

Research Article

Patterns of cognitive deficits in persons with spinal cord injury as compared with both age-matched and older individuals without spinal cord injury

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Context/Objective: Cognitive deficits can impact as many as 60% of individuals with spinal cord injury (SCI). In an effort to identify the nature of cognitive deficits in SCI, we examined neuropsychological test performance in individuals with SCI, age matched healthy controls and older healthy controls.

Design: Participants completed a motor-free neuropsychological test battery assessing attention, working memory, information processing speed, new learning /memory and executive control.

Setting: Outpatient rehabilitation research facility.

Participants: Participants included 60 individuals with chronic spinal cord injury [SCI; 32 with paraplegia (T2-T12) and 28 with tetraplegia (C3-T1)], 30 age-matched healthy controls (AMHC; 30–40 years old) and 20 older healthy controls (OHC; 50–60 years old).

Outcome Measures: Wechsler Intelligence Scale – 3rd edition (WAIS-III) Digit Span and Letter-Number Sequencing; Symbol Digit Modalities Test (SDMT) – oral version; California Verbal Learning Test-II; Paced Auditory Serial Addition Test (PASAT); Wechsler Abbreviated Scale of Intelligence (WASI); Delis-Kaplan Executive Function System; Verbal Fluency substest.

Results: Significant differences were noted between the SCI and AMHC groups on measures of information processing speed, new learning and memory, and verbal fluency. No significant differences were noted between the groups on tests of attention or working memory.

Conclusion: The current study documented differences in specific realms of cognitive functioning between a chronic SCI sample and AMHC. Implications for cognitive rehabilitation and overall quality of life are discussed. Additional research is needed utilizing a more comprehensive battery of motor-free neuropsychological tests that avoid the confound of upper limb motor limitations on cognitive performance.

Keywords: Spinal cord injury, Cognitive deficits, Memory, Processing speed, Executive functioning

Introduction

Traumatic spinal cord injury (SCI) is a significant public health concern¹ and decades of research have

focused on the associated physical limitations, as well as therapies for addressing these physical deficits. However, multiple studies have additionally documented cognitive deficits in persons with SCI, noted in attention,²⁻⁷ concentration,³⁻⁶ new learning and memory (NLM),^{3-5,7,8} abstract reasoning,³ verbal learning,³⁻⁵ and processing speed (PS),⁹ even in

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relatively young SCI cohorts (28–45 years).^{2,3,10,11} As many as 60% of individuals display some degree of impairment.^{2,3,10–12} The relative risk of cognitive impairment in persons with SCI is 13 times greater than that which has been documented in uninjured individuals.¹³ Previous research has identified distinct patterns of cognitive impairment in persons with chronic SCI, characterized by deficits in identifiable cognitive domains, namely in PS, NLM and executive functioning.¹² Most recently, Molina and colleagues¹⁴ demonstrated cognitive deficits in individuals with SCI during the acute period following injury (2–6 months post-SCI) that appear to worsen over time (one or more years post-injury).

Cognitive deficits exert a substantial negative impact on everyday life and quality of life (QOL) in various neurological populations,^{15–21} having a critical impact on functional outcomes after SCI in particular. Cognitive deficits after SCI have been associated with diminished functional gains during rehabilitation,²² increased aggressive behaviors,²³ higher likelihood of re-hospitalization,²⁴ and limited acquisition of novel, day-to-day skills required for successful community re-integration after SCI.^{9,25} Cognitive deficits thus detract from comprehensive rehabilitation efforts and social integration, and are also associated with poor self-perception and QOL.^{9,25,26} Indeed, persons living with SCI report significant decreases in cognitive functioning from before to after injury, which relate to decreased overall QOL.²⁷

