ($F_{10,340}$=2.00, p=0.033). Post-hoc testing revealed that an awareness effect developed after the fourth trial. Over the first four trials, the average time from perturbation onset to head acceleration onset was 29 ± 5 ms. In the remaining seven trials, the overall average remained the same, however, the onset of head acceleration occurred an average of 4 ms later in alerted subjects than in unalerted subjects.

Amongst the peak kinematic parameters used to characterize the response (labeled with hollow circles in Figure 4.3), only the positive (downward) peak in the vertical forehead acceleration (z_2) did not exhibit a significant variation in amplitude with repeated exposure (Table 4.3). Relative to the first trial, repeated exposure to the perturbation produced a decrease in the peak amplitudes of horizontal forehead acceleration (x_1, $F_{10,340}$=42.2, p<0.0001), horizontal mastoid acceleration (x_2, $F_{10,310}$=3.28, p=0.0005), downward mastoid acceleration (z_4, $F_{10,300}$=14.5, p<0.0001) and the angular head acceleration in flexion (α_2, $F_{10,340}$=2.06, p=0.027). In contrast, an increase in the peak amplitude with repeated exposure was observed in the vertical forehead acceleration (z_1, $F_{10,340}$=4.13, p<0.0001), retraction (r_x, $F_{10,290}$=8.47, p<0.0001), head angular acceleration in extension (α_1, $F_{10,340}$=3.10, p=0.0009), head angular velocity in extension (ω, $F_{10,340}$=8.46, p<0.0001) and peak head extension angle (θ, $F_{10,330}$=11.1, p<0.0001). A variable pattern, which consisted of an increased amplitude by the third trial followed by a return to baseline, was observed in the initial upward acceleration of the mastoid process (z_3, $F_{10,290}$=3.36, p=0.0004). The normalized habituation pattern for these variables and the percentage change between the first trial and the average of the last five trials are summarized in Figure 4.5.

The statistical analysis of two amplitude variables – horizontal forehead acceleration (x_1) and retraction (r_x) also produced significant awareness × trial interaction terms. Post-hoc examination of these data showed that there were no awareness effects present in any one of the eleven trials, but that awareness affected the rate of habituation over the first three trials for peak horizontal forehead acceleration (x_1) and the first four trials for peak retraction (r_x). In both variables, habituation occurred more rapidly in the unalerted subjects than in the alerted subjects. The initial upward acceleration of the forehead (z_1) was the only parameter in which peak amplitude varied with gender ($F_{1,34}$=4.99, p<0.032). Across all trials and awareness conditions, the peak amplitude of the upward forehead acceleration was 18 percent larger in male subjects than in female subjects (male:

Table 4.3 Mean (S.D.) of the amplitude of selected peaks in the kinematic data. Upper portion of table summarizes data as a function of awareness (unalerted, alerted), gender (female, male) and trial number (1 to 11). Trial data are summarized here for the first trial and the average of the last five trials only. Lower portion of table summarizes the results (F-statistics) of separate 3-way ANOVAs using awareness, gender and trial number as independent variables. The eleven trials were considered separately in the ANOVA. Peaks are labeled with hollow circles in the left panel of Figure 4.3. N, number of subjects in analysis; A, awareness; G, gender; T, trial.

		Linear kinematics							Angular kinematics			
		Forehead			Mastoid process			Retraction	Head			
		x_1 (g)	z_1 (g)	z_2 (g)	x_2 (g)	z_3 (g)	z_4 (g)	r_x (mm)	α_1 (rad/s²)	α_2 (rad/s²)	ω (rad/s)	θ (deg)
First Trial												
Female	Unalerted	1.20 (0.22)	-0.60 (0.13)	1.63 (0.62)	1.35 (0.41)	-0.19 (0.10)	0.66 (0.35)	-20.4 (3.8)	75 (23)	-145 (53)	3.2 (1.3)	12 (5)
	Alerted	1.11 (0.17)	-0.64 (0.21)	1.51 (0.41)	1.30 (0.40)	-0.19 (0.06)	0.51 (0.19)	-21.5 (3.1)	79 (31)	-133 (45)	3.5 (0.9)	12 (2)
Male	Unalerted	1.30 (0.37)	-0.70 (0.22)	1.64 (0.25)	1.17 (0.27)	-0.22 (0.08)	0.55 (0.24)	-20.4 (3.9)	68 (26)	-112 (24)	3.1 (0.6)	11 (2)
	Alerted	1.12 (0.33)	-0.74 (0.13)	1.50 (0.38)	1.07 (0.19)	-0.18 (0.07)	0.36 (0.10)	-19.6 (2.5)	67 (12)	-99 (29)	3.1 (0.5)	11 (2)
Last 5 trials												
Female	Unalerted	0.84 (0.14)	-0.67 (0.14)	1.61 (0.73)	1.12 (0.23)	-0.19 (0.09)	0.43 (0.25)	-23.9 (4.4)	80 (20)	-132 (72)	3.7 (1.1)	14 (5)
	Alerted	0.86 (0.14)	-0.69 (0.16)	1.49 (0.34)	1.21 (0.27)	-0.20 (0.08)	0.36 (0.14)	-22.6 (6.1)	80 (16)	-119 (42)	3.6 (0.6)	13 (3)
Male	Unalerted	0.89 (0.21)	-0.77 (0.26)	1.43 (0.32)	1.06 (0.16)	-0.24 (0.11)	0.34 (0.12)	-23.6 (3.9)	76 (20)	-103 (31)	3.6 (0.5)	14 (3)
	Alerted	0.88 (0.25)	-0.80 (0.15)	1.42 (0.46)	1.08 (0.16)	-0.19 (0.08)	0.28 (0.09)	-20.5 (4.0)	75 (15)	-94 (40)	3.5 (0.8)	13 (5)
N		38	38	38	35	33	34	33	38	38	38	37
F-statistics												
Aware												
Gender			4.99*									
Trial		42.2****	4.13****		3.28***	3.36***	14.5****	8.47****	3.10***	2.06*	8.46****	11.1****
Aware × Gender												
Aware × Trial		2.47**						1.87*				
Gender × Trial												
A × G × T												

* p<0.05, ** p<0.01, *** p<0.001, **** p<0.0001

Table 4.4 Mean (S.D.) of the time of peak amplitude in the kinematic data. Upper portion of table summarizes data as a function of awareness (unalerted, alerted), gender (female, male) and trial number (1 to 11). Trial data are summarized here for the first trial and the average of the last five trials only. Lower portion of table summarizes the results (F-statistics) of separate 3-way ANOVAs using awareness, gender and trial number as independent variables. The eleven trials were considered separately in the ANOVA. Peaks are labeled with hollow circles in the left panel of Figure 4.3. N, number of subjects in analysis; A, awareness; G, gender; T, trial.

		Forehead			Mastoid process			Retraction	Head			
		x_1 (ms)	z_1 (ms)	z_2 (ms)	x_2 (ms)	z_3 (ms)	z_4 (ms)	r_x (ms)	α_1 (ms)	α_2 (ms)	ω (ms)	θ (ms)
First Trial												
Female	Unalerted	169 (14)	88 (10)	170 (8)	115 (12)	58 (9)	124 (14)	149 (18)	110 (18)	168 (6)	131 (15)	177 (14)
	Alerted	171 (13)	93 (23)	169 (16)	112 (15)	62 (4)	125 (9)	160 (22)	108 (16)	173 (10)	136 (13)	184 (13)
Male	Unalerted	167 (9)	88 (11)	154 (10)	113 (9)	61 (7)	131 (12)	158 (22)	102 (5)	158 (10)	129 (6)	174 (10)
	Alerted	165 (15)	91 (9)	156 (14)	111 (9)	70 (10)	152 (29)	168 (16)	98 (8)	155 (14)	126 (10)	178 (18)
Last 5 trials												
Female	Unalerted	178 (18)	95 (12)	172 (21)	110 (9)	58 (11)	129 (24)	173 (34)	107 (11)	170 (16)	135 (10)	190 (21)
	Alerted	172 (16)	92 (14)	164 (15)	102 (10)	61 (5)	122 (13)	171 (30)	104 (12)	162 (16)	131 (12)	185 (20)
Male	Unalerted	174 (19)	93 (15)	164 (15)	111 (10)	59 (7)	137 (24)	186 (20)	107 (7)	162 (18)	134 (10)	196 (27)
	Alerted	167 (24)	96 (8)	158 (15)	107 (8)	70 (10)	157 (28)	178 (20)	101 (7)	155 (16)	128 (14)	192 (32)
N		38	38	38	35	33	34	33	38	38	38	37
F statistics												
Aware						6.64*						
Gender						6.06*	8.94**			4.70*		
Trial		3.62***			3.53***			10.0****				7.67****
Aware × Gender						4.38*						
Aware × Trial												1.87*
Gender × Trial									2.25*			
A × G × T												

* p<0.05, ** p<0.01, *** p<0.001, **** p<0.0001

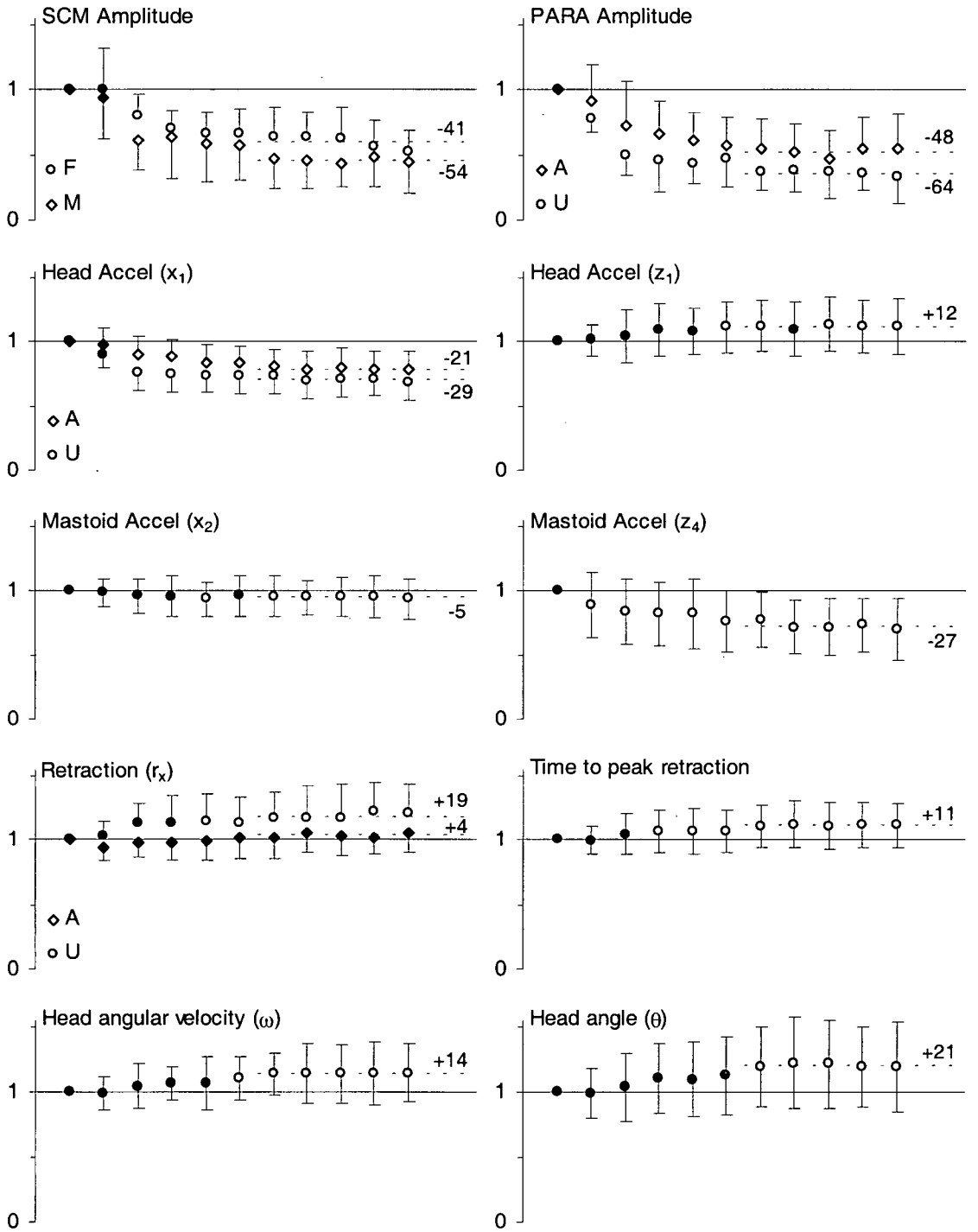


Figure 4.5 Mean (S.D.) of some normalized dependent variables over the eleven exposures. Subject data were normalized to the peak amplitude or the time of peak amplitude observed in their first trial before the normalized means (S.D.) depicted in this figure were calculated. Interactions with gender and awareness level shown where the effect was significant. Hollow markers indicate trials significantly different from the first trial. Number at right indicates the percentage change of the last 5 trials relative to the first trial. F, female; M, male; A, alerted; U, unalerted.

-0.77 ± 0.20g; female: -0.65 ± 0.14g). This gender effect was eliminated by the normalization process and therefore not shown in Figure 4.5.

Overall, repeated exposure to the perturbation affected the timing of fewer kinematic peaks than it did their amplitude (Table 4.4). Relative to the first perturbation, the mean time of the peak response over the last five perturbations was an average of 4 ms later for horizontal forehead acceleration (x_1, $F_{10,340}=3.62$, $p=0.00013$), 16 ms later for retraction (r_x, $F_{10,290}=10.0$, $p<0.0001$), and 12 ms later for head angle (θ, $F_{10,330}=7.67$, $p<0.0001$). Peak horizontal mastoid acceleration (x_2), however, occurred an average of 6 ms earlier over the last five trials when compared to the first trial ($F_{10,310}=3.53$, $p=0.0002$).

Both peaks in the vertical mastoid acceleration (z_3 and z_4) occurred earlier in male subjects than in female subjects. Averaged across all trials and awareness conditions, female subject reached peak downward mastoid acceleration 22 ms earlier than male subjects (z_4, $F_{1,30}=8.94$, $p=0.006$). The gender difference observed in the earlier upward acceleration of the mastoid process (z_3) was tempered by a gender × awareness interaction ($F_{1,29}=4.38$, $p=0.045$). Post-hoc analysis showed that peak acceleration occurred 11 ms earlier in alerted male subjects than the combined average of the other three groups ($p<0.02$). A gender effect was also present in the time to both peaks in the angular acceleration of the head. Peak angular acceleration in flexion (α_2) occurred 8 ms earlier in male subjects than female subjects when averaged across all trials and awareness conditions ($F_{1,34}=4.70$, $p=0.037$). A gender × trial effect in the extension angular acceleration (α_1) indicated that the peak angular acceleration occurred progressively earlier in female subjects and progressively later in male subjects with repeated perturbations (α_1, $F_{10,340}=2.25$, $p=0.015$). The difference between the first trial and mean of the last five trials was 4 ms (earlier) in female subjects and 4 ms (later) in male subjects.

4.3.3 Muscle Response

A consistent and stereotyped muscle response was observed in all subjects (Figure 4.3). Repeated exposure to the perturbation produced small isolated changes in muscle activation times and large, more uniform changes in muscle amplitude (Table 4.5). For masseter onset times, a significant 3-way interaction effect was present ($F_{10,300}=2.42$, $p=0.009$). Post-hoc analyses showed that an awareness × gender interaction, which consisted of activation 14 ms earlier in unalerted female subjects than in unalerted male subjects ($p=0.029$), was present in the first trial but eliminated by the second trial. A simpler interaction between gender and trial was present in the activation times of the SCM muscles ($F_{10,330}=2.35$, $p=0.011$). Post-hoc analyses revealed that an initial gender difference, in which female subjects activated their SCM muscles 6 ms earlier than male subjects ($p=0.001$), was no longer significant in the sixth and subsequent trials.

Table 4.5 Mean (S.D.) of EMG onset time and normalized RMS magnitude. Upper portion of table summarizes data as a function of awareness (unalerted, alerted), gender (female, male) and trial number (1 to 11). Trial data are summarized here for the first trial and the average of the last five trials only. Lower portion of table summarizes the results (F-statistics) of separate 3-way ANOVAs using awareness, gender and trial number as independent variables. The eleven trials were considered separately in the ANOVA. RMS, root-mean square; OO, orbicularis oculi; MAS, masseter; SCM, sternocleidomastoid; PARA, cervical paraspinal; N, number of subjects in analysis; A, awareness; G, gender; T, trial.

		Onset (ms)				RMS Magnitude	
		OO	MAS	SCM	PARA	SCM	PARA
First Trial							
Female	Unalerted	66 (8)	67 (4)	68 (5)	78 (8)	0.21 (0.09)	0.17 (0.09)
	Alerted	71 (13)	81 (12)	69 (5)	76 (7)	0.17 (0.04)	0.12 (0.08)
Male	Unalerted	64 (8)	81 (9)	74 (6)	81 (7)	0.29 (0.11)	0.19 (0.13)
	Alerted	67 (8)	74 (8)	75 (5)	75 (4)	0.31 (0.31)	0.1 (0.06)
Last 5 trials							
Female	Unalerted	68 (7)	72 (7)	70 (6)	78 (6)	0.1 (0.05)	0.06 (0.06)
	Alerted	71 (11)	76 (12)	67 (9)	77 (6)	0.11 (0.03)	0.06 (0.04)
Male	Unalerted	64 (9)	74 (8)	72 (6)	75 (6)	0.12 (0.07)	0.07 (0.05)
	Alerted	71 (10)	72 (10)	69 (8)	76 (6)	0.15 (0.15)	0.05 (0.03)
N		34	34	37	34	36	33
ANOVA F-statistics							
Aware							
Gender							
Trial						30.9****	28.5****
Aware × Gender							
Aware × Trial							4.41****
Gender × Trial				2.35*		3.03**	
A × G × T			2.42**				

* p<0.05, ** p<0.01, ****p<0.0001

The EMG amplitude in both neck muscles diminished with repeated exposures (Table 4.5, Figure 4.5), however the analyses showed different interaction effects in each of the two muscle groups. In addition to a reduction in SCM amplitude with multiple trials, a gender × trial interaction effect ($F_{10,320}$=3.03, p=0.0011) was also present. Post-hoc analyses showed that a gender difference in the EMG amplitude of the SCM muscle present in the first two trials was not present in the third and subsequent trials. The average amplitude of the SCM muscle response in females during the first two perturbations was 45 percent smaller than the SCM muscle response in males (p<0.036). Compared to the first trial, the mean SCM amplitude of the last five trials was reduced by 41 percent in female subjects and 54 percent in male subjects (Figure 4.5).

The amplitude of the PARA muscle response was also strongly affected by repeated exposure to the perturbation, however, the size of the amplitude reduction was different for the two awareness conditions ($F_{10,290}$=4.41, p<0.0001). Post-hoc analyses showed that a difference in the PARA amplitude existed between the alerted and unalerted subjects in the first two trials (p<0.031). This difference was no longer significant in the third and subsequent trials. The average PARA amplitude for the alerted subjects over the first two trials was 36 percent smaller than observed in the unalerted subjects. Compared to the first trial, the mean PARA amplitude over the last five trials was reduced by 48 percent in alerted subjects and 64 percent in unalerted subjects.

4.4 Discussion

The results of the current study showed clear evidence of habituation in the muscle responses and corresponding changes in the kinematic responses of human subjects exposed to sequential, low-intensity, whiplash-like perturbations. Habituation was observed in the amplitude of both neck muscles and related changes were observed in all but one of the eleven kinematic parameters examined here. Changes in muscle onset latencies and the time of the peak kinematic responses also varied, though in considerably fewer parameters than did amplitude. Depending on the variable, the effect of habituation became visible over a differing number of trials (Figure 4.5). In some variables, a significant decrease in response magnitude was already present by the second trial. In other variables, particularly some kinematic variables, these changes did not reach statistical significance until the fifth or sixth trial. For all of the variables that exhibited significant changes, the last five of the eleven trials were not significantly different from each other – a finding that suggested, but did not prove, that a stabilized response was achieved over the eleven perturbations used for this experiment. Based on the difference between the response observed in the first trial and the average of the responses observed over the last five trials, average reductions in the amplitude of the neck muscle responses varied between 41 and 64 percent. Average changes in the amplitude of the

kinematic responses varied between a 21 percent increase in peak head extension angle (θ) and a 29 percent reduction in peak horizontal head acceleration measured at the forehead (x_1). These large changes in both the muscle and kinematic responses with repeated exposure to the perturbation supported the primary hypothesis of this study and indicated that the process of habituation was a large potential confounder in the study of whiplash injury biomechanics using human subjects and repeated exposures.

In addition to the changes observed in the magnitude and timing of both the muscle and kinematic responses, gender and timing-awareness differences also underwent changes during the habituation process. Gender differences in the SCM onset, SCM amplitude and time of peak angular head acceleration (α_1) present in the first few trials were not evident in later trials. Similarly, an awareness difference in the PARA amplitude present during the first few trials was also not present in later trials. In contrast to the elimination of significant effects, an awareness-related difference in the onset time of vertical forehead acceleration not present initially developed after four trials. These findings indicated that gender or awareness differences that might be important to the understanding of whiplash injury might not be discernible in data acquired from subjects who have habituated to the stimulus. Alternatively, differences found in the data after habituation has occurred might not be relevant to whiplash injury. Both effects highlight the potential perils of using data from subjects who have habituated to the stimulus to investigate the biomechanics of whiplash injury.

The largest reductions in amplitude with repeated perturbations occurred in the neck muscle responses. Repeated perturbations produced a statistically significant decrease in muscle amplitude by the second exposure in the PARA muscles and by the third exposure in the SCM muscles. These responses subsequently appeared to stabilize at about 35 to 60 percent of their initial amplitude. Both the net change in muscle amplitude and the few trials over which this change occurred were consistent with previous reports of head, neck and shoulder muscle habituation in supine free-fall experiments (Bisdorff et al., 1994), acoustic startle experiments (Valls-Solé et al., 1997) and postural perturbation experiments (Woollacott et al., 1988; Allum et al., 1992).

These changes in neck muscle contraction levels have important biomechanical implications for the magnitude of head movement. The human cervical spine is a complex, multi-segmented structure that requires active muscle control to maintain its upright posture (Winters, 1988). Perturbations which disturb this upright posture produce dynamic changes in neck muscle activity aimed at controlling head movement and re-establishing a stable head position and orientation. Based on the current data, neck muscle activation appeared to serve two functions. First, a co-activation of the flexor muscles (represented by the SCM) and extensor muscles (represented by the PARA) appeared to be an attempt to stiffen the connection between the shoulders and head, and was similar

to a "strap down" strategy described by Nashner (1985). With repeated exposures, however, the need for this cocontraction appeared to diminish, leaving predominantly the second pattern – a task-specific flexor activation needed to overcome the induced posterior translation and extension rotation of the head with respect to the torso. Based on a similar adaptation pattern in both the SCM and PARA muscles during the current experiment, it appeared that habituation primarily diminished the co-contraction component of the neck muscle response. The slightly larger reduction observed in the PARA muscles was consistent with its antagonist role in the perturbation under study. Similar reductions, and in some cases abolition, of antagonist activity in the lower limb muscles during habituation to postural perturbations in standing have been observed by other researchers (Woollacott et al., 1988; Horak et al., 1989). Given that the neck requires muscle tone for static stability, complete abolition of the antagonist PARA muscle response would not be expected.

Most of the kinematic changes that occurred with habituation were consistent with a reduction in the neck stiffness secondary to a reduced level of neck muscle cocontraction. Larger amplitudes for the passively induced kinematics – angular head acceleration into extension (α_1), upward forehead acceleration (z_1), angular velocity in extension (ω), peak head extension angle (θ) and retraction (r_x) – were all biomechanically consistent with a more flexible neck connecting the head and body after the subjects had habituated. Similarly, lower amplitudes after habituation for the actively controlled kinematics – horizontal accelerations of the forehead (x_1) and mastoid process (x_2), angular acceleration of the head in flexion (α_2), and downward acceleration of the mastoid process (z_4) – were also consistent with reduced neck stiffness after habituation had occurred. Moreover, all of the kinematic peaks listed above occurred after the onset of neck muscle activity. This temporal sequence, combined with a similarity between the pattern of habituation in the muscle and the changes in the kinematic parameters (Figure 4.5), suggested that the altered kinematics were a direct result of the decrement in muscle response which occurred as subjects habituated to the perturbation.

Changes in the initial position and angle of both the head and torso over the eleven perturbations may have also contributed to the changes observed in the kinematics. The changes in initial head and neck posture, however, were small compared to the inter-subject variations in initial posture. In contrast, the changes in kinematics were large compared to their inter-subject variations. Based on their relatively small size, it was unlikely that the small changes in initial head, neck and torso posture that occurred over sequential trials were responsible for most of the observed changes in kinematics.

In two variables – the onset time of the upward acceleration measured at the forehead and the amplitude of the initial upward acceleration of the mastoid process (z_3) – the changes in initial posture

might have played some role. Both responses occurred before muscle activation and were therefore unrelated to habituation of the dynamic neck muscle response. The amplitude of the upward mastoid acceleration (z_3) and the initial horizontal position of the mastoid process both increased over the first three trials and then decreased over the remaining eight trials. This similarity suggested, though did not prove, that small changes in initial head position could affect the kinematic response. In the onset time of upward head acceleration, a small awareness-related difference not present initially emerged by the fifth trial. This awareness effect, in which onset of head acceleration occurred 4 ms later in alerted subjects than in unalerted subjects, was difficult to reconcile with the observed changes in initial posture since the postural changes were not related to subject awareness. Changes in baseline muscle tone could have produced this effect and a review of the pre-stimulus EMG activity in the neck muscles did show a small increase over the eleven perturbations. This small increment in baseline EMG levels, however, did not vary with subject awareness and therefore did not explain the awareness effect in the head acceleration onset data. Thus, based on the current analysis, the reason for the emergence of an awareness effect in the head acceleration onset data could not be determined.

Other awareness-related differences in the response data were also confined to interaction effects. In both horizontal forehead acceleration (x_1) and retraction (r_x), no significant differences between alerted and unalerted subjects were present in any one trial. In both variables, the significant awareness × trial interaction was isolated to the first three or four trials and indicated that awareness affected the rate at which these variables changed during habituation. In both cases, the response of alerted subjects changed more slowly than the response of unalerted subjects. While this effect was of marginal consequence for whiplash research, it did show that subtle protocol differences can alter the number of trials needed for subjects to habituate to a stimulus.

The minor role played by awareness in the current study was more consistent with the findings of Magnusson et al. (1999) than those of Kumar et al. (2000). Using an automobile seat and sled arrangement, Magnusson et al. (1999) reported no difference in the time to movement onset and muscle activation between alerted and unalerted conditions similar to those used here. Kumar et al. (2000), on the other hand, reported differences in both the time and amplitude of peak head acceleration between subjects in the alerted and unalerted conditions. The time of the peak kinematics reported by Kumar et al. (2000) were considerably delayed compared to those observed in the current study. In addition, the awareness effect reported by these authors appeared to be largest in the time of peak shoulder acceleration in male subjects, whereas this effect was largest in the time of peak head acceleration in female subjects. The low-back, molded-plastic seat used by Kumar et al. (2000) could account for this gender difference and could also explain why the peak response times observed in their study were so delayed with respect those observed here. Whatever the reason, these large

differences in peak kinematic response times suggested that this previous study and the current study may not be directly comparable. This was reinforced by the consistent onset times observed between the current study and that of Magnusson et al. (1999) – both of which used automobile seats.

4.4.1 Implications for Whiplash Injury

Some researchers have discounted a role for neck muscles in whiplash injury biomechanics (Foust et al., 1973; Snyder et al., 1975; Panjabi et al., 1998, Yoganandan, 1999). Their argument has been based largely on an observation that substantial levels of muscle force were generated too late to alter whiplash injury potential (Snyder et al., 1975). The current data clearly showed that large changes in peak kinematic response parameters occurred with habituation of the neck muscle response. This finding did not, however, directly answer the question of whether these kinematic changes occurred early enough to affect the potential for whiplash injury. Neck loads and intervertebral displacements have been shown to reach their peak magnitudes at about 150 to 200 ms after perturbations of about twice the velocity change used here (Kaneoka et al., 1999). These peak intervertebral kinematics, in particular, have been postulated to be responsible for facet joint injuries in some whiplash patients (Bogduk and Teasell, 2000). Peak SCM muscle activation in the current study occurred 104 ± 10 ms after perturbation onset, and all of the peak kinematic variables with habituation-related changes occurred within 200 ms of perturbation onset. If whiplash injury occurs in the postulated interval of 150 to 200 ms, then the results of the current study demonstrated that muscles are active early enough to affect the potential for whiplash injury.

Habituation of the neck muscle cocontraction may affect the study of whiplash injury in a number of ways. The level of cocontraction in the neck muscles affects the dynamic stiffness of the neck structure and therefore may also affect the magnitude and distribution of the intervertebral kinematics. Cocontraction may also set up other internal loads which are minimized or not present in subjects who have habituated to the perturbation. Winkelstein and Myers (2000) have shown that posterior neck muscles insert directly on the cervical facet joint capsular ligament and whiplash-like loads have been shown to produce injurious strains in this structure (Siegmund et al., 2000b). The larger posterior neck muscle contractions observed during the first exposure to the stimulus will produce a higher level of strain in the cervical facet joint capsular ligament than develops after multiple exposures to the perturbation. These two examples demonstrate how muscles might play a role in the aetiology of whiplash injury and highlight the importance of using experimental protocols which elicit the proper muscle response in order to study whiplash injury.

