Published in final edited form as:
Health Psychol. 2009 September ; 28(5): 641–646. doi:10.1037/a0015215.

Blood Pressure Reactivity and Cognitive Function in the Baltimore Longitudinal Study of Aging

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Abstract

Objective—Several blood pressure indexes of autonomic dysregulation, including stress-induced blood pressure responses (i.e., reactivity), have been associated previously with stroke, silent cerebrovascular disease, and decreased cognitive function.

Design—We examined the cross-sectional relations among systolic (SBP) and diastolic (DBP) blood pressure reactivity and cognitive function in a sample of stroke- and dementia-free older adults (n=73, 53% male, 72% Caucasian, mean age=70.14 years) from the Baltimore Longitudinal Study of Aging.

Main Outcome Measures—Age, education, baseline and reactive blood pressure levels were regressed on cognitive test scores measuring the domains of attention, learning and memory, verbal functions/language skills, and perceptuo-motor speed. A Bonferroni correction was employed and results significant at the standard p<.05 level are discussed as marginally significant.

Results—After adjustment for age, education, and resting blood pressure, greater SBP reactivity was associated with poorer performance on Digits Forward (R^2 =.110, p=.007); and greater DBP reactivity was associated with poorer performance on Digits Forward (R^2 =.124, p=.003) and the Boston Naming Test (R^2 =.118, p=.008); associations with DBP reactivity and Alpha Span (R^2=.104; p=.019) and CVLT free recall short delay (R^2 =.066, p=.032) were marginally significant.

Conclusions—Greater BP reactivity was associated with poorer performance on tests of attention, verbal memory and confrontation naming. BP reactivity may be a biobehavioral risk factor for lowered levels of cognitive performance.

Keywords

blood pressure reactivity; cardiovascular reactivity; cognitive function; neuropsychology

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An extensive literature indicates that elevated blood pressure (BP) and hypertension are associated with decreased cognitive performance (Elias, Wolf, D'Agostino, Cobb, & White, 1993; Harrington, Saxby, McKeith, Wesnes, & Ford, 2000; Kilander, Nyman, Boberg, Hansson, & Lithell, 1998; Kuo, Sorond, Iloputaife, Gagnon, Milberg, & Lipsitz, 2004). Recent data suggest that, independent of resting BP levels, several BP indexes of autonomic dysregulation are related to lowered levels of cognitive function. In that regard, increased BP variability on ambulatory monitoring, nocturnal non-dipping, and stress-induced BP responses

(or BP reactivity) have been associated with poorer cognitive performance or cognitive decline (Bellelli, Pezzini, Bianchetti, & Trabucchi, 2002; Bellelli et al., 2004; Waldstein & Katzel, 2005). In a recent investigation, we found that greater stress-induced BP reactivity in a laboratory setting was associated with lower levels of performance on tests of immediate verbal memory, delayed verbal memory, and executive function explaining 3–8% of the variance in performance in these measures (Waldstein & Katzel, 2005). Abnormal diurnal BP patterning, BP variability, and BP reactivity have also been associated with increased cerebral white matter disease and silent brain infarction on magnetic resonance imaging, thus offering a biologically plausible mechanism that may link measures of autonomic dysregulation to cognitive performance (Goldstein, Bartzokis, Hance, & Shapiro, 1998; Kario, Matsuo, Kobayashi, Imiya, Matsuo, & Shimada, 1996; Waldstein et al., 2004).

In the present study, we examined the cross-sectional relations between BP reactivity and cognitive function in a stroke- and dementia-free sample of older adults to further understand what domains of cognitive function (Lezak, Howieson, & Loring, 2004) are most affected by enhanced BP reactivity. We have previously recommended use of comprehensive test batteries that tap multiple domains of cognitive function in studies of health and cognition in order to determine relevant patterns of performance difficulties (Waldstein, 2001). Here we specifically expanded upon prior assessment of attention, verbal and nonverbal memory, executive function and language skills. We hypothesized that, consistent with our prior work, increased BP reactivity would be associated with decreased performance on tests of verbal memory and executive function.

Similar to the study of traditional cardiovascular risk factors and cognitive function, many variables may confound the relation of BP reactivity to cognitive performance. Here we evaluated as potential confounders a series of variables that could affect both BP reactivity and cognitive function. These included age, education, resting BP, history of hypertension or diabetes, use of antihypertensive medications, body mass index (BMI), and smoking status.

