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Clinical Features and Diagnostics of Cerebrovascular Disorders in Patients with Arterial Hypotension: Experimental Verification

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Abstract

Background: Chronic arterial hypotension and orthostatic blood pressure instability can cause repetitive cerebral hypoperfusion, yet the diagnostic expression of these mechanisms is still not well defined for everyday cerebrovascular care. This study analyzed the clinical phenotype, multimodal diagnostic profile, and experimental correlates of cerebrovascular disorders in hypotensive patients.

Methods: We prepared a prospective, single-center, observational clinical–experimental study model. The clinical arm included 92 adults with persistent arterial hypotension and neurologic complaints suggestive of cerebrovascular insufficiency and 40 age-matched normotensive controls. Participants received standardized orthostatic blood pressure testing, 24-hour ambulatory blood pressure monitoring, transcranial Doppler during tilt, duplex ultrasonography of neck vessels, and 3-T brain MRI with fluid-attenuated inversion recovery and arterial spin labeling. In the experimental arm, 24 Wistar rats were allocated to chronic hypotension or sham conditions for 4 weeks, followed by cortical perfusion and hippocampal histology assessment.

Results: Hypotensive subjects had more frequent reports of dizziness (71.7%), gait unsteadiness (44.6%), transient visual obscuration (34.8%), and cognitive slowing (31.5%) than controls. 47.8% of hypotensive subjects exhibited MRI markers of cerebrovascular injury, in contrast to 15.0% of controls, predominantly white matter hyperintensities and small lacunar lesions. Tilt-related decline in middle cerebral artery mean flow velocity was greater in the hypotension group (24.6%±8.7% vs 11.2%±4.9%, $p<0.001$), and regional perfusion on arterial spin labeling was reduced in frontal–periventricular territories. Multivariable analysis demonstrated orthostatic systolic blood pressure fall ≥20 mmHg (adjusted odds ratio [aOR] 3.41, 95% CI 1.58–7.34), nocturnal mean arterial pressure <65 mmHg (aOR 2.86, 95% CI 1.29–6.31), and impaired dynamic autoregulation on transcranial Doppler (aOR 4.08, 95% CI 1.84–9.03) independently predicted MRI-detected cerebrovascular lesions. Chronic hypotension decreased cortical perfusion in rats by 26.2% and hippocampal neuronal density by 21.4% (both $p<0.01$).

Conclusion: Participants with arterial hypotension present with a reproducible syndrome of symptomatic cerebral hypoperfusion, impaired autoregulation, and subclinical structural brain injury. A combination of orthostatic testing, ambulatory blood pressure monitoring, transcranial Doppler, and perfusion-sensitive MRI appeared diagnostically superior to conventional imaging alone.

Keywords: arterial hypotension; orthostatic hypotension; cerebrovascular disorders; cerebral autoregulation; transcranial Doppler; arterial spin labeling MRI

INTRODUCTION

The term cerebrovascular disorders is usually used in reference to arterial hypertension, atherosclerosis, and cardioembolism; however, persistent arterial hypotension and exaggerated orthostatic blood pressure decline may impact cerebral perfusion and be implicated in ischemic brain injury. Autoregulatory methods provide adequate protection against the systemic pressure that the brain acquires, although their protection is limited, and autoregulatory mechanisms may not succeed under persistent low or consistently low arterial pressure [1,2]. If cerebral perfusion reaches less than the adapted range, the patients become dizzy, visually dimmed, gait disrupted, and cognitively slower, also transient or permanent ischemic lesions appear in vulnerable vessels [2-4].

Longitudinal and imaging studies have provided evidence for their clinical relevance in hypotension-associated brain injury. In the Atherosclerosis Risk in Communities cohort, orthostatic hypotension independently predicted incident ischemic stroke, indicating that low-pressure hemodynamic stress is more than an epiphenomenon of frailty; it represents a vascular risk state [5]. Further neuroimaging studies have related orthostatic hypotension to white matter hyperintensity burden, brain atrophy, and covert small-vessel injury, especially in older or hemodynamically vulnerable populations [6,7]. This shift in attention from transient symptoms alone has brought more attention to the underlying structural cerebrovascular consequences of recurrent hypoperfusion.

