



## Attentional disengagement dysfunction following mTBI assessed with the gap saccade task

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### Abstract

Concussion, or mild traumatic brain injury (mTBI), leads to a number of cognitive, attentional, and sensorimotor deficits that can last a surprisingly long time after the initial injury. We have previously shown that the ability to orient visuospatial attention is deficient in participants with mTBI within 2 days of their injury, but then recovers to normal levels within a week. Orienting attention requires disengagement from the point of fixation, movement of attention to the location of interest, and reengagement at that location. Deficits in any or all of these processes could lead to the difficulties with orienting attention that we have observed in mTBI. To address this issue, we tested participants with mTBI using a gap saccade task. Because this task manipulates the temporal gap between the offset of the fixation target and the appearance of the peripheral saccade target, it isolates the contribution of the disengagement process to saccadic reaction time. We found that participants with mTBI had significantly longer saccadic reaction times than controls when the temporal gap was short but not when it was long. This gap-dependent difference in saccadic reaction time was present within 2 days of the injury and resolved within 1 week. This pattern of results suggests that as the contribution of the disengagement process is reduced, so too is the extent of the reaction time deficit in the participants with mTBI. Taken together, this is consistent with the idea that the deficits in orienting visuospatial attention in participants with mTBI are fully accounted for by difficulties with the initial disengagement process.

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Concussion, or mild traumatic brain injury (mTBI), has been defined as any transient neurological dysfunction that results from experiencing a biomechanical force to the head [14]. This mild form of brain injury can cause substantial damage in the form of focal lesions and/or diffuse axonal injury [6,17,18,22]. While more severe symptoms, such as loss of consciousness, are often associated with mTBI, studies have shown that the alteration of consciousness is sufficient for a diagnosis [32].

Individuals who have recently suffered an mTBI commonly display attentional deficits on tasks assessing the ability to maintain attention during single task performance, as well as appropriately distribute attention while performing multiple tasks simultaneously [4,5,7,12,25,33,34]. We have previously shown using the Attentional Network Test [11] that participants with mTBI are deficient in their ability to orient visuospatial

attention within 2 days of their injury, but then recover to normal levels within a week [15,36].

Directing attention in space requires the disengagement of attention from the central fixation point, the movement of attention to the peripheral location, and the reengagement of attention at the new location [26]. These processes have been shown to engage a diffuse network of areas, including the frontal, temporal, and parietal cortices [19,24,27-29,35]. In addition, many of these areas are also activated by saccadic eye movements [9], suggesting that spatial attention and saccade planning are closely associated.

The role of attentional disengagement in the process leading up to saccade generation can be characterized by making use of the gap paradigm. With this approach, a temporal gap is inserted between the offset of the central fixation point and the appearance of the peripheral target [20,31]. Healthy subjects exhibit a reduction in saccadic latency when the temporal interval is between 50 and 300 ms [30]. This has been termed the gap effect and is thought to reflect the release of attentional engagement

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35 from the central fixation point. In addition to this disengagement  
36 process, the offset of the target serves to alert the subject that  
37 a peripheral target will be presented shortly. The gap effect has  
38 been shown to be present in several attentional and oculomotor  
39 tasks including anti-saccades [3,13] and saccades made to  
40 peripheral exogenously cued targets and central endogenously  
41 cued targets [1]. Furthermore, it has also been established that  
42 neuronal activity in the several cortical and subcortical areas are  
43 modulated during performance of a gap saccade task [8].

44 In the present experiment, we tested participants with mTBI  
45 using the gap paradigm. We hypothesized that an alteration in the  
46 gap effect compared to control subjects would be evidence that  
47 part of the difficulty participants with mTBI have in directing  
48 attention in space is related to a deficit in disengaging attention  
49 from the current point of fixation. This alteration could be  
50 reflected in the data in a number of different ways. First, if  
51 participants with mTBI have difficulty fully disengaging attention  
52 from the fixation location, then there should be no, or very  
53 little, observable gap effect in their saccadic reaction time. Second,  
54 if participants with mTBI are able to disengage attention but the  
55 process is impaired by the effects of the injury, one would expect  
56 to see an increase in saccade latency at gap durations when the  
57 process of disengaging attention is taking place: 0–150 ms. Third,  
58 if participants with mTBI have difficulty with saccade preparation  
59 but not attentional disengagement, then the gap effect should be  
60 present but all the saccadic latencies would be shifted upwards.  
61

