A Microanalysis of EMG and EEG Changes during the Sleep Onset Period (SOP):

A Theoretical Investigation with Practical Applications.

By

Cory Roy Martin

A thesis
submitted in partial fulfillment
of the requirement for the degree
Master of Arts

Department of Psychology
BROCK UNIVERSITY
St. Catharines, Ontario

2004

© Cory Roy Martin, 2004
Acknowledgements

First and foremost, I would like to acknowledge the tremendous supervision and guidance afforded to me by my advisor Dr. Robert Ogilvie, or more informally Bob. Dr. Ogilvie was a pleasure to work with on this project. Along with being a very patient man, Bob is also a very kind and generous individual and I wish him all the best in his retirement.

I would also like to extend a debt of gratitude to the individuals that were involved in my thesis advisory committee, namely Dawn Good and David DiBattista who provided me with much needed advice and invaluable constructive criticism throughout this endeavor. In addition, I would like to thank Alistair MacLean from Queen’s University for serving as the external examiner for my thesis defense. It was a pleasure to have such a knowledgeable and respected member of the sleep research community present to provide support and challenging suggestions.

I also wish to thank the entire Psychology department at Brock for creating a fulfilling and beneficial learning environment during my time in the Master’s program.

This project could never have been completed without the help I received from fellow students in the sleep lab. Karen Baxter, Stan Lazic, Colin Massicotte, and Catherine Milner were always available and willing to assist with hook-ups and pilot testing. Their help was greatly appreciated.
Dedication

I would like to dedicate this project to my family. It is an honour to be the first member of my immediate family to attend university and see it through to the Master’s level. I hope this is an accomplishment that makes them proud, not only of my efforts but also of the support that they have always given me in each and every endeavor I have undertaken.
Abstract

The present study has both theoretical and practical aspects. The theoretical intent of the study was to closely examine the relationship between muscle activity (EMG) and EEG state during the process of falling asleep. Sleep stages during sleep onset (SO) have been generally defined with regards to brain wave activity (Rechtschaffen & Kales (1968); and more precisely by Hori, Hayashi, & Morikawa (1994)). However, no previous study has attempted to quantify the changes in muscle activity during this same process.

The practical aspect of the study examined the reliability of a commercially developed wrist-worn alerting device (NovAlert™) that utilizes changes in muscle activity/tension in order to alert its user in the event that he/she experiences reduced wakefulness that may result in dangerous consequences.

Twelve female participants (aged 18-42) spent three consecutive nights in the sleep lab (“Adaptation”, “EMG”, and “NOVA” nights). Each night participants were given 5, twenty-minute nap opportunities. On the EMG night, participants were allowed to fall asleep freely. On the NOVA night, participants wore the NovAlert™ wrist device that administered a Psychomotor Vigilance Test (PVT) when it detected that muscle activity levels had dropped below baseline.

Nap sessions were scored using Hori’s 9-stage scoring system (Hori et al, 1994). Power spectral analyses (FFT) were also performed. Effects of the PVT administration on EMG and EEG frequencies were also examined.

Both chin and wrist EMG activity showed reliable and significant decline during the early stages of Hori staging (stages H0 to H3 characterized by decreases in alpha
activity). All frequency bands studied went through significant changes as the
participants progressed through each of Hori's 9 SO stages. Delta, theta, and sigma
activity increased later in the SO continuum while a clear alpha dominance shift was
noted as alpha activity shifted from the posterior regions of the brain (during Hori stages
H0 to H3) to the anterior portions (during Hori stages H7 to H9). Administration of the
PVT produced significant increases in EMG activity and was effective in reversing
subjective drowsiness experienced during the later stages of sleep onset. Limitations of
the alerting effects of the PVTs were evident following 60 to 75 minutes of use in that
PVTs delivered afterwards were no longer able to significantly increase EMG levels.

The present study provides a clearer picture of the changes in EMG and EEG
during the sleep onset period while testing the efficacy of a commercially developed
alerting device. EMG decreases were found to begin during Hori stage 0 when EEG was
dominated by alpha wave activity and were maximal as Hori stages 2 to 5 were traversed
(coincident with alpha and beta activity). This signifies that EMG decrements and the
loss of resting alpha activity are closely related. Since decreased alpha has long been
associated with drowsiness and impending sleep, this investigation links drops in muscle
tone with sleepiness more directly than in previous investigations. The EMG changes
were reliably demonstrated across participants and the NovAlert™ detected the EMG
decrements when Hori stage 3 was entered. The alerting vibrations produced by the
NovAlert™ occurred early enough in the SO process to be of practical importance as a
sleepiness monitoring and alerting device.
**Table of Contents**

Acknowledgements ................................................................. ii
Dedication ........................................................................ iii
Abstract .............................................................................. iv
List of Tables ......................................................................... viii
List of Figures .......................................................................... ix
List of Abbreviations .............................................................. xi

Introduction ............................................................................... 1
  Sleep Stage Scoring ............................................................ 1
  Measurements of Sleep Onset ................................................ 2
  Behavioural vs. Passive Monitoring ........................................ 3
  The Nightcap ........................................................................ 4
  Hori’s 9-stage Continuum ..................................................... 5
  The Need for an Alertness-Monitoring Device ....................... 8
  Preventive Measures used to counteract sleepiness/drowsiness .. 11
  The NOVAAlert System ......................................................... 12
  Muscle Activity and the Sleep Onset Period .......................... 15
  Muscle Innervation and Control .......................................... 15
  Muscle Contraction and Regulation ...................................... 17
  Muscle Control ..................................................................... 19
  Hypotheses ........................................................................... 20
  Theoretical Predictions ....................................................... 20
  Practical Applications .......................................................... 22

Method ..................................................................................... 23
  Participants ........................................................................... 23
  Sleep Lab and Polysomnographic Data Acquisition Devices .... 23
  NOVAAlert ........................................................................... 24
  Procedure .............................................................................. 25
  Participant recruitment ....................................................... 25
  Overnight sessions ............................................................... 26
  Questionnaires ...................................................................... 29
  Analyses ................................................................................. 30

Results ...................................................................................... 32
  EMG Night ............................................................................ 33
  Chin EMG ............................................................................ 33
  Wrist EMG ............................................................................ 35
  EEG Activity .......................................................................... 36
  NOVA Night .......................................................................... 49
  Time of Night Effects .......................................................... 50
  Pre-PVT versus Post-PVT ..................................................... 51
  Chin EMG Power ................................................................. 51
  Wrist EMG Power ............................................................... 52
List of Tables

Tables

1. Differences Between H0 and all other Hori Stages for Wrist EMG .......... 36
4. Summary table of Paired Sample t-tests for Wrist EMG increases .......... 53
5. Summary ANOVA table for Psychomotor Vigilance Test (PVT) on EEG Power............................................................... 58
# List of Figures

<table>
<thead>
<tr>
<th>Figures</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Hori's 9-Stage Scoring</td>
<td>7</td>
</tr>
<tr>
<td>2. Chin EMG Power across Hori Stages</td>
<td>34</td>
</tr>
<tr>
<td>3. Wrist EMG Power across Hori Stages</td>
<td>35</td>
</tr>
<tr>
<td>4. EEG Power across Hori Stages</td>
<td>38</td>
</tr>
<tr>
<td>5. EEG Power (excluding Delta) across Hori Stages</td>
<td>38</td>
</tr>
<tr>
<td>6. Delta Power across Hori Stages as a function of Brain Region</td>
<td>40</td>
</tr>
<tr>
<td>7. Delta Power Laterality</td>
<td>40</td>
</tr>
<tr>
<td>8. Theta Power across Hori Stages as a function of Brain Region</td>
<td>41</td>
</tr>
<tr>
<td>9. Theta Power Laterality</td>
<td>42</td>
</tr>
<tr>
<td>10. Alpha1 Power across Hori Stages as a function of Brain Region</td>
<td>43</td>
</tr>
<tr>
<td>11. Alpha1 Power Laterality</td>
<td>43</td>
</tr>
<tr>
<td>12. Alpha2 Power across Hori Stages as a function of Brain Region</td>
<td>44</td>
</tr>
<tr>
<td>13. Alpha2 Power Laterality</td>
<td>45</td>
</tr>
<tr>
<td>14. Sigma Power across Hori Stages as a function of Brain Region</td>
<td>46</td>
</tr>
<tr>
<td>15. Sigma Power Laterality</td>
<td>46</td>
</tr>
<tr>
<td>16. Beta Power across Hori Stages as a function of Brain Region</td>
<td>47</td>
</tr>
<tr>
<td>17. Beta Power Laterality</td>
<td>48</td>
</tr>
<tr>
<td>18. Chin EMG Power before and after PVT across Naps</td>
<td>52</td>
</tr>
<tr>
<td>19. Wrist EMG Power before and after PVT across Naps</td>
<td>53</td>
</tr>
<tr>
<td>20. Mean # of PVTs per Ss as a function of Hori Stage</td>
<td>55</td>
</tr>
</tbody>
</table>
List of Figures (Continued)

21. Mean # of PVTs during each Nap......................................................... 56
22. Mean # of failed PVTs across Naps....................................................... 57
23. Total EEG Power before and after PVT across Naps............................... 58
24. Delta Power before and after PVT across Naps....................................... 59
25. Theta Power before and after PVT across Naps...................................... 60
26. Alpha1 Power before and after PVT across Naps.................................... 61
27. Alpha2 Power before and after PVT across Naps.................................... 61
28. Sigma Power before and after PVT across Naps..................................... 62
29. Beta Power before and after PVT across Naps....................................... 63
30. Mean SSS ratings across Naps................................................................. 64
31. Mean VAS ratings across Naps................................................................. 65
32. Mean time (in seconds) to the first PVT during each Nap......................... 66
33. Mean SSS ratings before and after PVTs.................................................. 67
List of Abbreviations

EEG – Electroencephalogram
EOG - Electrooculargram
EMG – Electromyogram
REM – Rapid Eye Movements
SOP – Sleep Onset Period or Sleep Onset Process
AOM – NOVA Alert-O-Meter
WU – NOVA Wrist Unit
PVT – Psychomotor Vigilance Test
PSG – Polysomnogram
ANOVA – Analysis of Variance
Introduction

Loomis, Harvey & Hobart (1935) implemented the use of polysomnographic recordings to monitor brain wave activity during sleep. These authors were the first to report that during part of the night the brain exhibited “many spontaneous bursts of waves,” while other parts of the night showed relatively few. Several types of EEG waves were described during sleep for the first time, including spindles, trains, saw-tooth, and random waves. Loomis et al. suggested that the level of consciousness was connected with changes in the type of wave exhibited. Further studies focused on brain potential change during the sleep onset period—a period that primarily consisted of two waves: alpha during waking, and low voltage activity without the presence of alpha (Davis, Davis, Loomis, Harvey, & Hobart, 1937).

Sleep Stage Scoring

As sleep studies became more frequent, EEG recordings became the standard method of determining the transition from wakefulness to sleep and discerning among the different sleep stages. Rechtschaffen & Kales (1968) developed a manual that outlined the standard guidelines for scoring sleep stages. The Rechtschaffen & Kales standard manual contains the stages W (wakefulness), 1, 2, 3, 4, and REM. Stage W consists of EEG that contains alpha waves and/or low voltage, mixed frequency waves. Stage W features less than 50% alpha mixed with beta and theta waves. Stage 1 contains greater than 50% alpha mixed with theta waves and is accompanied by slow rolling eye movements. Stage 2 is composed of sleep spindles (12-14 cps) and K-complexes with less than 20% delta waves. Stage 3 contains greater than 20% but less than 50% delta
waves while Stage 4 is indicated by greater than 50% delta (high amplitude, slow frequency). REM sleep is characterized by saw-toothed waves with episodic rapid eye movements and low EMG activity (Rechtschaffen & Kales, 1968).

**Measurements of Sleep Onset**

With the growing popularity of EEG-based determinants of sleep states came decreasing amounts of research interest in traditional behavioural-based methods of determining sleep states. However, some researchers were not so eager to abandon what they felt to be the more accurate methods of determining sleep onset.

In his classic work *Sleep and Wakefulness*, Nathaniel Kleitman commented briefly on the observation that muscle tonus falls during the passage from wakefulness to sleep. In perhaps one of the earliest studies of the relationship between muscle tonus and sleep onset, Kleitman asked participants to hold a spool between two fingers as they were falling asleep. As expected, when muscle tonus relaxed, the spool dropped. Kleitman observed that the spool tended to drop within 25 seconds after the disappearance of alpha waves and postulated that muscle tonus diminished shortly after the alpha pattern is lost (Kleitman, 1963).

Additional early studies reported reduced muscle activity during sleep onset; Max (1935) found that there was a reduction in action-current activity with the approach of sleep. Good (1975) was able to show that muscle tension decreased from Waking to Stage 1 but was unable to demonstrate a reliable pattern in the decline of EMG.
Behavioural vs. Passive Monitoring

Kleitman’s ‘spool-dropping’ experiment provided researchers with an excellent means of determining sleep onset using a passive behavioural measure. Similar measures have been adopted since. In another experiment, Kleitman (1939) also used a piece of paper instead of the spool. The paper was held between the participant’s thumb and forefinger. Sleep onset was determined by the time at which the paper dropped. Ogilvie, Wilkinson, & Allison (1989) used a ‘deadman switch’ (DM) in an attempt to more precisely estimate sleep onset. Participants were asked to continuously maintain pressure on the DM switch. When they did so, a single line would be present on the EEG recording. However, a double line on the EEG indicated that the participant had released the DM switch demonstrating that muscle tonus had decreased (with impending sleep) to a point where that participant could no longer hold the switch closed. There was a significant relationship between DM release and EEG-based arousal states indicating that muscle tonus relaxed and task awareness decreased as participants drifted towards sleep (Ogilvie et al., 1989). Sleep onset has also been estimated using active behavioural measures. Ogilvie et al. (1989) used a reaction time switch that was sewn into a squash ball in order to record participants’ responses to auditory tones while falling asleep. Ogilvie et al. found that reaction times were significantly related to EEG arousal states with responses fastest during waking, slower during Stage 1, and the slowest during Stage 2 sleep. Sleep was defined behaviourally as failure to respond to the tone (Ogilvie et al., 1989). MacLean, Arnedt, Biedermann & Knowles (1992) demonstrated that these results could be replicated using vibrotactile stimulation in the place of auditory tones.
The authors reported that failed responses to the stimulation were taken as evidence of entry into sleep.

