ABSTRACT

EFFECT OF PREDICTABILITY OF IMPOSED VISUAL MOTION ON THE OCCURRENCE OF MOTION SICKNESS

by Edward William Otten

The prediction and prevention of motion sickness in virtual environments will be critical to the acceptance of virtual environments as a training tool. The postural instability theory of motion sickness suggests that motion sickness results from prolonged exposure to situations where stable control strategies cannot be achieved. In the current research the extent to which the predictability of visual stimuli influences the occurrence of motion sickness was examined. The unpredictability of a complex stimulus versus a simple stimulus did not result in an increase in sickness incidence. Postural motion did show differences between sick and well participants during baseline trails, suggesting possible predisposition, and velocity successfully classified participants into sick and well groups, which may be useful in sickness prediction. Examination of the power of the postural motion revealed an ability of participants to adapt to a simple stimulus versus a relative inability to adapt to a complex stimulus.

EFFECT OF PREDICTABILITY OF IMPOSED VISUAL MOTION ON THE OCCURRENCE OF MOTION SICKNESS

A Thesis

Submitted to the

Faculty of Miami University

in partial fulfillment of

the requirements for the degree of

Master of Arts

Department of Psychology

by

Edward William Otten

Miami University

Oxford, Ohio

2005

Advisor_____
L. James Smart, Ph.D.

Reader_____
Allan J. Pantle, Ph.D.

Reader_____
Lynn A. Olzak, Ph.D.

Table of Contents

List of Tables

List of Figures

Effect of Predictability of Imposed Visual Motion
on Occurrence of Motion Sickness

Motion sickness has been a problem throughout history. Generally, it has been associated with transportation (e.g. ships, cars). In recent years, however, motion sickness has also been connected to the use of simulations. Sickness has been common in simulators that depict inertial motion, such as flight and driving simulations (Stoffregen & Smart, 1998). The use of simulator systems can offer a safe and cost-effective method of training, but acceptance of such systems will be severely reduced if motion sickness cannot be prevented (Biocca, 1992).

Sensory Conflict. Explanation for the occurrence of motion sickness has typically been based on the theory of sensory conflict (Oman, 1982). This theory is based on the assumption that stimulation of perceptual systems is ambiguous with respect to physical reality. It suggests that the individual sense organs are independently sensitive to the same events. It also suggests that the individual sense organs are normally redundant and match stored internal expectations created by past experience. In the event that the individual sense organs do not provide redundant information, and therefore do not match the stored expectations (expectancy violation), conflict ensues. As a result of this conflict, motion sickness occurs (Reason & Brand, 1975).

Although the sensory conflict theory provides an intuitive explanation for motion sickness, weaknesses exist that limit its utility. Chief among these weaknesses is that there is no way to objectively (empirically) measure conflict. Any quantification must be made post hoc, limiting predictive value. Without an objective measure of conflict, prediction of motion sickness using the sensory conflict theory is improbable. This lack of an objective measure also makes it difficult to empirically validate sensory conflict theory.

Postural Instability. Riccio and Stoffregen (1991) suggested an alternative theory based on the premise that sickness occurs in animals that are in situations in which they do not possess the necessary strategies to maintain a functional correspondence (taking into account both changing environment conditions and the goals of behavior) with the environment. This is manifested by postural instability.

Posture is defined as the overall configuration of the body and its segments (Riccio & Stoffregen, 1991). The control of posture is fundamental to the ability of an animal to engage in behavior. The animal seeks out a control strategy that is both stable and at the same time allows the behavior to occur. Most of the time, animals are successful in finding a stable control strategy and can, as a result, perform the behavior at a maximum level of performance. This adaptation occurs across a multitude of situations, and, in most circumstances, in a rapid fashion that minimizes any period of instability and allows the animal to continue and/or return to the behavior at the desired level of performance. However, in situations where a stable control strategy is not achieved, several consequences may result. In one case, the animal may lose control entirely, leading the cessation of the behavior. This loss of control is brief. The animal will quickly become stable once again, but the goals of the behavior will have not been met. In a second case, stability is not lost but rather degraded. In this case, the behavior is maintained, albeit at a lower level of performance. It is this prolonged instability that is believed to lead to motion sickness (Riccio & Stoffregen, 1991), and empirically postural

instability has been shown to precede motion sickness and be predictive of it (Stoffregen & Smart, 1998; Stoffregen, et. al., 2000, Smart, Stoffregen, & Bardy, 2002).

The postural instability theory of motion sickness suggests several important consequences for research. First, instability can be measured objectively. Measures of posture in non-naseogenic (non sickness inducing) situations can very easily be compared to posture in situations that cause sickness. Second, Riccio and Stoffregen (1991) suggested that the severity of motion sickness will scale directly to the duration of postural instability. Understanding the duration of instability that can be tolerated before the onset of sickness will be important for the prediction and prevention of sickness.

The occurrence of motion sickness depends greatly on the frequency of imposed motion. Frequencies between .08 to 0.4 Hz are very naseogenic, while frequencies outside this range appear to have little or no naseogenic characteristics, even after prolonged exposure (Kennedy, Hettinger, & Lilienthal, 1990). This might suggest that motion in the .08 to 0.4 Hz. range is the cause of sickness. However, unperturbed sway in humans is concentrated between 0.1 to 0.4 Hz (Bensel and Dzendolet, 1968). Since humans do not normally become sick as a result of their own postural sway, the frequency of imposed motion can not be the only factor leading to motion sickness. Other factors, such as amplitude, duration of exposure, axis of motion, or predictability, either alone or in combination with frequency, may provide a better explanation for the occurrence of motion sickness, and consequently need to be examined.

Predictability. One possibility that has been suggested is that the lack of predictability of complex stimulus waveforms might contribute to the increased incidence of sickness (Kennedy, Hettinger, & Lilienthal, 1990). The logic behind this possibility developed from idea that the execution of action relies on the successful detection of the emerging features of the environment (Reed, 1996). The ability of the organism to exhibit this forward-looking, or prospective, characteristic is especially critical for postural control (Gibson & Pick, 2000). Consequently, in situations where the characteristics of the environment change in an unpredictable (constantly changing) manner, prospective control of posture may be difficult. This inability to find a stable control strategy (adaptation), if prolonged, may result in sickness. However, in circumstances where the stimulus is highly predictable (relatively unchanging), adaptation should generally be achieved, and the occurrence of sickness should be low.

Venues. Motion sickness has typically been categorized into three distinct venues: vehicular or terrestrial, space sickness (i.e. orbital flight), and cybersickness (i.e. sickness produced by simulation/VE). These distinctions have primarily been made as a matter of empirical convenience, allowing researchers to examine sickness within a particular empirical framework (Smart, 2000). The general basis for these separate categories has been the existence of situation-specific clusters of symptoms (Kennedy & Fowlkes, 1992; Kennedy, et.al.,1992; Kennedy & Stanney, 1996). Some symptoms of motion sickness appear to occur more frequently in certain circumstances. However, this clustering of symptoms alone is not enough to conclude that the underlying mechanism of one venue is qualitatively different from another (Oman, et. al., 1986). The symptoms associated with motion sickness are not unique to motion sickness and could be the result of any number of factors (Reason & Brand, 1975). While distinct types of motion sickness may certainly exist, comparison of symptomology alone does not appear to provide an adequate basis for differentiation.

In an effort to examine motion sickness across venues directly, Smart (2000) compared a terrestrial venue (moving room) and a simulator venue (Synthesized Immersion Research Environment (SIRE) facility, Wright Patterson Air Force Base, OH). These venues varied in a variety of dimensions, the most important being the method of stimulus generation (physical displacement versus computer-generated simulation of displacement). Even with these differences, postural motion predicted sickness in both, suggesting the possibility of a single underlying cause of sickness. The use of postural instability theory to examine differences in sickness across venues may provide a better alternative to symptomology because postural motion can be directly measured and directly compared. The examination of postural instability theory as a means to directly compare sickness across venues will continue to be important for understanding the etiology of motion sickness.

Rationale. The current research sought to examine the extent to which the predictability of imposed motion is important in the occurrence of motion sickness. It was hypothesized that imposed motion of an unpredictable nature would lead to a greater occurrence of sickness than imposed motion of a predictable nature. The imposed motion was created through the use of a visual stimulus that either oscillated at one frequency (single sine wave stimulus), or at an oscillation constructed of 10 different frequencies (sum of sine waves stimulus). It is expected that due to the unpredictable nature of the sum of sines stimulus, it will be more difficult to achieve a stable control strategy, and as a result, instability will be more likely and more prolonged than in the single sine wave condition. Sickness should occur more often in this complex stimulus condition than in the simple stimulus condition.

In addition to investigating the effect of predictability of imposed motion, differences due to method of stimulus presentation were also examined. As stated earlier, the acceptance of virtual environments as a method of training will in part be dependent on the ability of the environments to be used without occurrence of motion sickness. Two methods of displaying virtual environments in common use are large screen projections and head mounted displays (HMD). Both provide large fields of view, high display resolution, and immersive experiences. No difference is expected in the occurrence of motion sickness as a result of the different methods of presentation.

Methods

Participants

Fifty-one students enrolled in PSY 111 were used as participants in this study. Participants were not permitted to take part in the study if any of the following were true:
1) the participant had eaten less then two hours before the study,
2) the participant had consumed alcohol in the 24 hours previous to the study,
3) the participant had taken in the 24 hours previous to the study or is currently taking any medications that may effect the balance of the participant, or
4) the participant was taller than six feet (any taller and the participant ran the risk of being out of range of the emitter).

Materials

Two different questionnaires were used in this study. The first asked for basic demographic information (Appendix A). The second questionnaire asked the participant

to rate their current condition on several symptoms that have been linked to motion sickness (Appendix B). This simulator sickness questionnaire (SSQ) (Kennedy & Lane, 1993) was used to determine the extent to which immersion in the virtual environment affected the participant.

A magnetic tracking system called a Flock of Birds (Ascension, Inc., Burlington, VT) was used to track the motion of the participants. The system consisted of an emitter that creates a low-level magnetic field that extends one meter in every direction. Four sensors were attached to the participant's head, hip, knee, and ankle. The motion of the sensors disturbed the magnetic field, and this disturbance was then recorded by the computer at 40 Hz. The coordinate system was set up so that X was in the direction that the participant was facing, Y was movement to the left or right of the participant, and Z was movement up or down. The system recorded the position of each sensor in 6 degrees of freedom (anterior-posterior (AP) (X), lateral translation (Y), vertical translation (Z), pitch (rotation around Y), roll (rotation around X), and yaw (rotation around Z)).

Two different stimuli were used (between subjects). The first stimulus consisted of a star field (a pattern of random white dots on a black background) that increased and decreased in size in a simple sinusoidal fashion. This change in size simulated movement of the star field toward and away from the participant in a predictable fashion, with a frequency of .25 Hz. The second stimulus was again a star field, moving towards and away from the participant, but this stimulus was created using the sum of ten sine waves (.08, .17, .25, .33, .42, .50, .58, .66, .75, and .83 Hz). While several of these frequencies alone are outside the range that is typically naseogenic, the combination of the frequencies into one single waveform was expected to be nauseogenic due to the lack of predictability. The amplitude of each wave was adjusted to insure that the total power of the complex waveform was the same as that of the simple waveform. The phase of each sine wave was randomly determined and four separate complex waveforms were created.

Two computers were used in this study. The first computer, in conjunction with the projector (Sharp PG-C30XU, Mahwah, NJ) or HMD (i-O Display Systems i-glasses, Menlo Park, CA), displayed the stimulus. In the projector condition, the participant stood 2.7 meters from the wall that the image was displayed on. The stimulus image ranged from a diameter of 1.20 meters when the stimulus was the farthest from the participant to 1.30 meters when the stimulus was the closest to the participant (a change of .1 meters). This resulted in a maximum visual angle of 25.7 degrees vertically and horizontally. In the HMD condition, the display simulated an image that was 3.3 meters away, with a stimulus that ranged from 1 meters when it was farthest from the participant to 1.1 meters when it was closest to the participant (a change of .1 meters). This resulted in a maximum visual angle of 18.4 degrees vertically and horizontally.

The second computer, using a program called The Motionmonitor (Innovative Sports Training, Chicago, IL), recorded the motion of the participant provided by the Flock of Birds. This program displayed the real time motion of the participant, as well as computed various measurements of movement (e.g. velocity, variability) from the collected position and orientation data. This computer was electronically synchronized with the computer that displayed the stimulus in order to insure that data collection began exactly at the start of the stimulus beginning.

