Blood Pressure and Cognitive Function in Older Adults

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KEYWORDS

• Blood pressure • Hypertension • Aging • Cognition • Dementia • Prevention • Treatment

KEY POINTS

- The relationship between blood pressure and cognitive function is complex and varies across different stages of life. Overall, elevated midlife blood pressure has the strongest association with late-life cognitive decline and dementia, supporting the need for a life course approach to detect and manage hypertension in clinical practice.
- In addition to hypertension, several lines of research suggest additional hemodynamic metrics may synergistically impair cognitive function and increase the risk of dementia. In particular, elevated blood pressure variability, abnormal blood pressure dipping patterns, and markers of vascular stiffness are linked to subsequent cognitive decline and an increased risk of developing incident dementia.
- The association between late-life hypertension control and cognition is less consistent, perhaps reflecting the passing of a critical period of intervention.

INTRODUCTION

Between 2000 and 2050, the global life expectancy at birth is expected to rise from 66 to 77, with the percentage of the population over the age of 65 years projected to rise

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from 7% to 16%.[1] This demographic shift poses an unprecedented challenge to the health care profession, necessitating an improved understanding of the health complications that accompany advancing age. Elevated arterial blood pressure (BP), or hypertension, affects an estimated 1.28 billion adults[2] and over two-thirds of adults aged over 65 years[3] and has been linked to cognitive decline and dementia.[4] Specifically, hypertension is recognized as the primary risk factor for vascular cognitive impairment and dementia (VCID) but is also associated with Alzheimer's disease (AD) dementia.[5] Dementia has a substantial global burden, affecting around 60 million individuals in 2023 with a projected 3 fold increase by 2050.[6] In the United States and other Western countries, the incidence of dementia appears to be declining, but given the rapidly aging population, its prevalence is actually increasing. A growing body of evidence supports hypertension as one of dementia's leading treatable risk factors, which could theoretically prevent or delay up to 20% of dementia cases.[7,8]

Hypertension is often underdiagnosed and poorly controlled, earning it the label of "silent killer" due to its subtle onset usually without overt clinical symptoms.[9] In fact, the World Health Organization estimates that 46% of adults with hypertension are unaware they even have the condition.[2] This underscores the importance of early hypertension detection and management, which if implemented effectively, may mitigate the deleterious effects across the life span and holds promise for alleviating the burden of the anticipated dementia epidemic. This review will consolidate the latest evidence on the relationship between hypertension and cognitive function, offering guidance for clinical practice and emphasizing the importance of early detection and management of hypertension to support brain health in the rapidly aging population.

OBSERVATIONAL RELATIONSHIP BETWEEN BLOOD PRESSURE AND COGNITION

The relationship between BP and cognitive function is complex and varies across different stages of life. Overwhelmingly, research has linked cumulative high BP exposure over the life span to an increased risk of dementia.[10] While this association is strongest for systolic blood pressure (SBP), it also holds true for diastolic blood pressure (DBP).[11] Midlife hypertension has the strongest association with later-life cognitive decline, but associations between elevated SBP in young adulthood, even below standard hypertension thresholds, and midlife cognitive decline have also been identified.[12–14] A nationwide cohort study of Swedish men further reinforces the concept of a cumulative accumulation of dementia risk from hypertension at any life stage, identifying that high SBP in adolescence is a risk factor for the subsequent development of dementia.[15]

A nationwide study of 4.5 million adults aged 60+ years found that the link between BP and dementia risk varies by dementia subtype. While the risk of overall dementia and AD showed a U-shaped association with SBP, with both high (SBP $\geq$160 mm Hg, for overall dementia, hazard ratio [HR] = 1.05, 95% confidence interval [CI] = 1.04–1.07; for AD, HR = 1.03, 95% CI = 1.01–1.05) and low (SBP <100 mm Hg, for overall dementia, HR = 1.13, 95% CI = 1.10–1.17; for AD, HR = 1.17, 95% CI = 1.13–1.22) levels having a significantly higher risk, the risk of probable vascular dementia (VaD) increased linearly with SBP levels (SBP <100 mm Hg, HR = 0.91, 95% CI = 0.83–1.01; SBP $\geq$160 mm Hg, HR = 1.23, 95% CI = 1.17–1.28).[16] All of these associations, however, are attenuated with increasing age.