In addition to the increases in reaction time, which had been documented for many years (Liberson & Liberson, 1966; Morell, 1966), Ogilvie, Simons, Kuderian, MacDonald, & Rustenburg (1991) have also reported sharp increases in EEG synchronization in conjunction with response failure as marking the point of sleep onset. Ogilvie et al. (1991) were also able to replicate the findings of Ogilvie et al. (1989) by reporting that shorter response times (to auditory tones) indicated alert wakefulness while slower responses suggested increasing drowsiness, and failed responses indicated sleep.

The Nightcap

The Nightcap (Mamelak & Hobson, 1989; Ajilore, Stickgold, Rittenhouse, & Hobson, 1993; Pace-Schott, Kaji, Stickgold, & Hobson, 1994) is a minimally invasive, home-based recording system. The cap consists of two sensors attached to a base monitor. The sensors are piezoelectric films that adhere to one eyelid and a mercury switch adhered to the forehead in order to measure eye movements and gross head movements respectively. Each minute, the base monitor records the number of eye and body movements. The cap is highly reliable in distinguishing Wake, NREM, and REM states. One validation study reported that the scoring determined by the Nightcap and gold standard laboratory scoring were in agreement on 87% of the epochs scored for 10 subjects (Ajilore et. al., 1994). Pace-Schott et al. (1994) reported that the Nightcap was reliable in accurately assessing sleep onset latencies in good and poor sleepers. Additionally, the Nightcap has been shown to reliably identify sleep onset according to
changes in perceived sleepiness and the appearance of hypnagogic dream characteristics (Rowley, Stickgold, & Hobson, 1998). Another study aimed at establishing the reliability of the Nightcap in determining sleep onset latency compared the Nightcap to standard polysomnography (PSG). The mean percentage of agreement between the Nightcap and PSG was 93%. The results suggested that the Nightcap could be a beneficial alternative to the PSG technique in the assessment of sleep onset in normal subjects in terms of cost and ease of administration (Cantero, Atienza, Stickgold, & Hobson, 2002).

In general, behavioural measures of sleep and EEG measures of sleep correlate quite well. Cote & Ogilvie (1994) reported an overall agreement between behavioural and EEG measures of 94%. However, when looking specifically at Stage 1, agreement levels dropped dramatically to just below 66%. The authors suggested that Stage 1 was more representative of a transitional period between wakefulness and sleep (Cote & Ogilvie, 1994). Additionally, Ogilvie (1985) found there was a reliable relationship between the active and passive behavioural measurements of impending sleep. However, he also found that the release of the microswitch (passive monitoring) typically occurred in advance of response cessation to the active monitoring (responding to tones). He also noted that this was more of a product of continuous muscle decrement.

**Hori’s 9-stage Continuum**

The need for a clearer picture of brain activity during this sleep onset period (the transition from Waking to Stage 1 to Stage 2 sleep) has been established and a finer group of stages has been devised. Hori, Hayashi, & Morikawa (1994) presented a 9-stage continuum that spans the sleep onset period from wakefulness to the onset of standard
Stage 2 sleep. The increased fidelity is the result of more precise EEG differentiation and increased temporal resolution (i.e. 5-second instead of 30-second epochs). Wakefulness, in the Hori Continuum is represented by Hori-Stage 0. Rechtschaffen & Kales' standard Stage W has been divided into two stages with Hori-Stage 1 (Alpha Wave Train) consisting of alpha wave trains with a minimum amplitude of 20 μV and Hori-Stage 2 (Alpha Wave Intermittent-A) in which the epoch is composed of greater than 50% alpha with a minimum amplitude of 20 μV. Hori's stages from 3 to 8 coincide with a systematic sequence of EEG changes which they detected within Rechtschaffen & Kales' standard Stage 1. Hori-Stage 3 (Alpha Wave Intermittent-B) contains 50% to 0% alpha with amplitudes < 20 μV. Hori-Stage 4 (EEG Flattening) is comprised of suppressed waves of less than 20 μV. Hori-Stage 5 (Ripples) is comprised of low-voltage theta wave activity. Hori-Stage 6 (Vertex Sharp Wave Solitary) is scored as an epoch containing one well-defined vertex sharp wave. Hori-Stage 7 (Vertex Sharp Wave Train or Bursts) follows as an epoch that contains at least two well-defined vertex sharp waves. Hori-Stage 8 (Vertex Sharp Wave and Incomplete Spindles) is observed when an epoch contains at least one well-defined vertex sharp wave and one incomplete spindle with a duration of less than 0.5 seconds. Finally, Hori's Stage 9 (Spindles) coincides with the beginning of Rechtschaffen & Kales' standard Stage 2 wherein an epoch must contain one well-defined spindle at least 0.5 seconds in duration and 20 μV in amplitude (Hori et al., 1994). Figure 1 illustrates Hori's 9-stage continuum visually.
null
Although a more detailed picture of the process of falling asleep has emerged from recent research, so far this understanding has not led to the development of effective detection and alertness devices. An attempt has been made involving the Brock Behavioural Response System (Ogilvie & Wilkinson, 1984). The system, referred to as the 'Sleepscope,' works as a reliable active behavioural monitor of sleep and waking. The device generates a series of random tones that last for a maximum of 5 seconds unless terminated by the user using a hand-held button. Termination of the tone within the 5-second window indicates wakefulness and failure to respond indicates sleep. Although a device such as the Sleepscope is reliably accurate in discriminating sleep from wakefulness it would not be practical to use such a device in order to predict and/or alert
the user of approaching sleep. Further, such a device would not be effective in postponing sleep onset.

The Need for an Alertness-Monitoring Device

According to the Center for National Truck Statistics, nearly 110,000 people are injured and more than 5,000 people are killed each year in the United States in motor vehicle accidents involving commercial trucks. Reports attribute as many as 56% of these accidents to fatigue (CNTS, 1996). However, the prevalence of driver drowsiness in crashes is believed to be greatly under-reported. There is usually little evidence upon which to base a finding that drowsiness was a factor in a crash (McCarrt, Ribner, Pack, & Hammer, 1996). Based on survey responses, McCarrt et al. reported that nearly 55% of drivers had driven while drowsy within the previous year. They also reported that nearly 23% of those surveyed reported that they had fallen asleep at the wheel and that almost 3% had crashed when they fell asleep. Knipling & Wang (1994), using data from the U.S. National Highway Traffic Safety Administration’s General Estimates System’s Fatal Accident Reporting System (FARS), reported that 3.6% of all fatal accidents between 1989 and 1993 had been the result of the driver having fallen asleep. Nearly 30% of all fatal accidents on rural roads can be attributed to the driver falling asleep (Fell, 1994). Of all crashes that were reported and attributed to the driver having fallen asleep, 60% of those crashes had occurred between the hours of 11 p.m. and 7 a.m. The U.S. National Transportation Safety Board (NTSB) has estimated that nearly one-third of heavy truck accident fatalities involve fatigue and that fatigue is implicated in a greater number of fatal heavy truck accidents than alcohol (NTSB, 1990). Considering the prevalence of the
situation, it is alarming and unacceptable that there is such a low level of knowledge concerning driver fatigue when compared to the high level of knowledge of alcohol-related accidents.

McCartt et al. (1996) reported that survey respondents indicated that being drowsy had a greater effect on their ability to drive safely than either adverse weather or having two drinks of alcohol. In addition, 26% of their respondents indicated that they knew someone who had had a crash due to falling asleep at the wheel. Crashes resulting from the driver falling asleep received less attention in traffic safety programs than the influence of alcohol or speeding. Utilizing a database from the Highway Safety Research Center, it was found that the fatality rate of sleep/drowsiness related accidents was of a similar magnitude to that in alcohol-related accidents and that the majority of sleep-related accidents occurred between the hours of midnight and 7 a.m. (Pack, Pack, Rodgman, Cucchiara, Dinges, & Schwab, 1995). During these late night/early morning hours, increases in sleepiness can result from sleep deprivation or inadequate sleep.

Performance lapses, loss of attentional vigilance, and slowed reaction times can all result from excessive sleepiness (Dinges, 1992). When drivers are extremely tired, they will drift into what Dinges refers to as ‘microsleeps’ in which drivers may fall asleep for brief periods of time and may be temporarily unaware of their surroundings, possibly resulting in an accident. Arnedt, Wilde, Munt, & MacLean (2001) reported that for tracking variability, and speed variability while driving, 18.5 and 21 h of wakefulness produced changes of the same magnitude as 0.05 and 0.08% blood alcohol concentration, respectively. The authors also reported that wakefulness prolonged by as little as 3 hours can produce decrements in the ability to maintain speed and road position in a driving
simulator as serious as those found at the legal limits of alcohol consumption. Pack et al. (1995) reported that crashes primarily occurred when the driver drove off the road to the right of left. This was far more common than alcohol-related causes; 78.5% to 48.4% respectively. However, sleep-related accidents more often involved single vehicle crashes than alcohol-related accidents, 77.5% to 50.7%, respectively. Pack et al. comment on previous opinions that there may be many more accidents in which sleepiness plays a part than are reported. Sleepiness may contribute to accidents attributed to loss of attention, and sleep deprivation significantly augments the impaired performance of someone who has been drinking. Thus, excessive sleepiness may play a larger role in causing accidents than the number of actually reported cases would imply. Unfortunately, there is no basic test for police officers to administer in an attempt to distinguish sleep-related, alcohol-related, or sleep/alcohol-related causes of accidents. In a study that examined the sleep of long-haul truck drivers, it was reported that drivers averaged only 4.78 hours of sleep per night over the five-day study period with no one having more than 5.38 hours of sleep on any night. These hours were significantly lower than the estimated 7.1 hours of self-reported ideal hours of required sleep for the drivers (Mitler, Miller, Lipsitz, Walsh, & Wylie, 1997).

It has been consistently shown that psychomotor performance is impaired if sleep is limited to less than five hours for two or more consecutive nights (Carskadon & Dement, 1981; Wilkinson, Edwards, & Haines, 1966). Sleep deprivation of this sort can lead to bouts of inattention and increased error rates. The drivers in their study obtained far less sleep than is required for adequate and safe alertness. Mitler et al. studied 80 male truck drivers and found that 56% had at least 1 six-minute interval of drowsiness while
driving, as judged by analysis of video recordings of their faces and two drivers had one episode of Stage 1 sleep while driving as determined by EEG. Several drivers exhibited electrographic features of drowsiness including slow rolling eye movements and elevated alpha activity. One driver in particular exhibited five episodes of Stage 1 sleep during a 41-minute stretch after he had been driving for 10 hours. The drivers were most susceptible to these sleepiness effects in the late-night hours and early morning. The periods of sleep latency were 19.3 minutes for drivers on steady day shifts compared to just 7.4 minutes for drivers on steady night shifts indicating that those drivers working steady nights were considerably more sleep deprived (Mitler et al, 1997). This level of sleepiness approaches levels of sleepiness deemed pathological in sleep clinics.

**Preventive Measures previously used to counteract sleepiness/drowsiness**

McCartt et al. (1996) found that when drivers were attempting to combat the effects of drowsiness, only 72% actually stopped driving. Just over half of the respondents (51.1%) attempted to change the environment in the car by opening the windows or using the radio, etc. and almost 33% of drivers consumed food or beverages in an attempt to stay awake. Roadside rest areas have been shown to provide an adequate means of allowing drivers to take a break from driving. McCartt et al. reported that 45% of their respondents had stopped at such a rest area in the previous year and nearly 67% of those who stopped reported that the rest helped to combat drowsiness a great deal.

Devices used to combat driver fatigue need to be designed with several ideas in mind. They need to detect drowsiness and provide some sort of warning and, in better scenarios, provide some sort of intervention. However, the detection of drowsiness varies
according to the indicators used for assessment of drowsiness or of impaired performance. Devices need to be designed for maintaining alertness by initiating some form of activation on the part of the user. Since drowsiness reflects lowered arousal due to lack of stimulation, an attention-demanding secondary task may increase the general level of arousal and improve performance (McCartt et al, 1996).

The NOVAalert™ System

The NOVAalert™ system was devised by Nova Interactive Technologies Ltd in 2000. It was designed as an alertness maintenance device for automobile drivers, train engineers, pilots, flight controllers, and anyone who performs tasks in which a significant amount of attention is needed. The NOVAalert™ system is intended as an early warning monitor. The device (approximately the size of a wristwatch) utilizes wrist muscle activity to detect the first lapses in alertness and performance, and aims to identify and reverse the effects of physiological and behavioural performance deficits. The NOVAalert™ is designed to identify signs of performance decrement, drowsiness, and the increasing propensity for impending sleep. The NOVAalert™ is claimed to be capable of temporarily alerting (by vibrations from the Wrist Unit) and arousing the user in order to prevent microsleeps resulting in accidents.

The NOVAalert™ system consists of two functional modes: the NOVAalert Stay-Alert Minder Driver’s System (SAM) and the NOVAalert Task Allocation Monitor Control Room Demo System (TAM). These are different functional modes of the NOVAalert-O-Meter™ (AOM) and accompanying Wrist Unit. The SAM mode is intended for use by drivers and measures isometric/dynamic muscle activity and
administers a psychomotor vigilance test (PVT) when muscle activity decreases below a set baseline (normal, adequate muscle tension). Baselines are set for each individual user when the unit is first placed on the wrist. The PVT is simply the Wrist Unit vibrating. This vibrating is felt on the user’s wrist. The TAM mode is intended for use in a laboratory setting and measures lack of total muscle activity without administering the vigilance test. Information is provided to the researcher about the point(s) at which the PVT would normally be administered.

