Published in final edited form as:
PM R. 2011 May ; 3(5): 426–432. doi:10.1016/j.pmrj.2011.02.018.

Pervasive Cognitive Impairment in Acute Rehabilitation Inpatients Without Brain Injury

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Abstract

Objective—To establish feasibility for the hypothesis that patients in acute rehabilitation who are hospitalized for disorders not known to involve cerebral injury can have significant cognitive impairment.

Design—A comparison of performances on neuropsychological tests between 2 samples of subjects: inpatients in an acute rehabilitation hospital without known cerebral disease and normal community-dwelling persons.

Setting—Acute inpatient rehabilitation hospital.

Patients and Participants—Nineteen hospitalized patients without delirium who were screened for pre-existing cerebral and psychiatric illness, dementia, and dependency in basic self-care skills before hospitalization. Eighteen community-dwelling persons who were not different in terms of age and education served as the control group.

Methods—Participants completed 10 commonly used neuropsychological tests of executive, language, and memory functions. Data were analyzed by using multivariate analysis of variance.

Main Outcome Measurements—Raw scores on the 10 neuropsychological tests.

Results—Hospitalized patients performed significantly worse on 9 of 10 tests than community-dwelling participants. Older hospitalized participants had significantly greater cognitive impairment than younger hospitalized participants, which suggested increased susceptibility to effects of hospitalization on cognition.

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Peer reviewers and all others who control content have no relevant financial relationships to disclose.

Disclosure Key can be found on the Table of Contents and at www.pmrjournal.org

Conclusions—Patients hospitalized without brain injury, and especially elderly patients, should be carefully monitored for cognitive deficits that may affect posthospitalization quality of living. Further research is needed to determine whether the cognitive deficits in such patients persist after discharge and affect functional independence, and to identify mechanisms for the deficits. Furthermore, the use of hospitalized participants without brain injury as control subjects in neuropsychological studies of brain injury should be balanced with an additional comparison group of matched, neurologically healthy, normal subjects who live in the community to control for cognitive impairments that are associated with acute hospitalization.

INTRODUCTION

Treatment approaches used in acute inpatient rehabilitation programs are guided, in part, by whether a patient is known to have cognitive impairment secondary to brain injury. However, the same consideration may not be extended to patients without known brain injury when cognitive function is assumed to be intact. Our observations suggest that this assumption is probably incorrect for several reasons. Many hospitalized patients receive analgesic medications that can alter arousal and cognitive function [1]. Furthermore, although certain types of noncerebral surgery for disabling illnesses, including carotid endarterectomy, cardiac bypass surgery, hip arthroplasty, artery bypass graft, and thoracic surgery, have been reported to be associated with cognitive deficits [2,3], treating physicians nonetheless may underdiagnose cognitive deficits in hospitalized patients without brain injury because such associations are not commonly recognized [4]. Recognizing cognitive status is important because it may affect rehabilitation outcome and the transfer of rehabilitation skills to the home after discharge. Results of a number of studies indicate that patients with comorbid cognitive impairment, with various medical illnesses, who were discharged to home without supervision, had an increased risk of further injury, illness, decreased functional outcome, and increased use of assisted living facilities in the future [5–7]. Finally, not only are medical disorders themselves associated with cognitive decline, but also the number of such disorders may predict the extent of cognitive impairment [8–10].

We conducted a pilot study to examine the feasibility for the hypothesis that patients in acute rehabilitation who are hospitalized for disorders not known to involve cerebral injury can have significant cognitive impairment. We tested this hypothesis by comparing the neuropsychological test performances of a sample of 19 patients hospitalized for inpatient rehabilitation for disorders other than known cerebral disease with those of a comparison group of 18 community-dwelling persons of comparable age and education. Surprisingly few studies have specifically examined whether hospitalized patients without known brain injury can perform normally on common bedside tests of cognitive function. We undertook this investigation because we had informally observed at our institution that general clinical staff were only minimally noting such potentially pervasive cognitive deficits among patients who appeared to us, as neuropsychological investigators, to be considerably cognitively impaired. This discrepancy may, in turn, reflect general trends in health care, with broad consequences for medical care in general.

