Introduction

Oculomotor System

Humans and other primates are largely visual animals (1); from reading the morning newspaper, to detecting a predator hiding in the bushes, vision is heavily relied upon for daily functions. Thus, a refined oculomotor system has developed to capture the environment onto the retina. It controls the orientation of the visual axis and allocation of visual-spatial attention. A key structure in control of coordinating the oculomotor system is the superior colliculus (SC). It is an evolutionarily conserved structure, located on roof of the vertebrate midbrain (2). The SC is divided into three layers: superficial, intermediate, and deep layers. The superficial layer receives direct projections from both retinal ganglion cells and striate cortex, while the intermediate and deep layer receive indirect input from the extrastriate cortex and also respond to visual stimuli (3). For simplicity, both intermediate and deep layers of the SC are referred as SCi. Neurons of the SC are organized into a retinotopically coded map of the contralateral visual space, with a larger representation of the central visual field than the peripheral visual field (3). The SC is highly interconnected to the cortical components of the oculomotor system via the thalamus. The SCi functions as an integration point of sensory information and goal-related motor responses as it receives convergent sensory, cognitive, and motor inputs from multiple cortical and subcortical sources (4). The SC coordinates a wide range of planning and execution of orienting movements of the eyes and head motor consequences of spatial attention shifts.

Movements of the Eye

The SCi serves a motor function by emitting high frequency bursts of neuronal activity to the premotor centres of the brain stem, for eye and head movements. The burst neurons for horizontal and vertical saccades are located in the pontine reticular formation and rostral interstitial nucleus, respectively. The brainstem burst generator is composed of excitatory burst neurons (EBNs) and inhibitory burst neurons (IBNs), which are collectively referred to as saccadic burst neurons (SBNs). EBNs excite while IBNs inhibit extraocular muscle motor neurons responsible for high velocity saccades. The SBNs are negatively regulated by omnipause neurons (OPNs), which are tonically active during fixation and inactive during a saccade. In order to generate a saccade, the strong inhibition from the OPNs must be removed for the groups of burst neurons to be activated. Consequently, a short, high frequency burst of activity is sent to the extraocular muscle motor neurons to bring forth a movement. Just prior to completion of a saccade, the OPNs are reactivated, re-inhibiting the brainstem burst generator. Through the inhibition of OPNs, the brainstem burst generator is able to differentiate saccadic and non-saccadic signals, from the other information received from the SCi. This prevents reflexive saccades from being made to every visual stimulus caused by a salient stimulus in the visual field.

Movements of the Head

Not only does the SCi send projections to the burst generator, but also the reticular premotor centres for head movements and body orienting movements (5). In humans and monkeys, the visual axis can be shifted by a saccadic eye movement as much as 50° to 60° (6); moreover, the head moves as well for shifts greater than 20° (7), and the body moves as well for shifts greater than 40°. (8). Studies have shown that high levels of stimulation current in the SCi initiate eye-head gaze shifts, while low levels of stimulation current (below the threshold of a saccade) evoke head-only gaze shifts (9). The neurons in the SCi (involved in saccades) encode the movement of visual axis, not the individual components of the eye and head movements (10). Downstream of the SCi, the eye-head gaze signal is broken down into eye and head components.

Kinematics of the Head and Eyes

The kinematics of eye-head gaze shifts are important as the eyes, head, and body have different biomechanical properties. The eye has very little mass and thus the rotational inertia is almost negligible. The anatomical arrangement of the six extraocular muscles is very simple and these muscle fibres are some of the fastest contracting fibres in the body (11). In contrast, the head has much greater mass and thus the rotational inertia is non-negligible. The anatomical arrangements of the neck muscles are extremely complicated, with redundancy amongst the muscle in their pulling directions. This introduces substantial lag between neck muscle contraction and onset of rotational head movement. However, measurement of electromyographic (EMG) activity of neck muscles shows precisely when the neural command was issued to the muscle, circumventing the inertial lag. It has shown that recruitment of neck muscles start prior to those of the eye (9). The selective inhibition by the OPNs on the saccadic burst generator may serve as a possible explanation for the independence of eye and head movements as it does not exert the same inhibition on head premotor structures. Therefore, the fate of the signal from the SCi is different if it is to the eye versus head premotor centres.

Microsaccades

Even during attempted 'fixation' of the eye upon a stable, non-moving target, there still is motion of the eye via fixational eye movements; these include drift, tremor and microsaccades (12). Microsaccades are very small (generally considered less than 1°) and rapid saccade-like movements of the eyes. Both microsaccades and saccades are conjugate movements, involving motor coordination of both eyes for bilateral fixation on a single target. Microsaccades prevent perceptual fading of static images on the retina by small eye movements to refresh the images

(13). Like saccades, microsaccades are generated in a similar manner, involving the SCi, brainstem burst generator and pause in OPN activity (14). Upon attempted fixation, microsaccade directions are randomly distributed (15); however, with the presence of a spontaneous stimulus, the direction of microsaccade can be skewed. The probability of microsaccade occurrences decrease shortly after a stimulus, but the few that do occur during this time are biased towards the direction of the stimulus (16).

Rationale, Hypothesis and Predictions

The amount of information potentially available through our vision is far too overwhelming for the brain. Thus, it limits what is perceived through attention, the allocation of cognitive processing resources to a selective location of the visual field. It acts analogous to a spotlight in a dark room, lighting up only a patch of the room to be seen. This visual attention must be moved around, and it can be shifted in two ways, overtly, and covertly. Overt shifts of attention occur when both the centre of visual axis and attention shifts towards the target of interest in the periphery. Covert shifts of attention occur when the centre of visual axis remains fixed, but attention shifts towards the target of interest in the periphery. Unlike overt shifts of attention, covert shifts do not exhibit large, obvious saccadic eye movements, so they are not so easy to detect. However, accumulation of past studies suggest the appearance of a stimulus in the visual periphery causes a visual response, which can be detected through neck muscle EMG activity and microsaccade detection. This study focuses on microsaccades and neck muscle EMG activity as indicators of covert attention as they seem to mirror the activity of the SC. Currently, only very crude behavioral activity examinations and expensive scans for covert attention are available; thus, these indicators may provide as inexpensive and noninvasive alternative methods for study and diagnostics. It is hypothesized that the activity in neck muscles and the frequency of cue-directed microsaccades reflect the activity of the SC in response to peripheral visual stimuli. It is predicted that higher frequencies of cue-directed microsaccades will be observed for trials with higher levels of neck EMG activity than trials with lower levels of neck EMG activity. It is also predicted that greater neck activity will be observed for trials with cue-directed microsaccades than trials without cue-directed microsaccades. If the results support the hypothesis and predictions, both neck muscle activity and microsaccades may serve as indicators of covert attention and be used to study clinical disorders such as hemi-spatial neglect.

