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THE EFFECTS OF INDIVIDUALIZED COGNITIVE REHABILITATION FOR IMPROVING PROSPECTIVE MEMORY IN ACQUIRED BRAIN INJURY

BY

Emily M. Aiken
B.S. *with honors* in Neuroscience
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(Date)

appear below, met wit this candidate for a final discussion of the thesis.

ADVISER Signature_____
Sarah Raskin

READER Signature_____
Molly Helt

Date: _____

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Abstract

Acquired Brain Injury (ABI) includes any damage to the brain resulting from traumatic (e.g. motor vehicle accident) or non-traumatic (e.g. stroke) incidence, that occurs after birth and is not resulting from genetic or congenital factors. Individuals with ABI report that prospective memory (PM) deficits are the most detrimental cognitive impairment following injury, persistently and negatively impacting their ability to function properly in everyday life. PM refers to the ability to remember to carry out intended tasks in the future, including the recall of both time and event regulated intentions. Using neuropsychological assessments to produce patient deficit profiles, this study examines the effectiveness of individualized cognitive rehabilitation therapies: attention process training (APT) or PM training, for improving PM in ABI. Participants were randomly assigned to groups, completing 10 sessions of either cognitive rehabilitation (n=4) or educational programming (n=3). Using the Memory for Intentions Screening Test (MIST), intra and inter treatment analyses examined the effectiveness of individualized cognitive rehabilitation for improving PM in ABI (N=7).

Introduction

Difficulties in daily life and degrees of cognitive deficit that present in individuals with acquired brain injury (ABI) are different for each person. Though there is variability from person to person, individuals with ABI report that prospective memory (PM) impairments are the most chronic (Tay, Ang, Lau, Meyyappan & Collinson, 2010) and detrimental to their ability to function properly in daily life (Shum, Fleming & Neulinger, 2002). Therapies for addressing and improving PM are still quite new and undeveloped, however it has been suggested that PM deficits may not be independent from other cognitive deficits faced by ABI patients (Mateer & Raskin, 1999). Furthermore, PM processing is rather complex and requires intact function across various cognitive domains (attention, memory and executive functioning) (Ellis, 1996). To better understand the most effect means of treating PM deficits, this study examines the effectiveness of an individualized approach to cognitive rehabilitation in comparison to active control treatment in adults with ABI.

Acquired Brain Injury

ABI refers to injuries to the brain that occur after birth which are not caused by biological or genetic factors (Brain Injury Association of America, 2014). Examples of ABI include but are not limited to traumatic brain injury (TBI), cerebral vascular accidents (CVA) and oxygen depletion (Taub, Bartuccio & Manio, 2012). In the United States, approximately 3.5 million Americans sustain an ABI (Brain Injury Alliance of Connecticut, 2016).

Traumatic brain injury (TBI) describes any injury to the brain that is caused by external force (Brain Injury Association of America, 2014). The Centers for Disease Control and Prevention (CDC) reports falls, blunt force trauma, assaults and motor vehicle incidents to be the leading causes of TBI (CDC, 2016). TBI diagnoses range from mild to severe, depending on the

occurrence and duration of loss of consciousness (BIAC, 2016). On average 1.4 million people in America suffer from TBI per year; approximately 17% require hospitalization, 6% experience long-term disability leading to inability to work, and 4% of cases report to be fatal (CDC, 2010; CDC, 2016).

Both CVA, such as stroke, due to blood depletion in a specific brain region, and oxygen depletion leading to hypoxia, are internally elicited ABIs (Brain Injury Association of America, 2014). Each year 15 million individuals around the globe suffer from a stroke. With a fatality rate of approximately 33% and a 33% incidence of permanent disability, stroke is considered a chronic from of ABI with numerous causes and risk factors (World Health Organization as cited in The Internet Stroke Center, n.d.).

Individuals with ABI may experience a variety of deficits following their injury. These difficulties include but are not limited to emotion regulation, language, sensorimotor abilities and cognitive functioning (BIAC, 2016). The severity of ABI correlates to the daily difficulties and cognitive deficits each individual experiences; with the most common difficulties involving attention, memory and executive functioning (Mateer & Raskin, 1999).

Attention

Definition & Identification of Subtypes. Attention as a cognitive process is responsible for the enhancement and inhibition of stimulus information, so that more in-depth processing can occur for desired information at a given time (Smith & Kosslyn, 2007). Divided into subsets: selective, sustained and divided; intact attention capabilities are required for successful daily functioning.

Selective attention facilitates the interactions between perception and corresponding action, thus facilitating concentration on specific stimuli within the environment (Houghton &

Tipper, 1994; McKay, Halperin, Schwartz & Sharma, 1994). Selective attention is responsible for controlling which sensory input is attended to, whereas sustained attention refers to the continued cognitive arousal or vigilance over a period of time (McKay et al., 1994). Thus, in practice, hearing your name aloud in a noisy space requires selective attention, and the ability to carry conversation or stay tuned to a lecture over a period of time, call upon sustained attention. Both attention subsets may occur consciously or subconsciously. More complex in nature, divided attention requires the ability to simultaneously perform two or more tasks, both of which require one's attention (Hahn, Wolkenberg, Ross et al., 2008), such as driving while listening to the radio. Alternating attention also involves two tasks, but requires one to alternate between the tasks, such as cooking a meal while periodically monitoring your child's homework.

Neural Correlates. The neural correlates of attention differ based on source of stimuli, such as visual versus auditory inputs. Environmental inputs follow their corresponding processing pathways until ultimately reaching the temporal lobe, where information inhibition and enhancement occur (Desimone, 2007). Attentiveness to certain stimuli is postulated to result from neural firing synchrony, mediated by frontal and parietal regions, rather than as a result of degree of neuronal activity within a given brain region (Desimone, 2007).

Numerous hypotheses have been made pertaining to the exact neural mechanism behind attention. Some researchers have proposed a series of independent attentional pathways, whereas others propose an interconnected circuitry responsible for attention. A meta-analysis of various attentional network mechanisms, suggests a triad of networks termed, altering, orienting and executive attention; that in combination form the overall attentional network (Raz & Buhle, 2006). By the most elementary explanation, alerting and orienting attention may be used synonymously with sustained and selective attention, whereas executive attention encompasses

divided attention bust also, in a more general sense, refer to more complex attentional tasks. Consolidation of findings from various neuroimaging studies have identified right dorsolateral prefrontal cortex, right anterior cingulate cortex, right inferior parietal region and left hemispheric temporal area activity in examining alternating attention; pulivinar, superior colliculus, superior parietal and temporal lobe, and tempoparietal junction involvement in orienting attention; and both dorsal and rostral anterior cingulate cortex activity in executive attention (Raz & Buhle, 2006). As illustrated here, the neural correlates for the various attentional domains are spread though frontal, parietal and temporal lobes; all regions which are susceptible to damage resulting from ABI.

Attention & ABI. Individuals with ABI often suffer from deficits of attention that, in turn, elicit difficulties in daily functioning. Attentional deficits resulting from ABI may alter a person's ability to learn new information or keep track of tasks, thus causing the individual to become easily frustrated and often embarrassed (Robertson, Manly, Andrade, Baggeley & Yiend, 1997). In a study comparing reaction time tasks between health and ABI persons, results for individuals across varying ABI severities indicate deficits in divided attention, speed of information processing and inability to remain focused on the tasks provided (Stuss et al., 1989). Additionally, similar deficits in speed of processing and selective attention have been identified with ABI damage that is localized to the medial thalamic region (Kraft et al., 2014). Attention dysfunction after ABI is variable, as there are so many domains of attention. However, improvement of attention deficits is critical for future improvement of the more complex cognitive deficits of ABI.

Executive Function

Definition. Executive functions consist of a wide range of cognitive processes thus making a universal definition difficult to produce. These interconnected complex cognitive processes encompass working memory, planning, organization, thought plasticity, and situational adaptability, which holistically elicit goal-mediated behaviors (D'Esposito & Gazzaley, 2005; Elliot, 2003). Executive functions are goal-driven processes that are necessary for proper mental control and self-regulation (Cooper-Kahn & Dietzel, as cited on LD OnLine, 2014). Proper executive function capabilities enable the completion of automatic cognitive processes controlled via planning, monitoring, activating, switching, and inhibiting functions, and depend on intact working memory and information modulation capabilities (Cicerone, Levin, Malec, Stuss & Whyte, 2006).

Neural Correlates. The terms executive function and frontal lobe function are often synonymously used (Elliot, 2003). Historically the frontal lobes have been identified as the primary area of activation with regards to executive functions, however additional brain regions have been identified (D'Esposite & Gazzaley, 2005; Elliot 2003; Perna, Loughan & Talka, 2012). As addressed by Elliot (2003), various clinical findings have implicated the prefrontal cortex, striatum and basal ganglia in disruptions within subsets of executive function.

Executive Function & ABI. As previously mentioned, ABI survivors suffer from executive function deficits that can pose difficulties in everyday life if left untreated. Perna, Loughan and Talka (2012), provide numerous examples of these daily difficulties faced by individuals with ABI; inability to initiate basic tasks such as trips to the grocery store, balancing a checkbook or ability to adjust to novel social situation, to name a few.

Prospective Memory

Definition. PM is remembering to do an intended action at a specific time or in response to a specific cue in the future (McCauley, McDanial, Pedroza, Chapman & Levin, 2009). The ability to carry out intended tasks in the future is vital for everyday activities such as remembering to take one's medications (Raskin & Sohlberg, 2009). There are two types of PM: event and time based. Remembering to take medications at the proper time each day exemplifies time-based PM, whereas remembering to mail a letter when seeing a mailbox, is an example of event-based PM.

Ellis (1996) discusses the complexity of PM processing in comparison to retrospective memory by suggesting that PM processing consists of five distinct steps: (1) the formation of an intended action, (2) retention of the intention, (3) remembering what was intended, (4) completing the intended actions, and (5) determining if the produced outcome was correct. Further analysis of Ellis' (1996) proposed steps of PM processing shows that the success of each step is dependent on other cognitive capabilities; step 1 depends on proper attention functioning, steps 2 and 3 rely on intact memory processes, and steps 4 and 5 require intact executive function capabilities.

Neural Correlates. Various brain regions are involved in PM processing; however, significant emphasis has been placed on the role of the frontal lobes in PM (McDaniel & Einstein, 2007). The bilateral frontal poles, right lateral prefrontal cortex, interior parietal lobe, and precuneus of the superior parietal lobe have all shown activation in response to maintaining an intention (Burgess, Quayte & Frith, 2001). Whereas increased thalamic activity and decreased right lateral prefrontal activation have been observed in response to intention realization (Burgess, Quayte & Frith, 2001).

PM & ABI. Deficits of PM associated with ABI threaten an individual's ability to live and function independently. Patients with diagnosed ABIs perform significantly worse on PM assessments in comparison to healthy participants, specifically on assessments focused on the ability to recall an intended action (Groot, Wilson, Evans & Watson, 2002; Raskin Buckheit & Waxman, 2012). More importantly, self-reports indicate that PM deficits are an individual's primary cognitive complain post ABI (Tay et al., 2010), and of all the memory impairments experienced by patients with ABI, deficits of PM appear to be the most detrimental to daily functioning (Shum, Fleming & Neulinger, 2002).

Recovery in ABI

Neural plasticity refers to the nervous system's ability to change in structure, connectivity or functionality (Chen, Epstein & Stern, 2010). Past research suggested that neural plasticity was limited to early development (Stiles, 2000), however more recent studies utilizing neuroimaging methods have provided evidence showing changes in brain structure and function in mature adult brains (Cerasa, Gioia, Valentino et al., 2013).

