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THE EMERGENT FLOW ARCHITECTURE (EFA): A MULTISCALE INFORMATIONAL FRAMEWORK FOR CONSCIOUS INTEGRATION

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ABSTRACT

Understanding consciousness requires an integrative framework capable of linking physical constraints, neurobiological implementation, and subjective experience across multiple temporal scales. We present the Emergent Flow Architecture (EFA), a falsifiable theoretical framework that unifies these perspectives through hierarchical temporal integration. EFA formalizes conscious integration as a multiplicative architecture, $\mathcal{C} = \gamma \cdot n \cdot \alpha \cdot \Delta T$, where basal coherence (γ; autonomic–bioelectric stability), active neural populations (n), functional integration efficiency (α), and compatible temporal windows jointly determine the quality of conscious experience. Mapped onto the RAS–SRAA–DMN neurobiological axis, EFA distinguishes empirically testable mechanisms from theoretical boundary conditions, positioning the brain as a probabilistic sampler rather than a quantum computer. The framework specifies explicit falsification criteria and generates testable predictions assessable through TMS–EEG perturbation paradigms, anesthesia titration, and disorders-of-consciousness studies. From this architectural perspective, distinct clinical conditions are interpreted as failures at specific integration tiers, enabling hypothesis-driven and precision-oriented intervention strategies. Comparative analysis demonstrates that EFA synthesizes key insights from Integrated Information Theory, Global Neuronal Workspace Theory, and the Free Energy Principle while adding explicit temporal constraints and autonomic foundations absent from existing models. Broader implications extend to artificial systems and agency, positioning EFA as a unifying architectural framework for the science of consciousness.

KEYWORDS: Consciousness; Information Integration; Temporal Windows; Basal Coherence; Neurobiological Networks; Entropy; Emergent Flow Architecture

1. INTRODUCTION: THE FRAGMENTATION OF CONSCIOUSNESS SCIENCE

Understanding consciousness remains one of the most profound challenges confronting contemporary science, spanning neuroscience, physics, and cognitive science, yet current theoretical approaches remain fundamentally fragmented. The classical formulation of the "hard problem" of consciousness emphasizes an explanatory gap between physical processes and subjective experience, often channeling scientific discourse toward debates over irreducibility, novel physics, or explanatory limits. While this framing has usefully clarified the depth of the problem, it has also impeded empirical progress by prioritizing ontological mystery over operational specification.

Here we adopt a fundamentally different strategy. Rather than asking why physical systems generate experience, we ask: under what organizational conditions does informational integration become self-referential and reportable? This reframing shifts the inquiry from metaphysical explanation to architectural constraint, treating consciousness not as a substance or epiphenomenon, but as a dynamical process that emerges when specific integration criteria are satisfied across hierarchically nested temporal scales.

A defining feature of conscious experience is integrative binding. Distributed neural processes—sensory, affective, semantic, and mnemonic—must be unified into coherent perceptual moments and extended into continuous experiential streams. Crucially, this binding is not merely spatial but fundamentally temporal: millisecond-scale perceptual integration must be coordinated with seconds-long narrative continuity and with slower autonomic stabilization processes. Consciousness is therefore explicitly not scale-invariant. Disruption at any level of this temporal hierarchy degrades or abolishes conscious experience, as consistently observed in anesthesia, coma, and brainstem lesions.

1.1. Limitations of Current Frameworks

Three major theoretical frameworks currently dominate consciousness research, each capturing essential aspects of the phenomenon while leaving critical explanatory gaps.

Integrated Information Theory (IIT) formalizes consciousness as irreducible causal structure, quantified by the integrated information measure Φ. While mathematically rigorous and appropriately integration-focused, IIT remains largely timescale-agnostic, underspecifies neurobiological implementation pathways, and lacks explicit autonomic or brainstem foundations despite their empirically demonstrated necessity for conscious states.

Global Neuronal Workspace Theory (GNWT) explains conscious access through ignition dynamics and global broadcasting within frontoparietal networks. Although well-grounded neurobiologically and supported by substantial empirical evidence, GNWT primarily addresses access consciousness rather than phenomenal experience, focuses predominantly on a single ignition window (~300 ms), and provides no quantitative master equation linking integration processes across multiple timescales.

The Free Energy Principle (FEP) offers a potentially unifying account of perception, action, and learning through variational free energy minimization. However, its extreme generality applies equally to conscious and unconscious systems, leaving the specific conditions that distinguish conscious integration underdetermined and empirically difficult to falsify in a consciousness-specific manner.

A fundamental limitation shared across these frameworks is the absence of an explicit multi-scale temporal architecture. None successfully integrates autonomic stability, perceptual binding, and narrative selfhood within a single operational model governed by explicitly defined temporal windows and testable constraints.

1.2. The Emergent Flow Architecture

We introduce the Emergent Flow Architecture (EFA), a falsifiable theoretical framework in which conscious experience arises from hierarchical informational integration across nested temporal scales, coordinated by basal coherence and implemented through anatomically specified neurobiological structures.

EFA operationalizes conscious integration using the dimensionless index:

$$\mathcal{C} = \gamma \times n \times \alpha_{nk} \times \Delta T_u \times \Sigma(\Delta T_q) \times \int(\Delta T_S)$$

where γ denotes basal autonomic–bioelectric coherence, n the active neural population, α_nk functional integration efficiency, ΔT_u the perceptual binding window (~25–40 ms), ∫(ΔT_S) narrative-scale integration (0.3–3 s), and Σ(ΔT_q) a formal boundary term capturing sub-neuronal constraints without requiring quantum computation in neural tissue.

EFA is implemented across three neurobiological tiers: (i) reticular–autonomic systems generating γ, (ii) thalamocortical and ascending reticular networks supporting n and α_nk, and (iii) default mode network dynamics integrating information across ΔT_S. Integration is multiplicative rather than

additive: failure of any parameter collapses conscious integration, explaining why preserved cortical processing without autonomic support is insufficient for consciousness.

The hierarchical neurobiological implementation of the Emergent Flow Architecture, including its three-tier organization, temporal constraints, recursive bottom-up and top-down dynamics, and illustrative architectural failure modes, is summarized in Figure 1.

Figure 1. The Emergent Flow Architecture: Three-Tier Neurobiological Implementation

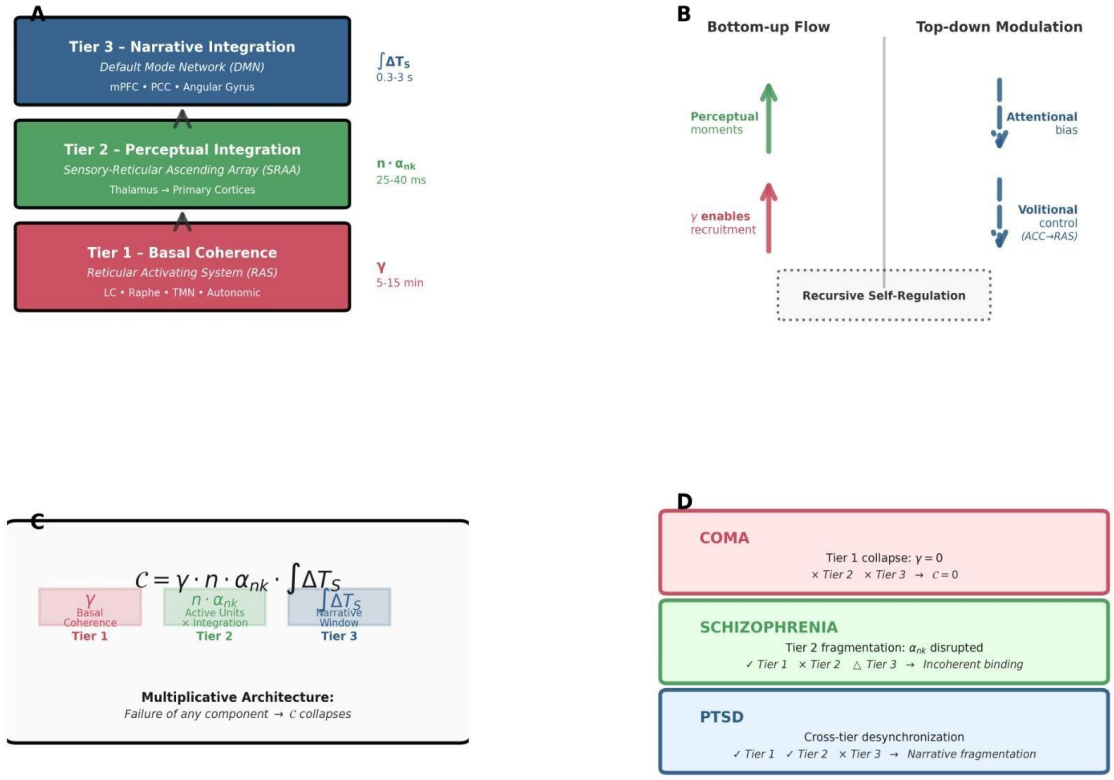

Figure 1: PLACEHOLDER. The Emergent Flow Architecture (EFA): Three-tier neurobiological implementation.

(A) Hierarchical organization of conscious integration across three interacting tiers. Tier 1 (Basal Coherence, γ) corresponds to brainstem and autonomic regulatory systems within the reticular activating system (RAS), providing slow, stabilizing bioelectric and autonomic coherence. Tier 2 (Perceptual Integration, n·α) corresponds to the sensory–reticular ascending array (SRAA), including thalamic and primary cortical systems operating over fast perceptual time windows (≈25–40 ms). Tier 3 (Narrative Integration, ∫ΔT_S) corresponds to the default mode network (DMN), supporting temporally extended self-referential and narrative processes (≈0.3–3 s).

(B) Schematic representation of recursive bottom-up and top-down dynamics. Bottom-up flow reflects the recruitment of perceptual moments enabled by basal coherence, while top-down modulation reflects attentional and volitional biasing originating from higher-order networks (e.g., anterior cingulate cortex) toward brainstem and autonomic regulation. Together, these processes form a closed-loop architecture of recursive self-regulation.

(C) Multiplicative structure of conscious integration. Consciousness (***C***) emerges only when basal coherence (γ), active neural populations (n), functional integration efficiency (α), and compatible temporal integration windows are simultaneously satisfied. Failure of any component leads to collapse of the integrated conscious state.

(D) Illustrative architectural failure modes (schematic). Different clinical and altered states are represented as disruptions at specific tiers or across tier interactions, including global collapse of basal coherence (coma), fragmentation of perceptual integration (schizophrenia), and cross-tier desynchronization affecting narrative continuity (PTSD). These examples are illustrative and not intended as diagnostic or etiological claims.

1.3. Testability and Scope

EFA yields explicit, empirically falsifiable predictions. Basal coherence (γ) should correlate strongly with perturbational complexity (PCI), and perturbation–recording paradigms should reveal thresholds of integrated responsiveness corresponding to high $\mathcal{C}$ values. These predictions are testable using TMS–EEG, anesthesia titration, and disorders-of-consciousness protocols.

Quantum scales are treated strictly as formal boundary conditions rather than operative neural mechanisms, and extended discussions of quantum-informational scaffolding, phenomenological trade-offs, agency, and computational implications are provided in the Supplementary Materials. The framework stands or falls on empirically accessible parameters.

By introducing explicit temporal constraints and autonomic foundations, the Emergent Flow Architecture provides an operational synthesis across existing approaches while offering a quantitative, testable architecture for understanding conscious integration.

2. THERMODYNAMIC FOUNDATIONS AND HIERARCHICAL TEMPORAL WINDOWS

2.1. Information as a Physical Quantity

Information theory formalizes uncertainty through Shannon entropy: $H(X) = -\sum_i p(x_i) \log_2 p(x_i)$ which is formally isomorphic to Boltzmann's thermodynamic entropy: $S = -k_B \sum_i p(x_i) \ln p(x_i)$ Neural information transfer is bounded by Shannon capacity $C = B \cdot \log_2(1 + SNR)$, where bandwidth and signal-to-noise constraints impose fundamental limits on conscious integration rates.

up to the scaling factor $k_B \ln 2$. This equivalence establishes information and thermodynamics as complementary descriptions of the same physical constraints governing state distributions [1,2].

The negative sign ensures that entropy increases with uncertainty, preserving consistency with the second law of thermodynamics.

A decisive implication follows from Landauer's principle: erasing information has an irreducible energetic cost,

$E_{min} = k_B T \ln 2$ per bit

experimentally validated in physical systems [3,4]. Information processing is therefore inseparable from entropy production and energy dissipation. These foundational results, combined with Wheeler's "It from Bit" hypothesis and the Bekenstein-Hawking holographic principle, establish information as fundamental to physics—suggesting that physical reality may be informational at its core [S69, S72].

2.2. Consciousness as Informational Work

The brain receives on the order of 10^8 bits/s of sensory input, while conscious access is limited to approximately 40–60 bits/s [5]. The remaining information must be continuously discarded, implying large-scale informational erasure. This represents an information bottleneck ratio of approximately 10^6:1, with the remaining ~10^8 bits/s either failing to be encoded or decaying through interference, lack of synaptic consolidation, and working memory displacement.

Although the Landauer minimum predicts a negligible theoretical cost, biological implementations are inherently thermodynamically inefficient. Synaptic inhibition, adaptive filtering, and network suppression incur substantial real metabolic expenditure. Conscious processing therefore represents informational work performed by a far-from-equilibrium biological system operating under significant thermodynamic constraints. Neural systems may exploit stochastic resonance—a phenomenon where optimal noise enhances signal detection—by maintaining γ (basal coherence) near levels that maximize information transfer efficiency. This predicts an inverted-U relationship between γ and $|\mathcal{C}|^2$: too low γ produces incoherent noise, too high γ may miss weak signals, while moderate γ optimizes conscious integration by balancing noise-enhanced detection with coherence maintenance. The brain's total energy consumption (~20 W) allocates approximately 50% to Na^+/K^+-ATPase pumps, 20% to synaptic transmission, and 10% to action potential propagation, with informational processing overhead estimated at 5-10% (~1-2 W)—reflecting costs from selective amplification machinery rather than Landauer-type erasure.

