The Relationship Between Memory in Individuals with Post-Concussion Syndrome with Comorbid Depression and/or Anxiety Symptoms

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THESIS APPROVAL

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Abstract

The purpose of this study was to assess memory in individuals who suffer from post-concussion syndrome (PCS) with comorbid anxiety and/or depressive symptoms. While much of the literature highlights concussions at the time of injury, symptom presentation, and short-term recovery, little research considers PCS, a common condition that consists of symptoms that last past the typical three-month recovery period. Many individuals with PCS complain of physical, affective, and cognitive symptoms. Cognitive symptoms, a primary impairment for 15% of sufferers typically include dizziness, headaches, memory loss, and a decrease in concentration. An overwhelming number of patients diagnosed with PCS also report depression and anxiety. Based on the literature, this study predicted that individuals with PCS and anxiety and/or depressive symptoms will perform worse on standardized cognitive measures compared to controls. This study utilized a convenience sample to qualitatively compose six control participants and one PCS participant. The researchers assessed percentile rank and performance of memory compared to controls. The patient performed in the highest percentile for six out of eight measures and scored in the lowest percentile for two of the eight measures. This study illustrates techniques that can be applied to larger studies aimed at evaluating the long-term consequences of concussions. Future research should strive for equal groups to increase power and assess significance.

The Relationship Between Memory in Individuals with Post-Concussion Syndrome and Comorbid Depression and Anxiety Symptoms

Concussions, a form of mild traumatic brain injury (mTBI), are a longstanding threat to the well-being of children, adolescents, adults, and older adults. These high incidence rates of concussions make head injuries a national concern. According to the Concussion Legacy Foundation (2023), nearly 3.8 million accounts of concussions occur in the United States per year as a result of sports and recreation alone. The CDC reports estimates of 3 to 5 million concussions each year, where the most common cause is motor vehicle accidents and elderly falls (Concussion Legacy Foundation, 2023). Seventeen percent of these reports are due to sport-related injuries (Conder et al., 2020). With these incident rates, improved diagnostic criteria and treatment and recovery approaches are key to improving the well-being of all sufferers. To add perspective, the CDC notes there are 266,400 cases of breast cancer and 795,000 cases of strokes diagnosed each year.

Consequences of concussions include an array of complications. Not only is the recovery process potentially lengthy and painful, but the aftermath of concussions may result in cognitive impairments long-term. Long-term cognitive deficits are believed to be associated with multiple concussions, although not exclusively. When comparing information processing speed in college football players with no previous concussions to players with multiple concussions, players with a history of injury performed worse (Iverson et al., 2004). To further this, Iverson and researchers (2004), reported findings of individuals' auditory processing with multiple concussions and only one concussion. Those with multiple concussions obtained lower processing scores compared to those with one concussion. Such data exemplifies the potential risk factors of concussions.

To study concussions, researchers often turn to animal models for evidence of concussion etiology and natural course of recovery. Animal studies allow researchers to explore head injuries, which often mimic the human response to injury. Typically, the onset of a concussion triggers a neurometabolic cascade, mostly in the central nervous system, that dysregulates connections between hyper-glycolysis and glucose metabolism, as well as a decrease in blood flow (Leddy et al., 2012). These studies show, homeostasis recovering after 7 to 10 days, on average, which reflects the typical human recovery recommendation of a few weeks to three months to regain full functioning (Conder et al., 2020). When symptoms persist beyond three months post-injury, individuals are diagnosed with post-concussion syndrome (PCS). Based on past studies and PCS symptom reports, estimates of 10% to 30% of concussion patients will be diagnosed with PCS (Clarke et al., 2012).

Several different definitions of PCS are found across the literature. In 2016, the *5th International Conference on Concussion in Sport* identified four criteria for a *sport-related concussion* as: (a) an indirect or direct force to the head, face, neck, or other location that impacts the head; (b) immediate or rapid neurological impairments that may evolve, but resolve spontaneously; (c) loss of consciousness, and evidence of functional injury rather than structural via neuroimaging; and (d) resolution of symptoms and impairments, although they may be prolonged (Conder et al., 2020).

