Spring 2010

From Blood Pressure to Physical Disability: The Role of Cognition

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Abstract—We examined the hypothesis that lowered cognitive performance plays a role in the relation between elevated blood pressure and physical disability in performing basic physical tasks. A community-based sample (N=1025) free from stroke and dementia (mean age: 61.1 years; SD: 13.0 years; 59.8% women) was used. Using path analysis, systolic and diastolic blood pressures (predictor variable) measured over multiple longitudinal examinations were averaged and related to multiple measures of cognition (intermediate variable) and physical ability (PA; outcome variable) measured at wave 6 of the Maine-Syracuse Study. PA was indexed by time required to execute standing, walking, and turning tests. A best-fit path model including blood pressure and multiple demographic and cardiovascular disease covariates was used. Paths from systolic blood pressure to global performance, verbal memory, and abstract reasoning (Similarities test) were significant ($P<0.05$), as were paths from diastolic blood pressure to global performance, executive functioning, visual spatial organization/memory, verbal memory, working memory, and abstract reasoning. Regardless of the blood pressure predictor, lower cognitive performance (intermediate variable) was related to lower PA (outcome) in the path from blood pressure to PA. The direct path from blood pressure to PA was significant only for systolic blood pressure. Cognitive performance mediates between blood pressure and PA. As compared with systolic blood pressure, more domains of cognitive functioning intervene between diastolic blood pressure and PA. **(*Hypertension*. 2010;55:1360-1365.)**

Key Words: blood pressure ■ cognitive performance ■ physical ability ■ physical disability ■ cardiovascular disease

Hypertension is one among a number of cardiovascular disease (CVD) risk factors for the development of functional disability.[1,2] Recently, Hajjar et al[3] examined the relation between blood pressure (BP) levels (in millimeters of mercury) and functional disability using self-report of activities of daily living data available from the Charleston Heart Study. They found that increases in remote and concurrent systolic BP (SBP), but not diastolic BP (DBP), were associated with greater functional disability.[3] This linear association was seen with statistical adjustment for self-reported comorbidities, including diabetes mellitus, CVD, and arthritis, and, thus, established a dose-response relation between SBP and disability.

We extend the work by Hajjar et al[3] to a consideration of objective measures of primary physical abilities (ie, standing, walking, and turning), which are strong and robust predictors of functional disability in activities of daily living[4,5] and mortality.[6] Positive associations between cognitive ability and physical functioning have been established,[5,7,8] as have inverse associations between BP and cognition,[9–12] but the hypothesis that cognition mediates the association between BP and physical functioning[3] has not been formally tested in a study using a comprehensive battery of cognitive tests.

Our group[13] proposed path analysis as a means of exploring whether cognitive ability intervenes between BP and physical ability (PA); a study using this method of analysis has not been reported. As with all regression-based techniques, path analysis neither requires nor establishes causality or a prospective design.[14] Rather, it is a systematic and simple way to evaluate plausibility of a set of hypothesized relationships among variables for a given data set.[14] Using path analysis, an extension of multiple regression, we asked questions that cannot be clearly addressed in a single regression model: does cognitive ability intervene between BP and PA, and, if so, do some specific cognitive domains play more important roles than others? On the basis of the fact that multiple brain regions, in association with the frontal regions, have parallel effects on a variety of cognitive abilities and PAs, such as walking, standing, and turning,[7] we hypothesized that multiple cognitive domains lie in the path between BP and PA, that is, BP→cognition→PA.

Hajjar et al[3] hypothesized that relations between SBP and self-reported functional disability were mediated by either executive functioning (EF) or fluid cognitive ability, a broader cognitive construct. Neither hypothesis has been formally tested, but a more recent latent profile analysis by

Received January 3, 2010; first decision January 30, 2010; revision accepted March 29, 2010.

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The content is solely the responsibility of the authors and does not necessarily represent the official views of the funding institutes.

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Hypertension **is available at http://hyper.ahajournals.org** **DOI: 10.1161/HYPERTENSIONAHA.110.149823**

Hajjar et al[15] indicated that EF ability but not verbal memory clustered (correlated) with impaired gait and depressed mood in elderly individuals. Using path analyses we were able to evaluate the role of these and other cognitive domains (composites of specific ability) in the association between BP and PA. Given that interventions to improve cognitive functioning and PA may benefit by focusing on specific cognitive abilities, it is important to know which cognitive skills[16] play a role in relations between BP and PA.