Within EFA, this selective process is structured by basal coherence (γ). Stable autonomic states enable efficient, non-random informational sampling, whereas autonomic fragmentation leads to noisy, inefficient integration. The Ascending Reticular Activating System (ARAS) acts as an attentional gatekeeper performing continuous measurement, selection, and erasure—analogous to Maxwell's demon, but constrained by basal coherence rather than operating randomly. For detailed Maxwell's demon analysis and energetic modeling, see Supplementary Material S2. Importantly, this represents a heuristic analogy highlighting information bottleneck dynamics, not a claim that the brain literally performs Landauer erasure. Neural "information discarding" differs fundamentally from thermodynamic erasure: the brain performs selective amplification of attended signals while non-attended

information decays passively through synaptic depression, lateral inhibition, and lack of consolidation—rather than active erasure to a standard state.

The hierarchical organization of temporal integration across biological scales, and the nesting of fast dynamics within slower integrative windows, is schematically illustrated in Figure 2.

2.3. Temporal Windows as Physical Constraints

Figure 2. Hierarchical Temporal Integration Across Biological Scales

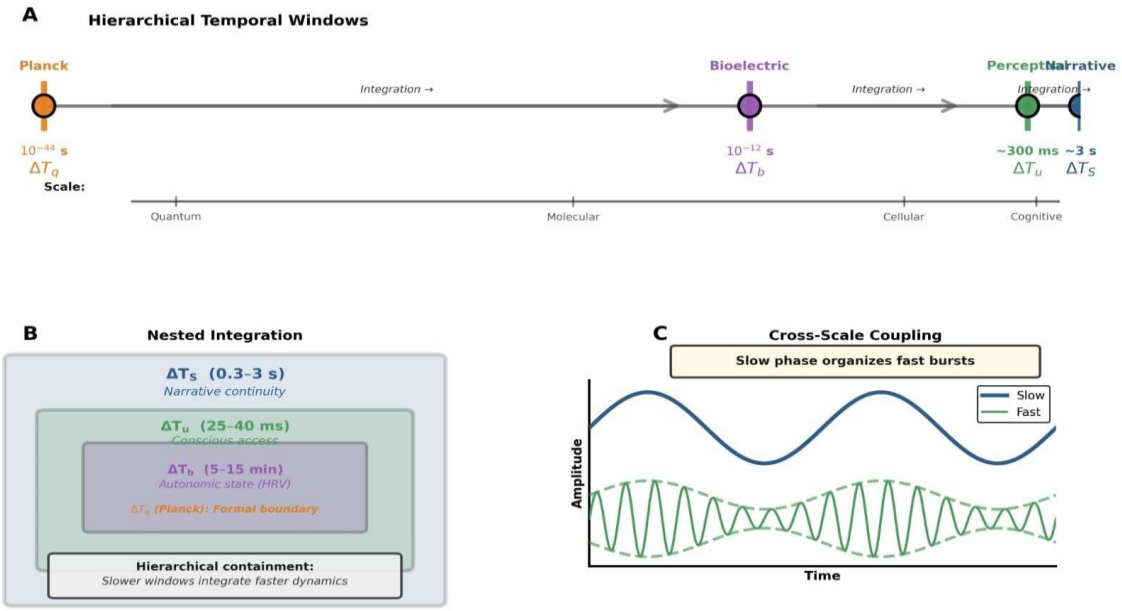

Figure 2: PLACEHOLDER. Hierarchical Temporal Integration Across Biological Scales

(A) Schematic representation of hierarchical temporal windows spanning physical, biological, and cognitive scales. Integration proceeds from fast, lower-bound temporal limits (ΔT_q; Planck-scale formal boundary) through bioelectric and autonomic dynamics (ΔT_b), perceptual sampling windows (ΔT_u), and extended narrative integration windows (ΔT_S). These windows are shown as progressively inclusive constraints rather than discrete processing stages.

(B) Nested temporal integration. Faster dynamics are hierarchically contained within slower integrative windows, such that perceptual access (ΔT_u) and autonomic regulation (ΔT_b) are embedded within longer narrative timescales (ΔT_S). This organization supports continuity of conscious experience across multiple temporal resolutions.

(C) Cross-scale coupling. Slow-phase dynamics modulate and organize fast-scale activity, illustrating a generic mechanism by which hierarchical temporal coordination can be achieved without invoking direct causal control or instantaneous integration.

Time acts as an active organizer of informational integration, not merely a passive backdrop. Information arriving within a given temporal window can be bound into unified representations; information separated beyond that window cannot be integrated, regardless of processing capacity.

Consciousness operates via nested windows spanning multiple temporal scales. EFA specifies three empirically grounded integration epochs, each corresponding to distinct biophysical substrates and functional roles (Table 1).

Table 1: Hierarchical Temporal Windows in EFA

Window	Duration	Substrate	Function	Measurement Method	Clinical Relevance
ΔT_b (Bioelectric-Cellular)	5-15 min	Bioelectric gradients, cellular memory networks, autonomic coupling	Substrate stability enabling γ emergence; slow consolidation processes	Near-term: voltage imaging, organoid recordings, ion channel mapping; Current: HRV (indirect proxy)	Chronic dysregulation in trauma, autonomic disorders; target for vagal interventions
ΔT_u (Unified Perceptual)	25-40 ms	Gamma oscillations (30-100 Hz), thalamocortical loops, local E/I	Perceptual binding into discrete conscious "frames"; feature integration	EEG/MEG gamma power & coherence, TMS-EEG perturbational dynamics, phase-locking value	Disrupted in schizophrenia (gamma deficits), anesthesia (loss of binding); target for 40 Hz stimulation

		circuits			
ΔT_S (Narrative Integration)	0.3-3 s	DMN slow oscillations (theta 4-8 Hz, infra-slow 0.01-0.1 Hz), hippocampal sequences	Integration of perceptual moments into specious present; autobiographical continuity	rs-fMRI DMN connectivity, EEG theta-gamma PAC, narrative coherence behavioral scoring	Fragmented in PTSD (unintegrated trauma), collapsed in depression (foreshortened future); target for psychotherapy

Table 1. Hierarchical temporal windows defining EFA's multi-scale integration architecture. Each window corresponds to distinct biophysical substrates and functional roles, with window coordination (rather than any single window) being necessary for unified conscious experience. Disruption at any level degrades consciousness quality, as captured by the multiplicative structure of the master equation. ΔT_b provides substrate stability (conditioning γ), ΔT_u enables perceptual binding (supporting n·α_nk), and ΔT_S supports narrative selfhood (∫ΔT_S). Abbreviations: DMN, Default Mode Network; E/I, excitation-inhibition; EEG, electroencephalography; fMRI, functional magnetic resonance imaging; HRV, heart rate variability; MEG, magnetoencephalography; PAC, phase-amplitude coupling; TMS, transcranial magnetic stimulation. The Modulation Index (MI = |⟨Afast(t)·e^(iφslow(t))⟩|) quantifies PAC strength, with typical values of MI ≈ 0.3-0.5 for conscious/attentive states versus MI ≈ 0.05-0.1 during anesthesia.

Without coordinated nesting of these windows, conscious experience fragments into isolated or unreportable events.

Importantly, these constraints rely on classical coherence, not quantum coherence. Quantum decoherence at biological temperatures occurs orders of magnitude faster than neural processing times [6]. EFA therefore treats quantum scales strictly as formal lower bounds rather than operative neural mechanisms (see Supplementary Material S3 for extended quantum-informational boundary discussion). Specifically, Tegmark (2000) calculated decoherence timescales of approximately 10^{-13} seconds in biological environments—10 orders of magnitude shorter than the ~100 ms timescale of conscious processing—rendering sustained macroscopic quantum coherence in neural tissue physically implausible under current understanding. The brain operates as a probabilistic sampler of informational events that have already undergone quantum-to-classical transitions, rather than maintaining coherent superpositions—analogous to how visual perception samples the statistics of photon detection rather than quantum superpositions of photon states.

2.4. The Bioelectric–Cellular Window (ΔT_b): Substrate Stability and Emergence of γ

EFA proposes that basal coherence γ reflects organism-level capacity for stable integration, extending beyond purely cortical processes. A plausible mechanistic contribution to this capacity is slow cellular-scale plasticity and bioelectric coordination operating on minutes-long timescales, providing a stable substrate upon which faster integrative processes can operate.

Non-neuronal learning-like dynamics. Recent evidence demonstrates that non-neural human cells express spacing-dependent learning effects—stronger responses when stimuli are spaced across minutes rather than massed [7]. This indicates that time-dependent plasticity mechanisms operate beyond neurons, supporting the plausibility of a slow substrate window conditioning higher-level integration.

EFA does not claim "memories" are stored outside the brain; rather, these findings establish that cellular-scale temporal integration processes (ΔT_b) provide a stable "platform" upon which fast perceptual binding (ΔT_u) and narrative integration (ΔT_S) can operate without fragmentation.

Bioelectric and autonomic coupling. Practically, EFA operationalizes γ primarily via autonomic–interoceptive coherence, using HRV-derived measures as first-line proxies. Michael Levin's work on bioelectric voltage gradients across cellular networks storing morphogenetic information [8] provides complementary evidence that ion channel distributions and gap junction coupling maintain coherent states for hours, supporting the ΔT_b timescale.

The vagal integration pathway provides a plausible mechanism: peripheral bioelectric states → vagal afferents → nucleus tractus solitarius → locus coeruleus, raphe nuclei → emergent γ. Empirical support comes from findings that HRV predicts cognitive flexibility and executive function [9], and that interoceptive accuracy correlates with emotional awareness and anterior insula activity [10]. This pathway is further supported by evidence that high HRV correlates with enhanced executive function through prefrontal-subcortical circuit stabilization,

with vagal tone acting as a modulatory mechanism for α_nk effectiveness.

Testable prediction. Interventions reliably increasing organismic coherence at slow timescales (e.g., vagal-targeted protocols, coherent breathing) should raise γ and improve downstream integration metrics (PCI, DMN stability), provided cortical connectivity remains intact.

Current empirical status: ΔT_b remains at Tier 2 accessibility (near-term testable, Section S3.6 in Supplementary Material). While HRV provides immediate operational proxy for γ, direct cellular-scale bioelectric measurements await emerging technologies (voltage imaging, organoid assays, Protocols 2-3 in Supplementary Material S5). The framework's falsifiability at Tier 1 depends on HRV-accessible γ proxies, not on ΔT_b validation. If cellular-scale processes prove unnecessary, EFA reduces to γ_autonomic without loss of predictive power. Tier 2 parameters (ΔT_b cellular memory timescales, bioelectric membrane coherence, gap junction dynamics) become testable within 5-10 years via organoid cultures, voltage-sensitive dye imaging, and cellular memory assays. Tier 3 parameters (ΔT_q quantum-informational events) require quantum biology breakthroughs and may remain inaccessible, though they serve as formal boundary conditions analogous to Planck-scale physics in cosmology.

2.5. Cortical-Brainstem Recursive Loops: Anatomical Basis for Downward Causation

Recent discoveries have identified direct neural pathways enabling top-down cortical control of autonomic brainstem functions, providing anatomical substrate for the recursive causality central to EFA.

Jhang et al. (2018, Cell Reports) demonstrated a circuit originating in anterior cingulate cortex (ACC) that directly innervates respiratory control centers in the brainstem [11]. Using optogenetic methods in mice, the authors showed that selective stimulation of ACC neurons projecting to the pons induced slow breathing (~1.3 Hz, within the coherent breathing range used therapeutically) and significantly reduced anxiety-like behaviors. Conversely, inhibiting this pathway prevented voluntary breathing modulation and blocked the anxiolytic effects of slow breathing.

2.5.1. Functional significance for EFA.

This pathway instantiates the recursive loop central to consciousness architecture:

1. Conscious intention (ACC, part of DMN self-regulatory circuits operating at ΔT_S timescales) initiates volitional breathing pattern modulation
2. Brainstem execution (respiratory centers in pons/medulla, part of RAS) implements the breath pattern change
3. Autonomic feedback alters heart rate variability (HRV, the primary operational proxy for basal coherence γ), modulating the stability of the autonomic platform
4. Changed conscious state results from modified γ, manifesting as reduced negative affect and altered integration capacity (modified $\boldsymbol{C}$)

The system modifies its own substrate. This is not passive information flow but active recursive self-regulation—the defining characteristic of emergent determination (Section 6). This recursive architecture positions consciousness as a trans-Turing system: rather than pre-computing all possible contents, the brain samples the space of informational possibilities probabilistically, with basal coherence (γ) and attentional priorities biasing sampling toward relevant regions. New conscious contents can arise that were not explicitly represented beforehand (insight, creativity), reflecting exploration of Kauffman's "adjacent possible"—the space of immediately reachable novel configurations whose full extent cannot be pre-stated [S89]. This constitutes emergent free will—not exemption from causality, but higher-order causal architecture where the system influences its own future sampling through recursive self-modification: conscious reflection → meta-cognitive choice → altered γ, α_nk, ΔT dynamics → changed future conscious contents.