Procedure

Upon entering the laboratory, the participants were presented with a consent form (Appendix C). This consent form explained the purposes of the experiment and described the rights of the participant. After signing the consent form, the participants were asked to complete several questionnaires; a demographic questionnaire and the SSQ. The participants were asked to keep the symptoms described in the SSQ in mind during the experiment, and, in the event of an increase of the symptoms, inform the researchers. For safety, the participants were asked to complete two balance checks. The first involved walking a line in heel-toe fashion. In the second check, the participant was asked to stand on one foot with their eyes closed for thirty seconds. If the participant was able to complete the two balance checks, they continued in the experiment.

The participants were asked to stand comfortably in the laboratory while the researchers measured their postural sway. The experiment consisted of 14 trials. The first four trials measured the baseline sway of the participant. These trials lasted 20 seconds each. On two of the trials the participants had their eyes closed. The next four trials were control trials. In each control trial, the participants watched a computer-generated stimulus (simple sine wave) for one minute. Again, for two of the trials the participants had their eyes closed. After the control trials, four experimental trials were run. These trials utilized either the simple or complex waveform, but in this case the trials lasted for 10 minutes each. In the case of the complex conditions, the participants were exposed to each of the four complex waveforms in a partially counterbalanced order. The first four participants started with waveform one, two, three, or four, respectively, and then continued with the remaining trials in numerical order (E.g. participant two started with waveform two, and then continued with three, four, and finally one). Each successive group of four participants repeated the same order as above. The participant had their eyes open for all of the experimental trials. Finally, one baseline trial (eyes open) and one control trial (eyes open) was repeated after the experimental trials. The stimulus was presented in one of two ways; either using a head-mounted display (HMD), or using a projector that displayed the stimulus on a wall in front of the participant.

The stimulus was expected to influence the stance of the participant. This change in posture may lead to instability on the part of the participant, and as a result, symptoms of motion sickness. In the event of the participant indicating symptoms of motion sickness, the trials were stopped. The participant once again filled out the SSQ indicating the new level of their symptoms. They were asked to stay in the laboratory for observation for 15 minutes. After this time, if the participant felt better, they were allowed to leave after first repeating the two balance checks. If the participant had no symptoms of motion sickness at any time during the trials, they were asked to complete the SSQ after the fourth experimental trial. As before, the participants were only allowed to leave after completing the balance checks. In either case, the participants were given a third copy of the SSQ. In the event that the participant exhibited symptoms at some time (up to 24 hours) after leaving the laboratory, they were asked to fill out the questionnaire at that time and return it. If the participant had no symptoms, they were asked to complete and return the questionnaire approximately 24 hours after immersion in the virtual environment.

Results/Discussion

Overview

Initially, the major focus of the analysis was intended to examine characteristics related specifically to motion sickness, including the past sickness history of the participants, incidence rates, and postural motion (velocity, variability, and range of motion). Analysis of the velocity, variability, and range included an step-wise discriminant analysis (a form of regression analysis) of the experimental trials.

In examining the data, several analyses related to aspects of postural adaptation were added, including both descriptive analyses and a power spectrum analysis. These analyses were intended to examine the ability of the participants to adapt to the stimulus over time. Adaptation, in this case, was defined as an increase in the power at the frequencies contained in the stimulus relative to the total power at all the frequencies.

Motion Sickness

History. A total of 51 participants participated in the study, with a mean age of 20.54 (*SD* = 3.43) years. Twenty-two (43.1%) of the participants were female and 29 (56.9%) were male. Thirty-one (60.8%) reported having experienced motion sickness previously, 15 (29.4%) reported never having experienced motion sickness previously, and 5 (9.80%) were not sure. The mean susceptibility to motion sickness score was 3.57 (*SD* = 1.98) on a 10 point scale with 1 being "not susceptible" and 10 being "very susceptible". A t-test revealed no significant difference between the susceptibility rating of those participants who later became sick (*M* = 3.93, *SD* = 2.59) and those who did not become sick (*M* = 3.51, *SD* = 1.91), $t(49) = .627$, $p = .534$.

Incidence. In the first condition (simple sine wave stimulus using large-screen projector (**SP**)), 4 (4 female, 0 male) out of the 14 participants reported being sick. In the second condition (simple sine wave stimulus using HMD(**SH**)), 3 (2 female, 1 male) of the 13 participants reported being sick. In the third condition (complex sum of sines wave using large-screen projector (**CP**)), 2 (1 female, 1 male) of the 12 participants reported being sick. In the fourth condition (complex sum of sines wave using HMD(**CH**)), 5 (2 female, 3 male) of the 12 participants reported being sick. Previous research (Stoffregen & Smart, 1998; Stoffregen, Hettinger, Haas, Roe, & Smart, 2000; Smart, Stoffregen, & Bardy, 2002) has shown incidence rates ranging from 23% to 50% using various visual stimulations. While the current study predicted an increase in incidence rates in the complex conditions versus the simple conditions, it made no specific numerical prediction. Therefore, it only seemed appropriate to compare the observed rate in the current study to a predicted rate consistent with that of the results of previous research. A non-parametric (binomial) analysis was conducted using a predicted incidence rate of 33% in order to examine whether the complex conditions differed from the simple conditions. The analysis found no significant differences between the predicted rate and observed rate for any of the conditions. Condition CH had the highest incidence rate while condition CP had the lowest (Table 1).

In attempting to understand the incidence rates of the various conditions, it is important to make the point that the experimenter relied on the verbal report of the

participants to determine if a participant was becoming sick. The experimenters did observe the participants, and in the event that the experimenter felt that participants were visibly becoming sick, even if the participants did not report being sick, the experimenters would stop the experiment. In the current study, that situation never occurred. Consequently, only when the participants verbally reported being sick was the experiment stopped. On occasion, however, the participants did not report being sick, but their post-exposure SSQ scores reflected increased symptomology, sometimes severe when compared with pre-exposure scores, consistent with the onset of sickness. This suggested that some participants may have been becoming sick, but did not report their condition. Examination of the SSQ scores was needed to determine if this reluctance to report sickness was important in the outcome of the study.

SSQ. The SSQ data was analyzed to examine differences in the symptomology of the sick participants versus the well participants for pre-exposure scores and post-exposure scores. In a separate analysis, differences in the scores across conditions were also examined, with no significant differences being found in either the pre-exposure scores or the post-exposure scores (Table 2).

Total (overall) scale. A Kruskal-Wallis (non-parametric) test showed non-significant pre-exposure scores ($\chi^2_{(1)} = 1.132$, $p = .287$) but significant post-exposure scores($\chi^2_{(1)} = 12.732$, $p < .001$), indicating that while the pre-exposure scores for sick participants ($M = 7.48$, $SD = 7.19$) did not differ from the scores for well participants ($M = 11.51$, $SD = 11.13$), the post-exposure scores for the sick participants ($M = 55.03$, $SD = 18.70$) were significantly greater than the scores for the well participants ($M = 29.92$, $SD = 30.14$).

Disorientation subscale. A Kruskal-Wallis (non-parametric) test showed non-significant pre-exposure scores ($\chi^2_{(1)} = .019$, $p = .891$) but significant post-exposure scores($\chi^2_{(1)} = 11.771$, $p = .001$), indicating that while the pre-exposure scores for sick participants ($M = 4.97$, $SD = 6.92$) did not differ from the scores for well participants ($M = 7.50$, $SD = 14.23$), the post-exposure scores for the sick participants ($M = 56.67$, $SD = 28.61$) were significantly greater than the scores for the well participants ($M = 26.41$, $SD = 41.12$).

Nausea subscale. A Kruskal-Wallis (non-parametric) test showed non-significant pre-exposure scores ($\chi^2_{(1)} = 1.529$, $p = .216$) but significant post-exposure scores ($\chi^2_{(1)} = 12.955$, $p < .001$), indicating that while the pre-exposure scores for sick participants ($M = 4.77$, $SD = 4.95$) did not differ from the scores for well participants ($M = 8.07$, $SD = 8.05$), the post-exposure scores for the sick participants ($M = 44.97$, $SD = 18.87$) were significantly greater than the scores for the well participants ($M = 21.03$, $SD = 25.07$).

Oculomotor subscale. A Kruskal-Wallis (non-parametric) test showed non-significant pre-exposure scores ($\chi^2_{(1)} = .870$, $p = .351$) but significant post-exposure scores ($\chi^2_{(1)} = 5.136$, $p = .023$), indicating that while the pre-exposure scores for sick participants ($M = 8.66$, $SD = 10.24$) did not differ from the scores for well participants ($M = 12.83$, $SD = 14.46$), the post-exposure scores for the sick participants ($M = 44.94$, $SD = 20.27$) were significantly greater than the scores for the well participants ($M = 29.54$, $SD = 24.46$).

The SSQ results indicated that for the overall scale and three subscales, the participants' pre-exposure scores were not different from one another. This was important because it suggested that the participants began the study as equivalent, and therefore differences in pre- and post-exposures scores were a result of the experiment. Also, differences in post-exposure scores for sick and well participants helped to corroborate the verbal reports provided by the participants. The sick participants should have had higher post-exposure SSQ scores, and that was the case.

As stated previously, there were several occasions where participants did not report being sick, but their SSQ scores suggested that they may have started to become so. If a large enough number of well participants' SSQ scores were consistent with sick participant's scores, it would have been unlikely to find a significant difference in the post-exposure scores, as the mean scores for the well participants would have been elevated to be equal with the mean scores for the sick participants. The existence of this difference suggests that the well participants who had elevated SSQ scores did not substantially raise the overall scores, and therefore, in general, the differences between sick and well participants appeared to be genuine.

Postural Motion

Baseline trials. Velocity, variability, and range of motion of head movement in the anterior-posterior (AP) direction were analyzed in order to examine differences between the trials that the participants had their eyes open versus those with their eyes closed (within-subjects) and to examine differences between the sick participants and the well participants (between-subjects).

Velocity. A 2(eyes open vs. closed) x 2(sick vs. well) ANOVA produced a marginally significant main effect of sickness ($F(1,99) = 3.417$, $p = .067$), indicating that the baseline velocity for people who would later get sick ($M = .21$ cm/s, $SD = .71$ cm/s) was slightly higher than the baseline velocity for people who remained well ($M = .09$ cm/s, $SD = .24$ cm/s; Table 3a). This result is consistent with previous findings (Stoffregen & Smart, 1998), and suggests that participants who are susceptible to motion sickness may have different postural sway even before exposure. This potential predisposition may prove useful in predicting, prior to exposure to sickness-inducing environments, who is likely to become sick.

Variability. A 2(eyes open vs. closed) x 2(sick vs. well) ANOVA showed a marginally significant main effect of eyes ($F(1,99) = 3.326$, $p = .071$), indicating that participants with their eyes closed ($M = .7$ cm^2, $SD = .8$ cm^2) had a slightly higher baseline variability than participants with their eyes open ($M = .5$ cm^2, $SD = .6$ cm^2; Table 3b). This finding is consistent with previous research (Lee & Lishman, 1975) which suggested that postural sway is greatest when eyes are closed compared to when they are open.

Range. A 2(eyes open vs. closed) x 2(sick vs. well) ANOVA showed no significant main effects or interactions. (Table 3c).

Control trials. Velocity, variability, and range of motion of head movement in the anterior-posterior (AP) direction was analyzed in order to examine differences between the trials that the participants had their eyes open versus those with their eyes closed

(within-subjects), to examine differences between the sick participants and the well participants (between-subjects), and to examine differences between the conditions in which the stimulus was presented using large-screen projection or HMD (between-subjects). A 2(eyes open vs. closed) x 2(sick vs. well) x 2 (HMD vs. Projector) ANOVA revealed no significant interactions or main effects for either velocity, variability, or range of motion (Table 4). These findings are in general inconsistent with previous research (Stoffregen & Smart, 1998; Stoffregen, et. al., 2000; Smart, Stoffregen, & Bardy, 2002), which often found differences between sick and well participants and differences between eyes open trials and eyes closed trials. However, only data in the A-P direction was examined, and it is possible that differences existed in other axes of motion.