Methods

Sample and Design

With the exception of BP values, taken from measurements (described below) within the first 6 waves (serial replications) of the Maine-Syracuse Longitudinal Study, all measures, including cognition and PA, were cross-sectional and taken from the sixth serial repetition (wave) of the Maine-Syracuse Longitudinal Study, a community-based study of CVD risk factors and cognition begun in Syracuse, New York, in 1974. Recruitment and data collection procedures for wave 6 (2001–2006) have been described in detail previously.[17,18] PA data were collected for the first time at wave 6. The ability to stand, walk, and turn was a requirement for the analyses performed in the present study. Fifteen participants failed to meet this criterion. Of the 1066 participants meeting study criteria, participants were excluded in the following sequence: (1) history of stroke (n=28); (2) probable dementia (n=8); and (3) dialysis (n=5), leaving a final sample of 1025.

Stroke, defined as neurological deficit of acute onset persisting >24 hours, was based on self-report and was confirmed by a record review indicating a diagnosis of acute stroke. The clinical diagnosis of dementia was based on cognitive data and medical charts, using the National Institute of Neurological and Communicative Diseases and Stroke/Alzheimer Disease and Related Disorders Association criteria.[19]

Procedure

After a fast from midnight, a blood sample for diagnostic assays was drawn in the morning and followed by a light breakfast and interview (including medical history). Subsequently, after supine rest for 15 minutes, 5 reclining, 5 standing, and 5 sitting automated BP measurements (GE DINAMAP 100DPC-120XEN, GE Healthcare) were obtained sequentially with a 5-minute interval between each set of measurements. Neuropsychological testing and then PA testing followed the BP measurements. All of the assay methods used to derive data on comorbidities and methods for identifying comorbidities have been described previously.[17,18,20]

Predictor Variable

The independent variable was the average of SBP or the average of DBP measures obtained for each participant using data from wave 1 through wave 6 (15 measurements per wave). Following Hajjar et al,[3] we adjusted for antihypertensive treatment at each examination using the Cui et al[21] method confirmed by Tobin et al[22] of adding 10 mm Hg to SBP and 5 mm Hg to DBP in participants being treated with antihypertensive agents at the time of BP assessments. These adjustments are as effective as complex algorithms.[21,22] Because only individuals with high BP receive treatment, statistical adjustment of observed BP values for antihypertensive medications by covariance analysis is ill advised.[21,22] It artificially reduces variability associated with BP, but only for those with the highest underlying BP values, and violates the assumption that predictors and residuals are independent.

Because it can be argued that the amount of exposure to high BP, regardless of treatment, is critical to lowered cognitive performance, secondary analyses were performed with BP values unadjusted for treatment. In all of the analyses, SBP and DBP are expressed in units of 10 mm Hg.

Covariates

Diabetes mellitus was defined by treatment with insulin, oral glucose-lowering agents, or by a fasting glucose level of ≥7 mmol/L (126 mg/dL).[23] Participants completed the Center for Epidemiological Studies Depression Scale[24] within 1 week before neuropsychological testing. Depressed mood was defined as a Center for Epidemiological Studies Depression Scale score >16 (clinical cut score).[24] Results were the same when adjusted for continuously distributed Center for Epidemiological Studies Depression Scale scores.

Additional covariates used in various analyses included age (years), education (years), sex, ethnicity (black versus nonblack), body mass index (in kilograms per meter squared), self-report of number of cigarettes smoked per week, alcohol consumption (grams per week), self-report of arthritis, and CVD confirmed by medical records and/or treatment. As in the Framingham Heart Study,[25] CVD was defined as the presence of any 1 of the following: (1) myocardial infarction (4.5%); (2) coronary artery disease (8.8%); (3) heart failure (2.4%); (4) angina pectoris (5.9%); or (5) transient ischemic attack (4.1%).

Dependent Variables

A composite measure of PA was created from 3 timed tests found in previous investigations[4,5] to be most predictive of activities of daily living dependence at 1 year: (1) walking back and forth over a 10-foot course; (2) turning in a full circle; and (3) standing up and sitting down from a hard-backed chair 3 times with arms folded. Participants were asked to perform each of these tasks as quickly as possible, and time required to complete the tasks was measured to the nearest hundredth of a second. Simply adding scores for the 3 tests gave too little weight to tasks that could be performed more quickly. Consequently, the times for walking, turning, and standing were standardized by transforming them to z scores and were then added. The resulting sums were then transformed again to z scores to obtain a composite measure of PA. Signs on the z scores were reversed so that poor PA was expressed as a negative regression coefficient.