The lack of consensus diagnostic criteria is problematic for recovery protocols, and then diagnosing and establishing best practices for PCS. Without a standard definition of concussion or PCS, medical providers rely on their subjective experiences to diagnose and treat the latter. In 2016, the *American Medical Society for Sports Medicine* and the *Berlin Consensus in Sport Group* agreed on four domains of PCS criteria: (a) a blow or force to the head with relatively

immediate symptomology; (b) symptoms including headache, difficulty balancing, fatigue, dizziness, sleep problems, sensitivity to noise and/or light, dysregulated mood, and vision changes; (c) symptoms persisting beyond a few weeks to months; and (d) a lack of alternate explanations for the other symptoms (Conder et al., 2020). The *International Classification of Disease, 10th Revision* (ICD-10) proposes a different set of criteria that include 3 or more symptoms including headache, dizziness, insomnia, irritability, fatigue, memory difficulty, concentration difficulty, and reduced tolerance to alcohol, emotional regulation, and stress. The *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition* (DSM-IV) required 3 symptoms from a different list of inclusion criteria including sleep disturbances, fatigue, irritability, headache, dizziness, personality change, apathy, and affective disturbances that last a minimum of 3 months post-injury. In addition, the DSM-IV required evidence of cognitive impairment in attention and/or memory, and difficulty functioning in social and/or occupational areas of life. However, the newest edition of the DSM, DSM-5, removed PCS from the manual and replaced it with *Major or Minor Neurocognitive Disorder Due to Traumatic Brain Injury*. This specification does not recognize the majority of the above symptoms as criteria for diagnosis. This new condition is only applicable if the individual exhibits loss of consciousness, posttraumatic amnesia, focal neurological signs via neuroimaging, or disorientation (Tator et al., 2016).

According to medical professionals, a more agreed-upon definition of PCS at this time is described as the onset of a variety of concussion symptoms that persist beyond the expected recovery time, typically ranging from 2 weeks to more than 3 months, although some may persist for a few years (e.g., Guty, et al., 2021 & Price et al., 2019). Although this definition is vague, it does reflect evidence from clinical studies. For instance, Ryan and researchers (2003) understood

PCS as a condition where symptoms may resolve within one month after onset; however, some may persist for fifteen or more years. Common complaints of PCS can be classified into ifive domains, which have been differentially validated and include (a) physical symptoms, such as balance difficulties, nausea, vomiting, dizziness, sensitivity to light, and sensitivity to noise; (b) affective disturbances, such as sadness, nervousness, and irritability; (c) cognitive symptoms, such as difficulty concentrating, memory problems, fogginess, and feeling slowed down; (d) sleep disturbances including fatigue, drowsiness, insomnia, and sleeping more or less than usual; and (e) headache (Clarke et al., 2012; Guty et al., 2021; Price et al., 2019).

Using the ICD-10's criteria, Tator and colleagues (2016) reported an average of 8.1 symptoms of PCS with a range of 3 to 23 total symptoms among a sample of 221 concussion victims. The most common symptoms reported by their participants included memory impairments, headaches, imbalance, dizziness, and concentration difficulties. Furthermore, neuroimaging techniques exhibited two common abnormalities, arachnoid cysts were present in 3.6% of participants, as opposed to .006% to 1.7% in the general public. As the most common type of brain cyst, arachnoid cysts are typically non-threatening but may need to be treated via draining the fluid or shunting if symptoms arise. Chiari malformations were also present in 4.5% of participants evaluated by Tator and colleagues (2016), as opposed to a prevalence of .00078% in the population. Chiari malformations occur when the cerebellum is pressed through the base of the skull and into the spinal cord. While this condition is generally non-life threatening, the symptoms may include persistent headaches and pain associated with concussions.