Although the etiology of the cognitive deficits in SCI remains elusive, several factors have been suggested as contributory including concomitant traumatic brain injury (TBI),^{2,3,10,11,28–34} secondary trauma as a result of cerebral edema, hypoxia and anoxia²⁷ and cardiovascular and cerebrovascular dysfunction.^{7,35,36} Recent studies also suggest that factors such as sleep disordered breathing/sleep apnea,³¹ core body temperature dysregulation,^{37,38} as well as medications prescribed for symptom management such as pain,^{39,40} and neurogenic lower urinary tract dysfunction⁴¹ may contribute to post-SCI cognitive dysfunction. Recent work by Bombardier and colleagues⁴² highlight the likelihood that factors other than TBI likely contribute to cognitive deficits post-SCI, because the number of patients reporting cognitive deficits exceeded physician-rated presence of TBI by 80% in their sample of 105 persons with SCI. Clearly, factors other than concomitant TBI are involved.

One candidate explanation for this pattern of increasing cognitive deficits post-SCI is *accelerated brain aging*. Across a variety of neurological (e.g. TBI; for review⁴³),

psychiatric (e.g. schizophrenia,⁴⁴ alcohol dependence⁴⁵), and medical (e.g. hypertension,⁴⁶ cardiac health⁴⁷) conditions, increasing evidence suggests that neural damage akin to the aging process is exacerbated, with concomitant medical conditions, often resulting in cognitive impairment⁴⁸ and disability.⁴⁹ As a number of these conditions are often associated with SCI, these individuals may be at increased risk, as compared to their able-bodied counterparts, for accelerated brain aging and subsequent cognitive aging.

In an effort to identify the nature of the cognitive deficits in persons with SCI, we examined neuropsychological test performance in persons with traumatic SCI compared to both age matched non-SCI healthy controls (AMHC) and a group of older non-SCI healthy controls (OHC). We hypothesized that the neuropsychological performance of persons with SCI would significantly differ from AMHC, with no difference noted between persons with SCI and OHC.

Methods

Participants: Participants consisted of 60 individuals with chronic SCI, including 32 individuals with paraplegia (T2–T12) and 28 individuals with tetraplegia (C3–T1). Thirty (30) age-matched healthy controls (AMHC; 30–40 years old) and 20 older healthy controls (OHC; 50–60 years old) were additionally included; group characteristics may be found in [Table 1](#). The upper age limit in the OHC sample was limited to age 60 to minimize the potential confound of an ongoing dementing processes. All participants were proficient in English and demonstrated visual acuity of at least 20/60 in the worst eye (with prescription eyewear). All participants with SCI were non-ambulatory (used a wheelchair ≥ 40 hours/week) with American Spinal Injury Association Impairment Scale (AIS) grade A, B or C and were at least 1 year post-injury. Potential participants were excluded in the presence of acute illness or infection or a documented history of chronic hypertension, diabetes mellitus, TBI, stroke, epilepsy or seizure disorders, multiple sclerosis, Parkinson's disease, psychiatric disorders (post-traumatic stress disorder, schizophrenia, bipolar disorder), illicit drug abuse within the past 6-months, Alzheimer's disease and dementia. Individuals scoring ≤ 22 the Mini Mental Status Examination (MMSE),⁵⁰ adapted to eliminate any motor requirements (i.e. changed 3-step command to non-motor tasks, oral sentence generation instead of written, and visuoconstruction task changed to visual discrimination), were also excluded.

Participants with SCI were recruited first. Individuals with SCI were recruited from the Northern New Jersey

Table 1 Demographic and disease characteristics by group.

	SCI N = 60	AMHC N = 30	OHC N = 20	Statistic	P
Age (years)	35.38 (7.01) range: 25–48	35.73 (7.35) range: 26–49	60.3 (2.68) range: 55–64	$F(2,107) = 117.07$	<0.001 ^a
Education (years)	13.65 (2.24)	15.50 (1.70)	16.15 (2.46)	$F(2,107) = 13.71$	<0.001 ^b
Ethnicity (% Caucasian)	48.3%	50%	90%	$\chi^2(14, N = 110) = 20.17$	0.125
Age at injury	25.55 (6.61) range: 16–45	n/a	n/a	n/a	
Years since injury	9.83 (7.32) range: 1–31				
Level of Injury	C3-T1: n = 32 T2-T2: n = 28	n/a	n/a	n/a	
WASI Vocabulary T Score	48.46 (9.80)	55.97 (8.67)	60.30 (11.02)	$F(2,107) = 13.37$	<0.001 ^b
WASI Matrix Reasoning T Score	46.95 (10.24)	52.47 (8.45)	57.15 (9.20)	$F(2,107) = 9.51$	<0.001 ^b

SCI, spinal cord injury; AMHC, age matched healthy controls; OHC, older healthy controls.

