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Cancelling planned actions following mild traumatic brain injury

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Abstract

Mild traumatic brain injury (mTBI) leads to a variety of attentional, cognitive, and sensorimotor deficits. An important aspect of behavior that intersects each of these functions is the ability to cancel a planned action. Thus, the purpose of this study was to determine the effects of mTBI on the ability to perform a countermanding saccade task. In this task, participants were asked to generate a saccade to a target appearing in peripheral vision, but to inhibit saccade execution if an auditory stop signal was presented. The delay between the appearance of the peripheral target and the presentation of the auditory stop signal was varied between 0 and 125 ms. We found that the change in the probability of cancelling the saccade as a function of this delay was no different between participants with mTBI tested within 2 days of their injury and matched controls. However, saccadic reaction times and the stop signal reaction time were unexpectedly faster in the participants with mTBI and, furthermore, they inaccurately inhibited saccades during 15% of the trials with no stop signal. Taken together, this data suggests that the ability to cancel planned actions is subtly yet adversely affected by mTBI.

Keywords: Mild traumatic brain injury; Countermanding saccades; Decision-making; Executive function

1. Introduction

The voluntary control of action is a vital aspect of behavior—it allows a person to change decisions during an action and make the best choice possible. Voluntary control can be studied experimentally using countermanding, or response inhibition, tasks (Band, van der Molen, & Logan, 2003; Logan & Cowan, 1984). In these tasks participants are required to generate a simple motor response upon the presentation of an imperative stimulus, but to inhibit the response if a stop signal is given. The interval between the presentation of the imperative stimulus and the stop signal is varied and the probability of cancelling the response is measured as a function of the interval. Typically, participants can successfully inhibit their responses if the interval is short, but become increasingly less likely to do so as the interval increases in duration. This lack of inhibitory control with increased delays provides insight into the voluntary control of action.

The neural mechanisms underlying voluntary control have been studied using singe-unit recordings in non-human primates (Schall, 2001; Schall, Hanes, & Taylor, 2000; Schall, Stuphorn, & Brown, 2002), and functional magnetic resonance imaging (fMRI) in humans (Aron & Poldrack, 2006; Chikazoe, Konishi, Asari, Jimura, & Miyashita, 2005; Curtis, Cole, Rao, & D'Esposito, 2004). This research has emphasized the role of a number of frontal cortical sites, in particular the right inferior frontal cortex, in the decision-making process. Studies in clinical populations have also provided some insight into the relation between different patterns of brain damage or dysfunction and voluntary control. Thus, people suffering from attention deficit hyperactivity disorder (Armstrong & Munoz, 2003; Hanisch, Radach, Holtkamp, Herpertz-Dahlmann, & Konrad, 2005), schizophrenia (Badcock, Michie, Johnson, & Combrinck, 2002), alcohol intoxication (Mulvihill, Skilling, & Vogel-Sprott, 1997), or cocaine dependency (Fillmore, Rush, & Hays, 2002) all display deficits in countermanding tasks. Typically, these can be characterized by reduced inhibitory control—that is, less success in inhibiting the planned action when the stop signal is presented. As with the neurophysiological studies mentioned above, these clinical studies have

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all emphasized the role of frontal cortical structures in the inhibitory control deficits observed in these patient populations. Indeed, Aron, Fletcher, Bullmore, Sahakian, and Robbins (2003) and Aron, Robbins, and Poldrack (2004) have explicitly demonstrated that damage to the right inferior frontal gyrus causes impaired inhibition during trials with a stop signal.

Concussion, or mild traumatic brain injury (mTBI), is a neurological injury that is a consequence of a force to the head. Previous studies have found that participants with mTBI have deficits in several different sensorimotor, cognitive, and attentional processes. For example, mTBI leads to instability during locomotion that is exacerbated when a secondary distracting task is also performed (Parker, Osternig, Lee, van Donkelaar, & Chou, 2005; Parker, Osternig, van Donkelaar, & Chou, 2006). This suggests that mTBI results in dysfunction in executive control—a notion that we have confirmed directly using the attentional network test (ANT) (Halterman et al., 2006; van Donkelaar et al., 2005). The executive component of attention allows one to switch between different task constraints, and thus engages many of the same mechanisms involved in deciding whether or not to act. Therefore, it seems plausible that participants with mTBI would also be deficient in countermanding tasks.