Methods

All experimental protocols were carried out by Brian D. Corneil, Douglas P. Munoz, Brendan B. Chapman, Tania Admans and Sharon L. Cushing, in accordance with the Canadian Council on Animal Care policy on the use of laboratory animals and also approved by the Animal Use Subcommittee of the University of Western Ontario Council on Animal Care. This project analyzed data from studies that have already been carried out. A total of four male rhesus monkeys (*Macaca mulatta)* were studied using two behavioral tasks; two monkeys (Jesse and Mooky) performed the saccade cueing task and the other two monkeys (Alex and Spike) performed the memory guided saccade task, both manipulating covert shifts of attention. They sat in a chair that limited the rotation about the torso to 10° or less. They faced an array of red light emitting diodes and performed the experiments with their head restrained. The monkeys were prepared for chronic recording of eye position (using eye trackers) and neck muscle EMG activities (using implanted electrodes). The ipsilateral, dorsal neck muscles (bringing forth horizontal movement) examined were: obliquus capitis inferior (OCI), rectus capitis posterior major (RCP) and splenius capitis (SP) as seen in Figure 10. All analog data were digitized at 10 kHz by a multichannel recording system [Plexon Inc; prior to digitization, EMG data were amplified (1000x) and filtered (100Hz – 4kHz)]. Offline, EMG signals were rectified and integrated into 1 ms bins. All aspects of the experimental paradigm were controlled by a real-time controller (LabVIEW, National Instruments).

In the first task, the monkeys (Jesse and Mooky) performed a saccade cueing task. They stared at a central fixation point for 500–1000 ms. While the fixation spot was on, a visual cue illuminated in the periphery, either on the left or right for 30 ms. After a 600 ms delay, the cue target onset asynchrony (CTOA), a target illuminated either on the left or right and the monkeys were required to look at the target. The eccentricity of both the cue and target was fixed within a block of trials. They varied between 10°, 15°, 20°, 27° and 35° across blocks of trials. Refer to Figure 11A representation.

In the second task, two monkeys (Alex and Spike) performed a memory guided saccade task. They stared at a central fixation point that illuminated for 500 ms before the peripheral cue appeared. The peripheral cue flashed for 100 ms either 20° to the left or right during the fixation period. The monkey had to maintain fixation at the central fixation point even during the appearance of the peripheral cue, and also until 700 – 900 ms after the peripheral cue had turned off. After the fixation point had been turned off, the monkey was required to saccade to the remembered location of the cue. Refer to Figure 11B for visual representation.

Using a Graphical User Interface (GUI) built by Brian D. Corneil, within Matlab 2009, ~5000 trials were marked for potential microsaccades (sample GUI screen seen in Figure 12). These manual markings recorded the following: trial number, onset time in trial, offset time in trial, trial type, horizontal amplitude, vertical amplitude, vectorial amplitude, direction, horizontal peak velocity, vertical peak velocity, peak velocity and duration. The criteria for microsaccades were vectorial amplitude of 0.1-2.0° and angle rotation of less than 75° from the horizontal meridian. The data set will be analyzed to observe the main sequence relationship of microsaccades, time-course of microsaccade frequencies, visual response on various neck muscles to cues, and examine microsaccades propensities through neck activity and vice versa. A student's paired t-test will be used for statistical analyses in determining whether a significant difference in the frequencies of cue-directed microsaccades is observed between trials with higher levels of neck EMG activity than trials with lower levels of neck EMG activity, and whether a significant difference in neck activity is observed for trials with cue-directed microsaccades than trials without cue-directed microsaccades.

Results

Data from three of the four monkeys (Mooky, Alex and Spike) were used to analyze the results as data from one monkey (Jesse) did not show microsaccades within the cueing task. As mentioned, trials with eye movement amplitude of 0.1 - 2.0° and angle rotation from horizontal meridian of less than 75° were considered as microsaccades. For counting microsaccades in a sliding window analysis, the half window width was set to 25 ms and the step sizes were set to 10 ms.

All the microsaccades (marked and considered under the initial parameters) across the three monkeys followed the main sequence trend, showing a linear relationship between amplitude and peak velocity as shown by an example of Mooky's in Figure 1. As shown in previous studies, all monkeys exhibited the same phenomenon, where the frequencies of microsaccades decreased shortly after cue onset (microsaccadic inhibition) as seen in Figures 2A, 3A and 4A. After the decrease, the frequencies of microsaccades increased again (microsaccadic rebound).

The visual response (pooled EMG neck muscle activity in response to appearance of a stimuli in the periphery of visual field) was observed on the various neck muscles of all monkeys. There were increased neck EMG for ipsilateral cues and decreased neck EMG for contralateral cues (from baseline levels) during the visual response to cue, as seen in Figures 2B, 3B and 4B. The visual responses in neck muscles were aligned with the decrease in microsaccade frequencies.

To address the first prediction, microsaccades were examined through neck activity. All trials were separated into two halves depending on the levels of neck EMG: low visual burst and high visual burst. The time-course of microsaccade freqeuncies of the lower half visual response trials and the higher half visual response trials did not show a difference in microsaccade patterns as seen in Figure 5.

To address the second prediction, neck activity was examined through microsaccades. As observed earlier, there was an increased neck EMG for trials with ipsilateral cues and decreased neck EMG for trials with contralateral cues (from baseline levels) during visual response cue. However, there was an even greater increase in neck EMG for trials that had a microsaccade towards the ipsilateral cue (cue-directed microsaccade) as seen in Figure 6. This heightened neck activity in trials with cue-directed trials coincides with the decrease in microsaccade numbers (microsaccadic inhibition) as seen in Figure 7.

The visual response above baseline of all trials with cue-directed microsaccades of various muscles of all three monkeys were compiled together onto one plot. It was observed that there is a subtle peak in neck EMG at around 100 ms after cue onset as seen in Figure 8. To compare the neck EMG of trials with cue-directed microsaccades to trials without cue-directed microsaccades (of various muscles of all monkeys), the average neck EMGs were plotted as seen in Figure 9. If the null hypothesis was true, there would be no difference of neck EMGs between trials with cue-directed microsaccades and trials without cue-directed microsaccades, and it would of followed the blue line. However, a paired student's T-test was performed and showed that trials with cue-directed microsaccades had significantly greater ($p= 0.0058$, $n=14$) neck EMGs than trials without cue-directed microsaccades.

Discussion

Visual Response on the Neck reflective of the SC

The presentation of a visual stimulus activates the oculomotor system transiently, inducing visual responses within the neuronal network without the presence of an eye-head gaze shift. This response is carried by neurons of the SCi that project to the downstream brainstem premotor circuits in control of eye and head movements. For the generation of a saccade, the level of activity in the SCi must reach a certain threshold, causing the temporary silencing of OPNs and burst of activity of the SBNs. It is suggested that the selective gating by OPNs on the saccadic burst generator, but not on the reticular head premotor centres, is the mechanistic explanation to why such a visual response from a stimulus does not cause a saccadic movement of the eye, but does cause recruitment of neck muscles. Since there are no gating mechanisms for the circuitry to the neck muscles, the visual response is not inhibited. These responses (to appearance of visual stimuli) that have been observed on neck muscles are thought to be reflexive covert orienting signals reflective of the visual responses seen in the SCi (17). Previous observations have shown that various muscles of the neck increased in activity following the presentation of an ipsilateral cue and decreased in activity following the presentation of a contralateral cue, perhaps in preparation of a movement.