Intermediate Variables: Cognitive Domains

We used the Similarities subtest from the Wechsler Adult Intelligence Scale[26] and 4 composite test scores derived from a previous factor analysis of individual tests in the Maine-Syracuse Longitudinal Study battery for this study population.[17,18] The 4 composite scores were visual-spatial organization and memory (visual reproductions immediate, visual reproductions delayed, matrix reasoning, block design, object assembly, and the Hooper visual organization test), scanning and tracking (trails A and B, digit symbol substitution, and symbol search), verbal memory (logical memory immediate and logical memory delayed and the Hopkins verbal learning test), and working memory (digit span forward and backward, letter-number sequencing, and controlled oral word associations). The Similarities subtest was used as a separate measure because it loaded on multiple composite scores in the previous factor analyses[17] and is highly correlated with general verbal intellect.[26] Detailed descriptions of the individual tests are summarized in Table S1 (available in the online Data Supplement, please see http://hyper.ahajournals.org).

The EF composite (trails B+controlled oral word associations) was constructed specifically for this study using widely agreed on measures of EF.[27] To construct the composite scores, the individual tests constituting each composite were expressed in z scores and added (after necessary transformations of trails A and B scores to satisfy normality assumptions). The composite scores were the resulting sums also transformed to z scores. This linear transformation results in a mean of 0 and an SD of 1.00 for each test or composite and enables expression of regression coefficients for the cognitive measures in terms of SD units. In addition to composite scores, a global composite score was calculated by adding the z scores for all of the individual tests used in each composite and then restandardizing this distribution to a mean of 0 and an SD of 1.00.

Statistical Analyses

Analyses were conducted in 2 phases. The first phase, multivariate regression analysis, was performed to verify that the relations between BP and self-report functional disability[3] would be replicated in the present study using PA and the Charleston Heart Study covariate set (full covariate set). The model used for this analysis (full model) was as follows: BP+age+education+sex+depressed mood+diabetes mellitus+ethnicity+arthritis+smoking+CVD+body mass index+alcohol consumption. The second phase, path analysis, was performed with the goal of testing the mediating role of cognition using the most parsimonious subset of predictors. Beginning with the full set of covariates, backward elimination (α=0.05) was used to determine which predictors of cognition and PA needed to be included in a preliminary path model. Next, nonsignificant paths in this model were fixed at 0 (removed) to obtain a final model. Overall fit for the final model was then evaluated by standard fit indices representing measures of population discrepancy (χ^2) and both incremental comparative fit index and absolute indices, goodness-of-fit index and root mean square error of approximation. We estimated additional models with different covariate sets and with and without elimination of nonsignificant associations, and the substantive conclusions were unaffected.

The University of Maine Institutional Review Board approved the protocol for this investigation. Informed consent for data collection was obtained from all of the participants.

Results

Preliminary Analysis

Sample characteristics are summarized in Table 1. Preliminary analyses indicated that the distributions of the BP scores, cognitive scores, and the disability measures were suitable for multiple regression and path analyses. All of the cognitive variables were correlated significantly (all $P<0.001$; r range: 0.39 [similarities and working memory] to 0.91 [global and EF]). See r matrix in Table S2.

Linear Regression Analysis: BP and PA

With adjustment for the full covariate set (defined above), multiple linear regression analysis indicated that treatment-adjusted SBP but not DBP was significantly associated with poorer PA scores (SBP [10 mm Hg]: $b=-0.036$, SE=0.015, $P=0.02$; DBP [10 mm Hg]: $b=-0.034$, SE=0.025, $P=0.18$). The same results were obtained with unadjusted BP as the predictor (SBP [10 mm Hg]: $b=-0.032$, SE=0.017; $P=0.05$; DBP [10 mm Hg]: b=−0.024, SE=0.027, $P=0.38$).

