

**INTEGRATED NUMERICAL MODELING AND FUNCTIONAL
ASSESSMENT OF CEREBRAL AUTOREGULATION IN PATIENTS WITH
CONCOMITANT PERIPHERAL AND CAROTID ARTERY DISEASE**

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Introduction

Multisystemic atherosclerosis simultaneously affecting the carotid and lower extremity peripheral arterial beds represents an aggressive phenotype of macrovascular disease. While macroscopic arterial narrowing is routinely evaluated using anatomical imaging, systemic endothelial dysfunction often leads to microvascular failure long before structural occlusion completes. Cerebral autoregulation (CA) and cerebrovascular reactivity (CVR) are vital physiological mechanisms maintaining steady cerebral blood flow despite systemic blood pressure fluctuations. In patients with multi-focal peripheral atherosclerosis, the continuous assessment of microvascular reserve dynamics remains a challenging yet critical task for preventing secondary ischemic events.

Materials and methods

A prospective clinical validation study was conducted on a cohort of 128 patients presenting with internal carotid artery stenosis (55%–75%) and concomitant lower-extremity peripheral artery disease (Fontaine stage II-III, verified by Ankle-Brachial Index (ABI) < 0.6). Continuous trans-cranial Doppler (TCD) ultrasonography of the middle cerebral arteries was paired with beat-to-beat non-invasive arterial blood pressure monitoring (Finapres system). Cerebral autoregulation was quantified using the transfer function analysis (TFA) parameters: phase shift, gain, and coherence in the low-frequency range (0.05–0.15 Hz). Functional vasomotor challenges included the breath-holding test for the Breath-Holding Index (BHI). A distributed lumped-parameter hydraulic circuit model (Windkessel-based numerical simulation) was constructed to calculate regional cerebral perfusion pressures under variable peripheral vascular resistance stresses.

Results and discussions

The clinical cohort demonstrated a profound structural-functional mismatch, where patients with severe PAD (lowest ABI scores) exhibited a critically low phase

shift (± 0.08 rad) and compromised BHI compared to control subsets, reflecting a degraded cerebral autoregulatory capacity. The predictive numerical model achieved exceptional predictive performance ($R^2 = 0.88$, $p < 0.001$). Multi-parametric sensitivity analysis identified the systemic ankle-brachial index, local carotid plaque instability scores, and microvascular phase-lag as the highest-weighted independent determinants of cerebral circulatory failure. The numerical framework successfully mapped localized territorial hypoperfusion risk with an overall accuracy of 88.5%.

Conclusion

Coupling non-invasive dynamic autoregulation metrics with lumped-parameter mathematical simulations provides a highly precise diagnostic platform. This integrated approach accurately identifies subclinical cerebrovascular failure in multi-focal peripheral vascular phenotypes, offering an objective tool for optimizing timing and stratification for multi-stage surgical or endovascular interventions.

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