Clarke and colleagues (2012) reported that 15% of those diagnosed with a concussion experience PCS, and among sufferers, cognitive symptoms are most common. Cognitive difficulties are substantial contributors to long-term impairment after a mild traumatic brain

injury (mTBI). Clarke and colleagues (2012) discussed a meta-analysis that showed significantly lower performance on measures of attention, executive functioning, processing speed, and memory in patients with mTBI compared to controls. Interestingly, objective cognitive deficits were not shown in patients who reported post-concussive symptoms, yet it has been shown that performance on objective cognitive tests is unrelated to complaints in patients with mTBI during the recovery period (Clarke et al., 2012).

Memory assessments can expose mTBI impairments. For instance, Custer and researchers (2016) reported a decline in both visual and verbal memory when comparing scores on the self-report post-concussion symptom scale (PCSS) at baseline and post-injury. Participants were grouped as asymptomatic and highly symptomatic at baseline. Findings suggested those with high symptomology performed worse on the verbal and visual memory assessments of the Immediate Post-concussion Assessment and Cognitive Test (ImPACT) compared to the asymptomatic group during the acute period (2-7 days post SRC) (Custer et al., 2016). Interestingly, the high symptomology group generated identical scores on the PCSS and the ImPACT during the acute phase and at baseline. Not only is this evidence that individuals with symptoms at baseline are at higher risk for negative outcomes after injury, but this evidence reveals impairments associated with verbal and visual memory, specifically.

Guty and colleagues (2021) described an overall reduction in memory performance over time for those suffering from PCS. Researchers assessed pediatric participants 7 months post-injury with various neurological difficulties. Participants who scored significantly lower on executive function and attention tasks reported their most prevalent symptom as a headache, which is consistent with other studies (Guty et al., 2021). An adult sample showed similar deficits specifically on assessments of verbal fluency, attention, processing speed, and cognitive

flexibility. These findings illustrate lasting impairments of concussions. Guty and researchers (2021) note that these results may have underlying connections to other psychological factors. The role that pre-existing mental health factors may play in unknown.

Depression and other affective disorders often mimic PCS symptoms (Thomas et al., 2022). Thomas and colleagues (2022) note that symptoms of depression and anxiety re common in PCS patients who had no symptoms prior to the concussion. In non-PCS patients, depression and anxiety symptoms can present as a decrease in concentration, lack of energy, memory disturbances, and irritability (American Psychiatric Association, 2013; Gillie et al., 2022). Depression in conjunction with concussion injuries may interact and prolong symptoms.

These findings suggest that neurological damage in addition to other unknown factors may lead to long-term PCS development. For example, researchers noted a correlation between cognitive symptoms and anxiety and depression scores in PCS patients. Clarke et al., (2012) suggest that those with elevated levels of both depression and anxiety also demonstrated more cognitive complaints. Following a sports-related concussion, 49% of athletes report emotional symptoms with 33% experiencing anxiety and 20% experiencing depression (Price et al., 2019).

While pre-existing emotional factors have been supported in the literature to exacerbate persistent concussion symptoms, little is understood about the development of emotional disorders following a concussion. Anxiety is seen in the recovery from a concussion and often presents as excessive worry with physiological arousal (Gillie et al., 2022). The presence of anxiety is associated with more symptoms reported and higher severity of symptoms on post-concussion symptom measures as well as increased concerns about academic success and self-esteem. (Gillie et al., 2022). Individuals with emotional disturbances following injury show poorer reaction time performance compared to other concussion groups (Datta et al., 2019).

Concussions likely precipitate physiological and chemical changes that lead to the development of anxiety. To assess these changes, researchers must obtain a baseline of anxiety prior to injury and after injury. Metabolic changes that result from head impacts may be a factor in the maintenance or development of depression and anxiety. On the other hand, research might benefit from assessing depression and anxiety as the impact of injury on life satisfaction and functioning (i.e. fear of reinjury, unable to attend school or work, stress on relationships, etc.). An understanding of these changes may lead to improved treatment for PCS.