Based on a significant change in PARA muscle amplitude by the second trial, the results of the current study indicated that subjects could only be tested once before their response showed

evidence of habituation. This conclusion, however, was based on sequential exposures with a relatively short inter-stimulus interval – a median of 2.2 minutes between the first and second perturbation. Longer inter-stimulus intervals of 20 minutes have also produced habituation of the acoustic startle response within 2 to 6 exposures (Brown et al., 1991b). Moreover, repeated exposures to startling acoustic stimuli over sequential days has produce long-term habituation effects (Maschke et al., 2000), although partial recovery has been observed over the space of a week after multiple sequential acoustic stimuli presented on two consecutive days (Foss et al., 1989). In perturbation tests of human subjects, Brault et al. (2000) reported no test order effect in subjects tested only twice with an inter-test interval of at least one week, a finding that suggested that a week may be an appropriate amount of time between tests to minimize the confounding effect of habituation.

In the current study, habituation was studied using a single perturbation intensity and direction. This was arguably an ideal protocol in which to observe habituation and a similar degree of habituation might not have occurred had perturbation intensity or direction been varied. Previous research has shown that even large increases in the amplitude of a perturbation have not restored the response to the level observed during the first exposure (Timmann and Horak, 1997). This finding suggested that part of the habituation process was attributed to the novelty of the stimulus and not necessarily its magnitude. This idea was supported by a number of researchers who characterized the response to the first trial as a startle-like response which then rapidly diminished as subjects became acquainted with the stimulus (Hansen et al., 1988; Allum et al., 1992; Timmann and Horak, 1997). A recent study in which perturbation direction was randomized between forward and backward perturbations reported no habituation over five trials in each direction or over repeated blocks of trials given several weeks apart (Vibert et al., 2001). The perturbation used in their study, however, was of insufficient intensity to evoke activation of the superficial neck muscles in most of the subjects exposed to the fore-aft perturbations. It therefore appeared that the neck response observed by these researchers was governed primarily by the passive properties of the neck tissues. Since habituation is a neural phenomenon, the absence of habituation observed by Vibert et al. (2001) was not surprising given that there was little or no neuromuscular activity to habituate.

The current results showed that previous studies using human subjects to investigate whiplash injury biomechanics need to be re-evaluated. Studies in which repeated exposures were used or data in which the muscle response of the cervical paraspinal muscles was reportedly low or absent (Gutierrez, 1978; Ono et al., 1997) likely suffer from the effects of habituation. As a result, the kinematics reported in some of the previous whiplash investigations using human subjects may not accurately reflect the kinematics that would occur in these same subjects during their first exposure.

A potential limitation of the current study was the relatively low perturbation magnitude used

here compared to what has been used in most whiplash experiments. A previously-observed gradation of both the muscle response (Brault et al., 2000) and kinematics (Siegmund et al., 1997) with pulse intensity, however, suggested that the effects observed here would also be present at greater perturbation intensities. In the current study, data from six subjects exhibited adaptations that were sufficiently large that some of the kinematic peaks were delayed by 200 ms or more. These response changes were larger than those observed in the other subjects, and might be better characterized as strategy changes rather than habituation. Similar large delays in returning to the initial position have been observed in perturbations to standing balance and were also considered to represent possible changes in postural strategy (Horak et al., 1989). To avoid biasing the current analysis, data from these six subjects were excluded from the analysis.

In summary, the results of this experiment showed large changes in the neck muscle response and head kinematics in subjects exposed to sequential whiplash-like perturbations. These changes occurred rapidly – by the second trial in some variables – and indicated that habituation was a large potential confounder in the study of whiplash injury biomechanics using human subjects and repeated exposures. The results of the current study suggested that human subjects should not be exposed to more than one perturbation if a muscle and kinematic response unaffected by habituation is important.

4.5 Bridging Summary

The current study showed clear evidence of habituation to multiple sequential exposures to a whiplash-like perturbation. In this regard, the hypothesis that habituation affects the muscle and kinematic responses of human subjects to whiplash-like perturbations was accepted. Together with the results of Experiment 2A, which showed that surprised subjects (subjects who were not event aware) respond differently than event aware subjects, the results of the current experiment indicated that the specific study of what factors affect the aetiology of whiplash injury must be undertaken in conditions that mimic the level of awareness and singular nature of most real whiplash collisions. These findings, however, did not preclude the use of repeated exposures to study specific aspects of the biomechanical response. More specifically, the response of subjects after they had habituation was still sufficiently representative of the biomechanical response of surprised subjects to allow certain features of the response to be studied in isolation. The remaining two studies in the thesis were just such studies. The next two studies used subjects from the previous two experiments to explore the effect of amplitude awareness on the muscle and kinematic responses (Experiment 2C, Chapter 5) and the effect of different perturbation properties on the muscle and kinematic responses (Experiment 2D, Chapter 6). Because they were conducted on subjects who had habituated to the perturbing stimulus, the absolute magnitude of the response variables observed in the next two experiments were not

directly applicable to the biomechanics of whiplash injury, however the relative magnitude of the response variables between the different conditions being studied likely reflect the pattern that would exist in subjects not habituated to the stimulus.

CHAPTER 5 AWARENESS OF PERTURBATION MAGNITUDE

5.1 Introduction

A defining feature of reflex responses is their gradation to stimulus intensity (Gordon and Ghez, 1991). Studies of automatic postural responses in perturbed standing, however, have shown that this gradation is not absolute. Habituation alters the absolute level of reflex responses, although once stabilized, these reflex responses remain graded to stimulus intensity (Timmann and Horak, 1997). Horak et al. (1989) have also shown that the medium and long latency reflex responses of the lower limb muscles can be altered by previous exposure to multiple sequential stimuli of a single amplitude. These authors showed that prior experience with a stimulus, combined with an expectation that the next stimulus would be similar, produced an inappropriate muscle response when the amplitude of the perturbing stimulus was unexpectedly changed. This predictive phenomenon, known as central set, was smaller than the reduction due to habituation (Hansen et al., 1988), but nonetheless altered the absolute gradation of reflex responses with stimulus intensity.

Awareness of an imminent event or task, independent of external physical preparations, creates anticipation of that event or task. Frank (1986) divided this anticipation into two components: event awareness, which described whether subjects knew an event would occur, and temporal awareness, which described whether or not the exact timing of an event was known. Using different reaction-time protocols, Frank (1986) showed that when temporal awareness was present, the facilitation of spinal reflexes occurred earlier in advance of a voluntary muscle contraction than when it was not present. Studies in which acoustically startling stimuli have been substituted for the 'go' stimulus in forewarned, simple reaction-time tasks have also shown that reflex responses can be altered when precise timing information (temporal awareness) was absent (Valls-Solé et al., 1999; Siegmund et al., 2001b). Unlike reaction time protocols, the muscle response to a perturbation is not voluntarily executed, but evoked reflexively by the externally imposed stimulus. Under these circumstances, knowledge of the amplitude of a perturbation might also create anticipation and therefore a third component of awareness – amplitude awareness – might also affect the reflex response. For the purpose of this study, amplitude awareness described whether a subject was aware of the amplitude of an imminent perturbation.

The primary goal of this experiment was to determine whether subjects made use of advance information regarding the intensity of a perturbation to make anticipatory changes that altered their reflexive neck muscle response. To examine the effect of amplitude awareness, seated subjects were exposed to a series of identical perturbations periodically interrupted by perturbations with a lower or

higher acceleration. In one block of perturbations, subjects were alerted to the amplitude of the next perturbation, and in the other block, they were not. It was hypothesized that in the alerted condition, subjects would anticipate the change in perturbation intensity and that this anticipation would produce a muscle and kinematic response that was different from that produced if the same perturbation was presented without advance warning.

A secondary goal of this study was to examine the effect of different accelerations on the muscle and kinematic responses. To date, many postural perturbation studies have varied only perturbation velocity and displacement and used back-to-back acceleration pulse – one in the positive direction and the other in the negative direction – to create their fixed displacement perturbations (Diener et al., 1988, Horak et al., 1989; Forssberg and Hirschfeld, 1994; Timmann and Horak, 1997). In this study, different forward accelerations up to a similar constant velocity were used to study the effect of acceleration in isolation. The results of this study will help determine whether advance knowledge of perturbation amplitude and the acceleration used to generate a perturbation are variables that needs to be controlled in the study of postural perturbations.

5.2 Methods

5.2.1 Subjects

Thirty-six subjects with no history of sensory or motor dysfunction participated in the experiment. Physical characteristics for the subjects are given in Table 5.1. All subjects gave their informed consent and were paid a nominal amount for their participation. The use of human subjects for this experiment was approved by the university's Ethics Review Board and the study conformed with the Declaration of Helsinki.

Table 5.1 Mean (S.D.) of subject age and physical characteristics.

	Female	Male
n	20	16
Age, yrs	26 (6)	28 (7)
Height, cm	166 (7)	177 (8)
Mass, kg	61 (11)	76 (17)

5.2.2 Instrumentation

Electromyographic (EMG) activity in the orbicularis oculi (OO), masseter (MAS), sternocleidomastoid (SCM) and cervical paraspinal (PARA) muscles was recorded bilaterally using

10 mm pre-gelled surface electrodes (H69P, Kendall-LTP, Huntington Beach, CA) and an Octopus AMT-8 amplifier (Bortec, Calgary, AB). Two uniaxial accelerometers (Kistler 8302B20S1; ±20g, Amherst, NY) and a single uniaxial angular rate sensor (ATA Sensors ARS-04E; ±100 rad/s, Albuquerque, NM) were strapped tightly to the midline of a subject's forehead, immediately above the glabella. The sensitive axes of the accelerometers were mutually orthogonal and oriented to measure horizontal (x') and vertical (z') acceleration in the mid-sagittal plane (Figure 5.1). The rotational axis of the angular rate sensor was oriented mediolaterally to measure flexion and extension motion in the sagittal plane. Sled acceleration was measured using an uniaxial accelerometer (Sensotec JTF3629-05; ±10g, Columbus, OH) oriented horizontally along the axis of motion. Displacement was measured using an Optotrak motion analysis system (Northern Digital, Waterloo, ON) and markers placed over the left mastoid process and midline at the subject's forehead, vertex of the head, manubrium and C7 spinous process (Figure 5.1). A sixth marker was placed on the left seat hinge. A force transducer (Artech S-Beam SS20210, ±2 kN, Riverside, CA) was used to measure reaction loads during normalizing contractions of the SCM and PARA muscles. EMG signals were bandpass filtered at 10 Hz to 1 kHz and transducer signals were low-pass filtered at 1 kHz before being simultaneously sampled at 2 kHz and stored for subsequent analysis. Optotrak data were acquired at 200 Hz.

5.2.3 Test Procedures

Subjects were seated in an automobile seat obtained from the front passenger location of a 1991 Honda Accord. The head restraint was removed from the top of the seat back to eliminate the potential for an externally applied load to the head during the perturbation. The seat was mounted on a custom fabricated sled powered by a feedback-controlled linear induction motor (Kollmorgen IC55-100A7, Kommack, NY). The sled generated no pre-perturbation signals, either audible or mechanical, which subjects could use to predict onset of the perturbation. Three perturbations with differing levels of acceleration were used to produce a velocity change of 0.5 m/s (Figure 5.2). The average accelerations of the low (L_a), medium (M_a) and high (H_a) intensity perturbations were 0.48g, 0.90g and 1.26g. The medium perturbation served as the standard stimulus and the low and high perturbations served as the test stimuli.

Each subject underwent 2 blocks of 36 perturbations. Each block consisted of 30 standard perturbations (M_a), 3 low-acceleration perturbations (L_a) and 3 high-acceleration perturbations (H_a). The six non-standard perturbations were spaced pseudo-randomly amongst the standard perturbations such that there were at least four standard perturbations between all non-standard perturbations. In one block of perturbations, subjects were warned of the intensity of the perturbations using one, two

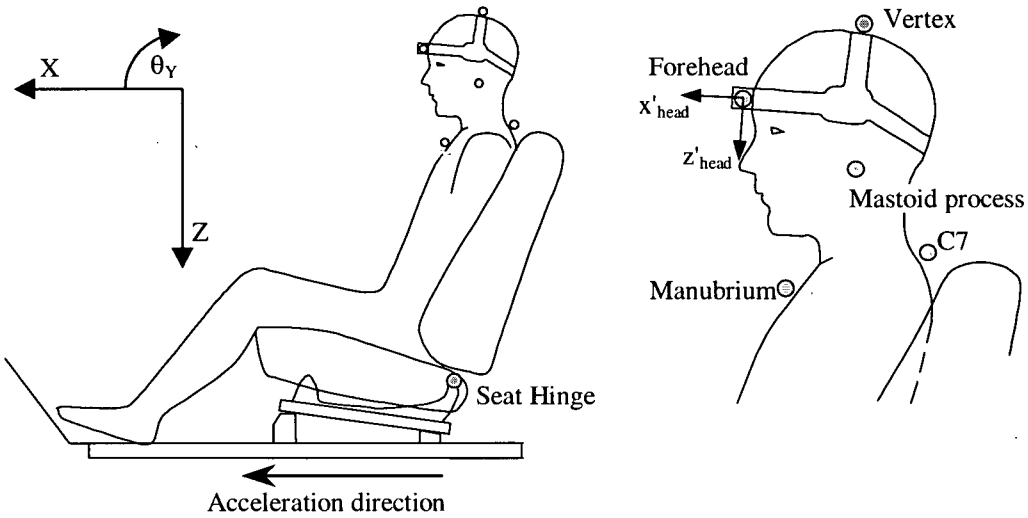


Figure 5.1 Schematic of the test configuration showing the locations of the Optotrak markers (shaded circles), the lab reference frame (X, Z) and the head reference frame (x', z'). The initial orientation of the head reference frame was determined by how the head band fit the subject and varied between 12 ± 4 deg relative to the lab reference frame.

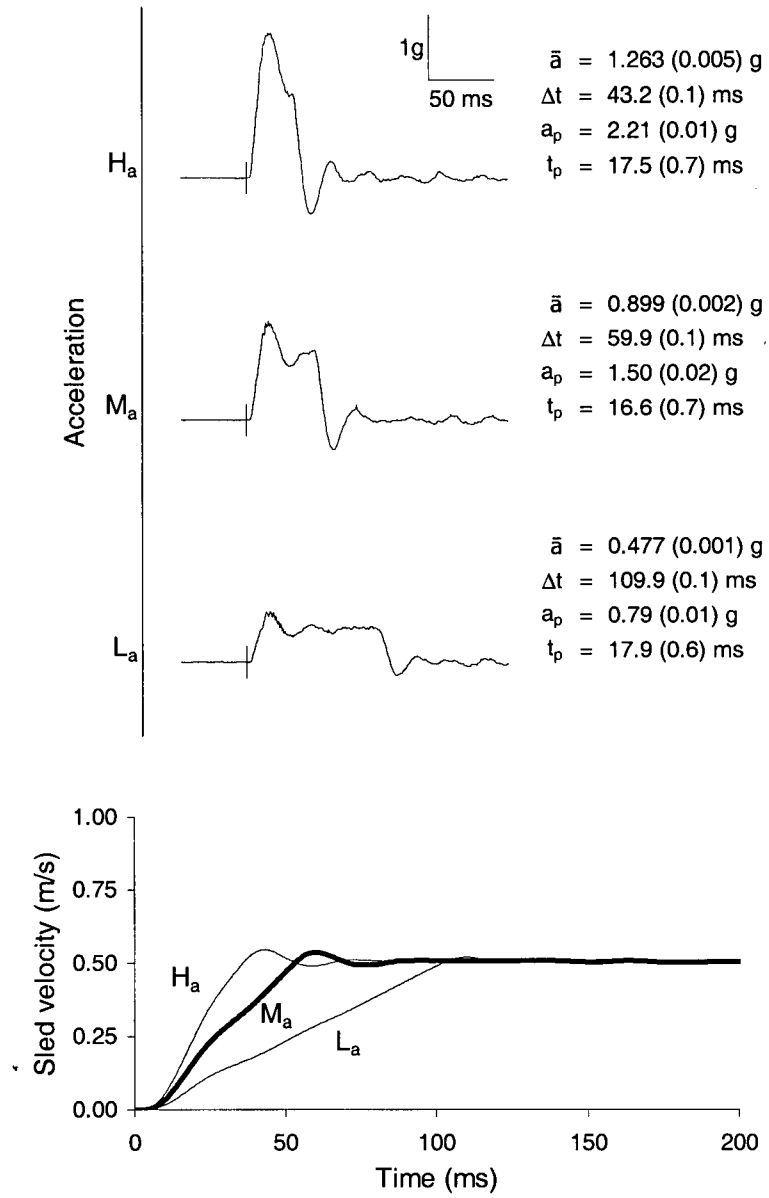


Figure 5.2 Acceleration vs. time graphs (top) for each sled acceleration (low L_a, standard M_a, and high H_a) and descriptive statistics for the average acceleration ($\bar{a}$), pulse duration (Δt), peak acceleration (a_p) and time of peak acceleration (t_p). Superimposed velocity vs. time plots (bottom) of the three perturbation pulses. The dark line depicts the standard perturbation.

or three auditory tones (1000 Hz, 80 dB, 40 ms duration) to identify the low, standard or high acceleration perturbations respectively. Warning tones were presented about 5 seconds after the completion of the preceding trial. In the other block, subjects received no warning tones. Half the subject received the warned block first; the other half received the unwarned block first. The time between perturbations varied randomly (uniform distribution) between 20 and 30 seconds and the rest period between blocks was about 5 minutes. For all trials, subjects were instructed to sit normally, face forward, rest their forearms on their lap and to relax their face and neck muscles. To achieve a stabilized response prior to testing, each subject first underwent eleven standard perturbations.

Prior to the above protocol, seated subjects performed sub-maximal isometric contractions in flexion and extension to generate normalizing data for the SCM and PARA muscles respectively. A strap attached to the load cell was placed around a subject's head and its length adjusted to ensure the subject's head was neutrally positioned. The strap was located immediately above the glabella for flexion contractions and at the height of the external occipital protuberance for extension contractions. Subjects were instructed to generate a force of 50 N with visual feedback, first in flexion and then in extension. EMG and load cell data were acquired for 5 s during each contraction.

5.2.4 Data Reduction

A subject's initial position was determined from the Optotrak data immediately preceding the perturbation. Initial head position was defined in the laboratory reference frame using the horizontal (X-axis; positive forward) and vertical (Z-axis; positive downward) positions of the mastoid process relative to the seat hinge (Figure 5.1). Initial head angle in the sagittal plane was determined from the forehead and vertex markers and reported relative to the positive X-axis ($+\theta_y$ rotation corresponded to extension). Initial torso position was defined using the horizontal and vertical positions of the midpoint between the manubrium and C7 spinous process markers relative to the seat hinge, and torso angle relative to the horizontal plane was determined using the same two markers. The RMS accuracy of the position measurements from the Optotrak system was less than 0.1 mm, and based on marker separation, the RMS accuracy of the calculated angles was less than 0.1 degrees.

The onset of head movement, and the amplitude and time of peak head accelerations were determined directly from the accelerometer data. The algorithm used to determine onset is developed in Appendix A. Forehead acceleration data were not resolved into the lab reference frame. Peak retraction, defined as the maximum horizontal translation of the top of the cervical spine rearward relative to the bottom of the cervical spine, was estimated using the maximum relative horizontal displacement in the lab reference frame between the Optotrak markers on the mastoid and manubrium. Peak angular velocity of the head was determined from the angular rate sensor data after

it had been digitally compensated to reduce the sensor's high-pass frequency to 0.002 Hz (Laughlin, 1998). Angular acceleration was then computed by finite differences (5 ms window) from the compensated angular velocity data. Total head angular displacement was determined from the Optotrak markers located at the forehead and vertex. Horizontal and vertical accelerations at the mastoid process were computed by double differentiating the Optotrak data. This technique produced results within 0.1g of the values computed using a rigid body transformation of the linear and angular accelerations measured at the forehead and was preferred because it relied on data from only one Optotrak marker rather than data from two accelerometers, an angular rate sensor and two Optotrak markers needed for the rigid body calculation. Mastoid accelerations were computed in the lab reference frame.

Pre-stimulus noise in each channel of the EMG data was quantified using the root mean squared (RMS) amplitude of the EMG signal over the 100 ms preceding the perturbation. EMG onset was defined as the time at which the RMS amplitude, computed from the raw EMG data using a moving 20 ms window, reached 10 percent of it maximum value and was then confirmed visually. For each muscle, the RMS amplitude of the EMG signal was calculated for the interval between EMG onset and peak head extension angle. The corresponding pre-stimulus noise for each muscle was then subtracted from this quantity. The SCM and PARA muscle EMG amplitudes were normalized by the RMS amplitude obtained during the 5 s sub-maximal contraction for the corresponding muscle.

5.2.5 Statistical Analysis

Data were parsed into sequences of five perturbations so that each sequence contained two standard perturbations before and two standard perturbations after a low or high acceleration perturbation. Within-subject means of each dependent variable were computed for each trial within these sequences separately for the alerted and unalerted blocks. These means were then normalized by the mean response for all 30 standard perturbations in the unalerted block. This normalization process reduced between-subject variance due to anthropometry and gender, but maintained within subject variance due to the different amplitude awareness levels and perturbation intensities. For each kinematic dependent variable, a two-way repeated-measures analysis of variance (ANOVA) was used to assess differences related to level of awareness (alerted, unalerted) and perturbation intensity (L_a, M_a, H_a). After first confirming that there were no left/right side differences in the muscle response data, the mean responses of the left and right EMG variables were calculated and then assessed using the same two-way repeated-measures ANOVA. Post-hoc testing for differences between the three perturbations was performed using a Scheffé test. All statistical analyses were performed using Statistica (v.5.1, Statsoft Inc., Tulsa, OK) and a significance level of α=0.05.

5.3 Results

The mean initial position and angle of the head and upper torso prior to each perturbation did not vary significantly with either warning level or perturbation intensity (Table 5.2). The perturbations themselves produced stereotypical responses in all subjects (Figure 5.3). The torso was accelerated forward first, followed by the head, which was accelerated linearly upward and forward, and angularly into extension. Most subjects responded to the perturbation by rapidly restoring their upright head position. Some subjects, however, altered their response strategy and restored their upright head position more slowly, particularly after the low intensity perturbations. In most of these trials, a clear peak was still visible in both the retraction (r_x) and head angle (θ) data (see left panel in Figure 5.3). In five subjects, peak head angle and peak retraction during the low perturbations did not occur within 500 ms of perturbation onset and therefore all data from these five subjects were removed from the statistical analysis. Clear SCM activity was present in all trials, however, PARA activity was often difficult to discern from the background noise. As a result, within-subject means for PARA activity were occasionally calculated using data from fewer than three trials.

Advance warning of perturbation intensity had no effect on the mean amplitudes or mean times of the EMG or kinematic variables (Figure 5.4). There were also no significant differences between the four standard perturbations considered in the analyses of variance. Significant differences, however, were observed in all dependent variables as a function of perturbation intensity. Descriptive statistics of the raw data and the results of inferential analyses on the normalized data are given in Tables 5.3 through 5.5. Post-hoc analyses of the perturbation intensity effect revealed that the amplitudes of the EMG and kinematic responses for all dependent variables were lower in the low intensity perturbation than in the standard perturbation ($p<0.048$). In addition, all peak kinematic responses occurred later in the low acceleration perturbations than in the standard perturbations ($p<0.017$). Differences in the onset latencies of the OO, MAS and PARA muscles were not significantly different between the low and standard pulses, however, SCM activation was delayed by 3 ms in the low perturbation compared to the standard perturbation ($p<0.0027$).

Despite significant main effects for pulse intensity when the low, standard and high acceleration pulses were analyzed together, post-hoc testing revealed that the high acceleration perturbation did not always produce significantly larger peak kinematic responses than the standard perturbation. The mean amplitudes of both peak retraction (r_x) and peak head angle (θ) were not significantly larger during the high acceleration trials than during the standard acceleration trials ($p>0.44$) (Figure 5.4). Similarly, some of the peak kinematic responses did not occur earlier in the high acceleration perturbation than in the standard perturbation. The mean time of the peak upward forehead acceleration (z_1) and peak downward mastoid acceleration (z_4) during high acceleration

Table 5.2 Mean (S.D.) of the initial position and angle of the head and torso. Upper portion of table summarizes data as a function of awareness (alerted, unalerted) and perturbation intensity (low, standard, high). Positions are relative to an origin at the seat hinge; angles are relative to the horizontal. There were no significant differences with either warning level or perturbation intensity. X, horizontal position, +ve forward; Z, vertical position, +ve downward; N, number of subjects in analysis.

		Mastoid process		Head	Manubrium-C7 midpoint		Torso
Awareness	Perturbation Intensity	X (mm)	Z (mm)	angle (deg)	X (mm)	Z (mm)	angle (deg)
Alerted	Low	12 (19)	-648 (36)	9.3 (6.3)	26 (16)	-565 (29)	-16.5 (4.8)
	Standard	12 (19)	-648 (36)	9.5 (6.3)	25 (16)	-565 (29)	-16.4 (4.8)
	High	11 (20)	-648 (36)	9.5 (6.6)	25 (17)	-565 (29)	-16.3 (4.9)
Unalerted	Low	9 (17)	-648 (36)	10.1 (6.6)	23 (14)	-565 (29)	-16.0 (4.6)
	Standard	9 (17)	-648 (36)	9.7 (6.4)	23 (14)	-565 (29)	-16.0 (4.6)
	High	9 (17)	-648 (36)	9.7 (6.6)	23 (14)	-565 (29)	-16.0 (4.6)
N		30	30	31	25	25	25

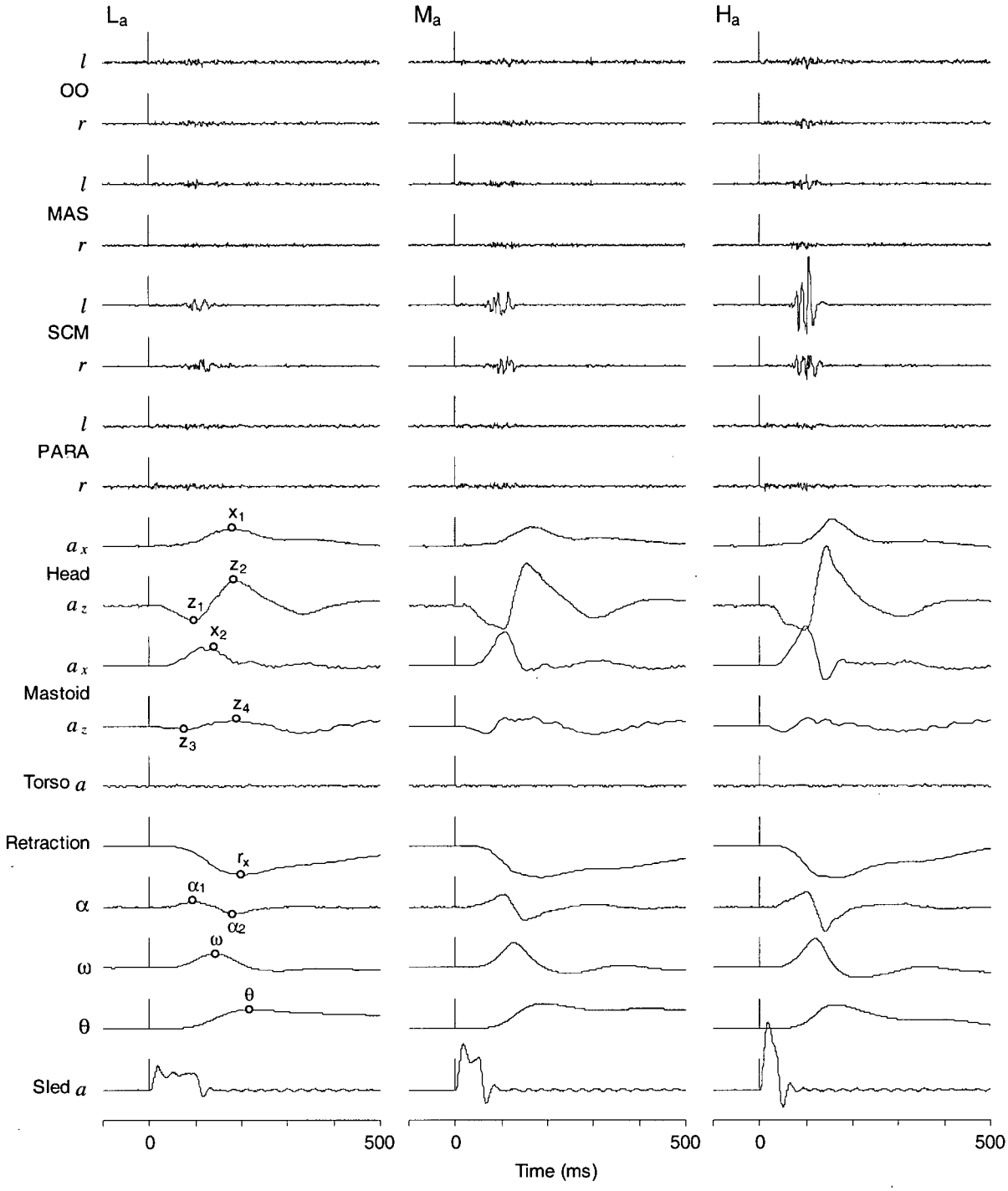


Figure 5.3 Sample data from a low acceleration (L_a), standard acceleration (M_a) and high acceleration (H_a) perturbation for a single subject. Labeled hollow circles in the left panel represent kinematic peaks used for subsequent analyses. The vertical scale bars are aligned with perturbation onset and are equal to 1g, 50 mm, 200 rad/s², 5 rad/s and 20 deg. OO, orbicularis oculi; MAS, masseter; SCM, sternocleidomastoid; PARA, cervical paraspinal, l, left; r, right; *a*, linear acceleration, subscript *x* and *z* refers to the x- and z-directions; α, head angular acceleration; ω, head angular velocity; θ, head angle.