^aSCI, AMHC < OHC; P < 0.05 based on ANOVA with LSD post-hoc.

^bSCI < AMHC, OHC; P < 0.05 based on ANOVA with LSD post-hoc.

SCI Model (NNJSCIS) System database, posted flyers at support groups and inpatient and outpatient treatment facilities. The AMHC and OHC participants were recruited from the local community, hospital personnel and flyers. AMHC and OHC were then matched to the SCI subjects for: sex, race, smoking status, socioeconomic status, IQ, and education (+/– 1 year of educational attainment), to the best of our ability. Participants with SCI were screened for level and severity of injury based on prior evaluation using the International Standards for the Neurological Classification of Spinal Cord Injury (ISNCSCI).⁵¹

There was no significant difference between the SCI group and the AMHC group in age. By definition, there was a significant difference between the OHC group and both the SCI group and the AMHC group ($F(2,107) = 117.14$, $P < 0.001$). There was additionally a significant difference between the groups in level of education, with the SCI group having less education than both HC groups ($F(2,107) = 13.71$, $P < 0.001$).

Pre-morbid IQ was assessed with the 2-subtest version of *Wechsler Abbreviated Scale of Intelligence (WASI)*⁵² which consists of the Vocabulary (verbal IQ; vocabulary and verbal knowledge) and Matrix Reasoning subtests (nonverbal reasoning). The WASI has shown excellent internal consistency reliability, and construct validity has been consistently supported by high intercorrelations between the WASI subtests and WAIS-III IQ scales (range: 0.66–0.92) and factor analyses.⁵³ Significant differences were noted between the groups on the IQ estimates, with the SCI group performing at significantly lower levels than both HC groups [WASI Vocabulary: ($F(2,106) = 13.67$, $P < 0.001$); WASI

Matrix Reasoning: ($F(2,107) = 9.50$, $P < 0.001$), which remained statistically significant after entering education as a covariate (Vocabulary: $F(2,106) = 3.53$, $P < 0.05$; Matrix Reasoning: $F(2,105) = 4.70$, $P < 0.05$). Education was a significant covariate in both statistical models (both Ps < 0.05). There were no significant differences between the groups on measures of depression (Chicago Multidimensional Depression Inventory) or anxiety (State Trait Anxiety Inventory).

Procedures: This was a prospective, cross-sectional investigation conducted at the Kessler Foundation (KF) in West Orange, NJ and supported by investigators at the James J. Peters Veterans Affairs Medical Center (JJPVAMC) in Bronx, NY. Institutional Review Board approvals were obtained at both institutions and informed consent was obtained prior to initiating study procedures. Complete assessment consisted of cardiovascular/cerebrovascular evaluation (heart rate [HR], finger blood pressure [BP], cerebral blood flow velocity [CBFv], and brachial BP at rest), which were collected while the subject was resting in the seated position and during neuropsychological testing. The neuropsychological test battery took approximately 2–2.5 hours to administer and included tasks of memory, attention, working memory, information processing speed and executive functioning. The current paper focuses on neuropsychological test performance; the cardiovascular and cerebrovascular data has been submitted for publication elsewhere.⁵⁴

Neuropsychological Assessment: A broad-based neuropsychological assessment was administered by a trained research assistant, who was approved for testing via a three-step process including two

postdoctorally-trained neuropsychologists. Test choice was limited by the upper limb motor paralysis in participants with tetraplegia. Thus, all measures were motor-free.