Microsaccades reflective of the SC

The explanation to microsaccades seems to be still up for debate as there currently are conflicting theories. Studies suggest that microsaccades occur as a result of subliminal activation of the oculomotor system by covert shifts of attention (16). It is believed that microsaccades have the same dynamics as larger saccades and are a part of a continuum defined merely by an arbitrary cut-off. This brings into question whether the available knowledge about the oculomotor system can allow for the presence of motor consequences of covert shifts of attention within its network. Direct recordings of the SCi during tasks manipulating covert attention shifts serve as strong evidence. It is known that the SC contains a spatially-coded map of the visual-motor space and the neurons within this map exhibit mutual inhibition. When attention is shifted to a peripheral target, the neurons encoding that certain position of the map (caudal SCi) exhibit transiently increased activity (18), while the neurons encoding the fixation point (rostral SCi) exhibit decreased activity. This weakens the neural excitatory signals of the fovea or fixation zone (within the map of the SCi) that project to the brainstem OPNs. It is believed that this reduction of neural responses may be just enough to momentarily reduce or shut down the activity of OPNs, allowing the transient activity of the SCi to bring forth a small eye movement.

However, this conflicts with the idea of a threshold that must be reached for the generation of a saccadic eye movement.

Suggested Model for Microsaccades

Hafed and Ignashchenkova (2013) have proposed a model to explain the microsaccadic inhibition, rebound and the direction bias after onset of peripheral cues. They believe that the phase resetting of an ongoing microsaccadic oscillatory rhythm play an essential role. Their purpose was to mechanistically explain the distinct dissociation observed between microsaccade frequency and direction after peripheral cue onsets. Within their's and this study, microsaccadic frequency was relatively stable prior to peripheral cue onset, but sharply declined to a minimum shortly after the cue, and to rebounded back again, returning back to stable baseline levels. During the sharp decrease in microsaccade numbers, the few microsaccades that did execute were highly directed towards the peripheral cue than away from it. These microsaccades had small amplitudes and not considered overt targeting saccades.

The hypothesized mechanism is that peripheral cue onsets initiate a competing motor command for the generation a new microsaccade, and that this new competing command interacts with the ongoing program to cancel it (19). A single microsaccade motor command considered an accumulation of some activity towards a certain threshold. Once this threshold has been reached, a microsaccade is executed and the activity drops back down. If a peripheral cue happens to appear sometime during this buildup phase of a microsaccade, a competing motor command to the ongoing command is initiated and this alters the buildup of the current command. If the cue onset appears early during the buildup phase, it may be powerful enough to bring the activity down to zero and cancel the microsaccade. If the cue onset appears late during the buildup phase, it may not be strong enough to bring the activity down to zero to cancel the microsaccade, resulting in an execution of a noncanceled microsaccade.

During steady-state fixation, microsaccades appear to have an ongoing oscillatory rhythm, consisting of a buildup to a threshold, execution of a microsaccade and again buildup to a threshold for the next microsaccade. Depending on timing of when a cue appears during the rhythmic pattern of microsaccades, it can either cause a canceled microsaccade or a noncanceled microsaccade. After the consequences of cue presentation, it is followed by a resumption of the ongoing oscillatory microsaccadic rhythm. The sharp decrease (inhibition) and increase (rebound) observed shortly after cue onset is a reflection of the phase resetting event of the ongoing rhythmic build up, caused by the appearance of the cue. Thus, this phase resetting may serve as a mechanism explaining the widely observed phenomenon microsaccadic inhibition and rebound.

The microsaccades that occur shortly after cue onsets are eye movements that were not canceled by the competing motor command brought on by the appearance of the cue. These rare microsaccades that do escape are highly correlated with the direction of the cue. It is thought that these microsaccades are highly correlated with the direction of the cue because not all microsaccades are equally easy to cancel by cue onsets. If a cue appears during a buildup phase of a microsaccade towards the side of the cue, it will be more difficult to cancel since this microsaccade will receive spatial support from the visual burst at the level of the SC upon cue onset (20). Likewise, it would be easier to cancel if the cue appeared during the buildup phase of a microsaccade away from the side of the cue. Therefore, there would be a greater likelihood of microsaccades towards the side of the peripheral cue during the critical window of time in which noncanceled microsaccades are expected to escape.

Findings and Applications

This study observed the relationship of the two potential indicators of covert attention, microsaccades and neck muscle activity. Microsaccades were examined through neck muscle activities, and also neck muscle activities were examined through microsaccades. It was found that a relationship did not exist both ways, but only when the visual responses on the various neck muscles were observed with versus without cue-directed microsaccades. This may be due to the very low occurrences of cue-directed microsaccades. In this study, microsaccades occurred in a very few minority of the trials, while the visual responses on the various neck muscles were fairly consistent and supported the strength of microsaccades as meaningful indicators. Measures of microsaccades and corresponding neck muscle recruitment could serve as potential indicators of covert attention as they parallel the insights that can be gained from the SCi.

These markers could serve as both discrete and continuous measures respectively, and may be applicable clinically to diagnosing and studying attention disorders such as hemispatial neglect. People with such deficits would be expected show low levels of or no microsaccades at all towards the side of attention deficit, and only distribute towards the side where attention is not deficient. Correspondingly, it is expected that the neck muscles ipsilateral to the side of attention deficit will not exhibit any sort of increase in activity, while the neck muscles contralateral to the side of attention deficit will not exhibit any sort of decrease in activity. Current diagnostics involves merely patients being asked to mark the halfway point of a line, draw a clock with its numbers, and etc. Perhaps these indicators of covert attention may be used to identify and quantitatively gauge the attention deficit of these patients while under clinical diagnosis, study or therapy.

Limitations

There were several potential limitations within the study that could have had affected the outcomes of this project. The initial selections of eye movements for potential microsaccades were marked manually, giving rise to consistency problems due to human errors. Adding on to that, parameters defining an eye movement as a microsaccade were decided arbitrarily using knowledge from past studies. Next, the noise signals sometimes made it difficult to identify a microsaccade from the background noise. The test subjects being monkeys, it was not feasible to have all trials of the behavioural tasks performed flawlessly. There were plenty of random saccadic eye movements and incorrectly performed trials that may bear an effect on the data. Lastly, there were very few microsaccades observed within the thousands of trials, making it difficult to draw significant conclusions about microsaccade patterns from the observation of neck muscle EMG activity.