62 Twenty participants with mTBI (12 males, 8 females; mean  
63 age:  $21 \pm 1.74$  years [age range: 18–24 years]; education:  
64  $16 \pm 1.65$  years) were recruited from the University of Oregon  
65 undergraduate student community. These same participants also  
66 completed testing on the Attentional Network Test (ANT) that  
67 we have reported on previously [15,35]. A demographics table  
68 for these participants is included in the former publication [15].  
69 All were involved in intercollegiate, club, or intramural sports,  
70 or recreational activities. They were initially identified by certified  
71 athletic trainers and/or attending medical doctors in the university  
72 intercollegiate athletic program or the student health center,  
73 and were referred for testing within 2 days (mean elapsed time:  
74  $37 \pm 11.5$  h; range: 12–50 h) following their injury. Additional  
75 testing occurred 1 week, 2 weeks, and 1 month after the injury.  
76 The cause of the injury varied from impacts to the head occurring  
77 during football games to accidents while participating in recreational  
78 sports and falls. Each of the participants was categorized as having  
79 a Grade 2 concussion according to the standards established by the  
80 American Academy of Neurology [2]. For a Grade 1 concussion,  
81 participants had to be disoriented as to time and place for less than  
82 15 min (for example, having difficulty knowing their location or the  
83 day and time), whereas for a Grade 2 concussion the disorientation  
84 could last longer than 15 min. Participants who sustained a Grade 3  
85 concussion, defined by a loss of consciousness for any period of  
86 time, and participants who had suffered a previous concussion  
87 within the last 12 months were excluded from the study. None  
88 of the participants with mTBI were taking any medication that  
89 directly or indirectly affected attention or oculomotor output.  
90 Control participants from the same undergraduate student pop-  
91

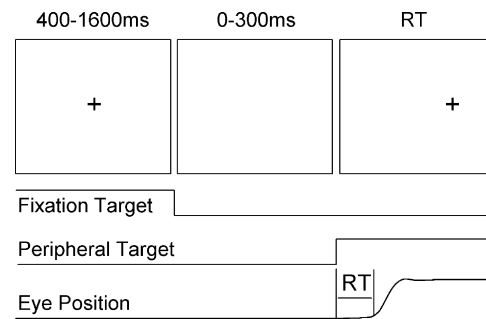


Fig. 1. Gap saccade task. Participants initially visually fixated a central target. After a variable delay the target disappeared and a temporal gap between 0 and 300 ms occurred prior to the appearance of a peripheral target 5° or 10° to the left or right of center. Participants were instructed to make a saccade to the peripheral target as quickly and accurately as possible. RT, saccadic reaction time.

92 ulation matched for age (mean age:  $21 \pm 1.81$ ; years age range:  
93 [18–24]), gender (12 males, 8 females), activity (e.g., football  
94 players were matched with teammates who played the same  
95 position), and education level ( $16 \pm 1.68$  years) to individual  
96 participants with mTBI were also tested. The experiments were  
97 conducted in accordance with the Declaration of Helsinki and  
98 all of the participants signed an informed consent form prior to  
99 partaking in the study and the local university human subjects  
100 compliance committee approved the experimental protocol.

101 Subjects were seated in a dimly illuminated room in front of  
102 a computer screen and asked to visually fixate a target presented  
103 on the screen 57 cm in front of them. The target consisted of  
104 a set of crosshairs (e.g. a plus sign subtending  $\sim 1^\circ$  of visual  
105 angle) (Fig. 1). After a variable delay (400–1600 ms), the target  
106 disappeared and subjects made a saccade to fixate a new target  
107 (another set of crosshairs subtending  $\sim 1^\circ$  of visual angle). On  
108 a given trial, the saccade target could appear to either the left  
109 or right of the fixation point at a distance of 5° or 10° of visual  
110 angle. On gap trials, a temporal interval (50, 100, 150, 200,  
111 250, or 300 ms) was inserted between the disappearance of the  
112 fixation point and the appearance of the saccade target. On ‘no  
113 gap’ trials, the temporal interval equaled zero. A small number  
114 of catch trials (three per block) in which no peripheral target  
115 appeared were also included to help reduce the predictability of  
116 the task.