It is important to provide the user with a secondary task that does not interfere with attentional resources needed to effectively and safely operate a vehicle, train, or plane, etc. With the NOVAalert™, this secondary task involves simply increasing the firmness of the user’s grip on the steering wheel or increasing the finger pressure of one finger on the monitored hand. This type of secondary task is claimed by the manufacturer to provide enough arousal without dividing the user’s attention from his or her primary task of driving the vehicle (NOVA Beta-Site Series User Guide, 2000).

The physical activity needed to perform this secondary task is monitored through wrist muscles responsible for finger activation, primarily the palmarus longus, the flexor carpi radialis, and the brachioradialis muscles. As the operator becomes tired, the muscle activity of the wrist should drop off, compared to their individual baseline. In this instance, the desire to stay awake may be weakening and the operator may experience microsleeps. This condition should be sensed and the wrist unit should then initiate the psychomotor vigilance task (PVT) in the form of a dual pulse vibratory stimulation. The purpose of the PVT is to arouse the person in order to alert him or her of the presence of a potentially dangerous condition. The purpose of the dual vibrations is to analyze the
user's state through the response to the vibrations. It is important for the user to ignore the first pulse and respond (with increased grip of the monitored hand) to the second pulse in order to ensure that the user was not inattentive and startled into responding to the first vibration. A correct response (responding to the second vibration) is compared to the individual, initial baselines (NOVA Beta-Site Series User Guide, 2000). Energy (latency and force) as well as accuracy is evaluated. The PVT is repeated every 10 seconds if the user: 1) responds to the first vibration; 2) correctly responds to the second vibration but with a grip of less firmness than baselines; or 3) fails to respond to the second vibration. If the user is inattentive and extremely fatigued, the unit will also sound an alarm buzzer to suggest that the driver stop immediately.

When the wrist unit is first placed on the user's wrist and turned on, the unit administers the dual pulses (vibrations). At that point, the user is to respond to the second pulse by flexing the fist of the monitored arm; this is done to allow the AOM to determine baseline wrist EMG levels to be compared to the PVT response during the testing mentioned above (NKVA Beta-Site Series User Guide, 2000). It should be noted that it is important to individually set EMG baselines. People will exhibit different levels of muscle tonus. Similarly, changes in muscle tonus and EEG can be expected to vary from person to person during the transition from wakefulness to sleep. For example, Ogilvie et al. (1989) described two participants' results when responding to auditory tones during the Sleep/Wake transition. The first participant responded to all 96 tones presented during EEG-defined wakefulness while the second participant failed to respond to 4 of the 39 tones presented during EEG-defined wakefulness. The first participant failed to respond to only 5 of the 53 tones during Stage 1 and failed to respond to all 23
tones presented during EEG-defined Stage 2 sleep. On the other hand, the second participant missed 27 of the 32 tones presented during Stage 1 and also failed to respond to 33 of the 34 tones presented during Stage 2 sleep (Ogilvie et al., 1989). This example is demonstrative of the fact that different people fall asleep in different ways. For this reason it is important that a device, intended to measure and detect the onset of sleep, be capable of discriminating individual differences. Similarly, a device such as the NOVAalert™ needs to be able to calibrate baseline levels for each individual user.

**Muscle Activity and the Sleep Onset Period**

The changes that occur during the process of falling asleep are becoming more and more clear with regard to EEG changes. Unfortunately, such a vivid depiction does not currently exist for EMG activity. The present study will aid in developing such a picture.

**Muscle Innervation and Control**

In order to properly study the relationship between muscle activity and the sleep onset period, it is important to fully understand how muscles are constructed and how they are controlled.

There are three types of muscles: skeletal, smooth, and cardiac. The muscles of particular interest in the present study are the skeletal muscles; those required for movement. The skeletal muscles are responsible for two primary types of movements: flexion and extension. A flexion movement is the result of the contraction of a flexor
muscle that produces the inward drawing of a limb. The opposite movement, an extension, is produced by the contraction of extensor muscles (Carlson, 1994).

A muscle is composed of thousands of individual muscle fibers. Muscles are connected to bone by tendons. Different muscles are arranged in a reciprocal fashion, such that when one muscle group contracts, the other is extended. These muscle groups are antagonists. Muscles that act together are called synergists. Skeletal muscles consist of two types of muscle fibers: extrafusal and intrafusal. The extrafusal muscle fibers are controlled by axons of the alpha motor neurons. One single axon of an alpha motor neuron serves many extrafusal muscle fibers. The number of muscle fibers served by a single axon varies depending on the precision needed to control a particular muscle. For example, fingers can have a ratio of less than one to ten while leg muscles can be one to several hundred. A motor unit consists of an alpha motor neuron, itsaxon and its associated extrafusal muscle fibers. A contraction of these extrafusal muscle fibers provides the motive force of the muscle. The intrafusal muscle fibers are sensory organs that are served by two axons, one sensory and one motor (Carlson, 1994).

The intrafusal muscle fibres are equipped with sensory endings that are sensitive to stretch applied to the muscle fibre. The intrafusal muscle fibre contraction is caused by the efferent axon of the gamma motor neuron. This contraction is successful in modifying the sensitivity of the fibre’s afferent ending to stretch. The reticular formation controls the activity of the gamma motor system and hence regulates muscle tonus. The reticular formation consists of a large number of nuclei located in the core of the medulla, pons, and midbrain (Galluscio, 1990).
**Muscle Contraction and Regulation**

A neuromuscular junction consists of the synapse between the terminal button of an efferent neuron and the membrane of a muscle fibre. The terminal buttons synapse on motor endplates that are located in grooves along the surface of the muscle fibers.

When an axon fires (produces an action potential), acetylcholine is released by the terminal buttons and causes a depolarization of the postsynaptic membrane. The acetylcholine is then broken down by its enzyme acetylcholine esterase. This specific action potential is referred to as an endplate potential. All of the muscle fibers innervated by that motorneuron respond to the acetylcholine by producing action potentials of their own. These potentials are vastly larger than an excitatory postsynaptic potential in synapses between neurons. In this case, an endplate potential always causes its accompanying muscle fibre to fire, in turn, propagating the potential along its length. This action potential causes a twitch, a contraction of the muscle fibre (Carlson, 1994).

Gates of voltage-dependent calcium channels are opened during this depolarization. These open gates allow calcium ions to enter the cytoplasm. It is this event that triggers the contraction. The calcium acts as a cofactor that permits the myofibrils to extract energy from the adenosine triphosphate (ATP) that is present in the cytoplasm.

A single muscle fibre consists of a bundle of myofibrils, each of which consists of overlapping strands of actin and myosin. The small protrusions on the myosin filaments are referred to as myosin cross bridges. These protrusions interact with the actin filaments and produce muscle contractions. The myosin cross bridges alternately attach to the actin strands, bend in one direction, detach, bend back, reattach to the actin at a point further
down the strand and so on. This action is often referred to as ‘rowing’ along the actin filaments (Galluscio, 1990). This ratchet-like action causes the myosin and actin strands to slide across each other in small steps, shortening the overall length of the muscle. The number of alpha neurons involved and their firing rate modulates the amount and the force of the contraction. The contraction of muscles takes energy. The energy comes from the ATP, which has high-energy phosphate bonds. When the ATP attaches to the myosin protrusions, it breaks down to adenosine diphosphate, releasing the energy from one of its phosphate bonds (Galluscio, 1990).

A single impulse of a motor neuron produces many twitches in a muscle fibre. Naturally, the effects of the twitch last substantially longer than the action potential itself. This is due to the elasticity of the muscle and the time required to rid the cell of calcium. An example of this neural plasticity is post-tetanic potentiation. When a rapid series of action potentials are induced in a motor nerve, the neuromuscular junctions are altered for a period so that subsequent single action potentials cause a stronger endplate potential in the muscle. This potentiation is caused by a build up in calcium ions in the presynaptic terminal leading to the release of more acetylcholine (Rosenzweig, Leiman, & Breedlove, 1996).

The strength of a muscular contraction is determined by the average rate of firing of the motor units involved in the contraction. The intrafusal muscle fibers contain sensory endings that are sensitive to stretch. These sensory endings are called muscle spindles. The intrafusal muscle fibers, arranged in parallel with the extrafusal muscle fibers, are stretched when the muscle lengthens and are relaxed when the muscle shortens. Despite the fact that these neurons serve as stretch receptors, they also provide
information as to the length of muscles. Muscle spindles consist of both afferent and efferent elements. There are two kinds of receptors in the muscle spindle, primary sensory endings and secondary sensory endings. The primary ending spirals around an area called the central region of the intrafusal fibre. The secondary fibres terminate near the ends of the spindle. When a muscle is stretched, the muscle spindle would also stretch and the deformation of the endings on the spindle would send up nerve impulses in the afferent fibers. These afferents inform the spinal cord, and the spinal cord then informs the brain about the degree of muscle stretch (Rosenzweig et al, 1996).

**Muscle Control**

The primary motor cortex lies on the precentral gyrus just rostral to the central sulcus. Penfield and Rasmussen (1950) were among the first to show that electrical stimulation of cortical tissue causes movements of particular parts of the human body.

The main cortical input into the primary motor cortex is the frontal association cortex. The frontal association cortex is located just rostral to the primary motor cortex. Most complex behaviours are planned here. The frontal association cortex receives axons from association areas of the occipital, parietal, and temporal cortex. The primary motor cortex also receives projections from the adjacent primary somatosensory cortex, located just across the central sulcus. Neurons in the primary somatosensory cortex that respond to stimuli applied to a particular part of the body send axons to neurons in the primary motor cortex that move muscles in the same part of the body. This organization appears to provide rapid feedback to the motor system during manipulation of objects (Evarts, 1974).
Hypotheses

The foregoing sections provide a detailed summary of how muscles work and how they are controlled. It is important to understand how muscles function before studies on their correlates can be examined. The EEG correlates of sleepiness and the Sleep Onset Period were also reviewed. The present study will examine both theoretical aspects and practical implications of evaluating EMG (muscle activity) during the Sleep Onset Period (SOP).

Theoretical Predictions

Very little previous research has focused on the specific EMG changes that are experienced throughout the transition from wakefulness to sleep and to the author’s knowledge, there are no studies examining EMG changes as a function of Hori’s nine stages of EEG changes across the SOP. Unfortunately, the general consensus has been that the decline in muscle activity during sleep (and sleep onset in particular) was too gradual to be of much interest or use. It has been reported that the only reliable changes in muscle activity are associated with REM sleep; even then it was only observed as a marked decrease in EMG activity (Jacobson, Kales, Lehmann, & Hoedemaker, 1964). However, Hadjiyannakis, Ogilvie, Alloway, and Shapiro (1997) did examine EMG during the transition from Stage 2 into REM sleep. The authors quantified EMG by using power spectral analysis. Their purpose was to determine whether decreases in EMG activity would correlate with significant differences in EEG power immediately before and after the decline in EMG activity (Hadjiyannakis et al., 1997). The authors reported that delta power decreased consistently during a 2-minute period centred around a drop in
EMG activity. For their purposes, a drop in EMG activity was demonstrated by a decrease in tonic submental EMG of 30% or more from one 20-second epoch to another. This decrease had to remain stable for at least 1 minute following the initial drop. In addition, Hadjiyannakis et al. examined correlations among Delta, Theta, Alpha, and Sigma during periods of either high EMG activity or low EMG activity. During high EMG activity they reported no significant relationships. However, during periods of low EMG activity, they reported significant positive correlations between Alpha power, Sigma power and EMG. Unfortunately, the EMG signal used in the Hadjiyannakis et al. study was sampled at a rate of only 102.4Hz, which, as observed by the authors, was lower than optimal. A much higher rate of sampling would provide a more detailed account of EMG activity. The present study examined EMG sampled at 1000 Hz, providing a fine-tuned analysis of EMG activity. The findings reported by Hadjiyannakis et al. suggest that there exists a close relationship between EEG and EMG as REM sleep is entered. The present study should provide a detailed analysis of muscle activity that may help reveal this underlying mechanism. The theoretical aspect of the present study should provide a greater understanding of the decline in muscle activity during the descent through Hori’s 9 stages of sleep onset using FFT analyses to quantify power changes from stage to stage. It is of interest to determine the relationship between EEG activity and EMG activity during the sleep onset process. This determination will be made by correlating changes in EMG with changes in EEG throughout each of Hori’s 9 stages. These correlations allow us to determine which Hori stages contained the largest and most frequent drops in EMG activity. It was predicted that decreases in EMG levels would relate to decreases in both Alpha and Beta activities since the disappearance of
these waveforms is indicative of decreases in attention and impending sleep onset. The
decline in muscle activity can also be compared to more subjective measures of
sleepiness such as the Stanford Sleepiness Scale (SSS) (Hoddes, Zarcone, Smythe,
Phillips, & Dement, 1973). It was predicted that the decline in EMG activity would
correlate negatively with higher ratings on the SSS: as participants report feeling 'more
sleepy', their accompanying EMG should be in decline.

Practical Applications

There is clearly a need for alertness monitoring devices as evidenced by the
alarming statistics on motor vehicle crashes attributed to fatigue that were described
earlier. The present study examined the effectiveness and reliability of a commercially
developed device designed to provide alertness monitoring. It is important to test the
reliability of the NOVAAlert™ in predicting levels of sleepiness that are confirmed by
subjective measures of sleepiness and by EMG and EEG measures of sleepiness used in
this study. The practical aspect of the present study examined these relationships to
ensure that the detection of sleepiness (as indicated by the NOVAAlert™) occurs in a
predictable manner in accordance with the EMG analysis during the Hori's stages that
indicate sleepiness and decreased awareness. It is important to determine whether muscle
activity/tension drops off in the middle stages in the S/W continuum only to rise again in
later stages leading to inaccurate sleepiness judgments made by the NOVAAlert™. It is of
practical importance to relate the changes in EMG to the Hori stages to ascertain whether
or not these changes are gradual or relatively abrupt (occur early or late in the SOP) and
whether they remain low or vary considerably. These characteristics could greatly affect
the usefulness of monitoring EMG as a 'sleepiness detector'. FFT analyses of EMG changes will be necessary to compare changes in EMG as indicated by the NOVAAlert™ system. This comparison is important since the administration of the PVT is dependent upon the NOVAAlert™ being sensitive to significant changes in EMG activity.