Countermanding saccade tasks are a simplified decision making task which require voluntary control over eye movements due to an infrequent stop stimulus signaling the subject to withhold the planned movement (Hanes & Carpenter, 1999). Although there have been no reports in the literature of countermanding saccade performance in participants with mTBI, there have been previous studies comparing voluntary reflexive saccades with antisaccades in participants with mTBI (Crevits, Hanse, Tummers, & Van Maele, 2000; Heitger et al., 2004). The results of these studies have been equivocal—with one showing antisaccade deficits and the other normal antisaccade performance. Antisaccades require a participant to inhibit the normal reflexive saccade to a target appearing in peripheral vision and instead generate a saccade to the analogous location in the opposite visual hemifield. Thus, as with countermanding saccades, response inhibition is required. However, with an antisaccade task, this inhibitory signal is consistently present on every trial as a cognitive rule that the participant must apply. By contrast, during countermanding saccade, the inhibitory signal occurs more infrequently. Thus, although antisaccades probe some of the same executive functions that contribute to countermanding tasks, the nature of countermanding tasks allows deeper insight into the temporal dynamics of response inhibition.

In this study, we used the countermanding saccade task to examine the potential differences in the dynamics of the decision making process between participants with mTBI and controls. A saccade as opposed to manual countermanding task was chosen because we wanted to compare the results from this task to those from several other saccade experiments we have undertaken in participants with mTBI. We hypothesized that both the controls and participants with mTBI would display a decreased probability of cancelling the planned saccade as the stop signal delay increased, but that, given their deficits in executive function, the participants with mTBI would be less successful at each delay.

2. Materials and methods

2.1. Participants

Seventeen participants with mTBI (nine males, eight females; mean age: 21.8 ± 4.2 years; education 14.5 ± 3.0 years) were recruited from the University of Oregon community. The participants were identified by athletic trainers and/or physicians from the athletic department or student health center, and each of them was tested within 2 days following the injury. The cause of mTBI varied between participants from impacts to the head during football and soccer games to falls and biking accidents. For our purposes we consider an mTBI to be equivalent to a Grade 1–3 concussion according to the criteria set forth by the American Academy of Neurology (1997). In particular, a Grade 1 concussion leads to disorientation as to time and place for less than 15 min (for example, having difficulty knowing their location or the day and time), a Grade 2 concussion to disorientation for longer than 15 min, and a Grade 3 concussion to a loss of consciousness. Based on these criteria, all of the current participants were classified as having suffered Grade 2 concussions and none had suffered a previous concussion in the preceding 12 months. We have used these selection criteria in our previous studies which have demonstrated significant attentional orienting, executive function, and gait stability deficits in similarly categorized participants with mTBI (Halterman et al., 2006; Parker et al., 2005, 2006; van Donkelaar et al., 2005; van Donkelaar, Osternig, & Chou, 2006). In addition, the same participants that took part in the current study also completed an attentional blink paradigm and displayed deficits in the ability to adequately distribute attention across time (McIntire et al., 2006). Thus, we felt confident that the current participants could be accurately categorized as having suffered an mTBI. Seventeen control subjects from the University of Oregon were matched to the mTBI subjects for age (22.6 ± 3.5 years), gender (eight males, seven females), education (15.9 ± 2.7), and activity level (e.g., football players with mTBI were matched to teammates who played the same position).

2.2. Apparatus

The subjects were seated 57 cm away from a computer monitor displaying visual stimuli. During each experimental session the horizontal movement of the left eye was monitored using an infrared corneal reflection device (Iris Skalar) attached to a semi-rigid adjustable band placed on the head of the subject. This system provided a signal proportional to the position of the eye with respect to the head, with an optimal resolution of 2 min arc and linearity within 3% between -25° and $+25^\circ$. The system was calibrated by having subjects make saccades to targets at known eccentricities prior to, and several times during, data collection. A dental impression bite bar was used to stabilize the head throughout the experimental conditions.

