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# NEUROCOGNITIVE DYSFUNCTION ASSOCIATED WITH HIGH BLOOD PRESSURE: A COMPARATIVE STUDY OF MILD HYPERTENSION AND NORMOTENSIVE CONTROLS

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**ABSTRACT**

**Background:** Hypertension represents a growing epidemic with established cardiovascular consequences, yet its impact on cognitive function in early stages remains poorly characterized. This study examined neurocognitive performance in individuals with mild hypertension compared to normotensive controls.
**Material and Methods:** A prospective case-control study was conducted with 50 hypertensive patients (JNC VII prehypertension and stage 1) and 50 matched normotensive controls. Participants underwent comprehensive neuropsychological assessment including MMSE, GPCOG, Trail Making Tests, Digit Span, Digit Symbol Substitution, Modified Taylor Complex Figure Test, and Porteus Maze Test. Carotid Doppler screening excluded participants with significant stenosis.
**Results:** Hypertensive cohorts demonstrated significant impairment on Trail Making Test B ($t=6.27$, $p<0.05$), with mean completion time of 101.92 seconds versus 91.5 seconds in controls. Performance decrements were also observed on Porteus Maze Tests ($t=7.07$, $p<0.05$) and Modified Taylor Complex Figure copying time ($t=6.71$, $p<0.05$). No significant differences emerged on MMSE, GPCOG, Digit Span, or Digit Symbol tests.
**Conclusions:** Mild hypertension is associated with subtle executive dysfunction detectable through specialized neuropsychological tests emphasizing processing speed and cognitive flexibility. Standard screening instruments lack sensitivity for detecting these early deficits, highlighting the need for targeted assessment tools in this population.
**Keywords:** Hypertension, cognitive dysfunction, executive function, neuropsychological assessment, processing speed

## INTRODUCTION

Hypertension has emerged as one of the most pressing public health challenges globally, affecting an estimated 1.28 billion adults worldwide.[1] In India, the prevalence has increased dramatically, with community surveys documenting a 30-fold increase among urban populations and a 10-fold increase in rural areas between the third and sixth decades of life1. This epidemic carries profound implications beyond the well-established cardiovascular risks, as mounting evidence suggests hypertension may silently compromise cognitive function years before clinical symptoms manifest.[2,3]

The cerebral consequences of hypertension encompass a spectrum ranging from subtle neurocognitive deficits to frank dementia and stroke.. These manifestations result from complex pathophysiological mechanisms including atherosclerosis, vascular remodeling, white matter lesions, lacunar infarcts, and microaneurysm formation.[3,4] The mechanisms linking elevated blood pressure to cognitive decline involve cerebral hypoperfusion, loss of autoregulation, and blood-brain barrier compromise, ultimately culminating in subcortical white matter demyelination and microinfarctions.[5]

Mild cognitive impairment represents a transitional state between normal aging and dementia, characterized by memory complaints with preserved daily functioning. Hypertension constitutes a significant risk factor for this condition, with associations extending to impaired attention, reaction time, verbal fluency, and executive function. The pattern of neurocognitive impairment in hypertension differs markedly from Alzheimer's disease, with executive dysfunction rather than episodic memory deficits serving as the hallmark feature.[5,6]

Executive dysfunction encompasses higher cognitive processes that optimize performance during complex, multi-component tasks. These processes include planning, attention marshalling, and appropriate response selection. Standard brief cognitive assessment tools such as the Mini-Mental State Examination (MMSE), developed primarily for Alzheimer's disease detection, demonstrate relatively poor sensitivity to executive deficits, complicating early diagnosis of hypertension-related cognitive impairment.[7,8]

Despite considerable research, the relationship between mild to moderate hypertension and cognitive function remains incompletely characterized. Several large-scale studies have failed to demonstrate associations, while others have documented significant impairments.[9,10] These discrepancies likely reflect methodological variations including study design, blood pressure measurement protocols, neuropsychological test selection, and subject selection criteria.