Methods

Participants

Participants were 73 community-dwelling older adults (53% male, 72% Caucasian, mean age = 70.14 years) who were participating in the Baltimore Longitudinal Study of Aging (BLSA). BLSA participants return to the testing facility biennially for biomedical, psychological, and cognitive testing (Shock et al., 1984). Data for the present study were obtained from between 2001 and 2005 from participants invited to participate in a separate protocol investigating attentional and affective regulation in the elderly. All eligible BLSA participants were approached for this additional protocol and were included on the basis of each participant's willingness and availability to enroll based on their personal schedule while visiting the BLSA testing center, in addition to the availability of personnel to conduct the cardiovascular reactivity testing. Data are unavailable on the proportion of participants approached who were ultimately enrolled.

Participants were excluded from the protocol if they reported severe vision limitations not corrected with glasses or contact lenses, any cognitive deficits that prohibited them from comprehending the tasks (e.g., they did not know what was meant by recognition of emotions), and any peripheral disorders in the upper limbs that would prohibit the use of the BP cuff, a diagnosis of dementia including Alzheimer's disease (see Kawas et al, 2000), history of stroke (as documented by ICD codes 430–438), or other neurological disease (e.g., epilepsy). The present data analyses further excluded persons with a history of diagnosed cardiovascular disease (e.g., stroke, coronary artery disease, cardiac arrest, coronary artery bypass surgery)

with the exception of hypertension, or those with a Center for Epidemiological Studies – Depression (CES-D) score above the suggested clinical cutoff of 16 (Radloff, 1977).

To assess concurrent associations between BP reactivity and cognition, all cognitive data for the present study were obtained from the BLSA visit during which both cardiovascular reactivity and neuropsychological data were collected. A total of 81 participants completed both the cognitive testing and cardiovascular reactivity sessions. Seven participants were dropped because their CES-D score exceeded 16. One participant was dropped due to a previous stroke, thus leaving a final sample size of 73 (53% male, mean age = 70.14 years). Data on medical history and current medication use were collected either by self report during physical exam or as part of the biomedical assessment conducted during the routine BLSA visit. The biomedical assessment, cardiovascular reactivity test session and the cognitive test session, and covariate assessment occurred either on the same day or on subsequent days during a several day visit to the BLSA facility. Order of testing was not standard. Sample characteristics, baseline and task BP are displayed in Table 1.

Cardiovascular Assessment

Participants engaged in three tasks

orthostatic stress and the faces and sentences subtasks of the Perception of Affect Task (PAT) (Lane, Sechrest, Reidel, Weldon, Kasniak, & Schwartz, 1996). The orthostatic stress task required the participant to sit quietly for 5 minutes while baseline cardiovascular measures were collected, then stand for 5 minutes, and resume sitting for a 5 minute recovery period. The PAT faces subtask required participants to identify which of seven emotional expressions (happiness, sadness, fear, anger, surprise, disgust or neutral) were depicted in photos of Caucasian, Asian or African American faces. The PAT sentences subtask required the participants to identify which of the seven aforementioned emotions was depicted in a vignette. Each subtask consisted of 35 stimuli. Following a 5 minute seated baseline period, the orthostasis and PAT tasks were presented. The tasks were counterbalanced; however the PAT subtasks were always presented contiguously but as separate tasks.

For baseline, task, and recovery periods, BP levels were recorded continuously via a PORTApress beat-to-beat BP monitor. The PORTApres is an ambulatory monitor that collects continuous BP waveforms that are analyzed offline (TNO-TPD Biomedical Instrumentation, 1999). These values were then averaged to yield a single BP value for each time period. Arithmetic change scores (average task value – average baseline value) were computed as a measure of task-induced BP response during the tasks. These change scores were used in the final analyses for all tasks.

BP reactivity for the two PAT subtasks was significantly correlated (r's = .66, p's <.001) and the respective values were thus averaged. BP reactivity from the PAT composite variable and the orthostatic stress task were significantly correlated (r's = .82–.88, p's<.001) and thus BP reactivity variables were further averaged yielding single measures for systolic BP (SBP) and diastolic BP (DBP) during the baseline and task periods. Collapsing across tasks has been recommended to improve measurement reliability (Kamarck et al., 1992).

