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Delayed Cybernetic Feedback and Hopf Instability in Insular–Autonomic Interoception: A Science 4.0 Hypothesis of Paroxysmal Panic Transitions

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Abstract

Unexpected panic attacks can include abrupt episodes of fear, cardiorespiratory arousal, and altered perception of internal bodily signals without an identifiable external trigger. Interoceptive inference frameworks implicate distributed cortical and brainstem networks, but do not uniquely specify a dynamical mechanism for the onset of such episodes. This theoretical perspective proposes a deliberately reduced, stochastic delay-differential model of a viscerosensory–insular–autonomic control loop. Its dimensionless feedback gain g and effective delay τd govern the local stability of a homeostatic equilibrium. Linearization yields an explicit Hopf-type crossing threshold: for g > 1, the equilibrium first becomes unstable when τd/τc exceeds arccos(−1/g)/√(g²−1). Illustrative deterministic simulations show damped and persistent oscillatory behavior on opposite sides of this threshold for one parameter set. Whether the nonlinear bifurcation is supercritical, or whether such dynamics explain clinical panic, is not established by the linear calculation. We outline falsifiable physiological observations and a prospective EEG–ECG–capnography study. This work introduces a phenomenological hypothesis; no clinical dataset was analyzed and no diagnostic or treatment claim is made.

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Introduction and Scope

Panic disorder includes recurrent unexpected panic attacks featuring marked fear or discomfort together with autonomic and somatic symptoms. Multiple explanatory levels are relevant, from cognitive interpretation and threat learning to brainstem respiratory regulation and cortical representation of bodily state [1,2]. Contemporary interoception research investigates how visceral afferents are integrated with predictions within insular and cingulate networks [3-5].

These concepts motivate dynamical models but do not, by themselves, establish a unique causal mechanism for any individual attack.

Delayed negative feedback is a well-studied source of oscillatory instability in physical and physiological control systems [6,7]. A corrective response that arrives too late may reduce, rather than enhance, effective stability. The present question is whether this principle offers a compact, falsifiable mathematical analogy for some paroxysmal aspects of panic, rather than a complete model of panic phenomenology. Previous author-described Science 4.0 publications address stress and sleep-related biological control; the present proposal is directed at a distinct, acute interoceptive feedback problem [8-10].

The model does not reconstruct measured anatomical connectivity, derive microscopic synaptic conductances, or claim that panic events are generally periodic. Its primary mathematical result concerns the stability boundary of an explicitly stated surrogate system.

Minimal Delayed-Feedback Model

Let z(t) denote a dimensionless collective state summarizing interoceptive prediction-error deviation from a reference level z = 0. Positive and negative deviations represent opposite directions of a coarse-grained error variable, not directly measured firing rates or catecholamine concentrations. The model contains an intrinsic relaxation time $\tau c > 0$, a feedback amplitude $\gamma fb > 0$, sensitivity $\beta > 0$, an effective loop delay $\tau d \geq 0$, and a phenomenological stochastic intensity $Dint \geq 0$.

$$\tau_c\, dz(t) = [-z(t) - \gamma_{fb} \tanh(\beta z(t - \tau_d))]\, dt + \sqrt{2\tau_c D_{int}}\, dW_t \quad (1)$$

Here Wt denotes standard Brownian motion. The hyperbolic tangent limits the deterministic feedback term as |z| becomes large; it does not strictly bound trajectories driven by additive Gaussian noise. The delay is a model-level effective parameter, not an experimentally established axonal conduction delay or a direct estimate from scalp EEG.

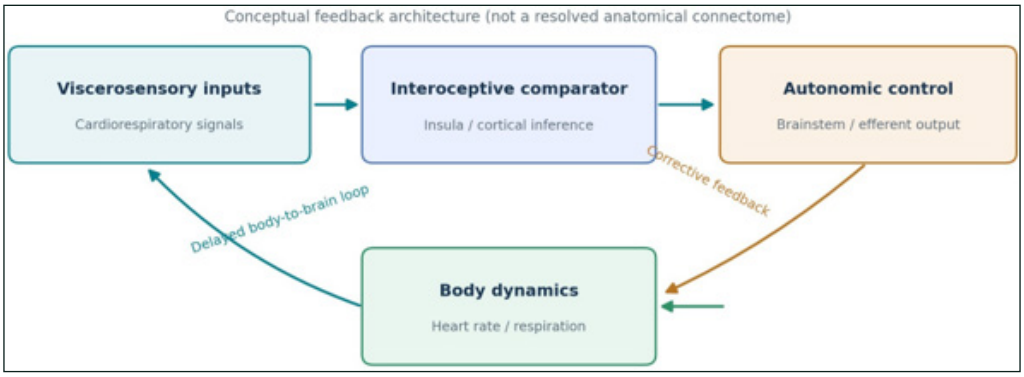


Figure 1: Conceptual Delayed-Feedback Architecture. Functional Blocks do not Assert a Literal Single-Path Connectome or Measured Synaptic Time Delays.