Experimental trials. A stepwise discriminant analysis was conducted using velocity, variability, and range of motion in the A-P direction as predictors. The rationale for this analysis was two-fold; first, the discriminant analysis could examine all three of the motion characteristics described above and determine which combination of these characteristics, if any, best separated the sick and well participants. This was done in a step-wise manner, using each characteristic one at a time. Second, due to the fact that a much larger number of trials existed in the well conditions versus the sick conditions, a discriminant analysis was better suited to deal with the unequal size groups than the Analysis of Variance used in the baseline trials and control trials. The analysis resulted in one discriminant function, Wilk's $\lambda = .960$; $\chi^2_{(1)} = 6.76$, $p = .009$. Differences in velocity classified participants into sick and well groups, accounting for 4% of the variance. The resulting function was:

$$y = 62.64*Vel - 0.47$$

The function accurately classified 78.3% of the cases into sick or well groups. The velocity was significantly greater for the sick participants than the well participants, $F(1, 149) = 6.918$, $p = .009$, but there was no significant difference between the sick and well participants for variability or range of motion (Table 5). Smart (2000) conducted two discriminant analyses in separate experiments that resulted in each case with one discriminant function, where vertical variability and lateral velocity, respectively, classified participants into sick and well groups. It is interesting to note that in the previous research the function was based on differences in axes other than that of the stimulus, while in the current research the function was based on differences in the axis of the stimulus (A-P).

Adaptation

Initially, the change in total power from the beginning of the trial to the end of the trial was examined, separately for the two conditions. For the simple conditions, a t-test revealed a significant difference between the total power at the beginning of the trial ($M = 1.47$, $SD = 2.15$) when compared to the end of the trial ($M = 6.37$, $SD = 16.1$), $t(180) = -2.88$, $p = .004$, indicating that the total power at the beginning of the trial was significantly less than at the end of the trial. For the complex conditions, a t-test revealed no significant difference in the overall power ($t(154) = -1.67$, $p = .098$), indicating that

the power at the beginning of the trial (M = 1.42, SD = 1.79) was not significantly different from the power at the end of the trial (M = 2.76, SD = 6.85).

Although the total power gave some indication of changes in the postural movement of the participants, it was important that the postural data be examined at the level of each frequency, especially those frequency(ies) that were contained within the stimulus that was presented. In examining the postural data at this level, only the participants classified as well were included in the analyses. This is because in order to understand the adaptation process over a time period that was both equal across participants and sufficiently long enough to observe adaptation, it was important that the participants were able to complete all four of the experimental trials, which the sick participants did not. For simplicity, effects due to the method of presentation were not examined, but rather were focused on the first and last minute of each of the four experimental (10 minute) trials (Trials 9 through 12, respectively).

Raw Data. The raw data were examined in order to identify from inspection whether or not the postural motion of the participants appeared to follow the pattern of the stimuli; in other words, if the participants were adapting to the stimulus. In the simple conditions, this would be reflected by motion that oscillated at .25 Hz, or 15 cycles in the one minute trial. In the complex conditions, because of the combination of 10 frequencies in the stimulus, it was not possible to simply look for evidence of movement at a particular number of cycles/minute. Rather the actual *pattern* of movement had to be examined to see if the participants' postural movement matched that of the stimulus. Representative examples of the data are shown in Figures 1-2 and 3-4, respectively.

For the simple conditions, it appeared that within a trial, the participants started out at frequencies lower than that of the stimulus (between 5 and 10 cycles/minute), but over time increased to frequencies that were closer to that of the stimulus (between 12 and 15 cycles/minute). This trend appeared to occur between trials as well, with later trials displaying frequencies closer to that of the stimulus. The adaptation did not totally carry over from one trial to the next, but rather the participant would often begin at frequencies lower than that of the end of the previous trial, but higher than that of beginning of the previous trial. However, in general, the participant ended the succeeding trial at frequencies higher than that of the preceding trial. This suggests that the breaks between trials were slightly detrimental to the adaptation process, but did not completely prevent it. Overall, it appeared that the participants were adapting to the stimulus over time.

For the complex conditions, there was little evidence to indicate that the participants were adapting to the stimulus. Occasionally, the data would appear to partially match the stimulus, but even this was relatively rare. In fact, comparing the data to the 20 second baseline trials (assuming that the baseline trials would be fairly consistent over 60 seconds), the complex data almost appeared to be closer to that of the baseline trials than the stimulus to which the participants were exposed (Figure 5). This suggested that the complex stimulus did not have a significant effect on the participants.

Power spectrum. Inspection of the raw data seemed to indicate that the participants were adapting to the simple stimulus but not to the complex stimulus, but further analysis using objective measures was needed to help support this. Fourier analysis of the postural

motion allowed for the examination of the power at the frequencies contained in the stimulus, as well as the surrounding frequencies. In both the simple and complex conditions, the frequencies examined ranged from 1 cycle/minute (.02 Hz.) to 50 cycles/minute (1 Hz.). This guaranteed that all 10 of the frequencies in the complex conditions were included.

In the simple conditions the power spectrum, like the raw data, indicated that the power at the stimulus frequency (15 cycles/minute) increased within a trial and continued to increase between trials (Figure 6). This again suggests that the participants were adapting to the simple stimulus over time. In the complex conditions the power spectrum, as was the case when looking at the raw data, indicated that the power at the stimulus frequencies was not increasing substantially, and in many cases, was decreasing (Figure 7). This suggests that the participants were not adapting to the stimulus over time. Looking at these power spectrums compared to a power spectrum of a baseline trial, the complex stimulus trials again look more similar to the baseline trials than the simple stimulus trials do. (Figure 8).

Analysis of Variance. A 4(trial) x 2(time) x 50 (cycles) ANOVA was conducted to examine differences in the distribution of power between trials and within trials. All three variables were within subjects. For the simple conditions, a trial by time by cycles interaction was found, $F(147, 2499) = 1.541$, $p < .001$, indicating that the distribution of power was significantly different both between trials and within a single trial. For the complex conditions, a trial by cycles interaction was found, $F(147, 2205) = 1.576$, $p < .001$, indicating that the distribution of power was significantly different between trials.

Power ratios. In order to understand the nature of the interactions resulting from the above ANOVA, distributions of the ratios of the power were created both between trials and within a trial for both the simple and complex conditions. Three distributions were created for the between trials, corresponding to the ratio of the second trial to the first, the third trial to the first, and the fourth trial to the first (Figure 9, 10). Within trials, four distributions were created, corresponding to the ratio of the last minute to the first minute, for each of the four trials individually (Figure 11, 12). These ratios, unlike simply calculating differences in power from one time period to the next, indicate the change in power based on a factor that is not influenced by the absolute power at a particular frequency.

In the simple conditions, the power ratios suggest a growing ratio as the trials progressed. The ratio of the third trial to the first trial (Figure 9b) has some increases in power at all frequencies, but especially around the stimulus frequency (15 cycles/minute) and interestingly, at 35 cycles/minute, which could be evidence of postural response at a harmonic of the stimulus frequency. This trend is even more strongly displayed in the ratio of the fourth trial to the first (Figure 9c). Again, there appears to be higher ratios at all frequencies, but especially at the stimulus frequency and possibly a harmonic of the stimulus frequency. Within a trial, the power ratios again show evidence of increases in power from the beginning of a trial to the end, especially during the fourth trial. This increase seems to occur most commonly at frequencies at and surrounding the stimulus frequency (and possible a harmonic), indicating that the effects found in the ANOVA are due in a substantial manner to changes in power at the stimulus frequency.

In the complex condition, the power ratios between trials (Figure 10) show increases at all frequencies, but not differentially at the frequencies contained within the stimulus. There does seem to be a greater increase at the higher frequencies when compared to the lower frequencies, and this may be the reason for the interaction found in the ANOVA, but there is no substantial increase at the stimulus frequencies like that of the simple stimulus conditions. Within a trial (Figure 12), there again is evidence of increases at all frequencies, but not specifically at the stimulus frequencies. Also there does not appear to be a differential change at any one particular group of frequencies versus another, which explains the lack of a time by power interaction found in the single stimulus conditions.

Conclusions

The relative unpredictability of the complex stimulus compared to the simple stimulus did not result in an increase in sickness incidence, and there appeared to be no significant differences in the incidence between the two venues. Across conditions, various aspects of postural motion showed differences between sick and well participants during non-stimulus (baseline) trails, suggestive of possible predisposition to sickness, and velocity was able to successfully classify participants into sick and well groups, indicating that postural motion may be useful in prediction of sickness. Examination of the power of the postural motion revealed an ability of participants to adapt to a simple stimulus versus a relative inability to adapt to a complex stimulus.

The lack of a significantly larger number of people becoming sick in the complex condition generally contradicts what was predicted, although condition CH did result in the most participants becoming sick. It was expected that the unpredictable nature of the complex stimulus would make it more difficult for participants to adapt, and this difficulty would lead to an increase in the occurrence of motion sickness. The lack of an increase in the occurrence of motion sickness could be explained by several reasons: 1) the complex stimulus was not unpredictable enough, 2) the participants were able to adapt to the complex stimulus, 3) the motion had no consequence for the action of the participants, and therefore the participants, in essence, were able to ignore the stimulus.

Since the power spectrum analysis indicated that the power at the stimulus frequencies did not increase over time in the complex conditions, it seems unlikely that the participants were adapting to the stimulus. Likewise, it is unlikely that the participants completely ignored the stimulus, due the fact that at least some of the participants became sick, and it would be unlikely that they would become sick in the absence of any stimulation. An interesting observation to be made concerns condition CH (complex stimulus, HMD). Although a significantly greater number of people did not become sick in this condition, the fact that five participants became sick compared to two in condition CP may suggest that there is something different about the two conditions. That difference may be that in condition CH the participants were physically attached to the HMD, and consequently it would be more difficult for the participants to “look away” (and hence ignore it) from the stimulus than in the projector condition. This inability to escape the stimulus may be the cause for the slight increase. However, this would also suggest an increase in sickness occurrence in condition SH compared to SP, which did not happen.

It is possible that the complex stimulus was not unpredictable enough to cause a significant increase in the occurrence of motion sickness compared to the simple stimulus. Previous research (Stoffregen & Smart, 1998) found that oscillations that mimic postural sway were nauseogenic, which suggests that as the motion of a stimulus becomes closer to that of postural sway, the more likely a person exposed to such a stimulus will become sick. The logic behind this is that motion similar to postural sway will interfere with the postural sway and cause a disruption. The unpredictable nature of the complex stimulus was closer in nature to that of postural sway than the simple stimulus because it contained multiple frequencies like normal postural sway, but perhaps it did not create a large enough interference effect. In future research, it would be interesting to use a stimulus that is dynamically created based on the motion of the actual participant. A stimulus such as this would directly mimic postural sway, be highly unpredictable, and should create a large interference effect. It would be expected that a large number of participants would become sick in such an experiment. From this, various frequencies could be removed until a relatively low number of participants became sick. This might suggest a threshold for how close a stimulus must be to postural sway to create an substantial interference effect.

A secondary purpose of the current study was to determine if differences in the occurrence of motion sickness would exist depending of the method of presentation utilized, or as described here, the venue. The incidence rates were not found to be significantly different for the HMD and projector conditions, as was predicted and is consistent with previous findings (Smart, 2000). This suggests that similar postural behaviors existed in both venues, which helps to support the suggestion that postural instability theory may be able to account for sickness across venues, reducing the necessity for the practice of creating separate categories of sickness, typically based on symptomology or merely as a matter of empirical convenience. However, as stated earlier, the slight increase in the number of participants sick in condition CH compared to CP might suggest that some difference may exist.

The ability of the participants to adapt to the simple stimulus even though they were not explicitly told to do so may provide some clue as to the mechanism for the coordination of posture in novel situations. It appears that the most stable strategy for control was to become entrained with the stimulus and allow the environment to specify the most appropriate behavior. This is evidenced by the shift in power from baseline frequencies around .02-.04 Hz to frequencies similar to that of the simple stimulus (.25 Hz) and the first harmonic of the stimulus (.50 Hz) (a possible indication of a non-linear response to the stimulus). This, however, may not be the case in all situations. As the complex stimulus condition suggested, there may be instances when adaptation is either not possible, or not beneficial. Again, the power analysis suggested that participants were not shifting power from the baseline frequencies to coincide with the frequencies of the complex stimulus. Future research that might help understand further when adaptation occurs and how it is implemented includes studying the coordination patterns between multiple joints and segments, the addition of a superpostural task to change the goal of the behavior, and the further manipulation of frequency, amplitude, phase, and complexity.