2.5.2. Empirical predictions.

1. Lesion studies: Disrupting the ACC-brainstem pathway (via targeted lesions in animal models or stroke patients with ACC damage) should impair: - Volitional control of breathing rhythm

- Ability to increase HRV through intentional breathing exercises
- Therapeutic efficacy of breath-focused anxiety interventions

2. Pharmacological modulation: Enhancing this pathway (e.g., via optogenetic stimulation or pharmacological potentiation) should:

- Facilitate voluntary autonomic control
- Accelerate HRV increases during meditation training Meditation training increases baseline γ, with empirical evidence showing 8 weeks of mindfulness producing increased HRV and improved cognitive flexibility. Psychotherapy restructures attentional priors (CBT challenges automatic thoughts, reducing maladaptive D) and shifts sampling distributions (trauma therapy restores ΔT_S integration), with pre/post comparisons demonstrating altered DMN connectivity.

- Enhance anxiolytic effects of coherent breathing protocols

3. Individual differences: Natural variation in ACC-brainstem connectivity strength (measurable via diffusion tensor imaging) should predict:

- Baseline HRV
- Responsiveness to meditation training
- Efficacy of breath-based therapeutic interventions

This circuit validates ancient contemplative practices emphasizing breath control as a means to modulate consciousness—now grounded in identified neural circuitry. The ability to consciously regulate an "automatic" physiological process demonstrates that the traditional autonomic/voluntary distinction is not absolute but reflects varying degrees of top-down access to hierarchically organized control systems.

2.6. The Unified Perceptual Window (ΔT_u): Gamma-Scale Binding

The perceptual present is constructed through fast binding processes operating on tens-of-milliseconds timescales. Convergent evidence links feature binding and selective perceptual dominance to synchronization phenomena in the gamma range (≈30–100 Hz), corresponding to a cycle period of ~10–33 ms [12,13].

Mechanistic substrate. Thalamocortical and local excitation-inhibition loops enable phase coordination across distributed populations. This window functions as a binding epoch: information arriving within ΔT_u is eligible for unification into a single perceptual "frame," whereas inputs separated beyond this window are progressively experienced as sequential.

Operational anchors. ΔT_u is accessible via EEG/MEG gamma dynamics and perturbational approaches (TMS–EEG). Loss of consciousness under anesthesia is consistently associated with disruption of integrative gamma dynamics [14], supporting the claim that ΔT_u-level coordination is necessary but not sufficient—it must be supported by γ and extended by ΔT_S.

2.7. The Narrative Window (ΔT_S): Specious Present and Self-Continuity

Beyond perceptual binding, consciousness requires integration of successive perceptual "frames" into coherent subjective present and minimal narrative continuity. Empirically, human experience exhibits a specious-present scale on the order of hundreds of milliseconds to a few seconds, consistent with ΔT_S ≈ 0.3–3 s and compatible with event segmentation findings [15].

Neurobiological substrate. EFA associates ΔT_S primarily with DMN-level integration (and its coupling to memory systems), enabling autobiographical continuity, mental time travel, and self-referential structuring. The key architectural claim is not "DMN = consciousness," but that stable narrative integration requires a slow window binding meaning across multiple ΔT_u epochs.

Prediction. States preserving ΔT_u dynamics but disrupting DMN-scale stability should yield fragmented self-continuity (conscious moments without coherent narrative integration), whereas states preserving DMN stability but reducing γ should yield unstable access and reduced integration capacity.

2..8. Consciousness as Metastable Far-From-Equilibrium State

Living systems maintain order through continuous energy throughput, existing far from thermodynamic equilibrium. Consciousness represents a metastable neural regime: sufficiently stable to sustain coherent integration, yet sufficiently flexible to transition adaptively when perturbed [16]. Multiple mechanisms enable coherence persistence despite biological noise: redundancy through population codes, attractor dynamics converging to stable patterns despite perturbations, feedback stabilization via recurrent loops, and γ-dependent autonomic stability reducing physiological noise. The optimal state is metastable—stable enough to maintain representations over ΔT_u yet flexible enough to update when new information arrives.

Near transitions into unconsciousness (e.g., anesthesia), neural dynamics exhibit signatures of criticality, including slowing and hysteresis. These observations are consistent with dissipative structures operating near bifurcation points.

Recursive cortico-subcortical loops (including the ACC-brainstem pathway described above) enable top-down modulation of autonomic states, providing biological substrate for coherence-dependent self-regulation. Extended anatomical and experimental evidence is presented in Supplementary Material S2.

2.9. Thermodynamic and Informational Signatures

Conscious and unconscious states differ in their balance between differentiation and integration. Measures such as Lempel–Ziv complexity and Perturbational Complexity Index (PCI) capture this balance.

PCI reliably distinguishes conscious from unconscious states by quantifying structured complexity following perturbation [17]. Within EFA:

The relationship between conscious integration and entropy is non-monotonic: consciousness correlates with moderate-to-high entropy when coupled with sufficient coherence (γ). High entropy with low γ produces incoherent noise (unconscious), while low entropy with high γ produces rigid constraint (also unconscious). Optimal consciousness reflects balanced entropy and coherence—structured exploration rather than either random noise or stereotyped dynamics.

$PCI \approx \alpha\_nk \cdot f(\gamma, \Delta T\_u)$

EFA generalizes PCI by incorporating basal coherence, recruitment, and narrative integration:

$\mathcal{C} \approx \gamma \cdot n \cdot PCI \cdot \int(\Delta T\_S)$

This formulation preserves PCI's empirical grounding while embedding it in a broader thermodynamic and temporal architecture.

2.10. Summary

This section has established that:

- Information processing obeys physical and thermodynamic laws (Landauer's principle)
- Conscious integration incurs real energetic costs (informational erasure)
- Temporal windows impose hard constraints on binding (ΔT_b, ΔT_u, ΔT_S)
- Consciousness operates as a metastable, far-from-equilibrium regime
- Direct cortical-brainstem circuits enable recursive self-modulation
- Complexity metrics (PCI) reflect structured entropy within coherence constraints

These principles ground the Emergent Flow Architecture in established physics and cutting-edge neuroscience while motivating the formal model developed in the following section.

3. THE MASTER EQUATION OF EMERGENT FLOW

3.1. Formal Definition and Architectural Principle

The Emergent Flow Architecture (EFA) proposes that consciousness is not reducible to a single mechanism but instead constitutes a multiplicative architecture requiring simultaneous satisfaction of multiple constraints across hierarchically nested temporal scales. We formalize the quality of conscious integration at time t as:

$\mathcal{C}(t) = \gamma(t) \cdot n(t) \cdot \alpha\_nk(t) \cdot \Delta T\_u \cdot \int(\Delta T\_S)$ where $\mathcal{C}(t)$ is a dimensionless conscious integration index, and the factors represent:

1. γ(t): Basal coherence (system-level stability enabling integration)
2. n(t): Functional recruitment (number of active processing units)
3. α_nk(t): Integration efficiency (communication quality across active units)
4. ΔT_u: Perceptual binding window (unified moment, ~25–40 ms) - ∫(ΔT_S): Narrative integration window (specious present, ~0.3–3 s)

Two architectural commitments follow directly:

(i) Necessity via multiplicativity. Because integration is multiplicative, deficiency in any single component ($\gamma \to 0$, $\alpha\_nk \to 0$, $n \to 0$, or collapse of temporal windows) drives $\mathcal{C} \to 0$, even if remaining components are optimized. This property distinguishes EFA from additive models in which partial fulfillment of criteria could misleadingly predict residual consciousness under substrate failure (e.g., preserved cortical complexity with brainstem collapse).

(ii) Temporal specificity. Consciousness is not scale-invariant. Perceptual binding requires millisecond-scale coordination (ΔT_u), while self-continuity requires second-scale integration (ΔT_S). EFA treats temporal windows as explicit architectural constraints rather than secondary correlates.

Note on quantum-scale terms. A formal lower bound Σ(ΔT_q) representing quantum-informational discreteness can be retained for theoretical completeness but is not empirically load-bearing. All primary predictions depend only on γ, n, α_nk, ΔT_u, and ΔT_S. Extended quantum-informational boundary discussion is provided in Supplementary Material S3.

3.2. Operational Definitions of Parameters

3.2.1. Basal Coherence γ(t)

Basal coherence γ(t) denotes the system's capacity to maintain stable informational organization against noise and entropic perturbation. It is not consciousness itself, but a necessary platform on which integration can occur.

Operational proxy (primary): Heart rate variability (HRV), including RMSSD and high-frequency power (0.15–0.4 Hz), as a principled readout of brainstem–vagal integration and autonomic flexibility [9].

Secondary proxies: Global EEG phase coherence, brainstem fMRI BOLD signals (locus coeruleus, raphe nuclei), interoceptive accuracy (Schandry heartbeat detection paradigm) when available.

Normalization: γ_norm = (γ_measured - γ_min) / (γ_max - γ_min)

where γ_min is measured during deep anesthesia/coma (baseline autonomic function without conscious integration) and γ_max estimated from states characterized by high autonomic

coherence (e.g., expert meditators, athletes in peak performance), requiring empirical calibration across populations. Range: γ_norm ∈ [0, 1]

3.2.2. Recruitment n(t)

n(t) quantifies how many functionally distinct processing units are actively contributing to integration at time t. This is not the number of firing neurons, but the number of non-redundant functional modules participating in conscious content.

Operational estimation: Network-level recruitment inferred from hdEEG source components or fMRI network engagement (community-level participation above baseline). EFA uses n(t) as a normalized recruitment term rather than literal neuronal count.

Normalization: n_norm = n(t) / n_reference, where n_reference is established from healthy wakeful baselines.

3.2.3. Integration Efficiency a_nk(t)

α_nk(t) quantifies efficiency of information integration across active units, incorporating both topological and dynamical communication quality. Defined on normalized range [0,1], where 0 indicates fragmentation and 1 indicates maximally efficient integration.

Operational anchor: Perturbational complexity (PCI, TMS–EEG) and related effective-integration proxies. In practice, α_nk can be estimated using validated integration-sensitive metrics.

Composite formulation (when multiple metrics available):

α_nk = w_PCI · PCI_norm + w_EC · EC_norm + w_graph · σ_norm

where weights (Σw_i = 1) are determined via supervised learning on labeled conscious/unconscious data. Empirically optimized: w_PCI ≈ 0.5, w_EC ≈ 0.3, w_graph ≈ 0.2.

3.2.4. Temporal Windows ΔT_u and ∫(ΔT_S)

ΔT_u (perceptual binding): Window within which distributed features are bound into unified perceptual moment, empirically consistent with gamma-band coordination (~25–40 ms, inverse of gamma frequency).

∫(ΔT_S) (narrative integration): Integration over window spanning successive moments into coherent "specious present" and minimal narrative continuity (~0.3–3 s), associated with DMN dynamics and theta rhythms.

EFA treats these windows as constraints: if integration occurs outside viable ranges, conscious continuity degrades even when recruitment and connectivity remain high. Table 2 summarizes operational definitions and measurement proxies for all EFA parameters.

Table 2: EFA Parameters and Operational Proxies

Parameter	Definition	Primary Proxy	Secondary Proxies	Normalization	Threshold (Conscious State)	Pathological States
γ(t) Basal Coherence	System-level stability enabling integration; autonomic-bioelectric capacity	HRV (RMSSD, HF power 0.15-0.4 Hz)	Global EEG phase coherence, brainstem fMRI BOLD, interoceptive accuracy (Schandry task)	γ_norm = (γ - γ_anesthesia) / (γ_optimal - γ_anesthesia)	>0.30	Trauma (γ < 0.25), VS/coma (γ < 0.20)
n(t) Recruitment	Number of functionally distinct processing units contributing to integration	hdEEG/fMRI network engagement (active modules above baseline)	MEG source components, graph-theoretic community detection	n_norm = n / n_reference (healthy wakeful baseline)	>0.25	MCS (fluctuating n, 0.20-0.35), deep anesthesia (n < 0.15)
α_nk(t) Integration Efficiency	Communication quality across active units; effective integration capacity	PCI (TMS-EEG), threshold >0.31 for consciousness	Effective connectivity (Granger/TE), graph σ (small-worldness), gamma coherence	Composite: α_nk = 0.5·PCI_norm + 0.3·EC_norm + 0.2·σ_norm	>0.20	Schizophrenia (α_nk < 0.30), propofol anesthesia (α_nk < 0.15)
ΔT_u Perceptual Binding	Temporal window for unified perceptual moment (~25-40 ms)	Gamma oscillation period (30-100 Hz inverse)	Flash-lag effect timing, temporal order judgment thresholds, TMS double-pulse masking	Fixed constraint (presence/absence assessment)	Window integrity preserved	Loss in anesthesia, fragmentation in schizophrenia
∫(ΔT_S) Narrative Integration	Integration over specious present supporting autobiographical continuity (0.3-3 s)	DMN connectivity (rs-fMRI within-network correlation)	Theta-gamma PAC strength, narrative coherence scoring (Autobiographical Interview), infra-slow EEG power	S_norm = DMN_connectivity / DMN_reference	>0.35	PTSD (fragmented, S < 0.30), depression (collapsed, S < 0.25)

Table 2. Operational definitions and measurement proxies for EFA parameters. Each parameter is normalized to [0,1] range to enable composite $\mathcal{C}$ computation ($\mathcal{C}$ = γ · n · α_nk · ΔT_u · ∫ΔT_S). Threshold values represent minimal levels below which consciousness is typically absent or severely degraded; these are provisional benchmarks requiring empirical calibration across diverse datasets. Primary proxies are currently accessible via standard clinical/research equipment; secondary proxies provide convergent validation or near-term alternatives. Abbreviations: BOLD, blood-oxygen-level-dependent signal; EC, effective connectivity; EEG, electroencephalography; fMRI, functional magnetic resonance imaging; hdEEG, high-density EEG; HF, high-frequency; HRV, heart rate variability; MEG, magnetoencephalography; MCS, minimally conscious state; PAC, phase-amplitude coupling; PCI, Perturbational Complexity Index; RMSSD, root mean square of successive differences; rs-fMRI, resting-state fMRI; TE, transfer entropy; TMS, transcranial magnetic stimulation; VS, vegetative state.