Pilot work suggested that there are indeed quantifiable changes in EMG activity during sleepiness that may be correlated with EEG changes. This work should lead to both a theoretical advancement of our understanding of muscle activity as sleep approaches and to the assessment of a potentially useful alerting device.

Method

Participants

Participants were 12 female undergraduate students at Brock University. Participants were either paid $15.00 per night or were given participation credit for their First-Year Psychology course. The mean age of the participants was 22 with a range of 18 to 42 years. Only female participants were used because of an insufficient number of male volunteers. By studying only one gender, the present study does not provide further insight into the role of gender on the process of falling asleep or the potential alerting aspects of the NOVAAlert™ system.

Sleep Laboratory and Polysomnographic Data Acquisition Devices

The Brock University sleep laboratory was used for the collection of all data. The laboratory contains two bedrooms, a monitoring control room, and a bathroom. Each bedroom is electrically shielded, sound attenuated, and light and temperature controlled.
Each bedroom measures approximately 3 x 3 meters in size. The mean temperature at ‘lights off’ was 21.7 with a range of 19.1 to 24.0 and the mean temperature at ‘lights on’ was 21.7 and ranged from 17.0 to 24.6. An infrared video camera recorded the participants continuously throughout the night with the information displayed in the control room. Video recordings were only used as a safety device and tapes were erased with the recording of the following participant. Two-way communication was available via microphones and speakers placed in each room.

Polysomnographic data including electroencephalogram (EEG), electrooculogram (EOG), and electromyogram (EMG) were collected from each participant for three consecutive nights. Gold standard electrodes were used for all polysomnographic measures.

EEG and EOG data was sampled at 250 Hz and EMG data was sampled at 1000 Hz. Data were recorded on the hard drive of a Pentium III, 450 MHz computer. The data was copied to compact disc for storage. Stellate Systems Harmonie™ 5.0 software was used to record, score, and analyze the data. PSG records were scored in 5-second epochs according to Hori’s (1994) stage scoring paradigm. Using small segments of EEG and EMG recordings allows for detection of fine changes in muscle and brain activity.

NOVAAlert™

The NOVAAlert™ was developed and constructed by Interactive Technologies Ltd, a division of Atlas Researches Ltd, and is composed of 2 parts, the Wrist Unit (WU) and the Alert-O-Meter (AOM). The wrist unit is worn by the research participant while the Alert-O-Meter remains in the control room with the researcher. The WU includes dry
muscle activity sensors, a skin vibrator/stimulator, one 3.6V Lithium battery, an RF wireless transmitter (approx. 10 meter range), muscle bio-amplifiers, filters, a digital signal processor and CPU/algorithm memory, a LED indicator, and an ON/OFF push button. The AOM includes an RF receiver, a decoder, a Battery Status LED, a User Status LED, one 9V alkaline battery, an ON/OFF switch, and a buzzer.

Procedure

Participant recruitment

Participants were recruited via sign-up sheets (Appendix C) posted around the Psychology Department. Students were required to write their student identification number and phone number or e-mail address on the sheet. For reasons of safety and confidentiality, students did not write their names on the sign-up sheet. Names of potential participants were obtained from the TA’s office prior to phoning. Potential participants were contacted and appointments for interviews and lab tours were arranged.
During the initial interview, approximately one week before the experiment, participants were given a tour of the sleep lab and the procedure for over-night recording was fully explained. Participants were given a Letter of Information (Appendix D) and two copies of an Informed Consent Form (Appendix E), one of which they signed and returned to the experimenter and one that they kept for themselves.

**Overnight sessions**

Participants were scheduled two at a time for three consecutive nights. The first night served as an adaptation night in order to account for possible first-night effects (Agnew, Webb, & Williams, 1966). The other two nights served as the experimental nights.

During the second and third experimental nights, each sleeper was allowed 5 twenty-minute Nap opportunities. This was done in order to increase the number of sleep entries while also increasing the need for sleep. Nap opportunities were terminated after 20 minutes. Participants were kept awake for 10 minutes between each of the nap opportunities in order to wake up, get a drink, go to the bathroom, etc. The inclusion of the vibrotactile stimulation (administration of the PVT) was randomly alternated between the two experimental nights. The reasons for separating the protocol into two parts were as follows: On the no-stimulation night the participant was not wearing the alerting device. During these nights we tried to get a clear picture of EMG changes throughout each of Hori’s 9 stages and into deep sleep. This allowed us to determine the relationship between EEG and EMG activity at sleep onset. This night would allow examination into whether reliable changes in EMG could be correlated with reliable changes in EEG
patterns. Namely, does the drop in EMG correlate with the disappearance of Alpha and Beta EEG activity? If so, does it also correlate significantly with other EEG waveform patterns? On the stimulation night, participants were wearing the alerting device. During these nights we again tried to gain a clearer idea of the EMG changes accompanying the descent from wakefulness to sleep in addition to assessing: 1) when the NOVAAlert algorithm identified sleepiness and 2) the effectiveness of the NOVAAlert in reversing the move towards sleep.

When a participant was wearing the NOVAAlert, the AOM (in the control room with the researcher) displayed a series of LED signals, each of which was annotated during the overnight recordings on the Harmonie system. A solid green light indicated acceptable muscle tension (i.e. compatible with wakefulness) when compared to each individual’s baseline. A flashing green light indicated the first warning sign of reduced muscle tension. A flashing red light indicated the beginning of the second warning period and a solid red light accompanied by the AOM alarm (an audible alarm) indicated a failed response to the PVT. These markers were stored in memory for off-line comparison with EMG and EEG changes.

Participants were asked to arrive between 1 – 1.5 hours before their regular bedtime so that they could be prepared for recording. Upon arriving at the lab, participants changed into clothes that they planned to sleep in. The primary investigator and another female researcher conducted the electrode hook-up. The following EEG electrode sites were used from the International 10-20 System: F7, F3, Fz, F4, F8, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, O1, O2, A1, and A2. Each of these sites was referenced to linked mastoids (A1 + A2), and a ground electrode was placed in the
middle of the forehead. The two chin EMG electrodes were placed over the submentalis muscle in accordance with common polysomnography (PSG) practice; the two wrist EMG electrodes were placed over the group of muscles that included the palmarus longus, the flexor carpi radialis, and the brachioradialis muscles (the same muscle group utilized by the NOVAalert™), and the EOG electrodes were placed on the outer right and left canthi in a diagonal fashion. For sleep stage scoring generally only C3 or C4 is needed, however, it was thought to be important to examine coherence and topographical changes in relation to EMG drops and NOVAalert™ signals. Both coherence and topographical measurement requires that multiple EEG sites be sampled.

In preparation for each overnight data collection session, the amplifiers were calibrated, and technical instruments were tested and set; for example, intercom system checked, VCR and computer set to record. Once the participants were hooked up, electrode impedances were checked. Any electrodes with unacceptable impedances (>10 kΩ for EMG and >5 kΩ for EEG) were removed and reapplied. Once the participants were in bed, a bio-calibration procedure was performed in order to obtain EEG and EMG baselines for comparison with experimental recordings. Participants were asked to lie relaxed on the bed with their eyes open for at least 30 seconds followed by lying relaxed with their eyes closed for an additional 30 seconds. Participants were then asked to look left-to-right several times, up-and-down several times, and to blink several times. Participants were also asked to grind their teeth, cough, swallow, and to flex the arm that was equipped with the NOVAalert™. Upon completion of the bio-calibration exercises, the lights were turned off and one of the two experimental conditions was carried out. Participants were informed that the sessions would be ended after 20 minutes regardless
of whether they had fallen asleep or not and that they then would have 10 minutes to ‘wake up.’ One the night with the NOVAalert™ monitoring, participants were asked to hold a small sensor between their thumb and forefinger of the monitored hand.

Once all of the 5 Nap sessions were completed, the participants were allowed to sleep uninterrupted until morning. Participants were awakened at 6 o’clock in the morning to conclude the experimental night; at this time the electrodes were removed. The participants were allowed to clean-up in the laboratory bathroom or head directly home.

**Questionnaires**

*Stimulus Evaluation Questionnaire (SEQ).* The SEQ (see Appendix A), constructed by the author, was used to assess the participant’s response to the presented stimuli. The stimulus in question was the psychomotor vigilance test (PVT) administered by the Wrist Unit. The questions asked on the SEQ included: “How awake/sleepy did you feel when you felt the vibration?”; “How awake/sleepy did you feel after the vibration was presented?”; “How effective would this device be in keeping a sleepy driver awake?”; and “How effective would this device be in warning a driver to take precautions to avoid dangerous levels of drowsiness/sleepiness?”. Each question was rated on a 7-point Likert scale. The participant responded by indicating a rating between 1-“Alert and wide awake” and 7-“Very tired, hard to stay awake.” The following forced-choice question was also asked: “Which did you feel was more effective in alerting/arousing you, the PVT or the AOM alarm?” The alarm in the control room was clearly audible through the laboratory intercom system.
Stanford Sleepiness Scale (SSS). The SSS, developed by Hoddes, Zarcone, Smythe, Phillips, and Dement in 1973, is a 7-point Likert scale designed to assess levels of sleepiness. The SSS has been shown to have a relationship with the S/W transition (Carskadon & Dement, 1977). The SSS correlates well with behavioural and physiological variables used in identifying the transition from wakefulness to sleep in that subjective sleepiness increases as participants move from Waking to Stage 1 (Ogilvie et al, 1989). See Appendix B for full scale.

Analyses

On the nights without vibrotactile stimulation, Hori's 9-stage scoring system was used to more precisely define the relationship between EEG and EMG activity. Each record was divided into 5-second epochs with each visually examined by the author and labelled as one of the 9 stages in Hori’s continuum. Reliability was ensured by comparing random samples of scoring with 3 researchers experienced in scoring according to Hori’s system. EEG analysis took two forms: 1) Hori scoring and 2) FFT analysis using 6 EEG frequency bins. Analysis of Variance (ANOVAs) tests were performed on each different FFT frequency to see how they changed through the 9 Hori stages. In addition, correlations were calculated to determine which changes in EEG measures correlate best with changes in EMG. FFT analyses of EMG changes were also used to obtain a total power measure for any timeframe (5-second windows were used in accordance with Hori’s 9-Stage scoring system). This power figure was also used to compare changes in EMG as indicated by the NOVAAlert™ system. This comparison is important because the
NOVAert™ system must be sensitive to changes in EMG activity in order to effectively administer the alerting task (PVT).

On the nights with psychomotor vigilance test (PVT), we again looked at EMG changes throughout the S/W process as well as the Hori stage in which the NOVAert™ algorithm initiated the stimulus, and studied both the rate at which the NOVAert™ reversed the sleepiness and the stage or sequence of stages through which the person ascended. Examining the percentage of trials in which the NOVAert™ system produced the desired alerting response assessed the reliability of the NOVAert™ system. This can be looked at as a function of time of night and practice to make sure people didn't habituate to the stimulus and then sleep through it.

Hori's 9-stage scoring system was again used to more precisely identify the EEG state in which the NOVAert™ system activated the PVT. This was accomplished by observing which of Hori's stages had the greatest number of Alert-O-Meter™ warnings. This observation allowed us to examine those stages that saw the largest and most frequent drops in EMG activity. An effective monitoring system should have a predictable pattern of detecting EMG drops with very little activation early on followed by a sharp increase in the number of warnings and levelling off with no subsequent decreases. It is important to ensure that muscle activity/tension does not drop off in the middle stages in the S/W continuum and rise again in later stages that would, in a sense, outsmart or invalidate the NOVAert™ system.
Results

Analyses were divided into two experimental nights. First, data were analyzed for the night when participants were given 5, twenty-minute opportunities to fall asleep. Each session was followed by a period of 10 minutes of wakefulness before allowing the next twenty-minute sleep opportunity. This night was referred to as the ‘EMG Night.’ Second, data were analyzed for the night when participants were required to wear and respond to PVTs administered by the NOVAAlert™ system again during 5, twenty-minute sessions. Ten-minute breaks between each Nap were used again on the second night to maintain conformity with the first night. These ten-minute breaks were used to re-establish EMG baselines for each participant. This night was referred to as the ‘Nova Night.’ Only the Hori Stages that were exhibited by all of the participants were used in the following analyses. This resulted in the exclusion of Hori Stage 1. Stage 1 requires a continuous train of Alpha pattern EEG throughout an entire 5-second epoch. Since there were not a substantial number of such epochs in all of the participants, Hori Stage 1 was dropped from the following analyses. With the inclusion of Wakefulness, termed Hori Stage 0, the number of Hori Stages in the following analyses remains at nine. Inter-rater reliability was satisfied by comparing random samples of scored epochs from the author with epochs scored by 3 experienced researchers with experience using the Hori system.
EMG Night

In order to examine changes in EMG and EEG across the Sleep Onset Period, each was broken into smaller parts (EMG into Chin EMG and Wrist EMG and EEG into 6 frequency bins), analysed individually and then compared together.

Chin EMG

Each of the 5 twenty-minute nap sessions was divided into, and scored, in 5-second epochs in accordance with Hori’s scoring guidelines (see Figure 1). Each 5-second epoch was visually judged by the author and labelled as one of Hori’s stages and then grouped with other similar epochs. FFT analyses were then conducted to obtain power figures representing the amount of muscle activity exerted during a particular stage as well as mapping the changes in EMG throughout the stages.