Path Analysis

After backward elimination, significant predictors of cognition were age, education, sex, ethnicity, arthritis, depressed mood, smoking, and diabetes mellitus. Significant predictors of PA were age, education, sex, ethnicity, arthritis, depressed mood, CVD, and body mass index. Path models using these variables fit the data very well (eg, $\chi^2[4]=2.4$, $P=0.80$, goodness-of-fit index: 0.9996, comparative fit index: 1.0000, root mean square error of approximation: 0.0000 for the global composite with SBP in the model and for variants of the model with DBP and the other cognitive scores). However, examination of the path model regression coefficients indicated that the direct path from education to PA was nonsignificant for the global composite ($P=0.61$), as well as other cognitive scores used in the indirect path (all $P>0.05$) used in separate analyses. Consequently, the path from education to PA could be fixed at 0 (deleted) in the final model without adversely affecting model fit.

Table 1. Demographic and Comorbidity Characteristics of the Sample at Wave 6 and BP Values at Waves 1 to 6

Variable	Mean or %	SD
Unadjusted SBP, mm Hg*	130.4	19.0
Unadjusted DBP, mm Hg*	73.7	10.9
Adjusted SBP, mm Hg†	133.7	21.0
Adjusted DBP, mm Hg†	75.4	11.7
Age	61.1	13.0
Education	14.6	2.7
BMI, kg/m^2	29.4	6.0
Alcohol, g/wk	35.9	75.0
Smoking, cigarettes per week	10.3	40.4
Total cholesterol, mmol/L	5.17	1.05
HDL, mmol/L	1.39	0.40
LDL, mmol/L	3.09	0.87
Triglycerides, mmol/L	1.58	1.25
Women, %	59.8	
White, %	86.2	
Diabetic, %	12.8	
Arthritis, %	46.9	
Depressed Mood, %	11.4	
CVD, %	12.4	
Hypertension, %	60.2	
Antihypertensive medication, %‡	81.0	
Obese, %	39.1	

HDL indicates high-density lipoprotein; LDL, low-density lipoprotein.

*Data show waves 1 to 6.

†Data show waves 1 to 6, adjusted for antihypertensive treatment at each examination using the method of Cui et al.[21]

‡Data show the percentage of hypertensive participants on antihypertensive medication.

The Figure shows a general form of the final model. This was the best-fitting model for SBP and DBP regardless of the cognitive measure used in the model. For example, across models the goodness-of-fit index ranged from 0.9991 to 0.9997 for both SBP and DBP. Details on additional fit statistics for the final model for each cognitive variable may be found in Table S3. All of the paths in the final models were statistically significant ($P<0.05$) or marginal ($P<0.10$) for the global composite. Marginal paths from ethnicity and arthritis were not deleted because they were significant in models with other cognitive variables. Table S4 shows the path models for the covariates for the global composite score.

Table 2 shows results for the direct and indirect paths starting from treatment-adjusted SBP (increments of 10 mm Hg). Regression coefficients for the SBP→cognition paths were significant for the global composite, verbal memory, and Wechsler Adult Intelligence Scale similarities but not statistically significant for the other cognitive variables. All of the cognition→PA and direct SBP→PA paths were statistically significant ($P<0.05$), with the exception of the SBP→PA path with the global composite included in the model ($P<0.06$).