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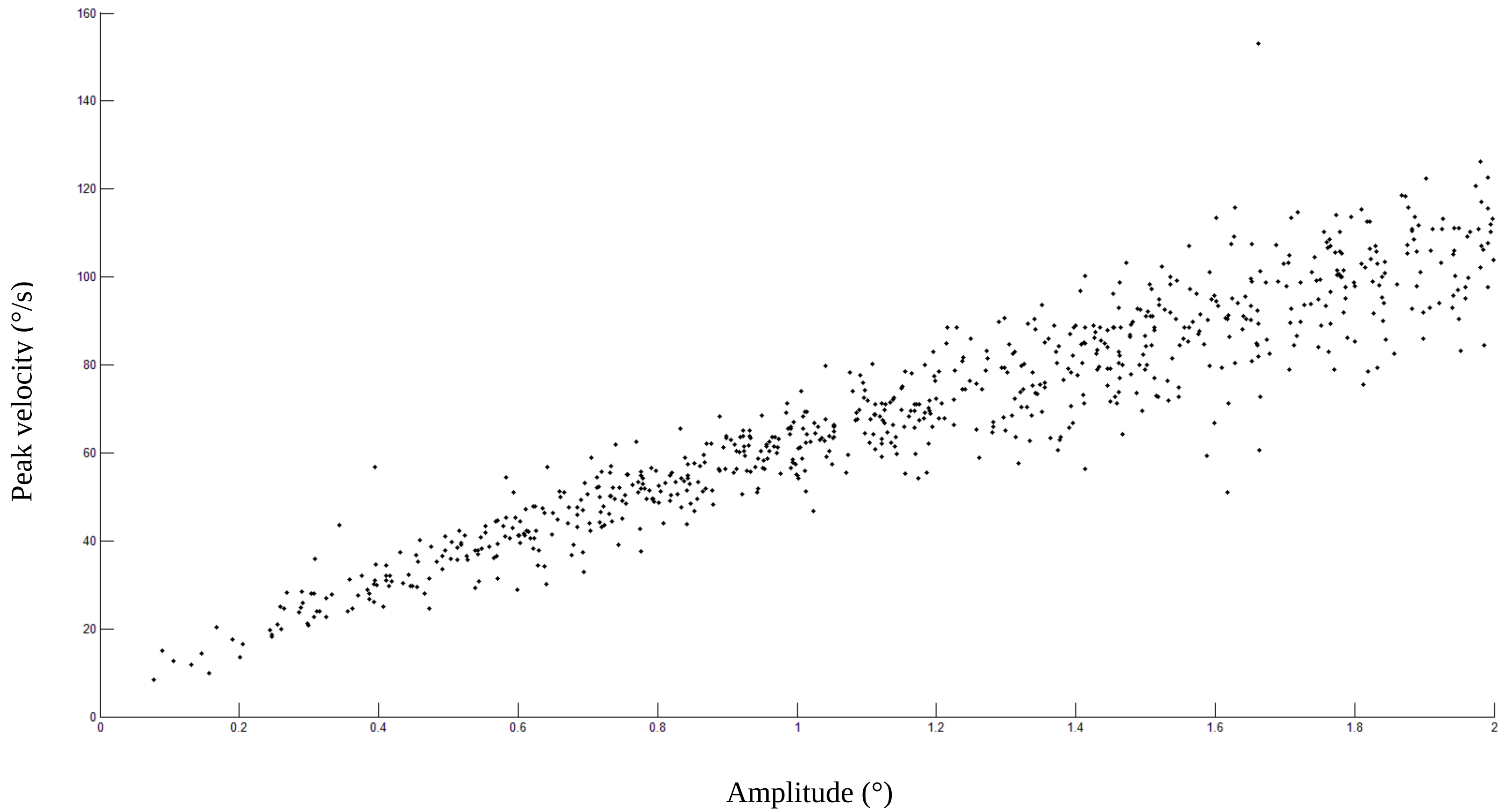


Figure 1.

Microsaccadic main sequence for Mooky

The amplitude and peak velocity of microsaccades show a positive, linear relationship.

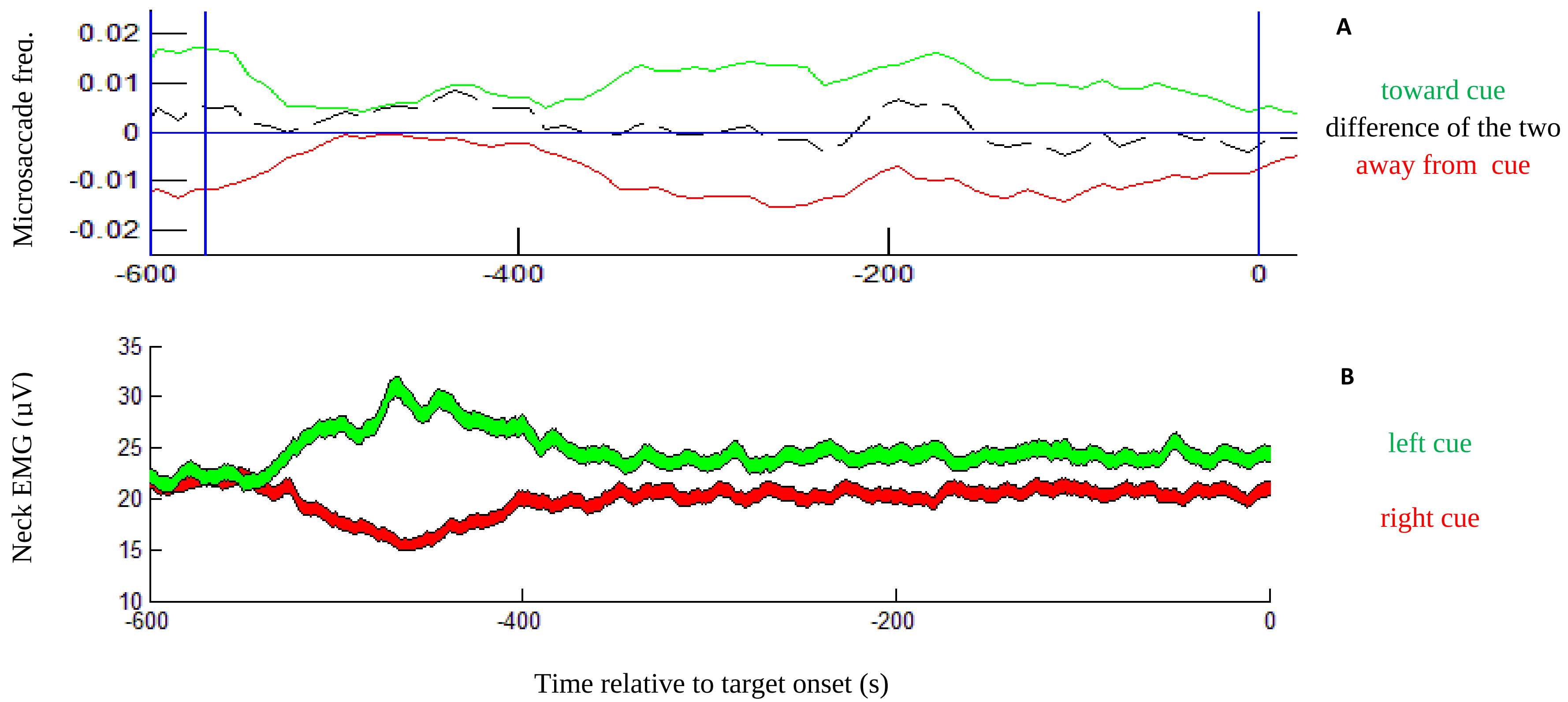


Figure 2.