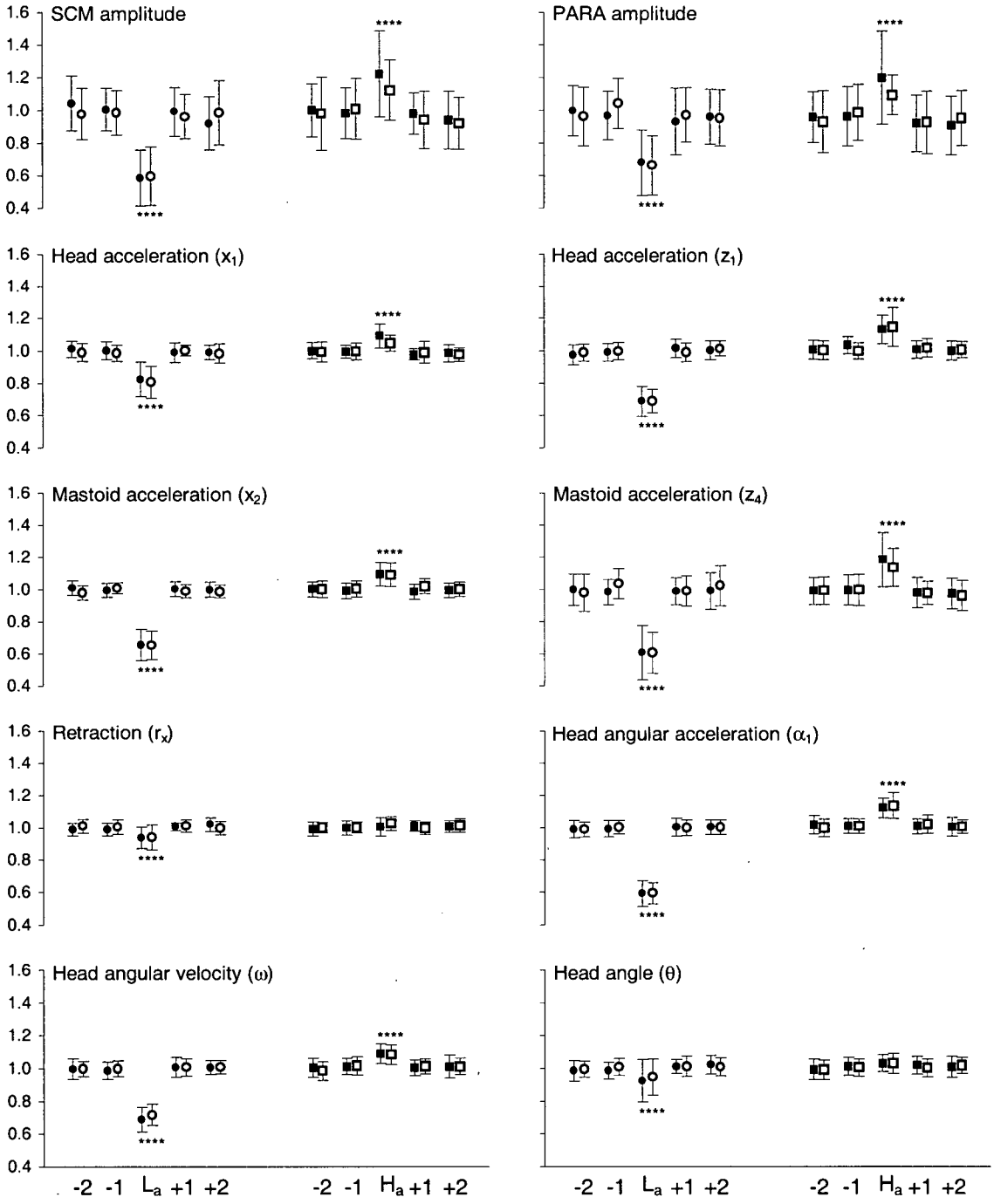


Figure 5.4 Mean and standard deviation of selected normalized electromyographic and kinematic amplitude data for two standard perturbations (M_a) either side the two test stimuli – a low acceleration (L_a) shown on the left half of each graph (● alerted, ○ unalerted), and a high acceleration (H_a) shown on the right half of each graph (■ alerted, □ unalerted). –2 and –1 are the penultimate and ultimate trials preceding the test stimulus, and +1 and +2 are the first and second trials immediately following the test stimulus. L and H values which are significantly different from the standard perturbation are indicated by asterisks: **** $p<0.0001$.

Table 5.3 Mean (S.D.) of the peak amplitude of selected kinematic data. Upper portion of table summarizes data as a function of amplitude awareness (alerted, unalerted) and perturbation pulse intensity (low, standard, high). Lower portion of table summarizes the results (F-statistics) of the ANOVAs for the effect of awareness and perturbation intensity on each of the peak responses. Individual peaks are labeled with hollow circles in Figure 5.3. Descriptive statistics are from the actual data and the results of the inferential statistics are from the normalized data. N, number of subjects in analysis.

		Linear kinematics							Angular kinematics			
		Forehead			Mastoid process			Retraction	Head			
Awareness	Pulse	x_1 (g)	z_1 (g)	z_2 (g)	x_2 (g)	z_3 (g)	z_4 (g)	r_x (mm)	α_1 (rad/s²)	α_2 (rad/s²)	ω (rad/s)	θ (deg)
Alerted	Low	0.69 (0.11)	-0.54 (0.12)	0.89 (0.20)	0.72 (0.12)	-0.09 (0.03)	0.20 (0.08)	-24.3 (5.8)	51 (11)	-60 (20)	2.7 (0.6)	14 (3)
	Standard	0.86 (0.19)	-0.79 (0.18)	1.58 (0.57)	1.11 (0.22)	-0.18 (0.06)	0.34 (0.14)	-25.8 (5.7)	86 (17)	-126 (64)	4.0 (0.8)	16 (3)
	High	0.94 (0.23)	-0.89 (0.22)	1.83 (0.67)	1.22 (0.22)	-0.24 (0.09)	0.40 (0.16)	-25.9 (5.7)	96 (19)	-152 (75)	4.3 (0.9)	16 (4)
Unalerted	Low	0.67 (0.12)	-0.54 (0.13)	0.92 (0.20)	0.71 (0.11)	-0.10 (0.04)	0.19 (0.08)	-24.5 (5.1)	51 (11)	-64 (28)	2.9 (0.5)	15 (3)
	Standard	0.84 (0.17)	-0.79 (0.19)	1.57 (0.55)	1.10 (0.21)	-0.18 (0.06)	0.32 (0.13)	-26.1 (4.9)	86 (17)	-127 (67)	4.0 (0.8)	16 (3)
	High	0.88 (0.18)	-0.90 (0.19)	1.77 (0.60)	1.20 (0.24)	-0.24 (0.09)	0.37 (0.15)	-26.6 (4.6)	97 (17)	-147 (66)	4.3 (0.8)	16 (3)
N		31	31	31	30	26	29	30	31	31	31	31
ANOVA F-statistics												
Awareness												
Pulse		61.2****	150****	136****	205****	84.8****	75.6****	12.4****	329****	123****	165****	7.5****
Awareness x Pulse												

**** $p<0.0001$

Table 5.4 Mean (S.D.) of the time of peak amplitude for selected kinematic data. Upper portion of table summarizes data as a function of amplitude awareness (alerted, unalerted) and perturbation pulse intensity (low, standard, high). Lower portion of table summarizes the results (F-statistics) of the ANOVAs for the effect of awareness and perturbation intensity on each of the peak responses. Individual peaks are labeled with hollow circles in Figure 5.3. Descriptive statistics are from the actual data and the results of the inferential statistics are from the normalized data. N, number of subjects in analysis.

		Linear kinematics							Angular kinematics			
		Forehead			Mastoid process			Retraction	Head			
Awareness	Pulse	x_1 (ms)	z_1 (ms)	z_2 (ms)	x_2 (ms)	z_3 (ms)	z_4 (ms)	r_x (ms)	α_1 (ms)	α_2 (ms)	ω (ms)	θ (ms)
Alerted	Low	199 (22)	107 (10)	196 (23)	132 (16)	77 (13)	148 (15)	218 (24)	111 (12)	194 (21)	151 (13)	238 (20)
	Standard	179 (19)	101 (10)	164 (13)	110 (7)	62 (7)	135 (14)	185 (24)	107 (9)	163 (13)	134 (11)	199 (22)
	High	171 (18)	95 (12)	157 (13)	103 (9)	53 (7)	127 (15)	172 (26)	101 (10)	157 (13)	128 (11)	187 (23)
Unalerted	Low	200 (19)	107 (9)	195 (21)	130 (15)	80 (15)	148 (16)	221 (18)	113 (11)	192 (22)	153 (14)	236 (18)
	Standard	178 (17)	100 (11)	164 (13)	108 (7)	62 (7)	134 (15)	186 (22)	106 (8)	163 (12)	134 (9)	198 (19)
	High	171 (15)	96 (11)	156 (12)	100 (8)	52 (5)	125 (18)	179 (21)	100 (7)	156 (11)	127 (9)	188 (19)
N		31	31	31	30	26	29	30	31	31	31	31
ANOVA F-statistics												
Awareness												
Pulse		100****	12.2****	93.0****	112****	47.0****	18.5****	78.7****	32.9****	103****	76.8****	138****
Awareness x Pulse												

**** $p<0.0001$

Table 5.5 Mean (S.D.) of EMG onset time and normalized RMS magnitude. Upper portion of table summarizes data as a function of awareness (alerted, unalerted) and perturbation pulse intensity (low, standard, high). Lower portion of table summarizes the F-statistics and the results of the ANOVA for awareness and perturbation intensity. Descriptive statistics are from the actual data and the results of the inferential statistics are from the normalized data. RMS, root-mean square; OO, orbicularis oculi; MAS, masseter; SCM, sternocleidomastoid; PARA, cervical paraspinal; N, number of subjects in analysis.

		Onset (ms)				RMS Magnitude	
Awareness	Pulse	OO	MAS	SCM	PARA	SCM	PARA
Alerted	Low	74 (13)	78 (8)	76 (8)	76 (5)	0.07 (0.04)	0.04 (0.05)
	Standard	73 (8)	76 (7)	73 (6)	76 (3)	0.13 (0.09)	0.06 (0.09)
	High	66 (12)	72 (9)	70 (6)	72 (6)	0.15 (0.11)	0.07 (0.10)
Unalerted	Low	78 (18)	78 (12)	77 (8)	76 (4)	0.07 (0.03)	0.03 (0.03)
	Standard	74 (9)	77 (8)	73 (6)	76 (3)	0.12 (0.06)	0.06 (0.07)
	High	67 (13)	72 (10)	69 (6)	71 (5)	0.13 (0.08)	0.06 (0.07)
N		30	29	31	31	31	31
ANOVA F-statistics							
Awareness							
Pulse		4.65****	7.66****	18.8****	8.83****	33.8****	26.0****
Awareness x Pulse							

****p<0.0001

perturbations did not occur earlier than during the adjacent standard perturbations. All of the remaining kinematic peaks occurred significantly earlier during high acceleration pulses than during standard pulses ($p<0.023$). EMG amplitude in the neck muscles was significantly larger and onset latencies in the neck muscles were significantly shorter in the high acceleration perturbation than in the standard perturbation ($p<0.0001$). MAS activation occurred earlier in high acceleration trials than in standard trials ($p<0.006$), however, OO activation did not.

Overall, the decrease in response amplitude observed in the low perturbations was greater than the increase in response amplitude observed in the high perturbations (Figure 5.4). Relative to the standard perturbations, the average decrease in amplitude for all of the dependent variables in the low acceleration perturbations was 31 ± 13 percent (range 6 to 45). This decrease was significantly larger than the average increase in amplitude observed for all dependent variables in the high acceleration perturbations (13 ± 8 percent; range 2 to 32) (paired t-test: $p<0.0001$). The large range in these percentages was attributable to peak retraction (r_x) and peak head angle (θ), both of which were not significantly larger in high perturbations than in standard perturbations. When values for both peak retraction (r_x) and peak head angle (θ) were removed from this comparison, the average decrease in response amplitude during low perturbations was 36 ± 8 percent (range 18 to 45) and the average increase in response amplitude during high perturbations was 14 ± 7 percent (range 6 to 32). Similar values were observed when the muscle responses were examined in isolation. SCM amplitudes decreased 41 percent during low perturbations and increased 17 percent during high perturbations, whereas PARA amplitudes decreased 33 percent during low perturbations and increased 14 percent during high perturbations.

5.4 Discussion

The goal of this experiment was to determine whether subjects used information regarding the intensity of a perturbation, i.e., amplitude awareness, to make anticipatory changes that altered their reflexive neck muscle response. Anticipation has previously been shown to facilitate spinal reflexes in advance of voluntary, reaction time movements (Frank, 1986) and readiness to perform a ballistic, reaction time task has previously been shown to alter both the magnitude and pattern of the reflex neck muscle response evoked during a startle reflex (Siegmund et al., 2001b). The results of this study clearly showed, however, that advance warning of the acceleration magnitude of a horizontal perturbation did not affect the muscle or kinematic responses of seated human subjects and disproved the primary hypothesis of this study. Although the three perturbation levels produced statistically significant changes in the amplitude and timing of all muscle and kinematic variables, it was possible that the range of perturbations used in this study was too narrow to discern a warning

effect. Had one of the perturbations been perceived as considerably more noxious than the other two, a sensitization might have occurred and a warning effect may have materialized. Therefore the results of the current study cannot be used to exclude a role for amplitude awareness in altering reflex responses to all perturbation intensity levels. Instead, the results can only be used to conclude that amplitude awareness had no effect on reflex responses evoked by the type and range of perturbations used in this study.

The current data showed a clear gradation of both muscle and kinematic responses to differences in the acceleration used to reach a constant perturbation velocity. This gradation was consistent with reflex activation of the neck musculature (Gordon and Ghez, 1991) and indicated that the mechanoreceptors modulating the reflex response were sensitive to perturbation acceleration. Previous perturbation studies in standing balance have focused on variations in the displacement and velocity of the perturbation and showed that early components (first 75 ms after activation) of the automatic postural response in leg muscles were graded to stimulus velocity and later components (150 to 500 ms after activation) were graded to stimulus displacement (Diener et al., 1988). The current study showed that early components of reflex neck muscle responses were graded to the acceleration of the perturbation. Whether the mechanoreceptors responsible for transducing the stimulus responded to acceleration directly, like the utricle and saccule of the vestibular system, or responded to velocity and then rely on temporal summation to produce an acceleration sensitivity, like muscle spindles, cannot be discerned from this experiment. Both the vestibular system and somatic mechanoreceptors in the trunk have been proposed as sources for triggering reflex muscle responses during postural perturbations (Allum et al., 1997).

Although a response gradation with acceleration was observed, this gradation did not appear to be linear. In comparison to the amplitude of the muscle and kinematic responses observed in the standard perturbations, the decrement in response amplitude observed in the low acceleration perturbations was 2.4 to 2.6 times larger than the increment in response amplitude observed in the high acceleration perturbations. For neck muscle amplitudes in isolation, this factor was 2.4 for both the SCM and PARA muscles. Even if linearly adjusted for the slight difference in the average acceleration of the perturbations (L_a was 47 percent less than M_a, whereas H_a was only 40 percent greater than M_a), this factor was still greater than 2.0. Although there was no reason to expect a linear variation in kinematic responses with changes in perturbation acceleration for a mechanical system as complex as the head and neck, these findings indicated that this non-linearity extended to the sensorimotor system mediating the neck muscle response.

Relative to the standard perturbation, activation of the SCM muscles was delayed by 3 ms during low acceleration perturbations and activation of both neck muscles was advanced by 4 ms

during high acceleration perturbations. Changes in muscle onset latencies have not been observed in postural perturbation studies in which only the velocity and displacement of the perturbation have been varied (Diener et al., 1984; Diener et al, 1988). Similar changes in reflex onset latency with stimulus intensity have been observed, however, in eye blinks to acoustically startling stimuli (Blumenthal, 1996). The observed changes in reflex onset latency with perturbation acceleration corroborated the observed amplitude gradation and suggested that perturbation acceleration was an important regulator of the intensity and timing of reflex responses in the neck muscles.

Peak retraction (r_x) and peak head extension angle (θ) did not vary significantly between the standard and high acceleration perturbations, but did vary significantly between the low and standard perturbations. Of all the dependent variables evaluated in this study, these two variables were the most temporally remote from the onset of the perturbation. Peak retraction (r_x) occurred about 185ms after perturbation onset and peak head extension angle (θ) occurred about 200 ms after perturbation onset. The standard and high perturbations, however, had a duration of 43 ms and 60 ms, respectively. Therefore, both peak kinematic responses occurred well after acceleration was complete, and therefore might be expected to exhibit a weaker relationship to perturbation acceleration than more proximate variables. Moreover, inspection of the temporal data in Table 5.4 showed that variance increased with temporal remoteness from the onset of the perturbations. As a result, differences in timing which were statistically significant for dependent variables that were temporally close to the perturbation were rendered insignificant by the increased variance when temporally more remote from the perturbation.

Low EMG amplitude in the PARA muscles resulted in PARA onset and amplitude data being discarded in some trials. Such reduced or absent antagonist activity in response to postural perturbations have been previously observed and attributed to habituation and practice (Woollacott et al., 1988; Horak et al., 1989; Allum et al., 1992). Despite difficulty quantifying some PARA responses, the analysis of PARA amplitude and onset latency produced similar results to those obtained for the considerably larger and more pronounced SCM responses. This similarity between the SCM and PARA results suggested that the analysis of the PARA muscles, though based on fewer trials, was valid. A change in response strategy, similar to that which eliminated five subjects from the current analysis, has also been observed in habituated subjects during standing balance (Horak et al., 1989). In this previous study, subjects did not return immediately to their initial upright posture, but temporarily stabilized at a new equilibrium and then slowly returned to their initial position. The similarly-altered response observed in some subjects in the current study suggested that these subjects were willing to compromise their head posture in order to optimize some other performance criterion. This willingness suggested that the magnitudes of the stimuli used here were not perceived by these

subjects to be threatening – a possible reason cited earlier for the absence of a warning effect.

The muscle and kinematic responses to the standard perturbation presented immediately after a non-standard perturbation were not affected by the insertion of a non-standard stimulus in either the warned or unwarned conditions. This robust response to the standard stimulus suggested that subjects were grading their response entirely to the initial properties of the perturbation stimulus. Although the protocol used in this experiment was not designed to test whether central set might play a role in the response to the standard perturbation, the absence of a discernible amplitude awareness effect on the response to either the non-standard perturbation, or the standard perturbation immediately following a non-standard perturbation, suggested that central set was not present.

In summary, the results of the current study indicated that the reflexive response of the neck muscles was not altered when subjects had advance knowledge of the acceleration of a horizontal forward perturbation. Despite no warning effect, clear evidence of gradation in both the latency and amplitude of the reflex response was observed with perturbation acceleration. Therefore, acceleration appeared to be an important regulator of the reflex response and should be controlled in studies of postural perturbations.

5.5 Bridging Summary

The results of the current study indicated that advance knowledge of perturbation intensity did not affect the muscle or kinematic responses. This negative finding could not, however, be extrapolated to many experiments of whiplash injury biomechanics. Whiplash experiments are often conducted at stimulus intensities larger than used in the current study. Some of the high intensity perturbations used in these other experiments might be perceived as noxious by some subjects. The perturbations in the current experiment did not appear to be perceived as noxious by the subjects, and therefore the current findings may not be applicable to other high intensity perturbations. As a result, it remains unclear whether awareness of the amplitude of a perturbation needs to be controlled during experiments involving repeated exposure to higher-intensity whiplash-like perturbations.

Considering the results from Experiments 2A (Chapter 3), the issue of amplitude awareness may be of only secondary importance in whiplash experimentation. If a true whiplash response can only be evoked in the laboratory by deception, then the effect of advance knowledge of stimulus intensity is unimportant. If future experiments show that amplitude awareness does alter the response to noxious stimuli, then it may be possible to combine amplitude awareness with deception to reverse the habituation process observed in Experiment 2B (Chapter 4).

An interesting finding in the current experiment was the reflex gradation with perturbation acceleration. In whiplash biomechanics, the intensity of an impact has commonly been quantified

using only the velocity change of the occupant's vehicle. Recent epidemiological evidence, however, has shown an increased risk of long-term symptoms in collisions with high-force and short-duration impulses (Krafft et al., 2000). Based on these findings, a parametric study of collision pulse properties was conducted to evaluate the relative contribution of acceleration and velocity change to the response of a BioRID II dummy specifically designed for whiplash research (Siegmund and Heinrichs, 2001). This study revealed a significant positive correlation between the amplitude of the dummy kinematics and the average perturbation acceleration for a series of impulses that produced the same velocity change. The results of the current experiment were consistent with these findings and indicated that the reflex muscle response also correlated positively with perturbation acceleration. Given these results, the final experiment of this thesis was designed to investigate the relative contributions of acceleration and velocity change to the reflex muscle response and induced kinematics in human subjects.

CHAPTER 6 GRADATION OF RESPONSE TO PERTURBATION PROPERTIES

6.1 Introduction

Reflex responses are graded to stimulus intensity (Gordon and Ghez, 1991). In simple reflexes, such as the monosynaptic stretch reflex, a single mechanoreceptor is known to play a dominant role in transducing the stimulus, and the specific property of the stimulus that evokes the response is relatively well defined. In more complex reflexes, such as automatic postural responses, multiple muscles and joints are involved and multiple mechanoreceptors may transduce the stimulus and trigger the reflex response (Keshner et al., 1988; Allum et al., 1997). The potential involvement of multiple mechanoreceptors means that different aspects of the stimulus may be simultaneously transduced in different parts of the body and a summation of the resulting sensory afference may trigger or shape different parts of the reflex response relatively independently. Understanding which properties of a reflex-evoking stimulus affect the response may provide insight into the how the stimulus is sensed and which mechanoreceptors are involved in the sensing process.

Perturbation studies in standing balance have shown that early components (first 75 ms) of the lower limb muscle response are graded to platform velocity, and that later components (150 to 500 ms) of the muscle response are graded to platform displacement (Diener et al., 1988; Horak et al., 1989). Lower-limb muscle responses to perturbations during gait have been shown to vary with support surface acceleration and displacement (Dietz et al., 1987). Reflex activation of the neck muscles has been measured using perturbations in standing postures (Woollacott et al., 1988; Keshner et al., 1988), seated postures (Gresty et al., 1989; Forssberg and Hirschfeld, 1994; Brault et al., 2000) and supine postures (Bisdorff et al., 1994, 1999; Ito et al., 1995, 1997), however the gradation of the neck muscle response with stimulus intensity has not been widely reported. Using whiplash-like perturbations, Brault et al. (2000) reported that the amplitude of the neck muscle response increased with stimulus intensity. Because perturbation velocity and acceleration varied simultaneously in this previous study, it was not possible to discern which factor was responsible for grading the muscle response. To date, there has been no systematic exploration of how neck muscle reflexes vary with different kinematic properties of a stimulus that perturbs posture.

The stimulus used in many postural perturbation studies has consisted of a fixed platform displacement achieved by an initial positive acceleration, a short period of constant velocity, and a final negative acceleration to rest. The duration from onset of the initial acceleration to termination of the final acceleration has typically varied from 125 ms to 300 ms. Since acceleration, and not velocity, is the destabilizing component of the stimulus, these fixed-displacement perturbations have

actually exposed subjects to two sequential perturbations: an initial acceleration that destabilized posture and a final acceleration that may have helped to restore posture. With practice, subjects may have incorporated this predictable final acceleration into their response strategy (McIlroy and Maki, 1994), a phenomenon that might partially explain the previously-observed gradation of late muscle components with platform displacement (Diener et al., 1988). In order to minimize possible contamination of the muscle and kinematic responses by the final acceleration, either the time interval between the initial and final accelerations can be increased, or the amplitude of the final acceleration, and therefore its potential posture-restoring contribution, can be decreased (McIlroy and Maki, 1994). The resulting perturbation, consisting predominantly of a single destabilizing acceleration, can then be used to study how the muscle and kinematic responses are graded to specific kinematic properties of a single perturbing stimulus.

The goal of this study was to examine how the neck muscle response and the kinematics of the head and upper torso varied with different kinematic properties of a perturbing stimulus. Seated subjects, with back support, were used to concentrate the postural response to the head and neck. Although a fixed-displacement perturbation was used, the duration of the constant velocity interval was increased and the amplitude of the final acceleration was decreased to better isolate the perturbation to a single acceleration. Three kinematic properties of the initial acceleration pulse were studied: its acceleration, its velocity (or velocity change) and its duration. These kinematic descriptors of the perturbation pulse were physically related, and therefore could not be varied independently. For this reason, each of these three parameters was in turn held constant while the other two parameters were varied simultaneously. It was hypothesized that the timing and amplitude of both the reflex muscle response and the induced kinematic response would vary with different stimulus properties. The resulting stimulus-response relationships might provide insight into the relative contributions of different mechanoreceptors to triggering the reflexive muscle response produced during seated postural perturbations.

6.2 Methods

6.2.1 Subjects

Thirty subjects with no history of sensory or motor dysfunction participated in the experiment. Physical characteristics for the subjects are given in Table 6.1. All subjects gave their informed consent and were paid a nominal amount for their participation. The use of human subjects for this experiment was approved by the university's Ethics Review Board and the study conformed with the Declaration of Helsinki.

Table 6.1 Mean (S.D.) of subject age and physical characteristics.

	Female	Male
n	15	15
Age, yrs	21 (3)	25 (6)
Height, cm	166 (7)	175 (8)
Mass, kg	59 (8)	78 (14)

6.2.2 Instrumentation

Electromyographic (EMG) activity in the orbicularis oculi (OO), masseter (MAS), sternocleidomastoid (SCM) and cervical paraspinal (PARA) muscles was recorded bilaterally using 10 mm pre-gelled surface electrodes (H69P, Kendall-LTP, Huntington Beach, CA) and an Octopus AMT-8 amplifier (Bortec, Calgary, AB). Two uniaxial accelerometers (Kistler 8302B20S1; ±20g, Amherst, NY) and a single uniaxial angular rate sensor (ATA Sensors ARS-04E; ±100 rad/s, Albuquerque, NM) were strapped tightly to the midline of a subject's forehead, immediately above the glabella. The sensitive axes of the accelerometers were mutually orthogonal and oriented to measure horizontal (x') and vertical (z') acceleration in the mid-sagittal plane (Figure 6.1). The rotational axis of the angular rate sensor was oriented mediolaterally to measure flexion and extension motion in the sagittal plane. Torso acceleration was measured using an uniaxial accelerometer (Kistler 8302B20S1; ±20g, Amherst, NY) applied with adhesive over the manubrium about 2 cm below its superior margin. The sensitive axis of the torso accelerometer was orthogonal to the skin over the manubrium and therefore could only be used for within-subject comparisons. Sled acceleration was measured using an uniaxial accelerometer (Sensotec JTF3629-05; ±10g, Columbus, OH) oriented horizontally along the axis of motion. Displacement was measured using an Optotrak motion analysis system (Northern Digital, Waterloo, ON) with markers placed over the left mastoid process and midline at the subject's forehead, vertex of the head, manubrium and C7 spinous process (Figure 6.1). A sixth marker was placed on the left seat hinge. A force transducer (Artech S-Beam SS20210, ±2 kN, Riverside, CA) was used to measure reaction loads during normalizing contractions of the SCM and PARA muscles. EMG signals were bandpass filtered at 10 Hz to 1 kHz and transducer signals were low-pass filtered at 1 kHz before being simultaneously sampled at 2 kHz and stored for subsequent analysis. Optotrak data were acquired at 200 Hz per marker.