Linear Stability and the Hopf Crossing

Setting Dint = 0 and linearizing tanh(βz) around z = 0 yields the effective loop gain g = γfb β and the linear delay equation:

$$\tau_c \frac{dz(t)}{dt} = -z(t) - g\, z(t - \tau_d), \qquad g = \gamma_{fb}\beta \tag{2}$$

For exponential perturbations z(t) ∝ exp(λt), the characteristic roots satisfy:

$$\tau_c \lambda + 1 + g\, e^{-\lambda \tau_d} = 0 \tag{3}$$

At zero delay, the only root is λ = −(1+g)/τc < 0. At a possible oscillatory crossing, substitute λ = iω0 with ω0 > 0 and separate real and imaginary parts:

$$\cos(\omega_0 \tau_d) = -\frac{1}{g}, \qquad \sin(\omega_0 \tau_d) = \frac{\tau_c \omega_0}{g} \tag{4}$$

The resulting positive crossing frequency and necessary gain condition are:

$$\omega_0 = \frac{\sqrt{g^2 - 1}}{\tau_c}, \qquad g > 1 \tag{5}$$

For g > 1, the first (smallest positive) critical delay is:

$$\tau_{d,\,crit}(g) = \frac{\tau_c}{\sqrt{g^2 - 1}} \arccos\left(-\frac{1}{g}\right) \tag{6}$$

Consequently, the linear equilibrium is asymptotically stable for 0 < g ≤ 1 at every finite delay and, for g > 1, remains stable below the first critical delay. Above this first threshold it loses linear stability. The crossing is compatible with a Hopf bifurcation; however, linearization does not determine whether the nonlinear branch is supercritical or subcritical. A normal-form calculation, center-manifold analysis, or rigorously validated continuation would be required to establish that classification. The term "unstable equilibrium" should not be read as a clinical diagnosis or a guarantee of an attack.

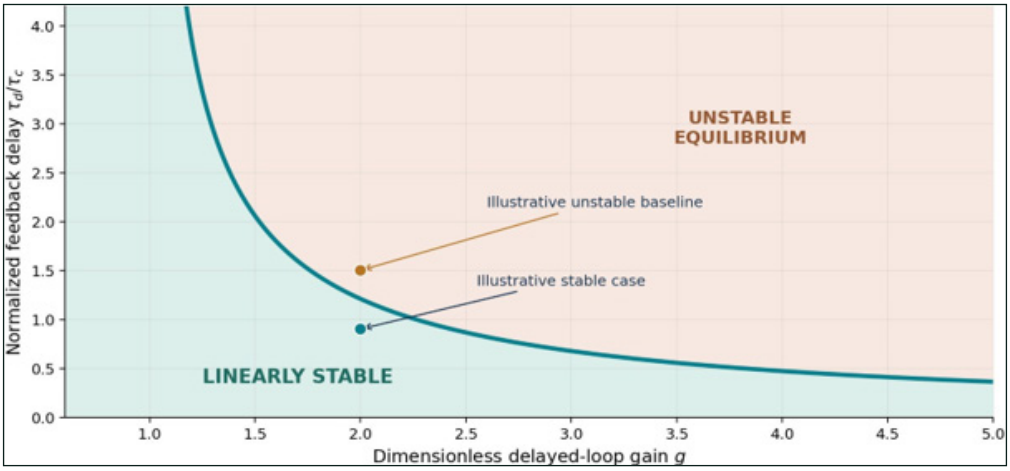


Figure 2: Exact First Linear-Stability Boundary of the Stated Reduced Model. Only the Equilibrium's Stability is Classified. The Shaded Unstable Region does not Establish Clinical Panic or the Existence of a Stable Nonlinear Limit Cycle.

Illustrative Nonlinear Dynamics

To illustrate the difference between local damping and persistent oscillatory behavior, a deterministic version of equation (1) was integrated numerically with $\tau c = 1$, $\beta = 1$, $\gamma fb = 2$ (thus $g = 2$), $Dint = 0$, and a constant initial history $z(t) = 0.15$ for $t \leq 0$. The exact linear threshold is $\tau d,crit/\tau c = \arccos(-1/2)/\sqrt{3} \approx 1.209$. An explicit small-step integration (step = 0.002 in normalized units) gives decay at $\tau d = 0.9$ and sustained oscillatory behavior over the simulated observation window at $\tau d = 1.5$.