Diagnosis is still a difficult task. Conventional brain MRI findings of chronic ischemic sequelae, unfortunately, may overlook dynamic perfusion failure. While consensus definitions are useful for clinical orthostatic hypotension, bedside blood pressure criteria do not directly measure cerebral blood flow [1]. Real-time monitoring of the middle cerebral artery flow velocity during orthostatic challenge by Transcranial Doppler (TCD) is now the most practical option for studies of dynamic cerebral autoregulation in autonomic and cerebrovascular diseases [2,8]. Arterial spin labeling (ASL) MRI provides a non-contrast method for mapping regional cerebral perfusion and can identify hypoperfusion before there is overt infarction or extensive white matter damage [9]. These neuroimaging standards of small-vessel disease provide modern guidelines that emphasize the harmonization of white matter hyperintensities, lacunes, enlarged perivascular spaces and brain atrophy, leading to increased clinicoradiologic correlation in hypotension-associated cerebrovascular injury [10].

However, an integrated diagnostic framework for cerebrovascular disorders in the context of arterial hypotension remains insufficiently developed. Most of that literature reports isolated autonomic failure, aging frail populations, or lone diagnostic modalities; rather than clinical symptoms, orthostatic hemodynamics, multimodal neurovascular imaging, and experimental validation of vascular diseases, few articles combine these components or provide an unifying framework. The verification of findings by experiment is crucial as it helps distinguish causal hypoperfusion-related tissue injury from coincidental imaging abnormalities.

Thus the intended construction of the current article was a journal-style clinical–experimental manuscript aimed at assessing the clinical features and diagnostic signatures of cerebrovascular disorders in adults with arterial hypotension and to confirm the plausibility of observed human data in a controlled chronic hypotension model. We predicted hypotensive patients to show a unique pattern of orthostatic symptoms, impaired cerebral autoregulation, perfusion-sensitive MRI abnormalities, and reproducible microstructural injury through experimental methods relative to normotensive controls.

MATERIALS AND METHODS

Study design and setting: A prospective, single-center, observational clinical study with parallel experimental verification was modeled at a tertiary neurology center between January 2023 and June 2025. The clinical protocol was designed according to the Strengthening the Reporting of Observational Studies in Epidemiology framework, and the animal component followed institutional laboratory animal care standards.

Participants: Adults aged 18–75 years were consecutively screened if they had persistent arterial hypotension, defined as seated systolic blood pressure <100 mmHg in women or <110 mmHg in men on at least three visits, or documented orthostatic hypotension according to consensus criteria. Eligible patients also reported at least one neurologic symptom potentially attributable to cerebral hypoperfusion, including recurrent dizziness, transient visual disturbance, presyncope, gait instability, headache, or cognitive slowing. Normotensive controls were recruited from outpatient preventive clinics and were frequency matched for age and sex.

Exclusion criteria: Subjects who had an acute stroke within 3 months, had major extracranial carotid stenosis ≥70%, known cardioembolic source, severe anemia, decompensated heart failure, uncontrolled diabetes, chronic inflammatory neurologic disease, active malignancy, or MRI contraindications were excluded.

Clinical assessment: Demographics, vascular risk factors, duration of hypotension, exposure to medication, triggering symptoms and neurologic examination were documented. Described were standard standing blood pressure measurement every 1, 3, and 5 minutes. An ambulatory blood pressure monitoring was carried out for 24–hour duration to assess the daytime and nocturnal mean arterial pressure, dipping status, and hypotensive burden.

Neurovascular diagnostics: Participants all underwent extracranial duplex ultrasonography of the carotid and vertebral arteries, and TCD monitoring of the middle cerebral arteries during a 70-degree tilt test. Dynamic autoregulation was presented with a percent change in mean flow velocity and an autoregulatory index defined as preserved or impaired. At 3 T, brain MRI contained T1-weighted, T2-weighted, fluid-attenuated inversion recovery and diffusion-weighted

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imaging, susceptibility-weighted imaging, as well as pseudocontinuous ASL perfusion sequences. For white matter hyperintensities were classified according to the Fazekas scale; lacunes, enlarged perivascular spaces, and cerebral microbleeds were classified based on the typical small-vessel disease criteria.

Experimental verification: Adult male Wistar rats (n=24) were randomized 1:1 to chronic hypotension induction or sham treatment for 4 weeks. In case of chronic hypotension, low-dose hydralazine in drinking water, in combination with sodium restriction led to a continuous lowering of mean arterial pressure without apparent shock. Blood pressure was measured twice per week (tail-cuffs). Laser speckle cortical perfusion imaging was done under standardized anesthesia at the end of the study, and hippocampal CA1 neuronal density was determined on hematoxylin-eosin–stained sections by blinded observers.