The examination of the circumstances under which motion sickness occurs is critically important for our understanding of how to prevent it. In this study, comparing a

relatively complex stimulus versus a relatively simple stimulus gave some insight into how the manipulation of predictability influences motion sickness, and also in what situations postural adaptation occurs. This study serves as a starting point for further examination of both predictability and adaptation, and their consequences for motion sickness.

References

Bensel, C.K. & Dzendolet, E. (1968) Power spectral density analysis of the standing sway of males. *Perceptual Psychology, 4,* 285-288.

Biocca, F. (1992). Will simulation sickness slow down the diffusion of virtual environment technology? *Presence, 1,* 334-343.

Gibson, E.J., & Pick, A.D. (2000). *An Ecological Approach to Perceptual Learning and Development*. New York: Oxford University Press.

Kennedy, R.S. & Fowlkes, J.E. (1992). Simulator sickness is polygenic and polysymptomatic: Implications for research. *International Journal of Aviation Psychology, 2*(1), 23-38.

Kennedy, R.S., Hettinger, L.J. & Lilienthal, M.G. (1990). Simulator Sickness. In Crampton, G.H. (Eds.), *Motion and Space Sickness,* (pp. 317- 341). Boca Raton: CRC Press. Inc.

Kennedy, R. S., Lane, N. E., Berbaum, K. S., & Lilienthal, M. G. (1993). Simulator sickness questionnaire: An enhanced method for quantifying simulator sickness. *International Journal of Aviation Psychology, 3,* 203–220.

Kennedy. R.S., Lane, N.E., Lilienthal, M.G., Berbaum, K.S. & Hettinger, L.J. (1992). Profile analysis of simulator sickness symptoms: Applications to virtual environment systems. *Presence, 1*(3), 295-301.

Kennedy , R.S., & Stanney, K.M. (1996). Postural instability induced by virtual reality exposure: Development of a certification protocol. *International Journal of Human-Computer Interaction, 8*(1), 25-47.

Oman, C.M. (1982). A heuristic mathematical model for the dynamics of sensory conflict and motion sickness. *Acta Otolaryngologica, 44,* (Suppl. 392).

Oman, C.M., Lichtenberg, B.K., Money, K.E., & McCoy, R.K. (1986). MIT/Canadian vestibular experiments on the Spacelab-1 mission: 4. Space motion sickness: symptoms, stimuli, and predictability, *Experimental Brain Research, 64*, 316-334.

Reason, J.T., & Brand, J.J. (1975). *Motion sickness*. London: Academic Press.

Reed, E.S. (1996). *Encountering the World: Toward an Ecological Psychology*. New York: Oxford University Press.

Riccio, G. E. & Stoffregen, T. A. (1991). An ecological theory of motion sickness and postural instability. *Ecological Psychology, 3,* 195-240.

Smart., L.J. A comparative analysis of visually induced motion sickness. (Doctoral dissertation, University of Cincinnati, 2000).

Smart, L.J., Stoffregen, T.A., & Bardy, B.G. (2002). Visually induced motion sickness predicted by postural instability. *Human Factors, 44*(3), 451-465.

Stoffregen, T.A., Hettinger, L.J., Haas, M.W., Roe, M.M., & Smart, L.J. (2000). Postural instability and motion sickness in a fixed-base flight simulator. *Human Factors, 42*(3), 458-469.

Stoffregen, T. A., & Smart, L.J. (1998). Postural instability precedes motion sickness. *Brain Research Bulletin, 47,* 437-448.

Appendix A

Motion Sickness History

Participant # ____

Age:

Gender:

Height:

Weight:

1. Corrected Vision? Yes / No
    a. If yes, wearing glasses / contacts

2. Have you ever been motion sick? Yes / No / Not sure
    a. If yes, where and under what circumstances?

_____

_____

3. How would you rate your susceptibility to motion sickness on a scale of 1(not susceptible) – 10(very susceptible)? ____

4. Have you ever used virtual reality prior to today? Yes / No
    a. If yes, what kind and under what circumstances?

Experimenter Use:

1. Vection?

2. Other Comments/ Observations?

Appendix B

Short Form:
Instructions: Circle the items that apply to you RIGHT NOW.

SYMPTOM	RATING			
1. General Discomfort	None	Slight	Moderate	Severe
2. Fatigue	None	Slight	Moderate	Severe
3. Headache	None	Slight	Moderate	Severe
4. Eye Strain	None	Slight	Moderate	Severe
5. Difficulty Focusing	None	Slight	Moderate	Severe
6. Salivation Increased	None	Slight	Moderate	Severe
7. Sweating	None	Slight	Moderate	Severe
8. Nausea	None	Slight	Moderate	Severe
9. Difficulty Concentrating	None	Slight	Moderate	Severe
10. "Fullness of the Head"	None	Slight	Moderate	Severe
11. Blurred Vision	None	Slight	Moderate	Severe
12. Dizzy (eyes open)	None	Slight	Moderate	Severe
13. Dizzy (eyes closed)	None	Slight	Moderate	Severe
14. Vertigo	None	Slight	Moderate	Severe
15. Stomach Awareness**	None	Slight	Moderate	Severe
16. Burping	None	Slight	Moderate	Severe

17. Other: Please describe _____

** "Stomach Awareness" usually is used to indicate a feeling of discomfort that is just short of nausea.

Part # _____ Date _____ Pre / Post

Appendix C

Letter of consent

Research Title: Postural Responses to Virtual Environment Immersion || **Project Director**: Dr. L. James Smart

Before signing form, please answer the following:

Do you have any history of disease of malfunction of your vestibular (inner ear) apparatus, or of postural instability, recurrent dizzyness, falls, etc.? Have you had any recent illnesses less than 24 hours prior to now? Have you taken any allergy/cold medication or consumed more that 16 oz of alcohol in the past 24 hours? Have you eaten within the past two hours?

yes_____ no_____

If you respond 'yes' to any of the above, you will be excused from this study with full credit given.

We are conducting research on the relationship between postural control and virtual environment use. You will be asked to sit comfortably in the laboratory while we measure your postural activity (sway). You will be wearing a head-mounted display (HMD) that will display a computer generated environment; this visible motion may influence your stance. The entire testing procedure takes about 40 mintues. While you are in the experimental room, or shortly thereafter, you may begin to experience symptoms of motion sickness (discomfort, stomach awareness, nausea). If these symptoms begin, you should say so, and the experiment will be stopped immediately. Before and after use of the HMD you will be asked to sit still for a few seconds with your eyes open, and then closed.

It is expected that about 50% of participants will experience symptoms of motion sickness. If motion sickness symptoms do not develop, the experiment will end after about 30 minutes of exposure to the visual stimulus. If you do develop motion sickness symptoms, the experimenter will provide you with a place to lie down if you want to. You will be allowed to rest for as long as you wish. There is a very slight possibility that one could faint or momentarily lose consciousness, although this is extremely unlikely. During your exposure to the virtual scene, there will be 'spotters' to safeguard against falls. Whether symptoms of motion sickness are experienced or not, you will be required to remain in the laboratory for at least 15 minutes after exposure. You will also be given a take-home symptom checklist, which must be returned to the primary investigator after 24 hrs.

From this research we expect to learn something about the relationship between virtual motion and postural control. Your name will not be included in any reports or discussions of this work.

Your participation should take up to 60 minutes (including the 15 min recovery period). You are free to stop your participation in this experiment at any time for any reason. You will still receive full credit for participation (2 credit hrs). You may obtain a written summary of the results of this experiment by providing us with a stamped envelope with your permanent address.

If you feel that you need medical attention following your participation, you can receive care at the Campus Health Center (529-3000), located across from the Campus Avenue Building. If it is necessary to receive care, the investigator will arrange for transport and accompany you to the health center. No other compensation is offered in the event of an unusual incident, other than assistance for obtaining emergency care.

If you believe you have been injured as a result of this research, or if you have any other questions about this study, you should contact Dr. L. James Smart at 529-1656. You can also reach Dr. Karen Maitland Schilling (the department chair) at 529-2400. For additional information about your rights as a participant you may contact the Office for the Advancement of Scholarship and Teaching (OAST) at 529-3734.

"*I, the undersigned, have understood the above explanations and given consent to my voluntary participation in 'Postural Responses to Virtual Environment Immersion'.*"

_____ _____

Signature of participant or legal representative Date

_____ _____

Signature of investigator Date

Revised 7/15/02

Table 1

Non-parametric (binomial) analysis of sickness incidence rates across conditions.

Condition	Incidence		Observed Proportion		Test Proportion	Sig. (1-tailed)
	Sick	Well	Sick	Well		
SP	4	10	0.29	0.71	0.33	0.486
SH	3	10	0.23	0.77	0.33	0.332
CP	2	10	0.17	0.83	0.33	0.188
CH	5	7	0.42	0.58	0.33	0.359

Table 2

Mean SSQ scores across conditions.

Condition	Pre-exposure score		Post-exposure score	
	M	*SD*	*M*	*SD*
SP (N = 14)	12.4	13.3	46.3	40.9
SH (N = 13)	9.35	11.0	35.5	21.9
CP (N = 12)	11.5	6.90	29.6	23.6
CH (N = 12)	8.92	9.72	28.8	26.3

Note. A 2 X 2 ANOVA revealed no significant differences based on stimulus (p = .169) or condition (p = .335) for either the pre-exposure scores or the post-exposure scores (separately).

Table 3

Mean postural motion (baseline trials).

3a. Velocity (cm/s)

	Sick		Well	
	M	*SD*	*M*	*SD*
eyes open	0.19	0.70	0.11	0.42
eyes closed	0.23	0.73	0.06	0.05

3b. Variability (cm^2)

	Sick		Well	
	M	*SD*	*M*	*SD*
eyes open	0.40	0.30	0.60	0.60
eyes closed	0.70	0.90	0.60	0.80

3c. Range (cm)

	Sick		Well	
	M	*SD*	*M*	*SD*
eyes open	2.5	1.0	2.5	1.4
eyes closed	2.9	1.4	2.5	1.4

Table 4

Mean postural motion (control trials).

3a. Velocity (cm/s)

		Sick		Well	
		M	*SD*	*M*	*SD*
PRO					
	eyes open	0.04	0.04	0.03	0.03
	eyes closed	0.01	0.04	0.03	0.04
HMD					
	eyes open	0.02	0.03	0.02	0.02
	eyes closed	0.02	0.01	0.07	0.02

3a. Variability (cm^2)

		Sick		Well	
		M	*SD*	*M*	*SD*
PRO					
	eyes open	1.0	1.2	0.9	1.0
	eyes closed	1.1	0.9	1.4	2.7
HMD					
	eyes open	1.0	1.4	1.4	1.9
	eyes closed	1.3	1.2	1.2	1.3

3a. Range (cm)

		Sick		Well	
		M	*SD*	*M*	*SD*
PRO					
	eyes open	4.1	1.2	4.1	1.9
	eyes closed	4.4	1.3	4.5	2.6
HMD					
	eyes open	3.7	1.5	4.4	2.5
	eyes closed	4.4	1.9	4.3	2.2

Table 5

Mean postural motion (experimental trials, discriminant analysis).

	Sick		Well	
	M	*SD*	*M*	*SD*
Velocity (cm/s)	0.01*	0.040	0.002*	0.011
Variability (cm^2)	5.9	7.9	4.5	7.8
Range (cm)	13.4	11.3	10.8	7.00

* Significant difference, $p = .003$.