A. Time-course of microsaccade frequencies toward and away from cue in Mooky

There is a decrease in microsaccade frequency shortly after cue onset, a phenomenon referred to as microsaccadic inhibition

B. Visual response on left OCI of Mooky

There is an increased activity for trials with left cues and decreased activity for trials with right cues

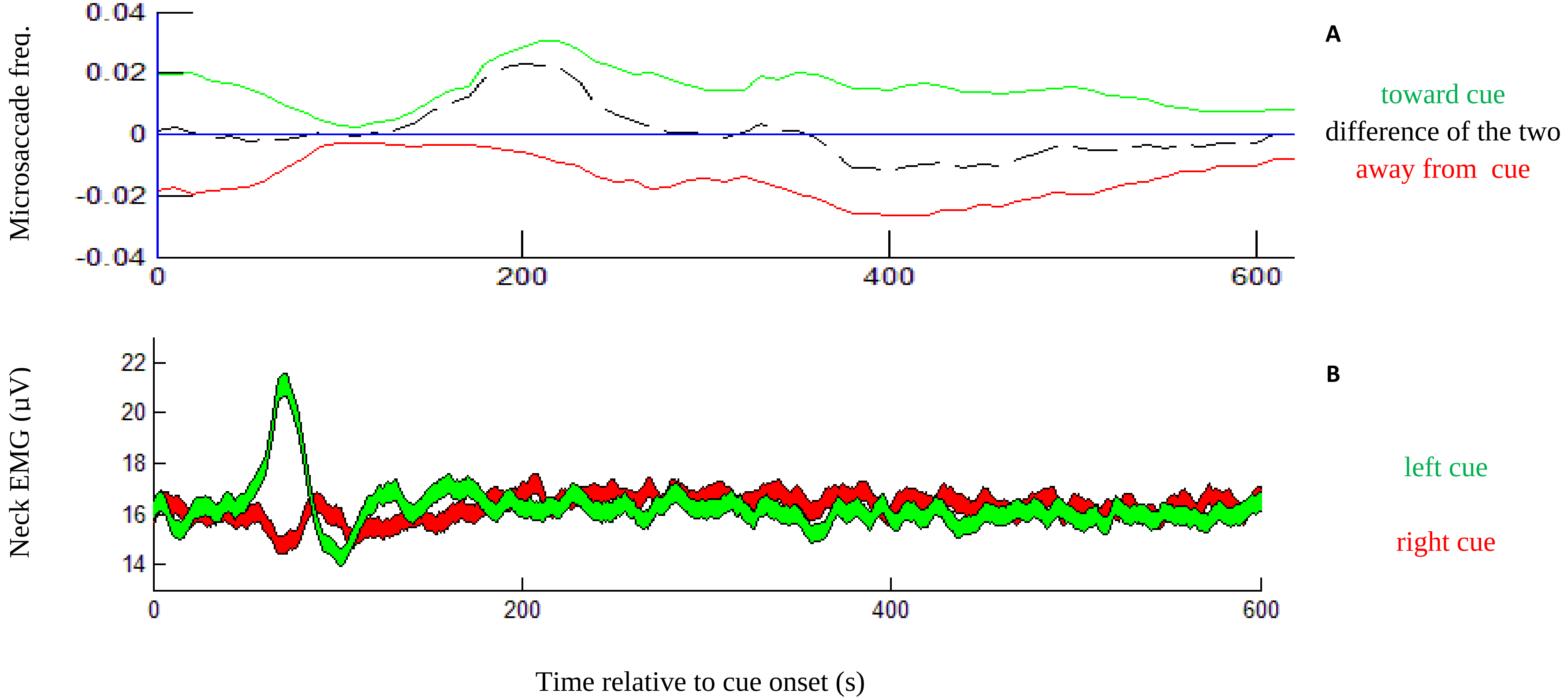


Figure 3.

A. Time-course of microsaccade frequencies toward and away from cue in Alex

There is a decrease in microsaccade frequency shortly after cue onset, a phenomenon referred to as microsaccadic inhibition

B. Visual response on left SP of Alex

There is an increased activity for trials with left cues and decreased activity for trials with right cues

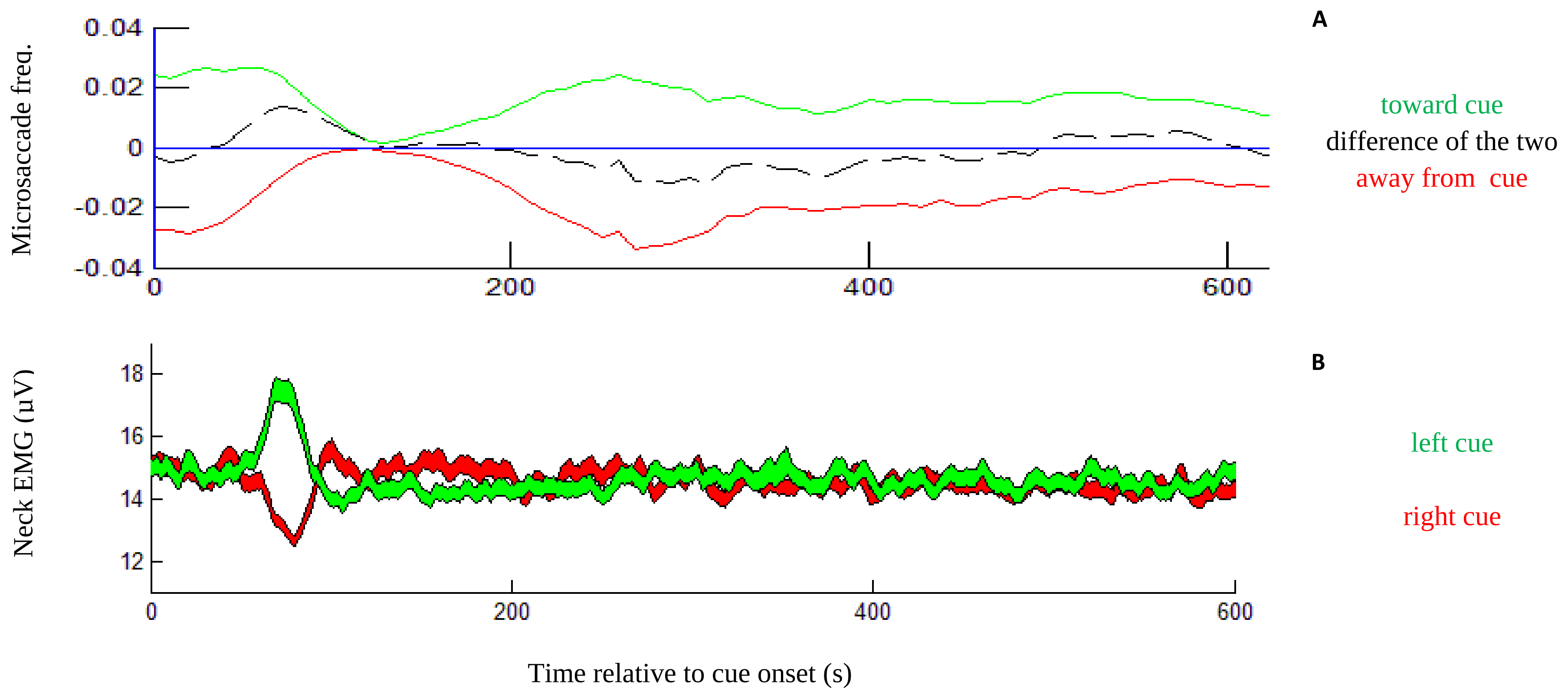


Figure 4.