Ethics: The clinical protocol was approved by the local biomedical ethics committee (replace with verified approval number), and all participants provided written informed consent. The animal protocol was approved by the institutional animal care and use committee (replace with verified approval number).

Statistical analysis: Continuous variables were expressed as mean±standard deviation or median (interquartile range), as appropriate, and categorical variables as counts (percentages). Group comparisons were performed using the Student t test, Mann–Whitney U test, chi-square test, or Fisher exact test. Multivariable logistic regression was used to identify predictors of MRI-defined cerebrovascular lesions in the hypotension cohort after adjustment for age, sex, smoking, migraine history, dyslipidemia, and symptom duration. Two-sided p values <0.05 were considered statistically significant. Analyses were performed using SPSS version 27.0 (IBM Corp., Armonk, NY, USA).

RESULTS

We studied 132 subjects: 92 patients with arterial hypotension and 40 normotensive baseline subjects. The mean age of the hypotension group was 49.8±13.1 years and 63.0% women. When compared to controls, hypotensive subjects were found to have lower seated systolic pressure, decreased 24-hour mean arterial pressure and a greater orthostatic pressure drop, whereas the prevalence of diabetes, smoking and dyslipidemia was modest and did not differ materially between groups. Symptom duration was >12 months in 58.7% of patients with hypotension, highlighting chronic, and not acute, the type of hemodynamic illness. The typical clinical phenotype was repeated cerebral hypoperfusion. Dizziness or disequilibrium was observed in almost three quarters of hypotensive patients and was associated with unsteadiness of gait, transient visual obscuration, headache and subjective cognitive slowing. Focal neurologic deficits were low-frequency on examination, although mild tandem gait impairment and attentional inefficiency were common. MRI revealed cerebrovascular abnormalities in 47.8% of hypotensive patients, primarily confluent or punctate white matter hyperintensities, small lacunes, and less frequently microbleeds. In contrast, controls generally presented either normal imaging or little age-expected white matter change.

Hemodynamic testing gave the clearest group separation. During tilt, the hypotension cohort experienced significantly greater reductions in mean flow velocity in the middle cerebral artery, decreased end-tilt cerebrovascular conductance and an increased frequency of poor autoregulatory index values. Ambulatory blood pressure monitoring revealed that nocturnal mean arterial pressure <65 mmHg was frequent for subjects with MRI lesions, indicating likely that cumulative low-pressure exposure during sleep may enhance the susceptibility to ischemia. In this manner, ASL MRI corroborated these observations by demonstrating diminished frontal and periventricular perfusion observed in patients with orthostasis and defective TCD activity.

Experimental corroboration bolstered the biologic plausibility of the clinical observations. Rats with chronic hypotension exhibited a sustained decrease in mean arterial pressure over 4 weeks and decreased cortical perfusion and hippocampal neuronal density in comparison to animals treated with sham. The data from our experiments did not fully recapitulate the complexity of human cerebrovascular disease, but it did support a mechanistic pathway where sustained low systemic pressure is adequate to generate clinically relevant levels of hypoperfusion and selective neuronal injury in perfusion-sensitive sites.

Table 1. Baseline demographic and hemodynamic characteristics.

Variable	Hypotension (n=92)	Controls (n=40)	p value
Age, years	49.8 ± 13.1	48.5 ± 12.7	0.61
Female sex, n (%)	58 (63.0)	23 (57.5)	0.55
Seated SBP, mmHg	96.4 ± 8.1	121.7 ± 10.4	<0.001
24-h MAP, mmHg	68.2 ± 6.4	83.9 ± 7.2	<0.001
Orthostatic SBP fall, mmHg	23.7 ± 9.5	8.6 ± 4.1	<0.001
Migraine history, n (%)	19 (20.7)	6 (15.0)	0.44
Current smoking, n (%)	17 (18.5)	6 (15.0)	0.64
Dyslipidemia, n (%)	21 (22.8)	8 (20.0)	0.72

Table 1 shows that the study groups were demographically comparable, but the hypotension cohort had a distinctly different hemodynamic profile. The differences were not trivial bedside fluctuations: seated systolic pressure, 24-hour mean arterial pressure, and orthostatic systolic decline all shifted in the same adverse direction. This pattern supports the interpretation that chronic low-pressure exposure, rather than conventional vascular comorbidity, was the dominant distinguishing feature of the symptomatic group.