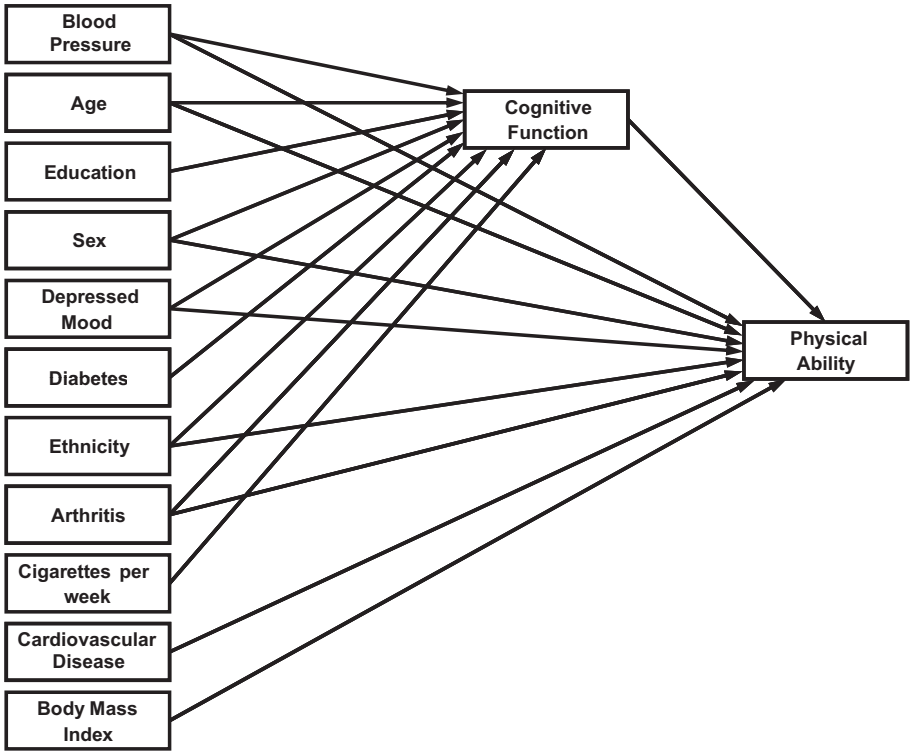

Figure. General form of the final path model. All of the exogenous variables (in this case, BP and the covariates) were permitted to covary. These covariance paths and error terms are not shown on this simplified diagram.

As can be seen in Table 3, regression coefficients for the DBP→cognition paths were significant ($P<0.05$) for the global composite, EF, visual-spatial organization and memory, verbal memory, working memory, and similarities. The cognition→PA paths were all significant ($P<0.05$), but none of the direct paths from DBP to PA were significant.

Results for all of the analyses were the same for BP values unadjusted for treatment and when examination 1 to 5 SBP or DBP values were substituted for examination 1 to 6 values in secondary analyses (mean years between examinations: 5.1; SD: 0.8).

Table 2. Path Coefficients (b) and SEs for Paths Beginning With SBP (10 mm Hg) by Cognitive Variable Included in the Model

		Indirect Path		Direct Path
Cognitive Variable	Measure	SBP→Cognition→PA		SBP→PA
Global composite	b	−0.027*	0.230†	−0.028§
	SE	0.013	0.033	0.015
Executive function	b	−0.019	0.161†	−0.035*
	SE	0.015	0.030	0.015
Visual-spatial organization/memory	b	−0.023‡	0.231†	−0.029*
	SE	0.014	0.031	0.015
Verbal memory	b	−0.030*	0.090‖	−0.038‖
	SE	0.015	0.029	0.015
Working memory	b	−0.025	0.066*	−0.039‖
	SE	0.016	0.028	0.015
Scanning and tracking	b	−0.009	0.224†	−0.034*
	SE	0.013	0.033	0.015
Similarities	b	−0.031*	0.151†	−0.034*
	SE	0.014	0.029	0.015

*$P<0.05$.
†$P<0.001$.
‡$P<0.10$.
§$P<0.06$.
‖$P<0.01$.

Table 3. Path Coefficients (b) and SEs for Paths Beginning With DBP (10 mm Hg) by Cognitive Variable Included in the Model

		Indirect Path		Direct Path
Cognitive Variable	Measure	DBP→Cognition→PA		DBP→PA
Global composite	b	−0.064*	0.235†	−0.019
	SE	0.022	0.033	0.025
Executive function	b	−0.054‡	0.164†	−0.030
	SE	0.025	0.030	0.025
Visual-spatial organization/memory	b	−0.054‡	0.236†	−0.022
	SE	0.023	0.031	0.024
Verbal memory	b	−0.053‡	0.093*	−0.038
	SE	0.026	0.029	0.025
Working memory	b	−0.068*	0.068‡	−0.039
	SE	0.027	0.028	0.025
Scanning and tracking	b	−0.023	0.227†	−0.032
	SE	0.022	0.033	0.025
Similarities	b	−0.069*	0.154†	−0.029
	SE	0.025	0.029	0.025

*$P<0.01$.
†$P<0.001$.
‡$P<0.05$.

Discussion

As predicted by the Hajjar et al[3] study of physical disability, SBP but not DBP was associated with PA in the multivariate analysis model. In addition, using the best-fit path model we again found that SBP did but DBP did not have a direct path to PA.