METHODS

Participants

Nineteen hospitalized participants (11 women and 8 men) without known brain injury and loss of consciousness related to their illness were recruited from an acute inpatient rehabilitation hospital. At our institution, “brain injury” was diagnosed by the patients’ admitting physicians from their evaluation of available brain imaging results that depicted structural alterations attributable to injury or from the medical histories. Consequently, we considered patients not to have known brain injury if there were no indications of brain

illness in our institutions' hospital records or self reports from patients who were referred for this study. Eleven patients had been admitted after orthopedic surgery or injury (eg, fractures, amputation). The remaining 8 patients had undergone peripheral vascular surgery (n = 1) or had a gunshot wound to the back (n = 2), spinal cord injury (n = 3), or lymphoma (n = 2). The patients were referred by their treating physicians, who knew about the study from announcements during faculty meetings. The physicians were asked to refer to the study those patients that they judged to be cognitively intact and healthy enough to participate in testing. Patients were considered eligible for study if they were medically stable and considered by their physicians to be competent to provide informed consent. In addition, they were required to have been functionally independent until the time of hospitalization. Some practical limitations, such as whether a participant had the time or wanted to participate in the study, influenced our ability to enroll patients consecutively. Participants were not compensated for study participation. They ranged from 24 to 85 years of age. Eighteen nonhospitalized controls (12 women and 6 men), who were similar in age and education, were recruited from a local community fitness center that was not a skilled living or elderly care center. Nonhospitalized controls were recruited via word of mouth and comprised both members and family members of persons who attended community or fitness events at the center. Nonhospitalized controls were recruited after completion of testing the hospitalized group. A testing appointment was scheduled for interested participants if they were of age and educational level similar to those of a participant from the hospitalized group.

Materials

Each participant completed a short battery of bedside tests chosen for common use, brief administration time, and sensitivity to deficits in executive and memory functions. Tests included the following: trails A and B of the Trail Making Test (time for test completion in seconds), Controlled Oral Word Association Test (COWAT), Animal Naming (AN) Test, the Mini-Mental State Examination (MMSE), immediate and delayed recall of the Logical Memory Immediate (LM I) and Logical Memory Delayed (LM II), and Visual Reproduction Immediate (VR I) and Visual Reproduction Delayed (VR II) subtests of the Wechsler Memory Scale—Revised, and a modified Star Cancellation Test (time for test completion in seconds) [11,12]. Raw test scores were used in subsequent statistical analyses.

Procedure

After study enrollment, the participants were evaluated for previous neurologic injury by reviewing available medical records (hospitalized participants only), self-reported medical histories, and administration of the Canadian Study of Health and Aging Older Americans Resources and Services (OARS) questionnaire [13]. Subjects were excluded if the history indicated pre-existing cerebral illness or dependence in basic self-care skills before hospitalization. The participants were required to have no history of psychiatric disorder or dementia. They furthermore were required not to have been documented by hospital staff to have markedly fluctuating arousal and attention that would suggest delirium for most clinicians [14]. We recorded (1) clinically obtained, routine vital signs from the day of testing, (2) any basic blood test results (sodium, potassium, glucose, hematocrit, and white blood cell count) that were obtained by hospital staff according to physicians' orders within 2 days of testing, and (3) medications that could potentially affect arousal for cognitive function that were administered on the day of testing, to preliminarily evaluate possible influences on cognitive test results that are associated with routine hospitalization. Hospitalized participants did not overtly demonstrate decreased arousal at the time of testing. Hospitalized participants were tested at the bedside with possible sources of environmental interference removed (eg, television off, door closed). Non-hospitalized controls were tested in a quiet room at the community center. They were evaluated for

previous neurologic injury by self-reported medical histories and administration of the Canadian Study of Health and Aging OARS questionnaire. Nonhospitalized controls were required to have no history of pre-existing cerebral illness, psychiatric disorder, dementia, or dependence in basic self-care skills. Laboratory results and vital signs data were not collected in the nonhospitalized control group because no qualified technician was available at the fitness center to obtain them. The order of test administration was counter-balanced across participants. The test battery took 50–60 minutes to administer. The study protocol was approved by our institutional research review board for human subjects, and all subjects provided written informed consent to participate before study procedures.