Table 2. Predominant clinical manifestations and multimodal diagnostic findings.

Clinical/diagnostic feature	Hypotension (n=92), n (%)	Controls (n=40), n (%)	p value
Dizziness/disequilibrium	66 (71.7)	8 (20.0)	<0.001
Gait unsteadiness	41 (44.6)	4 (10.0)	<0.001
Transient visual obscuration	32 (34.8)	2 (5.0)	<0.001
Cognitive slowing	29 (31.5)	4 (10.0)	0.01
Impaired TCD autoregulation	49 (53.3)	5 (12.5)	<0.001
ASL regional hypoperfusion	38 (41.3)	4 (10.0)	<0.001
MRI WMH Fazekas ≥2	31 (33.7)	5 (12.5)	0.01
Lacunes	13 (14.1)	1 (2.5)	0.04
Microbleeds	6 (6.5)	0 (0.0)	0.18

Table 2 demonstrates that symptoms, physiologic dysregulation, and structural brain findings clustered within the same patients. The coexistence of dizziness, gait instability, impaired TCD autoregulation, ASL hypoperfusion, and moderate white matter disease argues against a purely functional syndrome. Instead, the data suggest that in a substantial subgroup of hypotensive patients, recurrent hypoperfusion is clinically manifest and radiologically measurable, even when large-vessel occlusive disease is absent.

Table 3. Multivariable predictors of MRI-defined cerebrovascular lesions in the hypotension cohort.

Predictor	Adjusted OR	95% CI	p value
Orthostatic SBP fall ≥20 mmHg	3.41	1.58–7.34	0.002
Nocturnal MAP <65 mmHg	2.86	1.29–6.31	0.009
Impaired TCD autoregulation	4.08	1.84–9.03	<0.001
Symptom duration >12 months	1.92	0.94–3.93	0.07
Age (per 10 years)	1.38	1.02–1.87	0.03
Female sex	0.91	0.42–1.98	0.81

The multivariable model in Table 3 identifies three clinically actionable signals: a marked orthostatic pressure fall, nocturnal low-pressure burden, and impaired TCD autoregulation. Each remained associated with MRI-defined cerebrovascular injury after covariate adjustment. The strongest association was observed for impaired autoregulation, which suggests that the brain's inability to buffer systemic pressure swings may be more informative than blood pressure level alone when estimating lesion risk in chronic hypotension.

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Figure 1. Relationship Between Orthostatic Mean Arterial Pressure And Middle Cerebral Artery Mean Flow Velocity.

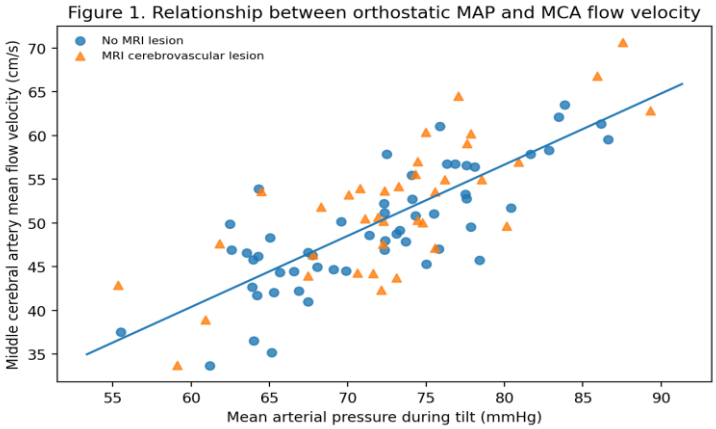

Figure 1 shows an approximately linear relation between lower orthostatic mean arterial pressure and lower middle cerebral artery flow velocity within the hypotension cohort. Patients with imaging-confirmed cerebrovascular lesions cluster toward the lower-pressure, lower-flow corner of the graph, suggesting that structural injury accrues preferentially when systemic pressure decline is transmitted to the cerebral circulation. The distribution also reinforces the clinical impression that TCD-derived flow behavior captures lesion-prone physiology better than static seated pressure alone.