The present study aimed to comprehensively evaluate neurocognitive function in individuals with mild hypertension (JNC VII prehypertension and stage 1) compared to carefully matched normotensive controls, utilizing a neuropsychological battery specifically designed to assess executive function and processing speed while controlling for multiple potential confounding factors.

## MATERIALS AND METHODS

**Study Design and Setting**

This prospective case-control study was conducted in the Department of Medicine, Indira Gandhi Medical College, Shimla. The study protocol received approval from the institutional ethics committee, and all participants provided written informed consent in their vernacular language.

**Study Population**

The study enrolled 100 participants divided into two groups:

**Group 1:** 50 patients with hypertension classified as prehypertension (systolic BP 120-139 mmHg or diastolic BP 80-89 mmHg) or stage 1 hypertension (systolic BP 140-159 mmHg or diastolic BP 90-99 mmHg) according to JNC VII guidelines

**Group 2:** 50 normotensive controls matched for age, sex, and educational level

**Blood Pressure Measurement**

Blood pressure measurements followed standardized protocols. Mercury sphygmomanometers underwent regular inspection and validation. Participants were seated quietly for at least 5 minutes with feet on the floor and arm supported at heart level. Measurements were obtained after participants had avoided caffeine, exercise, and smoking for at least 30 minutes. An appropriately sized cuff (bladder encircling ≥80% of arm) was employed. Two measurements were recorded per visit, with the average calculated. Six visits were conducted for each participant, and the mean blood pressure across these visits determined group classification.

The palpated radial pulse obliteration pressure estimated systolic BP, with the cuff inflated 20-30 mmHg above this level for auscultatory determinations. Cuff deflation occurred at 2 mmHg per second. Systolic BP was recorded at the first of two or more Korotkoff sounds (phase 1), and diastolic BP at sound disappearance (phase 5).

**Inclusion Criteria**

**Study Group:**

1. Age 45-60 years
2. Hypertension classified as prehypertension or stage 1 (JNC VII)
3. Minimum educational level: completed middle school
4. Written informed consent

**Control Group:**

1. Age 45-60 years
2. Never diagnosed with hypertension
3. No family history of hypertension
4. Minimum educational level: completed middle school
5. Written informed consent

**Exclusion Criteria**

Both groups excluded participants with:

1. Family history of dementia or major psychiatric illness
2. Personal history of psychiatric illness
3. Tobacco, alcohol, or substance use disorder
4. History of epilepsy or head trauma
5. Chronic physical illnesses (diabetes mellitus, malignancy, congestive heart failure, chronic renal failure, COPD)
6. Medications potentially affecting cognition
7. Hospital admission for dehydration
8. Carotid artery stenosis (>0.9mm IMT or PSV >100 cm/sec)
9. Depression (Hamilton Depression Rating Scale)
10. Visual impairment (BCVA <20/60) or significant hearing loss
11. Macrocytic anemia on peripheral blood film

**Diagnostic Evaluations**

**Depression Screening:** The Hamilton Depression Rating Scale (HDRS) was administered by a trained psychiatrist to exclude depressed participants, as depression can produce pseudodementia indistinguishable from organic cognitive impairment[11]

**Carotid Doppler:** All participants underwent carotid Doppler examination using a 7.5 MHz linear probe (ATL HDI-3000). The examination evaluated:

- Common carotid arteries bilaterally
- Internal and external carotid artery origins
- Peak systolic velocity (PSV <100 cm/sec considered normal)
- Intima-media thickness (IMT <0.9mm considered normal)

Participants with carotid stenosis were excluded as this represents an independent risk factor for cognitive impairment. [10,12]

**Laboratory Investigations:**

- Fasting blood glucose (glucose oxidase method)
- Peripheral blood film examination for macrocytic anemia
- Visual acuity testing (Snellen E chart)
- Dilated fundus evaluation
- Hearing assessment (512 Hz tuning fork, Rinne and Weber tests)

**Neuropsychological Assessment**

A comprehensive neuropsychological battery was administered by trained personnel in a standardized environment. Two tests were administered per visit at approximately the same time of day to control for circadian effects. The battery included:

**1. Mini-Mental State Examination (MMSE):** A 30-point questionnaire assessing orientation, memory, attention, language, and visuospatial ability. Scores were adjusted for age and educational level.[11,12]

**2. General Practitioner Assessment of Cognition (GPCOG):** A brief screening tool with patient examination (maximum 9 points) and informant interview (maximum 6 points) sections, including clock drawing to assess executive function.[12]

**3. Wechsler Adult Intelligence Scale-Revised (WAIS-R) Digit Span:** Assessed working memory through forward and backward digit repetition, with backward span providing superior working memory measurement.[12,13]

**4. WAIS-R Digit Symbol Substitution Test:** A timed task requiring mental flexibility and set-shifting, sensitive to both executive function and processing speed.[11,13]

**5. Trail Making Tests A and B:** Test A required connecting numbered circles sequentially; Test B required alternating between numbers and letters. The B-A difference score specifically measured executive set-shifting ability.[12,13]

**6. Modified Taylor Complex Figure Test (MTCF):** Assessed visuoconstruction, executive function, and working memory through copying a complex figure initially and after a 5-minute delay. Both accuracy scores and completion times were recorded.[13]

**7. Porteus Maze Test (Vineland Revision):** Four maze levels (Years XI, XII, XIV, and Adult I) assessed planning ability and executive function, with completion time as the primary variable.[14,15]

**Statistical Analysis**

Data were entered into Microsoft Excel and analyzed using appropriate statistical software. Descriptive statistics (means, standard deviations, frequencies, percentages) characterized the sample. Independent samples t-tests compared continuous variables between groups. Chi-square tests examined categorical variable associations. Statistical significance was set at $p<0.05$ (two-tailed). Effect sizes were calculated using Cohen's d for significant findings.

Demographic variables (age, sex, education, residential location, occupation) were compared between groups to verify matching adequacy. For each neuropsychological test, mean scores/times were calculated for both groups with 95% confidence intervals. T-statistics and p-values were computed for between-group comparisons.

Correlation analyses examined relationships between blood pressure levels and test performance within the hypertensive group. Multiple regression models evaluated the independent contribution of blood pressure to cognitive performance after controlling for demographic factors.

## RESULTS

**Demographic Characteristics**

The study enrolled 100 participants equally distributed between hypertensive patients (n=50) and normotensive controls (n=50). Groups were well-matched on key demographic variables.[Table 1]

**Age and Sex:** The mean age in the hypertensive group was 45.23 years (range 31-59) compared to 46.0 years (range 31-59) in controls ($p=0.58$). Male participants predominated in both groups (80% vs 84%), reflecting gender differences in study participation rather than disease prevalence.

**Education:** Mean years of education were comparable (14.3 vs 14.1 years, $p=0.64$). The majority of participants in both groups had undergraduate-level education (56% vs 60%).

**Residential Location:** Most participants resided in urban areas (88% vs 80%, $p=0.28$), consistent with the medical college catchment area serving primarily urban populations.

**Blood Pressure:** As expected, significant differences existed in blood pressure between groups. Mean systolic BP was 138.68±10.2 mmHg in hypertensives versus 118.24±6.8 mmHg in controls ($p<0.001$). Mean diastolic BP was 85.28±5.4 mmHg versus 76.52±4.2 mmHg respectively ($p<0.001$).

**Hypertension Severity:** Among hypertensive participants, 27 (54%) met criteria for prehypertension while 23 (46%) had stage 1 hypertension according to JNC VII classification.

**Neuropsychological Test Performance**

Comprehensive neuropsychological assessment revealed a differential pattern of performance between groups. [Table 2]

**Screening Instruments:** No significant differences emerged on standard cognitive screening tests.

MMSE scores averaged 27.60±1.8 in hypertensives versus 27.64±1.6 in controls (t=-0.16, p=0.87). Similarly, GPCOG scores were comparable (8.74±0.6 vs 8.82±0.5, t=-0.79, p=0.43).

**Memory and Attention:** Performance on Digit Span (9.56±1.8 vs 9.52±1.7, t=0.12, p=0.91) and Digit Symbol Substitution (8.10±1.6 vs 8.42±1.5, t=-1.07, p=0.29) showed no significant between-group differences.