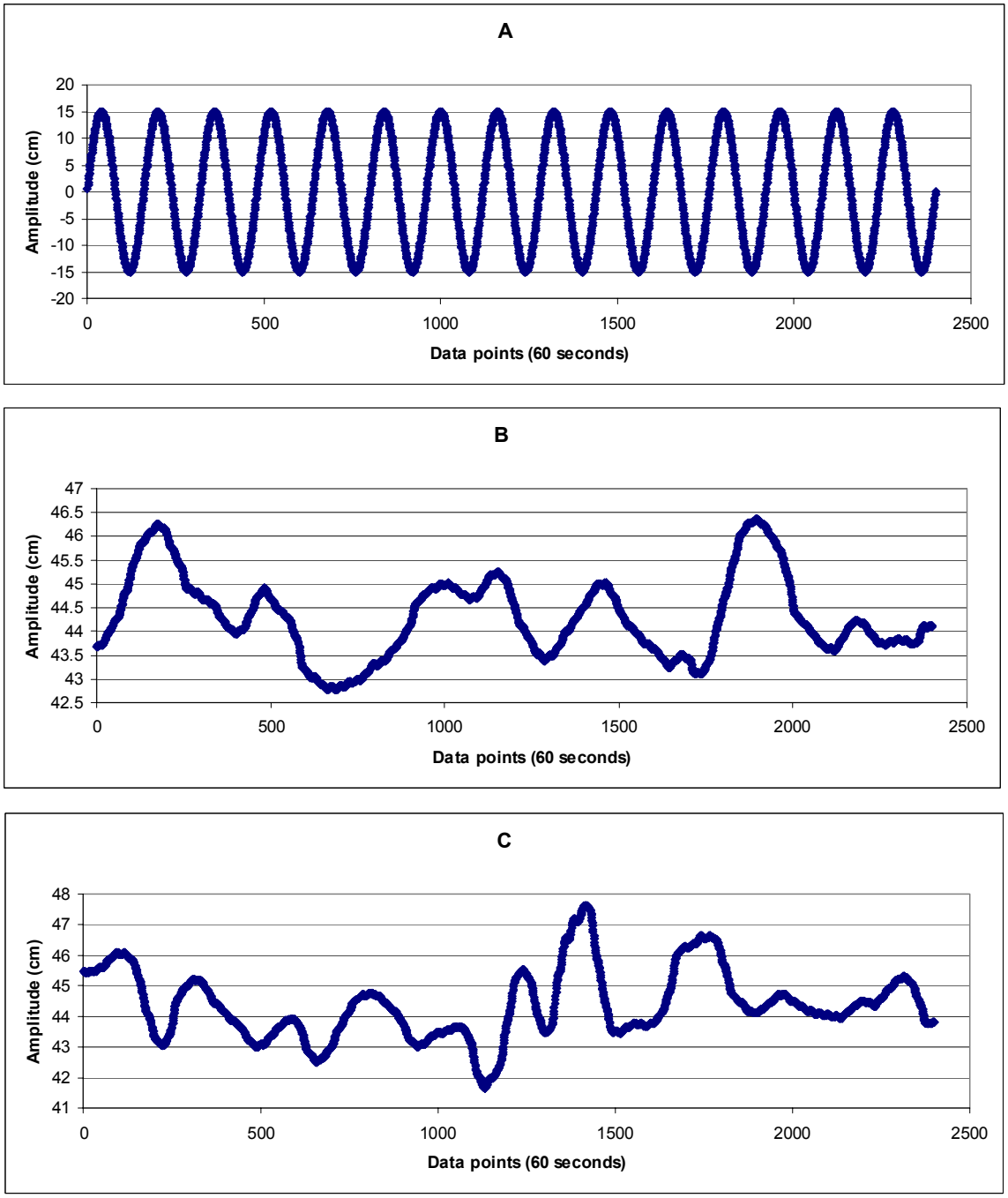


Figure 1. (A) One minute sample of the single sine wave stimulus. (B) First minute of first experimental trial (Trial 9 overall) for participant AL. (C) Last minute of first experimental trial (Trial 9 overall) for participant AL.

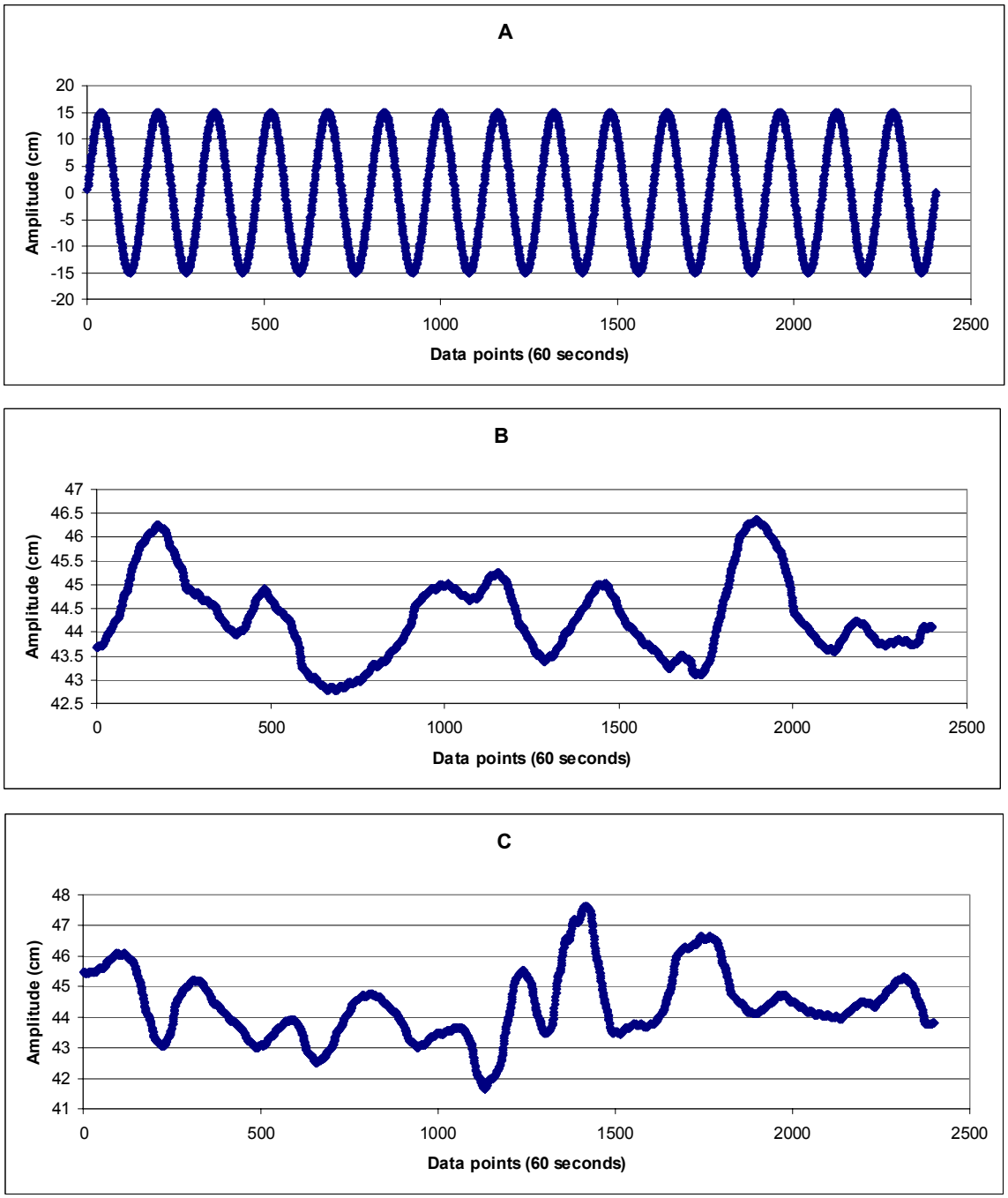


Figure 2. (A) One minute sample of the single sine wave stimulus. (B) First minute of last experimental trial (Trial 12 overall) for participant AL. (C) Last minute of last experimental trial (Trial 12 overall) for participant AL.

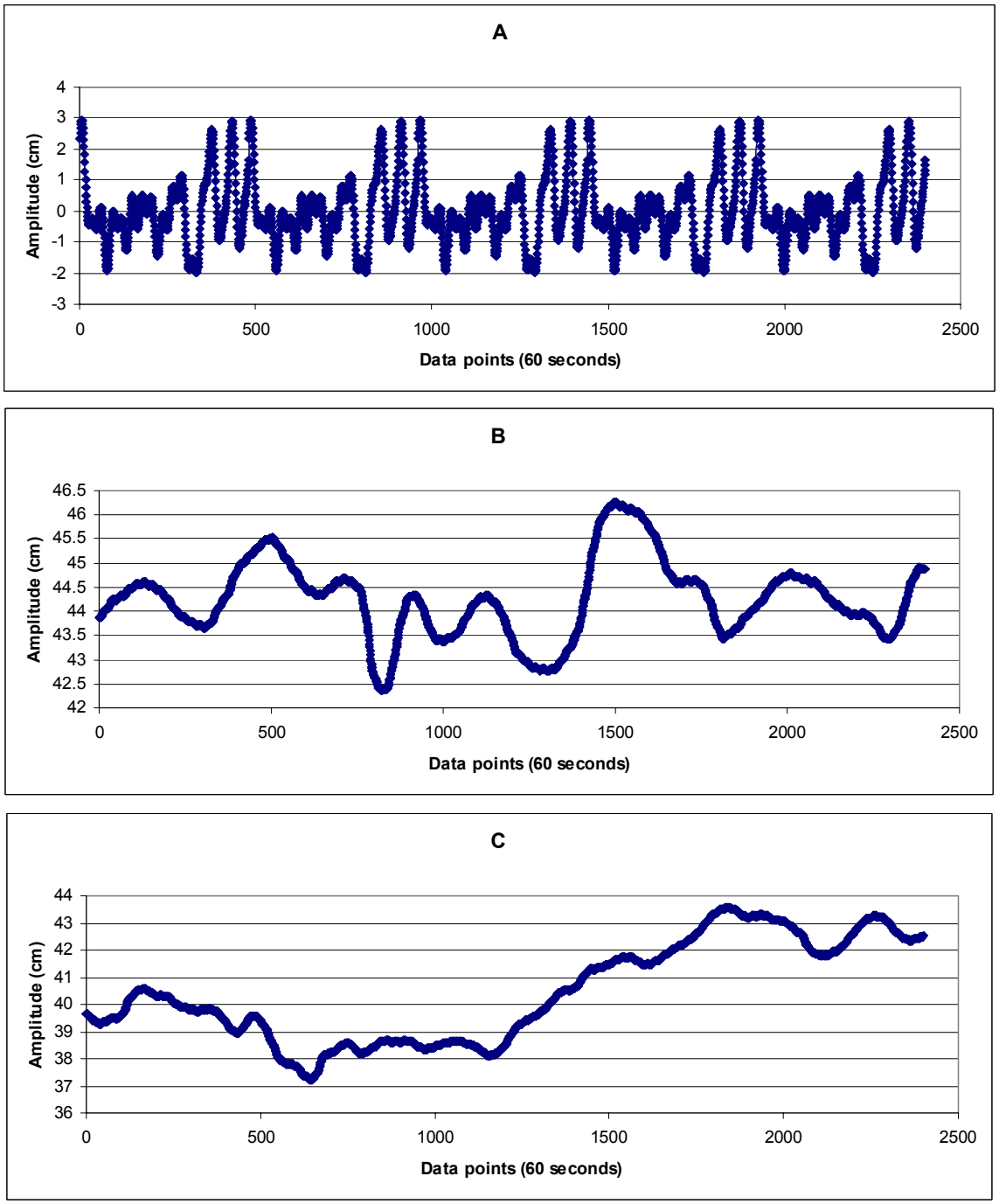


Figure 3. (A) One minute sample of one sum of sines wave stimulus. (B) First minute of first experimental trial (Trial 9 overall) for participant DO. (C) Last minute of first experimental trial (Trial 9 overall) for participant DO.