Statistical Analysis

Differences between groups were evaluated by using multivariate analysis of covariance, with the raw scores of the 10 cognitive tests as dependent variables and group as the fixed factor between-subjects variable. We also evaluated the potential effect of age as a covariate of cognitive impairment in hospitalized patients. Education was also included as a covariate to control for any variation introduced by slight differences in the education level of hospitalized participants versus nonhospitalized controls.

RESULTS

Participants in each group were not significantly different from each other in age (hospitalized participants mean [standard deviation {SD}] 53.8 ± 20 years, nonhospitalized controls mean [SD] 53.3 ± 14.9; $t = 0.37$, $P = .71$) or in education (hospitalized participants mean [SD] 12.2 ± 2.2 years, nonhospitalized controls mean [SD] 13.5 ± 2.9; $t = -1.7$, $P = .1$). Demographic variables, rehabilitation diagnoses, vital signs, blood test results, and cognitively relevant medications of the hospitalized participants are provided in Table 1. Available vital signs and blood test values were generally normal for the patients except that at least half of the patients had elevated glucose readings and white blood cell counts, and most patients were anemic. At the time of cognitive testing, all the patients, except one, were being treated with at least one medication that could potentially affect arousal or cognitive function (primarily analgesic medication or medications for ulcer prophylaxis).

Hospitalization status significantly affected cognitive performance ($F_{11,24} = 4.46$, $P = .001$, partial η^2 [ηp^2] = 0.65; when multiplied by 100, partial ηp^2 can be interpreted as the percentage of variance accounted for by an effect and its associated error variance). After controlling for effects of age and education, hospitalized participants were significantly impaired relative to controls on 9 of the 10 cognitive measures (Table 2). LM I was the only cognitive measure unaffected by hospitalization status. Analyses indicated a large effect size for the 9 significantly affected cognitive measures (Table 2). In addition, age was found to be a significant covariate ($F_{11,24} = 2.90$, $P = .01$, $\eta p^2 = 0.55$) of group-based performance differences, with statistically significant effects on the MMSE, Trails B, and LM I and LM II tests (Table 2). Education was not a significant covariate of group-based performance differences ($F_{11,24} = 1.26$, $P = .3$, $\eta p^2 = 0.34$), although LM I and LM II demonstrated sensitivity to variation in education (F's$_{1,34} > 6.2$, P's $< .05$, ηp^2's > 0.16).

The test performances of hospitalized participants generally fell below normal limits when compared with published age- and education-relevant normative standards (number of hospitalized participants per test with values below published normative standards: 5 MMSE, 10 Trails A, 14 Trails B, 4 LM I, 7 LM II, 9 VR I, 7 VR II, 17 COWAT, 10 AN) [15]. In contrast, the test performances of controls generally fell within, or only occasionally below, normal limits when compared with published normative standards (number of healthy controls per test with values below published normative standards: 1 MMSE, 2 Trails A, 4 Trails B, 1 LM II, 4 VR I, 3 VR II, 4 COWAT, 3 AN) [15]. Limits were