The precise etiology of PCS is poorly understood as is its link to affective symptoms (Ryan et al., 2003). PCS might be a consequence of axonal damage (Ryan et al., 2003). Other theories suggest that pre-existing mood disorders might complicate recovery following mTBI. For example, Price and colleagues (2019) reported findings from Yang (2015) and researchers and Yrondi (2017) and researchers where participants who reported preexisting depression were 4.6 times more likely to continue experiencing depression after injury. Similarly, those with anxiety prior to mTBI were 3.4 times more likely to report symptoms thereafter. Silverberg and Iverson (2011) propose an explanation for the neurobiological changes during recovery and the potential influence of psychological factors: Symptoms and injuries of PCS are organic in origin; however, the neurobiological damage begins to subside, and psychological factors arise from this unresolved metabolic dysregulation, sustaining PCS. In the instances where PCS continues for several months to years, it is probable that psychological symptoms are hindering functional and symptomatic recovery (Silverberg et al., 2011). Interestingly, Silverberg and Iverson (2011) added that neuropsychological assessments, specifically measuring post-traumatic amnesia, were not associated with PCS (r-value = .07 to .18), yet both anxiety and depression measures were strongly correlated (r-values = .60 to .65).

In 2018, Pearce et al. (2018) conducted a neuroimaging study to show that the corpus callosum in retired American football players presented microstructural changes correlating to cognitive impairments. Former professional soccer players showed cortical thinning, as well as a reduced performance in memory compared to controls (Pearce et al., 2018). Pearce et al. (2018) presented data on long-term neurophysiological, cognitive, and motor changes in retired professional rugby players with a history of concussion. The players showed slower reaction time in dexterity and visuomotor tests, as well as poorer cognitive performance and reduced cortical silent period (cSP) at suprathreshold stimulation intensities during transcranial magnetic stimulation (TMS). These assessments were done nearly twenty years post-concussion and provide evidence for long-term sequelae for individuals with a history of multiple concussions in contact sports.

In summary, research has begun to elucidate the immediate and long-term consequences of concussions. Questions regarding prevention, assessment, diagnostic criteria, and treatment have appeared in the recent literature, showing there is still much ambiguity regarding the factors that prolong the symptoms. The research clearly shows that cognitive symptoms are the most experienced and impeding symptoms of PCS. This study strives to address these symptoms.

Concussion victims can suffer from symptoms long after the average three-month recovery time. Persistent symptoms include physical, mood, and cognitive impairments. Further research needs to clarify which symptoms are more likely to persist and how these symptoms impact daily lives. This study investigates these phenomena.

The purpose of this study is to contribute to the current knowledge of PCS by providing a clearer understanding of the associations between PCS, memory, and possibly secondary factors. Further understanding may help future research treat these symptoms. This study hypothesizes

(a) memory performance will be poorer for individuals with PCS compared to controls, (b) memory performance will be poorer for individuals with PCS and present depressive symptoms compared to controls, (c) memory performance will be poorer for individuals with PCS and present anxiety symptoms compared to controls, and (d) memory performance will be poorer for individuals with PCS and present depressive and anxiety symptoms compared to controls.

Method

Participants

Participants were recruited by the principal investigator via convenience sampling from Millersville University students. Inclusion criteria included anyone between the ages of 18 and 50 and anyone with a current diagnosis of post-concussion syndrome (PCS) comorbid with current depression and/or anxiety symptoms. Individuals without a current PCS diagnosis, as well as no current depression or anxiety symptoms, were eligible to participate as members of the control group. Individuals recovering from a concussion at the time of testing were not eligible nor were any individuals with present learning disabilities or developmental delays.

The patient assessed in this study is a 25-year-old, white female currently enrolled in graduate studies. She reported having 6 previous concussions and being diagnosed with PCS in October 2018. The patient identified multiple, current symptoms in 3 of the symptom domains. The patient experiences physical symptoms 30% of the time including sensitivity to light and noise, and headaches; affective symptoms 30% of the time including depression, irritability, and anxiety; and cognitive symptoms 100% of the time including attentional difficulties, and memory impairments.