Following brain damage, two forms of recovery have been demonstrated to occur. Spontaneous recovery or reorganization refers to the changes (structure, function and/or network connectivity) that occur naturally after brain injury, without any assistance or intervention (Chen, Epstein & Stern, 2010). Spontaneous recovery occurs over varying time frames given the nature of the brain damage, and in some cases may not occur at all. The second form of recovery or reorganization that can occur following brain damage results from training or interventions (Chen, Epstein & Stern, 2010). This form of recovery is exemplified through the use of restorative cognitive rehabilitation (CR) to alter cognitive functionality but by changing brain structure or neuronal activity.

Cognitive Rehabilitation in ABI

Cognitive rehabilitation (CR) as with all other forms of rehabilitation, aims to improve an individual's ability to adapt and function in their daily life (Raskin, 2011). There are two primary means of cognitive rehabilitation: (1) utilization of compensatory methods and (2) restorative approaches.

Compensatory Methods. Compensatory methods alter an individual's behaviors and/or environment, thus allowing them to compensate in areas where deficits may present themselves (Raskin, 2010). Compensatory methods do not treat the actual problem, rather they are meant to help the individual so that they can function better in day-to-day life. Posting a sticky note on the front door that reads "Don't forget your keys!", removing oneself from a noisy environment while doing work, or setting an alarm on one's phone to remind one to take their medication at the correct time, are all examples of compensatory CR approaches. These methods may provide some assistance; however, they are only beneficial if the affected individual remembers to utilize the aids and/or remembers what to do when provided with a stimulus cue.

Restorative Interventions. Often used in conjunction with compensatory methods, restorative CR approaches focus directly on the problem, aiming to improve or respite specific cognitive functions (Raskin, 2010). Restorative CR can target specific cognitive functions (attention, memory or executive function), thus allowing for an individualized design approach for treating and improving cognitive deficits.

Restorative intervention therapies, such as rote repetition, require participants to accurately complete tasks repetitively until they are able to show proficiency, and then to complete the same tasks after a longer time period (Raskin & Sohlberg, 2009). Consistent repetition of such interventions is said to be the important factor with regards to PM

improvement, based on the assumption that repetitive activation can lead to changes in cortical organization (Mateer & Raskin, 1999). PM training is a form of cognitive rehabilitation that requires retrospective recall of assigned tasks by participants while completing distractor tasks.

Attention process training (APT) is a cognitive rehabilitative program that focuses on the training and improvement of various attentional domains that may be impacted by ABI (Sohlberg, McLaughlin, Pavese, Heidrich & Posner, 2000). Persons with ABI may suffer from varying degrees of attentional difficulties and APT provides a program that can be tailored to the deficits expressed by a give individual. The APT has a series of structured hierarchical exercises targeted to each component of attention (sustained, selective, divided, alternating) as well as modules pertaining to working memory and suppression.

Effectiveness of CR. The use of APT in ABI populations has been shown to improve performance on neuropsychological assessments of attention as well as self-reported participant questionnaires focused on daily attention improvement (Palmese & Raskin, 2000; Sohlberg et al., 2000). Improvement has been observed on neuropsychological assessments corresponding to the specific attentional domain(s) targeted with APT (Sturm, Willmes, Orgass & Hartje, 1997). Additionally, rote repetition PM training has proven to effectively treat and improve PM deficits as indicated on clinical assessments (Raskin & Sohlberg, 2009).

Furthermore, restorative cognitive rehabilitation therapies lead not only to clinical and behavioral improvements, but have also been shown to cause neurophysiological changes and improvements. Studies utilizing CR in the form of computerized attention training in multiple sclerosis (MS) and Parkinson's populations show that CR not only improves an individual's performance on written measures of attention, but also that neurophysiological measures indicate significant increases in neuronal activation during attention tasks following CR (Cerasa et al.,

2013). Variations in functional magnetic resonance imaging (fMRI) activity following cognitive training has been shown to directly correlate with improved performance on corresponding clinical measures (Erickson, Colcombe, Wadhwa et al., 2007) and in brain regions associated with the cognitive domain being targeted by that therapy (Wykes, Brammer, Mellers et al., 2002).

Methods

Participants

Potential participants (n=30) were contacted from a database of ABI individuals who had previously expressed interest in receiving cognitive rehabilitation, previous participants from studies within the cognitive neuropsychology laboratory at Trinity College, and via patient referrals received by Dr. Sarah Raskin. Additionally, advertisements were posted around the community and distributed to coordinators of brain injury support groups, and local neuropsychologists and neurologists. From the 30 contacted individuals, 12 were unresponsive to multiple inquiries, and 18 underwent phone screenings. Following phone screenings, 5 participants dropped out, 2 were deemed ineligible, and 11 scheduled and completed pre-testing appointments. From the individuals that were pre-tested, 2 were deemed ineligible and 1 dropped out prior to study enrollment. A total of 8 participants were enrolled in the study with 1 discontinuing participation after 4 sessions.

Inclusion criteria for this study were:

1. diagnosis of ABI at least 1 year prior to participation;
2. ability to speak, read, and understand English;
3. obtainable medical records;
4. commitment to attend 10 scheduled appointments over period of 6 weeks.

Exclusion criteria were:

1. current neurological or psychiatric disorders including but not limited to schizophrenia, manic depression, bipolar disorder, dementia, seizure in the last year, etc.;
2. previous cognitive rehabilitation;
3. significant visual or auditory impairment which would interfere with proper participation;
4. current alcohol or recreational drug use;
5. prospective memory ≥ 15 minutes as indicated by the MIST.

Demographic and injury information for the participants that completed the study (N=7) are shown in table 1.

Table 1. *Participant demographic & ABI[2] details*

Participant	Age[1]	Education[1]	Injury Duration[1]	Etiology of Injury
P1	50	16	4	**Korsakoff's encephalopathy** Chronic alcoholic (30 yrs.)
P2	45	14	3	**TBI**[2]: motorcycle accident GSC[3] = 3 R[4] hematoma with midline shift 10 day coma
P3	35	12	6	**TBI**[2]**:** motor vehicle accident 3 month coma
P4	34	14	31	**Multiple ABI**[2] Cerebral cavernoma (age 3) Stroke (age 10) Notable L[4] thalamic damage
P5	56	18	4	**Stroke** NHISS[5] = 15 Hyper dense R[4] MCA[6] Total occlusion of R[4] ICA[6]
P6	58	18	7	**TBI**[2]**:** assault GCS[3] = 14 Subarachnoid hemorrhages Notable L[4] parietal subdural hematoma
P7	51	11	~ 45[7]	**TBI**[2]**:** multiple assaults as infant Struck in back of head with bat Fall down staircase
Means	47	14.7	14.3	
SD	9.52	2.75	16.75	

[1]In years [2]ABI=acquired brain injury, TBI=traumatic brain injury [3]GCS=Glasgow Coma Scale (Taesdale & Jennett, 1974)
[4]R=right sided, L=left sided [5]NHISS=National Institute of Health Stroke Scale (Brott, Adams, Olinger et al., 1989)
[6]MCA=middle cerebral artery, ICA=internal carotid artery [7]Estimate because P7 unaware of exact age at onset

Materials

Measure of Prospective Memory. The primary outcome measure is the Memory for Intentions Screening Test (MIST, Raskin, 2004). The MIST is comprised of both time (e.g., "In 12 minutes, tell me it is time to take a break") and event- (e.g., "When I hand you a postcard, self-address it") based items over a period of 30 minutes. Responses included both verbal and action responses. The cues used in the MIST are purposefully related to the natural response one should have to a given cue. There are both short (2 min) and long-term (15 min) prospective memory tasks. To measure more naturalistic prospective memory, participants were told to contact the test administrator via voicemail in 24 hours and report the number of hours they slept the night before. The MIST also looked at retrospective memory recognition by asking participants a series of multiple-choice questions at the end of testing, regarding the tasks they had just been directed to complete. Errors or task omissions were coded as: prospective memory failure, task substitution, loss of content, loss of time or random error. Both retrospective recognition and PM measures were scored using age and level of education corrections.

Neuropsychological Cognitive Assessments. The neuropsychological battery included measures of attention, memory, and executive functioning. Attention was assessed using the Brief Test of Attention (BTA, Schretlen, 1989), memory was assessed using the Hopkins Verbal Learning Test-Revised (HVLT-R, Benedict, Schretien & Groninger, 1998) and the Brief Visuospatial Memory Test-Revised (BVMT-R, Benedict, 1997), and executive functioning was assessed with the Stroop Neuropsychological Screening Test (Stroop Color-Word, Trenerry, Crosson, DeBoe & Leber, 1989) and forms A and B from the Trail Making Test (Tombough, 2004).

Measures of Generalization. Questionnaires used as measure of generalization to daily life were administered during both pre and post-testing (n=6). These included the Prospective Memory Questionnaire (PMQ) which consists of 52 ranking scale questions with subscales pertaining to long-term episodic memory, short-term habitual memory, internally cued memory and the use of memory aid techniques, scored from 0 to 8 (Hannon, Adams, Harrington, Fries-Dias & Gibson, 1995). The Everyday Memory Questionnaire (EMQ) consists of 31 likert scale questions, scored from 0 (never) to 4 (several times a day), and served as self-report of everyday difficulties attributed to memory deficits (Sunderland, Harries & Baddeley, 1983). The APT-II Attention Questionnaire (APT-II) contains 12 likert scale questions and a free response attention problem list, and scoring tools provide a measure of level of attention disruption, ranging from mild to profound, scored from 0 (little-mild disruption) to 48 (profound disruption), experienced by each participant (Sohlberg, Johnson, Paule, Raskin & Mateer, 1993). From the BADS, the Dysexecutive Questionnaire (Dex) consists of 20 likert scale questions, scored from 0 (never) to 4 (very often), pertaining to executive dysfunction (Wilson, Alderman, Burges, Emslie & Evans, 1996). In addition to self-reports of cognitive functioning, the shortened version of the World Health Organization Quality of Life Survey (WHOQOL-BREF) assessed overall quality of life across 4 domains: physical health, psychological health, social relationships and personal environment, with each section score transformed into scaled scores ranging from 0 to 100 (World Health Organization, 1996). In the case that a participant was unable to live independently as a result of their ABI (n=3), their healthcare proxy or a person whom they lived with on a daily basis was asked to complete these same questionnaires as confirmation.

Cognitive Rehabilitation. Participant profiles were created using normative data for each baseline neuropsychological measure (table 2). These profiles were used to create

individualized cognitive rehabilitation protocols for active treatment participants (n=4). Below average attention score (n=2) indicated primary use of Attention Process Training (APT-III, Sohlberg & Mateer, 2010), whereas participants (n=2) with average attention abilities but below average performance on memory measures were administered rote repetition prospective memory training (PMT, Raskin & Sohlberg, 2009).

Active Control Program. Participants assigned to active control group (n=3) were administered brain education. The education program provided a basic overview of the following topics: history of neuroscience, neuroanatomy, neurophysiology, neurochemistry, sensory systems, attention, memory and learning, executive functioning, types of ABI and resources for ABI. Education modules were in the form of PowerPoint slides, videos and interactive computer programs. Additionally participants were given quizzes throughout each session to ensure that their focus was maintained throughout the programming.

Procedure

Interested participants (n=18) received a telephone screen to determine if they were eligible to participate and answer any questions they had regarding participation. Once screened, participants (n=11) underwent pre-testing. In addition to collecting background information and informed consent, the MIST, neuropsychological assessment battery and measures of generalized were administered during pre-testing. Once pre-testing was completed, eligible participants (n=8) were randomly assigned to either active control or active treatment groups. Pre-testing neuropsychological assessment scores were used to create individualized cognitive rehabilitation programs for active control (n=4) participants (table 2). Participants (N=7) completed 10 1-hour sessions of either active control (n=3) or active treatment (n=4) programs in a one-on-one setting over a period of 6 weeks. At the completion of the 10 sessions, participants underwent post-

testing, which consisted of re administration of the MIST, neuropsychological assessments, and measures of generalization. Individuals that were blinded to participant pre-testing scores and assigned study group administered post-testing. Alternate forms of the MIST and neuropsychological assessments were used when available. Compensation was provided to participants after completing the first programming session ($10) and at post-testing ($50). Additionally, participants assigned to the active control group were provided a debriefing form at the completion of their post-testing, and offered to opportunity to receive 10 sessions of cognitive rehabilitation.