6.2.3 Description of Perturbations

An automobile seat obtained from the front passenger location of a 1991 Honda Accord was mounted on a custom fabricated sled powered by a feedback-controlled linear induction motor

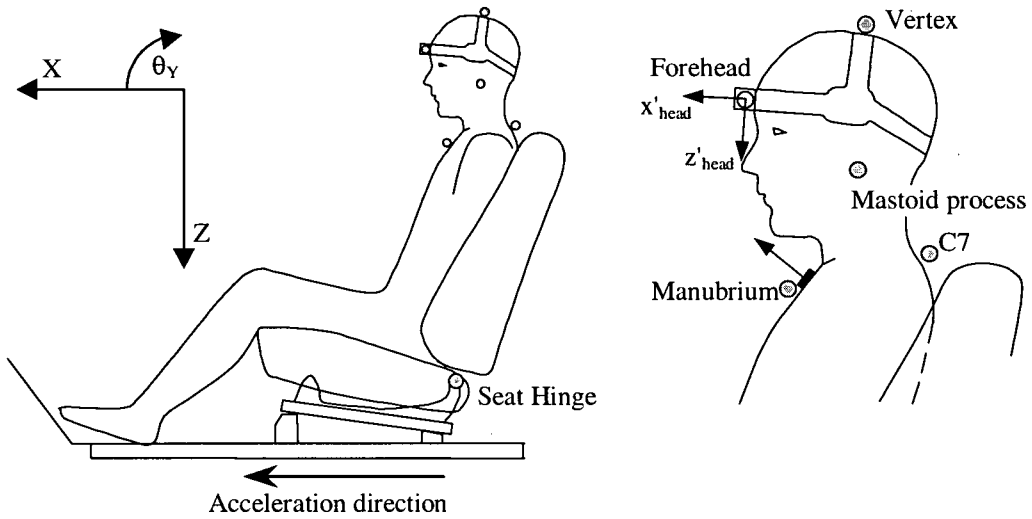


Figure 6.1 Schematic of the test configuration showing the locations of the Optotrak markers (shaded circles), the lab reference frame (X, Z) and the head reference frame (x', z'). The initial orientation of the head reference frame was determined by how the head band fit the subject and varied between +12 ± 4 deg relative to the lab reference frame. The sensitive axis of the torso accelerometer was also determined by a subject's body shape and varied between +30 ± 5 deg relative to the lab reference frame.

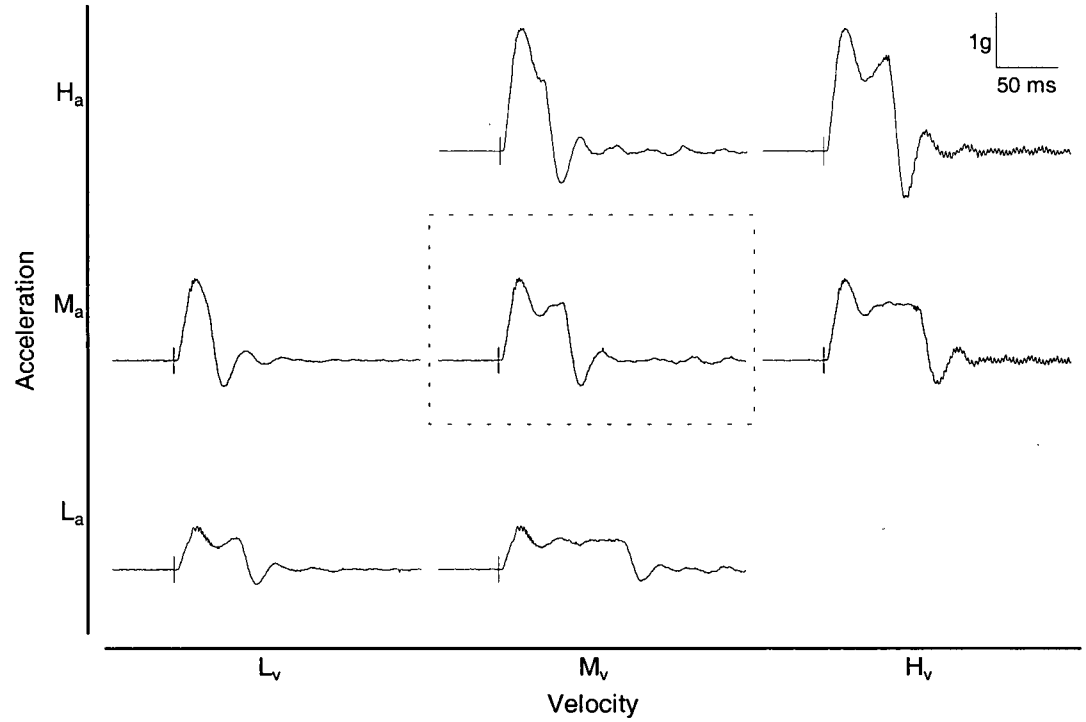
(Kollmorgen IC55-100A7, Kommack, NY). The head restraint was removed from the top of the seat back to eliminate the potential for an externally applied load to the head during the perturbations. The sled generated no pre-perturbation signals, either audible or mechanical, which subjects could use to predict onset of the perturbation.

Seven different perturbations were used in this study (Figure 6.2, Figure 6.3). Three velocity changes (Δv = 0.25, 0.50 and 0.75 m/s) and three average accelerations ($\bar{a}$ ~ 0.5, 0.9 and 1.3 g) were used in different combinations to produce the seven pulses. The pulses could be grouped in three ways: three pulses reached a common velocity of 0.50 m/s using the three different accelerations (middle column of Figure 6.2), three pulses used a similar average acceleration of about 0.9 g to reach three different velocities (middle row of Figure 6.2), and three pulses acted over a common duration of 60 ms using three different accelerations to reach three different velocities (diagonal in Figure 6.2). The "standard" perturbation, which consisted of the medium velocity (M_v=0.50 m/s) and medium acceleration (M_a=0.90 m/s^2) was common to each of the three groupings (dashed boxes in Figure 6.2). Low and high accelerations were designated L_a and H_a respectively, and low and high velocities were designated L_v and H_v respectively. Depending on the specific pulse, acceleration occurred over a distance of 4 mm to 32 mm. The sled slowed to rest at the end of its 750-mm throw at 0.05 g, about 10 percent of the lowest test acceleration. The total elapsed time from onset of initial acceleration to rest varied between 1.8 and 3.3 seconds.

These seven pulses were used to examine how muscle and kinematic responses varied with velocity, acceleration and pulse duration. The simple physical relationship between velocity change (Δv), average acceleration ($\bar{a}$) and pulse duration (Δt) for a square-wave pulse is depicted in Figure 6.4, wherein pulses of constant duration lie along straight lines radiating from the origin. The solid circles in Figure 6.4 depict the seven experimental pulses arranged in a similar manner to that in Figure 6.2. This two-dimensional matrix arrangement of the seven pulses was also used to report some of the results in later figures. A fourth variable, the product of the average acceleration and the velocity change, was also considered in the analysis. This $\bar{a}\Delta v$ product was a means of combining the effects of velocity change and acceleration, and the hyperbolic curves shown in Figure 6.4 represented curves along which this product remained constant.

6.2.4 Test Procedures

Each subject underwent a single block of 36 perturbations: 12 standard perturbations and 4 each of the other six perturbations. The order of presentation was randomized between subjects and the time between individual perturbations varied randomly (uniform distribution) between 20 and 30 seconds. Subjects were instructed to sit normally, face forward, rest their forearms on their lap and



Acceleration	L_v	M_v	H_v
H_a		$\bar{a}$ = 1.263 (0.005) g Δt = 43.2 (0.1) ms a_p = 2.21 (0.01) g t_p = 17.5 (0.7) ms	$\bar{a}$ = 1.354 (0.005) g Δt = 60.1 (0.2) ms a_p = 2.21 (0.01) g t_p = 17.6 (0.7) ms
M_a	$\bar{a}$ = 0.851 (0.005) g Δt = 33.4 (0.2) ms a_p = 1.50 (0.02) g t_p = 16.5 (0.8) ms	$\bar{a}$ = 0.899 (0.002) g Δt = 59.9 (0.1) ms a_p = 1.50 (0.02) g t_p = 16.6 (0.7) ms	$\bar{a}$ = 0.934 (0.003) g Δt = 85.0 (0.3) ms a_p = 1.50 (0.02) g t_p = 16.6 (0.8) ms
L_a	$\bar{a}$ = 0.451 (0.003) g Δt = 59.9 (0.3) ms a_p = 0.79 (0.01) g t_p = 18.0 (0.6) ms	$\bar{a}$ = 0.477 (0.001) g Δt = 109.9 (0.1) ms a_p = 0.79 (0.01) g t_p = 17.9 (0.6) ms	
	Δv = 0.25 m/s	Δv = 0.50 m/s	Δv = 0.75 m/s
Velocity	L_v	M_v	H_v

Figure 6.2 Matrix of sled pulses and descriptive statistics. Acceleration vs. time graphs (top) for each combination of sled velocity (low L_v, medium M_v, and high H_v) and acceleration (low L_a, medium M_a, and high H_a). Descriptive statistics (bottom) for the velocity change (Δv), average acceleration ($\bar{a}$), pulse duration (Δt), peak acceleration (a_p) and time of peak (t_p) corresponding to each of the seven pulses. The standard pulse (M_v, M_a), in the dashed box, was used for the initial eleven practice trials.

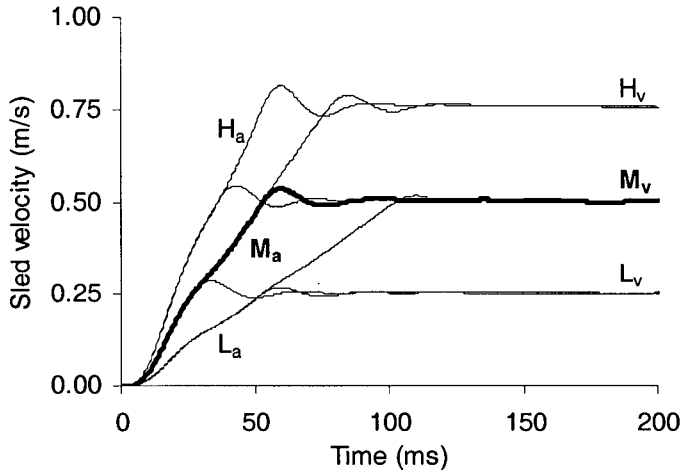


Figure 6.3 Superimposed velocity v. time plots for each of the seven perturbation pulses. The dark line depicts the standard perturbation (M_aM_v).

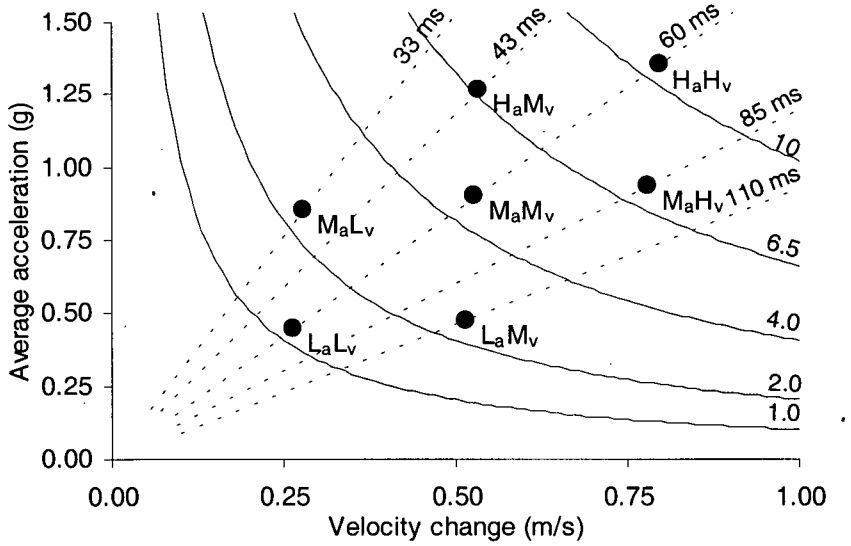


Figure 6.4 The relationship between average acceleration and velocity change for an idealized square wave perturbation pulse. The dashed lines radiating from the origin depict lines of constant pulse duration and the solid hyperbolic curves depict lines along which the product of acceleration and velocity is constant. The dark markers represent the seven pulses used in this experiment. Because actual pulses were not square waves, the peak velocity (plotted here) was larger than the actual velocity change. Units of time are milliseconds and units of the acceleration and velocity are m^2s^{-3}.

to relax their face and neck muscles. To achieve a stabilized response prior to testing, each subject first underwent eleven standard perturbations.

Prior to the above protocol, seated subjects performed sub-maximal isometric contractions in flexion and extension to generate normalizing data for the SCM and PARA muscles respectively. A strap attached to the load cell was placed around a subject's head and its length adjusted to ensure the subject's head was neutrally positioned. The strap was located immediately above the glabella for flexion contractions and at the height of the external occipital protuberance for extension contractions. Subjects were instructed to generate a force of 50 N with visual feedback, first in flexion and then in extension. EMG and load cell data were acquired for 5 s during each contraction.

6.2.5 Data Reduction

A subject's initial position was determined from the Optotrak data immediately preceding the perturbation. Initial head position was defined in the laboratory reference frame using the horizontal (X-axis; positive forward) and vertical (Z-axis; positive downward) positions of the mastoid process relative to the seat hinge (Figure 6.1). Initial head angle in the sagittal plane was determined from the forehead and vertex markers and reported relative to the positive X-axis ($+\theta_y$ rotation corresponded to extension). Initial torso position was defined using the horizontal and vertical positions of the midpoint between the manubrium and C7 spinous process markers relative to the seat hinge, and torso angle relative to the horizontal plane was determined using the same two markers. The RMS accuracy of the position measurements from the Optotrak system was less than 0.1 mm, and based on marker separation, the RMS accuracy of the calculated angles was less than 0.1 degrees.

The onset of head and torso movement, amplitude of peak head and torso acceleration and the time of the peak head and torso accelerations were determined directly from the accelerometer data. The algorithm used to determine onset is developed in Appendix A. Forehead acceleration data were not resolved into the lab reference frame. Peak retraction, defined as the maximum horizontal translation of the top of the cervical spine rearward relative to the bottom of the cervical spine, was estimated using the maximum relative horizontal displacement in the lab reference frame between the Optotrak markers on the mastoid and manubrium. Peak angular velocity of the head was determined from the angular rate sensor data after it had been digitally compensated to reduce the sensor's high-pass frequency to 0.002 Hz (Laughlin, 1998). Angular acceleration was then computed by finite differences (5 ms window) from the compensated angular velocity data. Total head angular displacement was determined from the Optotrak markers located at the forehead and vertex. Horizontal and vertical accelerations at the mastoid process were computed by double differentiating the Optotrak data. This technique produced results within 0.1g of the values computed using a rigid

body transformation of the linear and angular accelerations measured at the forehead and was preferred because it relied on data from only one Optotrak marker rather than data from two accelerometers, an angular rate sensor and two Optotrak markers needed for the rigid body calculation. Mastoid accelerations were computed in the lab reference frame.

Pre-stimulus noise in each channel of the EMG data was quantified using the root mean squared (RMS) amplitude of the EMG signal over the 100 ms preceding the perturbation. EMG onset was defined as the time at which the RMS amplitude, computed from the raw EMG data using a moving 20 ms window, reached 10 percent of it maximum value and was then confirmed visually. For each muscle, the RMS amplitude of the EMG signal was calculated for the interval between EMG onset and peak head extension angle. The corresponding pre-stimulus noise for each muscle was then subtracted from this quantity. The SCM and PARA muscle EMG amplitudes were normalized by the RMS amplitude obtained during the 5 s sub-maximal contraction for the corresponding muscle.

6.2.6 Statistical Analysis

Within-subject means of each dependent variable were calculated for each of the seven perturbations and then normalized by the within-subject mean of the twelve standard perturbations. A one-way repeated-measures ANOVA was then used to assess differences between the seven perturbations. Post-hoc testing for differences between individual pulses was performed using a Scheffé test. To examine the relationship between the dependent variables and the four different measures of the pulse (acceleration, velocity, duration and the $\bar{a}\Delta v$ product), separate linear regression analyses were performed for every combination of dependent variable and measure of perturbation intensity. The coefficients of determination were then examined to evaluate which measure of perturbation intensity correlated best with the dependent variables. A multiple regression analysis was not conducted because the four measures of perturbation intensity were not independent of each another. All ANOVAs were performed using Statistica (v.5.1, Statsoft Inc., Tulsa, OK) and a significance level of α=0.05. Linear regression analyses were conducted using Excel (v.7.0, Microsoft Corp., Redmond, WA).

6.3 Results

The initial position and angle of the head and upper torso prior to each perturbation did not vary between the different pulses. During each perturbation, a stereotypical response was observed in all subjects (Figure 6.5). The torso was accelerated forward first, followed by the head, which was accelerated linearly upward and forward, and angularly into extension. Most subjects responded to the perturbation by rapidly restoring their upright head position, although some subjects periodically

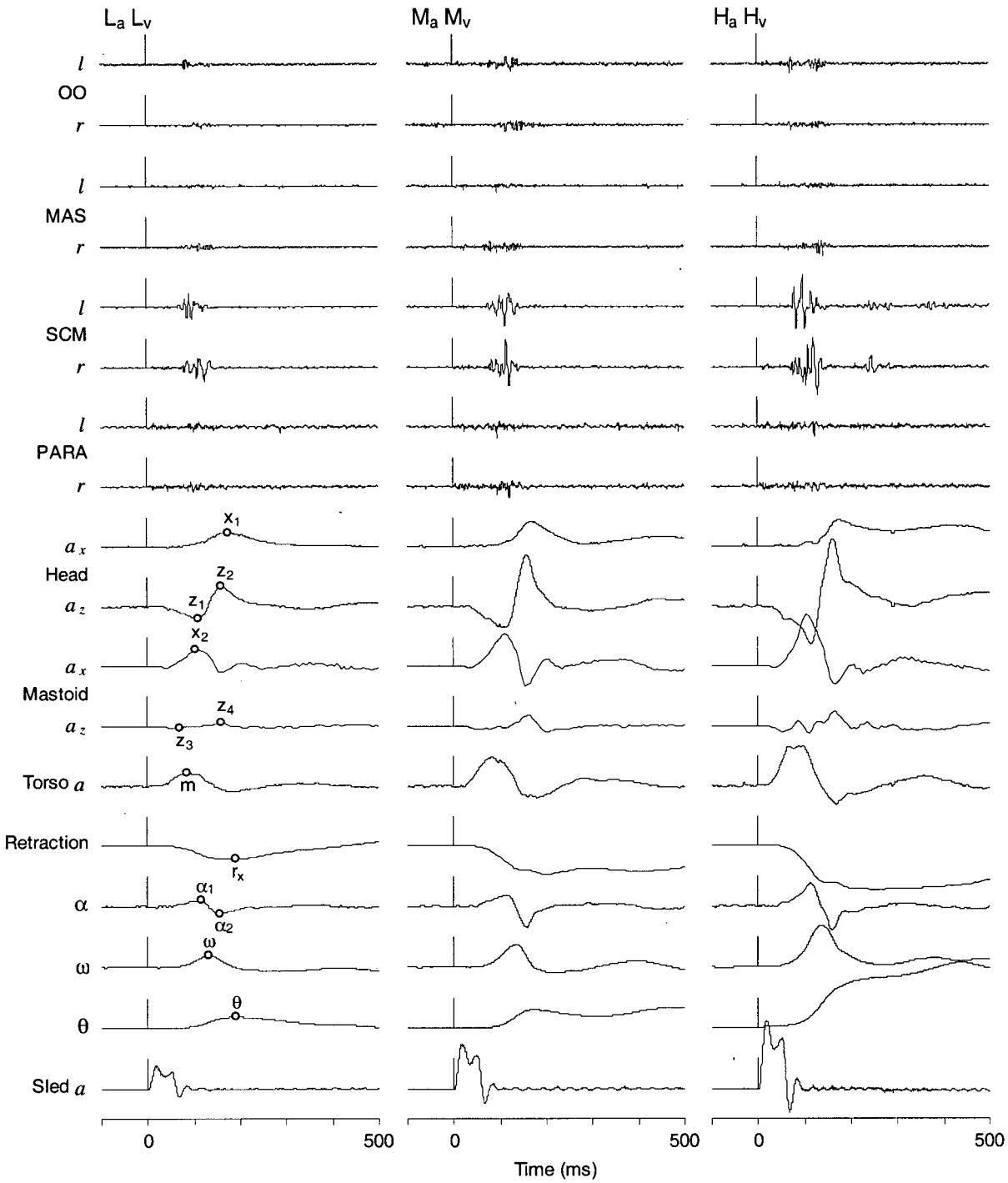


Figure 6.5 Sample data from a low velocity, low acceleration (L_vL_a), medium velocity, medium acceleration (M_vM_a), and high velocity, high acceleration (H_vH_a) perturbation for a single subject. Labeled hollow circles in the left panel represent kinematic peaks were used for subsequent analyses. The vertical scale bars are aligned with perturbation onset and equal to 1g, 50 mm, 200 rad/s², 5 rad/s and 20 deg. OO, orbicularis oculi; MAS, masseter; SCM, sternocleidomastoid; PARA, cervical paraspinal, l, left; r, right; *a*, linear acceleration, subscript *x* and *z* refers to the x- and z-directions; m, torso acceleration; r_x, retraction; α, head angular acceleration; ω, head angular velocity; θ, head angle.

altered their response and did not attempt to restore their upright head position until later. In most trials in which subjects responded in this manner, a clear peak was still visible in both the retraction and head angle data.

The amplitude of all EMG and kinematic parameters (identified by the labeled hollow circles in Figure 6.5) varied significantly between the perturbations ($p<0.0001$ for all variables; see Appendix C for a complete tabulation of all un-normalized data). Post-hoc comparisons revealed that the high acceleration, high velocity (H_a,H_v) perturbation consistently produced the largest amplitude response (Figure 6.6). In keeping with this finding, the lowest amplitude responses occurred in the low acceleration, low velocity (L_a,L_v) perturbation, though these responses were often not significantly different from those that occurred in perturbations from adjacent cells in the test matrix (e.g., L_a,M_v and M_a,L_v). Homogenous groups, determined via post-hoc testing, are shown in Figure 6.6 using outlines of increasing weight to represent increasing amplitude. Overall, the pattern visible in the weighted outlines in Figure 6.6 indicated that amplitude of both the EMG and kinematic responses increased with both acceleration and velocity change. To explore this pattern further, the amplitude of each of the EMG and kinematic variables was linearly regressed against each of the four descriptors of the perturbing pulse ($\bar{a}$, Δv, Δt and $\bar{a}\Delta v$). Aside from the regression analyses against pulse duration, all of the regression analyses yielded coefficients of determination that were statistically significant (Table 6.2). A comparison between the time at which each kinematic peak occurred in the standard pulse and its corresponding coefficient of determination showed that the amplitude of the early kinematic peaks correlated strongly with pulse acceleration, but that the strength of these correlations declined for later kinematic peaks (Figure 6.7). In contrast to average acceleration, the correlations with velocity change were weaker for the amplitude of the early kinematic peaks and stronger for the amplitude of the later kinematic peaks. Unlike either average acceleration or velocity change, the correlations between the $\bar{a}\Delta v$ product and the dependent variables remained uniformly high ($r^2>0.75$) across the whole time interval spanned by the kinematic response peaks considered here. The coefficient of determination observed for the $\bar{a}\Delta v$ product was only exceeded by the other pulse descriptors in four of the 12 response peaks: once by the regression against acceleration for the first kinematic peak (z_3) and three times by the regressions against velocity change for the last three kinematics peaks (x_1 ,r_x, and θ) (Table 6.2).

The timing of all EMG and kinematic parameters also varied significantly between the perturbations ($p<0.0001$ for all variables). Post-hoc comparisons for the neck muscle latencies and some early kinematic parameters revealed two patterns, both of which were different from the pattern observed for response amplitudes. In Figure 6.8, homogeneous groups were again outlined, however in this figure, increasing outline weight represented increasingly short latencies relative to

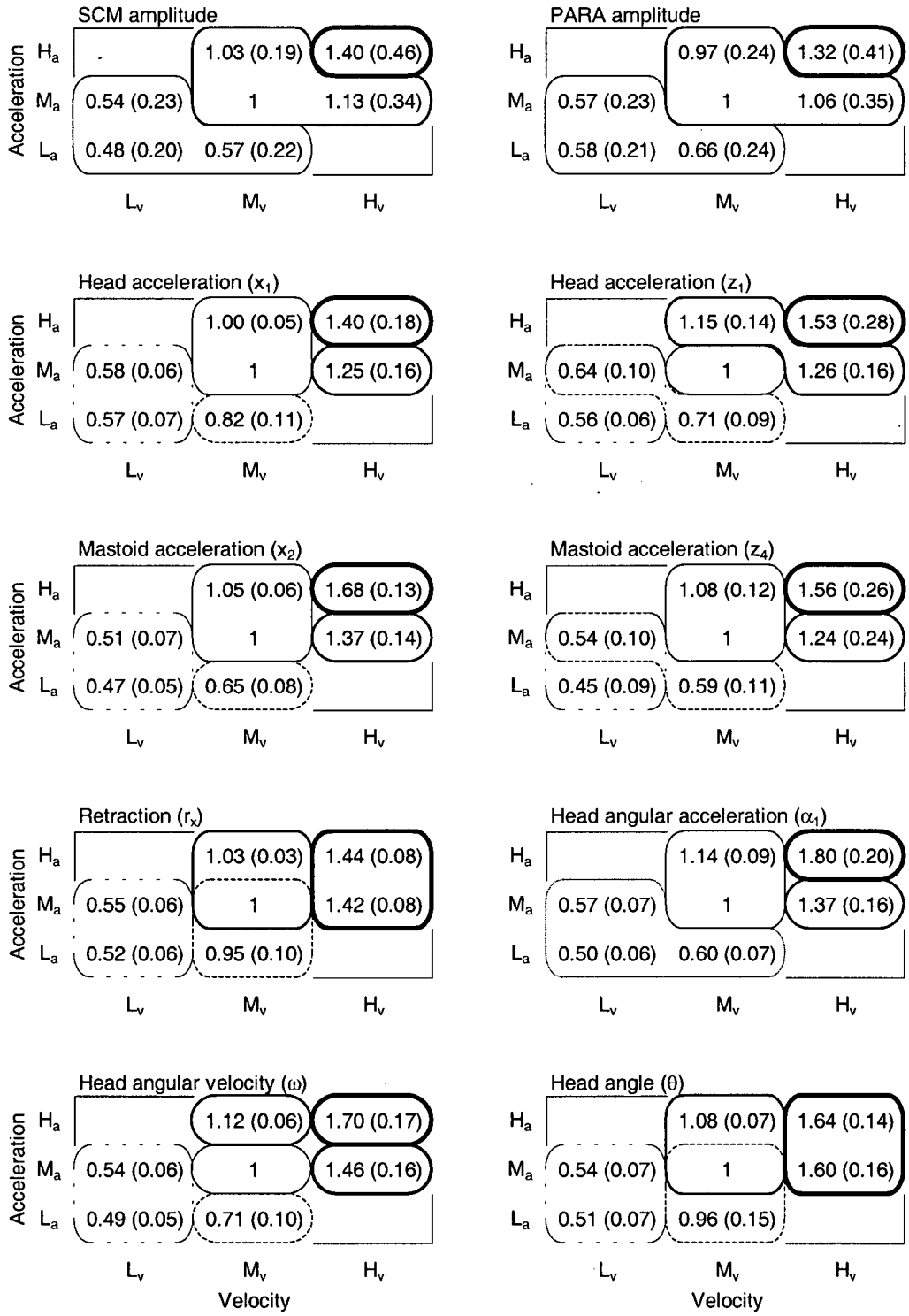


Figure 6.6 Mean and standard deviation of normalized electromyographic amplitude and peak kinematics as a function of perturbation pulse parameters. The borders encircle amplitudes which are not significantly different from each other according to a post-hoc Scheffé test on the normalized data. Increasing line weight corresponds to increasing amplitude. Note that larger amplitudes are consistently in the top right and lower amplitudes are consistently in the bottom left of each table.

Table 6.2 Coefficients of determination (r^2) for every combination of normalized dependent variable and perturbation pulse parameters. Kinematic amplitudes sorted temporally based on the average time observed in the standard perturbation. The largest r^2 for each variable in bold text. An r^2=0.02 was significant at the p=0.05 level. Δv, velocity change; $\bar{a}$, average acceleration; Δt, pulse duration; $\bar{a}\Delta v$, product of average acceleration and velocity change.