The initial control group consisted of 9 individuals who have never experienced a concussion, have never been diagnosed with PCS, and were not experiencing anxiety or

depression at the time of their participation. Of these 9 participants, 3 were excluded due to incomplete assessments; thus, the final control group included 3 male and 3 female participants. The control group completed an identical assessment as the patient.

Materials

Subtests from the Weschler Memory Scale IV (WMS-IV) were used to assess memory. The six subtests included Visual Reproduction I, Logical Memory I, Visual Reproduction II, Logical Memory II, Verbal Paired Associates I, and Verbal Paired Associates II. Each of these measured their respective domains of memory in both short-term and long-term capacities. This study also utilized the Beck Depression Inventory (BDI) and the Beck Anxiety Inventory (BAI) to assess those conditions.

Procedure

The principal investigator sent an email to all psychology and health and wellness professors at Millersville University. This email described a brief overview of the study and attached links for students to use to sign up for their best-fit testing times. Each participant was assessed individually for one three-hour session. The researcher verbally walked the participant through informed consent and upon receiving a signed consent form, the researcher continued onto the first assessment.

Three domains of memory were assessed including visual memory, logical memory, and verbal memory. Each of these domains consisted of paired assessments, where the first assessment evaluated short-term memory, and the second test (delivered after a 20-minute delay) evaluated long-term memory. During each delay, researchers either began the next short-term memory assessment and/or provided a filler task (e.g. crossword puzzles, word searches,

sudoku). The delay tasks engaged the participants and were designed to prevent rehearsal of previously learned information from the short-term memory assessments.

Visual Reproduction I (VRI) was delivered first and assessed short-term memory by requiring participants to draw a design they had been shown ten seconds earlier. The researcher recorded the time it took the participant to complete their drawing. To measure long-term memory, a 20-to-30-minute delay was required between each paired assessment. Next, the researchers provided the Beck Anxiety Inventory (BAI) to assess for current anxiety symptoms and symptom severity. Following this, the researcher delivered Logical Memory I (LMI) to assess short-term, narrative episodic memory that required the participant to verbally retell the short story that was just read aloud to them. If the participant completed LMI with time remaining from the delay, the researcher provided filler tasks. Once the delay was satisfied, the researcher delivered Visual Reproduction II (VRII) and asked the participant to draw the designs they could recall, followed by pointing to the designs they recognized from VRI. Next, if the delay time had not yet reached 20-to-30- minutes, the researcher supplied the participants with a filler task. Once the delayed time was satisfied, the researcher delivered Logical Memory II (LMII). Participants were asked to recite the two stories they heard from LMI and provide as much detail as they could recall. Following, the researcher delivered Verbal Paired Associates I (VPAI) and read aloud word pairs as the participant listened. This assessment evaluated the participant's associative and episodic memory. After each pair was read aloud, the researcher read aloud the first word in the pair and instructed the participant to say aloud the second word in the pair. This was completed four times, and each time, the order of the word pairs was scrambled. Next, the researcher provided the participant with the Beck Depression Inventory (BDI). Following, the participant was given filler tasks to satisfy the 20-to-30-minute delay

between assessments. Once the 20-to-30-minute delay was satisfied, the researcher delivered Verbal Paired Associates II (VPAII) and again instructed the participant to say aloud the second word of the word pair after hearing the first. After this section was completed, the researcher then instructed the participant to answer "yes," or "no," after hearing a word pair, indicating that it was or was not a word pair learned in VPAI.

Following the completion of these six subsets and Beck assessments, the researcher asked the participant to complete a demographic questionnaire. After completion of the demographic questionnaire, the researcher then asked the participant to fill out a compensation form, indicating if they would prefer compensation in the form of cash or gift cards. Finally, the participant was debriefed and dismissed.