A one-way, repeated-measures ANOVA was conducted with the independent variable being the nine Hori stages and the dependent variable the power of the Chin EMG. As predicted, there was a significant main effect of Hori Stage for Chin EMG, $F(8,88) = 2.73, p = .01$, indicating that Chin EMG declined significantly as individuals progressed through the Hori stages during sleep onset. Figure 2 illustrates the decline in Chin EMG across each of Hori’s stages.
There were no significant sequential stage differences (i.e. H2 did not differ from H3 and H5 did not differ from H6, etc.). Although there was an overall effect of stage, no two successive stages differed significantly. However, there were some significant stage differences in Chin EMG throughout the SOP. Hori Stage 0 (Wake) was significantly different from Stage 5, $t(11) = 2.98, p = .012$, Stage 7, $t(11) = 3.31, p = .008$, Stage 8, $t(11) = 3.38, p = .007$, and Stage 9, $t(11) = 3.31, p = .008$. Hori Stage 2 was different from Stage 7, $t(11) = 2.32, p = .049$, Stage 8, $t(11) = 2.76, p = .025$, and Stage 9, $t(11) = 2.56, p = .033$. Finally, Hori Stage 3 was different from Stage 5, $t(11) = 2.44, p = .032$, Stage 7, $t(11) = 3.30, p = .008$, Stage 8, $t(11) = 3.49, p = .006$, and Stage 9, $t(11) = 3.36, p = .007$. After Hori Stage 3 there were no more significant differences among the stages. This finding indicated that there existed a significant decline in Chin muscle activity following the progression through Hori Stage 3. Following Stage 3, Chin EMG activity
remained at a decreased level without any significant changes throughout the remainder of the Sleep Onset Process.

**Wrist EMG**

A one-way, repeated-measures ANOVA was conducted with the independent variable being the nine Hori stages and the dependent variable the power of the Wrist EMG. As predicted, there was a significant main effect of Stage on Wrist EMG, $F(8,88) = 2.82, p = .008$, indicating that Wrist EMG declined significantly as participants progressed through the Sleep Onset Period. Figure 3 illustrates the decline in Wrist EMG during the SOP.

![Figure 3](image.png)

Hori Stage 0 (Wake) was the only stage that differed significantly from other stages. Refer to Table 1 for a summary of stage differences with H0 with respect to Wrist EMG.
Table 1
Summary table for differences between H0 and all other stages (Wrist EMG)

<table>
<thead>
<tr>
<th>Stage</th>
<th>P</th>
<th>Stage</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>H2</td>
<td>.251</td>
<td>H6</td>
<td>.176</td>
</tr>
<tr>
<td>H3</td>
<td>.052</td>
<td>H7</td>
<td>.013</td>
</tr>
<tr>
<td>H4</td>
<td>.065</td>
<td>H8</td>
<td>.016</td>
</tr>
<tr>
<td>H5</td>
<td>.022</td>
<td>H9</td>
<td>.013</td>
</tr>
</tbody>
</table>

Wrist EMG activity demonstrated a significant decline compared to wakefulness when participants entered and progressed through Hori’s Stage 3 and beyond (excluding a brief rise in activity associated with Hori Stage 6). This significant level of decline differed from that found significant for Chin EMG which reached significance following Hori Stage 3. This finding indicated that fatigue effects were more quickly identified in Wrist EMG than in Chin EMG activity levels. Chin EMG activity was used in this study since this is the standard measure of muscle activity used in clinical and research polysomnography and is clearly outlined in the International 10-20 System.

**EEG Activity**

EEG activity was divided into 6 frequency bins: Delta (1.95 – 3.91Hz), Theta (3.91 – 7.81Hz), Alpha1 (7.81 – 10.74Hz), Alpha2 (10.74 – 13.67Hz), Sigma (13.67 – 17.58Hz), and Beta (17.58 – 29.69Hz) while EMG activity was summed over 29.69 – 495.0Hz. Analyses were conducted to determine whether changes, both within and across frequency bins, occurred throughout Hori’s stages.

Each of the five, 20-minute segments was divided into, and scored, in 5-second epochs in accordance with Hori scoring guidelines. Each 5-second epoch was labelled as one of the Hori stages and then grouped with other epochs similarly scored. FFT analyses were then conducted to obtain power figures for each frequency bin (including EMG) for
each Hori stage. A series of ANOVAs were performed in order to determine time-of-night effects, differences in brain topography and frequency differences.

**Overall Total EEG Power**

A 6 X 9 ANOVA was calculated to examine changes in Total EEG Power on the EMG, or undisrupted, night. The 6 X 9 ANOVA represented an examination of Frequency Bin (6 levels including Delta, Theta, Alpha1, Alpha2, Sigma, and Beta) and Hori Stage (9 levels; H0 through H9). There was an overall main effect of Frequency Bin, F(5,55) = 60.95, p < .001 indicating that there were enormously significant differences among all of the frequency bins. This effect may be skewed by the particularly large increase in Delta activity late in Hori’s continuum (refer to Figure 4). There was also a main effect of Hori Stage, F(8,88) = 11.84, p < .001 indicating that total EEG power changed across the course of the Sleep Onset Period. In addition, the Frequency Bin by Hori Stage interaction was significant, F(40,440) = 9.81, p < .001 indicating that as individuals progressed through the stages of sleep onset, some frequency bins increased in power while others decreased (or remained relatively unchanged) in power.

Figure 4 shows EEG power for each of the 6 frequency bins across each of the Hori stages. The large increase in Delta activity commencing at Hori Stage 7 makes observable changes in the other bins difficult to interpret. For this reason, two separate graphs are presented. Delta activity is easily observable from Figure 4 while a clearer picture of the remaining 5 frequency bins can be seen in Figure 5.
Each Frequency Bin was then examined more precisely by conducting six additional analyses (one for each standard frequency band) in which changes over Hori Stage (9 levels) and Brain Site (5 regions) were examined. EEG activity from all 17 sites was grouped into 5 regions representing the major areas of the brain. The Frontal region consisted of the F3, F4, Fz, F7, and F8 sites. The Central region was comprised of the C3,
C4, and Cz sites. The Temporal region consisted of the T3, T4, T5, and T6 sites. The Parietal region included the sites P3, P4, and Pz while the Occipital region included the O1 and O2 sites. Additional analyses were conducted to determine whether there existed different changes in Frequency across Hori Stage with respect to brain Laterality. Laterality analyses involved grouping EEG activity into 3 regions: Left (F3, F7, C3, T3, T5, P3, and O1), Right (F4, F8, C4, T4, T6, P4, and O2), and Midline (Fz, Cz, and Pz).

**Delta Power**

The 6 X 9 ANOVA (Frequency Bin X Hori Stage) conducted and illustrated previously, in Figure 4, revealed that there was a significant main effect of Hori Stage, $F(8,88) = 10.32, p < .001$, indicating that Delta power increased over the course of the Sleep Onset Period.

A 5 X 9 ANOVA comparing Brain Site (Frontal, Central, Temporal, Parietal, and Occipital) with Hori Stage revealed a significant main effect of Brain Site, $F(4,44) = 97.64, p < .001$, indicating that Delta power increased significantly more in some brain areas than in others. Referring to Figure 6, it can be seen that Delta power increased in Frontal, Central, and Parietal areas more dramatically than in Temporal and Occipital areas. There was also a significant interaction between Hori Stage and Brain Site, $F(32,352) = 8.07, p < .001$ indicating that the differences between Delta activity in each of the brain regions became more pronounced with the emergence of the onset of Hori stages 7 and 8.
In order to determine whether changes in Delta activity were similar for Midline sites in comparison to either the Left or Right hemisphere, a 3 X 9 ANOVA examined Laterality (Left, Right, and Midline) and Hori Stage. This analysis revealed a significant main effect of Laterality, $F(2,22) = 51.98$, $p < .001$, indicating that there was a greater increase in Delta power along the Midline compared to either the Left or Right hemispheres. This Midline dominance emerges significantly with the onset of Hori Stage 7. Figure 7 illustrates Delta power laterality.
Theta Power

The 6 X 9 ANOVA (Frequency Bin X Hori Stage) conducted and illustrated previously, in Figure 5 (EEG Power Across Hori Stages (excluding Delta)), revealed there was a significant main effect of Hori Stage, $F(8,88) = 14.70$, $p < .001$, indicating that Theta power increased as individuals progressed through the Sleep Onset Period.

A 5 X 9 ANOVA comparing Brain Site (Frontal, Central, Temporal, Parietal, and Occipital) with Hori Stage revealed a main effect of Brain Site, $F(4,44) = 34.68$, $p < .001$, indicating that Theta power increased more in some brain areas than in others. There was also a significant interaction between Stage and Site, $F(32,352) = 9.39$, $p < .001$.

Referring to Figure 8, it can be seen that Theta power was higher in Frontal, Central, and Parietal areas compared to Temporal and Occipital areas. This difference emerged with the onset of Hori Stage 7.

A 3 X 9 (Laterality X Hori Stage) ANOVA revealed there was a significant main effect for Laterality, $F(2,22) = 46.02$, $p < .001$, indicating that there was a significantly
greater increase in Theta power along the Midline compared to either the Left or Right hemispheres. Figure 9 illustrates Theta power laterality. There it can be seen that this Midline effect grows stronger during the latter stages of Hori Stages.

![Figure 9](image.png)

**Figure 9**
Theta power laterality

**Alpha1 Power**

As shown in Figure 5, which represents the initial 6 (Frequency Bin) X 9 (Hori Stage) ANOVA, there was no significant main effect of Hori Stage for Alpha1, F(8,88) = 1.53, p = .13. However, a 5 (Brain Site) X 9 (Hori Stage) ANOVA reveals that there was a significant main effect of Brain Site, F(4,44) = 34.68, p < .001. There was no significant Hori Stage by Brain Site interaction, F(32,352) = 0.99, p=.21. Figure 10 illustrates the changes in Alpha1 power across the SOP. There it can be seen that site differences were most pronounced early (Hori Stages 0 to 3) and then again later (Hori Stages 7 to 9) in the SOP.
Figure 10
Alpha1 power across Hori stages as a function of brain region

A 3 (Laterality) X 9 (Hori Stage) ANOVA revealed that there was a significant main effect of Laterality, $F(2,22) = 8.23$, $p < .01$, indicating that there was a greater increase in Alpha1 power along the Midline sites compared to either the Left or Right hemispheres. Figure 11 illustrates Alpha1 power laterality. There it can again be seen that these site differences are more pronounced early on (Hori Stages 0 to 3) and appear again later (Hori Stages 7 to 9) in the SOP.

Figure 11
Alpha1 power laterality
Alpha2 Power

As shown in Figure 5, which represents the initial 6 (Frequency Bin) X 9 (Hori Stage) ANOVA, there was a significant main effect of Stage, $F(8,88) = 5.81$, $p < .001$, indicating that, as with Alpha1, Alpha2 power decreased as individuals progressed through the Sleep Onset Period.

A 5 (Brain Site) X 9 (Hori Stage) ANOVA revealed there was no significant main effect of Brain Site, $F(4,44) = 1.54$, $p = .26$. However, there was a significant interaction between Hori Stage and Brain Site, $F(32,352) = 6.61$, $p < .001$. Figure 12 illustrates that Alpha2 power is greater during the first third and final third of the sleep onset process and that, as for Alpha1, site by stage differences were most apparent early (Hori Stages 0 to 3) and late (Hori Stages 7 to 9) in the SOP.

Figure 12
Alpha2 power across Hori stages as a function of brain region

A 3 ( Laterality) X 9 (Hori Stage) ANOVA showed that there was a significant main effect of Laterality, $F(2,22) = 21.28$, $p < .001$, indicating that there was a greater
increase in Alpha2 power along the Midline sites compared to either the Left or Right hemispheres. Figure 13 illustrates Alpha2 power laterality and shows that its effect was most dramatic in Hori Stages 7 through 9.

![Figure 13](image)

**Figure 13**
Alpha2 power laterality

**Sigma Power**

There was a significant main effect of Hori Stage, \( F(8,88) = 9.51, p < .001 \), indicating that Sigma power increased as individuals progressed through the Sleep Onset Period.

There was also a significant main effect of Brain Site, \( F(4,44) = 7.32, p < .001 \), indicating that Sigma power was greater in Central and Parietal areas compared to all others. There was a significant interaction between Hori Stage and Brain Site, \( F(32,352) = 9.56, p < .001 \). Figure 14 illustrates that Sigma power is greater during the final third of the sleep onset process for only a few of the brain areas (Parietal and Central) and not for the others.
There was a significant main effect of Laterality, $F(2,22) = 18.23$, $p < .001$, indicating that there was a greater increase in Sigma power along the midline compared to either the left or right hemispheres. Figure 15 shows that the Midline effect is particularly prominent late in the SOP (Hori Stages 7 to 9).
Beta Power

The initial 6 (Frequency Bin) X 9 (Hori Stage) ANOVA revealed no significant main effect of Hori Stage for Beta power, $F(8,88) = 1.98, p = .17$. There was also no significant main effect of Brain Site found in the 5 (Brain Site) X 9 (Hori Stage) ANOVA, $F(4,44) = 2.09, p = .12$. Nor was the interaction between Hori Stage and Brain Site significant, $F(32,352) = 0.32, p = .41$. Figure 16 illustrates that Beta power remains relatively unchanged across the 5 major regions of the brain throughout the Sleep Onset Period and while Beta appears to decrease from Hori Stage 2 to Stage 4, it does not do so in a significant manner.

Figure 16
Beta power across Hori stages as a function of brain area

![Graph of Beta power across Hori stages as a function of brain area]

However, the 3 X 9 (Laterality X Hori Stage) ANOVA showed that there was a significant main effect of Laterality, $F(2,22) = 8.95, p < .001$, indicating that there was greater Beta activity along the Midline sites compared to either the Left or Right...
hemispheres. Figure 17 illustrates this effect and shows it to be consistent across all Hori Stages.