Results from path analysis confirm the hypothesis that several different cognitive domains mediate between SBP and PA in stroke- and dementia-free individuals who are physically capable of performing walking, standing, and turning tasks. The regression coefficients in the path between SBP and cognition were statistically significant for the global composite, verbal memory, and similarities (abstract reasoning). However, additional cognitive measures were associated with DBP. Diastolic BP→cognitive path coefficients were significant for the global composite, EF, visual-spatial organization and memory, verbal memory, working memory, and similarities. The regression coefficients for SBP and DBP are not directly comparable in a clinical context because a rise of 10 mm Hg for SBP may have different implications than a rise in 10 mm Hg for DBP. However, for all but one of the cognitive variables (verbal memory), the regression coefficients for DBP were more than double the values for SBP.

In studies with older individuals, SBP has predicted a greater number of cognitive abilities than DBP.[10] However, in a number of studies with younger participants or for samples with a broad age range (as in our study), more cognitive abilities have been related to DBP than to SBP.[11,12] We had insufficient power to exclude individuals <50 years of age (younger adults) in the present study and also to meet power requirements for path analysis. Additional path analysis studies with elderly individuals are indicated.

Each of the measures of cognition was related to PA (cognition→PA) regardless of whether SBP or DBP was used in the model. There are 2 possible explanations, among others: intellectually capable individuals are more likely to make decisions and actions leading to better health, including lower BP and less disability, and/or multiple regions of the brain exert parallel influences over PA and cognition, such that injury results in impairments of both functions.[7,28–31] Many of these same brain injury mechanisms appear to play an important role in relations between BP and cognition.[9–12] This literature is consistent with our finding of associations among BP, cognition, and PA, reflected in the BP→cognition→PA paths.

In contrast to the Hajjar et al[15] study of BP and PA, we did not find a relation between SBP and executive performance or fluid abilities (visual-spatial organization and memory and working memory). We did find these associations for path analyses with DBP. It is not clear why the pattern of cognitive deficits was different for SBP and DBP, although this phenomenon has been reported in previous studies.[9–12] The brain injury mechanisms affecting cognition may be different for SBP and DBP.[11]

In our study the SBP→verbal memory→PA path was significant, as were the DBP→verbal memory and DBP→working memory paths. This finding is consistent with reviews indicating that damage to the hippocampus results in poorer gait and memory impairment, as well as poorer performance in other cognitive domains.[7,29,31] Moreover, it is clear that physical abilities are affected by brain regions other than the hippocampus,[7,28,31] a finding that is also consistent with our findings that multiple cognitive domains intervene between DBP and PA.

Absence of a relation between BP and verbal ability in the study by Hajjar et al[3] may relate to the fact that they defined verbal ability via a single clinical measure. Thus, we advocate further studies in which cognitive domains are indexed by multiple clinical tests.

Hajjar et al[3] suggested a number of possible brain injury mediators between BP and disability aside from cognitive functioning: white matter hyperintensities in the brain, cerebrovascular function, overall lean muscle mass, inflammation, or change in the renin-angiotensin system. Most of these have also been suggested as brain injury mechanisms intervening between BP and cognition.[11] Path analysis and structural equation modeling provide future opportunities to investigate these associations.[13]

Limitations of the present study were as follows. Except for BP assessments, the design was cross-sectional. There is a need for longitudinal analyses with a temporal separation between measurements of cognition and PA. Correlations among the cognitive scores precluded an examination of tasks with unique demands on ability. This high "cognitive congruence" phenomenon is a robust characteristic of cognitive studies reflecting true associations among various domains of functioning in human cognition and the fact that clinical tests measure abilities in addition to what they are designed to measure (test impurity).[31–34] Effect sizes and regression coefficients are modest with respect to BP associations with cognition within the BP→cognition→PA path, but the data have implications at a population level and in terms of the construction and testing of theories involving relations among BP and PA.

In summary, although DBP may play an unimportant role in direct paths to PA in the work by Hajjar et al[3] and in the present study, both DBP and SBP play an important role in the path to PA via cognition. In this context, global performance and several cognitive domains were related to SBP; a greater number of cognitive domains were related to DBP.

Perspectives

Remedial treatments and prevention strategies are important concerns for treatment of BP-related cognitive and PA disabilities and for disabilities involving other CVD risk factors. Prevention of hypertension, aggressive treatment, and BP control are important and obvious intervention strategies. Moreover, there is evidence that remedial training of cognitive skills can improve cognition, activities of daily living, and PA.[16] Clinical trials will be necessary to determine whether remedial training of cognitive skills is an efficient approach to reducing hypertension-related physical disability. The targeting of fluid and EF abilities is a reasonable but not optimal strategy, because multiple cognitive domains were related to DBP. Moreover, verbal ability and abstract reasoning ability were related to SBP and DBP.