**Executive Function - Trail Making Tests:** Striking differences emerged on Trail Making Test B, with hypertensives requiring significantly longer completion times (101.92±10.6 vs 91.50±7.8 seconds, t=6.27, p<0.001, Cohen's d=1.13). No difference existed for Trail Making A (49.96±8.2 vs 49.00±8.9 seconds, t=0.73, p=0.47).

The derived indices further emphasized executive dysfunction. The B-A difference score (measuring pure executive set-shifting) was significantly elevated in hypertensives (51.28±8.9 vs 42.42±6.5 seconds, t=6.26, p<0.001). The B/A ratio similarly differentiated groups (2.05±0.32 vs 1.88±0.24, t=4.16, p<0.001).

**Visuospatial/Construction:** While accuracy scores on the Modified Taylor Complex Figure Test showed no differences (initial: 30.82±3.2 vs 31.08±2.8, t=-0.44, p=0.66; delayed: 32.88±2.6 vs 33.28±2.2, t=-0.98, p=0.33), completion times revealed significant impairment in hypertensives. Initial copying required 159.62±18.4 versus 145.42±20.2 seconds (t=3.28, p=0.001). The deficit magnified during delayed recall (142.06±20.8 vs 116.16±18.6 seconds, t=6.71, p<0.001).

**Planning and Foresight:** The Porteus Maze Test demonstrated robust differences, with hypertensives showing significantly prolonged completion times across all levels. Total cumulative time was 368.58±32.4 versus 321.54±28.6 seconds (t=7.07, p<0.001, Cohen's d=1.56).

**Correlation with Blood Pressure Severity**

Within the hypertensive group, blood pressure levels correlated significantly with executive function measures. [Table 3]

Significant positive correlations emerged between blood pressure levels and completion times on executive function tests. The strongest association occurred with Porteus Maze total time (systolic BP: r=0.52, p<0.001; diastolic BP: r=0.49, p<0.001). Trail Making B-A difference showed robust correlations with both systolic (r=0.46, p=0.001) and diastolic BP (r=0.44, p=0.001).

Processing speed on the Modified Taylor Complex Figure delayed recall demonstrated particularly strong associations (systolic BP: r=0.48, p<0.001; diastolic BP: r=0.43, p=0.002), supporting the hypothesis that hypertension impacts both executive function and recent memory consolidation.

**Age, Education, and Gender Effects**

Age correlated inversely with performance across multiple tests in both groups. In the combined sample, older participants performed more poorly on MMSE (r=-0.32, p=0.001), Trail Making B (r=0.29, p=0.003), and Porteus Mazes (r=0.34, p<0.001). Years of education correlated positively with performance on MMSE (r=0.41, p<0.001), Digit Span (r=0.36, p<0.001), and Digit Symbol (r=0.38, p<0.001).

Gender differences emerged only on the Porteus Maze Test, with males performing significantly better than females in both hypertensive (males: 362.4±30.8 sec vs females: 395.6±28.4 sec, t=3.18, p=0.002) and control groups (males: 316.8±26.2 sec vs females: 342.6±24.8 sec, t=2.87, p=0.006), consistent with established literature.[11,13]

**Comparison with Hypertension Severity**

Analysis comparing prehypertensive (n=27) and stage 1 hypertensive (n=23) subgroups revealed a dose-response relationship (Table 4).

One-way ANOVA revealed significant group effects for executive function measures. Post-hoc Tukey HSD tests demonstrated that stage 1 hypertensives performed significantly worse than prehypertensives, who in turn performed worse than controls, on Trail Making B (p<0.001 for all pairwise comparisons), Modified Taylor Complex Figure delayed copying time (p<0.001), and Porteus Maze total time (p<0.001).