In older adults, low BP has emerged as a risk factor for poor cognitive performance and even the development of dementia.[11,17–21] Some evidence suggests that higher BP may result in improved cognitive testing results, perhaps reflecting improved cerebral perfusion.[22] However, the relationship is confounded by other medical

comorbidities and the high prevalence of antihypertensive treatment by later life as well as residual confounding inherent in these observational studies. Taking those factors into account, numerous studies provide strong evidence that untreated hypertension in later life also contributes to dementia risk and progression.[23–25] Overall, the observational evidence is consistent that hypertension at any age is a risk factor for the development and progression of cognitive impairment.

MECHANISM OF HYPERTENSION'S ASSOCIATION WITH DEMENTIA

Hypertension places stress on blood vessels throughout the body and brain, resulting in damage that leads to neuronal injury or death through numerous diverse mechanisms (Fig. 1). Prolonged hypertension can lead to decreased cerebral perfusion

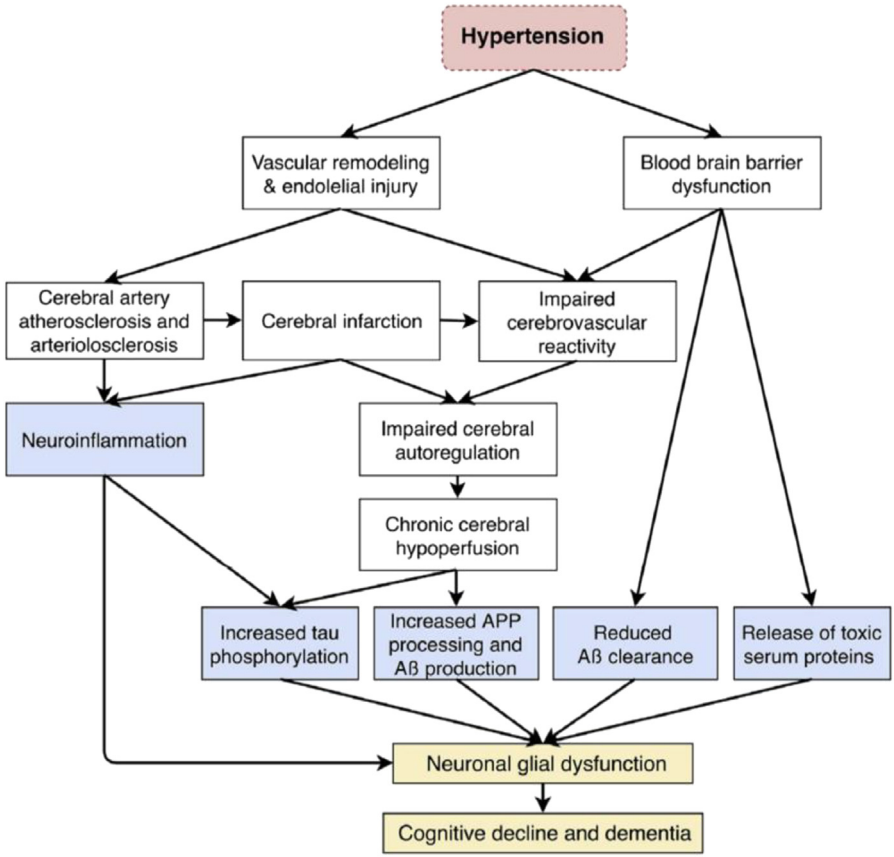

Fig. 1. Hypertension can cause structural and functional changes to cerebral vasculature, leading to impaired cerebral autoregulation, cerebral hypoperfusion, and cerebrovascular disease, such as lacunar infarctions and microhemorrhages. It can also promote neuroinflammation and Alzheimer's-specific pathophysiological processes, including tau phosphorylation and Aβ synthesis and oligomerization, each of which is known to cause neuronal and glial dysfunction and can lead to neurodegeneration and cognitive decline. Aβ, beta-amyloid; APP, amyloid precursor protein. (*From* Walker KA, Gottesman RF. The role of blood pressure and hypertension in dementia. In: Martin CR, Preedy VR, eds. *Diagnosis and Management in Dementia*. Academic Press; 2020:111-126. https://doi.org/10.1016/B978-0-12-815854-8.00008-2; with permission.)