![Figure 17: Beta power laterality](image)

The foregoing results illustrate a strong relationship between EMG and EEG changes and the Sleep Onset Period. Both Chin EMG and Wrist EMG showed significant decreases in observed power across the SOP as participants traversed the 9 stages of Hori’s continuum. Nearly all Frequency Bins, with the exception of Beta power, demonstrated a significant relationship with the progression through the Sleep Onset Period. While the changes in Alpha1 power were not overall significant with respect to Hori Stage, there was definitely an observable pattern in that Alpha1 decreased through the middle stages of Hori’s continuum only to rise again towards the end of the SOP. Again, in nearly all Frequency Bins, with the exception of Alpha2 and Beta, there existed a strong relationship between Hori Stage and power observed within a particular region of the brain. Delta, Theta, and Alpha1 power increased more prominently in the Frontal, Central, and Parietal regions compared to the Temporal and Occipital regions while
Sigma power increased more prominently in the Central and Parietal regions than observed in the Frontal, Temporal, and Occipital regions. Alpha2 and Beta power showed no significant changes with relation to Brain Site across the Hori continuum. In each Frequency Bin there existed a strong effect of Laterality in that observed power was consistently strong along the Midline of the brain as opposed to either the Left or Right hemispheres of the brain. Additionally, this midline effect grew stronger towards the end of the SOP for each of the Delta, Theta, Alpha1, Alpha2, and Sigma frequency bins.

**NOVA Night**

In order to assess the effectiveness of the NOVAlerf™ system as an alerting device, participants were required to wear the NOVAlerf™ wrist unit for 5, twenty-minute segments (NAPS). The NOVAlerf™ administered a Psychomotor Vigilance Test (PVT) to the participant when muscle activity decreased below a baseline set individually for each participant for each of the 5 nap opportunities. Individual baselines were established by the NOVAlerf™ prior to the start of each nap. This was accomplished by having the participants squeeze the fist of the monitored hand following an example of the PVT. FFT analyses were conducted for the 15 seconds immediately preceding and the 15 seconds immediately following the first PVT administered in each twenty-minute session. The 15 seconds before the PVT was referred to as the Pre-PVT condition and the 15 seconds following the PVT was referred to as the Post-PVT condition.

EEG power analyses were conducted in order to examine for time of night effects (from Nap 1 through Nap 5) and to look for changes in arousal between before and after PVT conditions.
Time of Night Effects

A 5 (Frequency Bin) X 5 (Nap; 1 through 5) ANOVA revealed there were no significant time-of-night effects. None of the frequency bins showed significant increases or decreases from Nap to Nap. In other words, Delta activity showed reliable patterns of activity from Nap 1 through to Nap 5 and likewise for each of the other 5 EEG Frequency Bins. In addition, neither of the EMG measures showed significant increases or decreases across nap trials. Refer to Table 2 for an ANOVA summary of the time-of-night effects on EEG. Chin and Wrist EMG were combined to form a total EMG power level that did not change significantly from Nap to Nap and can be see in Table 3. There were no changes in EEG or EMG power that could be attributed to the progression from Nap 1 to Nap 5.

<table>
<thead>
<tr>
<th>Source</th>
<th>Df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Power Error</td>
<td>4</td>
<td>52425.38</td>
<td>39516.54</td>
<td>1.30</td>
<td>.38</td>
</tr>
<tr>
<td>Error</td>
<td>44</td>
<td>322197.98</td>
<td>30357.79</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Delta Error</td>
<td>4</td>
<td>1600155.54</td>
<td>1327232.48</td>
<td>1.38</td>
<td>.35</td>
</tr>
<tr>
<td></td>
<td>44</td>
<td>9306397.28</td>
<td>964886.88</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Theta Error</td>
<td>4</td>
<td>330.15</td>
<td>155.20</td>
<td>2.15</td>
<td>.18</td>
</tr>
<tr>
<td></td>
<td>44</td>
<td>1228.25</td>
<td>72.16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alpha1 Error</td>
<td>4</td>
<td>677.95</td>
<td>391.04</td>
<td>1.69</td>
<td>.22</td>
</tr>
<tr>
<td></td>
<td>44</td>
<td>3211.77</td>
<td>231.56</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alpha2 Error</td>
<td>4</td>
<td>785.24</td>
<td>443.73</td>
<td>1.10</td>
<td>.33</td>
</tr>
<tr>
<td></td>
<td>44</td>
<td>5725.50</td>
<td>404.42</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sigma Error</td>
<td>4</td>
<td>5526.19</td>
<td>4645.39</td>
<td>0.81</td>
<td>.43</td>
</tr>
<tr>
<td></td>
<td>44</td>
<td>55051.00</td>
<td>5784.58</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beta Error</td>
<td>4</td>
<td>12315.74</td>
<td>9353.55</td>
<td>0.76</td>
<td>.46</td>
</tr>
<tr>
<td></td>
<td>44</td>
<td>129268.66</td>
<td>12272.11</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

There was no significant main effect for Time-of-night, F(4,44) = 1.66, p = .22, indicating that Chin muscle activity remained relatively unchanged throughout each of the Nap opportunities. As with Chin EMG there was no main effect of Nap number.
indicating that Wrist EMG remained relatively consistent throughout each of the naps.

Interpretations of the effects of multiple naps on EMG are difficult because of the vast variability in total power of the Chin EMG and Wrist EMG. Table 3 provides a clearer interpretation of the differences between the two measures of EMG power.

Table 3
Summary ANOVA table for time-of-night effects on EMG power

<table>
<thead>
<tr>
<th>Source</th>
<th>Df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>EMG</td>
<td>4</td>
<td>349784.59</td>
<td>251593.18</td>
<td>2.41</td>
<td>.09</td>
</tr>
<tr>
<td>Error</td>
<td>44</td>
<td>11609207.35</td>
<td>1043787.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chin</td>
<td>4</td>
<td>56830.08</td>
<td>21298.40</td>
<td>1.66</td>
<td>.22</td>
</tr>
<tr>
<td>Error</td>
<td>44</td>
<td>27397.26</td>
<td>12834.64</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wrist</td>
<td>4</td>
<td>6191596.99</td>
<td>4642902.82</td>
<td>2.25</td>
<td>.10</td>
</tr>
<tr>
<td>Error</td>
<td>44</td>
<td>22008757.19</td>
<td>2062967.78</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Pre-PVT versus Post-PVT

A 2 (Pre-PVT and Post-PVT) X 5 (Naps 1 to 5) ANOVA showed a significant effect of PVT administration on EMG activity, $F(1, 11) = 7.25$, $p = .03$ indicating that muscle activity in general (ie. Chin and Wrist combined) significantly increased following the administration of a PVT from the NOVAAlert™ wrist unit. Chin EMG and Wrist EMG power levels following PVT administration were then looked at separately.

Chin EMG Power

A 2 X 5 ANOVA evaluating Pre- and Post-PVT by Nap (1 to 5) revealed a significant main effect for PVT administration $F(1,11) = 33.98$, $p < .001$, indicating that Chin EMG increased significantly following the administration of a PVT as can be seen from Figure 18. Each individual Nap was examined more closely using a series of t-tests. The increase in Chin EMG observed during Nap 1 was significance at $t(11)= -1.99$, $p < .05$. The increase during Nap 2 was significant, $t(11)= -2.51$, $p < .05$. The increase during
Nap 3 is also significant, \( t(11) = -2.02, p < .05 \). Increases in Chin EMG drop off considerably during the 4\(^{th}\) and 5\(^{th}\) NAPs, \( t(11) = -97, p = .18 \) and \( t(11) = -1.05, p = .19 \) respectively. Figure 18 represents changes in Chin EMG as a function of PVT presentation.

![Figure 18](image)

**Figure 18**
Chin EMG power before and after PVT across Naps

**Wrist EMG Power**

The 2 X 5 ANOVA showed a significant effect of PVT administration on Wrist EMG, \( F(1,11) = 6.72, p = .03 \) indicating that Wrist EMG significantly increased following the administration of the PVT. Four of the five individual Naps were witness to significant increases in Wrist EMG activity following the PVT administration. Refer to Table 4 for a summary of significance levels for each of the 5 Naps. Figure 19 shows the effects of PVT administration on Wrist EMG. There it can be seen that EMG activity before the administration of the PVT is at or near zero, hence the detected need for the alerting PVT.
Figures 18 and 19 illustrate the importance of the analyses demonstrating the alerting effect that the PVT administration had on EMG activity. In each case, muscle activity was increased following the presentation of the NOVAalert™ administered PVT. From these comparisons it appears that wrist EMG may be more sensitive to the alerting effect of the PVT than chin EMG.

Table 4
Summary table of Paired Sample t-tests for Wrist EMG increases

<table>
<thead>
<tr>
<th>Source</th>
<th>Df</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nap 1</td>
<td>11</td>
<td>.01</td>
</tr>
<tr>
<td>Nap 2</td>
<td>11</td>
<td>.03</td>
</tr>
<tr>
<td>Nap 3</td>
<td>11</td>
<td>.02</td>
</tr>
<tr>
<td>Nap 4</td>
<td>11</td>
<td>.03</td>
</tr>
<tr>
<td>Nap 5</td>
<td>11</td>
<td>.06</td>
</tr>
</tbody>
</table>
PVTs and Hori Stages

It is important to determine when during the Hori continuum the NOVAAlert™ detects that muscle activity has dropped significantly below levels required for adequate and safe alertness and performance. Each twenty-minute session on the Nova Night was divided into 5-second epochs with each visually scored and labelled as one of Hori's 9 stages. Then, the mean number of PVTs administered to each of the 12 participants during each stage were counted and presented in Figure 20. Hori Stage 4 was eliminated from analysis because of the limited number of occurrences of this stage. This problem was also encountered by Doerfling, Ogilvie, Murphy, & Lamarche (1996). There was a significant increase in the number of PVTs administered by the NOVAAlert™ from Hori Stage 0 (wake) to Hori Stage 2, \( t(11) = 3.31, p = .009 \) as well as another significant increase from Hori Stage 2 to Hori Stage 3, \( t(11) = 5.11, p < .001 \). Graphically, there are subsequent drops in the number of PVTs during Hori Stages 5 and 6, these are minimal and are not statistically significant decreases. These results demonstrate support for the effectiveness of the NOVAAlert™ system to accurately detect significant decreases in wrist muscle activity as seen in Figure 3 (Wrist EMG Power across Hori Stages) where wrist muscle activity drops off significantly from Hori Stage 0 through Hori Stage 3 and remains low through the end of the Sleep Onset Period. Stages 0 to 3 in Hori's system represent EEG changes from continuous alpha activity to the complete absence of alpha. As well, these results show that the NOVAAlert™ was consistent in administering PVTs into the late stages of Hori’s continuum.
Number of PVTs

The number of PVTs during each Nap was counted to determine whether participants were becoming increasingly sleepy to the point at which the NOVAAlert™ began to significantly increase the number of PVT tests administered. This increase would signify that participants were becoming fatigued to the point of marked performance decline. A one-way ANOVA revealed a main effect of Nap, $F(4,44) = 3.40$, $p = .03$, indicating that the number of PVT tests did increase as time progressed. Figure 21 illustrates the relationship between time of night and the number of PVT test administrations.
There was a significant increase in the number of PVTs from Nap 2 (9.18) to Nap 3 (13.27), \( t(11) = -2.70, p = .022 \). This increase signified a substantial decline in the ability of the participants to maintain sufficient muscle tone as required by the NOVAAlert™ to remain above baseline.

**Number of Failed PVTs**

The number of failed PVTs was also counted to determine whether participants were more apt to fail tests as the night progressed by either responding too weakly or by not responding at all. A PVT was considered failed if the participant failed to raise their EMG level back to baseline or simply failed to respond at all. A one-way ANOVA revealed no significant main effect of Naps, \( F(4,44) = 1.37, p = .28 \), indicating that the number of failed responses did not increase significantly as time progressed. However, this non-significant increase was in the expected direction. Figure 22 illustrates the relationship between failed responses and Nap number.
Figure 22
Mean number of failed PVTs across Naps

PRE-PVT and POST-PVT Effects on EEG

A series of 2 (Pre- versus Post-PVT) X 5 (Naps 1 – 5) ANOVAs revealed that there were no significant changes in Delta, Theta, and Alpha1 power as a result of PVT administration. However, there were significant increases in Alpha2, Sigma, and Beta power following the administration of the PVT. More detailed descriptions of the changes following the PVT are explained separately for each Frequency Bin. Refer to Table 5 for a summary of the effects of PVT administration.
Table 5
Summary ANOVA table for Psychomotor Vigilance Task (PVT) on EEG power

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>SS</th>
<th>MS</th>
<th>F</th>
<th>P</th>
<th>$\eta^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Power</td>
<td>1</td>
<td>26998.02</td>
<td>26998.02</td>
<td>2.49</td>
<td>.22</td>
<td></td>
</tr>
<tr>
<td>Error</td>
<td>11</td>
<td>86716.86</td>
<td>10839.61</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Delta</td>
<td>1</td>
<td>713888.82</td>
<td>713888.82</td>
<td>1.91</td>
<td>.30</td>
<td></td>
</tr>
<tr>
<td>Error</td>
<td>11</td>
<td>2977596.72</td>
<td>372199.59</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Theta</td>
<td>1</td>
<td>66.01</td>
<td>66.01</td>
<td>.51</td>
<td>.53</td>
<td></td>
</tr>
<tr>
<td>Error</td>
<td>11</td>
<td>1039.25</td>
<td>129.90</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alpha1</td>
<td>1</td>
<td>9.61</td>
<td>9.61</td>
<td>.05</td>
<td>.81</td>
<td></td>
</tr>
<tr>
<td>Error</td>
<td>11</td>
<td>1656.38</td>
<td>207.05</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alpha2</td>
<td>1</td>
<td>203.76</td>
<td>203.76</td>
<td>8.04</td>
<td>.02</td>
<td>.58</td>
</tr>
<tr>
<td>Error</td>
<td>11</td>
<td>202.84</td>
<td>25.36</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sigma</td>
<td>1</td>
<td>2234.54</td>
<td>2234.54</td>
<td>5.45</td>
<td>.04</td>
<td>.48</td>
</tr>
<tr>
<td>Error</td>
<td>11</td>
<td>3281.88</td>
<td>410.23</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beta</td>
<td>1</td>
<td>7127.49</td>
<td>7127.49</td>
<td>6.87</td>
<td>.03</td>
<td>.54</td>
</tr>
<tr>
<td>Error</td>
<td>11</td>
<td>8303.67</td>
<td>1037.88</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Total EEG Power**

Figure 23 represents overall changes in total EEG power before and after the presentation of the PVT from the NOVAAlert wrist unit. As noted in Table 5, there was no main effect of PVT presentation with respect to Total EEG power.