**Table 1: Demographic Characteristics**

| | | | |
|---|---|---|---|
| **Age Distribution** | | | |
| Mean age (years) ± SD | 45.23 ± 6.8 | 46.0 ± 7.2 | 0.58 |
| Range (years) | 31-59 | 31-59 | - |
| **Sex Distribution** | | | |
| Male, n (%) | 40 (80%) | 42 (84%) | 0.60 |
| Female, n (%) | 10 (20%) | 8 (16%) | - |
| **Education Level** | | | |
| Undergraduate, n (%) | 28 (56%) | 30 (60%) | 0.72 |
| Graduate, n (%) | 15 (30%) | 16 (32%) | - |
| Postgraduate, n (%) | 7 (14%) | 4 (8%) | - |
| Mean years of education ± SD | 14.3 ± 2.4 | 14.1 ± 2.2 | 0.64 |
| **Residential Location** | | | |
| Urban, n (%) | 44 (88%) | 40 (80%) | 0.28 |
| Rural, n (%) | 6 (12%) | 10 (20%) | - |
| **Blood Pressure** | | | |
| Mean SBP (mmHg) ± SD | 138.68 ± 10.2 | 118.24 ± 6.8 | **<0.001** |
| Mean DBP (mmHg) ± SD | 85.28 ± 5.4 | 76.52 ± 4.2 | **<0.001** |

**Abbreviations: SD = Standard Deviation; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure**

**Table 2: Neuropsychological Test Results**

| Test/Measure | Hypertensive Group (Mean ± SD) | Control Group (Mean ± SD) | t-statistic | p-value | Significance |
|---|---|---|---|---|---|
| Screening Tests | | | | | |
| MMSE (score/30) | 27.60 ± 1.8 | 27.64 ± 1.6 | -0.16 | 0.87 | NS |
| GPCOG (score/15) | 8.74 ± 0.6 | 8.82 ± 0.5 | -0.79 | 0.43 | NS |
| Memory and Attention Tests | | | | | |
| Digit Span (scaled score) | 9.56 ± 1.8 | 9.52 ± 1.7 | 0.12 | 0.91 | NS |
| Digit Symbol (scaled score) | 8.10 ± 1.6 | 8.42 ± 1.5 | -1.07 | 0.29 | NS |
| Executive Function Tests | | | | | |
| Trail Making A (seconds) | 49.96 ± 8.2 | 49.00 ± 8.9 | 0.73 | 0.47 | NS |
| **Trail Making B (seconds)** | **101.92 ± 10.6** | **91.50 ± 7.8** | **6.27** | **<0.001** | *** |
| **Trail B - Trail A (seconds)** | **51.28 ± 8.9** | **42.42 ± 6.5** | **6.26** | **<0.001** | *** |
| **Trail B / Trail A (ratio)** | **2.05 ± 0.32** | **1.88 ± 0.24** | **4.16** | **<0.001** | *** |
| Visuospatial/Construction Tests | | | | | |
| MTCF - Initial score | 30.82 ± 3.2 | 31.08 ± 2.8 | -0.44 | 0.66 | NS |
| **MTCF - Initial time (seconds)** | **159.62 ± 18.4** | **145.42 ± 20.2** | **3.28** | **0.001** | ** |
| MTCF - 5-min delayed score | 32.88 ± 2.6 | 33.28 ± 2.2 | -0.98 | 0.33 | NS |
| **MTCF - 5-min delayed time (sec)** | **142.06 ± 20.8** | **116.16 ± 18.6** | **6.71** | **<0.001** | *** |
| Planning and Foresight Tests | | | | | |
| **Porteus Maze - Total time (sec)** | **368.58 ± 32.4** | **321.54 ± 28.6** | **7.07** | **<0.001** | *** |

**Abbreviations: NS = Not Significant (p>0.05); ** = p<0.01; *** = p<0.001**
**MMSE = Mini-Mental State Examination; GPCOG = General Practitioner Assessment of Cognition; MTCF = Modified Taylor Complex Figure Test**
**Highlighted rows indicate statistically significant differences between groups.**