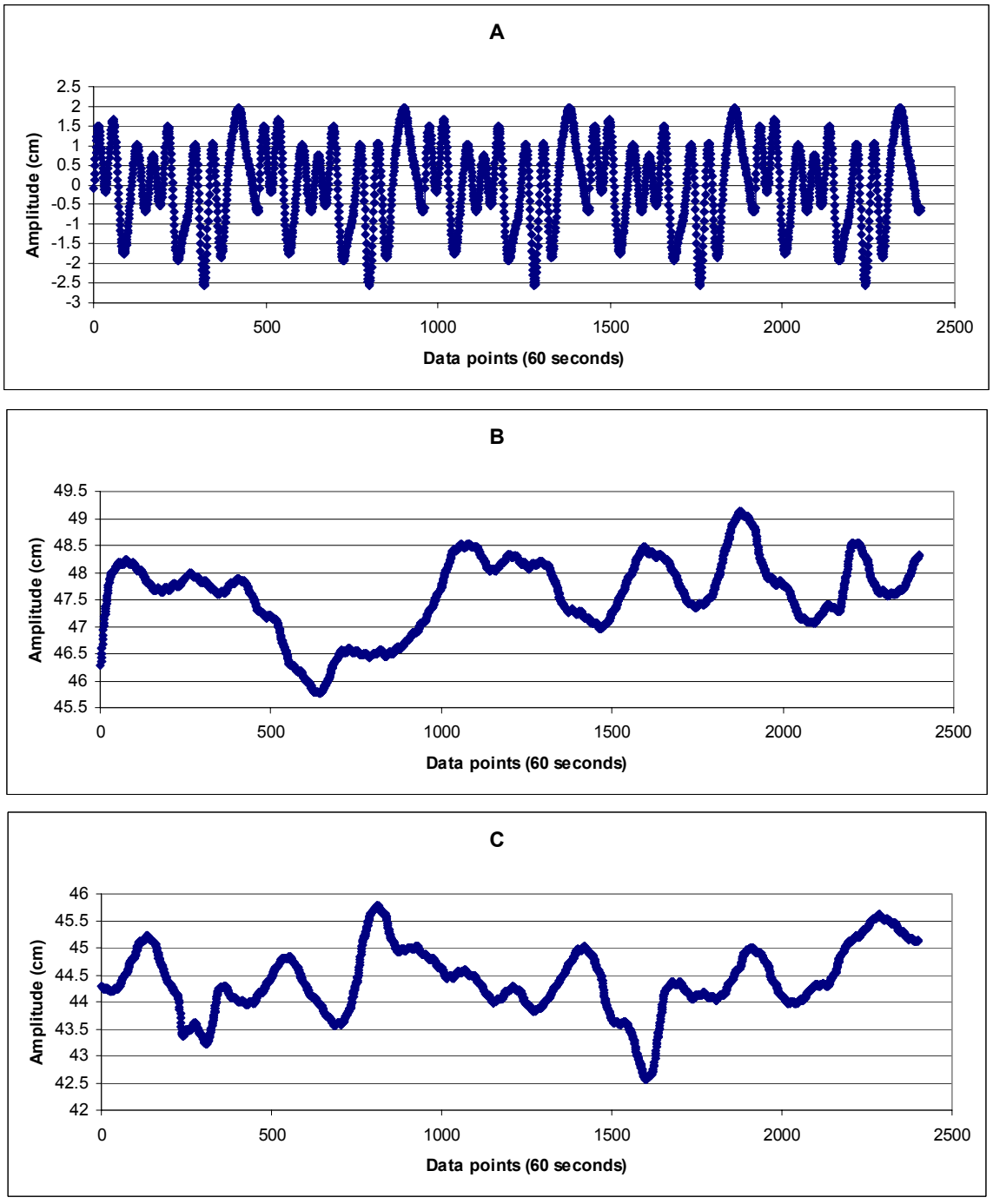


Figure 4. (A) One minute sample of one sum of sines wave stimulus. (B) First minute of last experimental trial (Trial 12 overall) for participant DO. (C) Last minute of last experimental trial (Trial 12 overall) for participant DO.

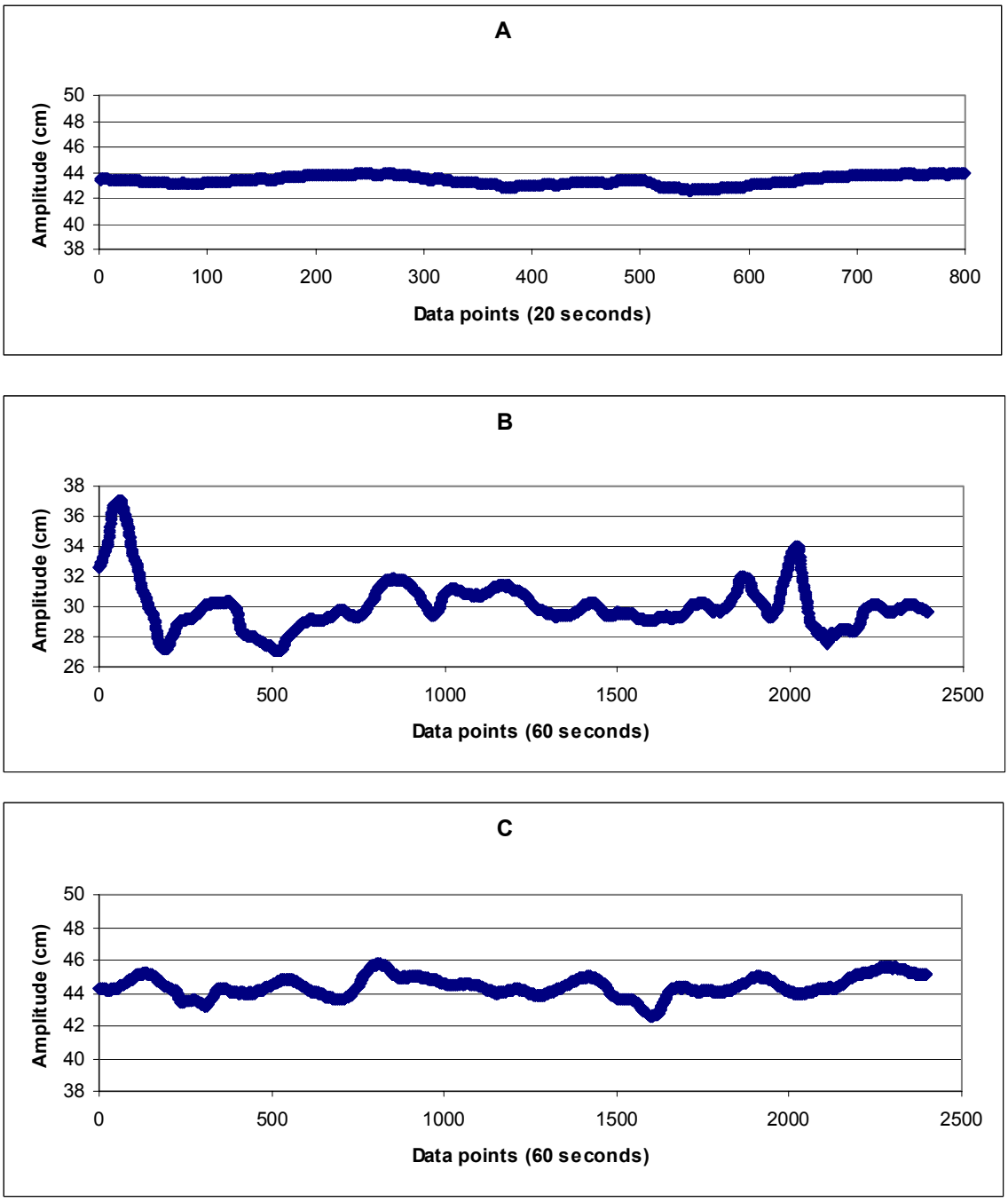


Figure 5. (A) Baseline trial. (B) Single sine wave stimulus trial. (C) Sum of sines wave trial.

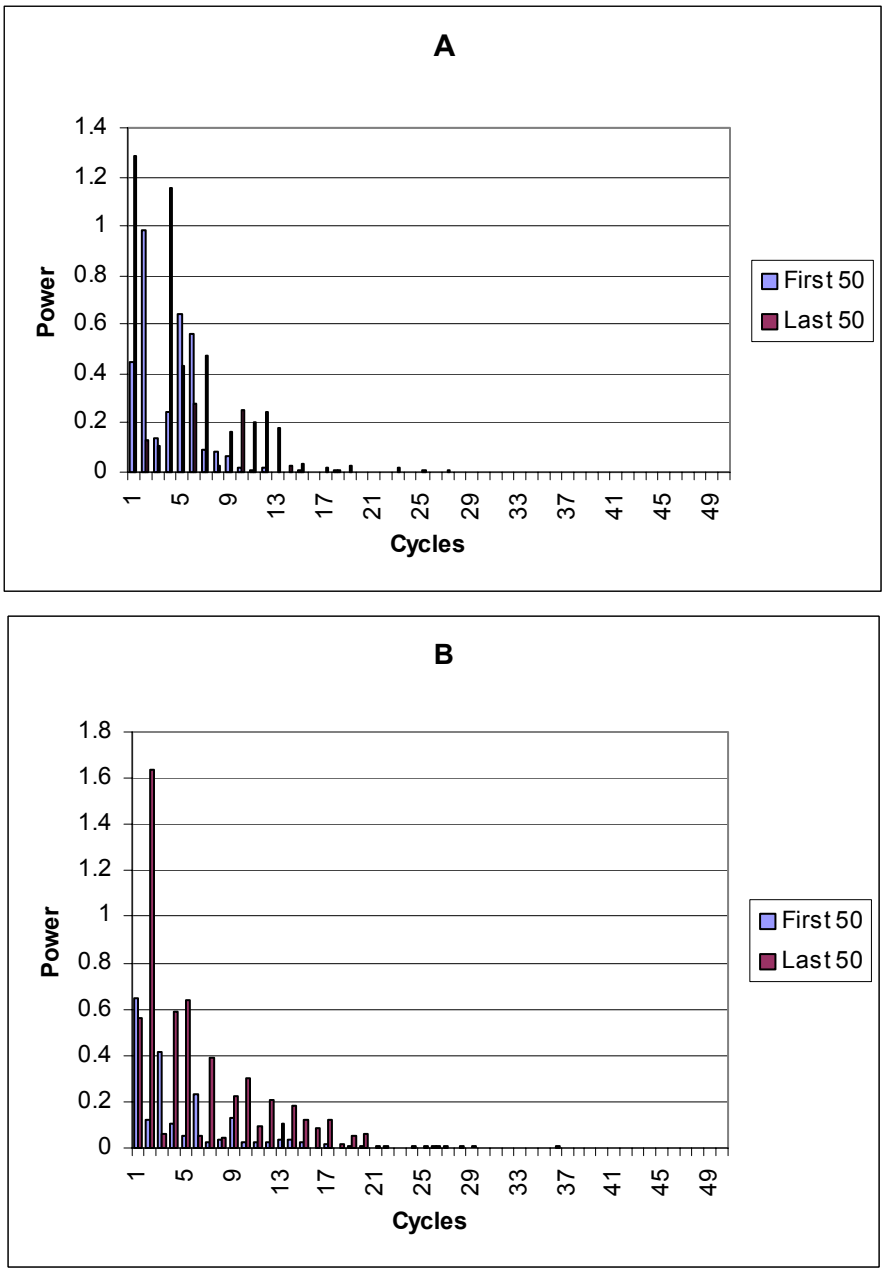


Figure 6. (A) Power spectrum of first experimental trial (trial 9 overall) for participant AL. (B) Power spectrum of last experimental trial (trial 12 overall) for participant AL.