		Dependent variable	Time (ms)	Coefficients of determination (r^2)			
				Δv	$\bar{a}$	Δt	$\bar{a}\Delta v$
Amplitude	EMG	SCM amplitude	-	0.47	0.47	0.00	**0.58**
		PARA amplitude	-	0.40	0.36	0.00	**0.48**
	Kinematics	Mastoid acceleration (z_3)	64	0.47	**0.80**	0.05	0.76
		Torso acceleration (m)	76	0.86	0.69	0.01	**0.94**
		Head acceleration (z_1)	95	0.69	0.66	0.00	**0.84**
		Head angular acceleration (α_1)	108	0.75	0.70	0.00	**0.93**
		Mastoid acceleration (x_2)	109	0.83	0.65	0.01	**0.93**
		Head angular velocity (ω)	135	0.84	0.63	0.01	**0.92**
		Mastoid acceleration (z_4)	142	0.69	0.66	0.00	**0.85**
		Head acceleration (z_2)	170	0.70	0.65	0.00	**0.85**
		Head angular acceleration (α_2)	171	0.59	0.62	0.00	**0.78**
		Head acceleration (x_1)	178	**0.83**	0.52	0.04	0.82
		Retraction (r_x)	191	**0.95**	0.42	0.12	0.80
		Head angle (θ)	212	**0.93**	0.44	0.10	0.81
Time	EMG	OO onset	-	0.05	0.01	0.08	0.01
		MAS onset	-	0.16	0.00	**0.21**	0.05
		SCM onset	-	0.06	0.07	**0.30**	0.00
		PARA onset	-	0.00	0.02	0.03	0.00
	Kinematics	Torso acceleration onset	-	0.12	**0.55**	0.20	0.35
		Head acceleration onset	-	0.09	**0.44**	0.14	0.27
		Torso acceleration peak	-	0.12	0.21	**0.90**	0.00
		Mastoid acceleration peak (z_1)	-	0.02	0.21	**0.56**	0.03

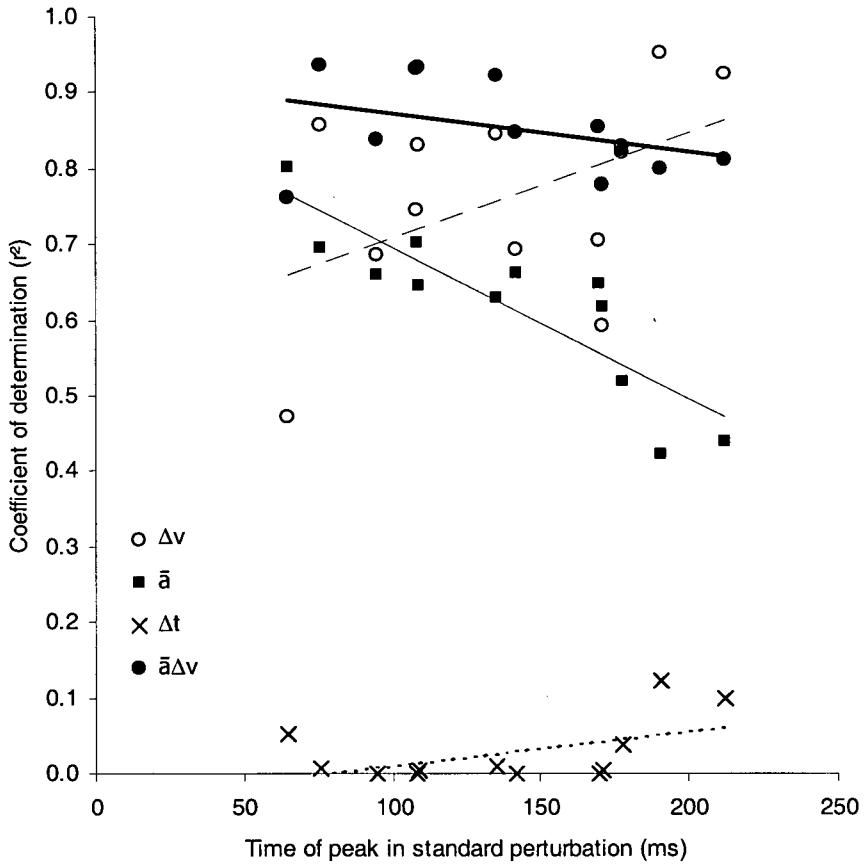


Figure 6.7 A plot of the coefficients of determination (r^2) produced by linear regression analyses between each of the dependent variables and the perturbation pulse parameters (acceleration, velocity change, duration, and product of acceleration and velocity) as a function of the time at which the corresponding peak amplitude occurred relative to the onset of the perturbation. Straight lines are least-squares best fit lines through the data.

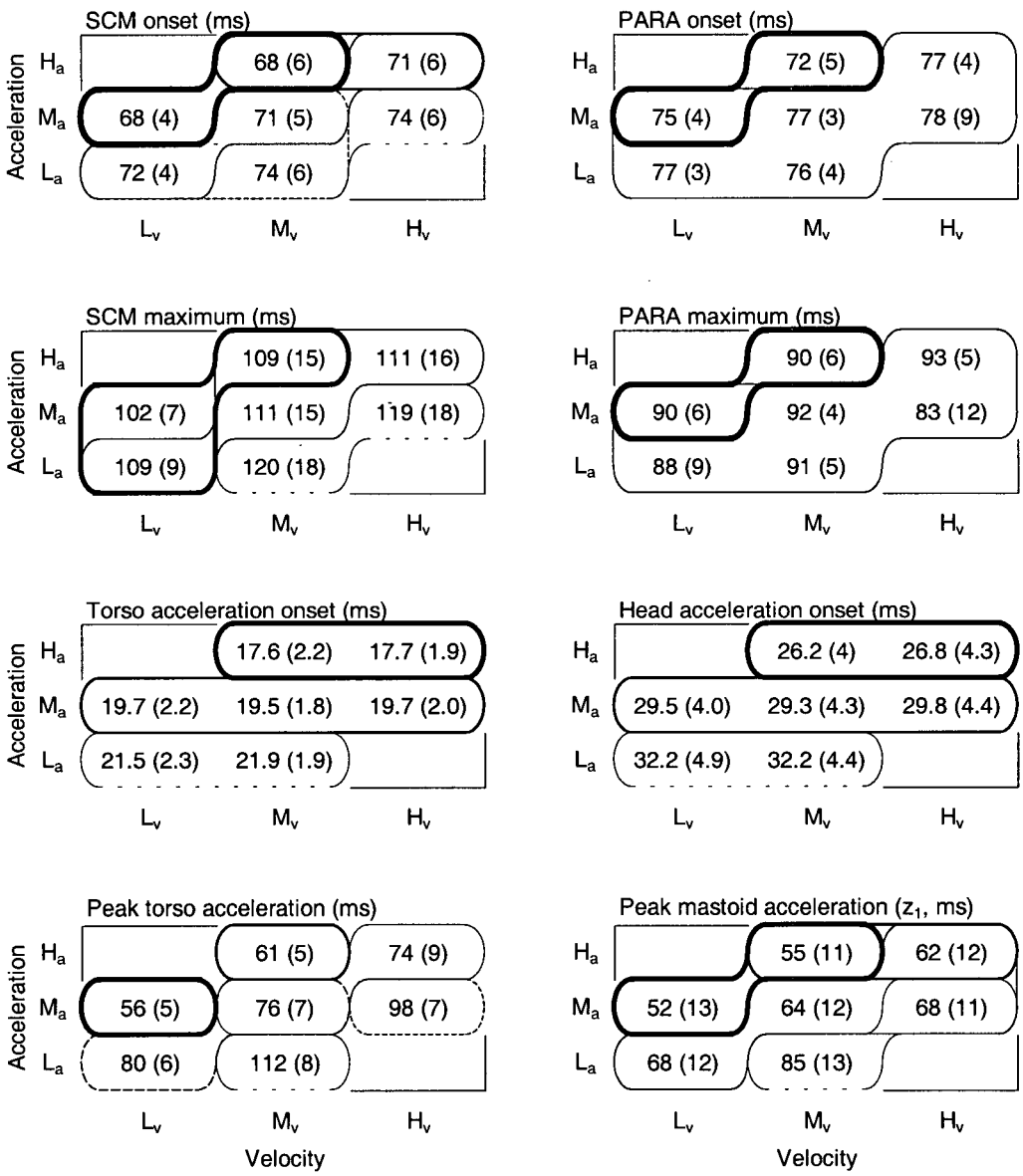


Figure 6.8 Mean and standard deviation of electromyographic onset times and the time of selected peak kinematics as a function of perturbation pulse parameters. The borders encircle times which are not significantly different from each other according to a post-hoc Scheffé test on the normalized data. Increasing line weight corresponds to increasingly early times.

perturbation onset. The times of the earliest kinematic parameters – onset of torso acceleration and onset of head acceleration – correlated most strongly to the acceleration of the pulse (Figure 6.8, Table 6.2). This relatively strong correlation with pulse acceleration was not observed in the first peak in the torso acceleration or in the first peak in the vertical head acceleration. Both of these two latter dependent variables correlated more strongly with pulse duration and reached minimum values for the shortest duration pulse (M_aL_v – 33 ms) and maximum values for the longest duration pulse (L_aM_v – 110 ms) (Figure 6.8). A similar pattern was present in the onset times of the SCM muscles, though not in the PARA muscles.

To investigate a possible role for the cervicocollic and vestibulocollic reflexes in triggering the response of the SCM muscle, specific correlations between different onset times were also determined. SCM activation times correlated significantly, though relatively weakly, with both the onset of torso acceleration (r^2=0.08, $p<0.0001$) and the onset of head acceleration (r^2=0.05, $p<0.0007$).

6.4 Discussion

The goal of this study was to quantify how the reflex response of neck muscles and the induced kinematic response of the head and neck varied with different properties of a horizontal forward perturbation in seated subjects. This goal was achieved by exposing habituated subjects to seven randomly-presented perturbations consisting of different combinations of average acceleration, velocity change and duration. The results of this study clearly showed that muscle and kinematic responses were graded to stimulus intensity, however, there was not one descriptor of stimulus intensity that explained the gradation seen in the peak amplitude and timing of all muscle and kinematic variables. Instead, these dependent variables could be divided into groups based on the stimulus property to which they most strongly correlated.

6.4.1 Kinematic Response

The strongest correlations between the perturbation properties and subject responses were observed in the amplitude of the kinematic variables. Of the four descriptors of perturbation intensity evaluated here, only perturbation duration (Δt) did not correlate significantly with the peak amplitude of all of the induced kinematics. Average acceleration ($\bar{a}$) exhibited strong correlations with the amplitude of early kinematic peaks, however the strength of these correlations diminished as the kinematic peaks became temporally more remote from the perturbation. Although more inconsistent than average acceleration, correlations between the peak amplitude of the kinematics and the velocity change (Δv) of the perturbation exhibited the opposite pattern. The strength of these correlations was

generally lower for early kinematic peaks and higher for temporally later kinematic peaks. Since some of the early kinematic peaks occurred before the sled reached its final velocity, a poor correlation between velocity change and the amplitude of these early kinematic peaks was not surprising. Similarly, kinematic peaks that occurred well after the end of the acceleration correlated more strongly with the net effect of the acceleration – namely the velocity change – than the acceleration used to produce that velocity change. This pattern, though perhaps obvious, revealed how different components of the kinematic response were related to different properties of the perturbation, and highlighted the difficulty of quantifying stimulus-response relationships for reflexes even within a simplified perturbation that contained only a single acceleration event.

Unlike either average acceleration or velocity change individually, the product of these two descriptors ($\bar{a}\Delta v$) correlated consistently strongly with the amplitude of the peak kinematics over the 250-ms interval spanning the kinematic responses considered in this study. Though the first kinematic peak (upward head acceleration, z_3) correlated more strongly with acceleration, and the last three kinematics peaks (forward head acceleration x_1, peak retraction r_x, and peak head angle θ) correlated more strongly with velocity change, the differences between these coefficients of determination and those achieved using $\bar{a}\Delta v$ were not large (Table 6.2). Thus, amongst the pulse-based measures considered here, $\bar{a}\Delta v$ was the best overall predictor of kinematic response amplitude.

In general, the peak kinematics occurred earlier for perturbations of shorter duration. This same pattern was not present in the onset times of the earliest kinematic events: onset of torso acceleration, which occurred about 17 to 22 ms after onset of the perturbation, and onset of upward forehead acceleration, which occurred about 26 to 32 ms after the onset of the perturbation. Both onset times exhibited the strongest correlations to average acceleration, a finding consistent with both events occurring prior to the end of the acceleration in even the shortest perturbation duration (M_aL_v Δt=33 ms). In contrast, the timing of the first two peak kinematic responses, peak upward mastoid acceleration at 52 to 85 ms and peak torso acceleration at 56 to 112 ms, no longer exhibited a strong correlation with average acceleration. Instead, these variables behaved like the times of the remaining kinematic peaks and correlated most strongly with perturbation duration. The change from a correlation with acceleration for the onset times of the earliest kinematics to a correlation with pulse-duration for the times of the initial peaks in these same kinematic variables suggested that the end of the pulse had a rapid effect on the kinematic response. This pattern further highlighted the difficulty of isolating different components of a perturbation in the study of stimulus-response relationships.

6.4.2 Muscle Response

Like the kinematics, the amplitude of the neck muscle response correlated more strongly with $\bar{a}\Delta v$ than with either average acceleration or velocity change. The strength of these correlations was lower for the amplitude of the neck muscles (r^2_{SCM}=0.58 and r^2_{PARA}=0.48) than for the amplitude of the peak kinematics (r^2=0.76 to 0.93), likely because of the proportionally larger variance present in the EMG data than in the kinematic data. These correlations were the same or stronger, however, than those observed between the amplitude of the EMG in the gastrocnemius muscle and the intensity of a perturbing stimulus in a study of stumbling reactions during gait (r^2=0.22 to 0.47; Dietz et al., 1987) and suggested a relatively strong gradation of reflexive muscle response to stimulus intensity.

The range of average onset latencies observed in the SCM muscles was small: between 68 and 74 ms. Their distribution across the different perturbations, however, revealed that shorter duration perturbations yielded shorter onset latencies. This pattern matched the timing of the kinematics peaks, which occurred earlier during short-duration perturbations than during long-duration perturbations. Similar small gradations in onset latency with stimulus intensity have been observed by others (Blumenthal, 1996), however the reason why the gradation observed here varied with pulse duration rather than some measure of stimulus amplitude was not clear. Muscle activation occurred both before some perturbations were complete and after others were complete – a situation that ruled out a causal relationship between pulse duration and muscle onset. Moreover, the strength of the correlation between SCM onset latency and pulse duration did not increase when the two longer perturbations (Δt=85 and 110 ms) were omitted from the regression analysis. Therefore, based on the current data, the graded pattern of SCM onset latencies appeared to be related to a variable which was not considered in this study but which was nonetheless related in some way to the duration of the perturbation.

Unlike the SCM muscle, a clear pattern was not observed in the onset latencies of the PARA muscles (Figure 6.8). The absence of a pattern in the PARA muscles may be related to the small response amplitudes observed in these muscles and the resulting difficulty encountered in determining its onset from the EMG data. Low levels of activity have been previously observed in the PARA muscles when they have acted as antagonists in subjects habituated to a perturbation (Allum et al., 1992; Forssberg and Hirschfeld, 1994). For this reason, further interpretation of the onset latency data was isolated to the SCM muscles.

The mechanoreceptors responsible for triggering neck muscle responses in postural perturbations have been debated in the literature (Woollacott et al, 1988; Keshner et al., 1988; Horak et al., 1994; Forssberg and Hirschfeld, 1994; Allum et al., 1997; Allum et al. 1998). Rotation about the ankles was originally thought to trigger automatic postural responses in perturbed standing

(Nashner et al., 1976), however, Keshner et al. (1988) showed that neck muscle activation occurred at about the same latency as lower limb muscle activation (about 95 ms) and concluded that ankle rotations alone did not adequately explain the neck muscle response. Forssberg and Hirschfeld (1994) subsequently perturbed seated subjects and observed SCM muscle activation at about 77 ms. Using various perturbation directions, they observed different muscle activation patterns despite the presence of similar head kinematics and therefore excluded a major vestibular component to the postural response. Instead, these authors concluded that mechanoreceptors in the pelvis, possibly related to contact with their rigid platform, were likely responsible for triggering the postural response. Bisdorff et al. (1994, 1999) used a whole-body free-fall in supine subjects to evoke large postural responses in the neck muscles at 54 to 60 ms. Based on similar responses in normal and avestibular subjects, these authors concluded that sudden changes in cutaneous pressure and joint forces might be responsible for triggering the neck muscle response. Head-only drop tests in supine subjects have produced short SCM activation times of 22 to 25 ms (Ito et al., 1995, 1997; Bisdorff et al., 1999). Due to an absence of this short latency response in avestibular patients (onset occurred at 67 ms in these patients), these short activation times were attributed to a vestibulocollic reflex (Ito et al., 1995). Rapid release of a load generated by a tonic neck muscle contraction has also been shown to evoke vestibulocollic reflexes at about 25 ms in the SCM muscles of normal subjects and stretch reflexes at about 41 ms in the SCM muscles of avestibular patients (Corna et al, 1996). Others have also reported stretch reflex latencies in the neck muscles of 50 to 60 ms (Horak et al., 1994; Ito et al., 1995), although the long duration of these stretch reflexes indicated that they were likely not similar to the monosynaptic stretch reflex observed in limb muscles, but more likely the polysynaptic cervicocollic reflex (Peterson, 1988).

Taken together, the results of these previous studies showed that the input of multiple mechanoreceptors converge on the motoneurones of the neck muscles and that all of them could play a role in triggering or modulating a postural response in the neck muscles. In the current study, mean onset times for torso acceleration varied from 17 to 22 ms, mean onset times for head acceleration varied from 26 to 32 ms, and mean onset latencies for the SCM muscles varied from 68 to 74 ms. Therefore SCM activation occurred about 50 ms after the onset of upper torso acceleration and 40 ms after the onset of head acceleration. Based on the previously-reported SCM onset latencies reviewed above, this was sufficient time for both the cervicocollic and vestibulocollic reflexes to mediate the SCM response observed in the current study and a simple timing analysis could not be used to exclude either reflex. The pattern of activation observed over the different perturbations used in the present study, however, provided some additional evidence with which the relative contribution of these reflex pathways could be evaluated further.

Torso acceleration was measured at the manubrium – the origin for the sternal heads of both sternocleidomastoid muscles. Forward acceleration of the manubrium relative to an initially stationary head would stretch the SCM muscle and could provide the necessary afferent input to trigger a cervicocollic reflex. If this was the primary sensory pathway mediating the SCM activation, then a strong correlation between the onset time of torso acceleration and the onset of SCM activation would be expected. A strong correlation, however, was not observed in the current data. The onset of torso acceleration correlated most strongly with pulse acceleration, whereas the onset of SCM activity correlated most strongly with pulse duration. A regression analysis between the onset of torso acceleration and SCM onset latency yielded a coefficient of determination of r^2=0.08. This low correlation suggested that cervicocollic reflexes played at most a minor role in the activation of the SCM muscles in the perturbations studied here.

The onset of head acceleration occurred about 40 ms before SCM activation, an interval about 15 to 20 ms longer than that needed for the vestibulocollic reflex to act (Ito et al., 1995; Bisdorff et al., 1999). Like the onset of torso acceleration, the onset of head acceleration correlated best with the average acceleration of the perturbation, whereas SCM activation correlated best with pulse duration. A regression analysis between the onset of head acceleration and the onset of SCM activation yielded a coefficient of determination of r^2=0.05, a result which suggested that the role of the vestibulocollic reflex in triggering SCM activation was small as well.

The apparently minor roles for both the cervicocollic and vestibulocollic reflexes in the SCM activation observed here were consistent with the conclusions reached by Forssberg and Hirschfeld (1994), even though peak linear head accelerations in the current study were 5 to 10 times larger and peak angular head accelerations were 10 to 15 times larger than those observed in this previous study. Despite the presence of a considerably larger muscle stretch stimuli and vestibular inputs, these reflex pathways still appeared to play only a minor role in triggering the SCM response. These large differences in head kinematics between the current study and the previous study of seated subjects by Forssberg and Hirschfeld (1994) were likely related to their perturbations pulse, which consisted of a fixed displacement of 80 mm at 0.36 m/s over 240 ms, and the absence of back support for their seated subjects. The perturbations used in the present study were both larger (up to 0.75 m/s), longer (750 mm) and consisted of a single large-amplitude initial acceleration with a much lower amplitude final acceleration. In addition, the presence of a seat back in the current study concentrated the kinematic response to the head and neck. The combined effect of these differences between studies likely explained these large differences in the amplitude of the head kinematics.

Horak et al. (1994) reported that the contribution of the vestibular system to postural control in standing was down-regulated in the presence of good somatosensory cues and up-regulated in their

absence. In the current study, the entire posterior aspects of the back, pelvis and thighs were in contact with the seat cushion and seat back, and would have provided subjects with widespread somatosensory information regarding the onset, duration and intensity of the perturbing stimulus. Given the apparently minor contribution of the two dominant head-and-neck-based reflexes for neck muscle activation, the trunk appeared to be the next most likely source of the somatosensory information that triggered SCM activation. Because of compliance in the seat back, the pelvis was likely the first part of the body to be accelerated forward by the perturbation. Spinal conduction velocities of 40 to 50 m/s have been measured in the ascending afferent pathways of the dorsal column of the human spinal cord (Cioni and Meglio, 1986; Halonen et al., 1989). Assuming a distance of 60 to 80 cm between the pelvis and brainstem, the afferent signals in the pelvis would require 12 to 20 ms to ascend the spinal cord. Given SCM onset latencies of about 70 ms, this analysis indicated that there was sufficient time for afferent signals in the pelvis and trunk to mediate the SCM response observed in the current study. Previous evidence of a 10 ms delay from the onset of lumbar paraspinal muscles to the onset of neck muscles in whiplash-like perturbations was consistent with this proposal (Szabo and Welcher, 1996). Therefore, the current findings supported a conclusion that the sensory information triggering a neck muscle response in seated perturbations originated remote from the head and neck and possibly in the pelvis, as predicted by Forssberg and Hirschfeld (1994), or more broadly from the trunk, as postulated by Allum et al. (1998). Whether the diffuse stimulation generated by seat contact excited mechanoreceptors in the skin, joints, muscles or other tissues could not be discerned from the results of the current study.

It has been postulated that the overall pattern of the response to a postural perturbation is centrally generated and then shaped by additional sensory input and biomechanical demands (Horak et al., 1994; Allum et al., 1998). Despite the poor correlation observed between SCM activation and movement onset in the upper torso and head, the amplitude of the SCM activity was strongly correlated to the peak amplitude of the upward mastoid acceleration (z_3, r^2=0.66) and the peak amplitude of the torso acceleration (r^2=0.77). Both of these kinematic peaks occurred at about the same time as SCM activation and well before peak SCM activity (102 to 120 ms). Although these strong correlations were not indicative of a causal relationship, they did indicate that the cervicocollic and vestibulocollic reflexes might have played a role in shaping the SCM muscle response.

6.4.3 Implications for Whiplash Injury

In the study of whiplash injury, the velocity change of a vehicle has been the most commonly used descriptor of collision severity. Its utility as a descriptor of collision severity, and by extension injury potential, was based on the assumption that the collision was complete before most of the

occupant motion occurred and that the aggregate effect of the acceleration – the velocity change – was therefore a valid predictor of the occupant response. Conceptually, the vehicle's velocity change has been thought of as the impact velocity of the seat back into the occupant (Siegmund and King, 1997).

The results of the current study, however, showed that the correlation between velocity change and peak kinematics varied with the time at which a particular kinematic peak occurred. Amongst the perturbation descriptors examined here, velocity change was only the best predictor of amplitude for the last three kinematic peaks. This result only supported the usefulness of velocity change as a descriptor of the peak occupant response and injury potential in whiplash collisions if the injury was produced by these late kinematics. To date, it remains unclear when during the induced kinematics an injury is produced, however, if the injury is related to earlier kinematics, then velocity change may not be the best predictor of these injury-inducing kinematics. Average pulse acceleration was a better predictor than velocity change of upward mastoid acceleration (z_3), but exhibited similar or lower correlations than velocity change for all subsequent kinematic peaks. The product of average acceleration and velocity change, $\bar{a}\Delta v$, appeared to offset the increasing correlations between velocity change and peak kinematic amplitude against the decreasing correlations between acceleration and peak kinematic amplitude. The $\bar{a}\Delta v$ product produced high correlations ($r^2>0.75$) with the amplitude of all kinematic peaks over the whole time span of the responses considered here and may therefore be a more robust measure with which to predict the peak amplitude of the occupant kinematics from a collision pulse. The inclusion of both acceleration and velocity change into a measure of collision severity would help to explain recent evidence of large variations in peak kinematics when different accelerations were used to generate a series of simulated collisions with the same velocity change (Siegmund and Heinrichs, 2001). It would also help to explain the increased incidence of long-term whiplash injuries observed in individuals whose vehicles were equipped with trailer hitches (Krafft et al., 2000). Impacts to the stiff hitch components produced collision pulses that had a higher acceleration and shorter duration than collisions of a similar velocity change for vehicles not equipped with a trailer hitch (Krafft et al., 2000). The $\bar{a}\Delta v$ product might therefore be a better measure than velocity change for predicting the magnitude of occupant kinematics in the kinds of collisions that produce whiplash injury.

Due to habituation, the absolute magnitude of the muscle and kinematic responses observed in the current study did not accurately reflect the response that would have been evoked in unprepared subjects exposed to only one perturbation. The onset latencies observed in the current study, however, were similar to those observed in subjects exposed to only one perturbation using the same experimental setup (Experiment 2A, Chapter 3), and suggested that the same reflex pathway was

mediating the SCM response despite habituation of the response magnitude. Therefore, the relative magnitude of the muscle and kinematic responses observed between the different perturbations used in the current experiment likely reflects the gradation in muscle and kinematics responses that would occur in unprepared subjects.

In summary, the results of the current experiment showed that no single descriptor of perturbation intensity predicted the gradation in amplitude and timing of the neck muscle response and the head and neck kinematics evoked in seated subjects exposed to horizontal forward perturbations. Overall, the product of the average acceleration and velocity change of the perturbation was the best perturbation-based predictor of the amplitude of the muscle and kinematic responses. The time of both activation and peak amplitude in the SCM muscles and the timing of all kinematic peaks correlated best with perturbation duration. A comparison of the temporal pattern of SCM activation and the onset of both torso and head acceleration over the seven perturbations used in this study suggested that neither the vestibulocollic reflex nor the cervicocollic reflex played a major role in activation of the SCM muscles during these perturbations. Other somatosensory information, such as a large cutaneous stimulus due to interaction with the seat, or muscle or joints receptors in the pelvis or trunk, was therefore a more likely source of the afferent signal triggering the SCM muscle response in these perturbations.

CHAPTER 7 GENERAL DISCUSSION AND CONCLUSIONS

The overall goal of this thesis was to assess two questions related to the testing of human subjects for whiplash injury research. The first question was whether, and to what degree, subject awareness regarding the presence, timing and amplitude of a whiplash-like perturbation affected the muscle and kinematic responses to that perturbation. The second question was whether, and to what degree, habituation over multiple perturbations affected the neck muscle response and the resulting head and neck kinematics. Both questions addressed the external validity of laboratory tests that have exposed human subjects to whiplash-like perturbations in order to study the biomechanics of whiplash injury. Real whiplash-producing collisions are typically one-time events that occur without warning. Laboratory tests, on the other hand, have often used multiple exposures or practice trials, and subjects participating in these tests have always known, to some degree, that a perturbation was imminent. These differences between the actual exposures that cause most whiplash injuries and the laboratory exposures used to study whiplash injuries may reduce the external validity of these human subject experiments.

Before exposing subjects to whiplash-like perturbations, the ability of subjects to alter a reflex neck muscle response was first studied using the acoustic startle reflex. In Experiment 1 (Chapter 2), readiness to perform a ballistic head movement in a simple reaction time protocol was shown to have a large effect on the reflex neck muscle response evoked by an acoustically startling stimulus. If subjects were prepared to execute a head flexion movement, then the startle-evoked response was a head flexion. Alternatively, if subjects were prepared to execute a head rotation movement, then the same startling stimulus evoked a head rotation. While it may be debated whether the prepared movement sculpted the startle response (Siegmund et al., 2001b) or whether the startle response released a prepared motor programme (Valls-Solé et al., 1999), the net effect was that the reflex response of the neck muscles had been altered by readiness to execute a specific movement. Although readiness to perform a ballistic movement in a simple reaction time protocol was likely an optimal setting in which to detect the alteration of a reflex response, the results of Experiment 1 nonetheless showed that the reflex response of the neck muscles was malleable. Experiment 1 also showed that readiness-induced alterations of the neck muscle reflex could occur without the movement being practiced. Together, these two findings suggested that conscious preparation of a response to a whiplash-like perturbation could potentially contaminate the muscle response during a subject's first exposure to a perturbation.

The ability to willfully alter a reflex response of the neck muscle, however, was not evidence

that subjects actually did so when exposed to whiplash-like perturbations. In Experiment 2A (Chapter 3), three groups of subjects were given different levels of information regarding the presence or timing of a single whiplash-like perturbation to examine whether they made pre-stimulus preparations that altered the reflex response of their neck muscles. Overall, the muscle and kinematic responses of subjects given precise information regarding the timing of their first perturbation (alerted subjects) responded in a similar manner to subjects given only imprecise information of when the perturbation would occur (unalerted subjects). In contrast, the response of a third group of subjects, who were deceived and perturbed unexpectedly (surprised subjects), was different from both the alerted and unalerted groups of subjects. The response differences consisted of a delayed neck muscle activation in all surprised subjects, a larger muscle contraction in surprised male subjects, and changes in the peak amplitude of some kinematic variables in female subjects. These results suggested that warning of the presence of a perturbation created anticipation, and that this event-related anticipation then facilitated the sensorimotor system and resulted in the earlier muscle activation observed in alerted and unalerted subjects than in surprised subjects. A similar sensorimotor facilitation may also have been responsible for the absence of habituation observed in Experiment 1. Precise or imprecise information of perturbation timing did not further alter the reflex muscle response – a finding that indicated that awareness of the perturbation (event awareness) had a larger effect on the response than did awareness of the timing of the perturbation (temporal awareness).

Experiment 2B (Chapter 4) showed that multiple sequential exposures produced a rapid attenuation of the neck muscle response consistent with habituation. The muscle response attenuated to about 35 to 60 percent of its original value and resulted in changes in the amplitude of peak kinematic parameters that varied between a 21 percent increase and a 29 percent decrease. This experiment showed that habituation of the neck muscle response to a whiplash-like perturbation resulted in kinematics that were significantly different from those observed during the first exposure and suggested that data from human subjects who had habituated to a perturbation might be of limited use in the study of whiplash injury mechanisms.