It is possible that lower intellect in general results in less than optimal or poor lifelong decisions, leading to higher prevalence and incidence of physical disability. Our data

suggest that improvement of cognitive skill performance, via education, may work to decrease physical disability related to elevated BP. Although education was unrelated to PA in the direct path from education to PA (education→PA), education plays an important role in cognition, a variable that intervenes between BP and PA. Thus, education and specific skill training are relevant treatment interventions for BP-related decrements in PA.

Acknowledgments

We thank Amanda L. Goodell and Suzanne Brennan, Hypertension Research Project University of Maine, for their help with data acquisition, data management, and copy editing.

Sources of Funding

This study was supported in part by the National Heart, Lung, and Blood Institute (grants HL67358 and HL81290) and the National Institute on Aging (grant AG03055) of the National Institutes of Health.

Disclosures

The salaries for M.F.E., M.A.R., and P.K.E. were paid from research grants from the National Heart, Lung, and Blood Institute (HL67358 and HL81290). The salary for A.D. was paid from a research grant from the National Heart, Lung, and Blood Institute (AG13180) and he was a consultant for the Study of Aging Amsterdam, Vrije Universiteit.

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ONLINE SUPPLEMENT

FROM BLOOD PRESSURE TO PHYSICAL DISABILITY: THE ROLE OF COGNITION

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Short Title: Blood pressure, cognition and disability

Total Word Count: 5960

Abstract Word Count: 246

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Table S1. Descriptions of the cognitive tests contributing to each composite score indexing a cognitive domain*.

Test Composite/ Tests Included in the Composite	Cognitive Ability Measured
Verbal Episodic Memory	
Logical Memory-Immediate Recall†	Immediate memory, verbal
Logical Memory-Delayed Recall†	Delayed memory, verbal
Hopkins Verbal Learning Test	Verbal learning and memory
Visual-Spatial Organization/Memory	
Visual Reproductions-Immediate Recall†	Immediate recall, visual memory, and visual-spatial problem solving
Visual Reproductions-Delayed Recall†	Delayed recall, visual memory and visual-spatial problem solving
Matrix Reasoning‡	Abstract reasoning and pattern recognition
Block Design§	Visual-spatial perception, organization and construction
Object Assembly§	Speed of visual-spatial organization
Hooper Visual Organization	Visual-spatial organization; some demands on executive function
Scanning and Tracking	
Trail Making A‖	Visual scanning and tracking; concentration and attention
Trail Making B‖	Trails A plus demands on executive function abilities
Digit Symbol Substitution§	Psychomotor performance
Symbol Search‡	Visual processing speed
Working Memory	
Digit Span Forward§	Attention and concentration
Digit Span Backward§	Attention, concentration, and working memory
Letter-Number Sequence‡	Information processing while holding information in memory
Controlled Oral Word Associations	Verbal fluency and executive functioning

Test Composite/ Tests Included in the Composite	Cognitive Ability Measured
Executive Function	
Trail Making B‖	Trails A plus demands on executive function abilities
Controlled Oral Word Associations	Verbal fluency and executive functioning
Similarities§	Verbal intelligence and abstract reasoning

*The tests employed in each composite score/domain define the abilities measured by that domain.
†Origin Wechsler Memory Scale-Revised
‡Origin Wechsler Adult Intelligence Scale III
§Origin Wechsler Adult Intelligence Scale
‖Origin Halstead-Reitan Neuropsychological Test Battery

Table S2. Pearson product moment correlations among the cognitive variables.

	Global	EF	VSOM	VM	WM	ST	Similarities
Global							
EF*	0.91						
VSOM†	0.89	0.90					
VM‡	0.71	0.53	0.53				
WM§	0.72	0.62	0.47	0.41			
ST‖	0.84	0.77	0.69	0.46	0.51		
Similarities	0.64	0.61	0.58	0.46	0.39	0.41	

All correlations are significant ($p < .001$)

*EF = Executive Function

†VSOM = Visual-Spatial Organization/Memory

‡VM = Verbal Memory

§WM = Working Memory

‖ST = Scanning and Tracking

Table S3. Fit statistics by BP and cognitive variables included in the model.