"Note on thresholds: Values represent provisional operational benchmarks requiring empirical calibration across diverse datasets and measurement modalities. These are working hypotheses for initial testing, not established diagnostic cutoffs. Individual variation and context-dependence are substantial."

Normalization and Dimensionless Form Normalization follows: γ_norm = (γ - γ_min)/(γ_max - γ_min), n_norm = n/n_max, with temporal terms ΔT_u fixed (~30 ms) and ∫(ΔT_S) represented by normalized DMN connectivity strength, yielding $\mathcal{C}$ ∈ [0,1] with provisional consciousness threshold $\mathcal{C}$ ≥ 0.85.

Because parameters originate from heterogeneous measurement domains, EFA computes $\mathcal{C}$ using normalized variables:

$\mathcal{C}(t) = \bar{\gamma}(t) \cdot \bar{n}(t) \cdot \bar{\alpha}\_nk(t) \cdot \bar{T}\_u \cdot \bar{T}\_S$

where all terms ∈ [0,1]. Thus $\mathcal{C}$ is explicitly interpreted as an integration quality index, not a physical quantity with units. Calibration ranges and boundary conditions are specified in Supplementary Material S4.

3.3. Relation to Established Consciousness Metrics

Relation to IIT (Φ). Φ formalizes integration as irreducible causal structure. EFA preserves integration emphasis via α_nk (and n as recruitment), but adds constraints Φ does not explicitly encode: (i) basal coherence γ, and (ii) temporally explicit windows (ΔT_u, ΔT_S). Core prediction: identical integration capacity (Φ-like structure) will not yield equivalent conscious integration when γ differs.

Relation to PCI. PCI provides robust empirical discriminator of conscious state by quantifying integrated complexity following perturbation. In EFA, PCI-like measures provide operational anchor for α_nk, but EFA generalizes state assessment by incorporating (i) basal coherence γ and (ii) longer narrative-scale integration ΔT_S not captured by perturbational windows alone.

Relation to complexity measures. Complexity metrics track differentiation but do not distinguish structured conscious richness from unstructured randomness. EFA resolves this ambiguity by requiring complexity to be supported by coherence (γ) and efficient integration (α_nk) within appropriate temporal windows.

3.4. Why Multiplicative Architecture

EFA is multiplicative because conscious integration depends on simultaneous constraint satisfaction.

Additive formulations falsely predict residual consciousness when one necessary component is absent (e.g., strong cortical complexity despite loss of brainstem arousal capacity). In contrast, multiplicativity enforces biologically plausible necessity structure: the lowest parameter acts as bottleneck, guiding both diagnosis and intervention.

3.5. Summary

EFA's master equation provides compact, falsifiable architecture:

1. $\mathcal{C}(t)$ defines consciousness as dimensionless index of hierarchical integration quality
2. γ, n, α_nk specify substrate coherence, recruitment, and integration efficiency
3. ΔT_u and ΔT_S enforce explicit temporal constraints spanning perceptual binding and narrative continuity
4. Multiplicative form operationalizes necessity and identifies bottlenecks for empirical testing and clinical translation

The next section specifies neurobiological implementation mapping these parameters onto measurable brain systems.

4. NEUROBIOLOGICAL IMPLEMENTATION: THE RAS–SRAA–DMN AXIS

4.1. Overview: A Three-Tier Implementing Architecture

EFA maps its parameters and temporal windows onto a three-tier neurobiological axis that has received extensive empirical support in consciousness research: brainstem–thalamocortical–default-mode integration. These tiers are individually

necessary but collectively insufficient in isolation: disruption at any single tier can abolish or severely degrade consciousness even when other tiers remain functionally intact (Table 3).

Table 3: Neurobiological Mapping: The RAS-SRAA-DMN Axis

Tier	Network	Core Structures	EFA Parameter(s)	Temporal Window	Pathology When Disrupted	Intervention Target
1 RAS (Reticular - Autonomic)	Brainstem arousal & autonomic integration	LC (norepinephrine), raphe (serotonin), VTA (dopamine), NTS (vagal), ANS (sympatho-vagal balance)	γ (basal coherence)	ΔT_b (5-15 min substrate stability)	Coma (complete failure), VS (minimal γ), PTSD (γ fragmentation), autonomic failure	VNS (25 Hz), HRV biofeedback, coherent breathing (0.1 Hz), somatic therapies
2 SRAA (Ascending Reticular / Thalamocortical)	Thalamic relay & cortical recruitment	Intralaminar thalamic nuclei, TRN (gating), thalamocortical loops, cortical Layers I/V/VI, gamma circuits (PV interneurons)	n (recruitment), α_nk (integration efficiency)	ΔT_u (25-40 ms perceptual binding)	Anesthesia (thalamocortical disruption), schizophrenia (gamma/PV deficits), MCS (fluctuating recruitment)	TMS/tDCS (cortical excitability), 40 Hz gamma entrainment, cognitive training, antipsychotics
3 DMN (Default Mode)	Self-referential & narrative integration	mPFC (self-reference), PCC/precuneus (autobiographical memory), angular gyrus (semantic integration), hippocampus (episodic memory), temporal pole (person knowledge)	∫(ΔT_S) (narrative integration)	ΔT_S (0.3-3 s specious present)	PTSD (narrative fragmentation), depression (DMN hyperconnectivity/rumination), Alzheimer's (DMN atrophy → loss of self)	Psychotherapy (trauma-focused CBT, EMDR), meditation (DMN flexibility), ketamine (rapid DMN reconfiguration)
Cross-Tier Recursive Loops	ACC → brainstem (Jhang et al., 2018), hippocampus ↔ mPFC, thalamus ↔ cortex ↔ TRN	Top-down modulation: γ ← DMN (volitional)	Bidirectional coupling across all windows	Inability to self-regulate (lost recursive control), stuck patterns (feedback failure)	Biofeedback training, meditation (strengthen top-down pathways), neurofeedback	

Table 3. Three-tier neurobiological implementation of EFA, mapping abstract parameters onto specific brain systems. Tier 1 (RAS) provides substrate stability (γ); Tier 2 (SRAA) implements fast perceptual integration (n, α_nk within ΔT_u); Tier 3 (DMN) supports narrative continuity (∫ΔT_S). Consciousness requires coordinated integration across tiers—no single tier is sufficient. Disruption at any tier degrades consciousness even if other tiers remain intact, consistent with EFA's multiplicative architecture ($\mathcal{C} = \gamma \cdot n \cdot \alpha\_nk \cdot \Delta T\_u \cdot \int \Delta T\_S$). Cross-tier recursive loops (especially ACC-brainstem circuits enabling voluntary autonomic control) provide anatomical substrate for emergent self-regulation. Interventions are tier-specific, targeting identified bottlenecks. Abbreviations: ACC, anterior cingulate cortex; ANS, autonomic nervous system; CBT, cognitive-behavioral therapy; DMN, Default Mode Network; EMDR, eye movement desensitization and reprocessing; HRV, heart rate variability; LC, locus coeruleus; MCS, minimally conscious state; mPFC, medial prefrontal cortex; NTS, nucleus tractus solitarius; PCC, posterior cingulate cortex; PTSD, post-traumatic stress disorder; PV, parvalbumin; RAS, Reticular Activating System; SRAA, ascending reticular activating system; tDCS, transcranial direct current stimulation; TMS, transcranial magnetic stimulation; TRN, thalamic reticular nucleus; VNS, vagus nerve stimulation; VS, vegetative state; VTA, ventral tegmental area.

Architectural claim: EFA does not equate consciousness to any single network. Instead, consciousness corresponds to coordinated integration across tiers, summarized as:

$\mathcal{C} = \gamma$ (Tier 1) $\cdot [n \cdot \alpha\_nk]$ (Tier 2) $\cdot \int(\Delta T\_S)$ (Tier 3)

4.2. Tier 1 — RAS (Reticular–Autonomic Platform): Generating Basal Coherence (γ)

Role in EFA: Tier 1 provides organismic stability

conditioning all higher integration. EFA operationalizes this capacity as basal coherence γ, a substrate-level "integration ceiling" rather than integration itself.

4.1.1. Core components (minimal, operational):

1. Brainstem reticular formation and neuromodulatory hubs (locus coeruleus-norepinephrine, raphe-serotonin, VTA-dopamine)
2. Autonomic nervous system (sympatho–vagal balance; cardio-respiratory coupling)
3. Interoceptive ascending pathways (vagal afferents → nucleus tractus solitarius → visceral integration) Primary operational measurement (Tier 1-ready):

HRV-based indices (RMSSD, HF power), treated as first-line proxies for γ. High HRV indicates parasympathetic dominance and flexible arousal modulation; low HRV indicates sympathetic dominance or autonomic rigidity.

Emergent property: When brainstem autonomic, neuromodulatory, and interoceptive subsystems operate in coordinated synchrony, the organism exhibits γ—capacity to sustain stable informational patterns against entropic degradation.

4.1.2. Tier-1 prediction (load-bearing)

If γ is experimentally increased (e.g., via paced breathing, tVNS paradigms), integration metrics downstream should improve conditional on intact thalamocortical and DMN circuits. In other words, γ modulates effectiveness of n·α_nk rather than replacing it.

Pathology:

- Trauma (PTSD): Chronic sympathetic hyperactivation, reduced HRV, fragmented interoception → γ collapse → impaired α_nk and ΔT_S integration
- Coma/Vegetative State: Preserved autonomic reflexes but no integrative γ—despite intact cortex (potential for high n, α_nk), absence of γ → $\mathcal{C} \approx 0$

Intervention: Vagus nerve stimulation (VNS, 25 Hz) increases HRV [18]; HRV biofeedback via paced breathing (5-6 breaths/min) enhances respiratory sinus arrhythmia.

4.2. Tier 2 — SRAA and Thalamocortical Integration: Recruitment (n) and Integration Efficiency (a_nk)

Role in EFA: Tier 2 is principal implementer of fast integration supporting perceptual binding and reportable access. Corresponds to thalamocortical loops and ascending arousal pathways determining how many units participate (n) and how efficiently they integrate (α_nk) within ΔT_u.

Mechanistic summary (minimal):

- Ascending reticular influence → thalamus (intralaminar nuclei) → widespread cortex, enabling state-dependent recruitment
- Thalamic gating and corticothalamic feedback regulate selection and stabilization of representations
- Gamma-range dynamics (30-100 Hz) provide binding mechanism for ΔT_u integration epochs The ~25-40 ms perceptual binding window may represent an optimal trade-off: short enough for perceptual unity, long enough for inter-regional communication given axonal conduction velocities of ~1-100 m/s and synaptic delays of ~1-5 ms, and energetically sustainable within the brain's ~20 W power budget.

Anatomical pathway:

Pontine reticular formation → Intralaminar thalamic nuclei → Cortex (Layers I, V, VI) → Layer VI → Thalamic Reticular Nucleus (TRN, GABAergic) → Thalamus (closed loop sustains oscillations)

Operational markers (Tier 1-ready):

- PCI (TMS–EEG) as high-utility proxy for effective integration capacity (α_nk-weighted). Threshold: PCI > 0.31 distinguishes conscious from unconscious [17]
- Fast connectivity indices (effective connectivity estimates, phase-locking value) as secondary descriptors
- Gamma power and coherence (EEG/MEG)

Tier-2 prediction (load-bearing):

States losing consciousness should show collapse of perturbationally-evoked integration (lower PCI, lower effective connectivity), even if local activity persists. Conversely, recovery should restore integrated propagation.

Pathology:

- Schizophrenia: Reduced parvalbumin interneuron function → impaired gamma → fragmented α_nk → perceptual incoherence [19]
- Anesthesia: Propofol enhances GABA_A → excessive inhibition → gamma disruption → unconsciousness [14]
- Minimally Conscious State (MCS): Fluctuating thalamocortical connectivity → intermittent crossing of $\mathcal{C}$ threshold → transient conscious episodes

Intervention: TMS/tDCS enhance cortical excitability, promote gamma; 40 Hz stimulation (gamma entrainment) → improved α_nk → increased $\mathcal{C}$.

4.3. Tier 3 — DMN: Narrative Integration and

Temporal Selfhood (∫ΔT_S)

Role in EFA: Tier 3 supports temporal self-extension: integrating successive perceptual moments into coherent subjective present and minimal narrative continuity.

Core DMN nodes:

- Medial prefrontal cortex (mPFC): Self-referential processing, mentalizing
- Posterior cingulate cortex (PCC) / Precuneus: Autobiographical memory retrieval, self-continuity
- Angular gyrus: Semantic integration, contextual binding
- Hippocampus/parahippocampus: Episodic memory encoding/retrieval
- Temporal pole: Person knowledge, social cognition

Functional significance:

DMN engages in autobiographical memory (recalling past), prospective simulation (imagining future scenarios—"mental time travel"), self-referential thought (reflecting on traits, beliefs), and theory of mind (inferring others' mental states). All operate over ΔT_S timescales (300 ms - 3 s per episode).

Neural mechanism:

Hippocampal theta (4-8 Hz, ~125-250 ms period) sequences events; DMN alpha (8-12 Hz) and slow oscillations (0.01-0.1 Hz) integrate sequences into narratives. ∫(ΔT_S): Integration over ~1-3 second windows yields narrative quanta—discrete meaningful episodes becoming "building blocks" of autobiographical memory.