Experiment 2C (Chapter 5) showed that pre-stimulus awareness of perturbation magnitude had no affect on the muscle or kinematic response, however the applicability of this conclusion was limited to the range of perturbation intensities used in this study. With more noxious perturbations, it was possible that different results may have been observed. Experiment 2C also showed that the neck muscle and kinematic responses were graded with the acceleration of the perturbation. This finding led to a systematic exploration in Experiment 2D (Chapter 6) of how the neck muscle response and kinematics of the head and neck were graded to different properties of the perturbation. This

experiment showed that the amplitude of the muscle and kinematic responses were graded to both the acceleration and velocity change of the perturbation. In addition, an analysis of neck muscle activation times in relation to the onset of torso and head acceleration suggested that neither the cervicocollic reflex nor the vestibulocollic reflex played a large role in triggering the neck muscle response to a whiplash-like perturbation.

7.1 Methodology – Advantages and Limitations

All of the experiments conducted here focused on the reflex response of the neck muscles. Two methods of generating a reflex response were used: a loud noise capable of evoking the startle reflex and a rapid horizontal translation that evoked a postural response. In Experiment 1, a stimulus was required that did not directly contact the subjects. This allowed the effect of motor readiness on the reflex response of the neck muscles to be studied in a setting where all of the resulting kinematics were generated by the evoked muscle response. Both the muscle and kinematic responses could then be examined for changes related to motor readiness. The acoustic startle reflex produced a neck muscle contraction without direct contact to the subject and was therefore well suited to this experiment.

Whereas the requirements for the stimulus needed in Experiment 1 were quite simple, the requirements of the rapid horizontal translation used for the remaining perturbation experiments were more complicated. If an overarching goal of these experiments was to assess the external validity of human subject studies into whiplash injury, then it was important that the perturbation experiments adequately represented both a real whiplash exposure and the conditions used in many human subject experiments. Given the wide variety of vehicles and seats available on the market, it was not possible to examine issues of external validity related to these factors. Instead, a single seat, taken from the passenger location of a 1991 Honda Accord, was used for these experiments. This seat was chosen because it had been used in previous human subjects experiments in which issues of habituation and subject awareness factored into the protocol (Siegmund et al., 1997; Brault et al., 2000). Although using only a single automobile seat reduced the external validity of the current study, there was nothing readily identifiable regarding the current experimental arrangement that would render the results observed in these experiments invalid had another seat been used.

The question of what type of perturbation pulse to use was more difficult to answer. An incomplete list of collision pulse descriptors includes the following: pulse shape, average acceleration, peak acceleration, duration, speed change, displacement, jerk and time to peak acceleration. As shown by the results of Experiments 2C and 2D (Chapters 5 and 6), at least some of these pulse descriptors affect the muscle and kinematic responses. The overriding criterion applied in

selecting the perturbations to be used in these experiments was subject safety. This was particularly important for the deceived subjects, since being unprepared for a collision has been identified as a risk factor for whiplash injury (Sturzenegger et al., 1994, 1995) and this level of subject awareness had not previously been studied. A second requirement of the perturbation was that it evoke a consistent neck muscle response in all subjects even after multiple sequential exposures. Based on pilot testing, a pulse with an average acceleration ($\bar{a}$) of 0.90 g, a velocity change (Δv) of 0.5 m/s, and a duration (Δt) of 60 ms was selected. This pulse intensity was below the lowest intensity pulse reported to have generated transient symptoms ($\bar{a}$=0.84 g, Δv=1.1 m/s, Δt=135 ms; Brault et al., 1998) and slightly larger than that used in previous seated perturbation studies (fixed displacement of 80 mm, v=0.36 m/s, Δt=240 ms; Forssberg and Hirschfeld, 1994).

The results of the four perturbation experiments contained in this thesis clearly showed that a reflex response of the SCM muscle was consistently evoked in all subjects over as many as 84 sequential perturbations. PARA muscle activation also occurred consistently, although after habituation the amplitude of the response in these muscles was small. The onset latencies of both the SCM and PARA muscles were similar to those observed in previous whiplash experiments (Table 1.1), and suggested that a similar reflex activation of the neck muscles was being evoked in the current experiments. Moreover, Experiment 2B (Chapter 3) showed that overall the reflex muscle response in surprised subjects, who represented unprepared occupants in real collisions, was similar, though delayed, to the reflex muscle response in alerted and unalerted subjects, who represented the two extreme awareness conditions used in previous whiplash experiments. The overall similarity of these muscle responses suggested that the reflex response observed in the current experiments were similar to those observed both in real whiplash collisions and in laboratory experiments using human subjects to study whiplash injury.

The overall kinematics produced by the current perturbations were also similar in pattern, but different in magnitude, to those observed in previous whiplash experiments (Severy et al., 1955; Mertz and Patrick, 1967; Gutierrez, 1978; McConnell et al., 1993; Geigl et al., 1994; Matsushita et al., 1994; Szabo et al., 1994; McConnell et al., 1995; Ono and Kanno, 1996; Szabo and Welcher, 1996; Castro et al., 1997; Ono et al., 1997; Siegmund et al., 1997; Davidsson et al., 1998; Pope et al., 1998; van den Kroonenberg et al., 1998). Moreover, the kinematic responses observed between the surprised and aware (alerted and unalerted) subjects in Experiment 2A (Chapter 3) were similar in overall pattern, though different in their relative amplitudes. As with the muscle response, the pattern of movement generated by the current perturbations was likely similar to that present in both real whiplash collisions and in laboratory experiments using human subjects to study whiplash injury. Therefore, from the perspective of both the reflex muscle response and the pattern of the induced

kinematic response, the rapid horizontal translation used to perturb subjects in these experiment appeared to be externally valid.

The external validity of these experiments therefore boiled down to a question of perturbation magnitude: were the differences observed at the perturbation intensities used in the current study applicable to the larger perturbations used in many whiplash experiments? Experiments 2C and 2D (Chapters 5 and 6) showed a similar gradation in both the amplitude of the muscle response and the amplitude of the kinematic response with perturbation intensity. Although it cannot be inferred from the current data that this simultaneous gradation of muscle and kinematics will remain intact at higher collision severities, a similar gradation between muscle and kinematic responses has also been observed in collisions conducted at velocity changes of 1.1 and 2.2 m/s (Siegmund et al., 1997; Brault et al., 2000). This evidence of a similar muscle and kinematic gradation at higher perturbation intensities than used in the current experiments suggested that the awareness-related, habituation-related, and stimulus-intensity-related phenomena observed in the current study were applicable up to a velocity change of 2.2 m/s.

Two factors ultimately limit the applicability of the current findings. First, as noted in Experiment 2C, at some increased intensity, the perturbation will be perceived as noxious and sensitization, rather than habituation, may develop. There are currently no data from which to predict at what perturbation intensity this transition might occur. Second, the force that can be produced by a muscle has an upper limit. Data from collisions at a velocity change of 2.2 m/s produced an integrated EMG amplitude in the SCM muscle that was about 50 percent of the amplitude produced during a maximum voluntary contraction (MVC) (Brault et al., 2000). These data suggested that gradation of the muscle response extended to collision severities greater than Δv=2.2 m/s, however, the perturbation intensity at which the reflex muscle contraction saturates has not been determined.

Based on the above discussion, there appeared to be no reason to conclude that the results observed in the experiments contained in this thesis would not be applicable to both real whiplash collisions and laboratory experiments using human subjects to study whiplash injury.

7.2 Comparison of Awareness and Stimulus Intensity Effects

Differences in the amplitude of some peak kinematic responses between surprised and aware subjects in Experiment 2A (Chapter 3) were similar or smaller than differences in the amplitude of peak kinematic responses observed between the different perturbation intensities used in Experiment 2D (Chapter 6). This raised the question of whether the different kinematics observed in surprised individuals could be addressed by scaling perturbation intensity.

In Experiment 2A, the peak retraction (r_x) observed in surprised female subjects was

25 percent larger than the peak retraction observed in both groups of aware female subjects. Data from Experiment 2D showed that this increase in retraction could be produced by *increasing* the intensity of the perturbation. Experiment 2A also showed that a corresponding 30 percent reduction in horizontal mastoid acceleration accompanied the increased retraction in surprised female subjects. Data from Experiment 2D showed that this decrease in mastoid acceleration could be produced by *decreasing* the intensity of the perturbation. Both kinematic changes observed in surprised female subjects could not be simultaneously produced by a single change in perturbation intensity. This comparison showed that the kinematic differences between surprised and aware subjects could not be accommodated by varying perturbation intensity and highlighted the complex interplay of different kinematic variables that differentiated the responses of surprised and aware subjects.

The multiple perturbations used in Experiment 2B (Chapter 4) produced an attenuation of the reflex muscle response. This attenuation was the result of a neural phenomenon known as habituation (Harris, 1943; Kandel, 1991), and did not produce an associated attenuation in all kinematic response variables. During habituation of the muscle response, the peak amplitude of some kinematic variables increased by up to 21 percent, whereas the peak amplitude of other kinematic variables decreased by up to 29 percent. When stimulus intensity was varied in Experiment 2D (Chapter 6), the amplitude of all kinematic peaks increased or decreased together. As a result, the kinematic changes that developed during habituation of the muscle response could also not be accommodated by varying perturbation intensity.

7.3 Neurophysiological Aspects of the Neck Muscle Reflex

As described above, the attenuation of the neck muscle response that occurred with habituation resulted in a non-uniform pattern of changes in the peak amplitude of various kinematic variables. Some variables decreased in magnitude, whereas others either increased or remained the same. The divergent effects of habituation on the amplitude of the peak kinematic responses underscored the complexity of the head and neck musculoskeletal system and indicated that habituation in complex musculoskeletal systems may not be reliably determined by examining only the response of isolated kinematic variables.

The rapid habituation of reflex muscle responses to sequentially-presented postural perturbations has led some researchers to equate the large initial response to a startle reflex (Hansen et al., 1988; Allum et al., 1992; Timmann and Horak, 1997). The startle reflex is a whole-body response that produces simultaneous activation of antagonist muscles, presumably to momentarily stiffen the body for protection or to better manage a blow (Yeomans and Frankland, 1996; Koch, 1999). It can be triggered with either acoustic, tactile or visual stimuli (Landis and Hunt, 1939) and

habituates rapidly to repeated exposures (Davis, 1984). In the current studies, both rapid habituation and co-activation of antagonist neck muscles were observed during a subject's first few perturbations. These similarities warranted a closer comparison of the startle-induced and perturbation-induced responses in order to determine whether they were indeed the same response.

The mean onset latencies of the neck muscles to the acoustically startling stimulus used in Experiment 1 (Chapter 2) were 52 ms for the SCM muscles and 60 ms for the PARA muscles. In Experiment 2A (Chapter 3), the mean onset latencies observed in all aware subjects were 72 ms for the SCM muscles and 78 ms for the PARA muscles. The delay in onset latencies between the acoustic and perturbation stimuli was about 18 to 20 ms for both muscles, and within each stimulus, the delay between SCM and PARA activation was about 8 ms. The delay between the acoustic and perturbation stimuli was remarkably similar to the 12 to 20 ms (calculated in Chapter 6) required for afferent information to ascend the spinal cord from the pelvis to the brainstem. Overall, the similar patterns of cocontraction, habituation, activation sequence, and overall activation times (if adjusted for a difference in mechanoreceptor location) between the acoustically-evoked startle reflex and the perturbation-evoked postural reflex suggested that the latter was either a startle response or some response indistinguishable from a startle response.

Unlike the neck muscles, the onset latencies of the orbicularis oculi (OO) muscles were delayed by 30 to 35 ms in response to the perturbation stimulus compared to the acoustic stimulus. This longer delay suggested that a different reflex pathway might be responsible for triggering the eye blink in response to the two stimuli. Brown et al. (1991b) observed that the onset latency of the OO muscles was the same whether evoked as part of a generalized startle reflex or evoked in isolation after the startle reflex had habituated. Based on this observation, these authors proposed a distinction between the auditory blink reflex, which did not readily habituate, and the startle response, which habituated rapidly. They proposed that the early OO activity observed in the acoustic startle reflex was actually the auditory blink reflex and that only later OO activity was related to the startle response. The perturbations used in the current study did not have a substantial acoustic component and were therefore unlikely to trigger the auditory blink reflex. As a result, the OO activity observed in the current data may be related entirely to the startle reflex independent of the auditory blink reflex. This interpretation of the data both explained the longer delay in activation of the OO muscles in the current experiments and provided support for the proposal advanced by Brown et al. (1991b).

7.4 Implications for Whiplash Injury Research

The experiments reported here provided new information regarding two aspects of whiplash injury: the external validity of human subject experiments and the aetiology of whiplash injury.

7.4.1 External Validity of Whiplash Experiments

Experiment 2A (Chapter 3) showed that surprised subjects responded differently to an identical perturbation than did either alerted or unalerted subjects. These differences included a larger peak retraction and lower peak horizontal mastoid acceleration in surprised female subjects, a larger PARA muscle response in surprised male subjects, and a delayed activation of both the SCM and PARA muscles in surprised subjects of both genders. Experiment 2B (Chapter 4) showed that multiple exposures to a perturbation resulted in habituation of the neck muscle response and complex changes in the kinematic response. The amplitude of the muscle response was significantly attenuated by the second trial and significant changes in some of the kinematic variables were also present by the second trial. These findings indicated that the responses of event-aware subjects, and in particular the responses of subjects presented with more than one perturbation, were different than the first response of surprised subjects. Since all of the previous experiments using human subjects to study the biomechanics of whiplash injury used subject-warning protocols that fell somewhere between the alerted and unalerted conditions used in Experiment 2A, the external validity of these previously conducted studies may be lower than previously thought.

Despite a reduced external validity, the combined results of the experiments conducted for this thesis did not rule out a role for human subject testing in the study of whiplash injury. The gross motion and muscle response of aware subjects exposed to a single perturbation might still provide some insight into the biomechanics of whiplash injury. For instance, some gender-based differences in the kinematic responses were present in both the surprised and aware subjects and could potentially be studied under aware conditions. In addition, the stabilized responses of subjects who have habituated to a perturbation might still provide useful information in parametric studies designed to measure the relative effect of some variable or intervention on subject response. Indeed, the latter two experiments in this thesis were based on this premise. The results of the current experiments did show, however, that human subject experiments need to be designed to account for subject awareness and habituation and the results of these experiments need to be interpreted carefully in the context of their reduced external validity.

7.4.2 The Aetiology of Whiplash Injury

Perhaps the most important outcome of these experiments was the knowledge that subjects responded differently to an unexpected perturbation than to a perturbation they knew was imminent. This finding paralleled epidemiological evidence which has shown that being unprepared for a collision increased the risk of whiplash injury (Sturzenegger et al., 1994, 1995). As a result, differences between the biomechanical response of the surprised and aware subjects might provide

some insight into factors that affect the aetiology of whiplash injury. In this regard, the increased retraction and decreased mastoid acceleration observed in surprised female subjects may be particularly important. These larger retraction motions were not associated with significantly larger head extension angles, a finding that suggested surprised subjects attempted to keep their head upright at the expense of increased retraction. A larger retraction would cause larger strains in the ligamentous tissues of the cervical spine. If the increased retraction observed in the surprised female subjects were distributed evenly between the joints of the cervical spine, about 1 mm of additional translation would occur between each pair of cervical vertebra. Based on a linear fit to data presented by Siegmund et al. (2000b), this additional motion would increase the maximum principal strain in the facet joint capsular ligaments by about 0.03. On average, this increase in strain was about 9 percent of the 0.35 ± 0.21 strain reported by these authors to cause sub-catastrophic failure of these ligaments. When the entire range of sub-catastrophic failure strains (0.11 to 0.80) reported by these authors was considered, the 0.03 increase in strain implied by the larger retraction motions amounted to between 4 and 27 percent of the sub-catastrophic failure strains. This comparison suggested that, in some individuals, the larger retraction observed here could result in relatively large increases in capsular ligament strain and increased potential for injury. This suggestion is consistent with clinical evidence that has isolated the cervical facet joints as the source of pain in about half of a population with chronic whiplash pain (Lord et al., 1996). Though in need of additional investigation, the results of the current study provided a possible biomechanical explanation for why unprepared female occupants have an increased risk of whiplash injury.

In Experiment 2A, a mechanism was proposed to explain why a larger retraction and lower mastoid acceleration was isolated to female subjects. This mechanism relied on a difference in how male and female subjects scaled the amplitude of their muscle response when perturbed unexpectedly. Possibly due to a large variance in the EMG data, this component of the mechanism did not reach statistical significance in post-hoc analyses performed on the current data. The likelihood of such a difference could, however, be inferred from the kinematic data. The mechanism underlying such a gender difference was not readily apparent. It might be that males have learned to scale their neck muscle response to unexpected stimuli through increased exposure to these types of stimuli, however, such an explanation is speculative. Further work is needed to explore these potential gender differences in muscle activation during unexpected perturbations and whether these differences contribute to the increased incidence of whiplash injury in females.

7.5 Conclusions

The results of the current studies indicated that differences in prior knowledge related to the

presence of a whiplash-like perturbation (event awareness) had a larger effect on the resulting muscle and kinematic responses than did prior knowledge related to the timing of the perturbation (temporal awareness). Prior knowledge of the perturbation intensity (amplitude awareness) did not affect the muscle or kinematic responses over the range of perturbation intensities used in these experiments. Multiple exposures to a whiplash-like perturbation resulted in habituation of the reflex muscle response. Kinematic changes related to this habituation produced both increases in the amplitude of some kinematic parameters and decreases in the amplitude of other kinematic parameters. Together, these results called into question the external validity of many previous experiments using human subjects to study the biomechanics of whiplash injury. Future human subject testing should consider the effects of both subject awareness and habituation, and incorporate these effects into both the design of their experimental protocol and the interpretation of their results.

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APPENDIX A DETECTION OF MOVEMENT ONSET

A.1 Introduction

The time at which movement begins in response to a stimulus is commonly determined from transducer data. Movement onset times estimated in this manner have previously been shown to be affected by the type of task, the test apparatus used, the specific transducer's properties and the type of signal processing performed before data acquisition (Corcos et al., 1992). These authors demonstrated that earlier and more accurate onset times could be determined if the movement task had a rapid onset, if the transducer was applied directly to the subject rather than to an object moved by the subject, if the transducer had a low detection threshold, and if a high gain was used to amplify the transducer's signal before data acquisition. Even after an experimental setup has been optimized as described above, a point in time within the data acquired from the transducer must still be identified as the onset of movement. The task of identifying the transition from a pre-movement signal containing only noise to a post-movement signal containing both movement and noise is not trivial. Often, a threshold level is selected based on some multiple of the pre-stimulus noise and then movement onset is considered to have occurred when the transducer signal exceeds this threshold. Unfortunately, this method produces an estimate of the onset time which is delayed relative to the true movement onset, i.e., movement must have occurred prior to the threshold being reached in order for the threshold to be reached. The goal of this analysis was to develop an algorithm that minimized this bias and thereby provided a better estimate of the actual movement onset time.

A.2 Mathematical Development

Assume that a measured signal $m(t)$ consists of noise $n(t)$ prior to the onset t_0 of movement and a combination of the actual movement signal $s(t)$ and noise $n(t)$ after the onset of movement (Figure A.1). If a fixed onset threshold C is used, then for every value of C greater than the amplitude of the noise $n(t)$, the onset time t^* determined using this threshold results in a delayed estimate, or positively-biased estimate, of the actual onset time t_0. The positive bias can be minimized by selecting a threshold C that is only slightly larger than the pre-movement noise level, however, the risk of falsely detecting onset increases as C is reduced. Therefore, in practice, a balance is sought between a low threshold to minimize the bias introduced in the onset time and a high threshold to minimize falsely detecting spurious noise as the onset of movement.

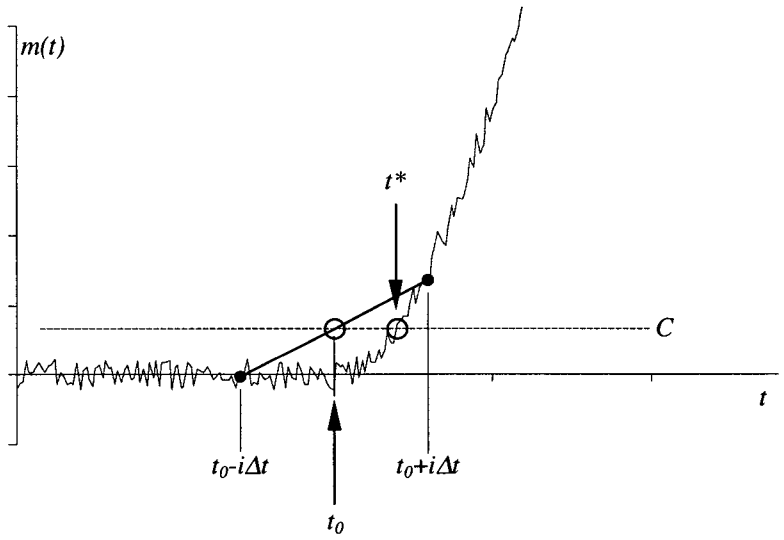

Figure A.1 Sample signal $m(t)$ and its actual onset time t_0. Using a set onset threshold C, the estimated onset time t^* is a biased estimate of the actual onset time t_0.

An alternate method of achieving improved onset times is to negatively bias the signal by an amount equal to the positive bias introduced by the threshold technique, and to then use a similarly transformed threshold on this negatively-biased signal. A common method of generating a phase-led version of a signal is differentiation. The phase-leading derivative $\dot{m}_i(t)$ leads the actual signal $m(t)$ by 90 degrees and therefore reaches threshold sooner. Unfortunately, differentiation also increases noise, a side-effect that eliminates the possibility of successive differentiation as a method of producing increasingly better estimates of movement onset times. This side-effect of differentiation can be overcome by only differentiating the measured signal once using a carefully selected window across which the derivative is computed by finite differences. The width of the finite-differences window determines the amount of negative bias introduced into $\dot{m}_i(t)$ at a given frequency. Therefore, the width of the differentiation window can be tuned to produce the negative bias needed to offset the positive bias introduced by using a threshold.

Let the width of the moving window used in the finite differentiation be equal to $2i\Delta t$, where i is half the number of discrete sampling intervals spanned by the window, and Δt is the sampling period used to acquire the data (Figure A.1). The derivative $\dot{m}_i(t)$ for a given half window width is then be calculated as follows:

$$\dot{m}_i(t) = \frac{m(t + i\Delta t) - m(t - i\Delta t)}{2i\Delta t} \qquad (1)$$

At the actual onset time, $t = t_0$, the value of this derivative is

$$\dot{m}_i(t_0) = \frac{m(t_0 + i\Delta t) - m(t_0 - i\Delta t)}{2i\Delta t} \tag{2}$$

From the pre-stimulus data, or data known to precede movement onset, the maximum value of $\dot{m}_i(t)$ within the noise-only portion of the signal can be determined for a given window width. If the maximum amplitude of the pre-stimulus noise is $n_{max} = \max |n(t)|$, then the maximum possible value of $\dot{m}_i^*(t)$ is given by the following equation:

$$\dot{m}_i^*(t) = \frac{(n_{max} - (-n_{max}))}{2i\Delta t}, \text{ or}$$
$$\dot{m}_i^*(t) = \frac{n_{max}}{i\Delta t} \tag{3}$$

A multiplier $c > 1$ can then used to define a threshold value $c\dot{m}_i^*(t)$, above which onset is deemed to have occurred. Equating $\dot{m}_i(t_0)$ in Equation 2 to $c\dot{m}_i^*(t)$ yields an equation in which the half window size i is defined in terms of the multiplier c, the sampling interval Δt, and the maximum level of noise in the pre-movement data n_{max}.

$$\dot{m}_i(t_0) = c\dot{m}_i^*(t) \tag{4}$$

Substituting Equations 2 and 3 into Equation 4 yields,

$$\frac{m(t_0 + i\Delta t) - m(t_0 - i\Delta t)}{2i\Delta t} = \frac{cn_{max}}{i\Delta t} \tag{5}$$

and simplification yields,

$$m(t_0 + i\Delta t) - m(t_0 - i\Delta t) = 2cn_{max} \tag{6}$$

Since on average, $m(t_0 - i\Delta t)$ is equal to zero, Equation 6 can be further simplified to,

$$m(t_0 + i\Delta t) = 2cn_{max} \tag{7}$$

In order to solve this equation for the half window width i, a mathematical model of the actual response signal must be used. This artificial representation of the signal $s(t)$ can then be used to

solve for i. Any increasing function $M(t)$ can be used and need not be continuous at t_0; it must however adequately model $s(t)$ in the region of $i\Delta t$ seconds after its onset. For purposes of this derivation, a partial sine wave of frequency f_a was used to model the onset of movement.

$$M(t) = A\left[1 + \sin\left(\omega(t - t_0) - \frac{\pi}{2}\right)\right] \qquad \text{for } t \geq t_0 \tag{8}$$

Substituting Equation 8 into 7 yields,

$$A\left[1 + \sin\left(\omega[(t_0 + i\Delta t) - t_0] - \frac{\pi}{2}\right)\right] = 2cn_{\max} \tag{9}$$

and solving for the half window width $i\Delta t$ yields,

$$i\Delta t = \frac{1}{2\pi f_a}\left[\sin^{-1}\left(\frac{2cn_{\max}}{A} - 1\right) + \frac{\pi}{2}\right] \tag{10}$$

Equation 10 demonstrates that the half window width is a function of the pre-movement noise in a signal ($n_{\max}$), the multiplier (c) chosen as the threshold value, and both the amplitude (A) and frequency (f_a) of the signal used to model the actual movement. Based on Equation 10, and an threshold of c=1.5, a relationship between the half window size in milliseconds and the ratio of peak noise in the measured signal to peak amplitude of the model signal was constructed (Figure A.2). This figure shows the optimum half window size needed to eliminate the bias introduced by using a fixed threshold value of c=1.5 for signals of varying frequencies. Windows shorter than that recommended in Figure A.2 yield onset times that are delayed relative to the actual onset of the signal. Longer windows yield onset times that are earlier than the actual onset of the signal. The shallow slope of the various frequency lines indicated that the optimal size of the half window was relatively insensitive to inexact estimates of either the noise ratio or the frequency of the sine wave used to model the actual signal.

A.3 Performance of the Algorithm

Because the algorithm incorporates the maximum values of a noise signal over a finite period of time, this value is necessarily an estimate of the actual maximum level of the noise that exists in the signal after time t_0. To evaluate the possible error introduced by this estimate of the maximum noise, a Monte Carlo simulation was performed. For this simulation, a signal described by Equation 8

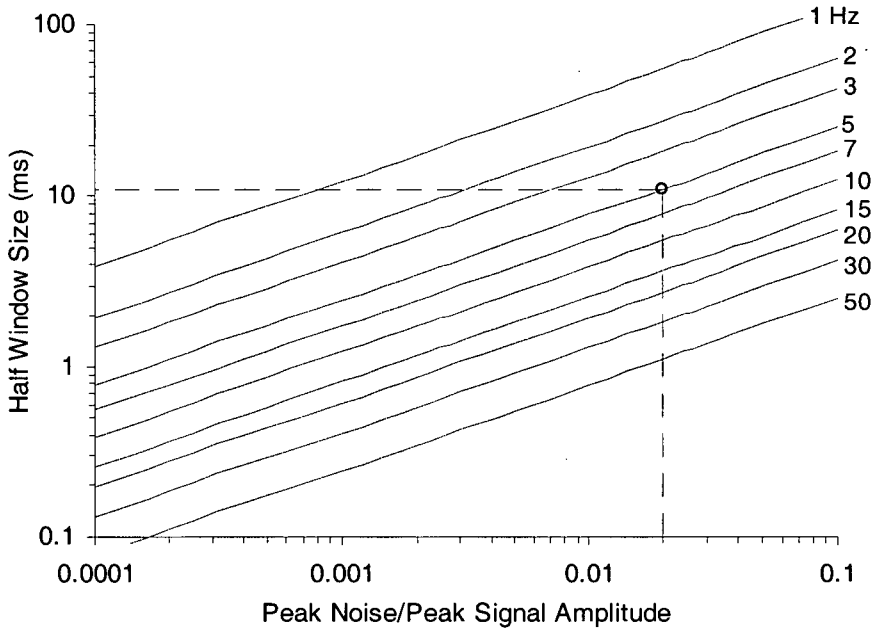

Figure A.2 Optimal half window size as a function of the peak noise to peak amplitude ratio and frequency for signals with onsets that can be modeled with a sinusoidal. The dashed line shows that for a 5 Hz signal with 2% noise, the half window size would be about 10 ms. Note logarithmic scales.

(A=1.0 and $\omega=2\pi f_a$, where f_a=5 Hz) and shown in Figure A.1 was used. This signal was similar to the torso and head accelerometer data observed in the studies performed in this thesis. For the simulation, onset occurred at 100 ms and uniformly distributed noise between ±2% of the peak amplitude of the signal was added to the signal (SNR~28 dB). A threshold multiple of c=1.5 was used. The dashed line in Figure A.2 shows that the appropriate half window size for this combination of parameters was 11 ms. A sampling rate of 2 kHz was simulated, thereby providing a resolution of 0.5 ms. All of the values selected for this simulation, aside from the onset time, were similar to values used for determining the onset time of the torso and head acceleration in the studies contained in this thesis.