Digit Span, Wechsler Intelligence Scale–III (WAIS-III) assessed attention and working memory.⁵³ Each segment (forward and backward) consists of seven pairs of random number sequences that the examiner reads aloud at the rate of one per second. In the digit span forward segment, the subject repeats the sequence in the same order presented. Conversely, in the backward segment, the subject repeats the sequence in the reverse order. WAIS III Digit Span has also shown high internal consistency reliability ($r = .90$) and high construct validity.⁵³

Symbol Digit Modalities Test (SDMT) – oral version⁵⁵ assessed information processing speed. The examinee substitutes a number for a randomized presentation of a geometric figure. The appropriate number is shown in a key containing the Arabic numbers 1–9, each with a different geometric figure. The SDMT has shown good test-retest ($r = .76$) and alternate forms ($r = .82$, $r = .84$) reliability. The sensitivity of the SDMT to cognitive effects has been demonstrated repeatedly in populations such as learning disabled children, commissurotomy patients (full and frontal), adults with cerebrovascular disease, chronic brain lesions (e.g. TBI), Huntington’s Disease, and Multiple Sclerosis (MS).⁵⁶ The SDMT has also been shown to be able to distinguish between individuals with depression and those with an organic dementia.⁵⁵

Letter-Number Sequencing, (WAIS-III LNS)⁵⁷ assessed auditory working memory. Individuals sequentially order a series of random numbers and letters orally presented in a specified random order. The subjects must first remember the numbers and letters and then reorganize the numbers in ascending order and the letters in alphabetical order. WAIS-III LNS has shown high internal consistency reliability ($r = .90$), construct validity and differential sensitivity to a variety of neurocognitive disorders.⁵³

The California Verbal Learning Test-II⁵⁸ assessed verbal new learning and memory. This test consists of a list of 16 words from 4 semantic categories presented orally over 5 trials and includes a 20-minute delayed recall trial as well as a recognition trial. The CVLT-II has demonstrated good reliability and validity, high internal consistency reliability ($r = .94$), split half reliability ($r = .83$), test-retest reliability ($r = .82$) and construct validity.⁵⁸

The Paced Auditory Serial Addition Test (PASAT)⁵⁹ assessed information processing, including speed and

working memory. The participant adds verbally presented randomized single digits so that each digit is added to the one immediately preceding it. Fifty digits are presented in each of 4 trials via audio recording, with the 4 trials varying in speed of presentation, differing by 0.4 seconds each (1 digit every 1.2 seconds to 1 digit every 2.4 seconds). Performance is evaluated by calculating the number correct or percent correct on each trial. The PASAT has shown good split half reliability ($r = .96$)⁶⁰ and high internal consistency.⁶¹ Factor analytic studies have shown the PASAT to load with other measures of information processing.⁶² It has also been shown to be sensitive to mild concussions,^{63,64} diffuse cerebral damage⁶⁵ and very sensitive to deficits in information processing ability.⁶²

The Verbal Fluency subtest of the Delis-Kaplan Executive Function System (D-KEFS: Delis et al.)⁶⁶ was administered to assess the fluency aspect of executive functioning. This test requires verbal generation of words in 60 seconds to the letter prompts “F”, “A”, and “S”, as well as to the semantic categories of “animals” and switching between “fruits” and “furniture”.

Data Analysis: Statistical analyses were performed using SPSS software (Version 21.0; IBM Corporation, Armonk, NJ, USA) and continuous data are reported as mean \pm standard deviation. Analysis of Covariance (ANCOVA) with group as the between subjects variable (SCI, AMHC, OHC) was conducted for scores on each neuropsychological test to examine differences between the groups within the specific cognitive domains of attention, working memory, information processing speed, verbal new learning and memory and verbal fluency. Raw scores (instead of normatively-corrected scores) were utilized for all analyses to facilitate the identification of patterns of performance between AMHC and OHC based on actual performance rather than a decline from previous functioning. Years of education was utilized as a covariate in all analyses due to significant group differences. LSD were calculated for pair-wise comparisons where appropriate. Significance was set at an alpha level of $P \leq 0.05$, and analyses were well-powered to detect medium effect sizes ($1 - \beta = 0.83$ for effect size $f = 0.31$).