2.3. Task

The countermanding saccade task consisted of a series of normal saccade trials interleaved with trials in which the saccade was to be inhibited. Fig. 1 displays the sequence of events during the task. Each trial began with a central fixation point (a plus sign subtending $\sim 1^\circ$ of visual angle) appearing in the center of the display screen for 500 ms. It then disappeared and was immediately replaced with a peripheral target (a circle subtending $\sim 1^\circ$ of visual angle) positioned 5° to the left or right of the center. The subject was required to perform a saccade to this peripheral target as quickly and accurately as possible unless a stop signal was given, in which case the subject was required to maintain central fixation. The stop signal was an auditory beep occurring on 33% of the trials. The stop signal delay (SSD), which is the time between the appearance of the peripheral target and the stop signal, ranged from 0 to 125 ms in 25 ms intervals. Note that we did not use the tracking procedure outlined by Band et al. (2003) in which the SSD varies dynamically across trials depending upon the degree of success on the preceding trials. After the eye tracker was calibrated, the participant completed 10–20 practice trials until they were comfortable with the task. They then completed five blocks of experimental trials with 36 trials per block. This resulted in ten countermanding trials for each SSD.

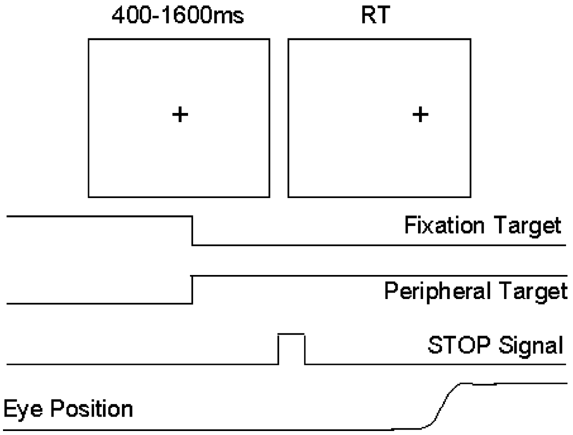

Fig. 1. Sequence of events in each trial. Top part of figure represents the visual events from the perspective of the participant during a typical trial. The bottom part displays the timing of fixation target offset, peripheral target onset, countermanding tone, and saccade response. In this particular example, the participant was unable to inhibit the saccade.

2.4. Data analysis

The main dependent variables of interest were the percentage of countermanding trials in which the subject successfully inhibited the saccade and the saccadic latency during trials in which a saccade was generated. We defined the occurrence of a saccade as a change in the position of the eyes of more than 0.5° in the direction of the target within a period of 1 s following the appearance of the target. Other than the latency, the characteristics of the saccades (i.e., amplitude, accuracy, velocity) were similar across the two groups and the different SSDs. Mixed model analyses of variance were completed to determine whether these variables were different across the two subject groups (mTBI and controls) and seven different task conditions (six SSD intervals and GO trials). The stop signal reaction time (SSRT) is an approximation of the time it takes to inhibit a planned saccade once the stop signal is presented. We calculated the SSRT according to the method outlined by Logan and Cowan (1984). This approach assumes that the SSRT represents the time between the start and finish of the stop signal. The start is demarcated by the SSD and the finish is inferred from the failure rate on STOP trials and the RT distribution on GO trials, where the latter construct represents the distribution of go signals on these trials. When the go signal finishes before the stop signal the saccade is unsuccessfully inhibited, implying that the upper limit of such responses represents the stop signal finishing time. Thus, the SSRT can be calculated for a particular SSD by rank ordering the GO trial reaction times, obtaining the reaction time at which the percentile rank is equivalent to the failure rate for that SSD, and subtracting the SSD from this value. For example, if the failure rate for an SSD of 100 ms is 75% and the rank ordered reaction time at the 75th percentile of the reaction time distribution for the GO trials is 300 ms, then the SSRT would be 200 ms.

3. Results

Fig. 2 displays the group averages for median saccadic reaction time during trials in which the stop signal was not presented (GO trials) and trials in which a saccade was made despite the presentation of the stop signal (unsuccessful STOP trials) at each of the different delays. There was no significant difference in reaction times at the different SSDs ($F[6, 224] = 1.26$, $p = 0.16$) or significant interaction between SSD and subject group ($F[6, 224] = 0.98$, $p = 0.24$), however, there was a significant main effect of group ($F(1, 224) = 5.945$, $p = 0.006$) due to the fact that participants with mTBI had shorter saccadic reaction times overall than the controls. Although the main effect of SSD was not significant, there was a trend for the reaction times on unsuccessful STOP trials to be quicker than those on GO trials—this being most apparent at the 75 ms SSD. This indicates that both groups were subtly but systematically faster to respond when they failed to stop the planned response. This has been demonstrated in previous studies (Curtis et al., 2004) and is thought to be due to the fact that failed STOP trials are those in which the GO signal makes it to the threshold more quickly than average.