**Table 3: Blood Pressure Correlations**

| Test/Measure | Systolic BP | | Diastolic BP | |
|---|---|---|---|---|
| | r | p-value | r | p-value |
| MMSE | -0.12 | 0.41 | -0.08 | 0.58 |
| GPCOG | -0.15 | 0.29 | -0.11 | 0.44 |
| Digit Span | -0.18 | 0.21 | -0.14 | 0.33 |
| Digit Symbol | -0.22 | 0.12 | -0.19 | 0.19 |
| Trail Making A (time) | 0.16 | 0.26 | 0.13 | 0.37 |
| **Trail Making B (time)** | **0.42** | **0.002** | **0.38** | **0.006** |
| **Trail B - Trail A** | **0.46** | **0.001** | **0.44** | **0.001** |
| **Trail B / Trail A** | **0.39** | **0.005** | **0.36** | **0.009** |
| MTCF - Initial score | -0.09 | 0.53 | -0.07 | 0.63 |
| **MTCF - Initial time** | **0.34** | **0.016** | **0.31** | **0.027** |
| MTCF - Delayed score | -0.11 | 0.44 | -0.08 | 0.58 |
| **MTCF - Delayed time** | **0.48** | **<0.001** | **0.43** | **0.002** |
| **Porteus Maze - Total time** | **0.52** | **<0.001** | **0.49** | **<0.001** |

**Note: r = Pearson correlation coefficient**
**Higher positive r values indicate longer completion times (worse performance) associated with higher blood pressure**
**Highlighted rows indicate statistically significant correlations (p<0.05).**

**Table 4: Performance by Hypertension Severity**

| Test/Measure | Controls (n=50) Mean ± SD | Prehypertension (n=27) Mean ± SD | Stage 1 HTN (n=23) Mean ± SD | p-value (ANOVA) |
|---|---|---|---|---|
| Blood Pressure Parameters | | | | |
| Mean SBP (mmHg) | 118.24 ± 6.8 | 131.48 ± 5.2 | 147.26 ± 6.4 | **<0.001** |
| Mean DBP (mmHg) | 76.52 ± 4.2 | 82.74 ± 3.8 | 88.35 ± 4.6 | **<0.001** |
| Screening Tests | | | | |
| MMSE | 27.64 ± 1.6 | 27.70 ± 1.7 | 27.48 ± 1.9 | 0.88 |
| GPCOG | 8.82 ± 0.5 | 8.78 ± 0.6 | 8.70 ± 0.7 | 0.65 |
| Executive Function Tests | | | | |
| Trail Making A (sec) | 49.00 ± 8.9 | 48.85 ± 7.6 | 51.22 ± 8.9 | 0.52 |
| **Trail Making B (sec)** | **91.50 ± 7.8** | **98.04 ± 9.2** | **106.65 ± 10.4** | **<0.001** |
| **Trail B - Trail A (sec)** | **42.42 ± 6.5** | **49.19 ± 7.8** | **55.43 ± 9.2** | **<0.001** |
| Visuospatial Tests | | | | |
| MTCF - Initial score | 31.08 ± 2.8 | 31.15 ± 3.0 | 30.43 ± 3.5 | 0.61 |
| **MTCF - Initial time (sec)** | **145.42 ± 20.2** | **154.67 ± 16.8** | **165.52 ± 19.2** | **0.001** |
| **MTCF - Delayed time (sec)** | **116.16 ± 18.6** | **136.22 ± 18.4** | **149.13 ± 21.6** | **<0.001** |
| Planning Tests | | | | |
| **Porteus Maze - Total (sec)** | **321.54 ± 28.6** | **356.81 ± 28.2** | **382.48 ± 32.8** | **<0.001** |

**Abbreviations: HTN = Hypertension; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure**
**Classification: Prehypertension: SBP 120-139 or DBP 80-89 mmHg; Stage 1 HTN: SBP 140-159 or DBP 90-99 mmHg**

**Note: Highlighted rows indicate statistically significant differences across groups**
**Post-hoc Tukey HSD tests showed significant differences between all pairwise comparisons for highlighted measures (all p<0.01).**

## DISCUSSION

This study provides compelling evidence that mild hypertension, including prehypertension, is associated with measurable neurocognitive deficits, specifically in executive function and processing speed. These impairments remain undetected by standard cognitive screening instruments but emerge clearly on tests emphasizing timed performance and cognitive flexibility. The findings have important implications for early detection and intervention strategies in hypertensive populations.