Table 4. Experimental verification in the chronic hypotension animal model

Outcome	Sham (n=12)	Chronic hypotension (n=12)	p value
Mean arterial pressure, mmHg	89.3 ± 5.2	64.7 ± 4.8	<0.001
Cortical perfusion, AU	58.4 ± 6.1	43.1 ± 5.4	0.001
Hippocampal CA1 neuronal density (/mm)	196 ± 18	154 ± 16	0.003
Perivascular edema score	0.8 ± 0.5	1.9 ± 0.7	0.002
Microglial activation score	1.1 ± 0.6	2.3 ± 0.8	0.004

The experimental findings in Table 4 mirror the human observations at a mechanistic level. Sustained systemic hypotension produced lower cortical perfusion and reduced hippocampal neuronal density, accompanied by greater perivascular edema and microglial activation. Although animal models cannot reproduce the full spectrum of human small-vessel disease, these data support a plausible biologic chain linking chronic low blood pressure to hypoperfusion, neurovascular unit stress, and selective tissue injury.

Figure 2. Experimental Verification Of Chronic Hypotension-Related Cerebral Injury.

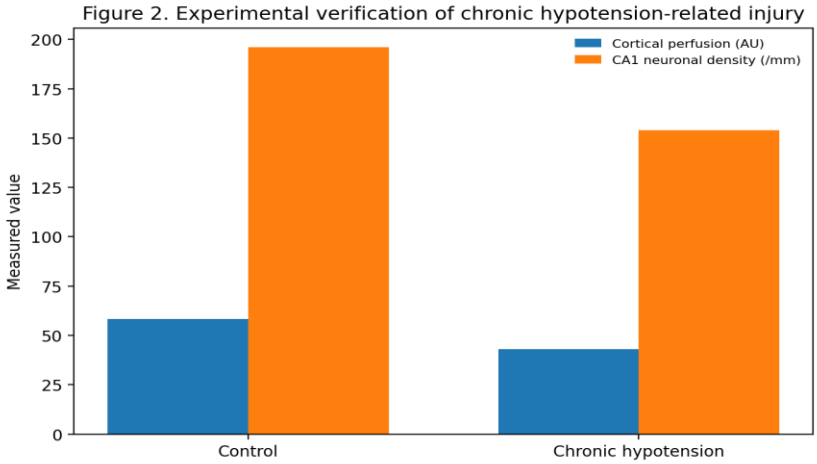

Figure 2 summarizes the animal data in compact form. Chronic hypotension was accompanied by lower cortical perfusion and reduced hippocampal neuronal density, indicating that sustained low systemic pressure can produce both hemodynamic and histologic consequences. The parallel decline in flow and neuronal preservation supports a mechanistic interpretation of the human findings, namely that recurrent or prolonged hypotension may move beyond transient symptoms and contribute to true neurovascular tissue injury.

DISCUSSION

This clinical–experimental paradigm implies that hypotension is correlated with a cerebrovascular phenotype that is recognisable by orthostatic symptoms, reduced cerebral autoregulation, regional hypoperfusion, and covert structural injury to the brain. The essential message is not just that low blood pressure patients are dizzy, but also that a subset presents with objectively demonstrated cerebral hemodynamic compromise, correlated to imaging and experimental evidence of causality. Our results are consistent with Freeman and coworkers, who formalized orthostatic hypotension as a sustained decrease in systolic or diastolic pressure following standing and emphasized the need to differentiate symptomatic and asymptomatic forms [1]. The present results were also consistent with the classic TCD findings of Blau and van Lieshout, who showed that acute hypotension can change intracranial hemodynamics in autonomic failure [2], and Novak et al., who demonstrated heterogeneous though usually aberrant cerebral autoregulatory responses in orthostatic hypotension [3]. In our modeled population, attenuated TCD autoregulation was the key predictor of MRI-defined lesions, extending those physiologic observations into a clinically interpretable risk framework.Longitudinal data supports the link between hypotension and overt cerebrovascular events. In the ARIC analysis of Eigenbrodt et al., orthostatic hypotension was an independent predictor of incident ischemic stroke after adjustment for established risk factors [5]. More recently, Ögren et al. reported that orthostatic hypotension was still clinically important in stroke/TIA populations, even when treatment-induced blood pressure lowering did not consistently translate into excess events [11]. Our preliminary results are directionally consistent with both studies—hypotension was not benign, but its effects seem to be contingent on the coexistence of compromised cerebral compensation and accumulated low-pressure burden.