Results

Descriptive statistics were calculated for each subtest and symptom scale (see Table 1-Table 3). Due to the study's little power, no statistical analyses for significance could be completed. Instead, the researchers identified percentile rank and considered mean scores and standard error (see Table 3 and Figure 1). The PCS patient scored in the top percentile for six out of the eight measures, including accuracy scores on VRI (71.4th percentile), VRII: Recognition (16.6th percentile), LMI (100th percentile), LMII (100th percentile), VPAII: Recall (60th percentile), and anxiety levels (100th percentile). These scores indicate no memory impairments on the above subtests. The PCS participant's anxiety score indicates higher levels of anxiety compared to controls, as the researchers suspected. The PCS participant scored in the lowest percentile for VRII: Recall (0 percentile), VPAI (0 percentile), and VPAII: Recognition (0 percentile). These scores support the notion that individuals with PCS may perform more poorly on memory assessments, specifically verbal and visual memory recall. The patient scored in the

80th percentile for current depressive symptoms. The researchers expected a higher depression score for the PCS participant relative to controls, although the PCS participant scored within the same range of scores relative to controls.

The control group's mean and standard error was calculated for each subtest and anxiety and depression questionnaires (see Table 3 and Figure 1). The mean accuracy score for VRI is 40.7 $\pm$ .92 (SE). The patient's raw score of 43 falls within the standard error relative to controls, indicating the PCS participant performed as accurate on VRI as controls. The mean accuracy score for VRII: Recall is 39.7 $\pm$ 1.36 (SE). The PCS participant's raw score of 28 falls below the standard error relative to controls, indicating the patient's performance on VRII: Recall was less accurate than controls. The researchers note the spread of SE for VRII: Recall may be less indicative of performance by the general population. The mean accuracy score for VRII: Recognition is 6.7 $\pm$ .33 (SE). The PCS participant's raw score of 7 falls within the standard error relative to controls, indicating the patient performed as accurate on VRII: Recognition as controls. A small SE indicates a more accurate representation of performance across the general population of controls. The mean accuracy score for LMI is 25.2 $\pm$ 3.24 (SE). The PCS participant's raw score of 39 falls above the standard error relative to controls, indicating the patient's performance on LMI was more accurate than controls. The mean accuracy score for LMII is 21.3 $\pm$ 2.84 (SE). The PCS participant's raw score of 36 falls above the standard error relative to controls, indicating the patient's performance on LMII was more accurate than controls. The mean accuracy score for VPAI is 46.7 $\pm$ 2.49 (SE). The PCS participant's raw score of 37 falls below the standard error relative to controls, indicating the patient's performance on VPAI was less accurate than the controls. The mean accuracy score for VPAII: Recall is 13.2 $\pm$ .37 (SE). The PCS participant's raw score of 14 falls within the standard error

relative to controls, indicating the PCS participant performed as accurate on VPAII: Recall as controls. The mean accuracy score for VPAII: Recognition is 40 $\pm$ 0 (SE). Due to all controls obtaining the same score, there is no variation to account for standard error. The PCS participant's raw score of 39 falls within the standard error relative to controls, indicating the PCS participant performed as accurate on VPAII: Recognition as controls. The mean score for anxiety is 11.2 $\pm$ 1.83 (SE). The PCS participant's raw score of 19 falls above the standard error relative to controls, indicating the PCS participant is experiencing higher levels of anxiety. The mean score for depression is 6.6 $\pm$ 2.58 (SE). The PCS participant's raw score of 15 falls above the standard error relative to controls, indicating the patient is experiencing higher levels of depression compared to controls.

Discussion

The researchers utilized percentile ranks and standard error to understand the current data. Based on the data, no conclusions can be made regarding the study's hypotheses.