Results

Information processing speed

A trend for significance was noted between the groups on the SDMT ($F(2,106) = 2.53$, $P = 0.08$). Post-hoc LSD indicated a significant difference between the AMHC and the OHC groups ($P < 0.05$; Hedge’s $g = 0.52$; medium effect) and a trend was noted between

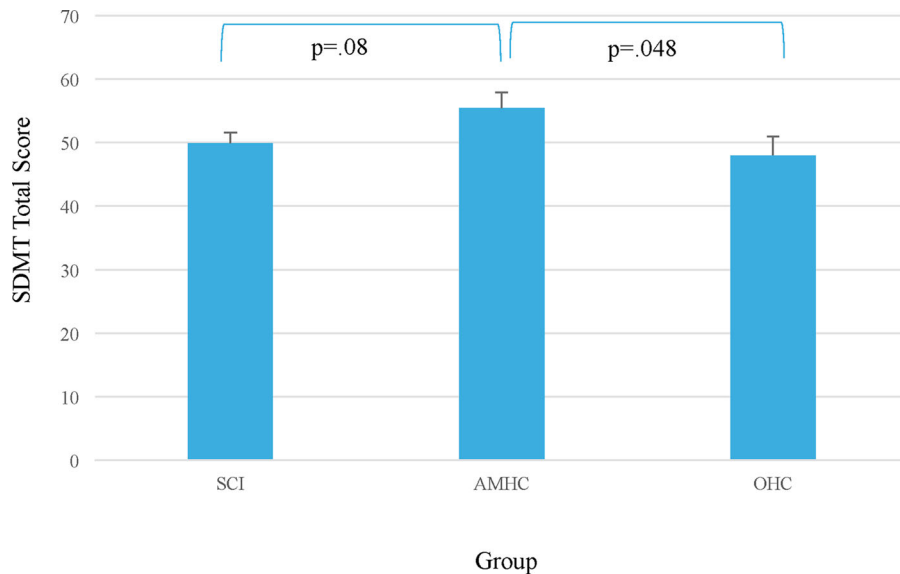


Figure 1 Information processing speed by group, as determined by the Symbol Digit Modalities Test – Oral Version.

the SCI group and the AMHC group ($P = 0.07$; Hedge’s $g = 0.55$; medium effect; see Fig. 1).

Attention and working memory

No significant group differences were noted between the groups on the PASAT ($P = 0.22$), letter number sequencing ($P = 0.80$) or Digit Span (total, forward or backward; all P s > 0.10).

Verbal learning & memory

In examining performance on a verbal list learning task, the CVLT-II, several significant differences were noted between the groups. After controlling for education (ns), there was a significant difference between the groups noted on the Trial 1 score ($F(2,106) = 4.59$, $P < 0.01$). Post-hoc LSD indicated significant differences between the SCI group and the AMHC ($P < 0.01$; Hedge’s $g = 0.79$; large effect). The difference