117 After  $\sim 10$  practice trials to become acquainted with the task,  
118 subjects performed 8 blocks of 29 trials each for a total of 232  
119 trials per experimental session. Of the 29 trials in each block,  
120 5 were no gap trials, 12 were trials in which the saccade target  
121 appeared to the right of the fixation point (6 at 5°; 6 at 10°) and  
122 12 were trials in which the saccade target appeared to the left  
123 of fixation (6 at 5°; 6 at 10°). The six trials at each peripheral  
124 location were associated with one of the six possible temporal  
125 intervals or gaps. Trial order was randomized within blocks.

126 During each experimental session the horizontal movement  
127 of the left or right eye was monitored at 200 Hz using an infrared  
128 corneal reflection device (Iris Skalar) attached to a semi-rigid  
129 adjustable band placed on the head of the subject. This system  
130 provides a signal proportional to the position of the eye  
131 with respect to the head with an optimal resolution of 2 min

arc and linearity within 3% between  $-25^\circ$  and  $+25^\circ$ . The system was calibrated by having subjects make saccades to targets at known eccentricities prior to each block of trials. A dental impression bite bar was used to stabilize the head throughout the experimental conditions.

Matlab computer software was used to examine and analyze the characteristics of the main dependent variable of interest: saccade reaction time. We defined the occurrence of a saccade as a change in the position of the eyes of more than  $0.5^\circ$  in the direction of the target within a period of 1 s following the appearance of the target. Reaction time was defined as the time between the appearance of the peripheral saccade target and the initial displacement of the eyes toward the saccade target. A graphical user interface was implemented in Matlab to allow the experimenter to mark the onset of the saccade on each trial for this purpose. Reaction times less than 80 ms and greater than 700 ms were discarded. These accounted for  $\sim 2\%$  of the trials. Saccades made in the wrong direction were also not included although they were very rare (five trials). Analyses of variance were performed to investigate the differences in saccadic reaction time across the different gap durations for each subject group. Specifically, 2 (subject group)  $\times$  7 (gap duration) mixed model ANOVAs were performed on the data from each testing day.

Initial analysis of the saccadic reaction times revealed that there were no differences within each group related to target direction or amplitude of the peripheral target, so the data from these different conditions were combined for each subject. Fig. 2 displays the resulting average median saccadic reaction times for each participant group at each of the gap durations. The different testing days are plotted in each panel. The gap effect

is apparent by the general trend for reaction time to decrease as gap duration increases from 0 to  $\sim 150$ – $200$  ms and then increase slightly as gap duration increases further. This is clearest during the later testing days. In addition, during the first, and to a certain extent second, testing days the participants with mTBI have markedly slower reaction times than the controls for the shortest gap durations. These effects were captured with ANOVAs performed separately on the data from each visit. During the first visit within 2 days of the injury, participants with mTBI were slower overall than controls ( $F[1, 38] = 18.753$ ,  $p < .0001$ ) and there was a significant gap effect for both groups ( $F[6, 228] = 7.582$ ,  $p = .011$ ). Post hoc Tukey's tests revealed that this latter effect was due to reaction times being significantly faster during trials with 150 and 200 ms gaps than with 0 and 50 ms gaps. In addition, there was a significant interaction between group and gap duration ( $F[6, 228] = 2.824$ ,  $p = .032$ ). This was due to reaction times in the participants with mTBI being markedly slower than that of controls for the shortest gap durations (0–100 ms), but similar to that of controls at the longer gap durations (150–300 ms). ANOVAs performed on the data from the subsequent visits revealed a consistent gap effect (Visit 2:  $F[6, 228] = 6.441$ ,  $p = .0128$ ; Visit 3:  $F[6, 228] = 41.621$ ,  $p < .0001$ ; Visit 4:  $F[6, 228] = 36.152$ ,  $p < .0001$ ), but no group effects or interactions between group and gap duration. As with the data from the first visit, post-hoc Tukey's tests revealed that the significant gap effect was generally due to reaction times at the mid-range gap durations (150–200 ms) being quicker than those at the shortest gap durations (0–50 ms). There was also a tendency for greater inter-individual variability in reaction time in the participants with mTBI during the first visit but not the later visits.