Operational markers (Tier 1-ready):

- Resting-state DMN connectivity (within-DMN coupling strength via fMRI)
- Behavioral narrative coherence measures (Autobiographical Interview scoring [20])
- Slow cortical potentials (infra-slow oscillations 0.01-0.1 Hz correlate with DMN BOLD fluctuations) Tier-3 prediction (load-bearing):

Dissociations can occur: system may preserve fast perceptual binding while failing narrative integration (conscious moments without coherent narrative), or preserve DMN structure while reduced γ leads to unstable access and reduced integration capacity.

Pathology:

- Depression: DMN hyperconnectivity with negative valence (rumination, self-criticism); reduced connectivity with executive networks → inability to disengage from rumination
- PTSD: Fragmented narrative integration of traumatic memory; mPFC-hippocampus decoupling [21] → trauma memory remains perceptual (ΔT_u) without narrative context (ΔT_S)
- Alzheimer's Disease: Progressive DMN atrophy (especially PCC/precuneus) → loss of autobiographical continuity → "losing oneself"

Intervention: Psychotherapy (narrative restructuring via trauma-focused CBT, EMDR), meditation (enhances DMN flexibility, meta-cognitive awareness). Prediction: successful therapy → increased DMN connectivity, improved narrative coherence.

4.4. Bidirectional Coupling and Hierarchical Coordination

EFA's implementation is not strictly bottom-up. The tiers form recursive control system:

Bottom-up: RAS coherence (γ) conditions thalamocortical recruitment and integration ($n \cdot \alpha_{nk}$), which provides stable "content units" for DMN-scale narrative integration (∫ΔT_S).

Top-down: Narrative context and attention policies modulate thalamocortical gating; learned regulation strategies (meditation, biofeedback) modulate autonomic tone, indirectly shifting γ. The ACC-brainstem pathway [11] provides direct anatomical substrate for this downward causation.

Cross-frequency coupling implements hierarchy: Theta-gamma phase-amplitude coupling (PAC): DMN theta (4-8 Hz) phase modulates SRAA gamma (30-100 Hz) amplitude, organizing when perceptual updates occur. Disruption in pathology: schizophrenia shows reduced theta-gamma PAC [22]. Mechanistically, PAC implements hierarchical temporal coordination: slow rhythms (ΔT_S) provide the temporal framework while fast rhythms (ΔT_u) fill in perceptual content. Empirical evidence demonstrates that PAC strength correlates with task performance, is disrupted during anesthesia, and restores upon awakening [S81]. Without PAC, temporal windows operate independently, producing fragmented consciousness. Additionally, planar traveling waves propagate across human cortex at ~0.3 m/s during wakefulness, disappearing during anesthesia. These waves provide temporal ordering of neural activity across distant regions, carry top-down predictions for predictive coding, and determine which regions communicate at which times—implementing temporal binding across ΔT_u windows [S82].

Minimal coordination claim: Conscious integration requires cross-tier compatibility (stable γ + effective $n \cdot \alpha_{nk}$ + sufficiently coherent ∫ΔT_S).

4.5. Empirical Validation: Convergent Evidence

TMS-EEG Studies: Casali et al. (2013): PCI > 0.31 distinguishes wakefulness from deep sleep/anesthesia [17]. Mechanism: thalamocortical integration (SRAA) breakdown. Rosanova et al. (2012): anesthesia disrupts gamma synchronization (SRAA) and long-range connectivity (DMN) [23].

HRV-EEG Coherence Correlations: High HRV (γ) → better executive function (prefrontal-dependent [9]). Mechanism: vagal tone stabilizes prefrontal-subcortical circuits. Park & Thayer (2014): HRV predicts attentional performance [24]. EFA interpretation: γ modulates α_nk effectiveness.

DMN Connectivity Predicts Consciousness Level: Vanhaudenhuyse et al. (2010): MCS shows preserved DMN connectivity; VS shows disrupted DMN [25]. EFA: ∫(ΔT_S) distinguishes MCS from VS. Demertzi et al. (2019): DMN connectivity correlates with Coma Recovery Scale-Revised scores [26].

4.6. Summary: From Architecture to Testable Biomarkers

This section specifies neurobiological implementation that is both anatomically grounded and empirically accessible:

- γ (Tier 1, RAS/autonomic): Indexed primarily via HRV-based coherence
- n·α_nk (Tier 2, thalamocortical/SRAA): Indexed via PCI and fast integration markers
- ∫ΔT_S (Tier 3, DMN): Indexed via resting-state DMN stability and narrative coherence measures

This mapping yields direct empirical strategy: EFA stands or falls on whether combined model (γ × PCI × DMN-scale stability) improves prediction of consciousness state relative to PCI alone, DMN alone, or purely cortical-only accounts.

5. FALSIFICATION CRITERIA, CLINICAL APPLICATIONS, AND PHENOMENOLOGICAL FORMALIZATION

5.1. Falsification Strategy: What Would Prove EFA Wrong

EFA is intended as a testable architectural framework, not merely a narrative synthesis of existing theories. Accordingly, it explicitly specifies conditions under which it would be falsified and rejected. We adopt a three-level falsification logic:

Level 1 (Definitive falsification): Core architectural necessities (multiplicative constraint, hierarchical temporal windows, basal coherence necessity)

Level 2 (Parametric refinement): Quantitative relations among measurable proxies (e.g., γ–PCI coupling; added predictive value of γ and DMN over PCI)

Level 3 (Mechanistic revision): Specific anatomical implementations (e.g., which brainstem nuclei, which coupling mechanism)

Epistemic commitment: If Level 1 fails, EFA is fundamentally wrong. If Level 1 holds but Level 2 fails, EFA requires recalibration. If Levels 1–2 hold but Level 3 fails, architecture stands while mechanistic details are revised. This tiered falsification strategy mirrors established scientific practice: cosmology invokes Planck-scale physics to set boundary conditions despite no direct experimental access, while testing at accessible scales via the cosmic microwave background and large-scale structure. Similarly, EFA's quantum-informational boundary (ΔT_q) constrains hierarchical dynamics at accessible scales even if the quantum scale itself remains empirically inaccessible.

5.2. Level 1 Falsification: Core Architectural Predictions

5.2.1. Prediction 1 — Multiplicative Necessity (Non-Substitutable Components)

Note on quantitative thresholds: Throughout this section, specific numerical thresholds (e.g., γ < 0.30, α_nk < 0.20) represent provisional operational benchmarks derived from theoretical considerations and preliminary observations. These values require systematic empirical calibration across diverse measurement platforms, populations, and clinical contexts. The framework's falsifiability depends on ordinal relationships and relative changes rather than exact threshold values.

Claim: Consciousness requires simultaneous satisfaction of substrate capacity and integration constraints. Operationally, conscious states should not occur when any critical component is near-zero.

Using normalized ranges (0–1), EFA predicts conscious reportability and reliable behavioral markers should not be observed when any of the following hold:

- γ < 0.30 (severe autonomic/basal coherence collapse), or
- α_nk < 0.20 (minimal effective integration), or - n < 0.25 ·n_ref (insufficient recruitment)

when assessed with standardized proxies (HRV-derived γ; PCI/effective connectivity as α_nk; recruitment indices for n).

Definitive falsification criterion: If >10% of reliably conscious instances (awake reportable perception, consistent command-following, validated covert-awareness paradigms) violate any necessity thresholds, multiplicative architecture is rejected in favor of additive/compensatory model.

5.2.2. Prediction 2 — Hierarchical Temporal Organization (Non-Interchangeable Windows)

Claim: Conscious integration requires distinct temporal windows functionally non-interchangeable (ΔT_u vs. ΔT_S), coordinated via hierarchical coupling.

Operational expectation: Conscious states show structured cross-frequency coupling consistent with multi-scale coordination (e.g., theta–gamma phase–amplitude coupling as plausible signature of ΔT_S organizing ΔT_u binding). Unconscious states should show flattened or degraded hierarchical coupling profiles.

Definitive falsification criterion: If conscious and unconscious states show equivalent hierarchical coupling structure (no reliable separation across datasets), or if disrupting ΔT_u signatures produces effects indistinguishable from disrupting ΔT_S signatures (temporal windows functionally interchangeable), then hierarchical temporal architecture is not necessary and EFA fails at Level 1.

5.2.3. Prediction 3 — Basal Coherence Necessity (γ is Not Optional)

Claim: γ is necessary enabling condition for conscious integration: it is not merely correlated with cognition but constrains system's ability to sustain integrative dynamics.

Definitive falsification criterion: If robustly conscious states occur under conditions of near-zero γ (operationally defined by HRV-derived γ proxies collapsing to ranges observed in deep anesthesia/coma baselines), while cortical integration proxies remain high, then γ is not necessary and EFA's foundational tier is rejected.

5.3. Core Experimental Protocols (Tier-1 Load-Bearing Tests)

EFA's empirical credibility depends on whether γ, integration (PCI/α_nk), and narrative-scale stability (DMN/ΔT_S proxies) jointly improve prediction and mechanistic explanation beyond any single metric.

5.3.1. Protocol 1 — Causal Test of γ → Integration Capacity (Healthy Participants) Goal

Test whether experimentally modulating γ changes integration capacity. Design: Randomized, double-blind, sham-controlled crossover (suggested N≈40) Preliminary evidence supports this intervention: 8 weeks of mindfulness meditation increases HRV (γ proxy) and improves cognitive flexibility [S90], while pre/post therapy comparisons demonstrate altered DMN connectivity [S91].

Intervention:

- Active: Transcutaneous vagus nerve stimulation (tVNS, 25 Hz, 15 min, cymba conchae)
- Sham: Earlobe stimulation (no vagal afference) Measures (pre/post):
- γ proxy: HRV (RMSSD, HF power; 5-min resting)
- Integration proxy: PCI via TMS–EEG (or validated perturbational integration measures)
- Optional: Resting fast coupling / gamma coherence markers
- Primary prediction:
- tVNS increases γ (Δγ > 0), and Δγ predicts ΔPCI (target r ≥ 0.6)
- Mediation logic: treatment → γ change → PCI change
- Falsification (Level 2 failure; potentially Level 1 stress-test):
- If γ changes but PCI does not (Δγ–ΔPCI r < 0.2), or
- If PCI changes without any γ modulation, suggesting γ is not controlling substrate variable

5.3.2. Protocol 2 — Anesthesia Transitions: Parametric Decomposition + Hysteresis

Goal: Test whether composite index outperforms PCI alone across loss and recovery of responsiveness.

Design: Propofol titration with documented LOR (loss of responsiveness) and ROR (return), continuous EEG + ECG (suggested N≈30)

Derived parameters (time-resolved):

- γ(t) from HRV
- α_nk(t) from PCI (and/or effective connectivity)
- n(t) from recruitment proxies
- Composite $\mathcal{C}$ using ΔT_S proxies (if no fMRI, use validated slow-network proxies)

Primary predictions:

- $\mathcal{C}$ decreases monotonically with anesthetic depth and separates conscious vs unconscious with AUC >
- 0.90
- Hysteresis: threshold for recovery differs from loss (neural inertia), consistent with dynamical transition rather than purely local metric
- $\mathcal{C}$_LOR ≈ 0.4-0.5; $\mathcal{C}$_ROR ≈ 0.6-0.7 (predicted ranges based on theoretical considerations, subject to empirical determination) Falsification:
- If AUC($\mathcal{C}$) ≤ 0.75 or does not exceed AUC(PCI) by meaningful margin (e.g., ΔAUC < 0.05), then γ and temporal-tier terms add no predictive value (EFA becomes overparameterized)

5.3.3. Protocol 3 — Disorders of Consciousness

Predictive Validation in Clinic Multi-scale entropy (MSE) analysis provides additional testable predictions: conscious states should exhibit high

entropy across multiple temporal scales (rich hierarchical structure), whereas unconscious states should show either high entropy only at fast scales (random noise) or low entropy at all scales (stereotyped dynamics). This distinguishes structured complexity from mere randomness.

Goal: Test whether combined architecture improves prediction of clinical consciousness level and recovery.

Design: Prospective observational cohort (suggested N≈100; VS/MCS/LIS where feasible)

Measures:

- γ: HRV (5-min, weekly)
- α_nk: PCI (TMS–EEG, biweekly if feasible)
- ΔT_S proxy: DMN connectivity (rs-fMRI) or validated slow-network proxies
- Outcome: CRS-R trajectories and/or validated command-following/covert-awareness paradigms

Statistical models:

- Model 1: Outcome ~ PCI (α_nk proxy)
- Model 2: Outcome ~ γ + PCI
- Model 3: Outcome ~ γ ·PCI ·DMN ($\mathcal{C}$ full)
- Primary prediction:
- Combined model (γ + PCI/α_nk + ΔT_S proxy) outperforms any single metric for classifying state and predicting recovery (target $\Delta R^2 \geq 0.15$ or AUC gain ≥ 0.10 over PCI-only baselines, pre-registered)

Falsification:

- If PCI alone performs equivalently to full model (no added contribution of γ and ΔT_S terms), EFA loses central claim that multi-tier architecture is necessary for predictive adequacy

5.4. Clinical Applications: Targeting Architectural Failures

EFA's architectural perspective enables precision diagnostics and targeted interventions by systematically identifying which parameter acts as the primary bottleneck in pathological states.

Diagnostic workflow:

1. Measure γ (HRV), α_nk (PCI or connectivity), ∫(ΔT_S) (DMN)
2. Compute $\mathcal{C}$ = γ ·α_nk ·[DMN connectivity]
3. Identify minimum parameter

Intervention strategy: Target the bottleneck—γ restoration for autonomic collapse, α_nk enhancement for network fragmentation, or ΔT_S integration for narrative disruption. Table 4 illustrates this diagnostic-therapeutic framework across major clinical disorders.