A. Time-course of microsaccade frequencies toward and away from cue in Spike

There is a decrease in microsaccade frequency shortly after cue onset, a phenomenon referred to as microsaccadic inhibition

B. Visual response on left SP of Spike

There is an increased activity for trials with left cues and decreased activity for trials with right cues

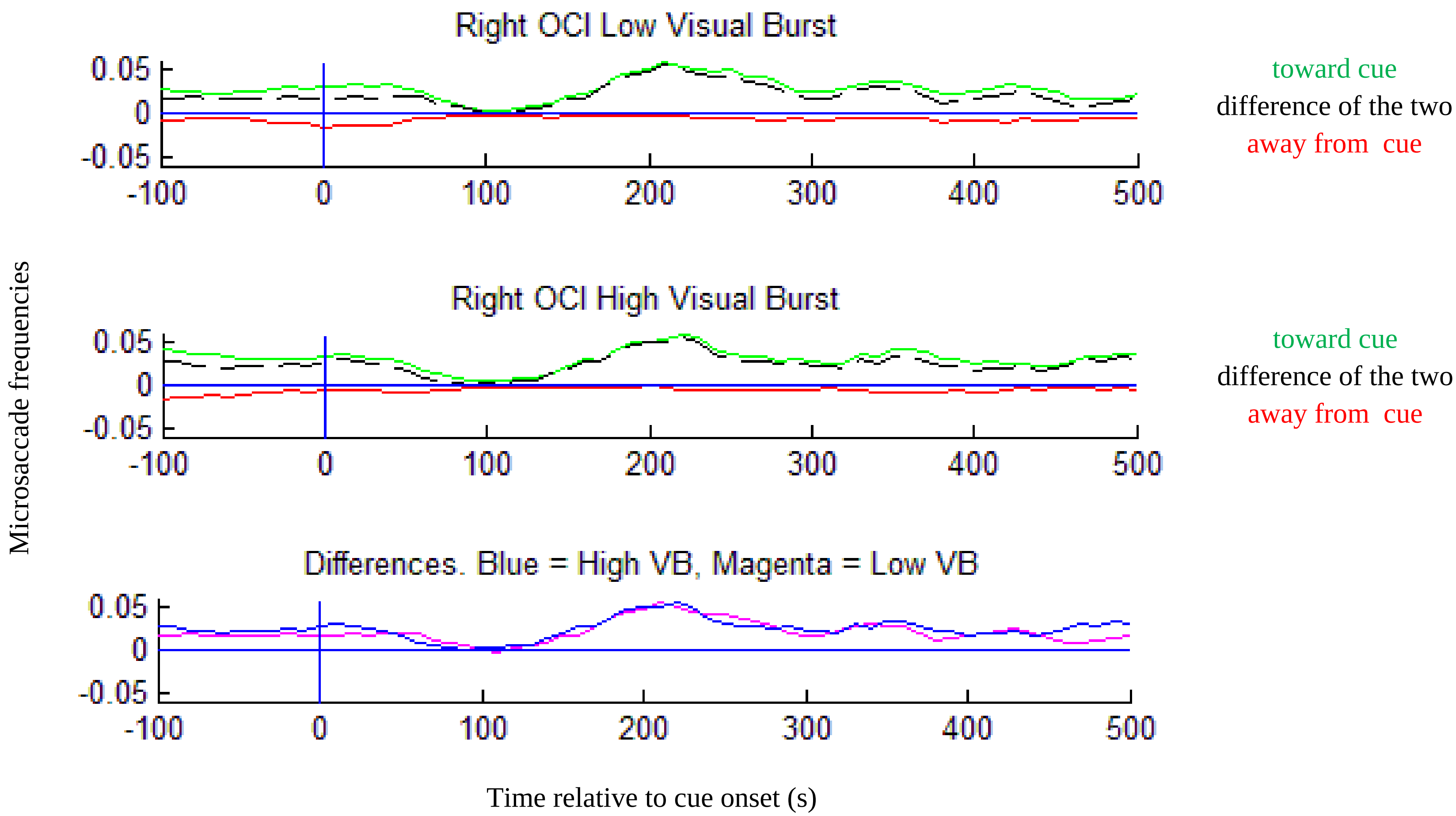


Figure 5.

Time course of microsaccade frequencies toward and away from cue, subdivided into lower half and higher half visual responses in right OCI of Alex

No difference in microsaccade frequencies is observed between high and low visual response groups

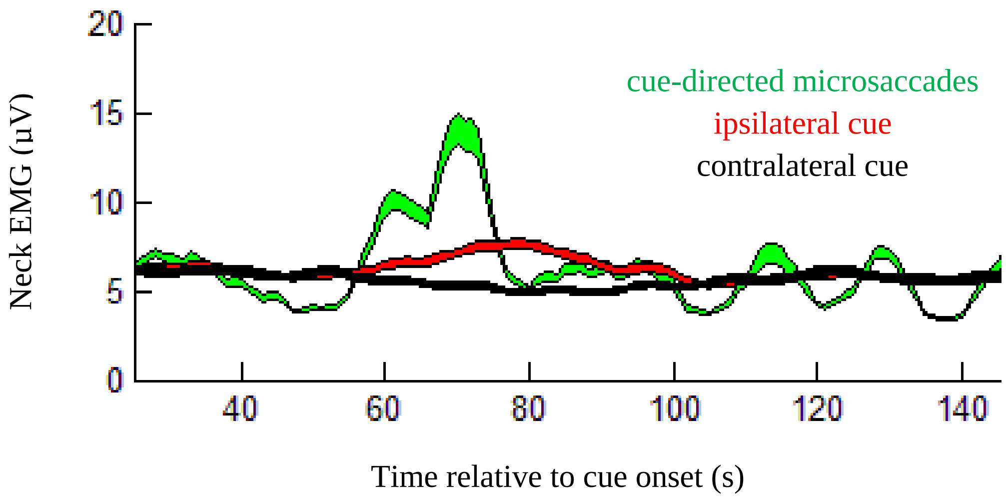


Figure 6.

Visual response on right SP of Spike

There is an increased visual response in trials with cue-directed microsaccades than trials ipsilateral or contralateral to the muscle.