caused by the proliferation of vascular smooth muscle cells, basal lamina alterations, endothelial hyalinosis, fibrosis, and, ultimately, luminal narrowing or vascular occlusion.[26–28] Moreover, it promotes the formation of free radicals and reactive oxygen species (ROS), which leads to cell apoptosis and breakdown of the blood–brain barrier (BBB).[29,30] Fluctuations in BP also induce stress on the vascular wall, affecting endothelial function,[31] and have been associated with cognitive deterioration and cerebrovascular pathology.[32] The association of episodic reduced cerebral perfusion and poorer cognitive test performance further reinforces this connection.[33–35]

The reduction in cerebral blood flow (CBF) secondary to chronic hypertension contributes to the neurodegenerative brain changes linked to hypertension. There is strong support that hypertension may lead to cognitive impairment through the occurrence of cerebral small vessel disease (CSVD),[36] evidenced by white matter hyperintensities (WMHs), microbleeds, lacunar infarcts, and enlarged perivascular spaces visible on brain MRI (**Fig. 2**). In particular, WMH on MRI has a well-established connection to hypertension.[37–40] Among all established MRI biomarkers, WMH has the strongest and most consistent association with cognitive impairment and dementia, both in cross-sectional and longitudinal analyses.[41–45]

Adults with hypertension also exhibit white matter microstructural disintegration, contributing to adverse effects on fluid intelligence, which highlights that WMH alone does not capture the entire pathophysiology of VCID.[46] Chronic cerebral hypoperfusion leads to white matter lesions (WMLs), gliosis and hyperintensities on MRI, microinfarcts, microhemorrhages, and even ischemic infarction, all of which have been associated with high BP and are known contributors to accelerated cognitive decline and dementia.[14,47–51]

Hypertension has also been associated with the accumulation of neurotoxic substances, such as beta-amyloid and phosphorylated tau, which can lead to neuronal dysfunction and neurodegenerative changes that are associated with AD cognitive impairment.[52–54] Hypertension is also linked to medial temporal lobe atrophy[55] and larger amounts of neuritic plaque in the neocortex and hippocampus, both hallmarks of AD.[56] Thus, it is possible that the association of hypertension with dementia is not only vascular but also mediated through the neurodegenerative pathways implicated in AD.

ANTIHYPERTENSIVE THERAPY'S EFFECT ON COGNITION

Observational studies have established that diuretics,[57] calcium-channel blockers (CCBs),[58] and ACE inhibitor/angiotensin receptor blockers[59] have a protective effect on cognitive function. A meta-analysis of randomized clinical trials with 92,135 participants found that BP lowering with antihypertensive medications compared with placebo or less-intensive BP lowering was significantly associated with a reduced risk of dementia or cognitive impairment (odds ratio [OR] = 0.93, 95% CI = 0.88–0.98) and cognitive decline (OR = 0.93, 95% CI = 0.88–0.99).[60] Another meta-analysis found antihypertensive use reduced dementia risk by 21% (risk ratio [RR] = 0.79, 95% CI = 0.70–0.89, I^2 = 68%).[11] However, these results are subject to unmeasured confounding given the heterogeneity of the trial cohorts, the interventions, the length of follow-up, and adjudication of cognitive outcomes.

Multiple trials have found no significant difference in cognitive outcomes between participants on active antihypertensive treatment versus control groups,[61–65] including Systolic Hypertension in the Elderly Program (SHEP), Hypertension in the Very Elderly Trial (HYVET), Heart Outcomes Prevention Evaluation-3 (HOPE-3) trial, and Study on Cognition and Prognosis in the Elderly (SCOPE). The Systolic Hypertension in Europe (Syst-Eur) trial study in the 1990s found that long-term antihypertensive treatment with