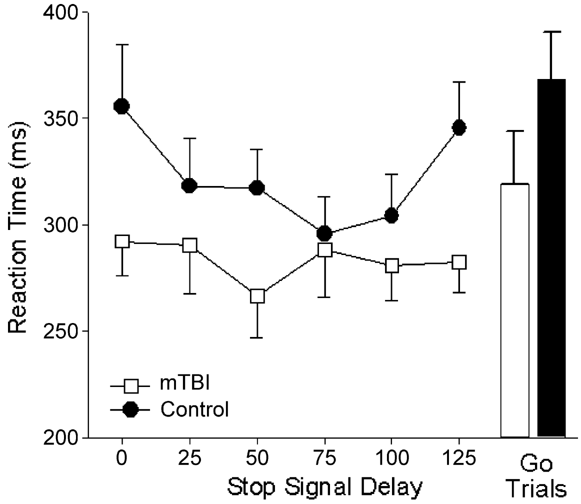

Fig. 2. Group averages for median saccadic reaction time plotted as a function of stop signal delay. Overall, reaction time was slower in the control subjects (*filled circles*) than in the participants with mTBI (*open squares*). GO trials are plotted separately in bars on the right. *Error bars*, 1 intersubject S.E.

Fig. 3 illustrates the probability of saccade execution plotted as a function of the SSD. Clearly as the SSD increases, the participants became more likely to generate a saccade. This was confirmed by a significant delay effect ($F[6, 224] = 10.372$, $p < 0.001$). Post hoc Tukey's tests demonstrated that the probability of saccade execution was significantly lower at the 0, 25, 50 ms SSDs than at the 110 and 125 ms SSDs and GO trials. This

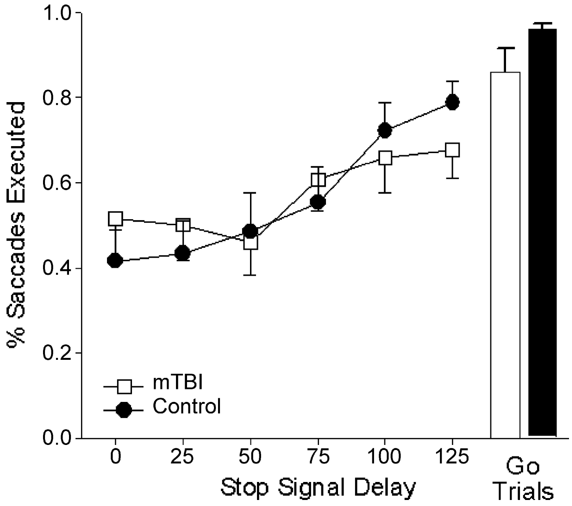

Fig. 3. Group averages for percentage of trials in which a saccade was executed plotted as a function of stop signal delay. Overall, as the stop signal delay increased the probability of saccade execution increased in both control subjects (*filled circles*) and participants with mTBI (*open squares*). GO trials are plotted separately in bars on the right. *Error bars*, 1 intersubject S.E.

change in the probability of saccade execution was similar across the two subject groups ($F[1, 224] = 0.093$, $p = 0.76$) and there was no significant interaction between SSD and subject group ($F[6, 224] = 0.76$, $p = 0.24$). Thus, to a first approximation participants with mTBI appeared to be just as able as controls to inhibit their planned actions. A peculiarity of this data is that even at the 0 ms SSD the participants could only stop themselves ∼60% of the time. This is consistent with previous countermanding saccade task performance in which the stop signal was auditory as opposed to visual (Cabel, Armstrong, Reingold, & Munoz, 2000). The important aspect of the current data is whether there is a relative change in successful response inhibition across the two subject groups.

Closer inspection of the data, however, revealed that there were subtle differences between the two groups. In particular, on GO trials the participants with mTBI actually failed to generate saccades 15% of the time. By contrast, the control group almost never failed to generate saccades on GO trials. This difference indicates that participants with mTBI have a subtle deficit in generating required saccades in the context of a countermanding task. This deficit could easily be present at the other SSDs, thus skewing the data from the participants with mTBI so that group differences were hidden.