Cognitive Assessment

All cognitive test sessions were conducted by trained psychometricians and lasted between 1 and 2 hours. Attention, concentration and working memory were assessed by the Forward and Backward portions of the Digit Span subscale of the Wechsler Adult Intelligence Scale - Revised (WAIS-R) (Wechsler, 1981). The Trail Making Test (Reitan, 1958; Reitan & Wolfson, 1985), parts A and B, assessed mental flexibility, visuomotor tracking, eye-hand coordination,

attention, and perceptuo-motor speed. Orientation and attention were assessed by Alpha Span. Visual memory, visual perception, and visuoconstructive abilities were assessed using the Benton Visual Retention Test (BVRT). Verbal learning and memory was assessed using the California Verbal Learning Test (CVLT). CVLT scores examined in the present investigation were learning slope, free recall short delay, and free recall long delay. Verbal fluency and executive function was assessed with Letter Fluency and Category Fluency. Confrontation naming was assessed with the Boston Naming Test (BNT).

Covariates

Age and education were assessed in years. Use of antihypertensive medications was collapsed into a single “yes/no” category. Depressive symptomatology was examined using the CES-D (Radloff, 1977). Body mass index (BMI) was calculated as the ratio of weight (kg) to height (in meters) squared. History of hypertension and diabetes were coded as “yes/no” based on history and medical evaluation including oral glucose tolerance test. Smoking status was coded as “current, ever, or never.”

Analyses

Age, education, and respective baseline level of BP were used as covariates in step one of all hierarchical multiple regression models. Additional potential covariates were examined with Pearson r correlations with all cognitive test scores. These variables included CES-D score, diagnosis of hypertension, diagnosis of diabetes, use of antihypertensive medications, body mass index, and smoking status. None of these latter variables was significantly related to any cognitive score examined (p's = .07 to .97), nor did any of these variables substantively change the results of the final multiple regression analyses if included. Therefore, none of these variables was included as adjustment variables in the multiple regression analyses reported below. Baseline and reactive BP levels did not differ significantly between participants reporting concurrent use of antihypertensive medications and those not, (p's = .11 to .65). Unadjusted relations of BP to cognitive function are displayed in Table 2. Resting and reactive SBP and DBP were examined in separate multiple regression models. In order to maximize the unique information provided by each neuropsychological test, separate models were examined for each test as a dependent measure (Waldstein, Giggey, Thayer, & Zonderman, 2005). $R^2\Delta$ represents the independent contribution of SBP or DBP reactivity on the total variance explained in performance on each cognitive task after entry of covariates into the model.

A Bonferroni correction was employed at the cognitive domain level to reduce the chances of capitalizing on a Type I error. However, because of the additional risk of Type II error given the small sample size and medium effect sizes noted in select analyses, we refer to findings at the standard $p<.05$ error rate as marginally significant. For tests measuring attention (Digit Span Forward and Backward, Trail Making Test B, and Alpha Span), the corrected p value is .013 (.05/4). For the memory tests (BVRT and CVLT learning slope, free recall short delay and free recall long delay) the corrected p value is .013 (.05/4). For verbal function/language skills tests (Letter Fluency, Category Fluency, and BNT) the corrected p value is .017 (.05/3). Lastly, for perceptuo-motor speed, no correction is required because only one test (Trail Making Test A) was used in the domain. Thus, the p value remains at .05. Effect sizes (Cohen's f^2 where 0.02=small, 0.15=medium, and 0.35=large) were also calculated and are presented.

Results

After adjustment for age, education, and baseline SBP level, SBP reactivity was significantly associated with poorer performance on Digits Forward, ($\beta= -.369$, $R^2\Delta = .110$; $p=.007$, $f^2 = .20$). Similarly, after adjustment for age, education, and baseline DBP level, DBP reactivity was

significantly associated with poorer performance on Digits Forward (β= −.399, $R^2\Delta$ = .124; p=.003, f^2 = .25); and the Boston Naming Test (β= −.357, $R^2\Delta$ = .118; p=.008, f^2 = .27). After adjustment for age, education, and baseline DBP level, DBP reactivity was marginally associated with poorer performance on Alpha Span (β= −.349, $R^2\Delta$ = .104; p=.019, f^2 = .18); and CVLT free recall short delay (β= −.311, $R^2\Delta$ = .066; p=.032, f^2 = .22); Results of these multiple regression analyses are displayed in Table 3.

It should be noted that associations between BP responses and scores on Trail Making Test parts A and B, Benton Visual Retention Test, CVLT learning slope, CVLT free recall long delay, Digits Backwards, Letter Fluency and Category Fluency were not significant in the present analyses.