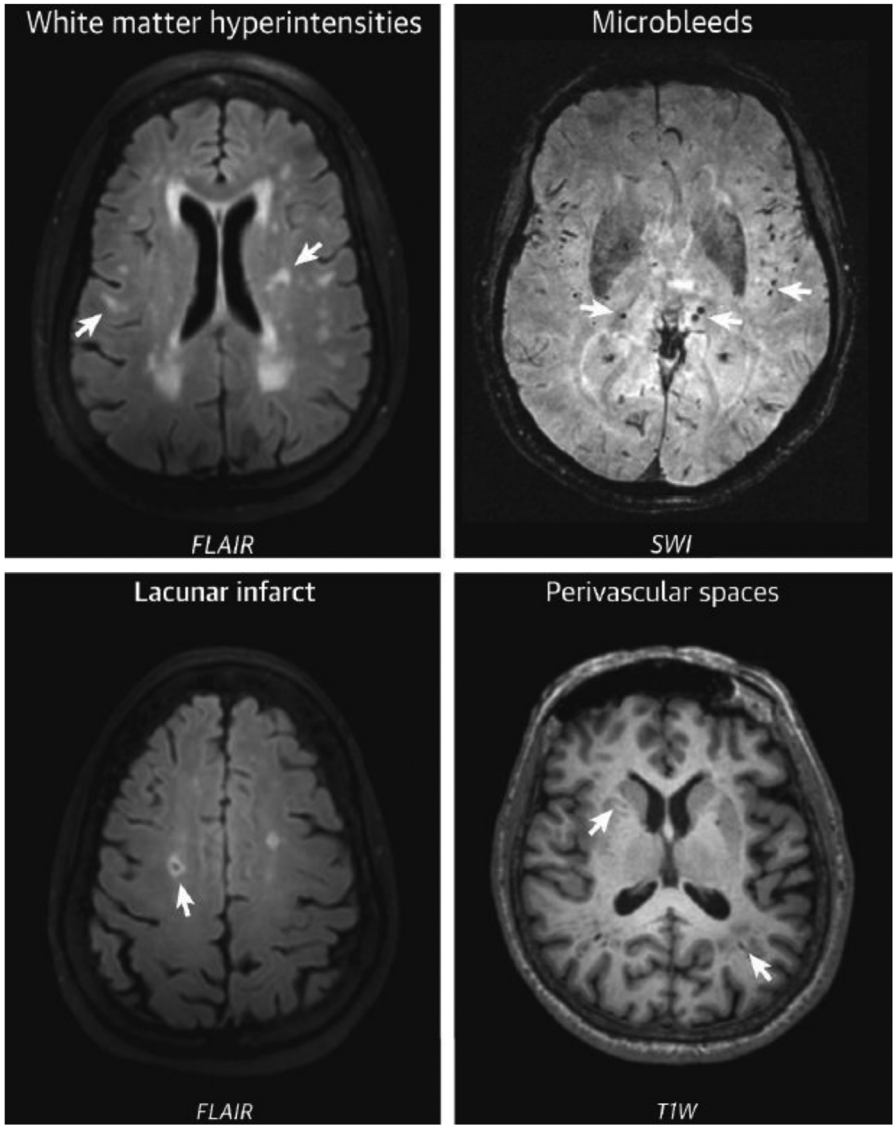

Fig. 2. Examples of Cerebral Small Vessel Disease on Brain MRI. *Arrows* indicate the presence of white matter hyperintensities, microbleeds, lacunar infarcts, and perivascular spaces. FLAIR, fluid attenuation inversion recovery; SWI, susceptibility-weighted imaging; T1W, T1 weighted. (*From* Amier RP, Marcks N, Hooghiemstra AM, et al. Hypertensive Exposure Markers by MRI in Relation to Cerebral Small Vessel Disease and Cognitive Impairment. JACC: Cardiovascular Imaging. 2021;14(1):176-185. https://doi.org/10.1016/j.jcmg.2020.06.040; with permission.)

nitrendipine, a dihydropyridine CCB, may reduce dementia risk by 50% (95% CI = 0.0–0.76; P = .05),[66] but had a total of only 33 dementia events in a cohort of 2418 participants followed for 2 years. The resulting imprecision of the effect size precludes definitive interpretation.

The Perindopril Protection Against Recurrent Stroke Study (PROGRESS) trial enrolled a cohort of 6105 participants with prior stroke or transient ischemic attack and found that BP lowering with perindopril and indapamide therapy produced a relative risk reduction of 19% (95% CI = 0.04–0.32; P = .01) for cognitive decline.[67] Additional analyses showed that this effect was due to a reduction in recurrent stroke. However, 2 other trials with a harmonized cohort of over 30,000 participants from Ongoing Telmisartan Alone and in Combination with Ramipril Global End Point Trial (ONTARGET) and Telmisartan Randomized AssessmeNt Study in ACE iNtolerant subjects with cardiovascular Disease (TRANSCEND), including a high proportion of participants with prior stroke, found no benefit of antihypertensive treatment on cognition (95% CI = 0.89–1.06; P = .53 and 95% CI = 0.95–1.27; P = .22, respectively).[68]

Action to Control Cardiovascular Risk in Diabetes MIND (ACCORD-MIND) was a substudy of the ACCORD trial in participants with type 2 diabetes that studied the effects of intensive BP lowering and glycemic control on cognitive function and brain volume.[69] The study found no significant differences in cognitive function between the intensive and standard BP treatment groups (MD = 0.32 for Digit Symbol Substitution Test score, 95% CI = −0.28–0.91; P = .2997), though there was a significantly higher total brain volume on MRI after 40 months in the group receiving intensive glycemic intervention (MD = 4.6 mL, 95% CI = 2.0–7.3; P = .0007). This finding introduces the possibility that structural changes related to hypertension may precede functional changes.