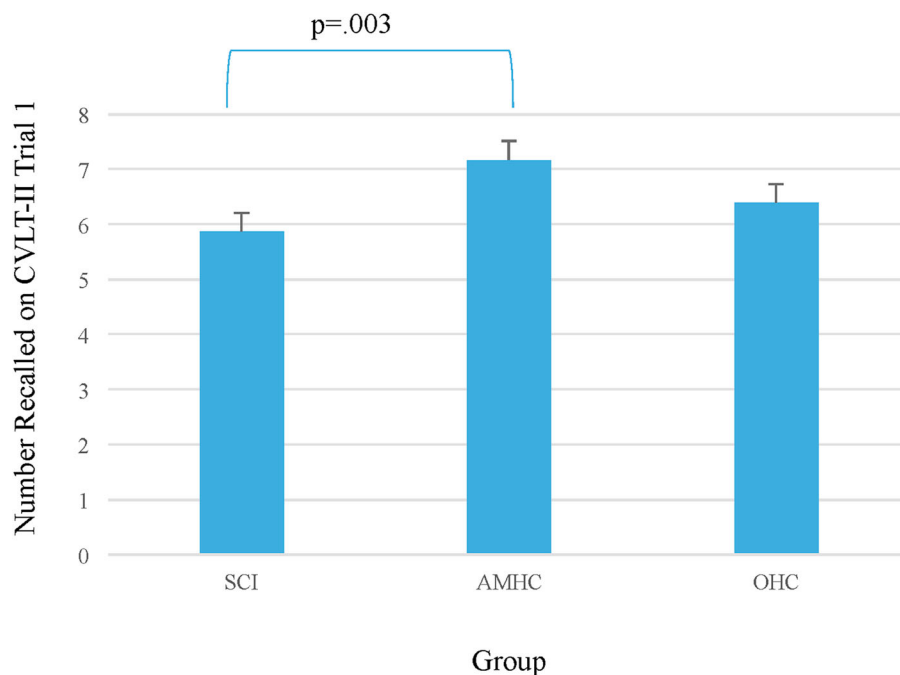


Figure 2 One-trial verbal learning by group, as determined by the California Verbal Learning Test – Second Edition, Trial 1.

between the SCI group and the OHC was not significant (Fig. 2).

After controlling for education (ns), there was also a significant difference between the groups noted on short delay free recall ($F(2,106) = 3.30$, $P < 0.05$). Post-hoc LSD tests indicated a significant difference between the SCI group and the AMHC ($P < 0.05$; Hedge's $g = 0.69$; medium-large). There was a trend toward a significance difference between groups on the CVLT-II Total Learning Score (Trials 1–5; $F(2,106) = 2.453$; $P = 0.09$), with a significant difference evident between the SCI group and the AMHC group ($P = 0.03$; Hedge's $g = 0.66$; medium-large effect). There was no significant difference between the SCI and OHC groups. There was no significant difference noted on the raw long delay free recall ($P = 0.28$).

Verbal fluency

A trend was noted between the groups for the letter fluency total score ($F(2,106) = 2.61$, $P = 0.08$) after controlling for variance associated with education ($F(1,106) = 7.42$, $P = 0.008$). Significant group differences were noted between the SCI group and AMHC ($P < 0.05$; Hedge's $g = 0.80$; large effect; Fig. 3). No significant difference was noted on category fluency ($P = 0.23$).

Discussion

Results indicated several significant differences in specific domains of neuropsychological functioning in persons with SCI as compared with AMHC and OHC. Significant differences from AMHC were noted in information processing speed, verbal learning and memory and verbal fluency, in the absence of significant differences from OHC. In contrast, simple attention and working memory appeared intact in our participants with SCI.

Deficits in information processing speed were noted on the SDMT such that persons with SCI performed at significantly lower levels than AMHC, but did not differ from OHC. The SDMT is a widely-accepted measure of information processing speed in neurological populations and often yields high sensitivity to cognitive impairment in such samples.^{55,67} Indeed, cognitive slowing is one of the primary deficits observed in normal cognitive aging and is frequently observed in the context of poor white matter integrity. In contrast, all measures of working memory (PASAT, Digit Span backwards, and Letter Number Sequencing) were intact in the SCI group indicating little to no difficulties with working memory. Thus, in the current dataset, the SCI groups performed at expected levels on working

memory tests, with a clear deficit in information processing speed noted.

With regard to verbal learning and memory, the SCI group differed significantly from the AMHC but not OHC in performance on several indices of the CVLT-II. Deficits were noted on Trial 1 of the CVLT, which best reflects learning in everyday life, given the fact that daily life learning does not typically provide an opportunity for repetition of the to-be learned information.⁶⁸ This finding may indicate that persons with SCI demonstrate difficulty learning new information in daily life situations. Deficits were similarly noted (albeit at an omnibus trend level) on total learning across the 5 trials of the CVLT, indicating a deficit in the initial learning of information, even in the presence of repeated presentation of that information. Short delay free recall was similarly impaired. However, it is interesting to note that there was no significant difference between the groups on any measure of memory for the newly learned information after a 20-minute delay. This indicates that although the SCI group demonstrated difficulty with the initial learning of information, the degradation of information over time was equivalent to that observed in both the AMHC and the OHC groups. This pattern of results has significant implications for cognitive rehabilitation. That is, in the presence of an immediate learning impairment, with intact delayed recall, cognitive rehabilitation must be targeted at improving new learning abilities and attempt to improve new learning abilities specifically in individuals with SCI. This would be expected to result in an overall improvement in memory performance, as has been seen in other neurologic populations.^{69,70}