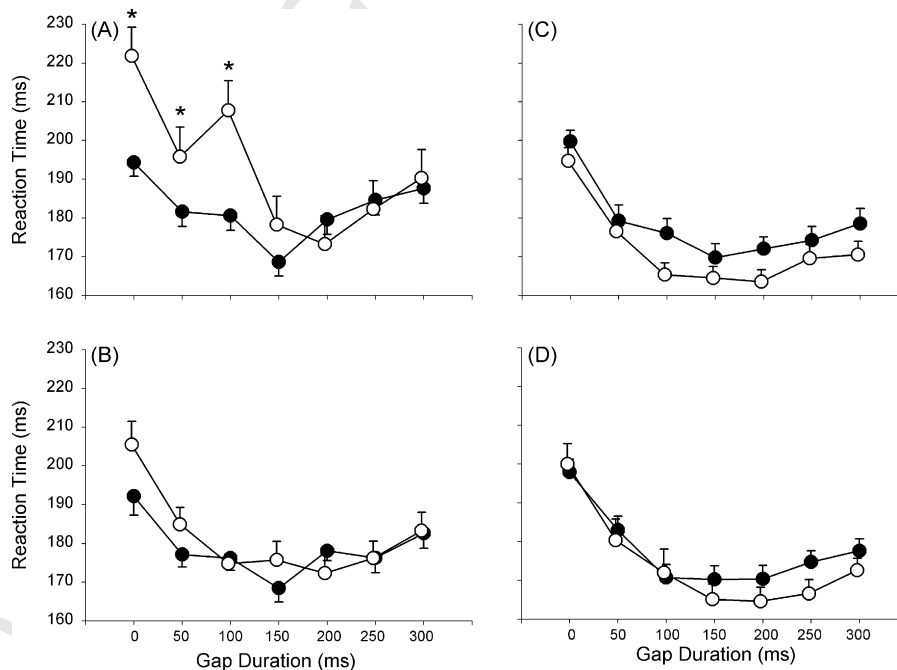


Fig. 2. Group averages for median saccadic reaction time for the participants with mTBI (open circles) and controls (filled circles) as a function of gap duration. The participants with mTBI were markedly slower to initiate their saccades when gap duration was short during the first visit (A). This difference was substantially reduced 1 week (B), 2 weeks (C) and 1 month (D) after the injury. Asterisks, mTBI latency significantly greater than control latency. Error bars, one intersubject S.E.

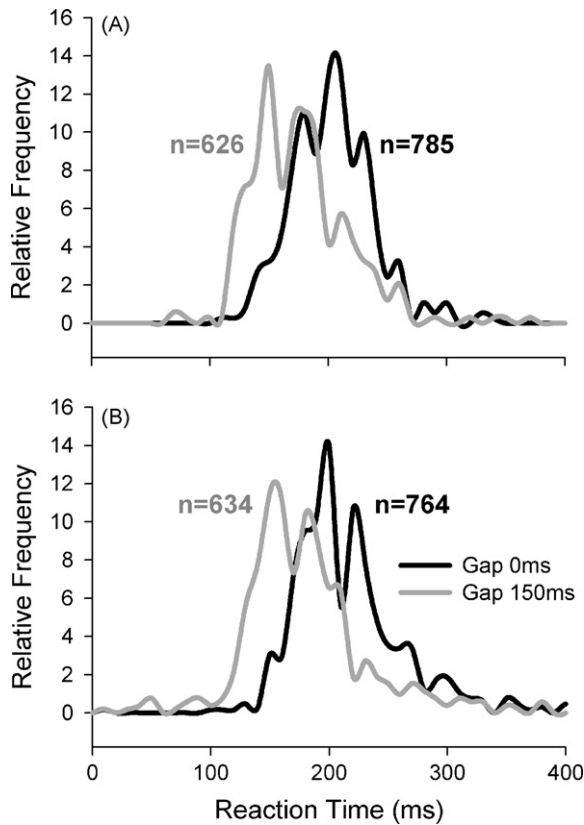


Fig. 3. Relative frequency distributions of saccadic reaction times from the first visit during trials with a 0 ms gap (black line) or 150 ms gap (gray line) for the controls (A) and the participants with mTBI (B). The relative frequency score refers to the percentage of trials within each condition that fell within each specific range of reaction times. The reaction time interval size was 10 ms.

