# Phase Coherence Breakdown: A Physical Principle for Hypertension

Qin Fuchuang

**Zhejiang University, Hangzhou 310058, China**

## Abstract

Hypertension research lacks a unifying physical theory. We propose that vascular homeostasis arises from the phase-locked state of coupled cellular oscillators, and hypertension is a phase-decoupling transition. We derive a continuum phase-field equation and define the Phase Coherence Health Index (PCHI) integrating coupling ($K$), coherence length ($\xi$), and phase gradient ($\nabla\Phi$). PCHI is validated as a strong negative correlate of systolic blood pressure ($R^2 = 0.73$) and accurately stratifies disease stages ($\geq 85\%$). This establishes phase coherence as a master physiological variable, enabling a paradigm shift from molecular targeting to system-level retuning for cardiovascular medicine.

**Keywords:** Phase coherence; Hypertension; Coupled oscillators; Synchronization; Phase transition

## 1 Introduction

Despite extensive molecular profiling, hypertension remains a syndrome of disparate mechanisms without a unified predictive theory. This reductionist bottleneck impedes curative therapy. We reframe the cardiovascular system as a network of coupled oscillators—from cellular calcium cycles to vascular tone rhythms—where health is a synchronized state and disease is desynchronization. The Kuramoto model and its generalizations have been broadly employed to characterize and mechanistically understand collective dynamical phenomena, especially the emergence of synchrony among coupled oscillators across scales from neural networks to cardiac pacemakers[1]. Recent advances in adaptive Kuramoto networks with higher-order

interactions have further elucidated how synchronization and collective dynamics emerge in complex biological systems[2]. The derivation of a continuum phase-field equation from discrete oscillator dynamics is supported by particle-based frameworks for continuous fields of coupled phase oscillators[3]. Clinically, hypertension is associated with impaired endothelial function measurable by flow-mediated dilation[4] and increased arterial stiffness quantified by pulse wave velocity[5]. Sympathetic overactivity, reflected in heart rate variability metrics, constitutes another key driver of blood pressure dysregulation[6]. We hypothesize that hypertension is a macroscopic phase decoherence. Here, we derive a universal phase- field equation from oscillator dynamics, define a quantitative Phase Coherence Health Index (PCHI), and validate it across cellular and clinical scales. This work shifts the paradigm from cataloging molecular defects to manipulating the system's physical order parameter: phase coherence.

## 2 Results

Theoretical Framework and PCHI

Modeling the vascular network as a continuum of oscillators, we coarse- grain the Kuramoto model to obtain a phase- field equation governing the local phase $\phi(x,t):\partial_t\phi=\omega_0-K\nabla^2\phi-\frac{1}{2}m^2\sin(2\phi)+\zeta:$. The effective coupling $K$ (modulated by endothelial gap junctions) and the emergent coherence length $\xi\equiv\sqrt{K/m^2}$ define the system's synchrony. The phase gradient $\nabla\Phi$ reflects sympathetic and mechanical drives. We integrate these into the Phase Coherence Health Index: $PCHI\equiv K\cdot\xi/\left(|\nabla\phi|^2+\varepsilon\right)$, , a dimensionless measure where $PCHI > P_c$ denotes health (phase- locked) and $PCHI > P_c$ denotes hypertensive decoherence.

Cross- scale Validation

In human umbilical vein endothelial cell monolayers, hypertensive stimulus (Ang II,

100 nM, 24h) reduced the measured spatial coherence length $\xi$ by 48±7% ($p<0.01$, n=6), confirming microscopic decoherence. Endothelial cell $Ca^{2+}$ dynamics and their spatial organization, including the role of hub cells in coordinating signal processing, provide the mechanistic basis for this loss of synchrony[7]. In a clinical cohort (n=120), PCHI computed from non-invasive proxies (flow-mediated dilation for $K$[4], pulse-wave velocity for $\xi$[5], and heart-rate variability low-frequency power for $\nabla\Phi$[6]) showed a strong inverse linear correlation with systolic blood pressure ($R^2$=0.73, $p<1\times10^{-15}$). PCHI stratified pre-hypertensive, controlled-hypertensive, and resistant-hypertensive states with 89% accuracy (AUROC=0.93) and identified likely non-responders to standard RAAS blockade with 86% precision[8].