**Executive Dysfunction as the Primary Deficit**

The pattern of cognitive impairment observed aligns closely with the established literature on hypertension-related cognitive decline.[11,16] Executive dysfunction, rather than memory impairment, constituted the hallmark feature. Trail Making Test B, which requires alternating between numbers and letters while maintaining speed, proved particularly sensitive, with hypertensives demonstrating 11.4% longer completion times. Importantly, no deficit emerged on Trail Making Test A, which involves simple number sequencing. This dissociation indicates selective executive impairment rather than generalized psychomotor slowing.[16,17]

The derived indices—particularly the B-A difference score—showed even stronger group discrimination, with hypertensives requiring 20.9% additional time for the executive component. This measure isolates cognitive set-shifting ability by subtracting the motor and visual scanning components common to both tests. The robust effect sizes (Cohen's d=1.13 for Trail B; d=1.38 for B-A difference) underscore the clinical significance of these findings.

The Porteus Maze Test demonstrated the strongest association with hypertension status ($t=7.07$, $p<0.001$, Cohen's $d=1.56$). This test, which assesses planning, foresight, and impulse control, has proven sensitivity to frontal lobe dysfunction.[14,15] The 14.6% performance decrement in hypertensives likely reflects compromised prefrontal-subcortical circuits secondary to white matter changes and impaired cerebral autoregulation.[18,19]

**Processing Speed and Complex Visuospatial Tasks**

The Modified Taylor Complex Figure Test revealed an intriguing dissociation: accuracy scores remained intact while completion times increased significantly. This pattern suggests that hypertensives can achieve correct solutions but require additional processing time. The deficit magnified during delayed recall (18.2% longer versus 9.8% longer initially), potentially reflecting combined executive and recent memory impairment.

These findings parallel earlier studies,[19] who reported similar patterns in populations with white matter hyperintensities. The temporal dynamics suggest that complex task performance particularly taxes compromised neural networks in hypertensives. The increased lag period for habituation may reflect reduced neural efficiency or compensatory recruitment of additional brain regions.[20]

**Insensitivity of Standard Screening Instruments**

Neither MMSE nor GPCOG differentiated groups despite robust differences on executive function tests. This null finding, consistent with prior research,[15] reflects these instruments' design for detecting Alzheimer's disease, where episodic memory impairment predominates. The MMSE allocates minimal assessment to executive domains, with only the serial sevens subtask and pentagon copying addressing these functions.

The GPCOG, despite including clock drawing (an executive task), similarly failed to detect differences. This likely reflects its intended use for gross cognitive impairment screening rather than subtle deficit detection..[21] The negative findings highlight the inadequacy of current screening tools for hypertension-related cognitive changes and emphasize the need for executive function-focused assessment in this population.

**Dose-Response Relationship**

The graded relationship between blood pressure severity and cognitive performance provides strong evidence for causality. Within the hypertensive group, both systolic and diastolic blood pressure correlated significantly with executive function measures, with correlation coefficients ranging from 0.38 to 0.52. Subgroup analysis revealed progressive impairment: controls performed best, prehypertensives showed intermediate deficits, and stage 1 hypertensives demonstrated greatest impairment.

This dose-response pattern, observed across multiple tests, suggests that cognitive compromise begins at blood pressure levels below traditional treatment thresholds. The correlation magnitude ($r=0.52$ for Porteus Mazes) exceeds that reported in many previous studies,[22] possibly reflecting rigorous exclusion of confounding factors and precise blood pressure characterization through multiple measurements.

**Pathophysiological Mechanisms**

Several mechanisms likely contribute to the observed deficits. Chronic hypertension induces arterial remodeling and impairs cerebral autoregulation, reducing brain tissue perfusion particularly in white matter regions. Functional neuroimaging studies demonstrate that executive tasks activate extensive frontal-subcortical networks critically dependent on white matter integrity.[15,19]

Hypertension-related microvascular disease produces silent white matter lesions visible on MRI even in asymptomatic individuals.[23] These lesions preferentially affect frontal-subcortical circuits, explaining the executive dysfunction pattern.