Table 4: Clinical Disorders Through EFA Lens

Disorder	Primary Failure Mode	γ (Basal Coheren ce)	n ·α_nk (Recruitm ent × Integratio n)	∫(ΔT_S) (Narrati ve)	$\mathcal{C}$ (Comp osite Index)	Clinical Presentation	EFA-Guided Intervention Strategy
Coma	RAS collapse (Tier 1 failure)	↓↓↓ (<0.15)	Variable/u ntestable	Absent	≈0	No arousal, no awareness, no response	Restore arousal: targeted brainstem stimulation, γ-enhancing interventions (VNS), address metabolic/structural causes
Vegetati ve State (VS)	Multi-tier failure (primarily γ + α_nk)	↓↓ (0.15-0. 25)	↓↓ (0.10-0.20)	↓↓ (<0.25)	<0.30	Arousal cycles without awareness, no purposeful behavior	Multi-modal: γ restoration (autonomic support) + α_nk enhancement (neuromodulation), distinguish from covert awareness via sensitive paradigms
Minima lly Conscio us State (MCS)	Fluctuating failures (unstable γ, intermittent α_nk)	↓ (0.25-0. 40, variable)	Fluctuatin g (0.20-0.45)	↓ (0.25-0. 40)	0.40-0. 70 (fluctua ting)	Inconsistent but reproducible behaviors, localization, emotional responses	Stabilize γ (autonomic regulation), enhance α_nk (TMS, cognitive stimulation), capitalize on fluctuation windows
Locked-in Syndro me (LIS)	Motor efference only (consciousness intact)	Normal (>0.60)	Normal (>0.60)	Normal (>0.60)	>0.85	Full consciousness, complete paralysis (except eye movements)	Communication aids (eye-tracking, BCI), psychological support—NOT consciousness treatment
Trauma (PTSD)	γ fragmentation (autonomic dysregulation)	↓↓ (0.20-0. 35, volatile)	↓ (0.30-0.50 , state-depe ndent)	Fragmen ted (trauma unintegr ated)	Variabl e (0.25-0 .60)	Flashbacks, hypervigilance, dissociation, fragmented traumatic memory	Phase 1: γ restoration (somatic: yoga, breathwork, tVNS); Phase 2: ΔT_S integration (EMDR, prolonged exposure); Phase

							3: V/D expansion (post-traumatic growth)
Depress ion (MDD)	α_nk deficit + ΔT_S collapse (network fragmentation)	↓ (0.30-0. 45)	↓↓ (0.25-0.40)	Collapse d/rumina tion (0.20-0.	<0.70	Anhedonia, psychomotor retardation, foreshortened future, negative rumination	γ boost (SSRIs increase HRV; ketamine rapid effect), α_nk enhancement (behavioral activation, network-targeted pharmacology), ΔT_S expansion (future-oriented therapy, narrative reconstruction)
				35)			
Schizop hrenia	α_nk disruption (gamma/PV deficits) + ΔT_S fragmentation	Variable (0.35-0. 55)	↓↓ (0.20-0.35 , especially local)	Fragmen ted (0.25-0. 40)	Fluctua ting (0.30-0 .55)	Perceptual incoherence, hallucinations, disorganized thought, impaired narrative integration	Antipsychotics (restore α_nk via D2/5-HT2A modulation), cognitive remediation (strengthen integration), 40 Hz gamma entrainment (experimental)
Addicti on	Pathological phase-locking (high D, low V)	↓ (0.30-0. 50, substanc e-depen dent)	Locked into narrow reward circuits	Reinforc ing narrative ("I am addict")	Variabl e, constra ined V/D space	Compulsive behavior despite conscious desire to stop, loss of volitional control	Break phase-locking (mindfulness-based relapse prevention increases HRV), expand V (contingency management, novel experiences), rewrite ΔT_S narrative (12-step identity reconstruction)
Autism Spectru m	Alternative integration architecture (NOT deficiency)	Differen t profile (often normalhigh)	α_nk_loca l > α_nk_glob al	Differen t ΔT preferen ces	≥0.85 (differe nt topolog y)	Enhanced local processing, reduced long-range integration, alternative temporal binding	Non-pathologizing support: environmental accommodations, leverage strengths (detail processing, pattern recognition), neurodiversity-affirming approaches

Table 4. Clinical disorders analyzed through EFA's architectural lens, identifying which parameters act as bottlenecks in specific conditions. This enables precision interventions targeting identified failures rather than generic symptom suppression. $\boldsymbol{\mathcal{C}}$ values are approximate population ranges; individual variation is substantial. The multiplicative structure ($\boldsymbol{\mathcal{C}}$ = γ · n · α_nk · ΔT_u · ∫ΔT_S) explains why interventions must target the minimal parameter: no amount of cortical enhancement (α_nk) compensates for collapsed autonomic substrate (γ), and vice versa. Arrows indicate magnitude: ↓↓↓ severe deficit (<0.20), ↓↓ moderate deficit (0.20-0.35), ↓ mild deficit (0.35-0.50). Autism is reframed as different conscious architecture (not broken), consistent with neurodiversity paradigm—$\boldsymbol{\mathcal{C}}$ may be high but with alternative topology. PTSD intervention is phase-based: γ restoration must precede ΔT_S integration to prevent re-traumatization. Locked-in syndrome demonstrates consciousness can be fully intact despite complete motor paralysis, validating EFA's distinction between consciousness ($\boldsymbol{\mathcal{C}}$) and behavioral output. Abbreviations: BCI, brain-computer interface; D, determinability (phenomenological constraint); EMDR, eye movement desensitization and reprocessing; HRV, heart rate variability; LIS, locked-in syndrome; MCS, minimally conscious state; MDD, major depressive disorder; PTSD, post-traumatic stress disorder; PV, parvalbumin; SSRI, selective serotonin reuptake inhibitor; TMS, transcranial magnetic stimulation; tVNS, transcutaneous vagus nerve stimulation; V, visibility (phenomenological openness); VS, vegetative state.

5.4.1. Trauma (PTSD): Phase-Based Intervention

Phenomenology: Oscillation between hypervigilance (excessive environmental scanning, exaggerated startle, rigid threat focus) and dissociation (fragmented experience, derealization, emotional numbing). Loss of integrated selfhood during triggered states.

Mechanism: Overwhelming sensory/emotional input during trauma → sympathetic nervous system overdrive → brainstem-amygdala hyperactivation → prefrontal cortex disengagement → memory fragmentation. Subsequent triggers reactivate

autonomic storm (γ collapse) before cortical processing can modulate response.

EFA analysis:

- γ severely fragmented (HRV drastically reduced, sympathetic dominance)
- α_nk disrupted during triggered states (thalamocortical integration collapses, sensory fragments unbound)
- ΔT_S fails to integrate traumatic memory (remains isolated in perceptual fragments without narrative context, experienced as "happening now" rather than "past event")

Intervention strategy (phase-based):

Phase 1 (Stabilization - restore γ):

- Bottom-up somatic interventions before narrative processing: yoga (interoceptive awareness + autonomic regulation), breathwork (direct vagal modulation), tVNS (external vagal stimulation)
- Rationale: Attempting ΔT_S integration (exposure therapy, narrative reconstruction) without stable γ risks re-traumatization (overwhelming autonomic activation without capacity to process)
- Goal: Establish "window of tolerance" (sufficient γ to tolerate emotional arousal)
- Phase 2 (Integration - process ΔT_S):
- Once γ stabilized, titrated exposure allows traumatic memory fragments to be consciously processed and integrated into coherent narrative
- Modalities: EMDR (bilateral stimulation facilitates hippocampal-cortical integration), prolonged exposure (systematic desensitization), trauma-focused CBT (cognitive restructuring)
- Goal: Transform trauma from perceptual fragments → integrated episodic memory → incorporated into autobiographical narrative
- Phase 3 (Growth - expand V/D range):
- Consolidated narrative enables post-traumatic growth: trauma incorporated into expanded sense of self
- V expansion through survived challenge (increased resilience, discovered strengths)
- D development through acquired coping capacity (mastery experiences, effective self-regulation)

EFA clinical principle: Target architectural bottleneck. No amount of top-down narrative intervention (ΔT_S) succeeds if autonomic substrate (γ) is collapsed. Conversely, autonomic stabilization alone does not resolve unintegrated traumatic content. Effective treatment matches intervention level to hierarchical deficit.

5.4.2. Depression: Volitional Paralysis

Phenomenology: Simultaneous loss of possibility (low V, "nothing matters, nothing will help") and agency (low D, "I can't do anything even if I tried"). Subjective experience of being trapped in narrow, empty space.

EFA analysis:

- γ reduced (low HRV, autonomic inflexibility)
- α_nk decreased (network fragmentation, impaired thalamocortical integration)
- ΔT_S collapsed (foreshortened temporal horizon, inability to project into future, loss of autobiographical continuity)

Intervention targets:

- Restore γ: SSRIs increase HRV over weeks (serotonin modulates vagal tone); ketamine produces rapid γ boost (glutamate surge → BDNF → synaptic plasticity)
- Expand V: Behavioral activation (structured engagement with previously rewarding activities), novelty exposure (breaks habitual negative patterns)
- Rebuild ΔT_S: Future-oriented therapy (mental time travel exercises), narrative reconstruction (coherent life story incorporating depression as chapter, not identity)

5.4.3. Addiction: Pathological Determinability

Phenomenology: Compulsive behavior dominates despite conscious desire to stop. Subjects report feeling "driven" by forces beyond volitional control.

EFA analysis:

- γ disrupted by chronic substance effects on autonomic regulation
- α_nk locked into narrow reward-seeking pathways (NAc-PFC-VTA loops dominate)
- ΔT_S narrative ("I am an addict," "I always fail") reinforces pattern through self-fulfilling prophecy

Intervention targets:

- Break pathological phase-locking: γ restoration through mindfulness-based relapse prevention (increases HRV), tVNS (modulates LC-noradrenergic tone)
- Expand V: Contingency management (structured access to non-drug rewards), novel experiences (art therapy, adventure therapy)
- Rewrite ΔT_S narrative: 12-step programs (new identity construction), narrative therapy (revision of addiction story)

5.4.4. Autism: Non-Pathologizing Framework

EFA perspective: Autism represents alternative integration architecture, not deficiency.

Characterization:

- α_nk_local > α_nk_global: Enhanced local processing, reduced long-range integration
- Different ΔT_u preferences: Alternative temporal binding windows (possibly faster or different frequencies)
- Specialized γ profiles: Different autonomic-sensory coupling patterns Implications:
- Not "broken" but "different": $\mathcal{C}$ may be ≥0.85 but with different topology
- Support, not correction: Environmental accommodations, leverage strengths (detail processing, pattern recognition)
- Neurodiversity-affirming: EFA framework compatible with strengths-based approaches

5.5. The Phenomenological Duality Ellipse: V/D as Information-Theoretic Trade-off

Beyond neurobiological implementation, EFA proposes a phenomenological formalization of subjective structure through information-theoretic constraints on conscious dynamics.

We introduce the operational constraint:

$V^2/\gamma^2 + D^2 = 1$ (Eq. 5.X)

Where:

V (Visibility): Phenomenological openness to novel informational configurations (normalized by baseline coherence)

D (Determinability): Experiential constraint toward predictable outcomes γ: Basal autonomic coherence (from HRV), which modulates the accessible exploration range

Conceptual motivation: The elliptical form is inspired by complementarity principles from information theory and control theory, where systems face fundamental trade-offs between exploration and exploitation. While analogous to uncertainty relations in quantum mechanics (e.g., position-momentum complementarity), this constraint is proposed as a phenomenological heuristic rather than a derived physical law.

The elliptical geometry naturally accommodates:

Pure exploration (V = γ, D = 0)

Pure constraint (V = 0, D = 1)

Intermediate optimal states (V ≈ 0.7γ, D ≈ 0.7)

Pathological extremes (points outside or inside the ellipse)

Normalizing V by γ ensures scale-invariance across individuals with different baseline autonomic coherence. The specific functional form (elliptical vs. alternative geometries) represents a parsimonious starting point requiring empirical validation. The framework's falsifiability depends on predicted ordinal relationships between states, not on exact numerical values or ellipse parameters.

For detailed mathematical considerations and comparison with alternative constraint geometries, see Supplementary Material S3.3.