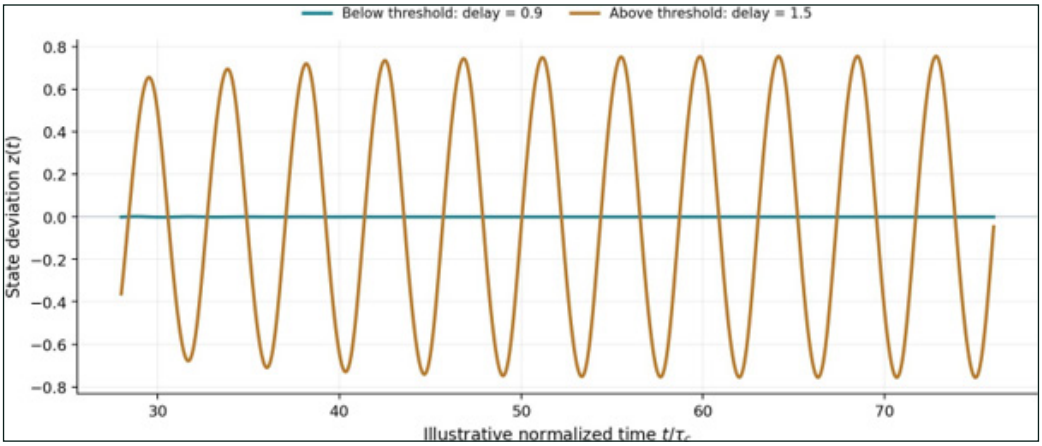


Figure 3: Original Illustrative Solutions of the Deterministic Reduced Delay Equation, not Recordings from Patients. These Numerical Traces are not a Proof of Global Stability or of Hopf Criticality.

Because a scalar delayed feedback oscillator can display dynamics unlike the short, irregular, context-sensitive time course of a panic attack, any biological mapping requires a separate measurement model and systematic comparison against rival explanations.

Falsifiable Predictions and Prospective Evaluation

Pre-episode Resonance

If the reduced stability mechanism has a useful physiological counterpart, some patients may show a change in damping or narrowband fluctuation structure in pre-episode cardiac and interoceptive time series. A potential measurement includes heartbeat-evoked potential (HEP) dynamics recorded with synchronized EEG and ECG. This is a hypothesis, not an established pre-ictal biomarker. Because cardiac field artifacts, attention, respiratory state, and other factors contaminate HEP estimates, predefined controls and reliability analyses are mandatory [11].

Cross-Modal Phase Relationships

The model predicts a frequency-dependent phase shift as loop delay changes. In a suitably identified observation model, cross-spectral associations between R–R intervals, respiration, and cortical proxies might vary with proximity to the inferred stability boundary. However, the phase between measured HEP amplitudes and R–R intervals must not be equated directly with $\omega\tau d$: physiological filtering, sampling and source mixing all intervene.

Gain Sensitivity and Model Discrimination

For the particular scalar linearization, reducing effective gain to $g \leq 1$ removes this delay-induced linear instability for finite delays. This does not imply that an intervention lowering a putative clinical precision-weighting parameter will abruptly eliminate panic attacks: the parameter is not directly measured and clinical disorders involve multiple mechanisms. A prospective study should compare the delayed-feedback model with autoregressive, state-space, and non-delay nonlinear alternatives, using held-out data and prespecified goodness-of-fit criteria. A future ethics-approved protocol could combine resting and standardized interoceptive tasks with high-density EEG, ECG, respiratory belt/capnography, symptom timing, and appropriately trained clinical oversight. Any CO2 challenge would require clinical ethics approval and medically supervised safety

procedures. Report uncertainty and identifiability for each fitted parameter; control for medications, respiratory physiology, activity, and cardiac artifacts. Data collection is proposed, not claimed.

Limitations, Originality and Clinical Boundaries

The proposed state z(t) merges multiple brain and body processes into one unobserved variable. The insula–autonomic network is distributed and is not faithfully captured by a single feedback loop. Effective gain and delay can be confounded in available observations; neither HEP nor HRV isolates a microscopic neural delay. Oscillation onset is not synonymous with a panic episode, and the clinical time course can be nonperiodic. The stability formula is mathematically precise only for the stated linearized equation.

Relative to the author's titles shown in the accompanying publication list, this draft addresses acute interoceptive instability rather than chronic HPA-axis adaptation or sleep-state motor gating. However, uniqueness of topic within a personal portfolio does not establish worldwide novelty, and the broader use of delayed physiological feedback and Hopf theory is established in prior literature [6,7,12]. A comprehensive literature review and journal-specific editorial check remain necessary before submission.

Conclusion

A minimal stochastic delayed-feedback formulation offers a falsifiable candidate framework for examining the stability of interoceptive–autonomic regulation. Its local equilibrium has an analytical delay-dependent instability threshold for $g > 1$, and deterministic numerical examples illustrate qualitatively different dynamics on either side. Translating this mathematical result into a mechanistic account of unexpected panic attacks will require independently recorded physiology, identifiability analysis, appropriate controls, and external validation. This contribution is a theoretical perspective, not evidence of clinical efficacy or a replacement for established psychiatric assessment.

Declarations

Data availability. No original clinical dataset was collected or analyzed; the figures are original analytical or illustrative numerical constructions.

Ethics. No human-participant data were used in preparing this theoretical contribution.

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