Prospective Memory Training (PMT). Active treatment participants (n=2) were assigned to rote repetition PM training as a result of their performance on pre-testing measures (table 2). Participants began PM training with 2-minute delay between task administration and task execution time, and a baseline activity packet was provided as a distractor task, which was later used to determine the appropriate difficulty and type(s) of distractors for each participant. Distractor tasks included word searches, crossword puzzles, word riddles, and Sudoku puzzles. After simultaneously completing 5 PM tasks accurately, the waiting interval was increased by 1 minute. Participants received 1 point for completing the correct task, and 1 point for the correct time. Accurate task completion was dependent on the participant completing the correct task at the correct time. The PM training log increased task difficulty as time delay interval increased, and distractor task difficulty was altered at time delay checkpoints throughout the duration of each participant's rehabilitation. Further details regarding PM training methodology and individualized participant protocols is provided in Appendix A.

Attention Process Training (APT-III). Active treatment participants (n=2) were given APT-III as a result of their performance on pre-testing measures (table 2). A computerized

version of APT training (APT-III, Sohlberg & Mateer, 2010) was used to create individualized rehabilitation programs. The computerized APT program generates performance scores for each task as well as collects self-reports that illustrate the degree of difficulty and effort put forth by the participant during each task. Means of increasing difficulty varied across tasks, however difficulty was not increased until successful consistency was observed. Success was defined as achieving a score greater than 80% and consistency was defined as achieving success in two consecutive sessions. Further explanation of individualized APT protocols and program examples are provided in Appendix A.

Data Collection & Analysis

IBM SPSS and Microsoft Excel were used to enter and analyze all participant data. In order to create individual participant neuropsychological profiles, normative data from neuropsychological assessments was used to convert pre-testing scores into percentiles, and to calculate intra-participant reliable change index scores (RCI, Jacobson & Truax, 1991) for clinical significance. The RCI for the study population was calculated to analyze significant change across groups and for each participant in the current study to determine effectiveness of cognitive rehabilitation.

Results

Design of Individual CR Protocols

Pre-testing neuropsychological assessments and MIST scores were used to create participant profiles which highlighted the level of impairment faced by each participant for each of the cognitive domains (attention, memory and executive function). Active treatment participants with impaired performance on pre-testing assessment of attention (BTA), were assigned APT-III (n=2), whereas active treatment participants with less impaired attention and majority of impairment on memory measures (HVLT-R & BVMT-R) were treated with PMT (table 2).

Significant Improvement Occurred Independent of Study Group

Cross-over analysis of clinically significant change ($p<0.05$) and reliable change index (RCI) calculated from the study population showed that individual participants showed significant improvement ($p<0.05$) on various neuropsychological assessments, and MIST variables, independent of participant study group (Table 3). Clinically significant improvement on the neuropsychological assessment of attention was only seen in participants that received APT-III (Table 3, Figure 2). Proportionally, the active treatment group improved on more measures of executive function and attention in comparison to the active control group, whereas the opposite was seen with regards to improvement on memory assessments (Table 3, Figure 1).

There was no significant difference between the average change on MIST variables between groups, however the active treatment group showed proportionally greater clinically significant improvement on MIST variables (Figures 3, 4).

Table 2. *Neuropsychological assessment profiles (percentiles) for eligible. Degree of impairment (below average, borderline impaired, impaired) derived form psychometric conversion table. Based on pre-testing performance active treatment participants were assigned either (a) PMT or (b) APT-III. Control participants (c) shown to illustrate non-bias in baseline testing scores across groups.*

Assessment	P1[a]	P2[a]	P3[b]	P4[b]	P5[c]	P6[c]	P7[c]	
Memory for Intentions Screening Test								*Prospective Memory*
Time	<1	35	6	17	81	29	63	
Event	<1	10	13	10	36	6	<1	
Prospective Memory Total	<1	10	5	10	46	7	10	
Retrospective Memory Total	25	38	7	>99	42	18	29	
Brief Visuopsatial Memory Test								*Memory*
Immediate Recall	NA[1]	58	<1	<1	12	50	<1	
Delayed Recall	NA	62	<1	1	18	73	<1	
Hopkins Verbal Learning Test								
Immediate Recall	<1	<1	1	1	1	27	<1	
Delayed Recall	<1	1	<1	3	2	34	<1	
Recognition Discrimination Index	<1	19	<1	6	30	53	<1	
Brief Test of Attention	50	8	<1	2	76	53	<1	*Attention*
Stroop Color-Word	78	8	<1	<1	22	50	<1	*Executive Function*
Controlled Oral Word Association Test[2]	20	NA	NA	NA	NA	NA	NA	
Trail Making Test								
Form A	NA	15	<1	<1	47	79	<1	
Form B	NA	2	<1	<1	67	73	<1	

[1] NA=Not available

[2] P1 is from pilot study that used the COWAT (Benton, Hamsher & Sivan, 1994) as an additional measure of executive function

Table 3. *Crossover of study significant & clinically significant improvement on assessments for all participants. ✔ indicates clinically significant (p<0.05) change between pre and post-testing and * indicates significant change (p<0.05) in comparison to study population. See Appendix B for detailed explanations.*

	Active Treatment				Active Control			
Assessment	P1[1]	P2[1]	P3[2]	P4[2]	P5	P6	P7	
Memory for Intentions Screening Test								*Prospective Memory*
2 minute	✔*		✔*					
15 minute	✔*	✔*	*	NC	*	NC		
Time			✔*	NC				
Event	✔*	✔*	*	*	✔*	✔*	*	
Verbal			NC	NC		NC		
Action	✔*	*	✔*		✔*			
Prospective Memory Total	✔*	NC	*		NC		NC	
Retrospective Memory Total	NC[3]	✔*	✔*		NC	NC	*	
Brief Visuospatial Memory Test								*Memory*
Trial 1	NA[4]				*			
Trial 2	NA	*				✔*		
Trial 3	NA		*	✔*	NC	✔*	✔*	
Immediate Recall	NA	*	✔*	*	*	✔*	*	
Delayed Recall	NA	✔*	NC		NC		✔*	
Hopkins Verbal Learning Test								
Immediate Recall	*		NC		NC	*	*	
Delayed Recall		NC	✔*	✔*	✔*		NC	
Recognition Discrimination Index		NC			NC	NC	✔*	
Brief Test of Attention			✔*	✔*				*Attention*
Stroop Color-Word		✔*	✔*	✔*	✔*		✔*	*Executive Function*
Trial Making Test								
Form A	NA		NA	✔*			✔*	
Form B	NA	✔	NA	✔*			✔	

[1] Prospective Memory Training Group [2] Attention Process Training Group [3] NC = No change [4] NA = Scores not available

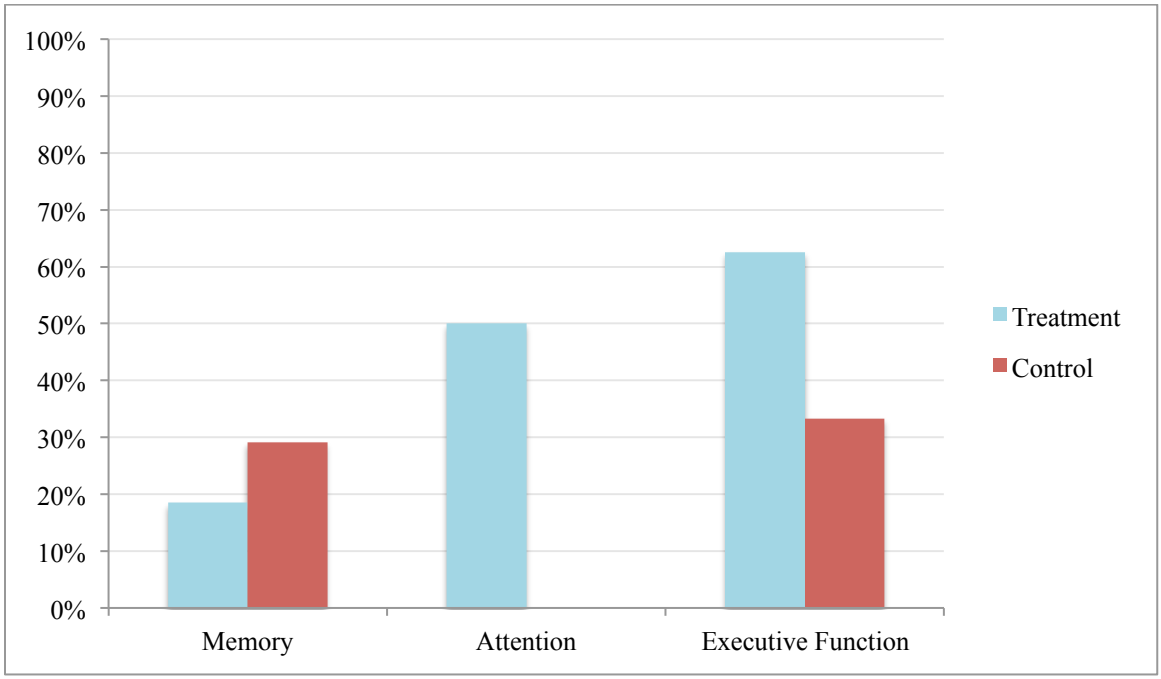


Figure 1. *Ratio of significant improvement variables between treatment and control groups for each cognitive domain.*

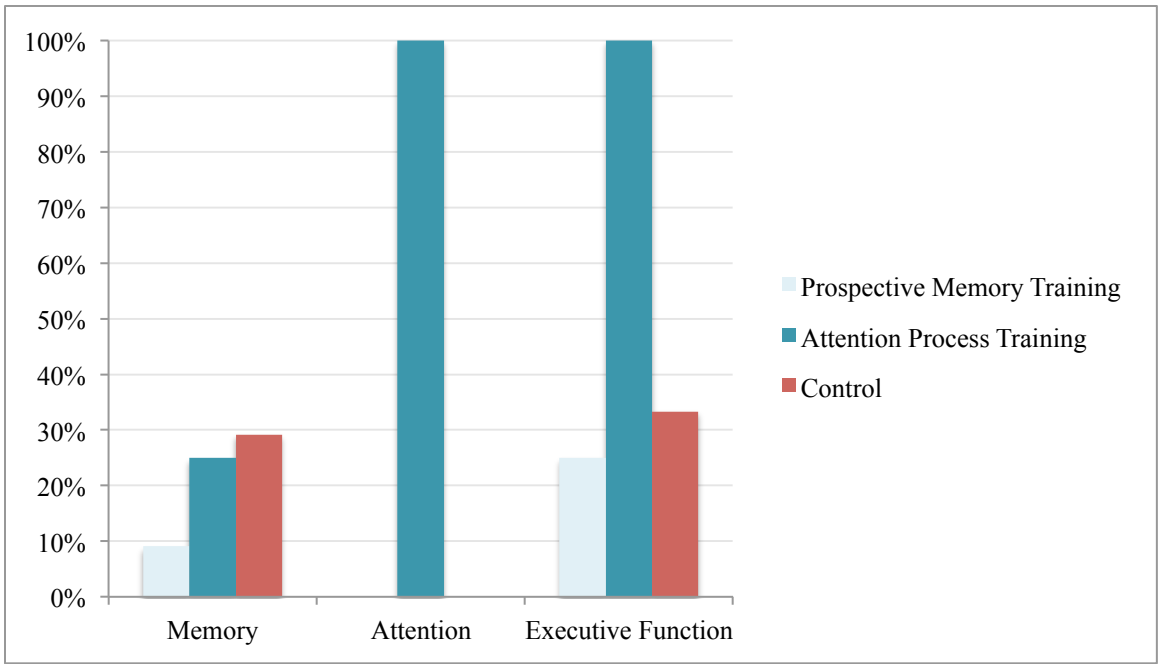


Figure 2. *Ratio of significant improvement vairbales comparing 2 treatment group subcategoires and control group.*