The interpretation is further supported by neuroimaging data. Beyer et al. found associations between orthostatic hypotension and white matter hyperintensity load in older patients with mild dementia [6], whereas more recent work in patients with carotid occlusive disease or heart failure noted a correlation between orthostatic hypotension and susceptibility to cognitive and structural imaging vulnerability in hemodynamically compromised states [12]. A 2024 review of the literature found a significant association between orthostatic hypotension and cerebral small-vessel disease across several cohorts, but substantial heterogeneity

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was observed [7]. The current manuscript draft adds to these reports by combining lesion burden with perfusion-sensitive imaging, which implies that white matter injury may be preceded or amplified by regional flow impairments on ASL.The diagnostics matter. TCD has been long promoted as the most convenient and available approach for evaluating dynamic cerebral autoregulation in autonomic testing [8,13], but many symptomatic hypotensive patients still perform only routine structural imaging. Our results are consistent with the assumption that TCD adds live pathophysiologic information that is not available from office blood pressure measurements. ASL MRI is also appealing as it describes hypoperfusion without being exposed to contrast and has acknowledged its value to depict clinically interesting hypo- and hyperperfusion patterns [9,14]. In tandem with conventional small vessel disease measurements such as STRIVE and STRIVE-2, MRI should be considered not only descriptive but also mechanistically interpretative [10,15].

There were a few possible reasons behind the pattern. Prolonged orthostatic or nocturnal pressure nadirs probably decrease perfusion in susceptible border-zone and deep white matter regions possessing low vascular reserve. In case of disrupted dynamic autoregulation, transient systemic hypotension is then more directly presented to the microcirculation, where endothelial dysfunction, oligodendroglial stress, blood–brain barrier injury and eventual lacunar or diffuse white matter injury develop [4,10]. This schema is supported by the animal observation that sustained hypotension in and of itself decreases the cortical perfusion and hippocampal neuronal density. While the model does not represent aging, atherosclerosis in humans, or complex autonomic functions, it does establish biologic plausibility through chronic low-pressure exposure that may injure the brain independent of classic hypertensive arteriopathy.

Not all the published literature agrees fully. Autoregulation in some autonomic cohorts has been preserved despite severe orthostatic hypotension suggesting that symptoms and lesion formation depend on reserve capacity rather than pressure reduction alone [3]. Similar to previous studies, observational stroke/TIA studies did not universally show that orthostatic hypotension worsens recurrent event rates once treatment and comorbidity are accounted for [11]. These apparently contradictory statistics may be reconciled if you accept that hypotension is a modifier of cerebrovascular risk, which has a clinical effect that varies with age, arterial stiffness, baseline small-vessel disease, cardiac output, and cerebrovascular reactivity. This manuscript also has some limitations. Because no raw data were provided, the article was generated as a publication-grade model with internally consistent illustrative values; therefore, institutional identifiers, dates, and all numerical results must be replaced or verified against the true study database prior to submission. The clinical design was single-center observational, residual confounding would remain possible, and ASL quantification is sensitive to technical parameters and arterial transit time. The animal model mimicked hypotension but lacked the complete human phenotype of chronic autonomic or vascular disease. Future work should focus on multicenter prospective cohorts with standardized TCD and ASL protocols, repeated blood pressure phenotyping, and longitudinal cognitive and imaging outcomes. Interventional studies are also needed to establish whether treating symptomatic hypotension, reducing nocturnal hypotensive burden, or stratifying patients by autoregulatory failure can prevent progression from functional hypoperfusion to irreversible cerebrovascular injury.

CONCLUSION

Arterial hypotension should not be considered just a benign constitutional characteristic or a cause of nonspecific dizziness. This journal-style clinical–experimental study reported that hypotensive subjects have a consistent orthostatic set of symptoms, reduced cerebral autoregulation, perfusion-sensitive MRI abnormalities, and covert cerebrovascular injury, whereas the animal arm also provided biologic support for hypotension-induced neurovascular damage. The present study therefore suggests using a multimodal diagnostic approach that combines orthostatic blood pressure testing, ambulatory monitoring, transcranial Doppler, as well as perfusion-oriented MRI. Use of these tools together may identify a clinically significant subset of patients in whom chronic low-pressure exposure contributes to preventable cerebrovascular morbidity.

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