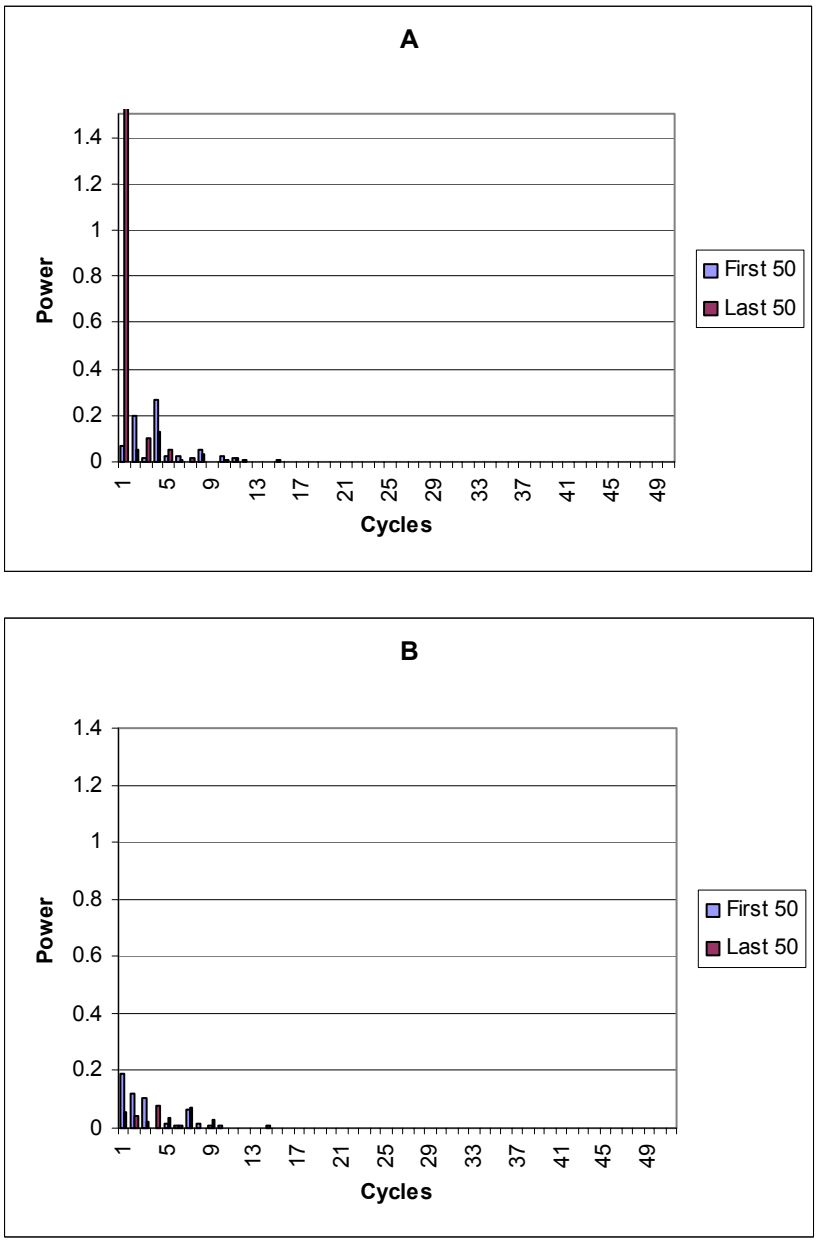


Figure 7. (A) Power spectrum of first experimental trial (trial 9 overall) for participant DO. (B) Power spectrum of last experimental trial (trial 12 overall) for participant DO.

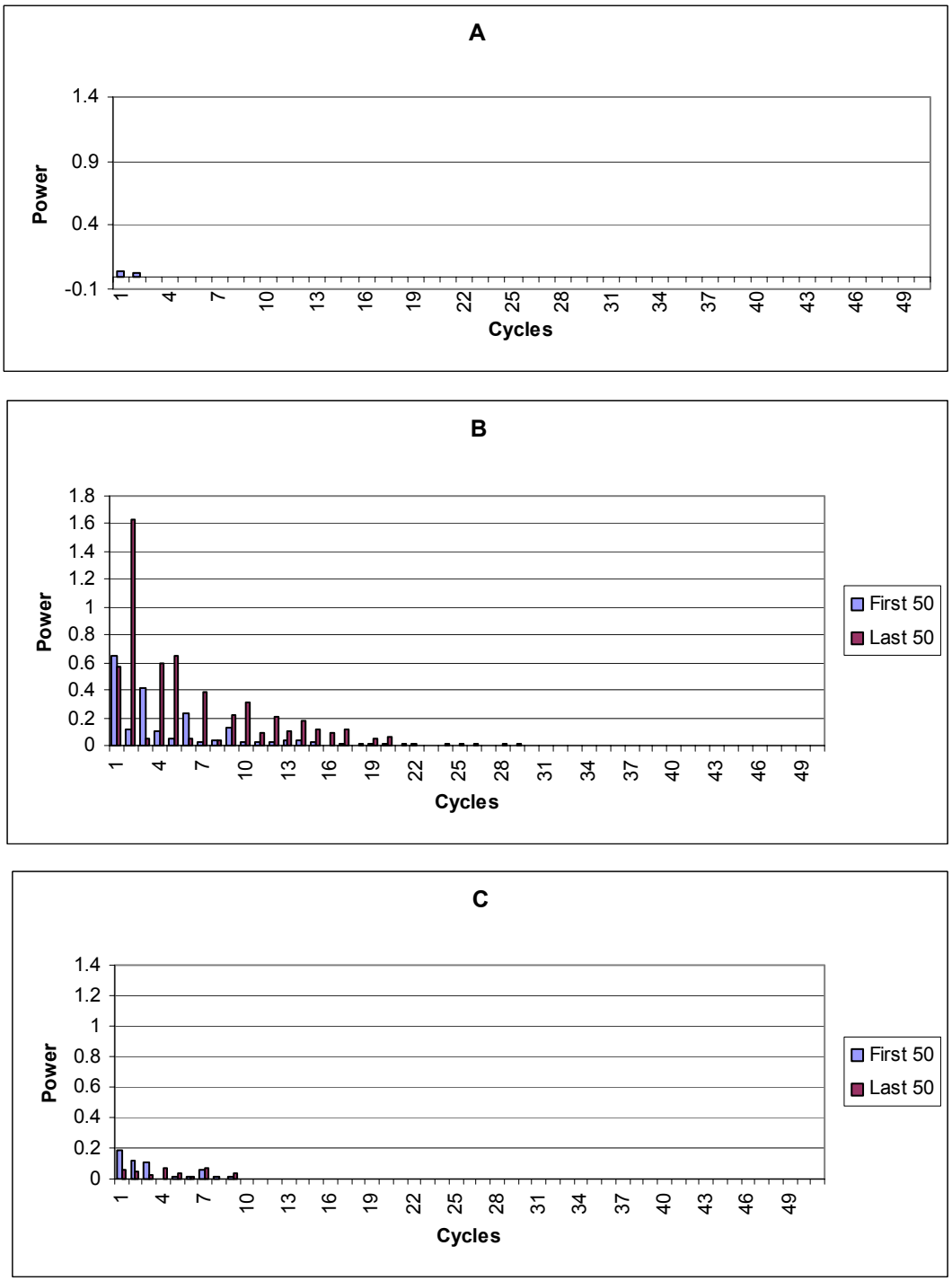
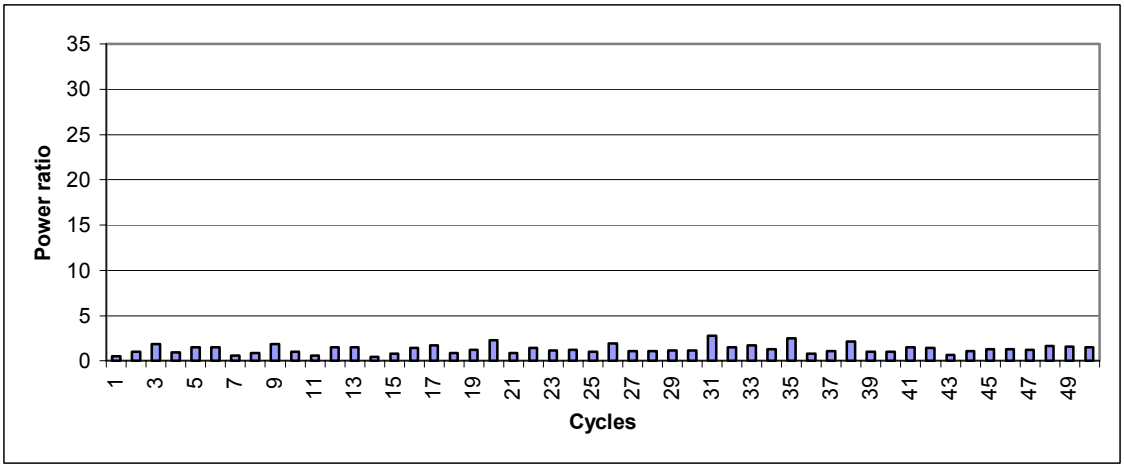
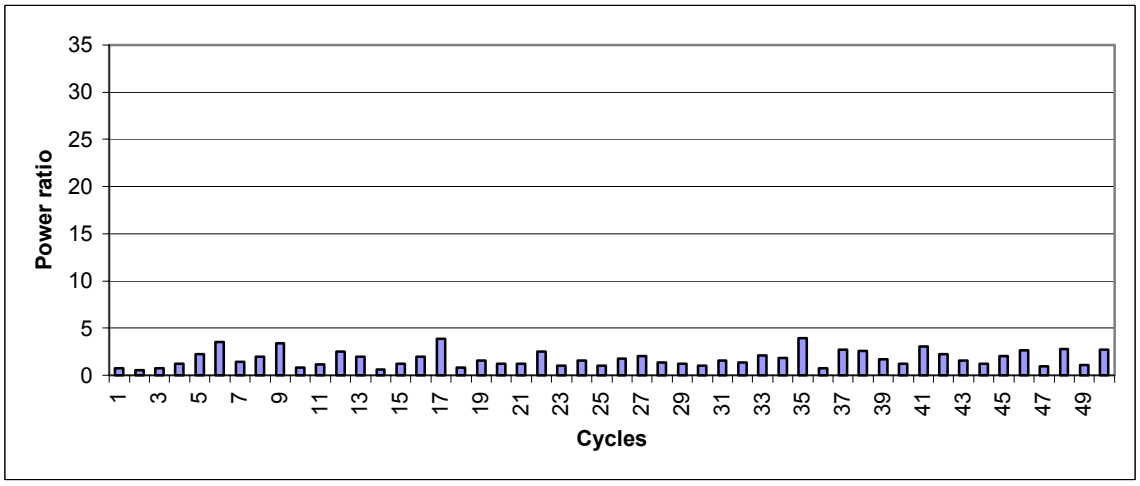


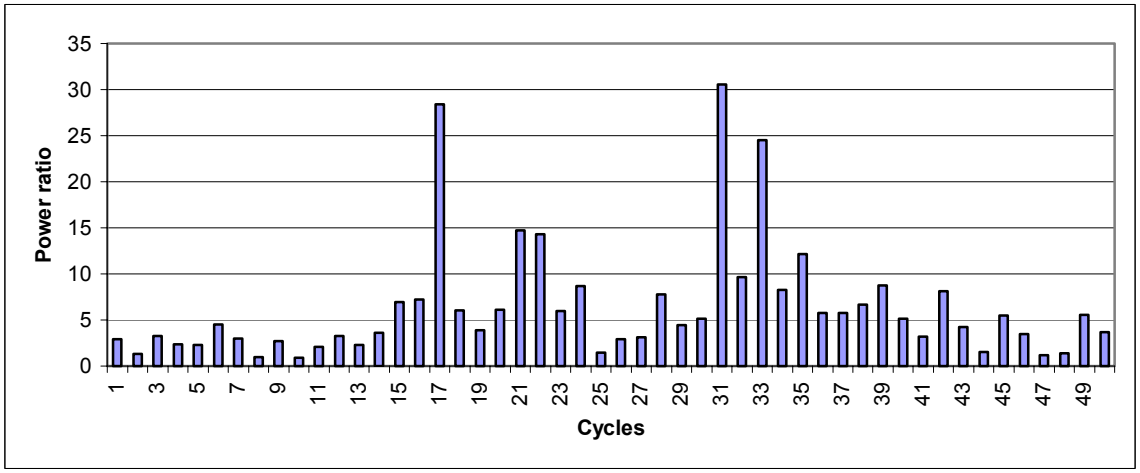
Figure 8. (A) Baseline trial. (B) Simple stimulus trial. (C) Sum of sines wave trial.