Other limitations to this study include the sampling method and sample size. A large sample size is often difficult to come by in neuropsychology research, as finding individuals with identical brain injury and symptom presentation is rare. Although concussions are a commonly studied brain injury, PCS is not. The researchers found difficulty locating clinics where individuals with PCS seek treatment; therefore, the sampling method was altered to convenience sampling on Millersville University's campus. Further research should be conducted from a clinic where individuals with PCS are diagnosed and/or treated to get a more accurate collection of data. This study also had a small sample size with unequal groups.

The administration of the BDI and the BAI pose limitations as well. Since they are both self-report measures, potential biases may occur due to the high face validities of these measures.

Participants are aware of what the items are assessing, raising the risk of faking good/bad (Richter et al., 1998). Depression and anxiety are understood as psychological and physiological conditions. One limitation of BAI specifically is the focus on physiological symptoms more than psychological, and cognitive symptoms.

These findings parallel certain findings from the current literature. Specifically, the patient reported higher levels of both anxiety and depression compared to controls and demonstrated poorer memory performance on verbal memory and visual memory recall. Possible explanations could include location of injury, abnormal structural or functional damage, or limited cognitive resources to cue recall. The patient's memory performance raises questions about the effect of injury on the type of memory required for recall and recognition tasks. Further investigation is necessary to understand these relationships.

Future research should strive for groups with equal variance and more than one individual with PCS. Since neuropsychological disorders and conditions present differently in each patient, it is even more essential to determine an experimental group with multiple members to achieve a more accurate understanding of PCS in the general population. With this stable group, future research will be able to assess for significance and possible regression models that can better inform the public, athletes, and medical professionals of the concerns surrounding PCS.

If this study achieved equal groups, an assessment of correlation and testing for group differences would provide information about the association between memory in individuals with PCS who are also experiencing anxiety and/or depressive symptoms. A regression model might also offer valuable intel about the patterns associated with these factors of PCS symptoms. If similar patterns appeared, the next step in this research would be to assess whether anxiety or

depression is a moderator or mediator in the maintenance of PCS memory impairments. Researchers should be mindful about pre-existing depression and anxiety in participants and consider baseline data prior to conducting assessments.

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Table 1

Descriptive Statistics of Control Group

Measures	*M*	*SE*	Median	Mode	*SD*	Sample Variance	Range
Age (years)	21.33	1.41	20.5	18	3.44	11.87	18 - 26
Education (years)	2.33	0.49	2.5	3	1.21	1.47	1 - 4
Number of Concussions	0	0	0	0	0	0	0
PCS Diagnosis	0	0	0	0	0	0	0

Note. $n = 6$.

Table 2

Demographics of PCS Patient

Measure	Raw
Age	*25*
Education Level	Graduate studies
Number of Concussions	6
PCS Diagnosis	October 2018

Note. $n = 1$.