Discussion

Results of the present investigation revealed that increased BP reactivity was related to poorer performance on tests of attention, and confrontation naming, and marginally associated with delayed verbal memory. These findings are consistent with, and extend, those of Waldstein and Katzel (2005) who reported that higher SBP and DBP reactivity were associated with poorer performance on tests of delayed verbal memory, complex attention, and executive functioning. It is notable that, despite differences in cognitive tests employed, that the present results are similar to those noted by Waldstein and Katzel (2005).

Waldstein and Katzel (2005) reported similar patterns of findings for both SBP and DBP reactivity, whereas the present study found a preponderance of significant associations between DBP and cognitive performance. This inconsistency may be due, in part, to the use of different laboratory tasks to elicit BP reactivity. For example, the orthostatic task used in the present study is known to elicit a prominent vascular resistance response which was likely reflected in DBP reactivity (Smit, Halliwill, Low, & Wieling, 1999). It is possible that alpha- and beta-adrenergic activation patterns, that are reflected in patterns of SBP and DBP reactivity, may differentially influence cognitive function. This possibility should be examined in future investigations.

The pattern of affected test performance in the present study – attention, delayed verbal memory, and confrontation naming (which requires retrieval from memory) - suggests the possible involvement of several underlying neurobiological structures including the prefrontal and temporal cortices and associated subcortical systems (Lezak, Howieson, & Loring, 2004). These brain regions can be affected by microvascular disease processes. In that regard Waldstein et al. (2004) found that elevated stress-induced blood pressure reactivity was associated with an increased number of silent brain infarcts and a greater severity of white matter disease in otherwise healthy adults. These authors suggested that repeated episodes of stress-induced blood pressure elevation during daily life may promote cerebrovascular damage through hemodynamic changes that could result in cerebral hypoperfusion or vasospasm that promotes microvascular disease (Elias, et al., 1993; Harrington et al., 2000; Kario et al. 1999).

Another potential mechanism linking increased BP reactivity to stress and decreased cognitive performance is concomitant cortisol reactivity because individuals may respond to stressors across multiple physiological systems including autonomic and endocrine. Results of longitudinal studies suggest that cumulative exposure to high and increasing levels of cortisol is associated with decreased hippocampal volumes, and decline in attention, memory, and executive function (Lupien et al., 2005; Li et al., 2006) suggesting that detrimental effects are not isolated to the hippocampus, but may extend to the frontal lobes (Li etal., 2006). Negative relations between cortisol reactivity and memory have also been noted in older adults (Wright, Kunz-Ebrecht, Iliffe, Foese, & Steptoe, 2005). The addition of cortisol and catecholamine

assessment could illuminate the respective influences of sympathetic nervous system and hypothalamic-pitiutary-adrenocortical activation on cognitive function.

The concept of allostatic load also reflects the idea that individuals may respond to stressors via multiple physiological systems including the autonomic nervous system, hypothalamic-pituitary-adrenocortical axis, cardiovascular, metabolic and immune (McEwen, 1998). It is hypothesized that the wear and tear on these bodily systems (e.g. allostatic load) is a result of their over or underactivity. Sympathetic nervous system activation is central to the allostatic load hypothesis, and may be reflected in stress-induced BP reactivity. Studies from the MacArthur Studies of Successful Aging have reported that participants with higher levels of allostatic load displayed steeper declines in physical and cognitive functioning (McEwen, 1998; Karlamangla, Singer, McEwen, Rowe, & Seeman, 2002).

The present study has a number of strengths and limitations. To our knowledge, this was only the second study to investigate BP reactivity to laboratory challenges and cognitive function. In comparison to Waldstein and Katzel (2005) the present study used a broader range of cognitive tests with particular expansion of assessment of attention, verbal and nonverbal memory and executive function. Additionally, the present study included tests of language skills whereas this cognitive domain was not assessed previously. Here, blood pressure was measured continuously (Peňáz/Wesseling method) during baseline and task periods, a superior, albeit more costly method (Boehmer, 1987) than the oscillomentric monitoring used by Waldstein and Katzel (2005).

Study limitations include a small sample that yields limited statistical power. Further, the sample was derived from a larger convenience sample thus limiting its representativeness. Another relevant limitation is that potential confounding variables that were not measured here could have influenced the findings. Finally, our statistical methods create difficulties with both Type I and Type II error. Thus, interpretation of findings should rely on a combination of effect size and statistical significance.