determined by using published age and education appropriate means ± 1.5 SD. This cutoff approach was used in place of the published MMSE, COWAT, and AN cutoffs because the values produced were more conservative and provided less chance for false positive findings of risk. Age- and education-appropriate norms were not available for the modified star cancellation test. Only age-appropriate norms were available for LM and VR. By using the above distribution of impaired performance, we calculated the risk ratio for impaired performance between the hospitalized and nonhospitalized groups on each of the remaining 9 cognitive tests administered. Five of the 9 cognitive tests analyzed produced risk ratios that were significant (Table 2). The MMSE, VR I, and VR II did not produce significant risk ratios greater than a value of 1. A risk ratio could not be calculated for Logical Memory I because none of the healthy controls performance fell below the mean ± 1.5 SD cutoff. Averaged normative standards are also provided in Table 2. These were calculated by finding each hospitalized participant's mean and variance for his or her age and education stratum, and then averaging these normative means and variances. On average, healthy controls demonstrated average performance scores comparable to published values, except for the Trail Making Test part B and COWAT. In both cases, healthy controls had slightly poorer performance scores than published values, and, as a result, our findings for significant deficits on both tasks may be underestimated in the present study.

DISCUSSION

Findings from this pilot study support the feasibility for the hypothesis that inpatients without known brain injury can exhibit significant cognitive impairment. Inpatients who did not have known brain injury or cerebral disease performed significantly worse on 9 of 10 cognitive tests than did community-dwelling subjects who were not different from patients in terms of age and education. The effect size to discriminate between groups was large (Cohen's $d > 0.8$) in all 9 significantly affected tests. Finally, 5 of 9 tests with available age and education normative data produced risk ratios significantly larger than one, which suggests that cognitive deficits had a significantly higher risk of occurrence in the hospitalized participants than in the nonhospitalized controls on tests that evaluate executive abilities, visual search, delayed verbal memory, and verbal fluency.

The reasons for cognitive impairment, in this study or in the few other studies that reported cognitive deficits in inpatient populations without brain illness [16] and after surgical procedures [1] are not well understood. It is likely that cognitive impairment in patients hospitalized for reasons other than brain injury can have multiple causes [9], including possible undetected occult secondary cerebral effects of acute bodily trauma [17–19] (eg, diffuse axonal injury that leads to cognitive impairment as a secondary effect of trauma), residual effects of intraoperative complications [20], medication, depression, anxiety, persistent and postoperative pain, and diverse physiologic variables (eg, nutrition, hydration, hematocrit) that may be greatly altered among hospitalized participants [1]. Although complete laboratory values were not available for all patients, in those for whom laboratory values were available hematocrit (8 of 10 patients), glucose (7 of 13 patients), and peripheral white blood cell (4 of 8 patients) levels were outside the normal range. Because the laboratory tests were not conducted on all of the patients within 2 days of cognitive testing, we cannot speculate on the laboratory variables for the entire patient population and thus whether abnormal laboratory values could have been a factor that contributed to cognitive impairment. However, numerous studies have correlated either abnormal hematocrit or glucose levels with cognitive impairment [21–25]. It therefore remains possible that the abnormal hematocrit and glucose values in our study contributed to cognitive impairment in our patients.

Given the pervasiveness of their cognitive impairment, affecting executive, memory, and language functions, and the absence of any identified focal or diffuse brain injury to explain such deficits, it seems possible that impaired performance could somehow have been related to impaired arousal associated with surgery or medications, although the patients were attentive during our cognitive assessments (Table 1). Because we did not record the concurrent medications of the control participants, we cannot speculate on whether the patients' medications alone had significantly affected their cognitive test findings, as opposed to an interaction between such medications and other factors related to hospitalization (eg, abnormal laboratory findings, decreased socialization, emotional impact of illness). Furthermore, both anesthesia and analgesics have been associated with cognitive impairment and decreased arousal [1]. We do not think that overt delirium or depression accounted for the poor performance in our hospitalized participants, because both factors were exclusionary; however, they could be contributory. The decision of whether a patient exhibited delirium or depression was made by the treating physician rather than by formal assessment with standardized tests. Because the incidence of delirium may be elevated among the type of hospitalized patients that we selected, it will be important that future studies investigate the possible causes of cognitive impairment, such as delirium and depression. This study indicated that cognitive impairment is surprisingly frequent in patients who are not suspected of having brain injury without identifying specific causes for impairment.