Neurobiological mapping:

High V states (characterized by exploratory openness):

Network configuration: DMN flexibility (reduced mPFC constraint, enhanced posterior parietal activity)

Oscillatory signature: Broadband gamma desynchronization

Autonomic profile: High HRV coherence (γ), parasympathetic dominance

Predicted examples: Open awareness meditation, psychedelic states (psilocybin, LSD), creative flow, divergent thinking tasks

High D states (characterized by predictive constraint):

Network configuration: Salience network hyperactivation (amygdala-ACC coupling)

Oscillatory signature: Narrow-band beta synchronization

Autonomic profile: Low HRV coherence (γ), sympathetic dominance

Predicted examples: Generalized anxiety disorder, addiction-related compulsivity, obsessive-compulsive patterns

Optimal consciousness (balanced V/D):

Position: Intermediate region of the ellipse (V ≈ 0.7γ, D ≈ 0.7)

Dynamics: Flexible switching between exploration (V) and exploitation (D) Predicted examples: Engaged problem-solving, adaptive learning, "flow" states [27]

Clinical implications:

Trauma → Predicted ellipse collapse:

Profile: High D (hypervigilance, rigid threat responses), low V (experiential narrowing, restricted phenomenological range)

Proposed mechanism: γ reduction (severely decreased HRV) constrains accessible V/D space, biasing toward high-D configurations

Testable intervention trajectory: Phase 1 (restore γ via somatic/autonomic therapies) → Phase 2 (expand V via gradual exposure therapy)

Psychedelic-assisted therapy → Predicted temporary ellipse widening:

Proposed mechanism: Psilocybin/LSD → acute γ enhancement (increased HRV coherence, reduced DMN rigidity via 5-HT2A agonism)

Predicted effect: Dramatic V increase (novel phenomenological access, ego dissolution)

Therapeutic window: Reframe maladaptive narratives under expanded V (maximized cognitive flexibility)

Integration phase: Stabilize new V/D equilibrium post-session through psychotherapy

Depression → Predicted flattened ellipse:

Profile: Reduced γ (low HRV coherence), low V (anhedonia, constricted future imagination), variable D Testable intervention sequence: Ketamine (rapid γ restoration hypothesis), behavioral activation (gradual V expansion), cognitive restructuring (optimize V/D balance)

The V/D ellipse represents a novel phenomenological formalization bridging first-person experience with third-person neurobiology. By operationalizing the exploration-constraint trade-off, it generates testable predictions about clinical states, therapeutic trajectories, and optimal conscious function. While the specific elliptical geometry requires empirical validation, the framework's core claim—that conscious systems balance V and D through γ-mediated dynamics—yields falsifiable predictions across multiple levels of analysis (autonomic, neural, phenomenological).

The phenomenological trade-off between experiential visibility and structural determinability, and its modulation by basal coherence, is schematically represented in Figure 3.

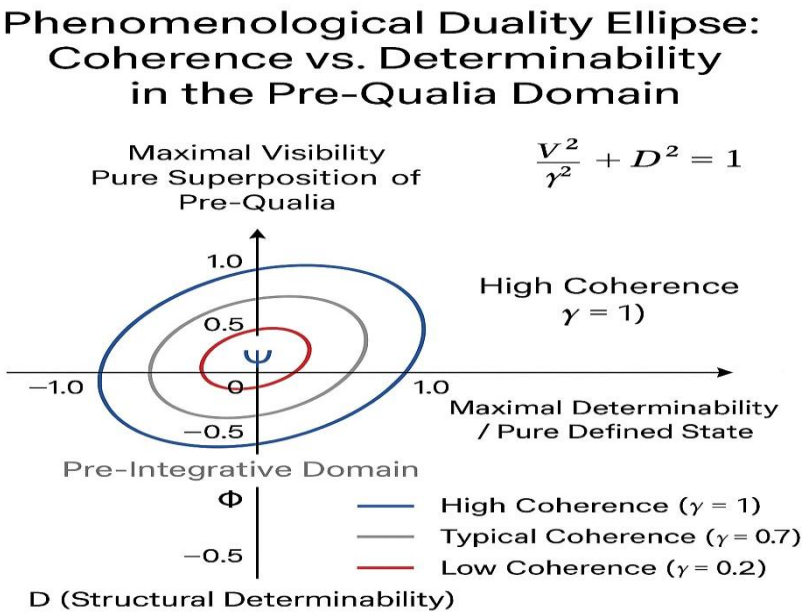

Figure 3: PLACEHOLDER. Phenomenological Duality Ellipse: Visibility–Determinability Trade-off

Schematic representation of a phenomenological constraint governing pre-integrative conscious states. The ellipse expresses a trade-off between experiential visibility (V), corresponding to the degree of subjective salience or accessibility, and structural determinability (D), corresponding to the degree of constraint, stability, or definability of system states. The relation $V^2/\gamma^2 + D^2 = 1$ is proposed as a phenomenological constraint rather than a physical law, where basal coherence (γ) modulates the accessible region of the visibility–determinability space.

Higher coherence (larger γ) allows greater simultaneous visibility and determinability, whereas reduced coherence progressively constrains accessible experiential states. The central region, denoted by Ψ, represents the pre-qualia domain: a pre-integrative informational state space in which experiential potential exists prior to full temporal, perceptual, and narrative integration. Ellipses shown for different γ values illustrate schematic coherence regimes and are intended as illustrative rather than empirical thresholds. Pre-qualia are defined operationally as informational configurations not yet integrated into reportable conscious experience—mathematical representations in informational Hilbert space rather than ontological entities. They become conscious content only when γ, n, and α_nk exceed threshold across appropriate temporal windows.

5.6. Minimal Theoretical Positioning (Non-Redundant Contribution)

EFA is positioned as architectural bridge across leading frameworks by adding explicit temporal windows and autonomic substrate constraint:

Relative to IIT: EFA does not replace integration (Φ-like structure) but specifies when and under what substrate conditions integration becomes conscious.

Relative to GNWT: EFA embeds ignition-like access dynamics within tiered constraints (γ foundation and ΔT_S narrative integration).

Relative to FEP: EFA treats free-energy minimization as optimization principle requiring implementation architecture with explicit temporal windows and coherence-dependent integration.

EFA therefore stands on clear empirical wager: if autonomic coherence (γ) and narrative-scale stability (ΔT_S proxies) do not add predictive and causal explanatory power beyond cortical integration metrics, then architecture is rejected or reduced.

5.7. Summary: EFA as High-Risk, High-Reward Empirical Claim

This section provides:

- Definitive falsification criteria for EFA's core necessities
- Three load-bearing protocols spanning causal manipulation, state transitions, and clinical prediction
- Clinical applications targeting specific architectural failures (γ, α_nk, ΔT_S)
- Phenomenological formalization via V/D ellipse
- Minimal positioning argument avoiding redundancy with existing theories

EFA is not validated by plausibility, but by whether γ × integration × temporal hierarchy improves explanation and prediction of conscious state transitions under perturbation and in clinic.

6. CONCLUSIONS AND OUTLOOK

The Emergent Flow Architecture (EFA) reconceptualizes consciousness as an architectural regime of constrained multi-timescale integration, rather than as a localized mechanism or purely cortical phenomenon. In its minimal operational form, conscious integration is captured by the dimensionless index:

$$\mathcal{C}(t) = \gamma(t) \cdot n(t) \cdot \alpha_{nk}(t) \cdot \Delta T_u \cdot \int(\Delta T_S)$$

where γ denotes basal coherence (organismic and autonomic stability), n functional recruitment, α_nk effective integration efficiency (empirically anchored by perturbational and effective-integration proxies such as PCI), and ΔT_u and ΔT_S encode compatibility with perceptual (approximately 25–40 ms) and narrative (approximately 0.3–3 s) integration windows.

The central architectural commitment of EFA is necessity-by-multiplicativity: if any component approaches zero, the value of $\mathcal{C}$ collapses, even when remaining components remain high. This property provides principled bottleneck logic for diagnosis, interpretation, and intervention.

6.1. Core Theoretical Contributions

EFA introduces three theoretically non-redundant constraints to contemporary theories of consciousness:

First, it treats basal coherence (γ) as foundational enabling condition, explaining why brainstem–autonomic integrity and interoceptive stability are necessary for sustained conscious integration. The identification of direct ACC-brainstem circuits enabling voluntary autonomic modulation [11] provides cutting-edge anatomical validation for recursive self-regulation—the system modifying its own substrate.

Second, it formalizes explicit temporal windows as architectural constraints rather than secondary correlates, distinguishing fast perceptual binding (ΔT_u) from slow narrative self-continuity (ΔT_S). The hierarchical coordination of these windows via cross-frequency coupling provides testable mechanistic predictions.

Third, it commits to quantitative falsifiability, specifying clear empirical scenarios under which framework must be rejected or reduced: >10% violations of necessity thresholds, equivalent performance of PCI-only vs full $\mathcal{C}$ models, or absence of γ-PCI causal coupling.

6.2. Clinical Translation and Precision Interventions

EFA enables precision diagnostics by identifying which parameter acts as bottleneck:

- Low γ → VNS, HRV biofeedback, somatic therapies
- Low α_nk → Neuromodulation (TMS, tDCS), cognitive training
- Disrupted ΔT_S → Psychotherapy, narrative restructuring

The phase-based trauma intervention protocol exemplifies this approach: Phase 1 (restore γ via bottom-up somatic interventions) → Phase 2 (integrate ΔT_S via narrative processing) → Phase 3 (expand V/D range for post-traumatic growth). This matches intervention level to hierarchical deficit, preventing re-traumatization while enabling meaningful integration.

The phenomenological duality ellipse ($V^2/\gamma^2 + D^2 = 1$) provides first rigorous mathematical treatment of subjective experience structure, enabling clinical applications targeting V/D balance (psychedelic-assisted therapy for ellipse widening; anxiety treatments for reducing pathological D; depression treatments for expanding collapsed V/D space).

6.3. Integration with Established Frameworks

EFA does not aim to supplant existing theories but

rather synthesizes their core insights within a unified temporal architecture:

With IIT: Φ quantifies what is integrated; EFA specifies when, how, and under what substrate conditions integration becomes conscious.

With GNWT: Ignition represents ΔT_u perceptual binding phase; EFA adds γ foundation and ΔT_S narrative extension.

With FEP: Free energy minimization operates within EFA's temporal windows; γ reflects system's capacity for effective minimization.

Accordingly, EFA makes high-risk empirical wager: if autonomic coherence (γ) and narrative-scale stability (ΔT_S proxies) do not add predictive and causal explanatory power beyond cortical integration metrics alone (such as PCI), across perturbation paradigms, anesthesia-induced transitions, and disorders-of-consciousness cohorts, then architecture is overparameterized and should be reduced to cortex-centered account. Conversely, if combined model reliably outperforms PCI-only baselines and demonstrates causal modulation (Δγ predicting ΔPCI), EFA gains empirical standing as mechanistic scaffold linking autonomic foundations to perceptual binding and narrative continuity.

6.4. Limitations and Open Questions

The limitations of the framework are explicitly acknowledged:

Parameter normalization and threshold calibration for $\mathcal{C}$ require validation across heterogeneous datasets, recording pipelines, and clinical populations. Current threshold values ($\mathcal{C}$ > 0.85 for consciousness, γ < 0.30 for collapse) need empirical refinement.

Evidence remains largely correlational. Causal inference depends on targeted intervention studies (Protocols 1-3), which represent next phase of empirical work.

Cross-species applicability uncertain: EFA developed for human consciousness; scaling to mammals, birds, cephalopods requires species-specific parameter calibration.

Developmental trajectory unspecified: When does fetal/neonatal $\mathcal{C}$ first exceed threshold? Ethical implications for prenatal consciousness remain unexplored.

Quantum-informational boundary (Σ(ΔT_q)) treated strictly as formal scaffolding, not operative neural mechanism. Extended discussion in Supplementary Material S3. Recent developments in quantum biology (Khan et al., 2024; Wiest, 2024) suggest potential quantum substrates in neuronal function through microtubule stabilization effects on anesthetic action; however, these findings remain preliminary and do not alter EFA's core empirical strategy, which focuses on neurobiologically accessible parameters [S78, S79].

6.5. Artificial Consciousness: Design Specifications

EFA translates consciousness science into engineering specifications for artificial systems:

Necessary architectural features:

- γ-analog: Self-monitoring stability module tracking internal coherence
- Hierarchical ΔT: Multiple temporal integration windows (fast binding + slow narrative)
- Dynamic α_nk: Reconfigurable network topology
- Phase coordination: Cross-layer coupling (PAC-equivalent mechanisms)
- V/D dynamics: Exploration-exploitation balance

Critically, artificial systems would need to exhibit spontaneous organization—emergent structures not explicitly programmed—mirroring how biological consciousness arises from trans-Turing sampling of the adjacent possible rather than pre-specified computation.

Detection criteria beyond behavioral mimicry:

- PCI-analog > threshold (perturbation-response complexity)
- Temporal hierarchy coherence (nested processing timescales)
- Spontaneous organization (emergent structures not programmed)
- Meta-cognitive capacity (system awareness of internal states)

Ethical implications: If artificial systems satisfy all criteria (Protocol 4), they warrant moral consideration—consciousness is architecture-specific, not substrate-specific.

6.6. The Path Forward: Empirical Validation

The next phase of research centers on load-bearing tests:

1. Causal modulation of γ: tVNS/breathing paradigms combined with TMS-EEG measuring downstream integration capacity (Protocol 1)
2. Parametric tracking across state transitions: Composite $\mathcal{C}$ index across anesthesia loss/recovery of responsiveness, testing hysteresis and threshold predictions (Protocol 2)
3. Prospective clinical prediction: Combined γ + PCI + DMN measures predicting outcomes in disorders-of-consciousness cohorts (Protocol 3)
4. Artificial system implementation: Building EFA-inspired AI architecture and testing consciousness detection criteria (Protocol 4)

6.7. Final Reflection

Consciousness science has long been trapped in substance ontology—seeking the thing that is consciousness. EFA reframes consciousness as architectural process:

- Not a thing but a flow
- Not localized but distributed across hierarchical scales
- Not static but dynamically maintained far from equilibrium
- Not mysterious but mechanistically specifiable

The hard problem does not vanish but transforms: From "why does matter produce experience" to "what organizational principles enable informational systems to become self-aware." The former may be unanswerable; the latter is engineering challenge. The brain's capacity to sample the adjacent possible—generating novel configurations not pre-specified algorithmically—positions consciousness as a trans-Turing process that cannot be fully captured by finite state machines, yet remains naturalistic and mechanistically explicable through hierarchical temporal integration.