Onset times were estimated by computing $\dot{m}_i(t)$ using a half window of 11 ms and then determining when this value exceeded the threshold given in Equation 5. For comparison, an estimate of movement onset was also computed using a threshold of 1.5×noise applied to the actual signal. The results of a simulation with 10 000 iterations are shown in Table A.1. These results showed that a threshold technique applied to the actual signal estimated an onset time that was delayed 7.3 ± 1.1 ms relative to the actual onset of the signal at 100 ms. The technique derived above estimated an onset time that was 0.5 ± 1.2 ms ahead of the actual onset, or within one sampling interval of the actual movement onset.

Table A.1 Mean (S.D.) of onset times for both algorithms from the Monte Carlo simulation.

	Estimated onset time (ms)
Threshold applied to actual signal m(t)	107.3 (1.1)
Derived algorithm	99.5 (1.2)
Difference	7.8 (1.7)

A sensitivity analysis was also conducted for the two model-signal parameters A and f_a. For this simulation, both parameters that uniformly varied between ±20 percent of the values used in the previous simulation. This sensitivity analysis revealed that 10 percent of the variance in the onset time was related to the frequency estimate f_a and 90 percent of the variance was related to the amplitude estimate A. Despite the introduction of relatively large errors in the estimates of the model-signal parameters, an onset time of 99.2 ± 2.7 ms was predicted by the algorithm during this simulation. The mean onset time was not considerably different from that estimated when only noise was varied. Despite a consistent mean, however, variations in A and f_a increased the standard deviation of the estimate by about 60 percent. These results suggested that relatively large errors in the model-parameter estimates reduced the confidence of the estimated onset time, however, if numerous trials were used to calculate the mean onset time, the results of the derived algorithm were considerably closer to the actual onset than the results obtained using the threshold technique on the actual signal.

A.4 Summary

Estimating movement onset times by applying a fixed threshold to transducer data produces a positively-biased (delayed) estimate of the actual movement onset time. This bias can be minimized by transforming the data into a phase-advanced form through differentiation. If the appropriate half window size is used to calculate the derivative by finite differences, the negative bias introduced by the differentiation process can be made to offset the positive bias introduced by using a threshold. The resulting movement onset time is a better estimate of the actual movement onset time than achieved using a similar threshold on the actual signal.

APPENDIX B TRIAL DATA FOR HABITUATION EXPERIMENT

The following tables list the trial-by-trial means (S.D.) for all of the dependent variables over the eleven sequential trials of the habituation experiment documented in Chapter 4. The data were divided based on gender (female, male) and level of temporal awareness (alerted, unalerted). The number of subjects (N) in each group is given below each table and group means and group standard deviations are given at the right of each table.

Table B.1 Mastoid process, X (mm)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	7 (19)	21 (18)	11 (16)	28 (12)	16 (18)
2	8 (18)	21 (18)	11 (17)	30 (16)	17 (19)
3	10 (18)	22 (15)	13 (16)	30 (18)	18 (18)
4	7 (19)	20 (17)	11 (16)	29 (20)	16 (19)
5	7 (18)	19 (16)	10 (13)	27 (19)	16 (18)
6	8 (20)	18 (17)	11 (14)	28 (19)	16 (19)
7	7 (20)	18 (16)	10 (13)	28 (19)	15 (19)
8	6 (18)	16 (16)	7 (13)	26 (19)	14 (18)
9	7 (18)	16 (17)	8 (15)	27 (21)	14 (19)
10	6 (18)	16 (16)	9 (14)	26 (22)	14 (19)
11	7 (17)	16 (16)	9 (15)	24 (19)	14 (18)
N	12	10	10	10	42

Table B.2 Mastoid process, Z (mm)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	-641 (35)	-672 (25)	-642 (23)	-661 (36)	-653 (32)
2	-637 (35)	-670 (26)	-640 (22)	-659 (36)	-651 (33)
3	-637 (36)	-669 (26)	-640 (22)	-659 (36)	-650 (33)
4	-637 (36)	-668 (26)	-640 (23)	-659 (36)	-650 (33)
5	-637 (36)	-668 (25)	-640 (22)	-659 (36)	-650 (33)
6	-637 (36)	-668 (25)	-640 (22)	-658 (36)	-650 (32)
7	-637 (36)	-668 (25)	-640 (22)	-658 (36)	-650 (32)
8	-637 (36)	-668 (26)	-639 (23)	-658 (36)	-650 (33)
9	-637 (36)	-668 (26)	-639 (22)	-658 (36)	-650 (33)
10	-637 (36)	-668 (26)	-639 (22)	-658 (36)	-650 (33)
11	-637 (36)	-667 (25)	-640 (21)	-658 (35)	-650 (32)
N	12	10	10	10	42

Table B.3 Manubrium-C7 midpoint, X (mm)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	21 (11)	28 (15)	21 (14)	38 (6)	27 (14)
2	20 (10)	28 (17)	21 (14)	40 (9)	28 (15)
3	19 (10)	29 (17)	21 (14)	39 (10)	27 (15)
4	16 (11)	28 (18)	20 (12)	39 (10)	26 (15)
5	16 (10)	28 (16)	19 (11)	38 (10)	25 (14)
6	16 (12)	26 (16)	18 (11)	38 (10)	25 (15)
7	16 (12)	26 (15)	18 (11)	38 (11)	25 (15)
8	15 (10)	25 (16)	17 (11)	37 (11)	24 (15)
9	16 (10)	25 (17)	17 (11)	37 (12)	24 (15)
10	16 (10)	25 (17)	17 (11)	37 (12)	24 (15)
11	16 (9)	26 (17)	18 (11)	36 (13)	24 (15)
N	9	9	10	10	38

Table B.4 Manubrium-C7 midpoint, Z (mm)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	-559 (28)	-584 (25)	-552 (19)	-572 (35)	-566 (29)
2	-557 (27)	-583 (25)	-550 (17)	-570 (35)	-565 (29)
3	-557 (28)	-582 (24)	-550 (17)	-570 (34)	-565 (28)
4	-557 (29)	-582 (24)	-550 (17)	-570 (34)	-565 (29)
5	-558 (29)	-582 (24)	-550 (18)	-570 (35)	-565 (29)
6	-558 (29)	-582 (25)	-549 (17)	-569 (34)	-564 (29)
7	-557 (29)	-582 (25)	-549 (16)	-570 (34)	-564 (29)
8	-557 (29)	-582 (24)	-549 (17)	-570 (35)	-564 (29)
9	-557 (29)	-582 (24)	-549 (17)	-570 (34)	-564 (29)
10	-556 (29)	-582 (24)	-549 (17)	-569 (35)	-564 (29)
11	-557 (29)	-582 (24)	-550 (16)	-569 (35)	-564 (29)
N	9	9	10	10	38

Table B.5 Initial head angle (deg)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	7.9 (3.4)	9.6 (4.8)	7.1 (1.9)	9.4 (4.0)	8.5 (3.7)
2	8.8 (3.4)	9.1 (4.7)	7.2 (1.7)	10.0 (5.0)	8.7 (3.9)
3	8.9 (3.6)	9.1 (5.6)	6.9 (2.4)	9.1 (6.2)	8.5 (4.5)
4	8.1 (3.1)	9.4 (5.5)	7.0 (3.0)	8.8 (7.8)	8.3 (5.0)
5	9.6 (2.6)	9.6 (5.4)	7.9 (3.4)	9.3 (6.2)	9.1 (4.4)
6	8.4 (3.5)	10.2 (5.7)	8.1 (3.8)	9.0 (7.1)	8.9 (5.0)
7	8.9 (3.1)	10.0 (5.2)	8.9 (3.3)	8.4 (7.7)	9.0 (4.9)
8	9.0 (3.9)	10.7 (4.9)	9.1 (4.0)	8.7 (7.4)	9.4 (5.1)
9	8.6 (4.2)	10.7 (5.7)	8.3 (4.6)	8.3 (7.5)	8.9 (5.4)
10	9.5 (3.7)	10.5 (6.0)	8.8 (4.6)	9.8 (7.3)	9.6 (5.3)
11	9.3 (3.1)	11.5 (6.0)	8.3 (4.9)	9.4 (6.8)	9.6 (5.2)
N	12	10	11	10	43

Table B.6 Initial torso angle (deg)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	-17.6 (4.0)	-17.2 (5.3)	-15.9 (4.1)	-15.3 (3.3)	-16.5 (4.1)
2	-17.2 (4.3)	-17.0 (5.3)	-16.2 (4.9)	-15.8 (3.5)	-16.5 (4.4)
3	-17.2 (4.2)	-17.2 (4.5)	-16.2 (4.7)	-15.7 (3.4)	-16.6 (4.1)
4	-16.5 (4.2)	-16.9 (4.6)	-16.1 (4.9)	-15.7 (4.1)	-16.3 (4.3)
5	-16.1 (4.3)	-17.0 (4.9)	-15.5 (4.4)	-15.4 (3.6)	-16.0 (4.2)
6	-15.9 (4.5)	-16.6 (5.1)	-15.8 (5.2)	-15.8 (3.8)	-16.0 (4.5)
7	-16.5 (4.9)	-16.7 (5.1)	-15.5 (5.1)	-15.5 (3.6)	-16.0 (4.5)
8	-15.9 (4.6)	-16.3 (4.7)	-15.3 (5.0)	-15.1 (3.6)	-15.6 (4.3)
9	-16.4 (4.2)	-16.3 (5.0)	-15.3 (5.1)	-15.3 (3.6)	-15.8 (4.3)
10	-16.4 (4.2)	-15.9 (4.9)	-15.8 (5.3)	-15.1 (3.8)	-15.8 (4.4)
11	-16.2 (4.0)	-16.1 (5.0)	-15.3 (5.8)	-14.9 (3.9)	-15.6 (4.6)
N	9	9	10	10	38

Table B.7 Forehead acceleration, x1 (g)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	1.11 (0.17)	1.12 (0.33)	1.20 (0.22)	1.30 (0.37)	1.18 (0.28)
2	1.09 (0.15)	1.05 (0.26)	1.08 (0.22)	1.18 (0.36)	1.10 (0.25)
3	1.03 (0.12)	0.92 (0.24)	0.94 (0.17)	0.93 (0.21)	0.96 (0.19)
4	0.98 (0.11)	0.95 (0.22)	0.89 (0.18)	0.96 (0.26)	0.95 (0.19)
5	0.93 (0.15)	0.91 (0.20)	0.90 (0.17)	0.92 (0.25)	0.92 (0.18)
6	0.90 (0.13)	0.95 (0.27)	0.88 (0.16)	0.94 (0.24)	0.91 (0.20)
7	0.90 (0.14)	0.88 (0.25)	0.86 (0.15)	0.92 (0.17)	0.89 (0.17)
8	0.85 (0.16)	0.88 (0.25)	0.85 (0.14)	0.85 (0.22)	0.86 (0.19)
9	0.86 (0.14)	0.88 (0.26)	0.84 (0.16)	0.90 (0.24)	0.87 (0.19)
10	0.84 (0.13)	0.89 (0.24)	0.86 (0.16)	0.89 (0.21)	0.87 (0.18)
11	0.85 (0.14)	0.87 (0.26)	0.78 (0.10)	0.90 (0.21)	0.85 (0.18)
N	12	9	8	9	38

Table B.8 Forehead acceleration, x1 (ms)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	171 (13)	165 (15)	169 (14)	167 (9)	168 (12)
2	165 (12)	162 (20)	172 (19)	163 (8)	165 (15)
3	166 (14)	169 (29)	172 (18)	166 (13)	168 (18)
4	165 (13)	159 (12)	177 (24)	168 (14)	167 (16)
5	169 (15)	162 (14)	173 (21)	167 (12)	168 (15)
6	171 (16)	165 (23)	175 (21)	168 (15)	169 (18)
7	173 (13)	171 (32)	178 (19)	169 (16)	173 (20)
8	172 (14)	169 (24)	176 (18)	178 (21)	174 (19)
9	170 (16)	169 (28)	179 (18)	178 (24)	174 (21)
10	172 (17)	161 (16)	178 (20)	172 (14)	170 (17)
11	171 (18)	165 (18)	181 (16)	172 (18)	172 (18)
N	12	9	8	9	38

Table B.9 Forehead acceleration, z1 (g)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	-0.64 (0.21)	-0.74 (0.13)	-0.60 (0.13)	-0.70 (0.22)	-0.67 (0.18)
2	-0.60 (0.08)	-0.76 (0.12)	-0.63 (0.14)	-0.70 (0.20)	-0.67 (0.15)
3	-0.59 (0.10)	-0.79 (0.13)	-0.63 (0.14)	-0.72 (0.16)	-0.68 (0.15)
4	-0.65 (0.11)	-0.78 (0.13)	-0.68 (0.13)	-0.75 (0.31)	-0.71 (0.18)
5	-0.63 (0.13)	-0.81 (0.13)	-0.67 (0.13)	-0.74 (0.25)	-0.71 (0.17)
6	-0.67 (0.15)	-0.80 (0.17)	-0.66 (0.11)	-0.80 (0.28)	-0.73 (0.19)
7	-0.69 (0.17)	-0.82 (0.16)	-0.68 (0.17)	-0.75 (0.20)	-0.73 (0.18)
8	-0.66 (0.15)	-0.78 (0.13)	-0.63 (0.12)	-0.78 (0.26)	-0.71 (0.18)
9	-0.69 (0.18)	-0.80 (0.15)	-0.67 (0.09)	-0.81 (0.31)	-0.74 (0.20)
10	-0.71 (0.17)	-0.81 (0.14)	-0.67 (0.13)	-0.74 (0.24)	-0.73 (0.18)
11	-0.68 (0.16)	-0.78 (0.18)	-0.69 (0.17)	-0.79 (0.29)	-0.73 (0.20)
N	12	9	8	9	38

Table B.10 Forehead acceleration, z1 (ms)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	93 (23)	91 (9)	88 (10)	88 (11)	91 (15)
2	94 (19)	95 (6)	90 (10)	88 (13)	92 (14)
3	86 (16)	94 (10)	89 (11)	92 (13)	90 (13)
4	89 (16)	91 (9)	94 (12)	91 (13)	91 (13)
5	90 (17)	94 (8)	93 (11)	88 (14)	91 (13)
6	92 (16)	93 (7)	93 (9)	94 (12)	93 (11)
7	91 (15)	96 (7)	101 (12)	89 (16)	94 (13)
8	92 (14)	96 (8)	91 (12)	92 (16)	93 (12)
9	92 (13)	96 (8)	96 (12)	95 (15)	94 (12)
10	92 (14)	96 (8)	96 (12)	92 (16)	94 (12)
11	94 (13)	96 (8)	91 (12)	94 (13)	94 (12)
N	12	9	8	9	38

Table B.11 Forehead acceleration, z2 (g)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	1.51 (0.41)	1.50 (0.38)	1.63 (0.62)	1.64 (0.25)	1.56 (0.41)
2	1.54 (0.41)	1.42 (0.32)	1.59 (0.71)	1.54 (0.38)	1.52 (0.45)
3	1.49 (0.38)	1.38 (0.42)	1.59 (0.83)	1.42 (0.24)	1.47 (0.48)
4	1.51 (0.34)	1.39 (0.37)	1.49 (0.85)	1.49 (0.36)	1.47 (0.48)
5	1.41 (0.32)	1.39 (0.42)	1.51 (0.72)	1.45 (0.37)	1.44 (0.45)
6	1.43 (0.34)	1.46 (0.51)	1.50 (0.72)	1.47 (0.40)	1.46 (0.47)
7	1.54 (0.38)	1.40 (0.41)	1.60 (0.80)	1.47 (0.40)	1.50 (0.49)
8	1.50 (0.32)	1.45 (0.54)	1.59 (0.73)	1.38 (0.31)	1.48 (0.47)
9	1.44 (0.36)	1.40 (0.43)	1.58 (0.63)	1.43 (0.39)	1.46 (0.44)
10	1.53 (0.33)	1.44 (0.47)	1.68 (0.66)	1.39 (0.23)	1.51 (0.43)
11	1.42 (0.31)	1.42 (0.45)	1.59 (0.81)	1.48 (0.27)	1.47 (0.47)
N	12	9	8	9	38

Table B.12 Forehead acceleration, z2 (ms)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	169 (16)	156 (14)	170 (8)	154 (10)	163 (14)
2	169 (16)	159 (18)	168 (14)	160 (9)	164 (15)
3	167 (12)	158 (15)	167 (16)	160 (10)	163 (13)
4	164 (13)	156 (11)	168 (17)	160 (10)	162 (13)
5	163 (12)	159 (14)	167 (13)	159 (10)	162 (12)
6	161 (18)	157 (14)	177 (26)	158 (11)	163 (19)
7	162 (14)	159 (13)	166 (16)	163 (15)	162 (14)
8	163 (14)	157 (13)	168 (11)	167 (20)	163 (15)
9	167 (16)	158 (15)	176 (26)	164 (15)	166 (18)
10	166 (16)	159 (17)	175 (27)	161 (9)	165 (18)
11	164 (17)	159 (16)	175 (25)	163 (18)	165 (19)
N	12	9	8	9	38

Table B.13 Mastoid process acceleration, x2 (g)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	1.30 (0.40)	1.07 (0.19)	1.35 (0.41)	1.17 (0.27)	1.22 (0.34)
2	1.30 (0.31)	1.13 (0.19)	1.17 (0.33)	1.11 (0.19)	1.18 (0.26)
3	1.26 (0.22)	1.06 (0.14)	1.17 (0.34)	1.06 (0.10)	1.14 (0.22)
4	1.27 (0.27)	1.07 (0.17)	1.09 (0.32)	1.07 (0.20)	1.14 (0.25)
5	1.18 (0.24)	1.06 (0.18)	1.14 (0.28)	1.05 (0.15)	1.11 (0.22)
6	1.22 (0.32)	1.06 (0.21)	1.17 (0.31)	1.12 (0.14)	1.15 (0.25)
7	1.24 (0.27)	1.08 (0.20)	1.11 (0.25)	1.08 (0.14)	1.14 (0.23)
8	1.19 (0.25)	1.05 (0.12)	1.16 (0.25)	1.07 (0.17)	1.12 (0.21)
9	1.21 (0.28)	1.08 (0.18)	1.12 (0.23)	1.08 (0.18)	1.13 (0.22)
10	1.20 (0.28)	1.11 (0.15)	1.12 (0.23)	1.05 (0.16)	1.12 (0.21)
11	1.19 (0.28)	1.08 (0.17)	1.06 (0.18)	1.04 (0.15)	1.10 (0.21)
N	11	9	7	8	35

Table B.14 Mastoid process acceleration, x2 (ms)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	112 (15)	111 (9)	115 (12)	113 (9)	113 (12)
2	105 (15)	112 (9)	112 (11)	112 (8)	110 (11)
3	105 (14)	111 (8)	113 (16)	109 (8)	109 (12)
4	101 (15)	109 (8)	111 (11)	109 (10)	107 (12)
5	105 (16)	108 (8)	109 (10)	109 (7)	107 (11)
6	105 (15)	107 (7)	115 (14)	109 (9)	109 (12)
7	104 (16)	106 (5)	110 (8)	113 (11)	108 (11)
8	103 (10)	108 (8)	110 (9)	112 (7)	108 (9)
9	101 (9)	105 (11)	109 (11)	108 (10)	105 (10)
10	102 (8)	107 (8)	111 (9)	110 (10)	107 (9)
11	101 (8)	109 (9)	110 (10)	111 (11)	107 (10)
N	11	9	7	8	35

Table B.15 Mastoid process acceleration, z3 (g)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	-0.19 (0.06)	-0.18 (0.07)	-0.19 (0.10)	-0.22 (0.08)	-0.19 (0.07)
2	-0.20 (0.07)	-0.19 (0.08)	-0.20 (0.09)	-0.24 (0.07)	-0.21 (0.07)
3	-0.22 (0.08)	-0.21 (0.07)	-0.22 (0.10)	-0.25 (0.08)	-0.22 (0.08)
4	-0.21 (0.08)	-0.19 (0.06)	-0.22 (0.10)	-0.25 (0.10)	-0.21 (0.08)
5	-0.21 (0.09)	-0.20 (0.07)	-0.21 (0.09)	-0.24 (0.10)	-0.21 (0.08)
6	-0.20 (0.09)	-0.20 (0.08)	-0.22 (0.09)	-0.25 (0.10)	-0.21 (0.09)
7	-0.21 (0.10)	-0.18 (0.07)	-0.20 (0.08)	-0.25 (0.10)	-0.21 (0.09)
8	-0.20 (0.07)	-0.18 (0.08)	-0.19 (0.09)	-0.25 (0.10)	-0.20 (0.08)
9	-0.19 (0.08)	-0.19 (0.09)	-0.18 (0.09)	-0.24 (0.10)	-0.20 (0.09)
10	-0.19 (0.07)	-0.20 (0.09)	-0.18 (0.08)	-0.22 (0.12)	-0.20 (0.08)
11	-0.19 (0.08)	-0.19 (0.08)	-0.20 (0.10)	-0.22 (0.11)	-0.20 (0.09)
N	12	8	7	6	33

Table B.16 Mastoid process acceleration, z3 (ms)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	62 (4)	70 (10)	58 (9)	61 (7)	63 (9)
2	62 (5)	72 (14)	63 (12)	63 (5)	65 (10)
3	59 (6)	70 (7)	59 (9)	64 (12)	62 (9)
4	58 (4)	65 (11)	59 (9)	59 (8)	60 (8)
5	59 (6)	78 (13)	59 (6)	59 (10)	63 (12)
6	61 (8)	73 (11)	61 (10)	61 (8)	64 (10)
7	63 (6)	69 (8)	60 (9)	60 (6)	63 (8)
8	61 (4)	68 (10)	57 (8)	59 (6)	61 (8)
9	60 (5)	69 (11)	60 (14)	62 (7)	62 (10)
10	60 (5)	74 (13)	59 (12)	55 (5)	62 (11)
11	60 (6)	71 (11)	56 (11)	58 (11)	61 (11)
N	12	8	7	6	33

Table B.17 Mastoid process acceleration, z4 (g)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	0.51 (0.19)	0.36 (0.10)	0.66 (0.35)	0.55 (0.24)	0.51 (0.24)
2	0.50 (0.23)	0.33 (0.11)	0.56 (0.35)	0.41 (0.17)	0.45 (0.23)
3	0.47 (0.16)	0.29 (0.09)	0.51 (0.28)	0.36 (0.14)	0.41 (0.19)
4	0.41 (0.16)	0.32 (0.08)	0.46 (0.27)	0.37 (0.14)	0.39 (0.17)
5	0.40 (0.16)	0.32 (0.12)	0.45 (0.28)	0.42 (0.16)	0.39 (0.18)
6	0.39 (0.18)	0.27 (0.08)	0.47 (0.29)	0.37 (0.13)	0.37 (0.18)
7	0.41 (0.15)	0.29 (0.10)	0.42 (0.22)	0.35 (0.10)	0.37 (0.15)
8	0.37 (0.14)	0.28 (0.11)	0.41 (0.23)	0.33 (0.09)	0.35 (0.15)
9	0.35 (0.17)	0.29 (0.08)	0.43 (0.26)	0.34 (0.13)	0.35 (0.17)
10	0.35 (0.13)	0.30 (0.07)	0.46 (0.30)	0.33 (0.13)	0.36 (0.17)
11	0.33 (0.10)	0.27 (0.07)	0.41 (0.27)	0.34 (0.16)	0.33 (0.16)
N	11	9	7	7	34

Table B.18 Mastoid process acceleration, z4 (ms)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	125 (9)	152 (29)	124 (14)	131 (12)	133 (20)
2	121 (9)	152 (29)	125 (18)	129 (16)	132 (22)
3	117 (9)	148 (34)	126 (17)	137 (19)	131 (24)
4	118 (14)	153 (27)	127 (29)	134 (26)	133 (27)
5	122 (13)	155 (29)	123 (28)	140 (24)	135 (27)
6	126 (13)	159 (30)	132 (17)	130 (25)	137 (25)
7	125 (12)	156 (22)	129 (26)	131 (21)	135 (23)
8	122 (16)	155 (28)	127 (20)	139 (19)	135 (24)
9	118 (15)	157 (27)	126 (29)	134 (31)	133 (29)
10	122 (11)	157 (31)	136 (20)	144 (24)	139 (25)
11	123 (10)	159 (30)	126 (26)	139 (22)	136 (26)
N	11	9	7	7	34

Table B.19 Retraction rx (mm)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	-22 (3)	-20 (2)	-20 (4)	-20 (4)	-21 (3)
2	-20 (4)	-19 (4)	-21 (4)	-20 (5)	-20 (4)
3	-21 (5)	-20 (4)	-22 (3)	-23 (3)	-21 (4)
4	-21 (5)	-20 (4)	-23 (4)	-22 (5)	-21 (5)
5	-21 (6)	-20 (4)	-23 (4)	-23 (3)	-22 (5)
6	-22 (7)	-20 (4)	-23 (5)	-22 (3)	-22 (5)
7	-22 (6)	-20 (4)	-24 (5)	-23 (4)	-22 (5)
8	-23 (6)	-21 (4)	-23 (4)	-24 (4)	-23 (5)
9	-22 (6)	-21 (4)	-24 (5)	-23 (4)	-22 (5)
10	-22 (6)	-20 (4)	-25 (3)	-24 (3)	-23 (5)
11	-23 (6)	-21 (4)	-25 (5)	-24 (4)	-23 (5)
N	12	8	7	6	33

Table B.20 Retraction rx (ms)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	160 (22)	168 (16)	149 (18)	158 (22)	159 (20)
2	153 (23)	164 (22)	154 (20)	163 (24)	158 (22)
3	153 (23)	175 (16)	166 (28)	178 (21)	166 (24)
4	158 (27)	173 (20)	174 (32)	180 (24)	169 (26)
5	158 (27)	175 (18)	168 (32)	185 (21)	169 (26)
6	162 (32)	173 (27)	171 (34)	179 (21)	170 (29)
7	168 (31)	178 (22)	174 (36)	184 (16)	175 (27)
8	174 (30)	178 (19)	170 (32)	193 (24)	177 (27)
9	170 (28)	178 (21)	174 (35)	185 (27)	175 (27)
10	172 (29)	176 (22)	174 (31)	188 (19)	176 (26)
11	172 (29)	179 (18)	176 (34)	181 (14)	176 (25)
N	12	8	7	6	33

Table B.21 Angular acceleration, a1 (rad/s²)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	79 (31)	67 (12)	75 (23)	68 (26)	73 (24)
2	78 (23)	69 (13)	70 (19)	67 (21)	71 (20)
3	79 (17)	70 (15)	74 (20)	72 (12)	74 (16)
4	77 (15)	70 (16)	76 (22)	74 (24)	75 (18)
5	77 (15)	72 (15)	74 (20)	70 (21)	74 (17)
6	77 (14)	73 (17)	79 (18)	78 (23)	77 (17)
7	80 (14)	73 (17)	83 (22)	73 (18)	77 (17)
8	78 (12)	74 (18)	80 (18)	77 (21)	77 (16)
9	80 (16)	75 (12)	79 (18)	77 (22)	78 (17)
10	82 (18)	77 (16)	82 (22)	74 (18)	79 (18)
11	81 (17)	74 (14)	77 (20)	78 (24)	78 (18)
N	12	9	8	9	38

Table B.22 Angular acceleration, a1 (ms)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	108 (16)	98 (8)	110 (18)	102 (5)	105 (13)
2	103 (14)	99 (8)	107 (17)	103 (8)	103 (12)
3	105 (13)	99 (8)	110 (16)	105 (6)	105 (12)
4	104 (11)	100 (8)	108 (11)	107 (7)	104 (10)
5	103 (12)	97 (7)	107 (11)	106 (6)	103 (10)
6	101 (14)	101 (8)	105 (10)	105 (6)	103 (10)
7	103 (13)	101 (5)	107 (10)	108 (6)	105 (9)
8	103 (10)	101 (7)	107 (10)	107 (7)	104 (9)
9	104 (12)	103 (7)	106 (10)	106 (6)	105 (9)
10	106 (12)	100 (8)	107 (11)	108 (8)	105 (10)
11	104 (12)	102 (8)	109 (12)	107 (6)	105 (10)
N	12	9	8	9	38

Table B.23 Angular acceleration, a2 (rad/s²)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	-133 (45)	-99 (29)	-145 (53)	-112 (24)	-122 (42)
2	-133 (47)	-94 (27)	-137 (67)	-115 (30)	-120 (46)
3	-123 (46)	-89 (36)	-133 (73)	-99 (22)	-111 (48)
4	-120 (38)	-93 (30)	-122 (76)	-108 (31)	-111 (45)
5	-115 (37)	-91 (35)	-124 (67)	-106 (34)	-109 (44)
6	-115 (38)	-97 (44)	-128 (71)	-108 (37)	-112 (47)
7	-127 (47)	-92 (38)	-133 (78)	-104 (37)	-114 (52)
8	-119 (45)	-97 (48)	-132 (69)	-99 (28)	-112 (49)
9	-114 (44)	-92 (38)	-129 (64)	-102 (35)	-109 (46)
10	-122 (37)	-95 (39)	-137 (70)	-98 (26)	-113 (46)
11	-113 (36)	-94 (38)	-131 (80)	-111 (27)	-112 (47)
N	12	9	8	9	38