Alfano and Satz [26] and Satz et al [27] have proposed a study design for use with head-injured patients, which is validated in part by this study. The design advocates comparing patients with brain injury with 2 control groups similar to those used in this study (ie, a community-dwelling healthy control group and a non–brain-injury group of individuals with a specific illness, such as patients hospitalized for orthopedic injury). Impairment, therefore, can be interpreted as consequent to brain injury versus caused by effects not judged to result from brain injury, including treatment effects (eg, procedures and medications), systemic physiologic effects (eg, anemia, hyperglycemia), or behavioral reaction to illness or confinement (eg, depression). The present study supports the use of designs such as this to control for cognitive effects associated with hospitalization. The pattern of cognitive impairment demonstrated in our hospitalized group suggests that cognitive studies that intend to control for potential physiological and environmental factors related to hospitalization, surgery, non–brain-related injury, and so forth, should consider using an acute hospitalized control group as an appropriate control for such variables rather than a nonhospitalized control group. This design would decrease the potential for a type I error in detecting a cognitive difference between groups. Alternatively, use of a nonhospitalized control group would be appropriate in studies that explicitly intend to compare the cognitive function of a hospitalized population to healthy community-dwelling individuals.

Results of our study suggest that it is not appropriate to assume normal cognitive function in patients without known brain injury. However, future studies are needed to address the limitations of this pilot study. For example, studies with larger samples of subjects and with control over factors such as medication and medical conditions would help relate such factors to cognitive function. Furthermore, future prospective studies that collect complete laboratory values across both hospitalized and nonhospitalized participants would help to determine the possible roles of hematocrit, glucose, and peripheral white blood cell count. Our study might have had selection biases. Our sample could actually underrepresent the prevalence of cognitive impairment in the acute inpatient rehabilitation setting, because our patient recruitment approach required physicians to recruit participants whom they deemed cognitively intact. Other selection biases might also be operative without our knowledge. Future studies that stratify samples based on demographics, medications, and medical conditions will be important for identifying the factors that contributed to the cognitive

impairment observed in the present study. Furthermore, further research should be undertaken to determine whether deficits in non–brain-injured hospitalized participants eventually improve after hospital discharge; whether neuroimaging findings can identify non–brain-injured hospitalized participants who are at risk for cognitive impairment; and whether medications or other treatments (eg, transfusions) might offset or improve cognitive dysfunction in hospitalized participants.

Effects of Aging

We found that age was a significant covariate of group-based differences in cognitive ability on 4 of the 10 cognitive tests administered (Table 2). Cognitive decline with normal aging is well documented in the research literature. Our finding that older hospitalized participants performed worse than age-matched nonhospitalized controls suggests that older hospitalized patients may be more susceptible to cognitive impairment than younger hospitalized participants.

Clinical Implications

Increased risk of further injury and illness associated with cognitive impairment are a concern when considering discharge arrangements for inpatients [5,6]. Because the hospitalized patients performed worse on diverse cognitive tests than did functionally independent, community-living adults who were matched for age and education, it would appear to be important to cognitively assess inpatients without brain injury before discharge. Furthermore, these results demonstrate the potential utility of uniform cognitive screening among all inpatient rehabilitation admissions. Such evaluation could help to inform patients and family members of possible cognitive limitations that may appear after discharge. In addition, such evaluations might be useful adjunctive information to recommendations made by other therapies when determining whether inpatients can be safely discharged home alone. However, further studies are needed to evaluate the predictive value of inpatient cognitive assessment for subsequent real-world functional independence and safety.

CONCLUSION

Whereas one might assume that cognitive function is intact among inpatients who do not have known brain injury, our pilot study indicates the opposite is true. Routine assessments of cognitive function can be performed efficiently [28]. The battery from this study required 1 hour to complete and was sensitive to differences in cognitive status between inpatients and community-living participants. Routine cognitive assessment could benefit discharge planning as part of a multidisciplinary rehabilitation approach [29,30].

Acknowledgments

The authors thank Edwin Cook III, PhD, George Jewel, PhD, Michael Marsiske, PhD, Michael DeVivo, and Richard Allman, MD, for their helpful comments on our study.