Figure 23
Total EEG power before and after PVT across Naps
The large visual differences between before and after PVT administration during Hori Stages 2 and 3 are not confirmed statistically, $t(11) = -1.7, p = .07$ and $t(11) = -1.46, p = .09$ respectively.

**Delta Power**

Table 5 notes that there was no main effect of PVT presentation on Delta power. Figure 24 represents the changes in Delta as a result of the PVT test.

![Figure 24](image)

*Delta power before and after PVT across Naps*

Referring back to Figure 23, it can be observed that the large amounts of Delta activity greatly influence the overall total EEG power numbers. Again, the large visual differences seen during Hori Stages 2 and 3 are not statistically significant, $t(11) = -1.59, p = .07$ and $t(11) = -1.50, p = .07$ respectively. These large increases in delta activity can
be due largely to body movements in response to the PVT itself and not a change in particular arousal levels.

**Theta Power**

Although not statistically significant, a trend can be seen across Naps in that Theta activity appears to increase gradually across time. In addition, it can also be seen that Theta activity is consistently lower following a PVT across all Naps, although not significantly lower. Figure 25 illustrates the relationship of Theta and PVT presentation.

**Figure 25**  
Theta power before and after PVT across Naps

![Theta Power Graph](image)

**Alpha1 Power**

No significant observations can be seen in Alpha1 activity across Naps. Figure 26 represents Alpha1 power as a result of the PVT presentation. Here is can be seen that the order of the means show no consistent patterns.
Alpha2 Power

According to Table 5, there was a main effect of PVT administration with Alpha2 activity increasing following the administration of the PVT. Statistically, the only individual Nap that showed a significant increase in Alpha2 power was Nap 5 ($t(11) = 3.26, p < .01$). Figure 27 represents the increases in Alpha2 in response to the PVTs.
Sigma Power

There was a significant main effect of PVT administration for Sigma activity. The differences in Sigma activity do not approach significance for Naps 1 or 4. Individually however, Naps 2 and 3 were significant, $t(11) = -2.01, p = .04$ and $t(11) = -2.11, p = .03$ respectively while the difference observed during Nap 5 is also significant, $t(11) = -2.73, p = .01$ suggesting that there existed far less variability in the levels of Sigma activity evidenced later in the night. Figure 28 shows the effects of PVT administration on Sigma activity across each of the 5 Naps.

Figure 28
Sigma power before and after PVT across Naps

Beta Power

There was a significant main effect of PVT administration across Naps with Beta activity increasing following the administration of the PVTs. These increases fall off in later naps. However, one of the individual Naps was significant; the difference observed during Nap 2, $t(11) = -2.06, p = .04$. Figure 29 shows the effects of the PVT on Beta power.
Subjective Data

Stanford Sleepiness Scale

Following each of the 5, twenty-minute sessions for the EMG night and each of the 5, twenty-minute sessions on the Nova night, participants were asked to complete the SSS. Data from all 12 participants indicated that there was a significant main effect of Nap Number, $F(4,44) = 31.00, p < .001$, indicating that participants demonstrated significant time-of-night effects in that participants, on both nights, rated themselves as more sleepy in the later sessions than in the earlier sessions.

There was no significant main effect of Night, $F(1,11) = 2.19, p = .17$, indicating that participants did not rate themselves as being more sleepy on the Nova night than on the EMG night. Additionally, the interaction between Nap Number and Night was not
significant. Figure 30 illustrates mean SSS ratings across Naps for both experimental nights.

![Figure 30](image)

**Visual Analog Scale**

In addition to the SSS, participants were also asked to complete the VAS (Akerstedt & Gillberg, 1990) following each of the 5, twenty-minute sessions for the EMG night and following each of the 5, twenty-minute sessions on the Nova night. As with the SSS, there was a significant main effect of Nap Number, $F(4,44) = 39.33$, $p < .001$, indicating that participants demonstrated significant time-of-night effects in that participants in both nights rated themselves as more sleepy in the later sessions than in the earlier sessions.

Again, as with the SSS, there was no significant main effect of Night, $F(1,11) = 2.54$, $p = .14$, indicating that participants did not rate themselves as being more sleepy on the Nova night than on the EMG night. As well, the Nap Number by Night interaction
was non-significant. Figure 31 illustrates mean VAS ratings across Naps for both experimental nights.

![Figure 31: Mean VAS ratings across Naps](image)

Participants' ratings of sleepiness on the SSS and the VAS were highly correlated with one another ($r = .86, p < .001$).

**Time to First PVT**

Time, in seconds, was measured to provide a measure of the rate in which participants tired while using the NOVAalert™ system. A one-way ANOVA revealed a weak trend towards the predicted main effect of Nap Number, $F(4,44) = 2.22, p = .14$, indicating that the time to the first test PVT did not lengthen during the experimental sessions. Figure 32 illustrates the relationship of time to first PVT and Nap Number.
Figure 32
Mean time (in seconds) to the first PVT during each Nap

![Graph showing mean time to first PVT during each nap.]

Graphically, there appears to be a large decrease in the time to first PVT from Nap 3 (248.64s) to Nap 4 (81.27s). This drop was significant, $t(11) = 3.80$, $p = .003$, indicating a significant decrease in the time to first PVT during the 4th Nap.

**PVT Effectiveness Ratings**

Participants were asked to indicate on the SSS how sleepy they felt immediately before and immediately after they were administered the PVT. A 2 (Pre- versus Post-PVT) X 5 (Naps) ANOVA revealed that there was a main effect of Time, $F(4,44) = 22.67$, $p < .001$ indicating that again individuals reported that they felt more sleepy as time progressed. There was also a main effect of PVT administration, $F(1,11) = 13.05$, $p = .005$, indicating that individuals felt significantly less sleepy immediately following the administration of the PVT. Figure 33 illustrates mean SSS ratings of sleepiness before
and after the PVT. There it can also be seen that the Nap by PVT administration was non-significant.

![Figure 33](image)

**Figure 33**
Mean SSS ratings before and after PVTs

As expected, individuals felt increasingly sleepier as the number of the Naps increased bringing them later and later into the evening as seen from the ratings on the SSS and the VAS. Participants did not rate themselves as more or less sleepy on the NOVA or the EMG nights. There was a significant drop in the time needed to elicit a PVT from the NOVAAlert™ following the third Nap session. The mean number of PVTs administered increased significantly from the second to the third Nap and remain at such a level. In addition, the mean number of failed PVT responses increased following the third Nap session indicating a point in which fatigue began to seriously affect performance.
Discussion

The primary intent of the present study has been to examine the relationship of EMG activity to the sleep onset period. Many studies have focused on EEG-based correlates and behavioural correlates to the process of falling asleep, but what can muscle activity tell us about this process? Can EMG measures provide information not obtainable from other measures?

Theoretical Implications

EMG

To the author's knowledge, there exist no studies examining EMG changes as a function of Hori's nine stages of Sleep Onset. Previous research had dismissed the prospect that measuring muscle activity during the Sleep Onset Period could be of significant use (Jacobson et al., 1964). Other research had suggested there existed a close relationship between EEG and EMG changes during the emergence of REM sleep from Stage 2 activity (Hadjyannakis et al., 1997) in that alpha and sigma power correlated positively with drops in EMG. However, the present study has shown an inverse relationship between EMG activity and both alpha and sigma activity as sleep begins. Both alpha and sigma showed significant increases in power during the latter stages of Hori's continuum while at the same time, EMG activity decreased significantly.

With respect to EMG measured from the chin, there does appear to be a significant relationship between muscle activity and sleep onset. From the present study it appears that chin muscle activity does drop off significantly over the entire Sleep Onset Period, however, the greatest drop in tone occurred after Hori Stage3. This is also the
point at which alpha activity drops off significantly. There were no significant sequential stage differences in that Chin EMG measured during Hori Stage 2 did not differ from Chin EMG measured during Hori Stage 3. Although there was an overall effect of stage, no two successive stages differed significantly. However, there were significant stage differences in Chin EMG throughout the SOP. Hori Stage 0 (wake) was significantly different from Stage 5, Stage 7, Stage 8, and Stage 9. Hori Stage 2 was different from Stage 7, Stage 8, and Stage 9. Finally, Hori Stage 3 was different from Stage 5, Stage 7, Stage 8, and Stage 9. After Stage 3 there were no more significant differences among the stages. This finding indicated that there existed a significant decline in Chin muscle activity following Hori Stage 3 (see Figure 2). Chin EMG activity could feasibly be divided into two areas of classification during the Sleep Onset Period: one area of elevated activity present during Hori Stages 1 – 3 following by a period of decreased activity evident during Hori’s Stages 5 – 9. Similar findings have been reported with respect to the decline in other muscle groups, namely the tensor palatini muscles. Worsnop, Kay, Pierce, Kim, & Trinder (1998) found that increases in airway resistance were evident with a transition from Alpha dominated (first third of Hori’s stages with adequate EMG levels) to Theta dominated sleep (final third of Hori’s stages with decreased EMG activity).

On the other hand, EMG activity measured from the wrist appears to drop off sooner than that of the chin. There was once again a significant reduction in overall Wrist EMG activity throughout the entire SOP however, the major significant drop came earlier, before the onset of Hori Stage 3 even before the disappearance of alpha activity. Previous research had shown that the ability to hold a microswitch closed significantly
decreased in the middle of Rechtschaffen & Kales' stage 1 'sleep' (Ogilvie, Wilkinson, & Allison, 1989; Perry & Goldwater, 1987). This observation lends support to wrist muscle activity providing a useful early-warning measure of impending drowsiness as utilised by the NOVAalert™ monitoring system. This finding establishes the basis for which the fundamentals of the NOVAalert™ functions. EMG activity was highly correlated with Beta activity indicating that decreases in psychological alertness were closely linked with decreases in physiological alertness. In addition, when these low levels of alertness were encountered, the wrist-worn NOVAalert™ device was effective in reversing both the level of Beta activity as well as EMG activity which in turn led to participants reporting that they felt more alert following the alarm emitted from the device.

**EEG**

It was important to separately analyse and report on EEG activity during the process of falling asleep (Hori's continuum) in order to provide a context against which to assess the EMG changes during the same period. This analysis also provided a standard against which to compare the changes observed following administration of the PVTs through the NOVAalert™ system.

Sleep onset has been nicely defined in terms of brain-based EEG patterns by Hori's 9-stage model. Using this model as a framework, the present study determined correlates of this model based on quantified EEG and EMG measurements, subjective ratings of sleepiness, and performance monitoring.

Early in the sleep onset process, EEG measures tend to exhibit limited variability. Changes in all major frequencies are carefully described by Hori staging. Hori (1985)
reported on average power spectral values following the beginning of stage 1 sleep, and found that variability in Delta, Theta and Alpha power increased as standard Stage 1 was entered and that the increased variability continued throughout standard Stage 1. Delta activity tends to remain low during the first and second thirds of the sleep onset period and rising sharply with the onset of vertex waves evident with Hori Stage 7, remaining elevated through Hori Stage 8 and falling again as an individual progresses to Stage 9. This effect is most prominent along the midline of the brain as opposed to either the left or right hemispheres and is most prominent in the frontal, central and parietal regions of the brain. The present study demonstrated that Theta activity follows a similar pattern as Delta in that Theta activity remained low during the first and second thirds of the SOP only to rise significantly with the onset of Hori Stage 7 and falling again through Hori Stage 9. However, this discovery came somewhat surprisingly. Ogilvie, Simons, Kuderian, MacDonald, & Rustenburg (1991) reported that Theta activity increased and remained elevated during the SOP. While easy to observe with the naked eye the slower patterns associated with vertex waves and vertex wave trains of Hori Stages 7 and 8, it is not so easy to pick out theta activity during those same stages. While scoring epochs according to Hori’s guidelines, one looks for theta rhythm activity primarily during Stage 5. However, the present FFT analysis has shown no increase in Theta frequency during Stage 5 but a sharp increase in Theta frequency during Stages 7 and 8. This discovery may prove useful in developing algorithms for more precise sleep stage scoring. Again, as with Delta activity, Theta was found to be most predominant along the midline of the brain as opposed to either the left or right hemispheres. As expected, Alpha activity was higher during the early stages of the sleep onset period predominantly in the posterior
regions (parietal and occipital regions) of the brain and then moving towards more anterior portions of the brain during the later stages of sleepiness. This finding is supported by previous research examining Alpha dominance transferring from the posterior regions of the brain to the anterior portions during sleep onset (Hori et al, 1994; Hasan & Broughton, 1994). Additionally, Alpha1 and Alpha2 activity was most predominant along the midline sites of the brain.

However, a laterality effect was most noticeable during the later stages of Hori’s continuum. Sigma activity followed a similar pattern as that of Delta and Theta activity in that all brain areas produced similar patterns during the first two thirds of the sleep onset process only to give way to a parietal-central-frontal dominance and increased variability during the final third of the process. And again, Sigma activity was greatest along the midline of the brain and this finding was most prominent during the later stages of Hori’s continuum.

Beta activity did not follow the same pattern of rising during the final stages of Hori sleep. Since Beta activity is believed to be an indication of arousal level, it is logical that these levels should drop off and stay low during the last moments of waking consciousness. One observation from the frequency bin analysis that proved interesting was that for all bins, activity was at its peak along the midline fissure compared to either the left or right hemispheres. This may very well be a result of the physical shape of the brain and not to any functional or behavioural differences. This realization may well prove useful in fine-tuning traditional sleep stage scoring according to standard R & K scoring. Researchers and clinicians alike are constantly looking for ways to better describe the sleep cycle by analyzing, in greater detail, the standard stages put forth by R
& K. One example has been used extensively during this present study; that of Hori's 9-Stage continuum based on R & K's standard Stage 1. Knowing that EEG power is significantly more prominent along the midline fissure would provide enhanced detail for examining more subtle changes in brainwave activity during the remaining standard sleep stages.