Despite the prior research, there continued to be equipoise because none of these trials conducted a follow-up period exceeding 4 years that also included a thorough expert review of cases involving dementia and mild cognitive impairment (MCI). Attempting to reduce the uncertainty regarding antihypertensive treatment and cognition, the Systolic Blood Pressure Intervention Trial Memory and Cognition in Decreased Hypertension (SPRINT-MIND)[70] ancillary to the SPRINT trial was designed with more rigor and longer follow-up. SPRINT-MIND found that intensive SBP control (<120 mm Hg; n = 4278) compared with a standard SBP treatment goal (<140 mm Hg; n = 4385) in patients with hypertension significantly reduced the risk of MCI by 19% (HR = 0.81, 95% CI = 0.70–0.95, P = .01) and combined MCI/probable dementia by 15% (HR = 0.85, 95% CI = 0.74–0.97, P=.02), but not dementia alone (HR = 0.83, 95% CI = 0.67–1.04, P = .10). Importantly, there were no clinically meaningful adverse effects of intensive SBP control in older patients, those with low baseline DBP, borderline kidney disease, or other pertinent subgroups. The results of the trials are summarized in **Table 1**.

Not all antihypertensives have neuroprotective properties, as they each have different mechanisms of action. Several studies have shown that dihydropyridine CCBs do have neuroprotective effects,[66,71–75] attributed to their high lipophilicity and ability to cross the BBB to regulate intracellular calcium levels. High intracellular calcium levels lead to vasoconstriction, reducing CBF, and have been associated with increased production of β-amyloid peptide,[76] ROS,[77] and tau accumulation,[78] all of which are linked to the development of AD and VaD. Dihydropyridines and CCBs, in general, work by blocking L-type voltage-gated calcium channels (L-VGCCs) to prevent high levels of intracellular calcium, thus mitigating its role in the pathogenesis of AD and VaD. This mechanism is illustrated in **Fig. 3**.

Several interesting substudies analyzing brain MRI at baseline and follow-up have emerged out of the SPRINT-MIND trial. One substudy showed that intensive BP treatment, compared with standard treatment, was associated with a smaller increase of WMLs (644.5 vs 1258.1 mm^3), a smaller decrease in fractional anisotropy (mean

Table 1
Characteristics of the selected randomized clinical trials examining the effect of antihypertensive therapy on cognition

Study	Total N (Treatment Group)	Age (SD) at Baseline	Follow-up (years)	Intervention/ Antihypertensive Agents Studied	Outcome of Interest	Effects of Treatment
SHEP[64]	N = 4608 (2317)	74	5	Diuretic and β-blocker vs placebo	Cognition	No effect
HYVET[62]	N = 3336 (1687)	84 (3)	2.2	Diuretic and ACE inhibitor vs placebo	Dementia	No effect
HOPE-3[65]	N = 1626 (Candesartan/ HCTZ = 405, rosuvastatin = 401, Combination = 406)	74 (3.5)	5.7	Candesartan/ hydrochlorothiazide, rosuvastatin, or their combination vs placebo	Cognition	No effect
SCOPE[61]	N = 4964 (2477)	76	4	ARB vs placebo	Cognition and dementia	No effect
Syst-Eur[59]	N = 2418 (1238)	70 (7)	2	CCB ± diuretic vs placebo	Dementia	Protective
PROGRESS[67]	N = 6105 (3054)	64 (10)	4	ACE inhibitor ± diuretic vs placebo	Cognition and dementia	Protective for patients with recurrent stroke
ONTARGET[68]	N = 25,620 (ARB = 8542, ACE inhibitor = 8576, combination = 8502)	66 (7)	4.6	ACE inhibitor, ARB, or their combination vs placebo	Cognition	No effect
TRANSCEND[68]	N = 5231 (2972)	67 (7)	4.7	ARB vs placebo	Cognition and dementia	No effect
ACCORD-MIND[95]	N = 2977 (1358)	62 (6)	3.3	Intensive glycemic treatment (HbA1c <6%) vs standard (HbA1c 7%–7.9%) and intensive BP treatment (<120 mm Hg) vs standard (<140 mm Hg)	Cognition	No effect
SPRINT-MIND[70]	N = 9361 (4678)	68	3.3	Intensive BP treatment (<120 mm Hg) vs standard treatment (<140 mm Hg)	MCI and dementia	Intensive BP treatment was protective against MCI and MCI/dementia combined