		BP Variable	
Cognitive Variable	Fit Statistic	SBP	DBP
Global Composite	Chi-square	2.4	2.2
	GFI*	0.9996	0.9997
	CFI†	1.0000	1.0000
	RMSEA‡ (90% CI)	0.0000 (n/a, 0.0282)	0.0000 (n/a, 0.0262)
Executive Function	Chi-square	1.3	1.7
	GFI	0.9998	0.9997
	CFI	1.0000	1.0000
	RMSEA (90% CI)	0.000 (n/a, 0.0111)	0.0000 (n/a, 0.0201)
Visual-Spatial Organization	Chi-square	6.0	6.0
	GFI	0.9991	0.9991
	CFI	0.9994	0.9994
	RMSEA (90% CI)	0.0144 (n/a, 0.0480)	0.0139 (n/a, 0.0478)
Verbal Memory	Chi-square	4.7	5.3
	GFI	0.9993	0.9992
	CFI	1.0000	0.9998
	RMSEA (90% CI)	0.0000 (n/a, 0.0425)	0.0071 (n/a, 0.0449)
Working Memory	Chi-square	4.0	5.0
	GFI	0.9994	0.9992
	CFI	1.0000	1.0000
	RMSEA (90% CI)	0.0000 (n/a, 0.0392)	0.0031 (n/a, 0.0441)

		BP Variable	
Cognitive Variable	Fit Statistic	SBP	DBP
Scanning & Tracking	Chi-square	1.8	2.0
	GFI	0.9997	0.9997
	CFI	1.0000	1.0000
	RMSEA (90% CI)	0.0000 (n/a, 0.0224)	0.0000 (n/a, 0.0244)
Similarities	Chi-square	2.8	3.3
	GFI	0.9996	0.9995
	CFI	1.0000	1.0000
	RMSEA (90% CI)	0.0000 (n/a, 0.0315)	0.0000 (n/a, 0.0354)

*GFI = Goodness of Fit Index
†CFI = Comparative Fit Index
‡RMSEA = Root Mean Square Error of Approximation

Table S4. Estimates for path coefficients (b) and standard errors (SE) for the paths from the covariate to cognition and from cognition to PA.

		SBP		DBP	
Cognitive Variable		Covariate → Cognition	Covariate → PA	Covariate → Cognition	Covariate → PA
Age	b	-0.0392*	-0.0273*	-0.0405*	-0.0288*
	SE	0.0021	0.0028	0.0020	0.0027
Education	b	0.0136*	NP†	0.1036*	NP
	SE	0.0091	NP	0.0090	NP
Sex	b	0.1311‡	-0.1162§	0.1079§	-0.1122§
	SE	0.0494	0.0547	0.0506	0.0562
Ethnicity	b	-1.0317*	-0.1666‖	-1.0137*	-0.1661‖
	SE	0.0831	0.0999	0.0834	0.1003
Arthritis	b	0.0966§	-0.1891*	0.0912¶	-0.1921*
	SE	0.0485	0.0555	0.0483	0.0555
Depressed Mood	b	-0.2418‡	-0.2119§	-0.2344‡	-0.2121§
	SE	0.0759	0.0850	0.0758	0.0852
Cigarettes per day	b	-0.0013§	NP	-0.0013§	NP
	SE	0.0006	NP	0.0006	NP
Diabetes	b	-0.2995*	NP	-0.2945*	NP
	SE	0.0727	NP	0.0723	NP
CVD#	b	NP	-0.1664§	NP	-0.1833§
	SE	NP	0.0830	NP	0.0829
BMI**	b	NP	-0.0194*	NP	-0.0214*
	SE	NP	0.0048	NP	0.0047

*p< .001
†NP = no path (path set to zero)
‡p< .01
§p< .05
‖p< .10
¶p< .06
#CVD = cardiovascular disease

**BMI = body mass index

Note. The coefficient for the path from SBP to PA is: b= -0.028, SE= 0.015, p< .06. The coefficient for the path from DBP to PA is: b= -0.019, SE= 0.025, p> .10. The coefficients for the path from global to PA are as follows: SBP in model, b= -0.230, SE= 0.333, p< .001; DBP in model, b= -0.235, SE= 0.033), p < .001. Negative values on coefficients indicate poorer performance.