To get a sense of whether the gap effect was due to similar alterations in the reaction time distribution across the two groups, we plotted these distributions for data from the first visit for the 0 and 150 ms gap conditions (Fig. 3). These conditions were chosen because the latencies in the 0 ms gap condition were markedly slower than those from the 150 ms gap condition, and this was especially apparent in the participants with mTBI. For both participant groups, the distributions appeared to be qualitatively similar. In particular, in the 0 ms gap condition the distribution peaked at ~200 ms, with the increased latencies overall for the participants with mTBI in this condition being due to a slight shift rightwards in the shoulders of the distribution (i.e., fewer trials with rapid reaction times and more with sluggish reaction times). By contrast, the distribution for the 150 ms gap condition had a prominent early component resulting in two peaks—one at ~150 ms and another at ~185 ms. Thus, it appears that the reaction time distributions of both participant groups were modulated in a similar manner by the introduction of the 150 ms gap.

When a temporal gap occurs between the disappearance of a foveated target and the appearance of a subsequent peripheral target healthy subjects produce faster saccadic reaction times [30]. This gap effect is characterized behaviorally as a reduction in saccade latency as gap duration increases from 0 to 150 ms followed by increased saccade latency as gap duration increases

from 150 to 300 ms. It is thought to be due to a reduction in the contribution of the disengagement component of attentional orienting as well as an alerting effect brought about by the disappearance of the fixation target. This study made use of the gap saccade task as a means of examining the effect of mTBI on these processes. Individuals with mTBI have been shown to have difficulties associated with appropriately distributing visuospatial attention [15,36]. The results from the current study demonstrated that the gap effect was present in participants with mTBI but that within the first 2 days of the injury, they had markedly slower reaction times for the shortest gap durations. In what follows, we discuss how our results compare to other studies investigating the effects of mTBI on attentional orienting.

Our results show that both the participants with mTBI and controls exhibited a gap effect across all testing sessions consistent with previous studies. The mTBI group, however, showed a pattern of results significantly different from controls during the first session. During this visit, saccade latency was significantly longer at the 0, 50, and 100 ms gap durations in the mTBI group but not at the longer gap durations. This implies that as the extent of the contribution of the disengagement process was reduced (with increasing gap durations), the saccadic reaction times of the participants with mTBI normalized. We suggest that this demonstrates that the deficit we have previously observed in the orienting of visuospatial attention in participants with mTBI [15,36] is due specifically to difficulties disengaging attention rather than to problems orienting and reengaging it at the peripheral target. The alterations in the gap effect in participants with mTBI could also imply that they are unable to take advantage of the alerting cue that occurs when the fixation target disappears. However, in our previous studies we have shown that the alerting component of attention is unaffected in this population [15,36]. Taken together these results suggest that the network of cortical and subcortical structures underlying visuo-spatial orienting, and particularly the disengagement component of orienting, is subtly susceptible to mTBI within the first two days following the injury. Recent neurophysiological studies have identified the superior colliculus and the frontal and parietal areas projecting to it as playing a vital role in the gap effect [10,21,23]. The current data implies that these sites are affected by an mTBI. The increased inter-individual variability in reaction time during this time is also consistent with the subtle effects of mTBI on brain function. The effects observed in the present study were quite short lasting relative to other previous work demonstrating more enduring attentional deficits [4-6]. This suggests that the patient population used in the current study suffered from a very mild form of TBI.

These results confirm and extend a recent study by Heitger et al. [16] who found saccadic abnormalities in participants with mTBI but only for more complex saccade tasks such as anti-saccades and memory-guided saccadic sequences. By contrast, visually triggered and guided saccades were essentially normal. This suggests that the deficits we have observed in the present study are more likely to be due to difficulty disengaging attention and not to any basic problem planning and executing saccades. If such problems were present this would have been evident in

longer reaction times in the participants with mTBI across all the gap durations.

In conclusion, we have demonstrated that individuals with mTBI present with deficits with the disengagement process of attentional orienting. This implies that the cortical and sub-cortical locations involved in the disengagement process are vulnerable to mTBI. This information could be of particular value to researchers investigating the decreased attentional capabilities following this form of brain injury.

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