Table B.24 Angular acceleration, a2 (ms)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	173 (10)	155 (14)	168 (6)	158 (10)	164 (13)
2	167 (16)	156 (18)	170 (11)	158 (10)	163 (15)
3	165 (12)	157 (17)	168 (11)	156 (11)	162 (13)
4	160 (13)	154 (13)	171 (16)	157 (10)	160 (14)
5	162 (13)	154 (15)	167 (13)	160 (14)	161 (14)
6	159 (16)	155 (17)	168 (13)	156 (11)	159 (15)
7	160 (14)	155 (15)	167 (11)	163 (23)	161 (16)
8	162 (14)	154 (14)	166 (10)	166 (23)	162 (16)
9	163 (17)	156 (17)	172 (26)	163 (17)	163 (19)
10	163 (16)	154 (16)	174 (19)	158 (9)	162 (16)
11	161 (18)	156 (16)	172 (15)	162 (19)	162 (17)
N	12	9	8	9	38

Table B.25 Angular velocity w (rad/s)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	3.5 (0.9)	3.1 (0.5)	3.2 (1.3)	3.1 (0.6)	3.2 (0.8)
2	3.4 (0.7)	3.2 (0.5)	3.0 (1.2)	3.1 (0.6)	3.2 (0.7)
3	3.3 (0.6)	3.3 (0.7)	3.3 (1.3)	3.3 (0.5)	3.3 (0.8)
4	3.4 (0.6)	3.2 (0.8)	3.4 (1.2)	3.5 (0.5)	3.4 (0.8)
5	3.4 (0.6)	3.3 (0.8)	3.4 (0.9)	3.4 (0.6)	3.4 (0.7)
6	3.5 (0.6)	3.4 (0.8)	3.5 (0.9)	3.5 (0.6)	3.5 (0.7)
7	3.6 (0.6)	3.5 (0.8)	3.8 (1.3)	3.5 (0.6)	3.6 (0.8)
8	3.6 (0.6)	3.4 (0.9)	3.6 (1.0)	3.7 (0.5)	3.6 (0.7)
9	3.5 (0.7)	3.5 (0.7)	3.8 (0.9)	3.6 (0.4)	3.6 (0.7)
10	3.7 (0.5)	3.5 (0.8)	3.7 (1.0)	3.4 (0.6)	3.6 (0.7)
11	3.5 (0.6)	3.5 (0.9)	3.7 (1.1)	3.7 (0.4)	3.6 (0.7)
N	12	9	8	9	38

Table B.26 Angular velocity w (ms)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	136 (13)	126 (10)	131 (15)	129 (6)	131 (12)
2	131 (12)	128 (12)	133 (16)	129 (9)	130 (12)
3	129 (12)	129 (11)	132 (14)	132 (9)	130 (11)
4	129 (10)	129 (10)	135 (16)	133 (9)	131 (11)
5	129 (11)	128 (12)	135 (9)	132 (9)	131 (10)
6	130 (13)	128 (13)	135 (10)	131 (10)	131 (12)
7	131 (10)	128 (13)	133 (13)	134 (10)	132 (11)
8	132 (11)	128 (13)	135 (10)	135 (10)	132 (11)
9	130 (12)	128 (14)	135 (10)	134 (11)	132 (12)
10	130 (13)	129 (14)	136 (9)	133 (10)	132 (11)
11	131 (12)	129 (14)	137 (9)	133 (11)	132 (12)
N	12	9	8	9	38

Table B.27 Head angle q (deg)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	12.4 (2.3)	11.0 (2.2)	11.6 (5.1)	10.8 (2.2)	11.6 (3.0)
2	11.6 (2.2)	11.4 (3.3)	11.5 (5.0)	10.4 (2.4)	11.3 (3.2)
3	11.0 (2.4)	12.6 (4.6)	12.3 (5.3)	11.6 (2.9)	11.8 (3.8)
4	11.7 (2.3)	12.2 (3.4)	13.4 (5.7)	12.8 (2.6)	12.4 (3.5)
5	11.6 (2.3)	12.5 (3.9)	12.7 (4.2)	12.6 (3.4)	12.3 (3.3)
6	12.6 (3.6)	12.6 (4.2)	13.2 (4.2)	12.1 (2.5)	12.6 (3.5)
7	13.2 (2.5)	14.0 (5.8)	13.9 (5.1)	13.0 (2.1)	13.5 (3.9)
8	12.9 (2.1)	13.6 (4.9)	13.6 (4.7)	14.8 (3.6)	13.6 (3.7)
9	12.9 (3.3)	13.4 (4.6)	14.3 (4.4)	14.0 (2.9)	13.6 (3.7)
10	13.2 (2.3)	13.1 (4.7)	14.5 (4.2)	13.0 (2.7)	13.4 (3.4)
11	12.6 (2.5)	13.3 (4.9)	14.6 (4.7)	13.4 (2.1)	13.4 (3.6)
N	12	9	8	8	37

Table B.28 Head angle q (ms)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	184 (13)	178 (18)	177 (14)	174 (10)	179 (14)
2	178 (11)	183 (27)	182 (19)	174 (17)	179 (18)
3	177 (10)	191 (34)	182 (17)	183 (20)	183 (21)
4	177 (14)	187 (20)	190 (24)	193 (28)	186 (21)
5	182 (21)	189 (25)	185 (19)	188 (21)	186 (21)
6	185 (28)	188 (31)	187 (21)	186 (20)	186 (25)
7	185 (18)	195 (38)	188 (19)	191 (23)	189 (25)
8	185 (14)	191 (31)	188 (19)	206 (35)	192 (25)
9	185 (22)	192 (32)	191 (24)	199 (34)	191 (27)
10	185 (22)	189 (31)	191 (21)	196 (27)	190 (24)
11	186 (21)	192 (30)	193 (22)	188 (17)	190 (22)
N	12	9	8	8	37

Table B.29 Orbicularis oculi onset (ms)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	71 (13)	67 (8)	66 (8)	64 (8)	67 (10)
2	66 (8)	65 (11)	64 (8)	63 (9)	64 (9)
3	70 (13)	68 (12)	64 (5)	65 (7)	67 (10)
4	62 (6)	67 (10)	69 (15)	65 (13)	66 (11)
5	68 (10)	67 (10)	64 (8)	65 (10)	66 (9)
6	69 (13)	69 (8)	65 (4)	66 (9)	67 (9)
7	72 (14)	72 (17)	67 (10)	66 (10)	69 (13)
8	72 (9)	73 (10)	64 (3)	64 (8)	68 (9)
9	70 (14)	69 (7)	67 (6)	63 (8)	68 (9)
10	72 (11)	70 (8)	73 (10)	64 (8)	70 (10)
11	69 (8)	73 (9)	66 (8)	61 (9)	67 (9)
N	8	9	8	9	34

Table B.30 Masseter onset (ms)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	81 (12)	74 (8)	67 (4)	81 (9)	76 (10)
2	74 (10)	73 (8)	71 (7)	77 (13)	74 (10)
3	75 (12)	72 (9)	71 (6)	76 (13)	74 (10)
4	71 (11)	73 (9)	73 (8)	73 (10)	72 (9)
5	73 (13)	69 (5)	71 (5)	75 (12)	72 (10)
6	76 (13)	73 (14)	73 (4)	78 (11)	75 (11)
7	78 (11)	70 (9)	70 (4)	76 (7)	74 (9)
8	76 (14)	71 (9)	71 (8)	75 (8)	73 (10)
9	73 (11)	72 (8)	73 (8)	75 (9)	73 (9)
10	75 (13)	73 (11)	72 (7)	71 (5)	73 (9)
11	76 (12)	74 (14)	73 (9)	73 (10)	74 (11)
N	9	9	7	9	34

Table B.31 Sternocleidomastoid onset (ms)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	69 (5)	75 (5)	68 (5)	74 (6)	72 (6)
2	65 (4)	73 (6)	70 (5)	73 (5)	70 (6)
3	65 (6)	72 (6)	70 (4)	74 (6)	70 (6)
4	64 (6)	70 (8)	73 (9)	72 (6)	69 (8)
5	63 (6)	72 (7)	68 (6)	75 (6)	69 (8)
6	69 (9)	69 (6)	70 (7)	73 (6)	70 (7)
7	68 (8)	70 (9)	72 (6)	73 (8)	70 (8)
8	69 (11)	70 (10)	69 (7)	73 (3)	70 (8)
9	68 (9)	69 (5)	70 (6)	74 (8)	70 (8)
10	66 (8)	69 (6)	70 (5)	72 (5)	69 (6)
11	65 (7)	68 (6)	71 (6)	70 (6)	68 (7)
N	11	9	8	9	37

Table B.32 Cervical paraspinal onset (ms)

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	76 (7)	75 (4)	78 (8)	81 (7)	77 (7)
2	77 (7)	81 (11)	80 (7)	77 (7)	79 (8)
3	77 (7)	74 (6)	78 (3)	79 (4)	77 (6)
4	74 (6)	76 (5)	77 (3)	74 (8)	75 (6)
5	72 (9)	75 (5)	78 (5)	74 (6)	74 (7)
6	72 (8)	75 (5)	80 (6)	77 (6)	76 (7)
7	78 (6)	73 (8)	79 (8)	74 (6)	76 (7)
8	80 (6)	75 (8)	78 (4)	76 (6)	78 (6)
9	78 (6)	74 (3)	78 (5)	76 (6)	77 (5)
10	73 (6)	79 (4)	76 (6)	74 (6)	75 (6)
11	78 (6)	77 (5)	79 (7)	73 (6)	77 (6)
N	11	7	7	9	34

Table B.33 SCM RMS amplitude

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	0.17 (0.04)	0.31 (0.31)	0.21 (0.09)	0.29 (0.11)	0.24 (0.17)
2	0.17 (0.03)	0.27 (0.22)	0.18 (0.06)	0.25 (0.16)	0.22 (0.14)
3	0.15 (0.05)	0.18 (0.15)	0.14 (0.05)	0.18 (0.11)	0.16 (0.10)
4	0.13 (0.03)	0.19 (0.19)	0.12 (0.05)	0.16 (0.09)	0.15 (0.11)
5	0.13 (0.05)	0.18 (0.21)	0.12 (0.05)	0.16 (0.08)	0.14 (0.11)
6	0.12 (0.04)	0.18 (0.20)	0.12 (0.06)	0.16 (0.10)	0.14 (0.11)
7	0.11 (0.04)	0.15 (0.17)	0.11 (0.05)	0.13 (0.09)	0.13 (0.10)
8	0.11 (0.03)	0.14 (0.13)	0.12 (0.06)	0.12 (0.07)	0.12 (0.08)
9	0.11 (0.04)	0.14 (0.13)	0.10 (0.05)	0.12 (0.08)	0.12 (0.08)
10	0.10 (0.03)	0.17 (0.17)	0.09 (0.04)	0.11 (0.06)	0.12 (0.09)
11	0.10 (0.04)	0.14 (0.16)	0.09 (0.06)	0.11 (0.05)	0.11 (0.09)
N	11	9	8	8	36

Table B.34 PARA RMS amplitude

	Alerted		Unalerted		
Trial	F	M	F	M	Group
1	0.12 (0.08)	0.10 (0.06)	0.17 (0.09)	0.19 (0.13)	0.14 (0.10)
2	0.10 (0.07)	0.07 (0.03)	0.14 (0.08)	0.14 (0.09)	0.11 (0.07)
3	0.09 (0.06)	0.05 (0.03)	0.09 (0.06)	0.09 (0.05)	0.08 (0.05)
4	0.07 (0.05)	0.06 (0.04)	0.07 (0.06)	0.08 (0.06)	0.07 (0.05)
5	0.08 (0.05)	0.05 (0.03)	0.08 (0.05)	0.07 (0.05)	0.07 (0.05)
6	0.06 (0.04)	0.05 (0.04)	0.08 (0.05)	0.09 (0.06)	0.07 (0.05)
7	0.07 (0.05)	0.05 (0.03)	0.06 (0.04)	0.06 (0.03)	0.06 (0.04)
8	0.05 (0.04)	0.04 (0.02)	0.07 (0.06)	0.07 (0.05)	0.06 (0.04)
9	0.05 (0.03)	0.05 (0.04)	0.06 (0.07)	0.08 (0.06)	0.06 (0.05)
10	0.06 (0.03)	0.05 (0.03)	0.07 (0.05)	0.07 (0.05)	0.06 (0.04)
11	0.06 (0.05)	0.05 (0.04)	0.05 (0.06)	0.07 (0.04)	0.06 (0.05)
N	11	7	7	8	33

APPENDIX C SUMMARY OF DATA FOR PERTURBATION PROPERTY EXPERIMENT

The following tables contain the un-normalized mean (S.D.) of all dependent variables for the perturbation properties experiment documented in Chapter 6. The data are presented in tables similar to those used for the normalized data in Chapter 6. The data presented in the following tables were separated into male and female groups because the absolute value of some variables varied with gender. This gender dependence was removed by normalizing the data for the analysis contained in Chapter 6.

Table C.1 Mastoid process, X (mm)

Female

Acceleration	L_v	M_v	H_v
H_a		6 (20)	6 (19)
M_a	6 (21)	6 (20)	6 (20)
L_a	6 (20)	6 (20)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		25 (14)	24 (14)
M_a	24 (14)	23 (14)	25 (14)
L_a	23 (14)	24 (14)	

Velocity

Table C.2 Mastoid process, Z (mm)

Female

Acceleration	L_v	M_v	H_v
H_a		-633 (32)	-633 (32)
M_a	-633 (31)	-634 (31)	-633 (31)
L_a	-633 (31)	-633 (32)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		-663 (29)	-663 (29)
M_a	-663 (29)	-663 (29)	-663 (29)
L_a	-663 (30)	-663 (29)	

Velocity

Table C.3 Manubrium-C7 midpoint, X (mm)

Female

Acceleration	L_v	M_v	H_v
H_a		16 (14)	15 (14)
M_a	16 (16)	16 (15)	17 (15)
L_a	15 (15)	16 (14)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		33 (14)	33 (14)
M_a	33 (14)	33 (14)	34 (14)
L_a	33 (14)	33 (14)	

Velocity

Table C.4 Manubrium-C7 midpoint, Z (mm)

Female

Acceleration	L_v	M_v	H_v
H_a		-547 (28)	-547 (28)
M_a	-547 (28)	-547 (28)	-547 (28)
L_a	-547 (28)	-546 (28)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		-573 (23)	-573 (24)
M_a	-573 (23)	-573 (24)	-573 (23)
L_a	-573 (24)	-573 (23)	

Velocity

Table C.5 Initial head angle (deg)

Female

Acceleration	L_v	M_v	H_v
H_a		7.7 (3.5)	7.7 (3.2)
M_a	7.7 (3.3)	7.4 (2.9)	7.5 (3.2)
L_a	7.8 (3.1)	7.9 (3.5)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		10.5 (5.7)	10.7 (5.8)
M_a	10.6 (5.0)	11.0 (5.5)	10.6 (5.1)
L_a	10.9 (5.8)	10.8 (5.4)	

Velocity

Table C.6 Initial torso angle (deg)

Female

Acceleration	L_v	M_v	H_v
H_a		-15.5 (3.9)	-15.4 (3.9)
M_a	-15.4 (3.9)	-15.6 (4.0)	-15.8 (4.0)
L_a	-15.4 (3.8)	-15.6 (4.0)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		-16.8 (4.7)	-16.7 (4.6)
M_a	-16.8 (4.2)	-16.6 (4.4)	-16.7 (4.2)
L_a	-16.5 (4.4)	-16.7 (4.3)	

Velocity

Table C.7 Forehead acceleration, x1 (g)

Female

Acceleration	L_v	M_v	H_v
H_a		0.70 (0.14)	1.02 (0.23)
M_a	0.44 (0.08)	0.72 (0.14)	0.90 (0.14)
L_a	0.42 (0.08)	0.61 (0.10)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		0.86 (0.18)	1.16 (0.25)
M_a	0.47 (0.11)	0.85 (0.16)	1.02 (0.15)
L_a	0.46 (0.10)	0.65 (0.09)	

Velocity

Table C.8 Forehead acceleration, z1 (g)

Female

Acceleration	L_v	M_v	H_v
H_a		-0.80 (0.24)	-1.12 (0.39)
M_a	-0.43 (0.07)	-0.69 (0.16)	-0.90 (0.23)
L_a	-0.38 (0.06)	-0.47 (0.09)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		-0.78 (0.14)	-0.98 (0.22)
M_a	-0.43 (0.08)	-0.68 (0.12)	-0.82 (0.18)
L_a	-0.38 (0.06)	-0.49 (0.10)	

Velocity

Table C.9 Forehead acceleration, z2 (g)

Female

Acceleration	L_v	M_v	H_v
H_a		1.49 (0.45)	2.22 (0.68)
M_a	0.72 (0.26)	1.35 (0.44)	1.87 (0.56)
L_a	0.62 (0.21)	0.76 (0.21)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		1.63 (0.43)	2.29 (0.47)
M_a	0.76 (0.25)	1.40 (0.36)	1.74 (0.34)
L_a	0.68 (0.23)	0.82 (0.20)	

Velocity

Table C.10 Mastoid process acceleration, x2 (g)

Female

Acceleration	L_v	M_v	H_v
H_a		1.25 (0.39)	2.01 (0.50)
M_a	0.59 (0.16)	1.21 (0.36)	1.61 (0.37)
L_a	0.55 (0.14)	0.76 (0.18)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		1.04 (0.12)	1.62 (0.18)
M_a	0.52 (0.09)	0.98 (0.12)	1.34 (0.14)
L_a	0.47 (0.08)	0.64 (0.07)	

Velocity

Table C.11 Mastoid process acceleration, z3 (g)

Female

Acceleration	L_v	M_v	H_v
H_a		-0.29 (0.08)	-0.35 (0.11)
M_a	-0.14 (0.04)	-0.22 (0.07)	-0.23 (0.07)
L_a	-0.08 (0.03)	-0.10 (0.04)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		-0.28 (0.10)	-0.33 (0.12)
M_a	-0.14 (0.05)	-0.21 (0.09)	-0.24 (0.08)
L_a	-0.10 (0.04)	-0.13 (0.04)	

Velocity

Table C.12 Mastoid process acceleration, z4 (g)

Female

Acceleration	L_v	M_v	H_v
H_a		0.42 (0.14)	0.64 (0.22)
M_a	0.21 (0.08)	0.41 (0.13)	0.52 (0.16)
L_a	0.18 (0.06)	0.24 (0.07)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		0.43 (0.10)	0.56 (0.13)
M_a	0.20 (0.04)	0.38 (0.09)	0.44 (0.11)
L_a	0.17 (0.04)	0.22 (0.06)	

Velocity

Table C.13 Torso acceleration, axz (g)

Female

Acceleration	L_v	M_v	H_v
H_a		1.44 (0.23)	1.83 (0.18)
M_a	0.71 (0.14)	1.19 (0.14)	1.59 (0.15)
L_a	0.56 (0.08)	0.86 (0.07)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		1.15 (0.17)	1.50 (0.19)
M_a	0.58 (0.10)	1.01 (0.13)	1.38 (0.18)
L_a	0.49 (0.08)	0.77 (0.08)	

Velocity

Table C.14 Retraction rx (mm)

Female

Acceleration	L_v	M_v	H_v
H_a		-27 (5)	-37 (6)
M_a	-15 (3)	-26 (5)	-37 (7)
L_a	-14 (3)	-24 (6)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		-25 (3)	-35 (4)
M_a	-13 (3)	-24 (3)	-35 (2)
L_a	-12 (2)	-24 (3)	

Velocity

Table C.15 Angular acceleration, a1 (rad/s²)

Female

Acceleration	L_v	M_v	H_v
H_a		88 (23)	142 (36)
M_a	42 (7)	77 (18)	110 (29)
L_a	37 (8)	45 (10)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		77 (12)	119 (22)
M_a	39 (5)	68 (12)	89 (16)
L_a	35 (5)	42 (7)	

Velocity

Table C.16 Angular acceleration, a2 (rad/s²)

Female

Acceleration	L_v	M_v	H_v
H_a		-117 (43)	-192 (88)
M_a	-54 (23)	-102 (41)	-143 (51)
L_a	-45 (19)	-49 (15)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		-111 (40)	-159 (48)
M_a	-50 (19)	-92 (32)	-107 (30)
L_a	-43 (17)	-46 (16)	

Velocity

Table C.17 Angular velocity w (rad/s)

Female

Acceleration	L_v	M_v	H_v
H_a		4.0 (0.8)	6.3 (1.0)
M_a	1.9 (0.4)	3.7 (0.7)	5.5 (1.0)
L_a	1.7 (0.4)	2.6 (0.7)	

Velocity

Male

	L_v	M_v	H_v
H_a		3.9 (0.6)	5.7 (1.0)
M_a	1.9 (0.3)	3.5 (0.6)	4.9 (0.7)
L_a	1.8 (0.3)	2.5 (0.5)	

Velocity

Table C.18 Head angle q (deg)

Female

Acceleration	L_v	M_v	H_v
H_a		16.4 (3.4)	25.3 (3.9)
M_a	7.9 (2.1)	15.4 (3.0)	24.7 (4.3)
L_a	7.4 (1.9)	14.8 (4.1)	

Velocity

Male

	L_v	M_v	H_v
H_a		16.3 (3.0)	24.3 (4.6)
M_a	8.6 (1.9)	15.0 (3.2)	23.6 (4.3)
L_a	8.2 (2.1)	14.5 (2.8)	

Velocity

Table C.19 Forehead acceleration, x1 (ms)

Female

Acceleration	L_v	M_v	H_v
H_a		177 (18)	187 (18)
M_a	170 (20)	180 (17)	201 (20)
L_a	174 (17)	201 (31)	

Velocity

Male

	L_v	M_v	H_v
H_a		172 (16)	179 (15)
M_a	170 (14)	176 (16)	194 (20)
L_a	181 (23)	203 (28)	

Velocity

Table C.20 Forehead acceleration, z1 (ms)

Female

Acceleration	L_v	M_v	H_v
H_a		90 (16)	97 (16)
M_a	89 (11)	95 (14)	105 (15)
L_a	94 (11)	104 (10)	

Velocity

Male

	L_v	M_v	H_v
H_a		90 (16)	100 (11)
M_a	85 (14)	95 (11)	102 (7)
L_a	93 (8)	106 (7)	

Velocity

Table C.21 Forehead acceleration, z2 (ms)

Female

Acceleration	L_v	M_v	H_v
H_a		160 (12)	168 (12)
M_a	157 (16)	166 (11)	180 (14)
L_a	169 (27)	194 (24)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		159 (7)	163 (9)
M_a	159 (11)	164 (8)	175 (12)
L_a	169 (12)	195 (18)	

Velocity

Table C.22 Mastoid process acceleration, x2 (ms)

Female

Acceleration	L_v	M_v	H_v
H_a		99 (7)	104 (8)
M_a	93 (14)	107 (9)	116 (11)
L_a	109 (9)	128 (16)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		104 (8)	114 (8)
M_a	92 (9)	112 (8)	125 (7)
L_a	109 (9)	134 (14)	

Velocity

Table C.23 Mastoid process acceleration, z3 (ms)

Female

Acceleration	L_v	M_v	H_v
H_a		53 (3)	58 (4)
M_a	51 (4)	63 (5)	64 (6)
L_a	67 (6)	82 (14)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		57 (16)	66 (17)
M_a	54 (18)	65 (17)	72 (13)
L_a	68 (17)	90 (10)	

Velocity

Table C.24 Mastoid process acceleration, z4 (ms)

Female

Acceleration	L_v	M_v	H_v
H_a		129 (12)	128 (12)
M_a	131 (13)	132 (13)	136 (14)
L_a	139 (11)	150 (14)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		147 (14)	147 (13)
M_a	151 (13)	153 (13)	154 (16)
L_a	157 (12)	169 (18)	

Velocity

Table C.25 Torso acceleration, axz (ms)

Female

Acceleration	L_v	M_v	H_v
H_a		60 (3)	71 (10)
M_a	59 (5)	74 (7)	97 (6)
L_a	79 (7)	111 (8)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		61 (6)	77 (8)
M_a	54 (3)	78 (7)	99 (7)
L_a	80 (4)	113 (7)	

Velocity

Table C.26 Retraction rx (ms)

Female

Acceleration	L_v	M_v	H_v
H_a		183 (34)	187 (28)
M_a	177 (23)	186 (25)	204 (34)
L_a	186 (20)	213 (29)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		200 (37)	205 (29)
M_a	186 (28)	198 (26)	221 (34)
L_a	194 (25)	239 (59)	

Velocity

Table C.27 Angular acceleration, a1 (ms)

Female

Acceleration	L_v	M_v	H_v
H_a		103 (7)	111 (8)
M_a	93 (7)	108 (7)	121 (12)
L_a	101 (7)	115 (15)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		103 (5)	111 (6)
M_a	90 (7)	107 (6)	118 (7)
L_a	100 (5)	111 (9)	

Velocity

Table C.28 Angular acceleration, a2 (ms)

Female

Acceleration	L_v	M_v	H_v
H_a		162 (11)	173 (21)
M_a	157 (13)	167 (15)	184 (18)
L_a	171 (26)	199 (27)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		156 (7)	163 (9)
M_a	157 (12)	164 (9)	174 (13)
L_a	171 (19)	200 (23)	

Velocity

Table C.29 Angular velocity w (ms)

Female

Acceleration	L_v	M_v	H_v
H_a		131 (11)	137 (10)
M_a	123 (12)	136 (10)	149 (11)
L_a	130 (13)	155 (18)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		130 (7)	138 (8)
M_a	123 (8)	134 (8)	147 (10)
L_a	132 (9)	152 (16)	

Velocity

Table C.30 Head angle q (ms)

Female

Acceleration	L_v	M_v	H_v
H_a		207 (42)	210 (31)
M_a	197 (48)	215 (42)	230 (34)
L_a	205 (39)	254 (45)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		199 (29)	214 (29)
M_a	200 (38)	209 (33)	232 (32)
L_a	211 (38)	251 (46)	

Velocity

Table C.31 Torso acceleration onset (ms)

Female

Acceleration	L_v	M_v	H_v
H_a		18.3 (2.6)	18.2 (2.0)
M_a	19.8 (2.8)	20.0 (2.2)	19.8 (2.3)
L_a	21.8 (2.8)	21.8 (2.4)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		17.0 (1.5)	17.2 (1.7)
M_a	19.5 (1.6)	19.0 (1.2)	19.7 (1.8)
L_a	21.1 (1.7)	22.0 (1.4)	

Velocity

Table C.32 Head acceleration onset (ms)

Female

Acceleration	L_v	M_v	H_v
H_a		26.5 (3.9)	27.4 (4.7)
M_a	29.7 (4.4)	29.7 (4.7)	30.4 (5.2)
L_a	31.9 (5.3)	32.1 (5.3)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		26.0 (3.3)	26.2 (3.9)
M_a	29.2 (3.7)	28.8 (3.9)	29.2 (3.6)
L_a	32.5 (4.7)	32.2 (3.6)	

Velocity

Table C.33 Orbicularis oculi onset (ms)

Female

Acceleration	L_v	M_v	H_v
H_a		63 (6)	67 (6)
M_a	65 (8)	68 (7)	72 (13)
L_a	69 (7)	71 (12)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		63 (6)	69 (9)
M_a	64 (8)	66 (7)	72 (12)
L_a	67 (9)	65 (7)	

Velocity

Table C.34 Masseter onset (ms)

Female

Acceleration	L_v	M_v	H_v
H_a		66 (7)	73 (8)
M_a	66 (5)	73 (5)	80 (11)
L_a	72 (4)	74 (8)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		69 (6)	78 (8)
M_a	69 (6)	72 (5)	79 (9)
L_a	72 (5)	77 (9)	

Velocity

Table C.35 Sternocleidomastoid onset (ms)

Female

Acceleration	L_v	M_v	H_v
H_a		65 (4)	68 (6)
M_a	66 (4)	70 (4)	73 (6)
L_a	70 (5)	73 (7)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		72 (5)	73 (5)
M_a	69 (4)	73 (4)	76 (5)
L_a	73 (3)	76 (3)	

Velocity

Table C.36 Cervical paraspinal onset (ms)

Female

Acceleration	L_v	M_v	H_v
H_a		70 (6)	76 (4)
M_a	74 (4)	76 (3)	75 (11)
L_a	75 (3)	75 (3)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		73 (5)	77 (4)
M_a	77 (3)	77 (2)	80 (7)
L_a	78 (2)	78 (3)	

Velocity

Table C.37 SCM RMS amplitude

Female

Acceleration	L_v	M_v	H_v
H_a		0.09 (0.04)	0.13 (0.05)
M_a	0.06 (0.05)	0.09 (0.05)	0.10 (0.05)
L_a	0.05 (0.03)	0.06 (0.03)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		0.13 (0.08)	0.16 (0.10)
M_a	0.06 (0.07)	0.13 (0.08)	0.12 (0.05)
L_a	0.06 (0.07)	0.05 (0.03)	

Velocity

Table C.38 PARA RMS amplitude

Female

Acceleration	L_v	M_v	H_v
H_a		0.05 (0.04)	0.08 (0.05)
M_a	0.03 (0.03)	0.06 (0.04)	0.06 (0.05)
L_a	0.03 (0.02)	0.04 (0.03)	

Velocity

Male

Acceleration	L_v	M_v	H_v
H_a		0.07 (0.04)	0.08 (0.05)
M_a	0.04 (0.03)	0.07 (0.03)	0.07 (0.03)
L_a	0.04 (0.03)	0.04 (0.02)	

Velocity