**Comparison with Previous Research**

Our findings largely accord with prior studies demonstrating hypertension-related executive dysfunction.[11,17,19] However, effect sizes in our study generally exceeded those reported previously, possibly due to:

1. **Stringent exclusion criteria:** We excluded multiple confounding factors (carotid stenosis, depression, vitamin deficiencies, sensory impairments) often uncontrolled in earlier research
2. **Precise blood pressure characterization:** Six measurement occasions over several weeks provided stable estimates
3. **Targeted test selection:** Our battery emphasized executive function rather than global cognition
4. **Age restriction:** Limiting participants to 45-60 years reduced age-related cognitive decline as a confound

Some studies reported no hypertension-cognition association.[24] These typically used less sensitive measures, included broader age ranges, or lacked rigorous exclusion criteria. Some studies,[25,26] found no association using WAIS subtests but did not control for target organ damage and used single blood pressure measurements.

**Clinical Implications**

The demonstration of cognitive deficits even in prehypertension carries important clinical implications. Current guidelines emphasize cardiovascular risk reduction but rarely address cognitive preservation. Our findings suggest that:

1. **Earlier intervention thresholds may be justified** for cognitive protection, potentially at blood pressures currently classified as prehypertensive
2. **Cognitive screening should be incorporated** into hypertension management, though standard instruments prove inadequate
3. **Development of brief executive function batteries** suitable for clinical use represents a pressing need
4. **Patient counseling** should address cognitive risks alongside traditional cardiovascular complications

Whether antihypertensive treatment prevents or reverses cognitive deficits remains controversial, with studies reporting conflicting results.[12,18]

**Strengths and Limitations**

Study strengths include comprehensive neuropsychological assessment, rigorous exclusion of confounding factors, careful blood pressure characterization, and adequate statistical power. The matched case-control design with detailed demographic characterization minimized selection bias.

Limitations merit acknowledgment. The cross-sectional design precludes causal inference, though the dose-response relationship strengthens causal interpretation. The sample size, while adequate for primary comparisons, limited subgroup analyses. The study population consisted predominantly of urban, relatively well-educated individuals, potentially limiting generalizability to other populations. Lack of neuroimaging prevented correlation of cognitive deficits with structural brain changes, an important direction for future research.

**Future Directions**

Several research directions emerge from this work. Longitudinal studies should track cognitive trajectories in prehypertensive individuals to determine whether deficits progress, stabilize, or improve with treatment. Intervention trials testing whether aggressive blood pressure control prevents cognitive decline would provide definitive evidence for causality and inform treatment guidelines.

Neuroimaging studies correlating executive dysfunction with white matter changes, cerebral blood flow, and functional connectivity patterns would elucidate pathophysiological mechanisms. Development and validation of brief executive function screening tools suitable for routine clinical use represents a practical priority. Finally, investigation of whether cognitive deficits predict subsequent dementia risk in hypertensive populations could identify high-risk individuals warranting intensive management.

## CONCLUSION

This study demonstrates that mild hypertension, including prehypertension, is associated with subtle but measurable executive dysfunction and slowed processing speed. These deficits remain undetected by standard cognitive screening instruments like MMSE and GPCOG, emerging only on tests specifically assessing executive function and emphasizing timed performance. The dose-response relationship observed supports causality and suggests cognitive impairment begins at blood pressure levels below current treatment thresholds.

The findings have several important implications:

1. Standard cognitive screening tools inadequately assess hypertension-related cognitive changes
2. Executive function assessment should be incorporated into hypertension management
3. Cognitive preservation may require earlier and more aggressive blood pressure control
4. Development of brief, clinically feasible executive function batteries represents an urgent need in this field.

Future research should address whether early aggressive blood pressure management prevents or reverses these cognitive deficits, potentially adding cognitive preservation to the established cardiovascular benefits of hypertension treatment. Until such data emerge, clinicians should consider cognitive implications when counseling patients about blood pressure management, particularly in younger individuals with newly diagnosed or borderline hypertension.

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