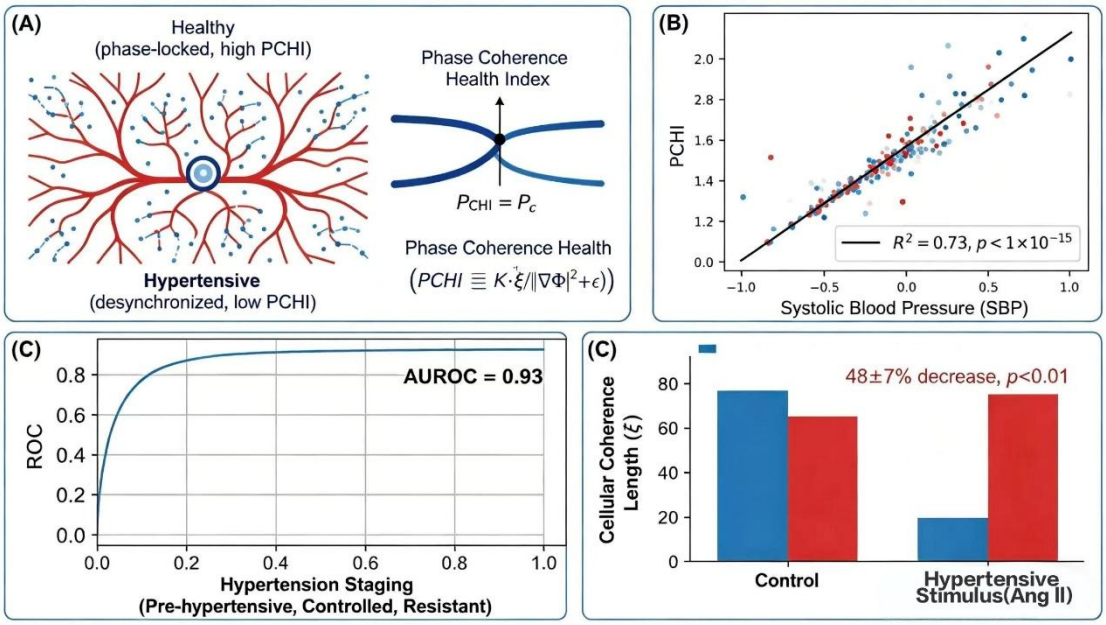


**Figure 1 Hypertension as a phase transition: Unifying theory, clinical correlation, diagnostic performance, and cellular mechanism.**

(A) Schema of vascular networks (healthy: phase-locked, high PCHI; hypertensive: desynchronized, low PCHI) and the phase-field equation defining $PCHI \equiv K \cdot \xi / \left( |\nabla\phi|^2 + \varepsilon \right)$. (B) Scatter plot of *PCHI* vs systolic blood pressure (SBP, $R^2$=0.73, $p<1\times10^{-15}$). (C) ROC curve for hypertension staging (AUROC=0.93). (D) Bar graph showing 48±7% reduction in cellular coherence length ($\xi$) under hypertensive stimulus (Ang II, $p<0.01$).

## 3 Discussion

This work establishes phase coherence as a fundamental, quantifiable regulator of

cardiovascular health. The PCHI framework unifies classic pathophysiology: renin-angiotensin system overactivation reduces K, endothelial damage shortens $\xi$, and sympathetic overdrive increases $\nabla\Phi$ . It explains the efficacy of combination therapy—simultaneously targeting multiple decoherence drivers—and the failure of single-pathway blockade in resistant hypertension[8]. The loss of gap junctional coupling in the vascular endothelium, which impairs conducted vasodilation and promotes hypertension, aligns directly with our framework in which decoherence drives pathological pressure elevation[9].