**Subjective Measures**

As expected, participants rated themselves as more sleepy as the number of Nap sessions increased. However, participants did not rate themselves as more or less tired on either the EMG night or the NOVA night when asked to report scores on the Stanford Sleepiness Scale. Similar findings were evidenced when participants were asked to complete the Visual Analog Scale. Participants again rated themselves as more tired as the number of Naps increased bringing them later into the evening and again no differences emerged between ratings of fatigue between the EMG night and the NOVA night.

Participants were also asked to rate their fatigue levels on the SSS following the administration of the NOVAalert™ PVT and as predicted, these individuals reported feeling less sleepy following the vibrotactile stimulation presented by the NOVAalert™. This effect remained persistent through all five of the nap opportunities in which the wrist unit was worn.

Even though ratings on the SSS and VAS were not significantly different between the EMG night and the NOVA night, the results were all in the predicted direction in that
ratings on the NOVA night were consistently lower than those from the EMG night and there were no obvious interaction effects.

**Practical Applications**

**Nova and the Detection of Sleepiness**

The NOVAler™ was designed as an early warning device to alert the user to impending performance detriments due to fatigue. The present study provided support for the effectiveness of the NOVAler™ in detecting significant decreases in wrist muscle activity as it is evidenced early in the Hori continuum (H0 to H3). Hori Stages H0 to H3 were the stages associated with significantly decreasing levels of Alpha and Beta activity; those waveforms associated with arousal. Support was also provided for the consistency of the device to continue detecting dangerously low levels of muscle activity (resulting in increased numbers of PVTs). The number of PVTs administered remained high through the entire SOP in response to persistently lowered levels of wrist muscle activity.

When participants were asked to wear the wrist unit on the NOVA night, the PVTs administered by the NOVAler™ significantly increased the amount of muscle activity in both the Chin and Wrist, as well as Beta and Sigma EEG activity, providing an alerting response. The number of PVTs increased significantly from the second Nap session to the third indicating that fatigue increased sufficiently during that time (approximately 50 to 60 minutes after the onset of the first Nap session) to induce performance detriments raising the need for an alerting response that was in turn provided by the NOVAler™. Chin muscle activity showed significant power increases following the administration of PVTs during each of the first three Nap sessions.
indicating that with the fourth Nap session the NOVAalert™ began to decrease in its effectiveness in eliciting an alerting response with regards to Chin EMG. Wrist EMG showed significant increases in activity following the administration of the PVTs for each of the first four Nap sessions indicating that the NOVAalert™ demonstrated powerful alerting capabilities with regards to Wrist EMG activity for nearly 60 minutes. The NOVAalert™ was designed as an alertness maintenance device for automobile drivers, train engineers, pilots, flight controllers, and anyone who performs tasks in which a significant amount of attention is needed. The present study provides evidence that alertness can be effectively maintained for approximate 60 minutes, allowing the user to safely find rest.

The present study provided evidence as to the effectiveness of the NOVAalert™ system by showing that its use eliminated the time-of-night effects displayed by the majority of the frequency bins throughout the SOP. When equipped with the wrist unit, all frequencies in question remained relatively unchanged throughout most of the ‘nap’ opportunities. This was not the case when participants were simply allowed to sleep when frequencies were prone to significant increases (or decreases in the case of Beta) during the process of falling asleep. Delta activity was not changed from Pre-PVT to Post-PVT. However, Delta activity remained consistently low during each of the 5 Nap sessions due to the continually alerting presence of the NOVAalert™; baseline levels (those evident before the presentation of the PVT) never exceeded those obtained past Hori Stage 5. Higher levels of Delta activity would be indicative of entrance into the later stages of Hori’s continuum. Theta levels also remained consistently low during each of the 5 Nap sessions with the NOVAalert™ remaining at levels consistent with Hori’s definition of
wakefulness. As with Delta, Theta levels were not significantly altered with administration of PVTs since they were never significantly elevated as with impending sleep. Alpha2 activity was significantly affected by the administration of PVTs in that power levels for Alpha2 increased following the PVT indicating an alerting response to levels similar to that of resting wakefulness. One characteristic used to help define sleep is that it is a state, which is reversible with stimulation (Flannigan, 1972). One step further would be to add the characteristic that sleep onset could be postponed with adequate stimulation.

The most evident effects of PVT administration (aside from that of the effects on EMG) occurred with Sigma and Beta activity. Although not statistically significant, it can be seen from Figures 23 and 24 that Sigma and Beta activity levels tended to increase following the presentation of a PVT for each of the first three Nap sessions. This alerting effect appears to dissipate with the fourth and fifth sessions suggesting that the NOVAalert™ is highly effective for the first 60 to 70 minutes of use and then its effectiveness begins to wears off. This is also approximately the time frame in which the number of PVT tests and failed PVTs significantly increased along with a significantly shortened interval from baseline-establishing PVT to the first test PVT. These limitations encountered during this time frame perhaps indicate the upper limit on the practical effectiveness of the NOVAalert™ as a means of postponing the effects of increased sleepiness. This is consistent with the intended use of the device as an early warning system but also shows that as expected, its sensitivity decreases with continued use and growing sleepiness.
Conclusions

Sleep researchers are generating a better understanding of what we experience physiologically during sleep. Once thought to be a state of ‘nothingness,’ sleep processes are becoming ever more clear. Of particular interest in relatively recent years and to relatively few researchers is the process of falling asleep itself. Sleep onset is no longer believed to be an exact moment in which our bodies and minds go from waking one second to fast asleep the next. Past behavioral and physiological evidence suggested that the exact moment of sleep onset varied according to the measure used to define sleep onset. However, many parameters change as we fall asleep. Our behaviors and brain waves change along with our subjective feelings about our own sleep/wake state changes. And naturally, our muscle tone changes as we fall asleep. It is important to use convergent methods of measuring sleep onset. Relying on one particular measure may lead to misinterpretation of sleep onset and falling prey to believing in an exact ‘moment’ of sleep. The present study has provided such a convergence of sleep onset measures that can be used to better clarify the sleep onset process.

EMG, particularly wrist EMG, drops precipitously early in the SOP (i.e. during Hori stages 0 to 3). This drop in EMG is paralleled by EEG changes – most dramatically by the appearance (during Hori stage 1) and disappearance (during Hori stage 3) of alpha activity. Theoretically, this suggests that the process (or processes) responsible for blocking alpha as sleep begins also may regulate EMG activity. If this is so, a basis may be established for the practical usefulness of EMG changes in monitoring sleepiness and hence, the effectiveness of alertness monitoring devices such as the NOVAAlert™.
While the present study goes a long way to better clarify the relationship between EMG, EEG, and sleep onset processes, there are considerations that need to be addressed with future research. With respect to future testing of alertness monitoring/maintenance devices such as the NOVAalert™ system, it would be prudent to test such devices in more practical situations. The present study simply had participants lying in bed with the device attached to their wrists. Future studies should take steps further, perhaps using such a device in conjunction with driving simulators or even simply having participants sitting upright instead of lying down in bed.

The present study has shown a reliable link between changes in EMG activity and EEG and subjective changes encountered during the Sleep Onset Period. However, as mentioned earlier, reliance on only one measure in determining the ‘moment’ of sleep would risk falling into the trap of believing that there exists a single ‘point’ of sleep onset. Sleep onset is a process that needs to be monitored by a series of convergent measures that, as a group, describe and paint a clearer picture of the process of falling asleep.
References


Appendix A

Stimulus Evaluation Questionnaire

Please rate the following from:

How awake/sleepy did you feel when you felt the vibration from the Wrist Unit?

1. Feeling active and vital; wide awake.
2. Functioning at a high level, but not at peak; able to concentrate.
3. Relaxed, not at full alertness; responsive.
4. A little foggy; let down.
5. Fogginess; beginning to lose interest in remaining awake.
6. Sleepiness; prefer to be lying down; fighting sleep; wozzy.
7. Almost in reverie; sleep onset soon; losing struggle to remain awake.

How awake/sleepy did you feel after the vibration was presented?

1. Feeling active and vital; wide awake.
2. Functioning at a high level, but not at peak; able to concentrate.
3. Relaxed, not at full alertness; responsive.
4. A little foggy; let down.
5. Fogginess; beginning to lose interest in remaining awake.
6. Sleepiness; prefer to be lying down; fighting sleep; wozzy.
7. Almost in reverie; sleep onset soon; losing struggle to remain awake.

How effective would this device be in keeping a sleepy driver awake?

1 2 3 4 5 6 7
Very Neutral Very Effective
Poor

How effective would this device be in warning a driver to take precautions to avoid dangerous levels of drowsiness/sleepiness?

1 2 3 4 5 6 7
Very Neutral Very Effective
Poor
Appendix B

Stanford Sleepiness Scale

Please circle the number which best describes how you feel based on the statements given below.

1. Feeling active and vital; wide awake.
2. Functioning at a high level, but not at peak; able to concentrate.
3. Relaxed, not at full alertness; responsive.
4. A little foggy; let down.
5. Fogginess; beginning to lose interest in remaining awake.
6. Sleepiness; prefer to be lying down; fighting sleep; wozzy.
7. Almost in reverie; sleep onset soon; losing struggle to remain awake.

Visual Analogue Scale

Please indicate how you feel by placing a mark on this line.

Very Sleepy---------------------------------------------------------------Very Alert
Appendix C

SLEEP STUDY

“A Microanalysis of EMG Changes during the Sleep Onset Period (SOP):
A Theoretical Investigation with Practical Applications”

<table>
<thead>
<tr>
<th>Student Number</th>
<th>Phone Number</th>
<th>Best Time to Call</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix D

A Microanalysis of EMG Changes in the Sleep Onset Period (SOP):

A Theoretical Investigation and Practical Applications

Brock University

Letter of Information

Procedure for overnight recordings:

You will be scheduled along with one other person (most likely of the same sex) for three consecutive nights. Upon arriving at the lab, you will change into the clothes that you plan to sleep in. The electrode hook-up will then commence. Each electrode site will be cleaned with alcohol, then scrubbed with a mildly abrasive paste and wiped clean with a cotton swab. The electrodes will then be applied and held in place by conductive paste (if in the hair), or tape (if on a hairless part such as the face). 21 electrodes will be applied to your head along with 1 on the outside of each eye, 2 under the chin, two on the inside area of your wrist, and one in the middle of your forehead. Also, on one of the experimental nights, a small wrist-worn device will be attached to the same arm as the wrist electrodes.

Once you are ready for bed, we will calibrate the recording equipment. You will be asked to blink several times, look left, look right, look up, look down, cough, swallow, and grind your teeth. You will then fill out the Stanford Sleepiness Scale (SSS). The wrist-worn alerting device will be turned on and calibrated. The lights will then be turned off and you will begin the first of six, 20-minute nap segments. After each of the nap sessions you will be asked to complete the SSS. After the sixth nap you will be allowed to sleep for the rest of the night uninterrupted. During the first night, there will be no nap sessions and on only one of the two experimental nights will you wear the wrist device. On the nights with the alerting device, you will also be asked to complete the Stimulus Evaluation Questionnaire along with the SSS.

In the morning when you awaken, you will be asked to complete the SSS once again. Once this is completed, the experiment will be concluded. We will then remove the electrodes.

I (please print) __________________________ have read and understood the above procedure.

Signature __________________________ Date: __________________________
Appendix E

A Microanalysis of EMG Changes in the Sleep Onset Period (SOP):

A Theoretical Investigation and Practical Applications

Informed Consent Form

Researchers: Cory R. Martin, B.A. and Professor Robert D. Ogilvie, PhD

Name of Participant: (Please print)

I understand that during the study I will be required to fill out various questionnaires pertaining to my sleep condition. I understand that I will be spending three nights in the Brock University Sleep Lab, monitored as outlined in the Letter of Information.

I have been given a tour of the sleep lab and read the Letter of Information, describing the overnight procedures in detail, and all of my questions have been answered.

I understand that my participation in this study is voluntary and that I may withdraw from the study at any time and for any reason, without penalty. I will receive three (3) course credit hours for my participation in the three overnight sessions (for PSYC 1F90). As an alternative, I may choose to receive $15 for each overnight session, or $50 for all three.

I understand that there is no obligation to answer any question or participate in any aspect of this study that I consider invasive, offensive, or inappropriate, and that I may choose to omit any information that I do not feel comfortable providing, without penalty.

I understand that all personal data will be kept strictly confidential and that all information will be coded so that my name is not associated with any information given or data collected. I understand that only the researchers named above will have access to the data.

I agree to refrain from using alcohol or caffeine for the three days on which overnight recordings will be conducted. Moreover, I understand that there will be no opportunity to smoke during the overnight sessions.

Participants Signature: _______________________________ Date: _______________________________

If you have any questions or concerns about your participation in the study, you can contact Cory Martin at (905) 688-5550 ext. 3795 or Professor Ogilvie at (905) 688-5550 ext. 3573.

I have fully explained the procedures of this study to the above participant.

Researcher’s Signature: _______________________________ Date: _______________________________
Appendix F

A Microanalysis of EMG Changes in the Sleep Onset Period (SOP):

A Theoretical Investigation and Practical Applications

Informed Consent Form

Having completed your participation in the present study, we would like to express our appreciation and provide you with the following information concerning the issues under investigation.

The present study has both theoretical and practical aspects. The theoretical intent of the study is to closely examine the relationship between muscle activity (EMG) and EEG state during the process of falling asleep. Sleep stages have been well defined with regards to brain wave activity (Rechtschaffen & Kales; Hori) during the sleep onset process. However, little work has attempted to quantify the changes in muscle activity during this same process, as this study will.

The practical aspect of the study will examine the reliability of a commercially developed wrist-worn alerting device that utilizes changes in muscle activity/tension in order to alert its user in the event that he/she experiences reduced wakefulness that may result in dangerous consequences.

Again, thank you for your participation in the study. Any further information you may require can be addressed to:

Cory Martin
Department of Psychology
Brock University
Email: cm99an@badger.ac.brocku.ca
Phone: 905-688-5550 ext. 3795

Prof. Robert Ogilvie, PhD
Department of Psychology
Brock University
Email: rogilvie@spartan.ac.brocku.ca
Phone: 905-688-5550 ext. 3573