The only indices of executive functioning included in the neuropsychological battery administered assessed verbal fluency. Interestingly, significant differences were noted between the groups on the letter fluency subtest, but not the category fluency subtest. Although it is tempting to attribute deficits in verbal fluency to speed of processing, as fluency measures required rapid speed of processing, the fact that deficits were noted on the letter but not the category fluency argues against this interpretation. Historically, the rapid retrieval of words by initial letter sound has been associated with frontal systems functioning, while such retrieval organized by word category has been associated with temporal lobe functioning. This indicates that persons with SCI are demonstrating a disruption in the frontal executive retrieval system necessary to carry out the task. Indeed, this pattern is consistent with other neurological disorders that have injury patterns secondary to

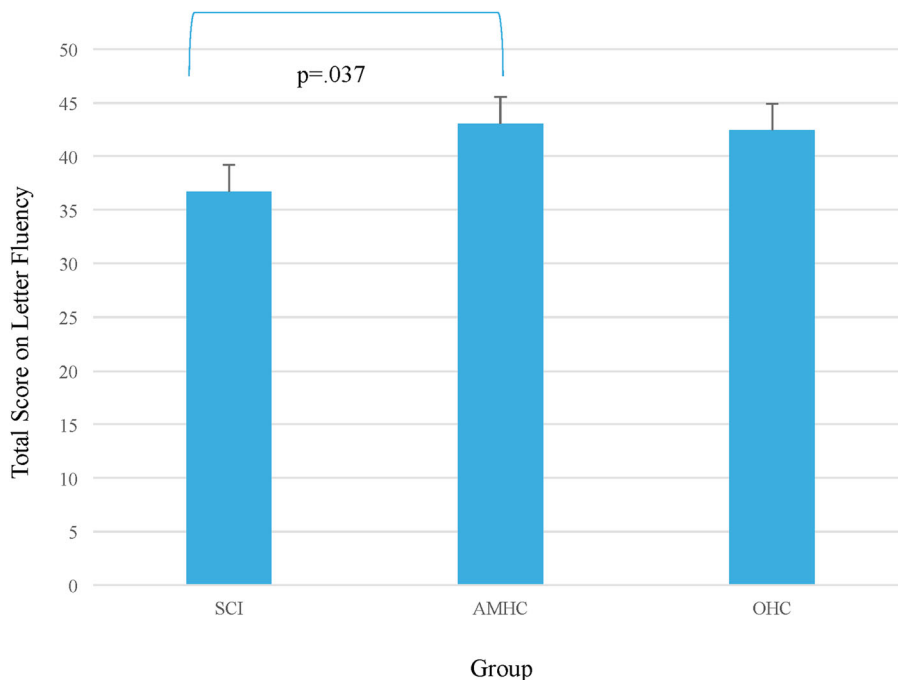


Figure 3 Verbal fluency (letter) by group, as determined by the Delis-Kaplan Executive Function System – Verbal Fluency Subtest (Condition 1: Letter Fluency).

both reduced perfusion (e.g. vascular dementia⁷¹) and traumatic injury (e.g. TBI⁷²).