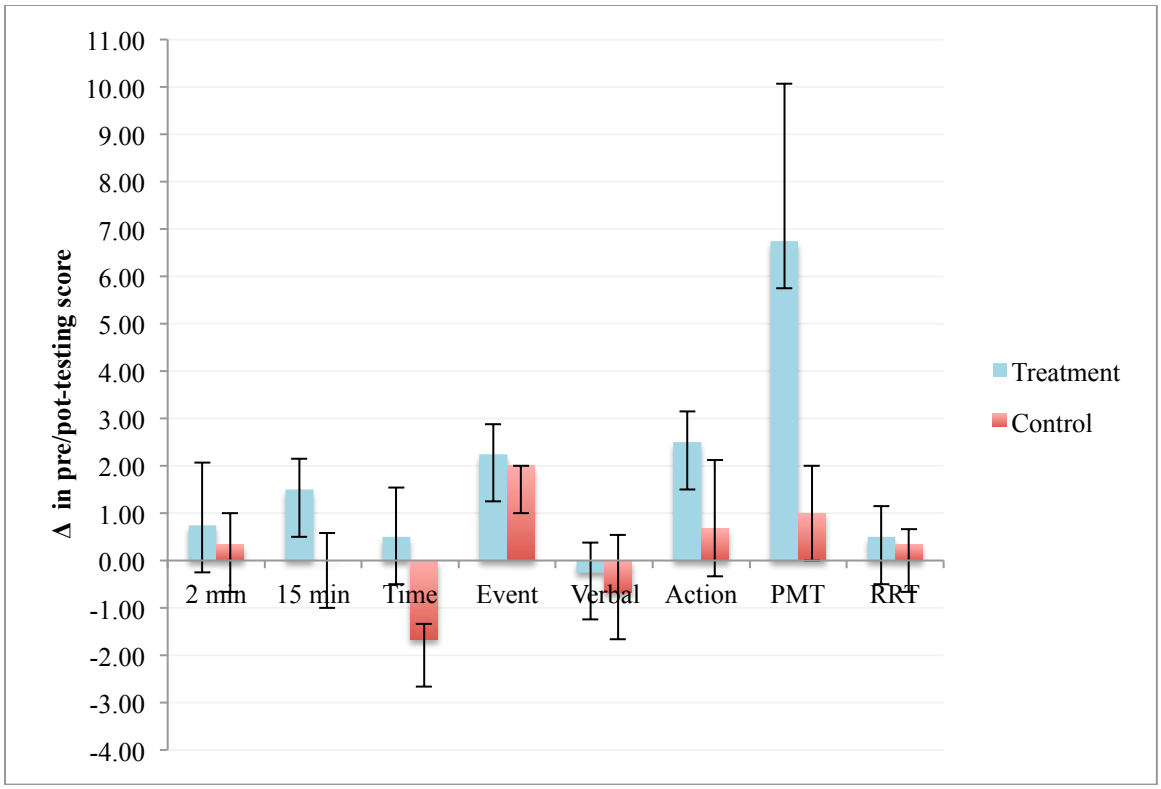


Figure 3. *Average change on MIST varibles between pre and post-testing for each study group.*

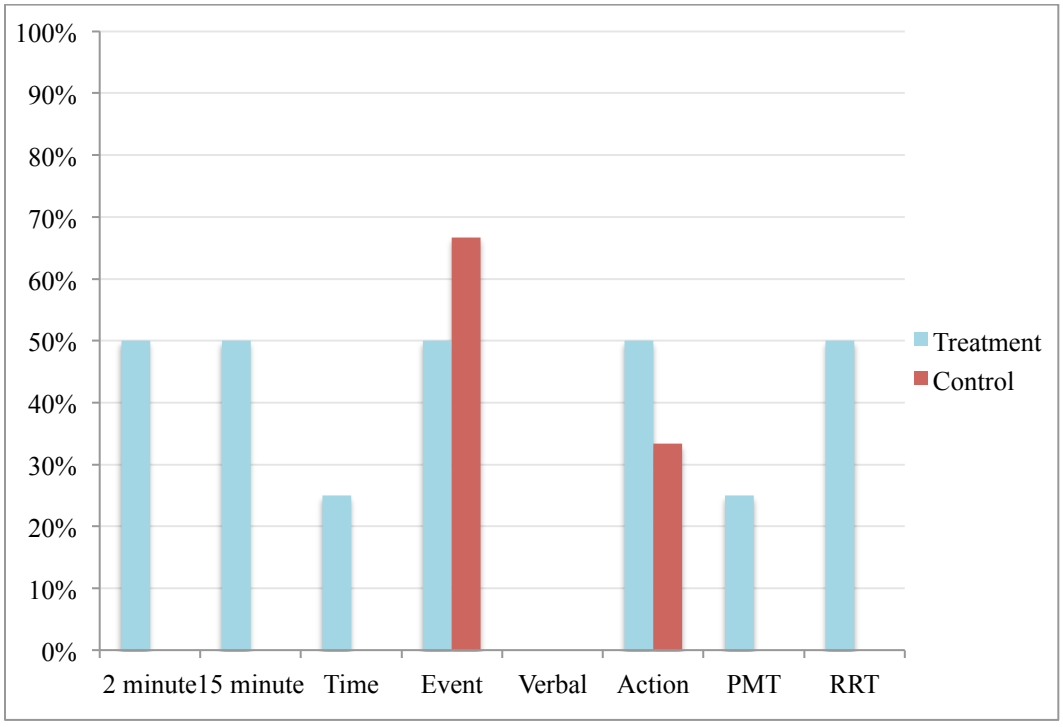


Figure 4. *Ratio of signifincat improvement on Memory for Intentions Screening Test variables as measures of prospective memroy between groups.*

Measures of Generalization

RCI for the study population indicated active treatment and active control participants indicated significant improvement ($p<0.05$) on generalized measures pertaining to quality of life, everyday memory, and prospective memory, whereas significant improvement pertaining to everyday memory and executive function was only observed in the active treatment group (Table 4). Proportionally, significant improvement on generalizing variables (attention, executive function, everyday memory, quality of life) was greater following active treatment (Figure 5).

Compared to the active control group, the active treatment group showed proportionally great improvement on everyday PM measure variables pertaining to long-term episodic memory, short-term habitual memory, and use of memory techniques, whereas no difference was observed on PM variables pertaining to internally cued memory and overall PM (Figure 6).

Table 4. *Significant improvement on measures of generalization for participants (n==6) compared to study population (*p<0.05). See Appendix B.*

	Active Treatment			Active Control		
	P2	**P3**	**P4**	**P5**	**P6**	**P7**
Quality of Life[1]						
Physical Health	*	*	*	NC	NS	NS
Psychological Health	NC[6]	NC	*	*	NC	NC
Social Relationships	NC	NS	NC	NC	NC	NS
Personal Environment	*	*	NC	*	NC	NC
Attention[2]	*	NS	NS	NS	NS	NS
Everyday Memory[3]	*	*	*	NS	NS	*
Executive Function[4]	*	NS	*	NS	NS	NS
Prospective Memory[5]						
Long-term episodic	NS[7]	*	*	NS	*	NS
Short-term habitual	*	*	*	*	NS	NA[8]
Internally cued	*	NS	*	NS	*	*
Memory techniques	NS	NS	*	NS	NS	NS
Total Prospective Memory	*	*	*	*	*	*

Measured by the: [1]QOL-BREF (World Health Organization, 1996), [2]APT-II (Sohlberg, Johnson, Paule, Raskin & Mateer, 1993), [3] EMQ (Sunderland, Harries & Baddeley, 1983), [4] BADS Dex (Wilson, Alderman, Burges, Emslie & Evans, 1996), and [5] PMQ (Hannon, Adams, Harrington, Fries-Dias & Gibson, 1995). [6] NC=No change [7] NS=No significant improvement [8] NA=Pre/Post-testing comparison not available

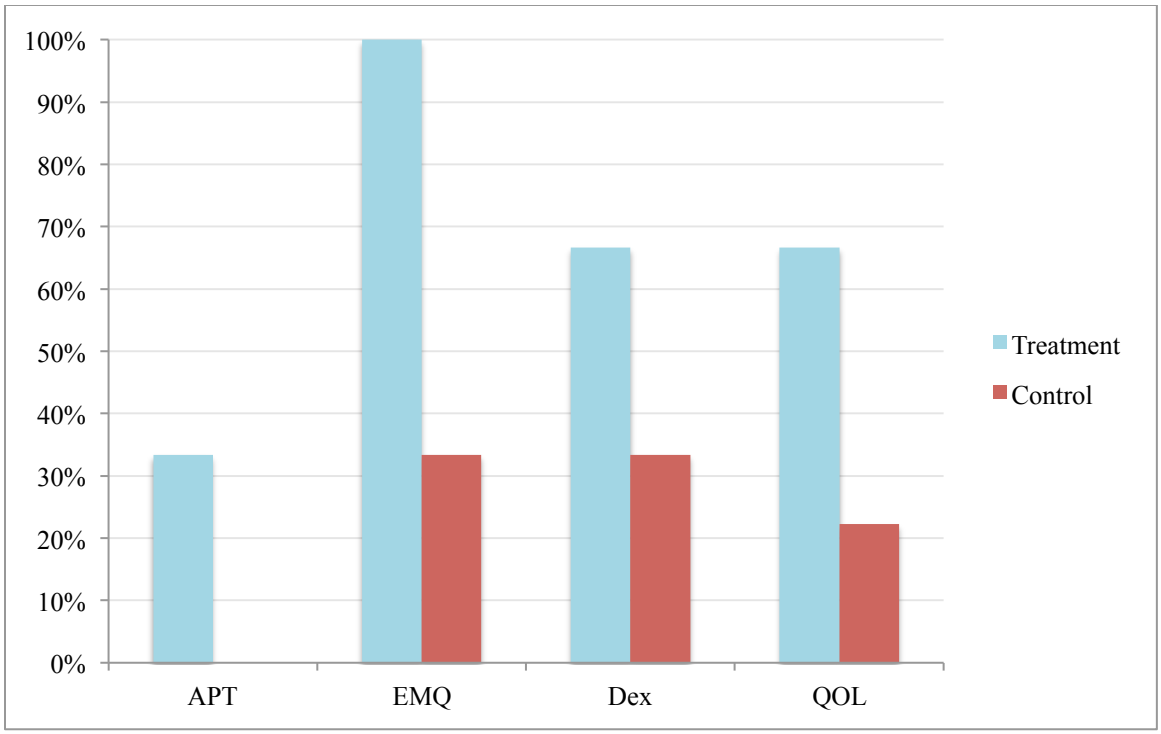


Figure 5. *Ratio of significant improvement on measures of generalization variables for attention, everyday memory, executive function and overall quality of life between groups.*