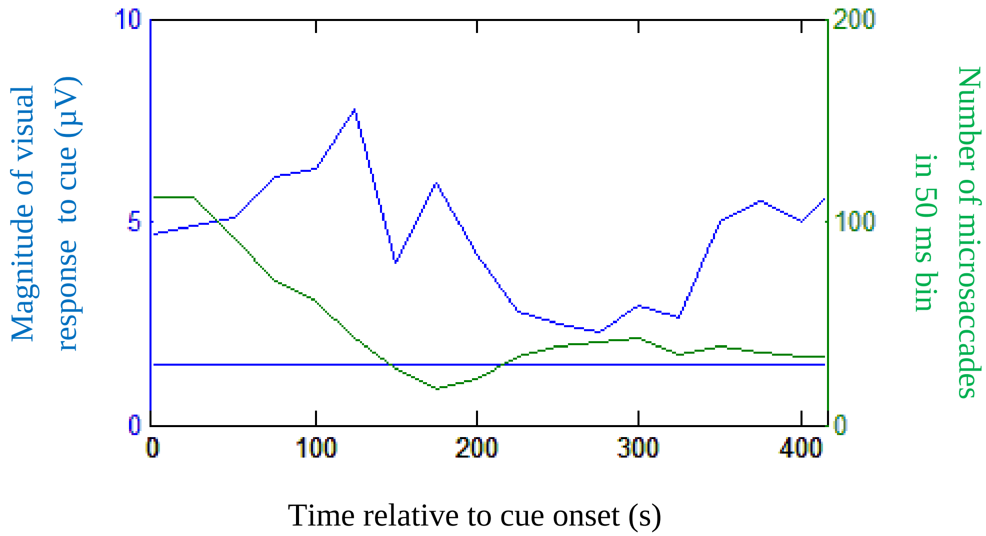
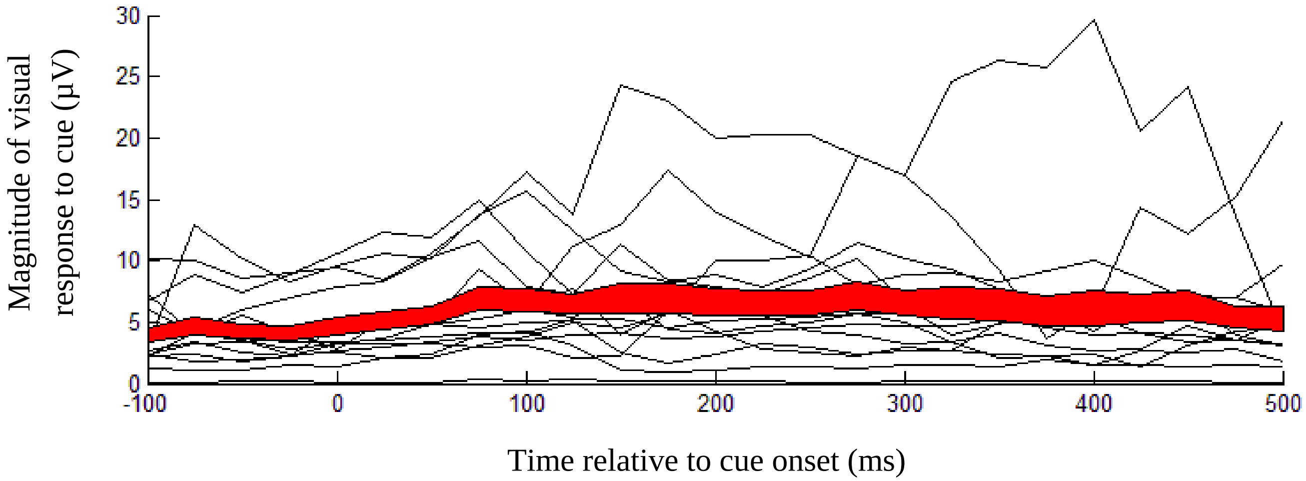


Figure 7.

Visual response on right SP of Spike

The decrease in microsaccades (microsaccadic inhibition) coincides with increase in magnitude of visual response above baseline



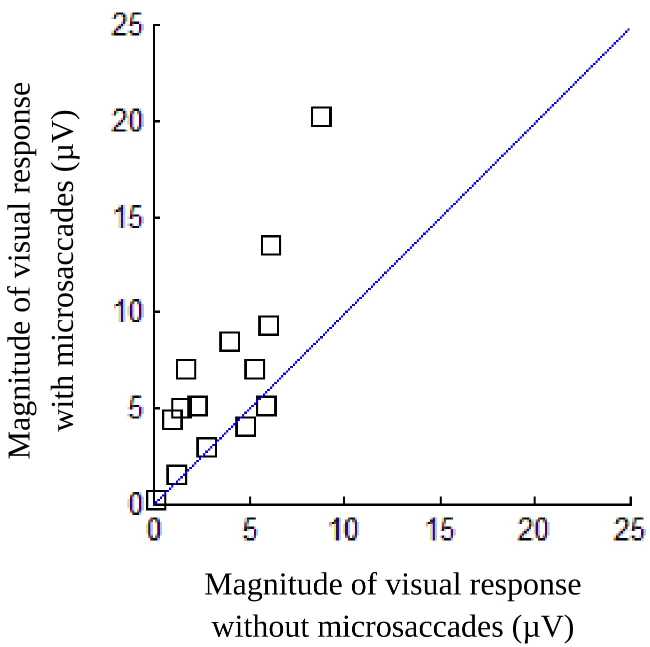


Figure 8.

Comparison of the magnitudes of visual response across various muscles in all monkeys with cue-directed microsaccades aligned to cue onset

There seems to be a small peak of activity in visual response around 100 ms after cue onset

Figure 9.

Comparison of the magnitudes of visual response across various muscles in all monkeys with cue-directed microsaccades and without cue-directed microsaccades

The magnitude of visual response was significantly greater when there were cue-directed microsaccades than when there were no cue-directed microsaccades ($p = 0.0058$, $n=14$)

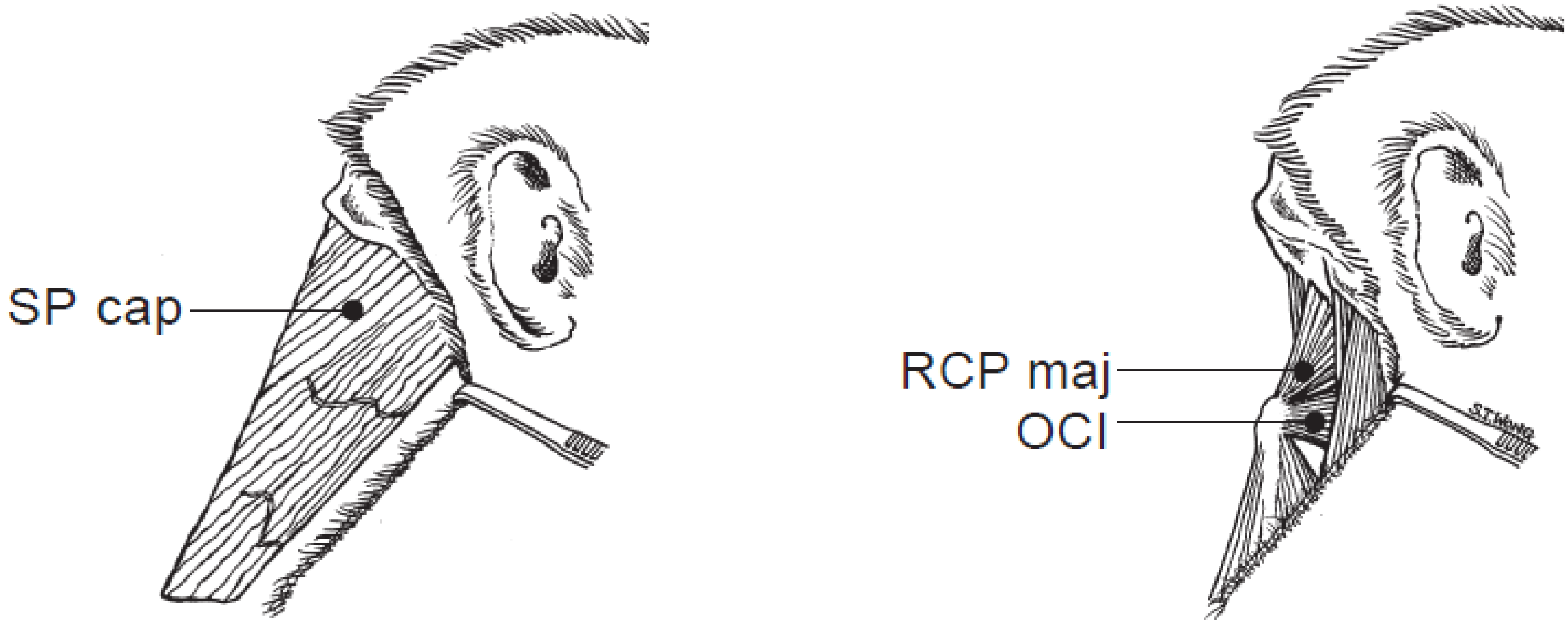


Figure 10.