Significant differences were noted between the groups in education level. This was an interesting and unexpected finding, and suggests that perhaps the educational accomplishments of the SCI sample may have been limited by the occurrence of the injury itself. Age at time of injury ranged from 16 to 45 years old (M(SD) = 25.55 (6.61); mode = 24). When examining the individual data, it is notable that of the 60 individuals with SCI included in the study sample only 1 participant reported an education level that would have been completed after the SCI was sustained. Although, the cognitive deficits noted in the SCI group compared to the AMHC are reported after controlling for years of education there is the potential that educational attainment in the SCI group was hindered by the presence of cognitive deficits in the years following the injury. Therefore, early intervention to diagnose and treat cognitive deficits in the SCI population should be considered to facilitate return to school and work and ultimately alleviate long-term cognitive dysfunction.

Beyond the documentation of the pattern of neuropsychological deficits in the current study, the inclusion of two control groups was unique and allowed a comparison of the SCI group to both an AMHC and OHC group. Of the domains assessed in this study, the

pattern of deficits in the SCI and OHC samples relative to our younger healthy control sample (AMHC) are consistent with what is consistently reported in the aging literature: dysfunction in information processing speed, new learning and memory, and executive functioning (e.g. Ref. 73; see Ref. 74 for a review). Importantly, we did not use normative scores that pre-corrected for the influence of age on the neuropsychological tests. As cognitive aging is a neurologically normal process, use of normative correction for age would have washed out the raw score differences observed between OHC and AMHC. Additionally, examination of raw scores estimates *actual ability* on a neuropsychological test, whereas normative corrections are generally utilized to help determine whether a decline from an individual's previous level of function is likely to have occurred. Given the focus on ability, uncorrected scores can be considered to be a better indicator of real life abilities on daily tasks (i.e. although a decline of 1 standard deviation may be significant for a high-functioning individual, the level of functioning may still be sufficient for performing simple instrumental activities of daily living). As such, acknowledgement of cognitive impairment in this way is important, because treatment focused on these cognitive deficits could have a substantial impact on, not only the deficit itself, but on overall functioning in daily life, including the ability to return to work and school and overall QOL.

There are several study limitations that deserve mention. First, the neuropsychological test battery employed was limited to tests with no motor component. As a result, there was limited availability of measures to reliably assess cognition in this sample. Several cognitive domains were thus not assessed including visuospatial processing, non-verbal memory, and aspects of executive control such as planning and problem solving. Additionally, brain imaging at the time of injury was not available for our participant pool. While there was no documented presence of TBI in any included participant, without neuroimaging we cannot be fully certain that mild TBI was not sustained at the time of injury. Additionally, neuroimaging techniques may more directly help to answer questions related to brain-based pathology, which may or may not confirm the hypothesis of accelerated aging. Furthermore, in order to more rigorously test the hypothesis of accelerated cognitive aging in SCI, a longitudinal study examining the relative cognitive trajectory in the SCI population (compared to their healthy counterparts) is warranted. With regard to generalization, a significant difference was noted between the groups in education. While this was appropriately managed through statistical analysis using years of education as a covariate, the ideal study would have education-matched participant groups. Finally, the relatively small sample size and strict inclusion criteria may preclude generalization of these results to the larger population of individuals with SCI.

Conclusion

Despite these limitations, the current study documented differences in specific realms of cognitive functioning between a chronically injured SCI sample and a sample of AMHC; of note there were no significant differences in cognitive performance comparing the SCI and OHC groups. Cognitive deficits were noted in information processing speed, new learning, and verbal fluency, which is consistent with normal aging. Attention, working memory and memory retrieval processed appear intact. Additional research is needed utilizing a more comprehensive battery of neuropsychological tests that are motor free in nature to avoid the confound of upper extremity motor limitations. Moreover, clinicians in the rehabilitation realm should be aware of the potential presence of cognitive deficits in individuals with SCI and how clinical interactions might be affected. As gleaned from recommendations in other neurological populations with comparable deficits, several ways to minimize the impact of cognitive impairment in treatment settings include: (1) providing information at a slower pace and

in multiple modalities (e.g. verbal and written instructions), (2) confirm understanding of medical instructions via independent recitation by the patient, and (3) encouraging use of compensatory strategies to enhance encoding of information to be learned (e.g. spaced self-testing) (see Ref. 75).

Disclosure statements

Conflicts of interest The authors report no conflicts of interest.

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