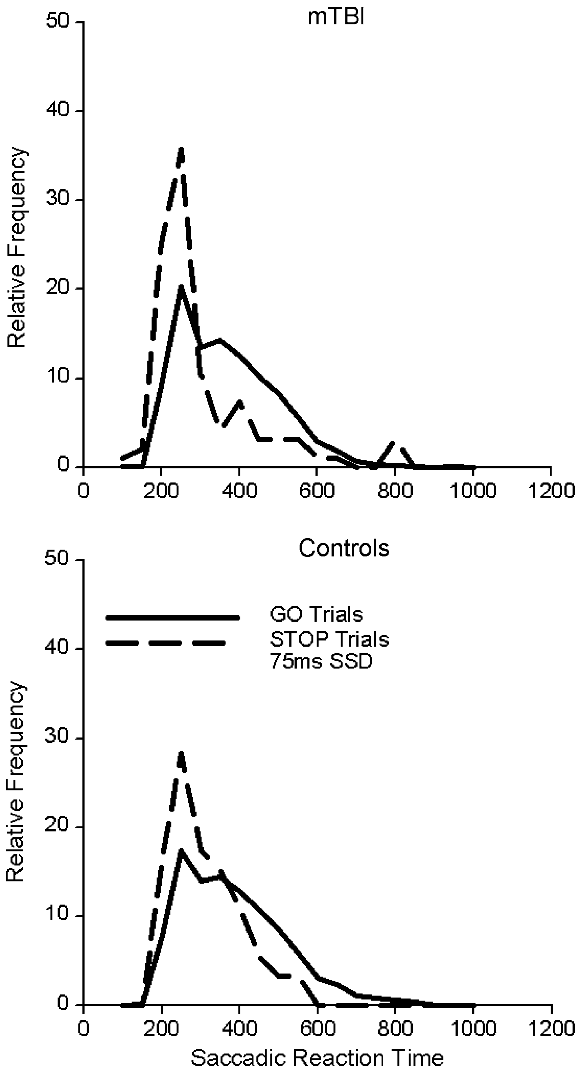

Fig. 4. Relative frequency distributions of saccadic reaction times during GO trials (*dark gray*) and failed STOP trials with a 75 ms SSD (*light gray*) for the participants with mTBI (*A*) and the controls (*B*). The relative frequency score refers to the percentage of trials within each condition that fell within each specific range of reaction times.

This subtle difference becomes more apparent in the analysis of the SSRT for each group. A sense of this difference is gained by inspection of the reaction time distributions. Fig. 4 shows the relative frequency distribution of saccadic reaction times during GO trials and during unsuccessful STOP trials for the participants with mTBI (Fig. 4A) and the controls (Fig. 4B) using the data from trials with a 75 ms SSD as an example. The intersection point between these distributions is at a shorter reaction time in the participants with mTBI than the controls. This appears to be due to a sharper peak on the unsuccessful STOP trial distribution combined with a smaller "shoulder" on the right-hand side of the GO trial distribution in the participants with mTBI. The intersection point is an estimate of the time required to process the stop signal and inhibit the planned response. This difference between the groups when comparing GO trials and unsuccessful STOP trials with a 75 ms delay was consistent across all the delays when calculated more directly using the SSRT measure. In the participants with mTBI the SSRT across all the SSDs (191 ± 37 ms) was significantly faster (t-test, $p < 0.05$) than that of the controls (262 ± 58 ms). Taken together, this data reveals that it takes a shorter period of time for the participants with mTBI to inhibit their planned action.

4. Discussion

In this study we used the countermanding saccade task as a way of examining the effect of mTBI on the voluntary control of action. Based on evidence from previous studies in different patient populations as well as our own studies on attentional deficits in mTBI, we hypothesized that participants with mTBI would display deficits in inhibitory control characterized by reduced success in countermanding saccades at each stop signal delay. This hypothesis was not supported. Instead, we found the following unique set of characteristics in the participants with mTBI: they failed to generate saccades on 15% of the GO trials; they had faster saccadic reaction times overall than controls; and their SSRT was significantly faster than that of controls. In what follows we discuss each of these characteristics.