Dorsal neck muscles responsible for ipsilateral horizontal movement used for recordings

Splenius capitis (SP), rectus capitis posterior major (RCP), obliquus capitis inferior (OCI)

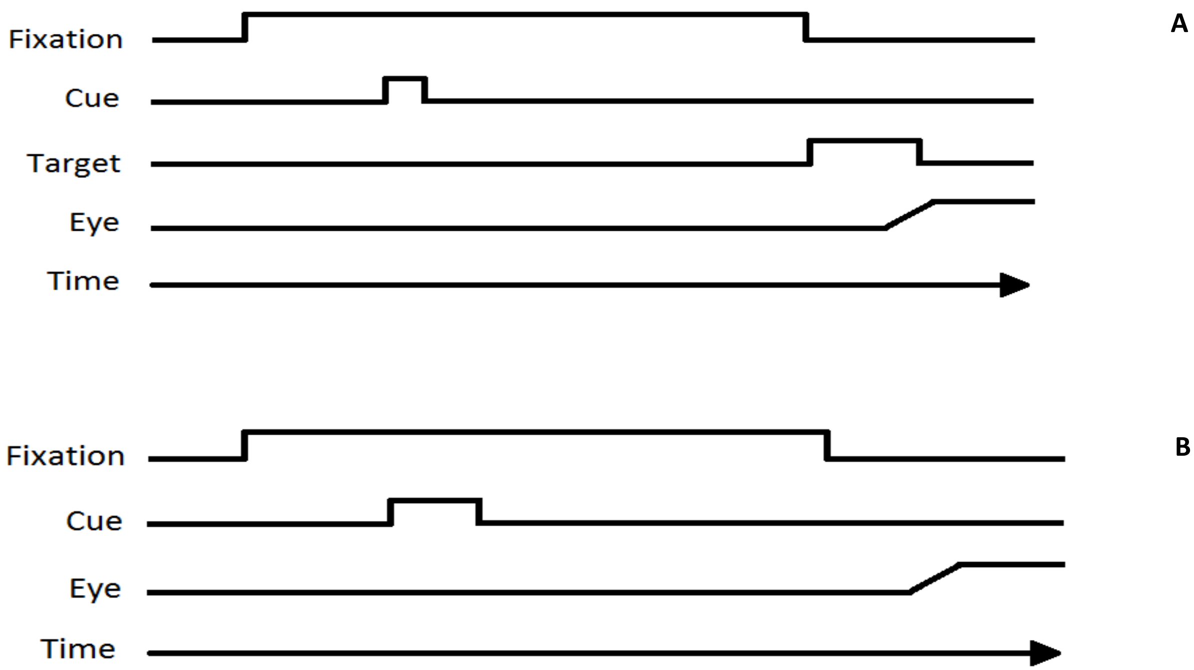


Figure 11.

A. Saccade cueing task

B. Memory guided saccade task

The rise in lines represent the onset of a cue or target, and movement of eye position.

Both tasks manipulate covert attention by presenting the visual cue. The time window of interest is between cue onset and target onset for the saccade cueing task, and between cue onset and fixation point disappearance ("Go" signal for a saccade) for the memory guided saccade task.

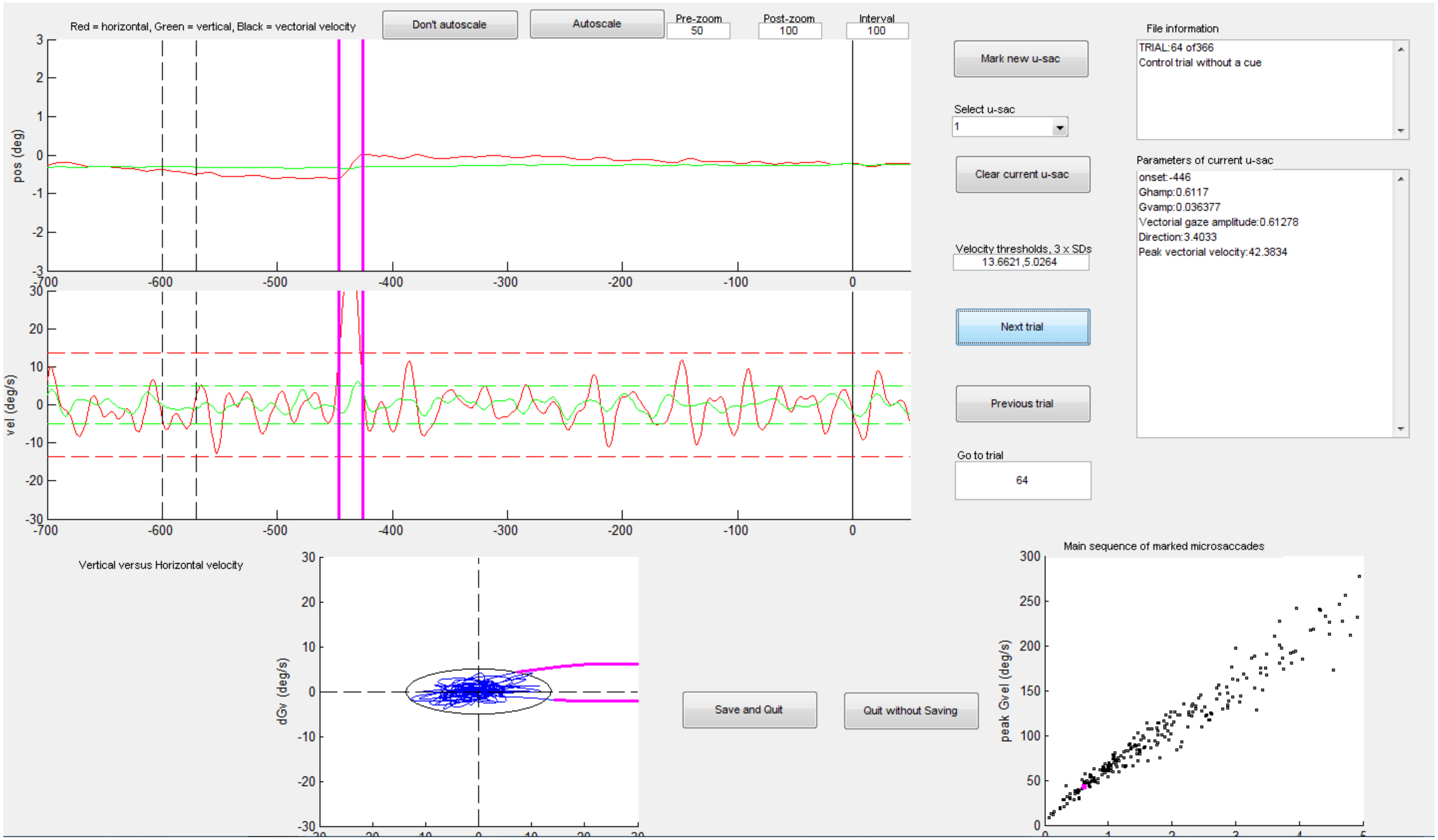


Figure 12.

Sample screen of GUI used within Matlab 2009 used to mark potential microsaccades in Mooky

The eye movements initially marked were further narrowed down to meet the set parameters of what we defined as microsaccades