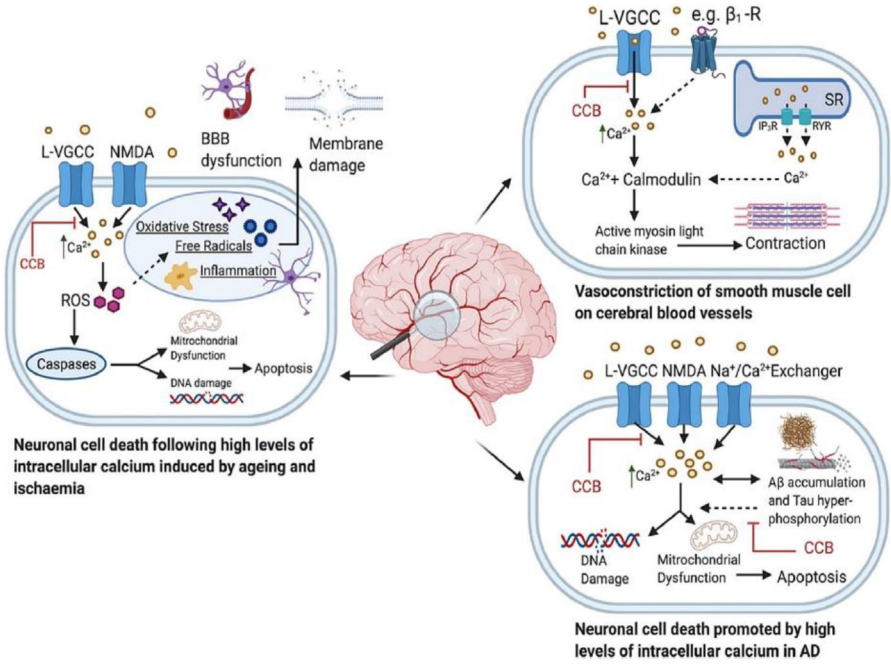

Fig. 3. Role of intracellular calcium in the pathogenesis of AD and VaD, illustrating the potential neuroprotective effect of CCBs. Aβ, amyloid-β; AD, Alzheimer's disease; β1-R, beta1-adrenoceptor; BBB, blood–brain barrier; CCB, calcium channel blocker; IP3R, 1,4,5-triphosphate receptor; L-VGCC, L-type voltage-gated calcium channel; NDMA, N-methyl-d-aspartate; ROS, reactive oxygen species; RYR, ryanodine receptor; SR, sarcoplasmic reticulum. (Created with BioRender.com.)

change, -0.0026 vs -0.0062 for deep white matter [DWM] regions and -0.0011 vs -0.0040 for superficially located white mattter (SWM) regions), a smaller increase in mean diffusivity (0.0044 vs 0.0184 for DWM regions and 0.0038 vs 0.0073 for SWM regions), and a larger increase in CBF (4.6 mL/100 mg/min vs 3.7 mL/100 mg/min).[79] Another interesting substudy in SPRINT-MIND found that at 48 months follow-up, in the intensive BP control arm, WMH volume increased by 0.28 versus a larger increase of 0.92 mL in the standard BP control arm ($P = .004$).[80] A similar result was also found in an analysis of 314 participants in the ACCORD-MIND trial, where those in the intensive BP control arm had a WMH increase of 0.67 versus 1.16 mL in the control arm ($P = .001$).[81] Similar results were found in a small trial of older participants with established CSVD.[82] Finally, an analysis of MRI data from SPRINT in combination with INFINITY (Intensive vs Standard Blood Pressure Lowering to Prevent Functional Decline in Older People) found that intensive SBP control was associated with less WMH progression than the standard SBP treatment (0.29 vs 0.48 mL, $P = .03$).[83] The relative consistency of this finding across different clinical trials shows that a putative benefit of hypertension control on cognition could be mediated through a reduction of WMH progression, in addition to reducing stroke risk.