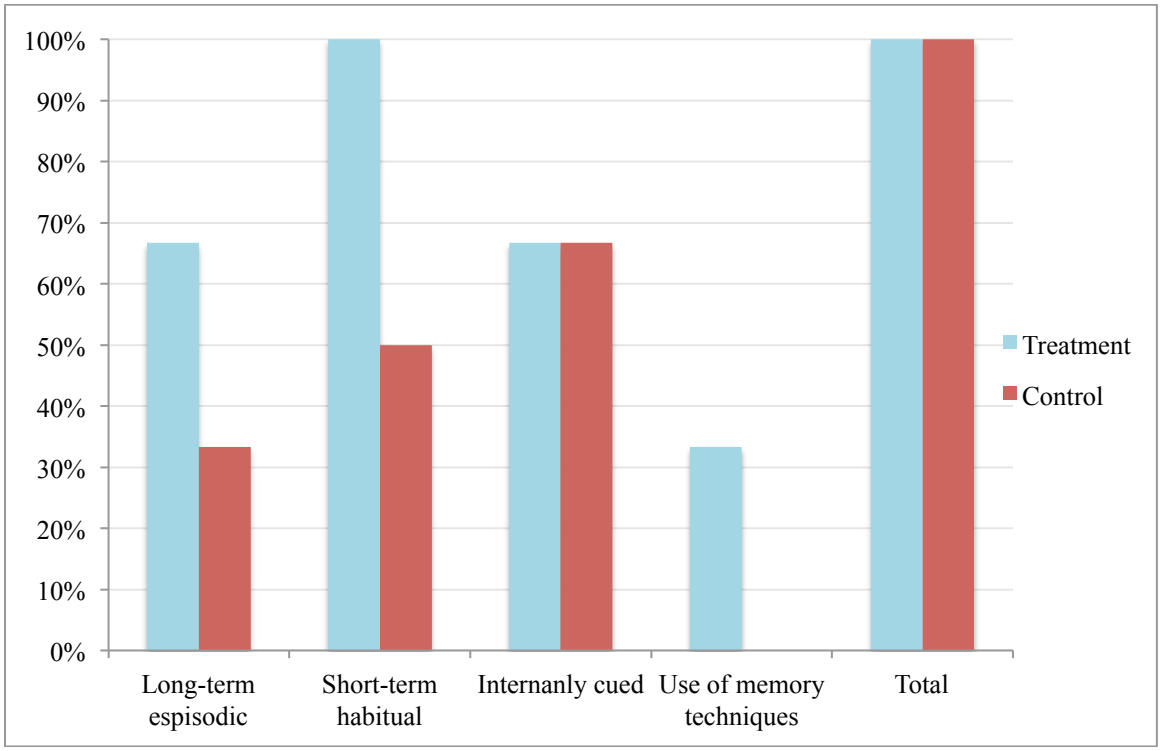


Figure 6. *Ratio of significant improvement on prospective memory variables pertaining to generalized prospective memory in daily life between groups.*

Discussion

Interpretation of Results

Neuropsychological Assessments.

Memory. All participants, independent of study group, showed significant improvement ($p<0.05$) on at least 1 memory variable in comparison to average change in testing scores for study participants (Table 3). Participants (P2-P7) showed clinically significant improvement ($p<0.05$) on at least 1 memory variable from neuropsychological assessments of memory (table 3), independent of study group. Crossover of study significant change and clinically significant change showed that participants (P2-P7) significantly improved on at least 1 variable from pre and post-testing on neuropsychological assessments of memory (Table 3). This suggests that mental simulation, whether CR or learning novel information can cause lead to a significant improvement in memory.

There was no significant difference in improvement on neuropsychological assessments of memory between study groups, however the treatment group showed proportionally less significant improvement compared to the control group on memory assessments (Figure 1). Furthermore, there was no significant difference in the treatment group between the PM training and APT, however the APT group should proportionally more improvement compared to the PM training group (Figure 2).

Attention. Only participants receiving APT showed significant change ($p<0.05$) between pre and post-testing scores pertaining attention in comparison to the study population (Table 3, Figures 1, 2). Furthermore, these changes were also determined to be clinically significant ($p<0.05$) (Table 3). Thus it can be concluded that APT leads to improved attention. A possible explanation for these results could be that significant improvement on the BTA would only be

observed in individuals that scored impaired on pre-testing BTA (Table 2), however this is refuted by control participant, P7, not showing significant improvement on this measure after participation.

Executive Function. Participants (P2, 3,4,5,7) showed clinically significant improvement ($p<0.05$) on at least 1 variable measuring executive functioning, independent of study group (Table 3). Furthermore, these same participants showed improvement that was significant ($p<0.05$) in comparison to the study population (Table 3). Similarly to neuropsychological assessments of memory, this suggests that mental stimulation of any form may have the potential to improve executive functioning.

There was no significant difference in improvement on neuropsychological assessments of executive function between study groups, however the treatment group showed proportionally greater significant improvement in comparison to the control group (Figure 2). Within the treatment group significant difference in improvement on measures of executive function was not found, however the APT group showed proportionally greater improvement in comparison to either the PMT group or control group (Figure 4). Interestingly, clinically significant and study significant improvement was found for all variables of executive function in the APT group, similarly to the results produced for variables of attention.

Measures of Generalization. Significant change ($p<0.05$) was found independent of study group for measures of generalization pertaining to everyday memory, prospective memory, and overall quality of life (Table 4), with proportionally more change occurring as a result of treatment in comparison to control (Figure 5). Only the treatment group showed significant improvement on the measures of generalization pertaining to attention and executive function in daily life, where as the control group did not (Table 4, Figure 5). Proportionally more significant

improvement ($p<0.05$) on measures of generalization for PM resulted from treatment as compared to the control group (Table 4, Figure 6). Participants were aware that the study aimed to improve PM following CR, thus it is possible that these trends on self-reports could be explained by all participants, independent of group, inflating their post-testing responses on measures of generalization pertaining to memory and PM as an attempt to show they had "improved" in a fashion that would be consistent with the study goal.

Prospective Memory. Clinically significant improvement on MIST variables was observed independent of study group (Table 3,), however the treatment group showed proportionally more improvement on MIST variables (Figure 4). Furthermore, there was no significant difference in improvement (positive change) between groups on MIST variables (Figure 3). From this it can be concluded that individualized cognitive rehabilitation was not more effective than generalized mental stimulation in improving PM.

These findings are significant in the sense that they suggest that individuals with ABI have the ability to improve on cognitive measures by learning novel information rather than only resulting from targeted CR therapies.

Study Limitations

The primary limitations of this study were poor attrition and, thus, low sample size. Using the database of previously interested participants and referrals received by Dr. Raskin was effective in drawing participant interest, however many potential participants do not function independently and thus rely on the accountability and availability of aids and family members to schedule and bring them to appointments.

With regards to study participants, the variability of initial degree of cognitive impairments and duration of injury may have also been factors that influenced results, which

should be controlled for in future studies. Additionally, retrospective analysis concluded that the randomization of participants did not allow for study groups to be age and education matched. This was most likely not a confounding factor, and with a larger sample size may have been ameliorated.

Furthermore, the brain injury education programming may have been too complex to be an accurate representation of a control treatment. In order to keep participants blinded to their group assignment, the topic of brain injury education was selected.

Future Directions

Prior to continuation of this study, it is recommended that modifications be made to the exclusionary criteria to prevent as much variability across participants. Increased sample size was increased, subgroups pertaining to age, level of education and baseline level of impairment may be just as effective in giving a reliable comparison between treatments. Additionally, a more simplistic control paradigm should be created to mirror mental stimulation that an individual would encounter in everyday life. If the control paradigm remained unaltered, a larger sample size could provide significant evidence with regards to the affinity of newly learning information to impact neural plasticity in ABI. Furthermore, it would be interesting to examine treatment efficacy as compared to controls 6 months and 1 year after program completion. This information would be useful to draw conclusions regarding the long-term effects of individualized rehabilitation.

Conclusion

Results from this study indicate that individualized CR is an effective approach for improving cognitive impairments in ABI. Interestingly, this improvement was not seen to be significantly greater in comparison to the control paradigm. With regards to properly designing treatment plans for individuals with ABI, our results suggest that mental stimulation pertaining to novel information may in fact activate and alter neural pathways, thus resulting in improved cognition. In order to affirm these conclusions, it is suggested that this study be continued with an increased population. Additionally retrospective follow-up assessments may provide additionally insight with regards to which protocol (treatment vs. control) resulted in long term change, thus proving to be more effective in improving cognition in ABI.