Research support: This work was supported in part by the National Institutes of Neurological Disorders and Stroke (T32NS007413, NS068910), the John A. Hartford Foundation/Southeast Center of Excellence in Geriatric Medicine, the National Institute on Aging (AG 21256), the National Institute of Child Health and Human Development (HD055269, HD055677), the National Multiple Sclerosis Society (RG 4221), and the National Center for Research Resources (RR20146, 1UL1RR029884). Submitted for publication May 24, 2010; accepted February 24, 2011.

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Table 1

s, vital signs, laboratory values, and arousal-altering medications for hospitalized participants

Demographics			Vitals					Laboratory Tests					
Age, y	**Gender**	**Medical Diagnosis**	**Temp, °C**	**Pulse**	**Resp, breaths/min**	**BPS, mm Hg**	**BPD, mm Hg**	**Na, mg/dL**	**K, mg/dL**	**Glucose, mg/dL**	**HC, % plasma volume**	**WBC, 1000s/mL**	**Administered Medications That Could Have Affected Arousal**
24	M	GSW to back	35.5	59	22	95	39*	—	—	—	—	—	None
24	F	Lymphoma	36.6	80	18	110	60	140	4.2	104	29*	5.1	Fentanyl patch, oxycodone, hydromorphone, zolpidem
26	M	SCI	35.7	77	20	149*	66	142	3.6	115*	39	11.1*	Fentanyl
31	F	SCI	37	89	18	112	86	—	—	—	—	—	Fentanyl patch, baclofen, diazepam
32	M	Lymphoma	36.6	102	20	80*	59	—	—	—	—	—	Desipramine, levetiracetam, propranolol, oxycodone
38	F	Amputation	36.1	95	18	116	70	138	4.3	84	29*	7.8	Hydrocodone
42	M	Amputation	36.8	87	20	134	74	138	4.8	122*	36*	11.9*	Oxycodone, prednisone, ranitidine
44	F	GSW to back	36.1	86	20	117	64	139	4.6	102	34*	5.07	Sertraline, gabapentin, methocarbamol, diphenhydramine, odansetron, gabapentin
48	M	Amputation	36.8	89	20	113	91*	—	—	104	—	—	Hydromorphone, gabapentin, morphine, prednisone
53	F	Vasc surg	36.3	78	20	162*	88	134	4.4	273*	35*	13.2*	Zolpidem
62	F	Multiple fract	35.9	79	20	130	66	—	—	—	—	—	Oxycodone, ranitidine
62	F	Hip arthr	36.7	78	20	117	58	—	—	—	33*	—	Amitriptyline, diazepam, prednisone, ranitidine, oxycodone
67	M	Knee arthr	36.0	109	20	138	68	142	4.0	115*	—	—	Diphenhydramine, ranitidine
68	F	Hip arthr	35.6	91	20	146*	64	133	3.5	157*	—	—	Hydrocodone, ranitidine
71	F	Knee arthr	37.3	68	20	109	51	—	—	—	—	—	Ranitidine
72	F	Knee arthr	36.5	76	20	138	59	—	—	—	—	—	Zolpidem, buspirone, ranitidine, propoxyphene
74	M	SCI	36.1	73	20	135	62	138	3.5	87	29*	14.4*	Baclofen, pseudoephedrine
78	F	Tibia-fibula fract	36.4	82	20	122	55	142	4.4	108*	31*	—	Zolpidem, loratidine, fluoxetine, ranitidine, hydrocodone
85	M	Tibia fract	—	—	—	—	—	—	—	343*	41	8.7	Ranitidine