Table 3

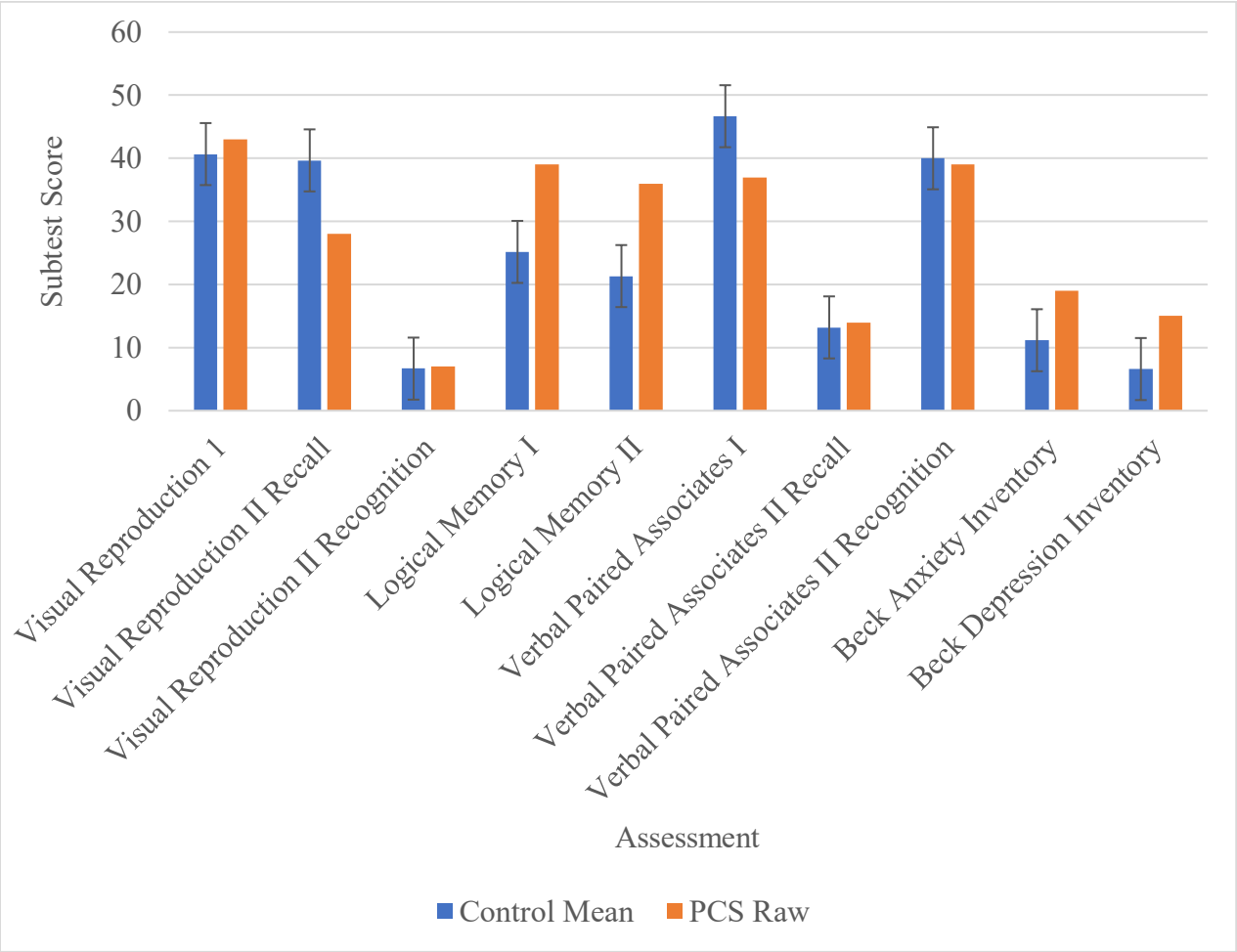
Descriptive Statistics of Memory Assessments and Depression and Anxiety Levels

Measures	*M*	*SE*	Median	Mode	*SD*	Sample Variance	Range	*n*	PCS Raw
VRI	40.67	0.92	41.5	42	2.25	5.07	37 - 43	6	43
VR II Recall	39.67	1.36	41	41	3.33	11.07	35 - 43	6	28
VR II Recognition	6.67	0.33	7	7	0.82	0.67	5 - 7	6	7
LM I	25.17	3.24	25.5	#N/A	7.94	62.97	12 - 36	6	39
LM II	21.33	2.84	22	#N/A	6.95	48.27	11-29	6	36
VPA I	46.67	2.49	46.5	53	6.09	37.07	37-53	6	37
VPA II Recall*	13.2	0.37	13	14	0.84	0.7	12-14	5	14
VPA II Recognition*	40	0	40	40	0	0	0	5	39
BAI	11.17	1.83	12.5	15	4.49	20.17	5 -15	6	19
BDI*	6.6	2.58	6	#N/A	5.77	33.3	1 - 16	5	15

*Participants data excluded due to incomplete assessment.

Figure 1

Memory Performance Accuracy and Levels of Depression and Anxiety Between the Control Group and PCS Patient with Standard Error



Note. The values shown represent the control group mean scores and the PCS patient's individual scores.