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Appendix A

Treatment Group Cognitive Rehabilitation Progressions

Prospective Memory Treating

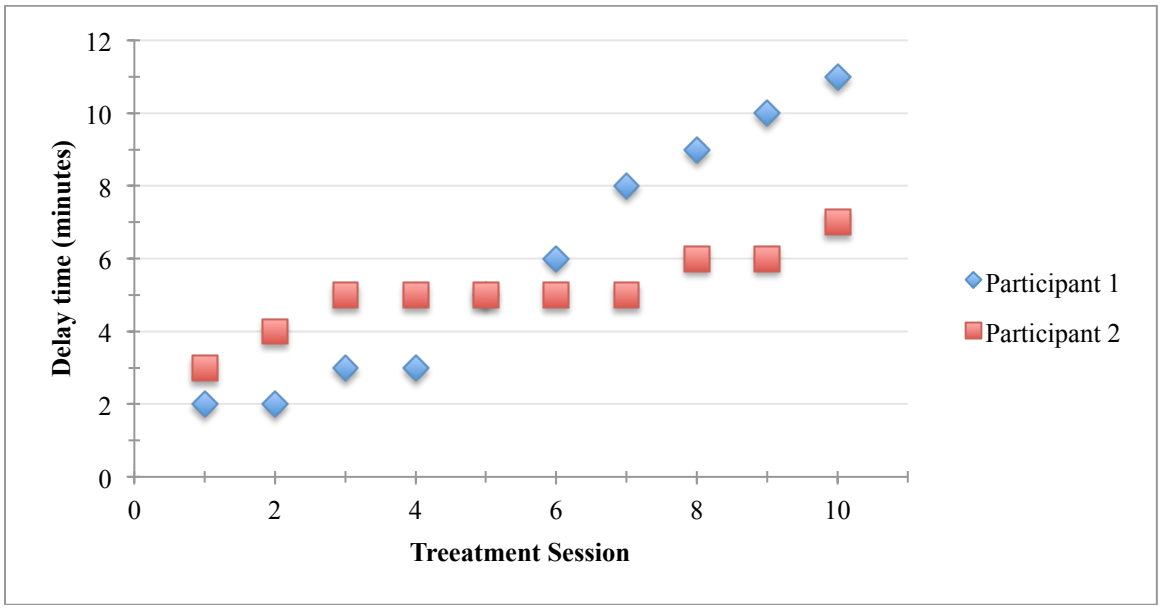


Figure A1. *Rote repetition PM training progression for participants 1 & 2.*

Attention Process Training Accuracy Progressions

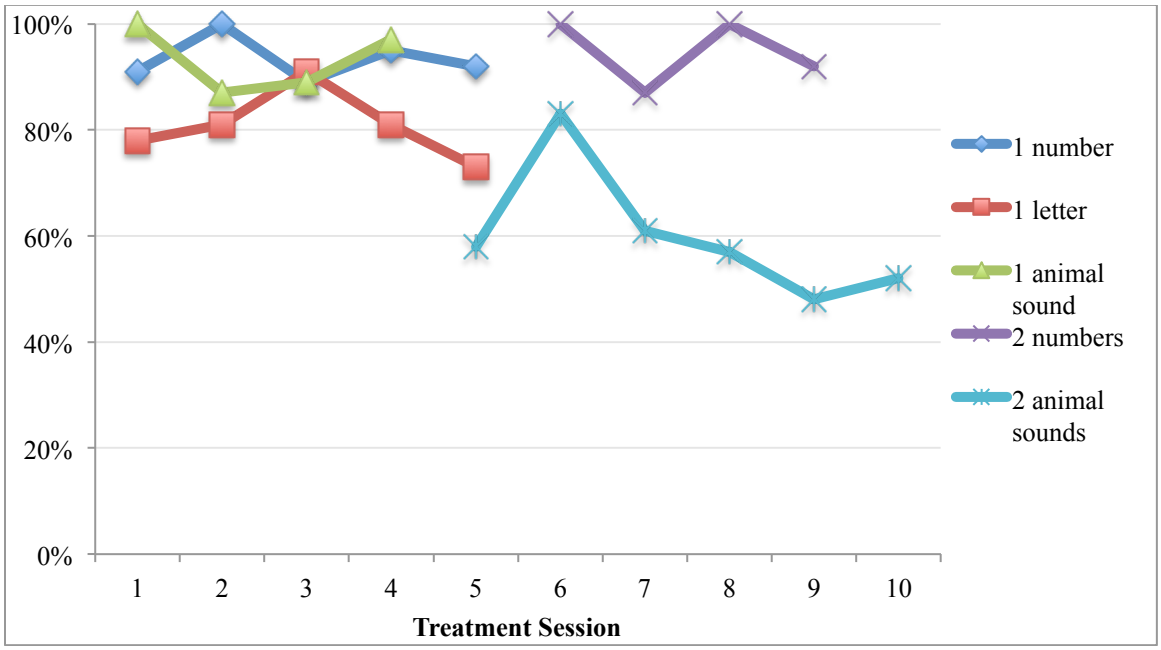


Figure A2. *Participant 3 progression on selective attention tasks.*

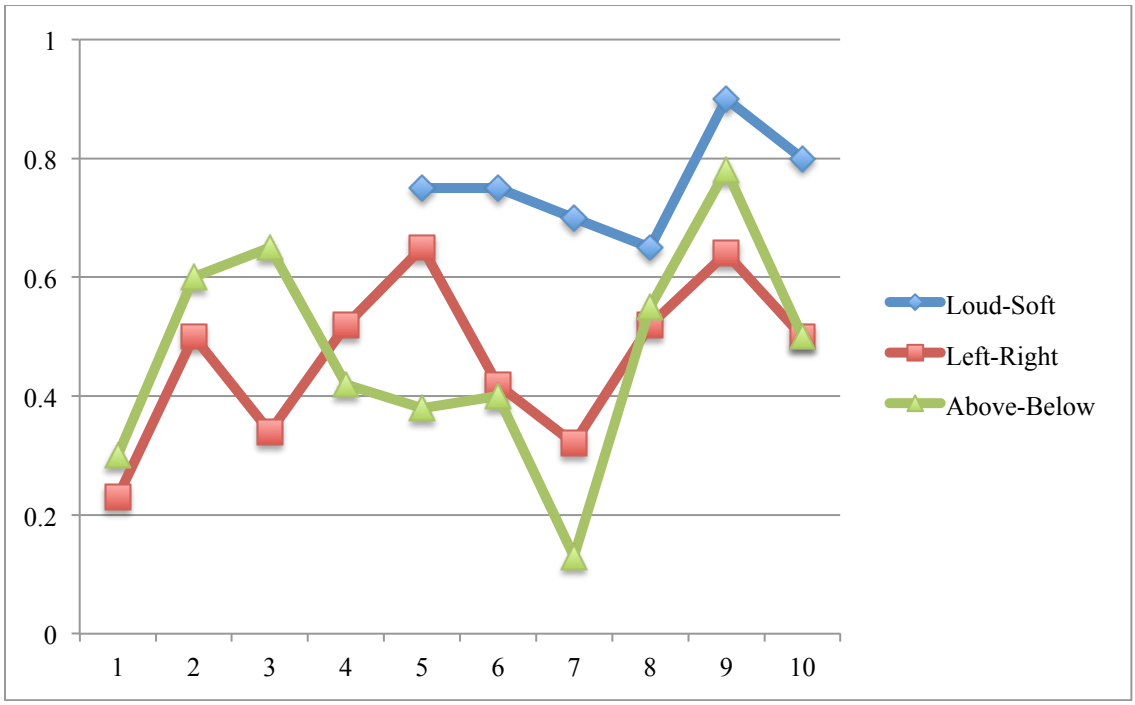


Figure A3. *Participant 3 progression on suppression tasks.*

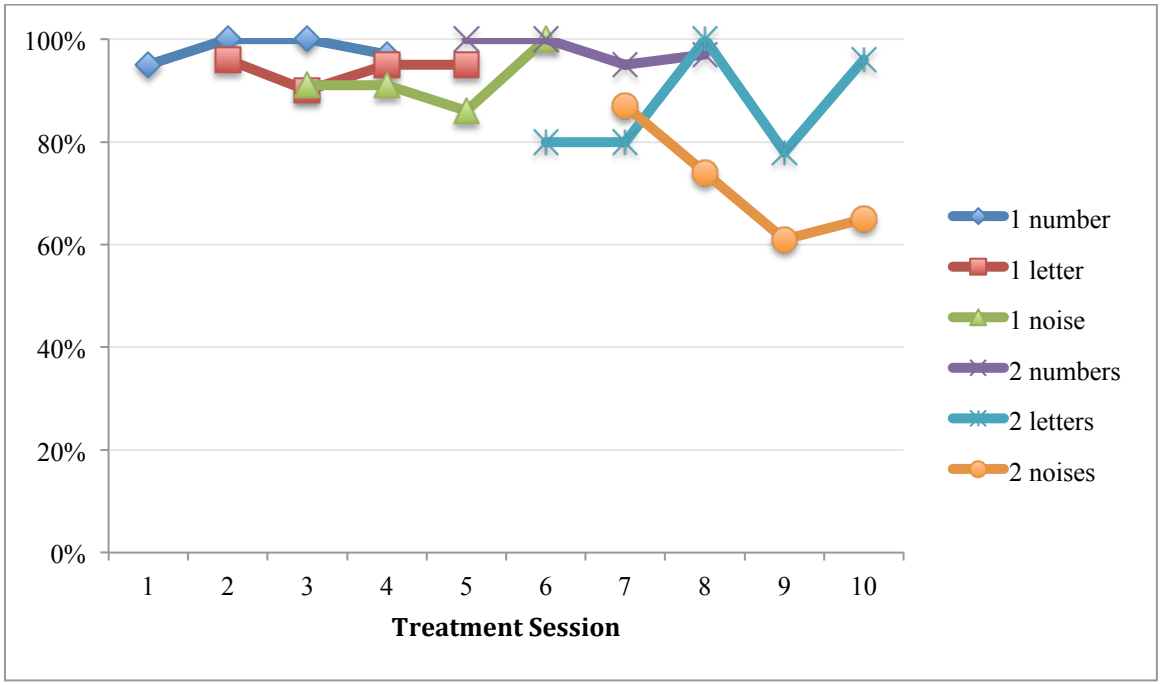


Figure A4. *Participant 3 progression on sustained attention tasks.*

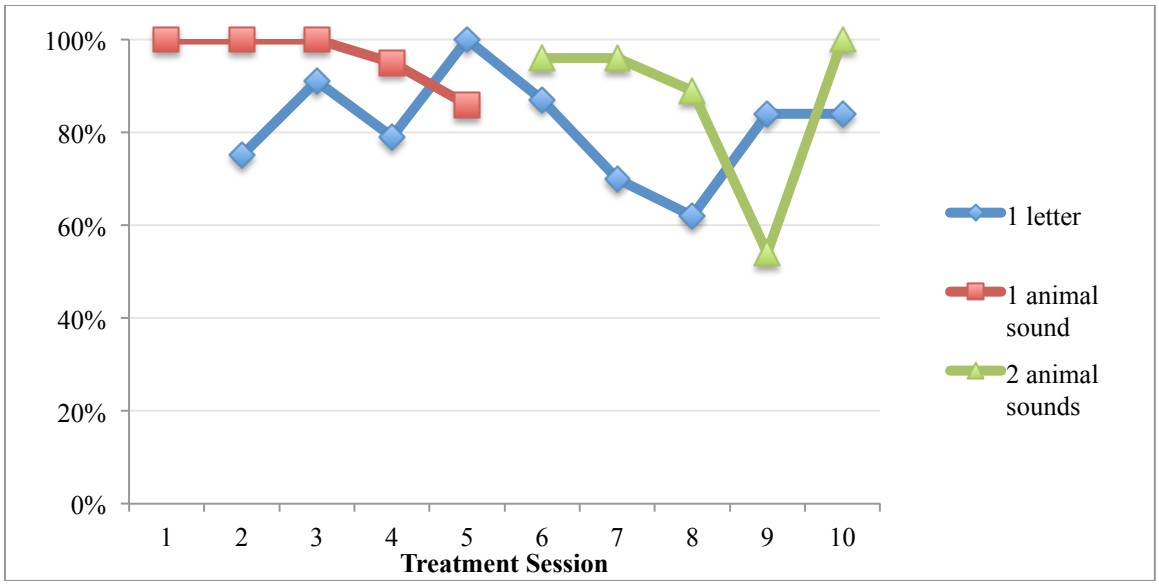


Figure A5. *Participant 4 progression on selective attention tasks.*

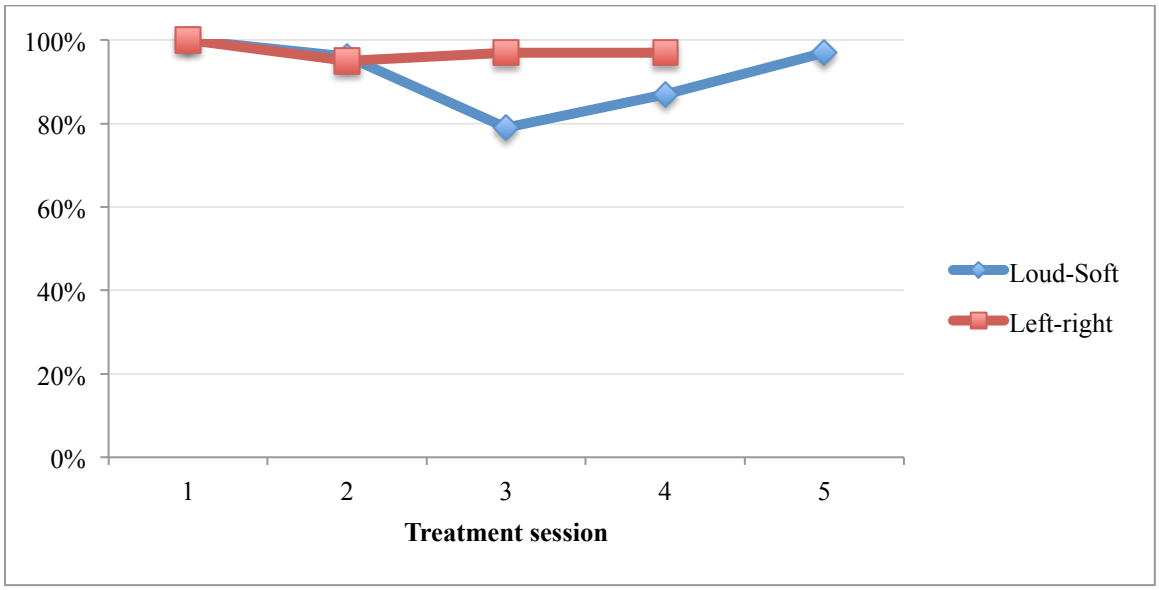


Figure A6. *Participant 4 progression on suppression tasks.*

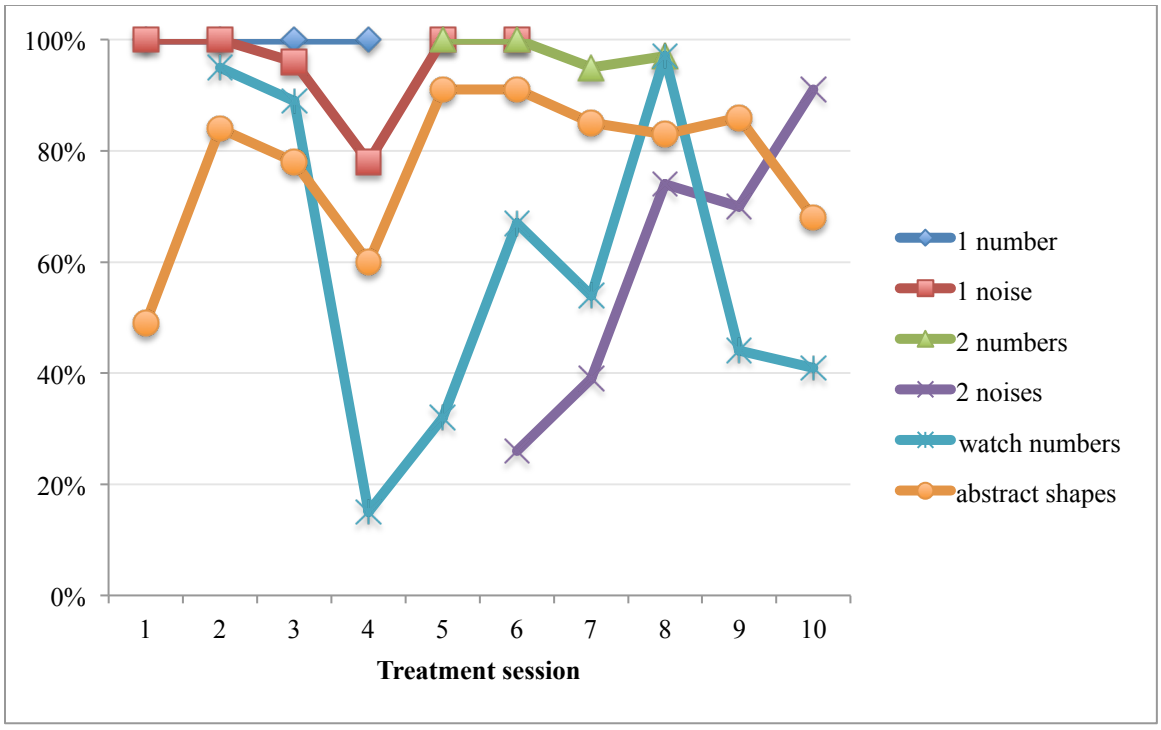


Figure A7. *Participant 4 progression on sustained attention tasks.*

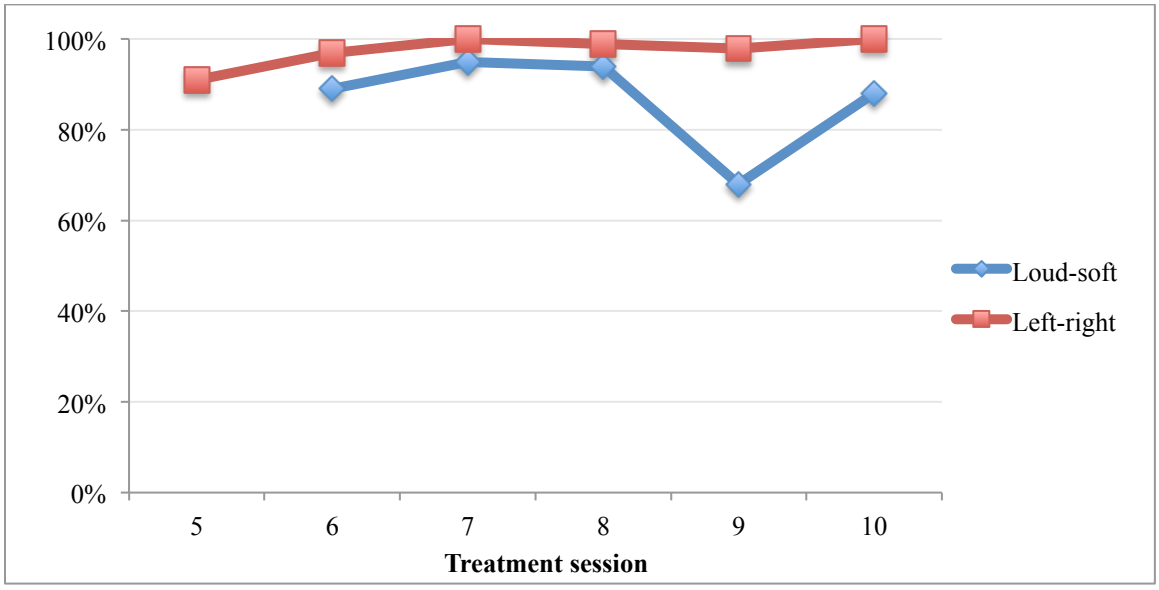


Figure A8. *Participant 4 progression on alternating attention tasks.*

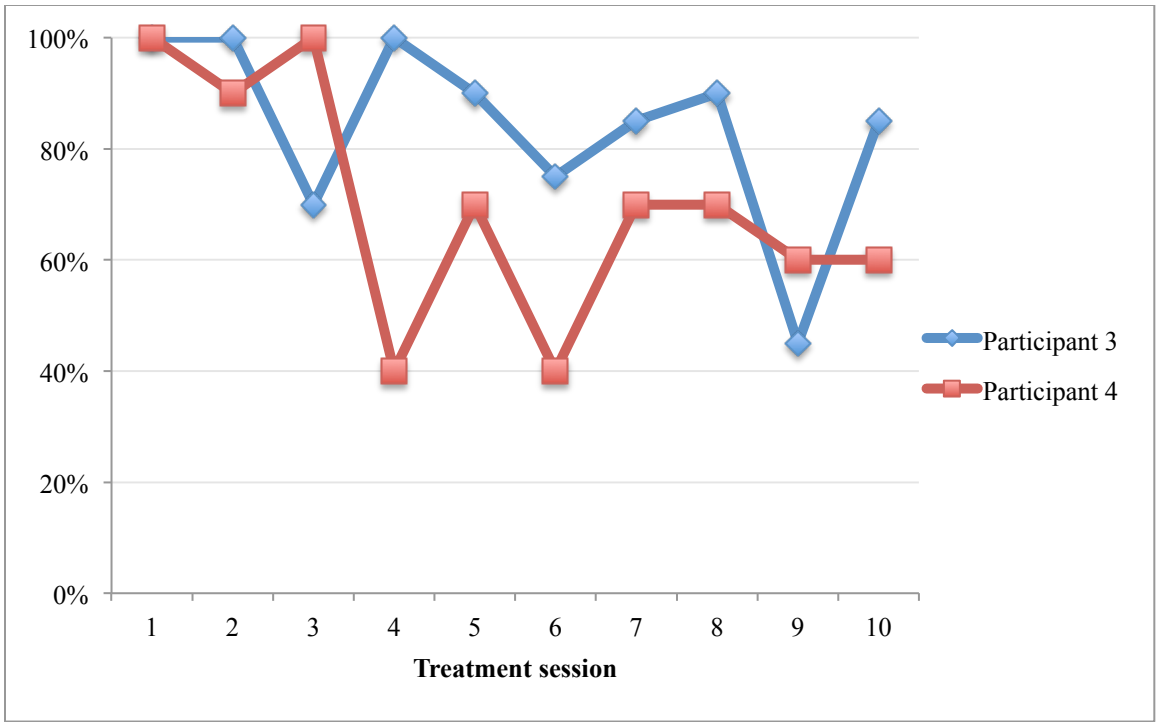


Figure A9. *Progression on ascending number sequencing working memory task for Participants 3 & 4.*

Appendix B

Result Calculations

Clinically Significant Change Calculations

Table B1. *Active Treatment Group Clinically Significant Improvement Scores (*p<0.05).*

			P1			P2			P3			P4		
			Pre[1]	Post[1]	RCI[2]	Pre	Post	RCI	Pre	Post	RCI	Pre	Post	RCI
Memory	**BVMT-R[3]**	Trial 1	NA[9]	NA	NA	7	8	0.55	3	1	-1.09	2	3	0.55
		Trial 2	NA	NA	NA	8	10	1.26	2	3	0.63	6	7	0.63
		Trial 3	NA	NA	NA	11	12	1.14	1	5	4.55*	6	9	3.41*
		Total Recall	NA	NA	NA	26	28	0.62	6	9	0.93	14	19	1.56
		Delayed Recall	NA	NA	NA	10	12	2.00*	2	2	NC	6	5	-1.00
	HVLT-R[4]	Total Recall	18	21	1.05	16	17	0.33	17	17	NC	20	17	-0.94
		Delayed Recall	1	0	-0.70	5	5	NC[10]	0	3	1.79*	3	6	1.79*
		RDI	6	-1	-3.97	10	10	NC	6	4	-1.44	7	9	1.44
Attention	**Brief Test of Attention[5]**		16	4	-5.74	12	14	1.01	4	9	2.53*	10	18	4.04*