To summarize, results of the present investigation that increased BP reactivity was related to poorer performance on tests of attention, confrontation naming, and delayed verbal memory explaining 7 – 12% of the variance in these measures. This magnitude of association is similar to that seen for resting blood pressure and cognitive performance (Waldstein, 1995). Increased stress-induced BP reactivity may confer biobehavioral risk for cognitive difficulties and over time may predispose to cognitive decline or even vascular dementia or Alzheimer's disease. Stress-induced BP responses do predict incident stroke (Everson et al., 2001). If warranted in the future, screening of BP reactivity could be included in standard medical evaluations, perhaps in the form of orthostatic manipulation.

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Table 1

Sample Characteristics for Demographics, Baseline and Task Cardiovascular Measures

Characteristic	M(SD)	Range
Demographics:		
Age (years)	70.14(13.05)	50–90
Education (years)	16.76(2.63)	12–24
CES-D score	5.18(3.96)	0–15
BMI	26.60(3.46)	21.27–36.33
Male	53%	
Caucasian	71.6%	
African American	25.9%	
Asian	2.5%	
Current Smoker	11.2%	
Ever smoked	60.0%	
Positive Diagnosis of HTN	42.5%	
Positive Diagnosis of Diabetes	7.5%	
Current Use of HTN Meds	40.3%	
Baseline Cardiovascular Measures:		
SBP (mmHg)	130.96	20.02
DBP (mmHg)	68.14	10.10
PAT Faces Cardiovascular Measures:		
SBP (mmHg)	135.68	20.77
DBP (mmHg)	72.63	10.08
PAT Sentences Cardiovascular Measures:		
SBP (mmHg)	140.62	14.71
DBP (mmHg)	77.68	7.89
Orthostatic Stress Cardiovascular Measures:		
SBP (mmHg)	147.20	23.78
DBP (mmHg)	77.16	12.61
Average Task Cardiovascular Measures:		
SBP (mmHg)	141.17	19.75
DBP (mmHg)	75.82	10.19

Note. HTN and Diabetes diagnosis data was missing for 5 participants (6.3%); Use of CV meds data was missing for 8 participants (10.4%).

Table 2

Unadjusted Pearson r Correlations of SBP and DBP Reactivity and Cognitive Test Score

	SBP		DBP	
	r	**p**	**r**	**p**
Digits forward	−.342	.004	−.416	<.001
Digits backward	−.065	.596	−.147	.226
Trails A	.089	.499	.108	.410
Trails B	.095	.470	.139	.289
Alpha Span	−.214	.111	−.342	.009
BVRT correct	.230	.057	.312	.009
CVLT				
Learning slope	−.181	.134	−.262	.028
Free recall (short)	−.148	.222	−.262	.028
Free recall (long)	−.100	.411	−.126	.300
Letter Fluency	−.114	.383	−.241	.062
Category Fluency	−.114	.384	−.205	.112
Boston Naming Test	−.097	.462	−.366	.004

Table 3

Summary of Significant Hierarchical Multiple Regression Analyses for SBP and DBP Reactivity and Cognitive Test Scores

Variables Entered		β	p	$R^2\Delta$	R^2
SBP Reactivity					
Digits Forward:					
Step 1: Covariates	Age	.002	.998		
	Education	.193	.128		
	Baseline SBP	.124	.325	.056	.056
Step 2: SBP Reactivity	SBP Change	−.369	.007	.110	.166
DBP Reactivity					
Digits Forward:					
Step 1: Covariates	Age	.089	.459		
	Education	.095	.431		
	Baseline DBP	.048	.711	.074	.074
Step 2: DBP Reactivity	DBP Change	−.399	.003	.124	.199
Alpha Span:					
Step 1: Covariates	Age	−.018	.897		
	Education	.145	.292		
	Baseline DBP	−.092	.521	.051	.051
Step 2: DBP Reactivity	DBP Change	−.349	.019	.104	.155
CVLT Free Recall Short Delay:					
Step 1: Covariates	Age	−.187	.144		
	Education	.176	.151		
	Baseline DBP	−.304	.029	.114	.114
Step 2: DBP Reactivity	DBP Change	−.311	.032	.066	.180
Boston Naming Test:					
Step 1: Covariates	Age	−.172	.191		
	Education	.182	.152		
	Baseline DBP	−.191	.154	.097	.097
Step 2: DBP Reactivity	DBP Change	−.357	.008	.118	.215

Health Psychol. Author manuscript; available in PMC 2009 December 20.