Perhaps the most surprising result is that the participants with mTBI failed to generate a saccade on 15% of the GO trials. By contrast, the controls, as expected almost never failed to generate a saccade on these trials. Thus, some aspect of generating saccades in the context of a countermanding task leads participants with mTBI to inappropriately inhibit the planned action despite the absence of the stop signal. The fact that saccadic reaction times on the GO trials (∼325 ms) are markedly longer than typical saccadic reaction times produced without the countermanding contingency (∼200 ms, e.g., Reddi & Carpenter, 2000) and that participants with mTBI display normal reflexive saccadic latencies (Heitger et al., 2004) suggests that the inhibitory stop signal contributes to saccade initiation processes even when the external inhibitory stimulus is not present. Clearly, this signal played a larger role in saccade initiation during the GO trials in participants with mTBI resulting in a much larger proportion of inappropriate failed GO trials. Based on this observation, we suggest that the stop signal is more easily triggered in participants with mTBI than it is in controls.

Another aspect of the current results that warrants discussion relative to these failed GO trials in the participants with mTBI is the rate of success in the STOP trials across the two subject groups. We found that both groups were equally successful at inhibiting saccades at each of the SSDs. This result is consistent with at least one previous study examining antisaccade performance in participants with mTBI (Crevits et al., 2000). However, there is no reason to assume that the explanation provided above for the failed GO trials in participants with mTBI does not also apply to the successful STOP trials. After all, the participants do not know as the trial evolves whether it will be a STOP or GO trial. As such, if the more easily triggered response inhibition in the participants with mTBI was not present, it would lead to a reduced likelihood that a saccade would be inhibited at each of the SSDs relative to the controls—an effect that is similar to that observed in countermanding tasks in other neurological conditions (Armstrong & Munoz, 2003; Aron et al., 2003, 2004; Badcock et al., 2002; Fillmore et al., 2002; Hanisch et al., 2005; Mulvihill et al., 1997).

In addition to these effects in terms of inhibiting responses, participants with mTBI were also quicker overall in initiating the saccades that they did generate. These reductions in reaction time were also apparent in the SSRT scores. Taken together, these latency results imply that participants with mTBI had difficulty withholding their reflexive saccades. Because the main goal of the countermanding task is to inhibit the planned action on a minority of trials, the more easily triggered stop signals apparent in participants with mTBI would allow them to both compensate for their difficulty withholding saccades and yet still achieve acceptable levels of response inhibition. The only drawback of this approach is that it leads to the relatively low cost of a small proportion of failed GO trials. Thus, the attempt to control errors during STOP trials caused an increase in errors on GO trials. Nevertheless, this highlights the fact the participants with mTBI were actively attempting to develop coping strategies to deal with the complex demands of countermanding tasks.

Given that participants with mTBI have a subtle deficit in inhibitory control one can speculate as to the areas of the brain that may be affected. In particular, a number of previous studies have emphasized the contributions from frontal cortical sites including the frontal eye fields, supplementary eye fields, anterior cingulate cortex, and right inferior frontal cortex (Aron & Poldrack, 2006; Chikazoe et al., 2005; Curtis et al., 2004; Schall, 2001). Based on the current results, it seems plausible that these areas are particularly susceptible to the effects of mTBI. Future studies using functional brain imaging should be conducted in this population to examine whether the ease of triggering response inhibition relates to differential activation in some or all of these sites, and in particular in the right inferior frontal cortex.

It is also possible to speculate on how changes in the underlying cortical physiology may account for the alterations in performance that were observed in participants with mTBI. Head impacts associated with mild forms of TBI can cause diffuse axonal injury due to stretching and twisting that have both acute and chronic effects (Gaetz, 2004). This can lead to alterations in the ability of ions to move across the axonal membrane that, in turn, can cause changes in the efficacy of synaptic transmission. GABA is the most common inhibitory neurotransmitter in the brain, so it seems likely that changes in behavior driven by inhibitory signals would be influenced by alterations in this molecule. In fact, there is evidence that mTBI leads to changes in the cortical silent period induced by transcranial magnetic stimulation over the motor cortex (De Beaumont, Lassonde, & Théoret, 2006)—an alteration associated with GABA activity (Werhahn, Kunesch, Noachtar, Benecke, & Classen, 1999). Further studies designed to address these issues should be completed in an attempt to understand these deficits across several levels of analysis.

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