Executive Function	**Stroop Color-Word[6]**		111	112	0.14	87	112	5.44*	29	43	4.83*	35	48	5.21*
	Trail Making[7]	Form A	NA	NA	NA	39	38	-0.10	NA	NA	NA	140	104	-5.99*
		Form B	NA	NA	NA	91	66	-3.50*	NA	NA	NA	467	253	-29.13*
	Controlled Oral Word Association[8]		36	42	0.82	NA	NA	NA	NA	NA	NA	NA	NA	NA

[1] Pre=Pre-test raw score, Post=Post-test raw score [2] RCI=Reliable Change Index [3] RCI calculated as compared to BVMT-R, Benedict, 1997
[4] RCI calculated as compared to HVLT-R, Benedict, Schretien & Groninger, 1998 [5] RCI calculated as compared to BTA, Schretlen, 1989
[6] RCI calculated as compared to Stroop Color-Word, Trenerry, Crosson, DeBoe & Leber, 1989 [7] RCI calculated as compared to Tombough, 2004
[8] RCI calculated as compared to COWAT, Benton, Hamsher & Sivan, 1994
[9] NA=Data not available [10] NC=No change between pre and post-testing

Table B2. *Active Control Group Clinically Significant Improvement Scores (*p<0.05)*

			P5			P6			P7		
			Pre[1]	Post[1]	RCI[2]	Pre	Post	RCI	Pre	Post	RCI
Memory	**BVMT-R[3]**	Trial 1	2	5	1.64	6	7	0.55	0	1	0.55
		Trial 2	7	6	-0.63	8	11	1.89*	1	2	0.63
		Trial 3	8	8	NC[8]	9	11	2.27*	3	5	2.27*
		Total Recall	17	19	0.62	23	29	1.86*	4	8	1.24
		Delayed Recall	7	7	NC	10	11	1.00	2	5	3.00*
	HVLT-R[4]	Total Recall	18	18	NC	25	29	1.37	8	11	1.02
		Delayed Recall	6	9	1.97*	9	10	0.66	2	2	NC
		RDI	10	10	NC	11	11	NC	3	8	3.21*
Attention	**Brief Test of Attention[5]**		19	20	0.51	17	18	0.51	5	4	-0.51
Executive Function	**Stroop Color-Word[6]**		78	103	3.41*	97	99	0.27	18	30	2.31*
	Trail Making[7]	Form A	34	30	-0.60	25	26	0.24	95	61	-5.73*
		Form B	59	58	-0.07	56	57	0.11	300	231	-10.70*

[1] Pre=Pre-test raw score, Post=Post-test raw score [2] RCI=Reliable Change Index [3] RCI calculated as compared to BVMT-R, Benedict, 1997
[4] RCI calculated as compared to HVLT-R, Benedict, Schretien & Groninger, 1998 [5] RCI calculated as compared to BTA, Schretlen, 1989
[6] RCI calculated as compared to Stroop Color-Word, Trenerry, Crosson, DeBoe & Leber, 1989 [7] RCI calculated as compared to Tombough, 2004 [8] NC=No change