(A)

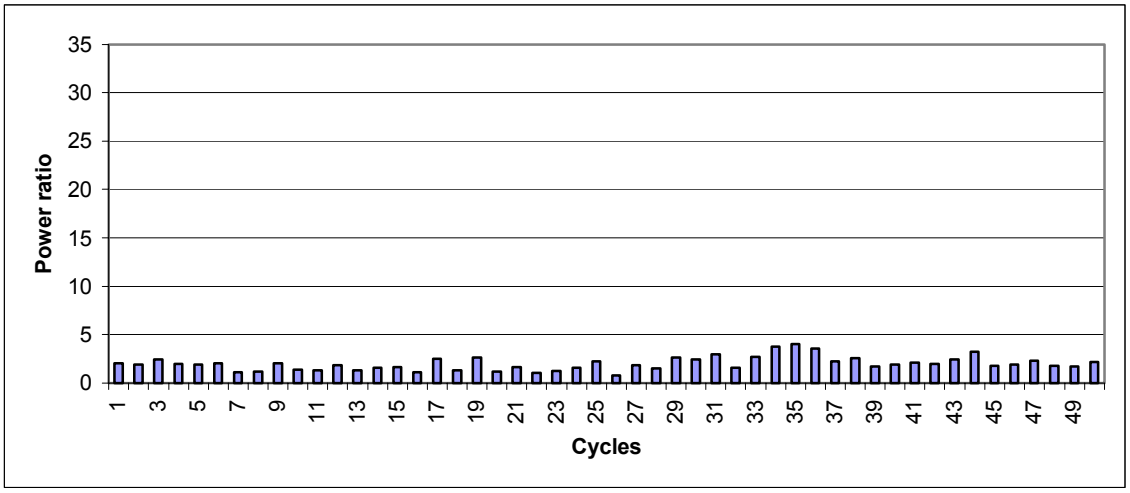


(B)

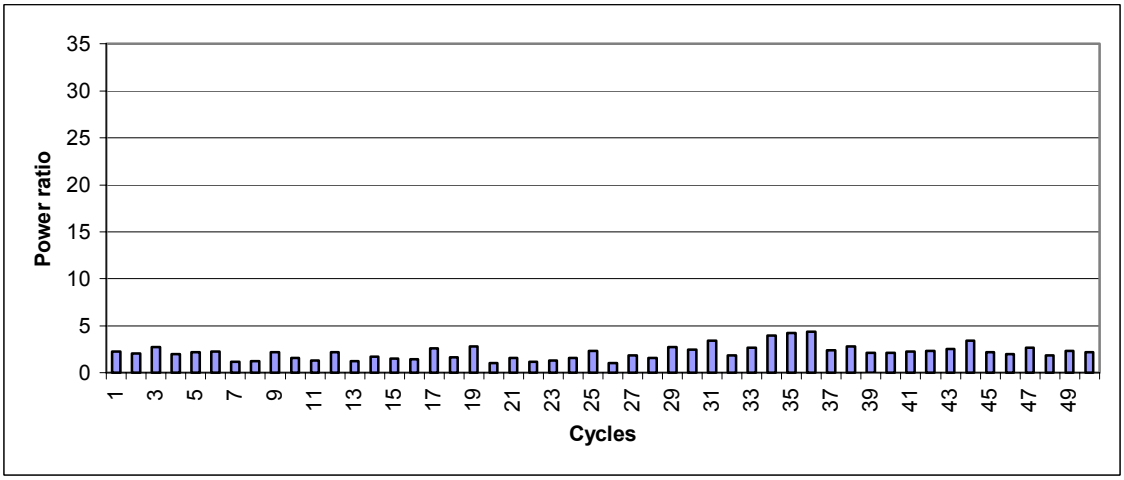


(C)

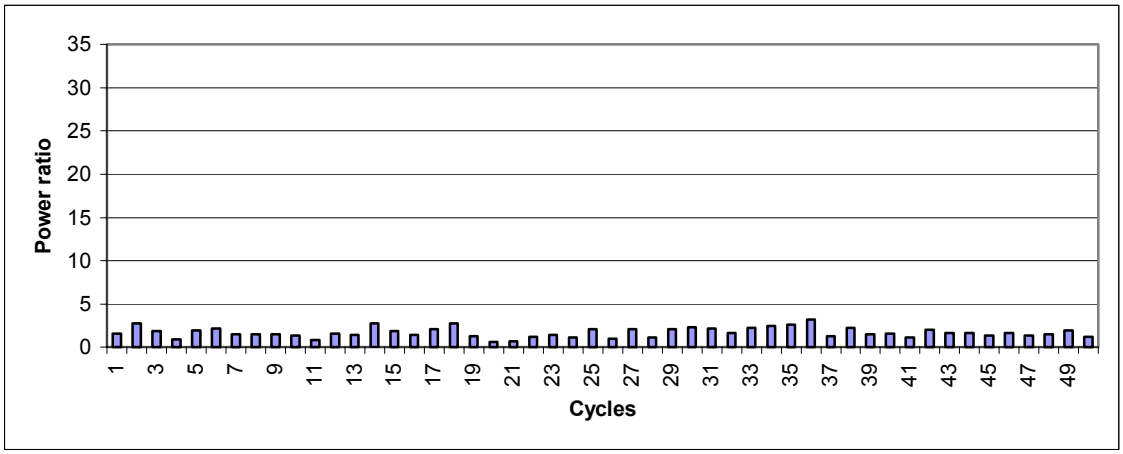
Figure 9. Power ratios, simple stimulus. (A) Trial 2/Trial 1. (B) Trial 3/Trial 1. (C) Trial 4/Trial 1.



(A)



(B)



(C)

Figure 10. Power ratios, sum of sines stimulus. (A) Trial 2/Trial 1. (B) Trial 3/Trial 1. (C) Trial 4/Trial 1.

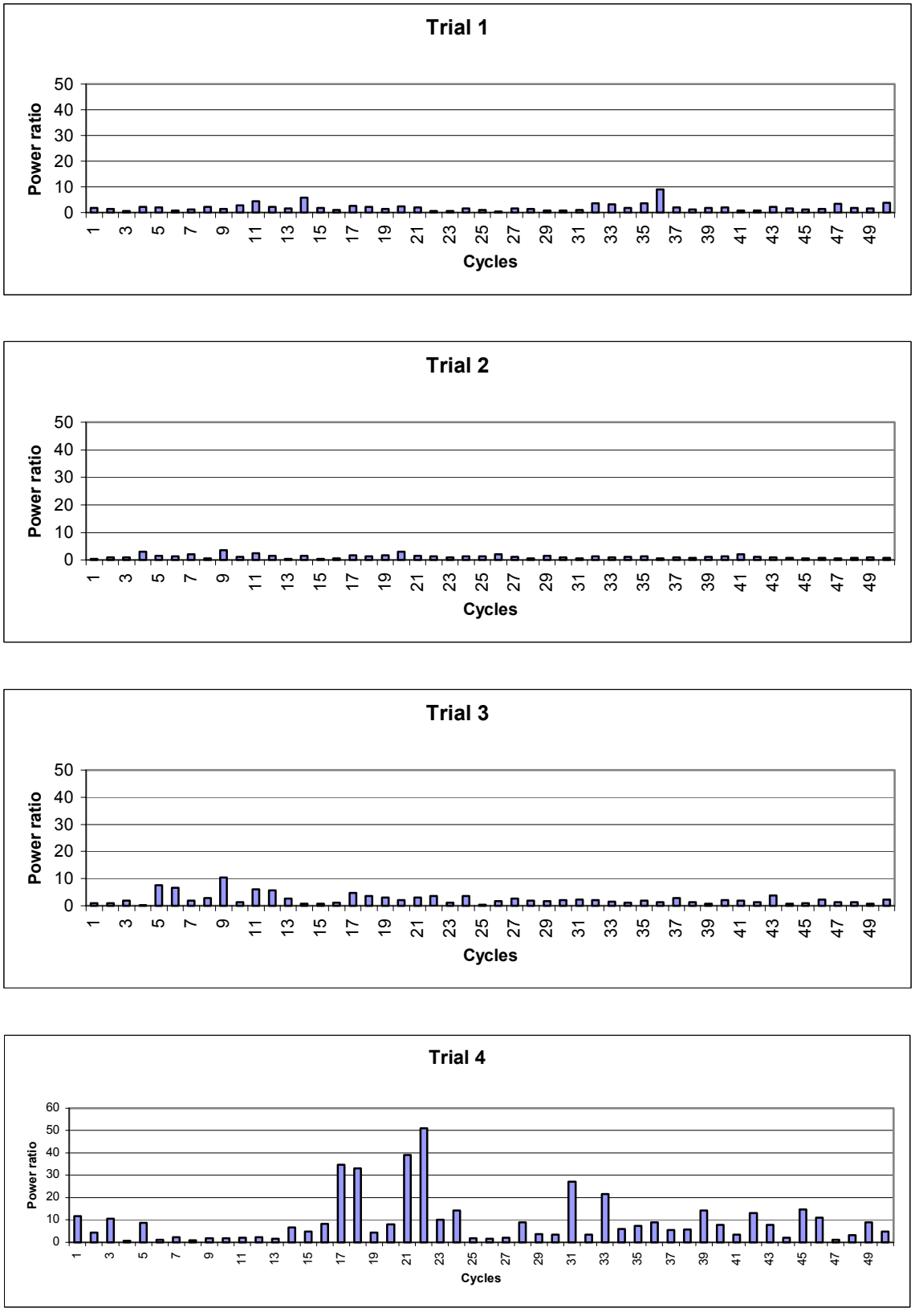


Figure 11. Within trial power ratios (minute 10/minute 1), simple stimulus.

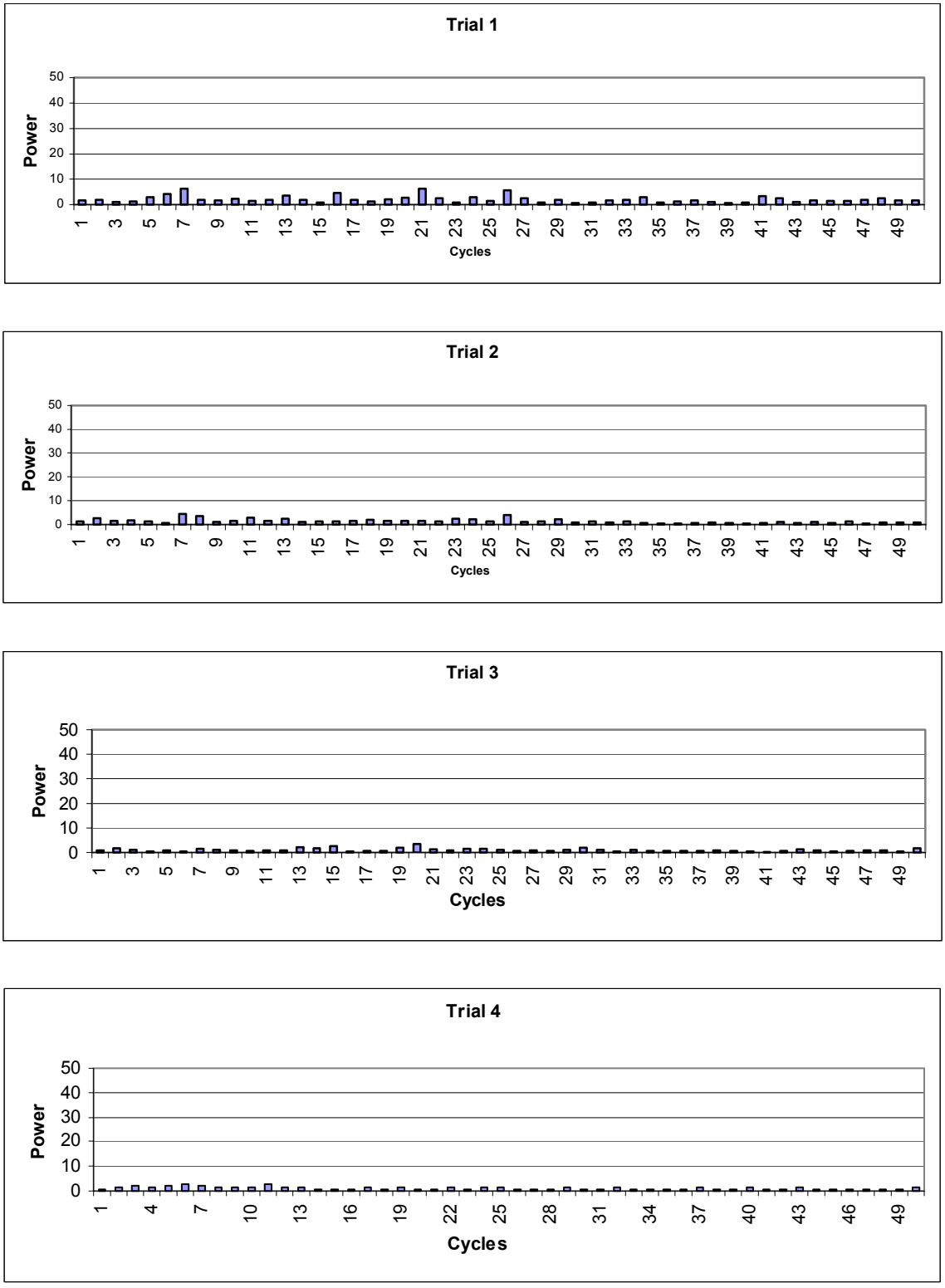


Figure 12. Within trial power ratios (minute 10/minute 1), complex stimulus.