OTHER BLOOD PRESSURE METRICS AND MEASUREMENT METHODS

New BP metrics and monitoring methods have provided valuable insights into the relationship of other BP-related factors with cognition, offering a more comprehensive

understanding of this relationship. Blood pressure variability (BPV) reflects fluctuations in BP over distinct time scales, encompassing very short-term to long-term variations. Higher BPV has emerged as an indicator of impaired cardiovascular regulation, correlating with adverse outcomes such as stroke, coronary artery disease, heart failure, end-stage renal disease, and dementia incidence.[32]

The Three-City Study found a significant association between a higher BPV and an increased risk of incident dementia. In this community-based older adult cohort, participants in the highest decile of BPV over the 8 year follow-up period had a 77% increased risk of dementia compared with those in the lowest decile (P = .007).[84] Work with the S.AGES cohort (Sujets AGES—Aged Subjects) linked higher visit-to-visit BPV to poorer cognitive function and greater risk of dementia, independent of baseline BP levels (1-SD increase of systolic BPV: HR = 1.23, 95% CI = 1.01–1.50, P = .04), with similar results reported for diastolic BPV (P < .01).[47] Substantiating this, the Coronary Artery Risk Development in Young Adults (CARDIA) study[85] demonstrated that elevated long-term systolic and diastolic BPV, independent of hypertension, in young adulthood are linked to worse midlife psychomotor speed (β standard error [SE]: −0.025 [0.006] and −0.029 [0.007], respectively; P<.001) and poorer performance on verbal memory tests (β [SE]: −0.016 [0.006] and −0.021 [0.007], respectively; P<.05). An analysis of data from the Atherosclerosis Risk in Communities study supports these findings, also associating greater visit-to-visit BPV during midlife with lower cognitive function over 25 years of follow-up.[86]

A post hoc analysis of the SPRINT-MIND trial found that the rate of dementia increased by ascending BPV tertile, even in cases with excellent BP control.[87] In the SPRINT-MIND cohort, compared to the lowest tertile of BPV, the highest tertile of BPV increased the risk of dementia in both unadjusted (HR = 2.36, 95% CI = 1.77–3.15) and adjusted (HR = 1.69, 95% CI = 1.25–2.28) models. They also found that the highest tertile of BPV was associated with both MCI (adjusted HR = 1.40, 95% CI = 1.14–1.71) and the composite of dementia/MCI (adjusted HR = 1.43, 95% CI = 1.20–1.71). A meta-analysis of studies found dementia and cognitive impairment to be associated with higher systolic BPV (OR = 1.25, 95% CI = 1.16–1.35), mean systolic pressure (OR = 1.12, 95% CI = 1.02–1.29), DBPV (OR = 1.20, 95% CI = 1.12–1.29), and mean diastolic pressure (OR = 1.16, 95% CI = 1.04–1.29). Further, they found that the BPV effect size was stronger than the mean BP effect size on dementia or cognitive impairment.[88] However, these are all secondary and post hoc analyses. Additional research is needed to understand how to reduce BPV and to determine if its reduction lowers the risk of cognitive impairment or dementia.

In addition to BPV, pulse pressure (PP) and pulse wave velocity (PWV) are highly correlated with cognitive decline. Elevated levels of PP and PWV have been associated with poorer cognitive test performance[89] and glymphatic dysfunction, a hallmark of AD. Moreover, PP and PWV serve as markers of arterial stiffness,[90] suggesting they may be better indicators of the chronicity of hypertension than hypertension itself (More information is provided in Tuday and colleagues[91]). However, similar to BPV, additional research is needed to determine their potential as a therapeutic target for dementia.[92]

24-hour Ambulatory Blood Pressure Monitoring

24-hour ambulatory BP monitoring (ABPM) is another method of BP monitoring that captures the physiologic variance of BP throughout the day, termed the “circadian BP rhythm.” A normal pattern is characterized by fluctuations in a diurnal pattern and a normal 10% to 20% drop in nighttime BP values.[32] Patients can be categorized as “dippers” (≥10%) who exhibit a normal dipping BP pattern, “nondippers” (<10%), and “reverse dippers” (dipping <0%). Longitudinal studies employing 24-hour ABPM

reveal that abnormal dipping (less than 10%) precedes cognitive decline, emphasizing the significance of analyzing BP profiles beyond office measurements.