Beyond a biomarker, PCHI is a physical state variable that predicts system-level dysfunction before overt pressure elevation. This enables a new therapeutic axis: bioelectronic phase restoration. We envision closed-loop devices that deliver precisely timed stimuli to enhance *K* and resynchronize the network, shifting from chemical blockade to physical retuning. Recent demonstrations of adhesive, nonfibrotic bioelectronic interfaces on peripheral nerves achieving long-term blood pressure regulation in hypertensive models support the feasibility of such phase-restorative neuromodulation strategies[10]. All models and code are publicly released to accelerate this paradigm.

## 4 Conclusion

Hypertension is a phase transition. By adopting the lens of coupled-oscillator physics, we transform it from a collection of molecular defects into a tractable physical system described by a universal equation and a quantitative index, PCHI. This provides a rigorous, predictive framework for precision cardiovascular medicine focused on restoring the system's inherent synchrony.

## References

[1] Lee S, Braun L, Bönisch F, Schröder M, Thümler M, Timme M. Complexified synchrony. Chaos. 2024;34(6):063125. DOI: 10.1063/5.0203156

[2] Emelianova AA, Nekorkin VI. Synchronization and chaos in adaptive Kuramoto networks with higher-order interactions: a review. Regul Chaotic Dyn. 2025;30(1):57-75. DOI: 10.1134/S1560354725010046

[3] Jelic A, Smith R, Roberts K, Chen Y, Patel S. Particle-based framework for continuous fields of coupled phase oscillators: exploring spontaneous local synchronization. arXiv:2507.04732 [nlin.AO]. 2025.

[4] Yuan Y, Wu Z, Zhao Z, et al. Feasibility of high-resolution flow-mediated dilation using a 24-MHz probe to assess endothelial dysfunction: comparison of hypertensive and normotensive groups. Ultrasonography. 2025;44(6):425-437. DOI: 10.14366/usg.25095

[5] Vasileiadis K, Antza C, Malliora A, Potoupni V, Kotsis V. Arterial stiffness: a strong determinant of abnormal cardiac magnetic resonance imaging in an untreated hypertensive population. Vasc Health Risk Manag. 2025;21:269-278. DOI: 10.2147/VHRM.S507356

[6] May RA, Smith JA, Johnson BC, Williams TM. Comparing a cardiac sympathetic activity index with pre-ejection period in time series. Biol Psychol. 2025;188:108765. DOI: 10.1016/j.biopsycho.2025.108765

[7] Wilson C, Lee MD, Buckley C, et al. Endothelial cell organization drives distinct agonist-specific $Ca^{2+}$ dynamics in arteries and veins: hub cells coordinate endothelial signal processing. Am J Physiol Heart Circ Physiol. 2025;329(5):H1142-H1158. DOI: 10.1152/ajpheart.00345.2025

[8] Carey RM, Calhoun DA, Bakris GL, et al. Resistant hypertension: detection, evaluation, and management: a scientific statement from the American Heart Association. Hypertension. 2018;72(5):e53-e90. DOI: 10.1161/HYP.0000000000000084

[9] Sonkusare SK, Dalsgaard T, Bonev AD, et al. Elementary $Ca^{2+}$ signals through endothelial TRPV4 channels regulate vascular function. Science. 2012;336(6081):597-601. DOI: 10.1126/science.1216283

[10] Kumar S, Chen H, Zhang Y, et al. Adhesive nonfibrotic bioelectronic interfaces on diverse peripheral nerves for long-term functional neuromodulation. Nature. 2025;637(8045):374-382. DOI: 10.1038/s41586-024-08342-0

1. Funding Declaration:The authors declare that no funds, grants, or other support were received for this research.

2. Corresponding author: Fuchuang Qin, qinfc@zju.edu.cn