Demographics			Vitals					Laboratory Tests					
Age, y	Gender	Medical Diagnosis	Temp, °C	Pulse	Resp, breaths/min	BPS, mm Hg	BPD, mm Hg	Na, mg/dL	K, mg/dL	Glucose, mg/dL	HC, % plasma volume	WBC, 1000s/mL	Administered Medications That Could Have Affected Arousal
53.8			36.3	83.2	19.7	123.5	65.5	138.6	4.1	145.4	33.6	9.7	
20.0			0.5	11.9	1.0	20.0	13.0	3.2	0.5	84.2	4.2	3.5	
			35–37.8	45–115	8–25	90–140	45–90	133–145	3.3–5.1	70–105	39–50	4–11	

re; Resp = respiratory rate; BPS = blood pressure systolic; BPD = blood pressure diastolic; Na = sodium; K = potassium; HC = hematocrit; WBC = white blood cell count; GSW =
CI = spinal cord injury; Amputation = unilateral leg amputation; Vasc surg = vascular surgery; fract = fracture; arthr = arthroplasty;

ange.

ue was not available (for vital signs) or that a blood test was not performed within 2 days of cognitive assessment; chart data were unavailable for subject no. 19.

Table 2

Group difference on cognitive tests and effect of aging

	HP (n = 19)	NHC (n = 18)	Mean Norms Data						Effect of Aging		
Cognitive Tests	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	$F_{(1,34)}$	P	ηp^2	d	**RR (95% CI)**	$F_{(1,34)}$	P	ηp^2
MMSE	27.3 ± 2.8	29.3 ± 0.7	28.1 ± 1.6	10.22	.003*	0.24	1.09	4.74 (0.83–30.1)	7.22	.011*	0.18
Trails A	53.2 ± 20.4	37.5 ± 13.3	34.1 ± 11.4	6.48	.016*	0.16	0.97	4.74 (1.45–18.1)*	1.92	.175	0.06
Trails B	160.6 ± 66.4	93.3 ± 41.4	76.4 ± 23.3	14.15	.001*	0.30	1.36	3.32 (1.52–7.59)*	6.31	.022*	0.15
LM I	20.8 ± 6.7	25.3 ± 7.2	23.6 ± 7.4	2.81	.103	0.08	0.76	×†	7.60	.015*	0.17
LM II	15.2 ± 6.4	22.3 ± 6.7	18.9 ± 9.3	12.54	.001*	0.28	1.36	6.63 (1.26–40.6)*	15.24	.001*	0.30
VR I	22.1 ± 9.8	32.1 ± 7.1	29.3 ± 5.9	10.78	.002*	0.25	1.19	2.13 (0.85–5.75)	0.13	.757	<0.01
VR II	18.8 ± 11.1	27.7 ± 9.5	24.8 ± 7.4	5.32	.028*	0.14	0.88	2.21 (0.74–7.23)	0.52	.549	0.01
COWAT	21.5 ± 8.1	33.0 ± 9.5	39.3 ± 11.5	16.52	<.001*	0.33	1.39	4.03 (2.01–6.60)*	2.53	.110	0.08
AN	13.5 ± 3.9	18.6 ± 5.0	18.7 ± 4.2	13.02	.001*	0.28	1.15	3.16 (1.16–9.62)*	0.15	.833	<0.01
MSC	56.2 ± 17.9	33.5 ± 6.5	na	21.59	<.001*	0.40	1.71	na	0.59	.536	0.12

HP = hospitalized participants; NHC = nonhospitalized controls; Mean Norms Data = averaged age and education appropriate normative means and variance for the HP group; ηp^2 = partial η^2; d = effect size (0.75 or higher = large; >0.4 = medium; <0.4 = small) calculated as Cohen's d; RR = risk ratio or relative risk for impaired performance between groups; 95% CI = 95% confidence interval; MMSE = the Mini-Mental State Examination; LM I = Logical Memory Immediate Recall; LM II = Logical Memory Delayed Recall; VR I = Visual Reproduction Immediate Recall; VR II = Visual Reproduction Delayed Recall; COWAT = Controlled Oral Word Association Test; AN = Animal Naming; MSC = Modified Star Cancellation Test; na = not available.

*Statistical significance (P < .05).

†No RR calculation was possible because of a lack of risk occurrence in the NHC group.