Table B3. *Clinically Significant Improvement Score on Memory for Intentions Screening Test variables (*p<0.05).*

	Active Treatment												Active Control								
	P1			P2			P3			P4			P5			P6			P7		
	Pre[1]	Post[1]	RCI[2]	Pre	Post	RCI	Pre	Post	RCI	Pre	Post	RCI	Pre	Post	RCI	Pre	Post	RCI	Pre	Post	RCI
2 min	4	7	3.65*	8	5	-3.26	4	6	1.75*	7	8	1.09	8	7	-1.06	5	6	1.06	3	4	0.69
15 min	0	2	1.90*	1	4	2.16*	2	3	0.62	2	2	NC	5	6	0.68	4	4	NC	1	0	-0.67
Time	2	3	1.01	5	3	-1.61	2	5	2.22*	4	4	NC	7	5	-1.71	5	4	-0.85	4	2	-1.49
Event	2	6	4.98*	4	6	1.72*	4	6	1.46	5	6	0.86	6	8	1.83*	4	6	1.83	0	2	1.28
Verbal	4	5	1.05	6	4	-1.92	4	4	NC	6	6	NC	8	5	-2.91	4	4	NC	3	4	0.75
Action	0	4	4.53*	3	5	1.59	2	5	1.96*	3	4	0.79	5	8	2.33*	5	6	0.78	1	0	-0.63
PMT[4]	12	27	3.52*	27	27	NC	20	29	1.25	27	30	0.50	39	39	NC	27	30	0.50	12	12	NC
RRT[5]	7	7	NC[3]	7	8	2.00*	5	7	2.56*	8	7	-2.00	7	7	NC	6	6	NC	5	6	0.71

[1] Pre=Pre-test raw score, Post=Post-test raw score [2] RCI=Reliable Change Index [3] NC=No change [4] Prospective Memory Total [5] Retrospective Recognition Total

Calculations of Significant Inter-study Participant Improvements

Table B4. *Inter-participant analysis of pre/post-testing improvement on Brief Test of Attention (*p<0.05).*

	Δ[1]	RCI[2]
P1[a]	-12	-4.67
P2[a]	+2	0.78
P3[a]	+5	1.95*
P4[a]	+8	3.11*
P5[b]	+1	0.39
P6[b]	+1	0.39
P7[b]	-1	-0.39
Mean	0.57	
SDD[3]	6.29	
SE_{diff}[4]	2.57	

[a] Active treatment group [b] Active control group
[1] Difference in pre/post-testing score
[2] RCI=Reliable Change Index
[3] SDD=Standard deviation of the difference
[4] SE_{diff}=Standard error of the difference

Table B5. *Inter-participant analysis of pre/post-testing improvement on Brief Visuospatial Memory Test variables (*p<0.05).*

	Trial 1		Trial 2		Trial 3		Immediate Recall		Delayed Recall	
	Δ[1]	RCI[2]	Δ	RCI	Δ	RCI	Δ	RCI	Δ	RCI
P2[a]	+1	1.40	+2	3.36*	+1	1.58	+2	2.74*	+2	3.04*
P3[a]	-2	-2.79	+1	1.68	+4	6.32*	+3	4.11*	0	NC
P4[a]	+1	1.40	+1	1.68	+3	4.74*	+5	6.85*	-1	-1.52
P5[b]	+3	4.19*	-1	-1.68	0	NC	+2	2.74*	0	NC
P6[b]	+1	1.40	+3	5.05	+2	3.16*	+6	8.23*	+1	1.52
P7[b]	+1	1.40	+1	1.68	+2	3.16*	+4	5.48*	+3	4.56*
Means	0.8		1.2		2.0		3.7		0.8	
SDD[3]	1.60		1.33		1.41		1.63		1.47	
SE_{diff}[4]	0.72		0.59		0.63		0.73		0.66	

[a] Active treatment group [b] Active control group
[1] Difference in pre/post-testing score
[2] RCI=Reliable Change Index
[3] SDD=Standard deviation of the difference
[4] SE_{diff}=Standard error of the difference

Table B6. *Inter-participant analysis of pre/post-testing improvement on Hopkins Verbal Learning Test variables (*p<0.05).*

	Immediate Recall		Delayed Recall		Retrospective Discrimination Index	
	Δ[1]	RCI[2]	Δ	RCI	Δ	RCI
P1[a]	+3	3.05*	-1	-1.44	-7	-4.65
P2[a]	+1	1.02	0	NC	0	NC
P3[a]	0	NC	+3	4.31*	-2	-1.33
P4[a]	-3	-3.05	+3	4.31*	+2	1.33
P5[b]	0	NC	+3	4.31*	0	NC
P6[b]	+4	4.07	+1	1.44	0	NC
P7[b]	+3	3.05	0	NC	+5	3.32*
Means	1.1		1.3		-0.3	
SDD[3]	2.41		1.70		3.68	
SE_{diff}[4]	0.98		0.70		1.50	

[a] Active treatment group [b] Active control group [1] Difference in pre/post-testing score [2] RCI=Reliable Change Index
[3] SDD=Standard deviation of the difference [4] SE_{diff}=Standard error of the difference

Table B7. *Inter-participant analysis of pre/post-testing improvement on measures of Executive Function (*p<0.05).*

	Stroop Color-Word		Trail Making Form A		Trail Making Form B	
	Δ[1]	RCI[2]	Δ	RCI	Δ	RCI
P1[a]	+1	0.25	NA	NA	NA	NA
P2[a]	+25	6.37*	-1	-0.08	-26	-0.70
P3[a]	+14	3.57*	NA	NA	NA	NA
P4[a]	+13	3.31*	-36	-4.86*	-214	-5.84*
P5[b]	+25	6.37*	-4	-0.49	-1	-0.02
P6[b]	+2	0.51	-1	-0.19	+1	0.03
P7[b]	+12	3.06	-34	-4.57*	-69	-1.88
Means	13.1		-15.0		-61.6	
SDD[3]	9.61		18.03		89.67	
SE_{diff}[4]	3.93		7.36		36.61	

[a] Active treatment group [b] Active control group [1] Difference in pre/post-testing score [2] RCI=Reliable Change Index [3] SDD=Standard deviation of difference [4] SE_{diff}=Standard error of difference

Table B8. *Inter-participant analysis of pre/post-testing improvement on MIST variables (*p<0.05).*

	2 min		15 min		Time		Event		Verbal		Action		PMT		RRT	
	Δ[1]	RCI[2]	Δ	RCI	Δ	RCI	Δ	RCI	Δ	RCI	Δ	RCI	Δ	RCI	Δ	RCI
P1[a]	3	4.00*	2	3.92*	1	1.39	4	11.76*	1	1.75*	4	6.35*	15	6.94*	0	NC
P2[a]	-3	-4.00	3	5.88*	-2	-2.78	2	5.88*	-2	-3.51	2	3.17*	0	NC	1	2.78*
P3[a]	2	2.67*	1	1.96*	3	4.17*	2	5.88*	0	NC	3	4.76*	9	4.17*	2	5.56*
P4[a]	1	1.33	0	NC	0	NC	1	2.94*	0	NC	1	1.59	3	1.39	-1	-2.78
P5[b]	-1	-1.33	1	1.96*	-2	-2.78	2	5.88*	-3	-5.26	3	4.76*	0	NC	0	NC
P6[b]	1	1.33	0	NC	-2	-2.78	2	5.88*	1	1.75*	-1	-1.59	0	NC	0	NC
P7[b]	1	1.33	-1	-1.96	-1	-1.39	2	5.88*	0	NC	1	1.59	3	1.39	0	NC
Means	0.6		0.9		-0.4		2.1		-0.4		1.9		4.3		0.3	
SDD[3]	1.99		1.35		1.90		0.90		1.51		1.68		5.71		0.95	
SE_{diff}[4]	0.75		0.51		0.72		0.34		0.57		0.63		2.16		0.36	

[a] Active treatment group [b] Active control group [1] Difference in pre/post-testing score [2] RCI=Reliable Change Index [3] SDD=Standard deviation of difference [4] SE_{diff}=Standard error of difference

Table B9. *Inter-participant analysis of pre/post-testing improvement on quality of life measures of generalization (*p<0.05).*

	Physical Health		Psychological Health		Social Relationships		Personal Environment	
	Δ[1]	RCI[2]	Δ	RCI	Δ	RCI	Δ	RCI
P2[a]	25	4.68*	0	NC	0	NC	13	5.18*
P3[a]	12	2.25*	0	NC	-12	-5.86	12	4.78*
P4[a]	18	3.37*	6	4.36*	0	NC	0	NC
P5[b]	0	NC	7	5.09*	0	NC	6	2.39*
P6[b]	-6	-1.12	0	NC	0	NC	0	NC
P7[b]	-6	-1.12	0	NC	-6	-2.93	0	NC
Means	7.2		2.2		-3.0		5.2	
SDD[3]	13.09		3.37		5.02		6.15	
SE_{diff}[4]	5.34		1.38		2.05		2.51	

[a] Active treatment group [b] Active control group [1] Difference in pre/post-testing score [2] RCI=Reliable Change Index
[3] SDD=Standard deviation of difference [4] SE_{diff}=Standard error of difference

Table B10. *Inter-participant analysis of pre/post-testing improvement on attention, memory & executive function measures of generalization (*p<0.05).*

	APT		EMQ		Dex	
	Δ[1]	RCI[2]	Δ	RCI	Δ	RCI
P2[a]	-28	-5.63*	-14	-1.98*	-11	-2.04*
P3[a]	-2	-0.40	-29	-4.10*	-7	-1.30
P4[a]	-5	-1.00	-22	-3.11*	-34	-6.29*
P5[b]	-7	-1.41	-7	-0.99	-9	-1.67
P6[b]	9	1.81	-13	-1.84	-9	-1.67
P7[b]	-2	-0.40	-55	-7.79*	7	1.30
Means	-5.8		-23.3		-10.5	
SDD[3]	12.19		17.31		13.23	
SE_{diff}[4]	4.98		7.06		5.40	

[a] Active treatment group [b] Active control group [1] Difference in pre/post-testing score [2] RCI=Reliable Change Index
[3] SDD=Standard deviation of difference [4] SE_{diff}=Standard error of difference

Table B11. *Inter-participant analysis of pre/post-testing improvement on prospective memory measures of generalization (*p<0.05).*

	Long-term episodic		Short-term habitual		Internally cued		Techniques to Remember		PMT Total	
	Δ[1]	RCI[2]	Δ	RCI	Δ	RCI	Δ	RCI	Δ	RCI
P2[a]	-0.6	-1.50	-0.7	-3.24*	-3	-6.31*	2	1.90	-1.4	-9.07*
P3[a]	-2.4	-5.99*	-0.8	-3.71*	-0.9	-1.89	0.7	0.66	-0.9	-5.83*
P4[a]	-2.1	-5.24*	-1.5	-6.95*	-3.3	-6.94*	2.9	2.75*	-0.9	-5.83*
P5[b]	-0.5	-1.25	-0.5	-2.32*	-0.7	-1.47	-3.4	-3.23	-1.3	-8.42*
P6[b]	-1.4	-3.49*	-0.2	-0.93	-1	-2.10*	-0.1	-0.09	-0.7	-4.53*
P7[b]	0.1	0.25	NA		-2.5	-5.26*	-3	-2.85	-1.7	-11.01*
Means	-1.2		-0.7		-1.9		-0.2		-1.2	
SDD[3]	0.98		0.48		1.16		2.58		0.38	
SE_{diff}[4]	0.40		0.22		0.48		1.05		0.15	

[a] Active treatment group [b] Active control group [1] Difference in pre/post-testing score [2] RCI=Reliable Change Index [3] SDD=Standard deviation of difference [4] SE_{diff}=Standard error of difference