

The Pulse That Cannot Reach the Tissue

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

Large-vessel pulsatility and microvascular exchange operate on different physical scales. The proposed question is whether altered arterial stiffness changes the temporal pattern of pressure and flow reaching fragile capillary networks, increasing local injury even before conventional organ-specific endpoints occur.

The biological route

Elastic arteries normally cushion the heart's ejection; stiffening can alter pulse-wave reflection and distal pulsatile energy. Small vessels have limited tolerance for excessive pressure fluctuation, particularly in the brain and kidney. Endothelial injury and impaired autoregulation can amplify local effects. The precise impact depends on anatomy, pulse shape and vascular adaptation, not solely systolic blood pressure.

An illustrative scenario

A patient with normal average blood pressure can nevertheless show marked variability in pulsatile flow, whereas another with elevated mean pressure has a different hemodynamic profile. Neither simple comparison establishes microvascular disease. What matters is whether pulse-wave characteristics reach a particular organ and independently change tissue injury or function. The theory should be judged against well-validated cardiovascular risk measures.

Alternative explanations and limits

Age, hypertension, diabetes and atherosclerosis correlate with both vascular stiffness and organ disease, so causation is difficult to isolate. Arterial stiffness may be a marker of accumulated injury, a driver of further injury, or both. Not every vascular bed responds identically.

What would change the conclusion?

The hypothesis predicts that pulsatility measures add prospective information beyond mean blood pressure and established risk factors, and that interventions reducing harmful pulsatility improve microvascular endpoints. If the association vanishes after adjustment, the independent-driver claim weakens.

The distinctive prediction

Distal pulsatile energy should predict regional damage independently of mean pressure and age. A failure to demonstrate an organ-specific relationship would make arterial stiffness a marker rather than an established driver in that case.

Clinical interpretation

The proposal complements conventional hypertension and vascular care. It is not evidence for unvalidated devices, supplements or changing proven blood-pressure treatment targets.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

Angleys H et al. The effects of capillary transit time heterogeneity on brain oxygenation. J Cereb Blood Flow Metab. 2015.
<https://pmc.ncbi.nlm.nih.gov/articles/PMC4420854/>

McEwen BS. Stress, adaptation, and disease: Allostasis and allostatic load. Ann N Y Acad Sci. 1998.
<https://pubmed.ncbi.nlm.nih.gov/9629234/>

Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.