A meta-analysis of studies found dippers to have a 51% (OR = 0.49, 95% CI = 0.35–0.69) lower risk of abnormal cognitive function (composite of cognitive impairment or dementia) and a 63% (OR = 0.37, 95% CI = 0.23–0.61) lower risk of dementia alone, compared to nondippers.[6] Moreover, they found reverse dippers to have a 6 fold higher risk (OR = 6.06, 95% CI = 3.15–11.64) of abnormal cognitive function compared to the normal dippers. Reverse dippers were also found to perform worse in global function neuropsychological tests compared with both normal and nondippers (standardized mean difference [SMD] = −0.66, 95% CI = −0.93 to −0.39).[6] This evidence is in line with studies that found that nondipping and reverse-dipping patterns are associated with WMH, silent cerebral infarcts, and brain atrophy.[93]

RECOMMENDATIONS

Given the strong association between midlife hypertension and late-life cognitive function, a life course approach is necessary in clinical practice to identify individuals who may be at elevated risk of cognitive decline secondary to uncontrolled BP. According to the 2017 American College of Cardiology/American Heart Association Hypertension Clinical Guideline,[94] the standards for accurate BP measurements are as follows:

- Normal: <120/<80 mm Hg
- Elevated: 120 to 129/<80 mm Hg
- Stage 1 Hypertension: 130 to 139 or 80 to 89 mm Hg
- Stage 2 Hypertension: ≥140 or ≥90 mm Hg

This guideline recommends that the diagnosis of hypertension should be confirmed with out-of-office self-monitoring of BP, such as home or 24-hour ABPM, which affords clinicians a more complete picture of one's BP profile and can tailor intervention at the individual level. Based on the cognitive results of SPRINT-MIND and the overall cardiovascular and mortality benefits of intensive SBP reduction to less than 120 mm Hg,[10,95–98] it is reasonable to recommend that patients with stage 1 or stage 2 hypertension receive antihypertensive medication to target a goal of less than 120/80 mm Hg, especially those at high risk for cognitive decline and dementia. In patients with elevated BP but not overt hypertension, a more conservative approach focusing on diet, exercise, and other nonmedication therapies seems to be reasonable. However, if that approach fails, initiation of antihypertensive medication should also be considered.

SUMMARY

This review underscores the critical role of hypertension management across the life span in reducing the risk of cognitive decline and dementia. Observational and clinical evidence solidifies the association between hypertension and various forms of cognitive impairment, indicating that effective BP control can serve as a pivotal intervention in preserving cognitive health. Novel metrics and monitoring methods, such as BPV and 24-hour ABPM, offer deeper insights into the complex relationship between hypertension and cognitive function, suggesting more personalized approaches to hypertension management. The findings advocate for a life course approach to BP monitoring and control, emphasizing early detection and intervention. Ultimately, addressing hypertension effectively not only promises to enhance cardiovascular health but also to mitigate the burgeoning global burden of dementia, highlighting

the necessity for health care systems to adapt and prioritize hypertension management in clinical practice.

CLINICS CARE POINTS

- While there is no definitive evidence that control of hypertension prevents dementia, there is compelling mechanistic evidence supporting the association between hypertension and Alzheimer's and VCID. This underscores the critical role of hypertension prevention rather than treatment in clinical practice for the preservation of cognitive function.
- Nonetheless, given the benefit in reducing the incidence of MCI seen in SPRINT-MIND and the difficulty of studying this outcome, we recommend aggressive control of hypertension, particularly among younger patients, for the maintenance of cognitive function in later life.
- We recommend at-home BP monitoring and lifestyle modifications in conjunction with antihypertensive therapy as a strategy to personalize care for individuals with hypertension or those at risk for dementia.

DISCLOSURE

Dr A. de Havenon has received consultant fees from Novo Nordisk, royalty fees from UpToDate, and has equity in TitinKM and Certus. Dr K.N. Sheth reports compensation from Sense and Zoll, for data and safety monitoring services; compensation from Cerevasc, CSL Behring, Rhaeos and Astrocyte for consultant services; a patent for Stroke wearables licensed to Alva Health. Dr A.M. Brickman serves as a scientific advisor/consultant to Cognito Therapeutics, Cognition Therapeutics, and CogState. He serves on data safety and monitoring boards for Albert Einstein College of Medicine and University of Illinois. Dr A.M. Brickman has a patent for white matter hyperintensity quantification (patent # 9867566) and a patent pending for microbleed detection (publication #20230298170). Dr E.A. Mistry receives consultant fees from AbbVie and RAPID AI.

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