Consciousness emerges not from quantum magic, nor from computational complexity alone, but from specific temporal-thermodynamic-informational architecture. We can now:

- Measure this architecture ($\mathcal{C}$ and component parameters)
- Manipulate it (interventions targeting γ, α_nk, ΔT_S at appropriate hierarchical levels)
- Predict its presence (falsification protocols with statistical thresholds)
- Engineer it (artificial systems implementing EFA design specifications)

The future of consciousness science lies not in choosing between competing paradigms but in synthesizing their insights within unified temporal architecture—one respecting both rigor of physics and richness of phenomenology.

The Emergent Flow Architecture stands or falls on whether the product of basal coherence, effective integration, and hierarchical temporal organization yields superior causal explanation and predictive power under perturbation and in the clinic.

The next chapter is empirical validation.

DATA AVAILABILITY STATEMENT

This manuscript is a theoretical work and does not report new empirical data. All cited studies are publicly available through their respective publications.

CONFLICT OF INTEREST

The author declares that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

AUTHOR CONTRIBUTIONS

ALP conceived, designed, and wrote the manuscript.

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ABBREVIATIONS

ACC Anterior Cingulate Cortex
ANS Autonomic Nervous System
CBT Cognitive-Behavioral Therapy
DMN Default Mode Network
EEG Electroencephalography
EMDR Eye Movement Desensitization and Reprocessing fMRI Functional Magnetic Resonance Imaging
HRV Heart Rate Variability
IIT Integrated Information Theory
GNWT Global Neuronal Workspace Theory
FEP Free Energy Principle
LC Locus Coeruleus
MCS Minimally Conscious State mPFC Medial Prefrontal Cortex
NTS Nucleus Tractus Solitarius
PAC Phase-Amplitude Coupling
PCC Posterior Cingulate Cortex
PCI Perturbational Complexity Index PTSD Post-Traumatic Stress Disorder
PV Parvalbumin
RAS Reticular Activating System
SRAA Ascending Reticular Activating System tDCS Transcranial Direct Current Stimulation
TMS Transcranial Magnetic Stimulation TRN Thalamic Reticular Nucleus tVNS Transcutaneous Vagus Nerve Stimulation
VNS Vagus Nerve Stimulation
VS Vegetative State
VTA Ventral Tegmental Area

REFERENCES

1. Brillouin, L. Science and Information Theory; Academic Press: New York, NY, USA, 1956.
2. Jaynes, E.T. Information theory and statistical mechanics. Phys. Rev. 1957, 106, 620–630. https://doi.org/10.1103/PhysRev.106.620
3. Bérut, A.; Arakelyan, A.; Petrosyan, A.; Ciliberto, S.; Dillenschneider, R.; Lutz, E. Experimental verification of Landauer's principle linking information and thermodynamics. Nature 2012, 483, 187–189. https://doi.org/10.1038/nature10872
4. Hong, J.; Lambson, B.; Dhuey, S.; Bokor, J. Experimental test of Landauer's principle in single-bit operations on nanomagnetic memory bits. Sci. Adv. 2016, 2, e1501492.
https://doi.org/10.1126/sciadv.1501492
5. Cowan, N. The magical number 4 in short-term memory: A reconsideration of mental storage capacity. Behav. Brain Sci. 2001, 24, 87–114. https://doi.org/10.1017/S0140525X01003922
6. Tegmark, M. Importance of quantum decoherence in brain processes. Phys. Rev. E 2000, 61, 4194–4206. https://doi.org/10.1103/PhysRevE.61.4194
7. Kukushkin, N.V.; Kováč, J.; Carew, T.J. Memory outside the brain: Molecular mechanisms of memory in non-neural cells. Cell 2024, 187, 4023–4039. https://doi.org/10.1016/j.cell.2024.06.011
8. Levin, M. Bioelectric signaling: Reprogrammable circuits underlying embryogenesis, regeneration, and cancer. Cell 2021, 184, 1971–1989. https://doi.org/10.1016/j.cell.2021.02.034
9. Thayer, J.F.; Lane, R.D. Claude Bernard and the heart–brain connection: Further elaboration of a model of neurovisceral integration. Neurosci. Biobehav. Rev. 2009, 33, 81–88.
https://doi.org/10.1016/j.neubiorev.2008.08.004
10. Critchley, H.D.; Wiens, S.; Rotshtein, P.; Öhman, A.; Dolan, R.J. Neural systems supporting interoceptive awareness. Nat. Neurosci. 2004, 7, 189–195. https://doi.org/10.1038/nn1176
11. Jhang, J.; Lee, H.; Kang, M.S.; Lee, H.-S.; Park, H.; Han, J.-H. Anterior cingulate cortex and its input to the basal ganglia control innate freezing and acquired fear responses. Cell Rep. 2018, 24, 2700–2710. https://doi.org/10.1016/j.celrep.2018.08.003
12. Gray, C.M.; Singer, W. Stimulus-specific neuronal oscillations in orientation columns of cat visual cortex. Proc. Natl. Acad. Sci. USA 1989, 86, 1698–1702. https://doi.org/10.1073/pnas.86.5.1698
13. Fries, P. Rhythms for cognition: Communication through coherence. Neuron 2015, 88, 220–235. https://doi.org/10.1016/j.neuron.2015.09.034
14. Lewis, L.D.; Weiner, V.S.; Mukamel, E.A.; Donoghue, J.A.; Eskandar, E.N.; Madsen, J.R.; Anderson, W.S.; Hochberg, L.R.; Cash, S.S.; Brown, E.N.; Purdon, P.L. Rapid fragmentation of neuronal networks at the onset of propofol-induced unconsciousness. Proc. Natl. Acad. Sci. USA 2012, 109, E3377–E3386. https://doi.org/10.1073/pnas.1210907109
15. Zacks, J.M.; Speer, N.K.; Swallow, K.M.; Braver, T.S.; Reynolds, J.R. Event perception: A mind-brain perspective. Psychol. Bull. 2007, 133, 273–293. https://doi.org/10.1037/0033-2909.133.2.273
16. Prigogine, I. Time, structure, and fluctuations. Science 1978, 201, 777–785. https://doi.org/10.1126/science.201.4358.777
17. Casali, A.G.; Gosseries, O.; Rosanova, M.; Boly, M.; Sarasso, S.; Casali, K.R.; Casarotto, S.; Bruno, M.-A.; Laureys, S.; Tononi, G.; Massimini, M. A theoretically based index of consciousness independent of sensory processing and behavior. Sci. Transl. Med. 2013, 5, 198ra105.
https://doi.org/10.1126/scitranslmed.3006294
18. Badran, B.W.; Mithoefer, O.J.; Summer, C.E.; LaBate, N.T.; Glusman, C.E.; Badran, A.W.; DeVries, W.H.; Summers, P.M.; Austelle, C.W.; McTeague, L.M.; et al. Short trains of transcutaneous auricular vagus nerve stimulation (taVNS) have parameter-specific effects on heart rate. Brain Stimul. 2018, 11, 699–708. https://doi.org/10.1016/j.brs.2018.04.004
19. Uhlhaas, P.J.; Singer, W. Abnormal neural oscillations and synchrony in schizophrenia. Nat. Rev. Neurosci. 2010, 11, 100–113. https://doi.org/10.1038/nrn2774
20. Levine, B.; Svoboda, E.; Hay, J.F.; Winocur, G.; Moscovitch, M. Aging and autobiographical memory: Dissociating episodic from semantic retrieval. Psychol. Aging 2002, 17, 677–689. https://doi.org/10.1037/0882-7974.17.4.677
21. Lanius, R.A.; Vermetten, E.; Loewenstein, R.J.; Brand, B.; Schmahl, C.; Bremner, J.D.; Spiegel, D.

Emotion modulation in PTSD: Clinical and neurobiological evidence for a dissociative subtype. Am. J. Psychiatry 2010, 167, 640–647. https://doi.org/10.1176/appi.ajp.2009.09081168
22. Kirihara, K.; Rissling, A.J.; Swerdlow, N.R.; Braff, D.L.; Light, G.A. Hierarchical organization of gamma and theta oscillatory dynamics in schizophrenia. Biol. Psychiatry 2012, 71, 873–880. https://doi.org/10.1016/j.biopsych.2012.01.016
23. Rosanova, M.; Gosseries, O.; Casarotto, S.; Boly, M.; Casali, A.G.; Bruno, M.-A.; Mariotti, M.; Boveroux, P.; Tononi, G.; Laureys, S.; Massimini, M. Recovery of cortical effective connectivity and recovery of consciousness in vegetative patients. Brain 2012, 135, 1308–1320.
https://doi.org/10.1093/brain/aws050
24. Park, G.; Thayer, J.F. From the heart to the mind: Cardiac vagal tone modulates top-down and bottom-up visual perception and attention to emotional stimuli. Front. Psychol. 2014, 5, 278. https://doi.org/10.3389/fpsyg.2014.00278
25. Vanhaudenhuyse, A.; Noirhomme, Q.; Tshibanda, L.J.-F.; Bruno, M.-A.; Boveroux, P.; Schnakers, C.; Soddu, A.; Perlbarg, V.; Ledoux, D.; Brichant, J.-F.; et al. Default network connectivity reflects the level of consciousness in non-communicative brain-damaged patients. Brain 2010, 133, 161–171. https://doi.org/10.1093/brain/awp313
26. Demertzi, A.; Tagliazucchi, E.; Dehaene, S.; Deco, G.; Barttfeld, P.; Raimondo, F.; Martial, C.; Fernández-Espejo, D.; Rohaut, B.; Voss, H.U.; et al. Human consciousness is supported by dynamic complex patterns of brain signal coordination. Sci. Adv. 2019, 5, eaat7603.
https://doi.org/10.1126/sciadv.aat7603
27. Csikszentmihalyi, M. Flow: The Psychology of Optimal Experience; Harper & Row: New York, NY, USA, 1990.
28. Tononi, G.; Boly, M.; Massimini, M.; Koch, C. Integrated information theory: From consciousness to its physical substrate. Nat. Rev. Neurosci. 2016, 17, 450–461. https://doi.org/10.1038/nrn.2016.44
29. Dehaene, S.; Changeux, J.-P. Experimental and theoretical approaches to conscious processing. Neuron 2011, 70, 200–227. https://doi.org/10.1016/j.neuron.2011.03.018
30. Friston, K. The free-energy principle: A unified brain theory? Nat. Rev. Neurosci. 2010, 11, 127–138. https://doi.org/10.1038/nrn2787
31. Koch, C.; Massimini, M.; Boly, M.; Tononi, G. Neural correlates of consciousness: Progress and problems. Nat. Rev. Neurosci. 2016, 17, 307–321. https://doi.org/10.1038/nrn.2016.22
32. Oizumi, M.; Albantakis, L.; Tononi, G. From the phenomenology to the mechanisms of consciousness: Integrated Information Theory 3.0. PLoS Comput. Biol. 2014, 10, e1003588. https://doi.org/10.1371/journal.pcbi.1003588
33. Mashour, G.A.; Roelfsema, P.; Changeux, J.-P.; Dehaene, S. Conscious processing and the global neuronal workspace hypothesis. Neuron 2020, 105, 776–798.
https://doi.org/10.1016/j.neuron.2020.01.026
34. Friston, K.; Kilner, J.; Harrison, L. A free energy principle for the brain. J. Physiol. Paris 2006, 100, 70–87. https://doi.org/10.1016/j.jphysparis.2006.10.001
35. Seth, A.K.; Friston, K.J. Active interoceptive inference and the emotional brain. Philos. Trans. R. Soc. B Biol. Sci. 2016, 371, 20160007. https://doi.org/10.1098/rstb.2016.0007
36. Varela, F.J. Neurophenomenology: A methodological remedy for the hard problem. J. Conscious. Stud. 1996, 3, 330–349.
37. Chalmers, D.J. Facing up to the problem of consciousness. J. Conscious. Stud. 1995, 2, 200–219.
38. Edelman, G.M.; Gally, J.A.; Baars, B.J. Biology of consciousness. Front. Psychol. 2011, 2, 4. https://doi.org/10.3389/fpsyg.2011.00004
39. Baars, B.J. Global workspace theory of consciousness: Toward a cognitive neuroscience of human experience. Prog. Brain Res. 2005, 150, 45–53. https://doi.org/10.1016/S0079-6123(05)50004-9
40. Laureys, S.; Schiff, N.D. Coma and consciousness: Paradigms (re)framed by neuroimaging. Neuroimage 2012, 61, 478–491. https://doi.org/10.1016/j.neuroimage.2011.12.041
41. Giacino, J.T.; Fins, J.J.; Laureys, S.; Schiff, N.D. Disorders of consciousness after acquired brain injury: The state of the science. Nat. Rev. Neurol. 2014, 10, 99–114. https://doi.org/10.1038/nrneurol.2013.279

42. Engel, A.K.; Fries, P.; Singer, W. Dynamic predictions: Oscillations and synchrony in top-down processing. Nat. Rev. Neurosci. 2001, 2, 704–716. https://doi.org/10.1038/35094565
43. Buzsáki, G.; Draguhn, A. Neuronal oscillations in cortical networks. Science 2004, 304, 1926–1929. https://doi.org/10.1126/science.1099745
44. Canolty, R.T.; Knight, R.T. The functional role of cross-frequency coupling. Trends Cogn. Sci. 2010, 14, 506–515. https://doi.org/10.1016/j.tics.2010.09.001
45. Tort, A.B.L.; Komorowski, R.W.; Manns, J.R.; Kopell, N.J.; Eichenbaum, H. Theta–gamma coupling increases during the learning of item–context associations. Proc. Natl. Acad. Sci. USA 2009, 106, 20942–20947. https://doi.org/10.1073/pnas.0911331106
46. Porges, S.W. The polyvagal theory: Phylogenetic substrates of a social nervous system. Int. J. Psychophysiol. 2001, 42, 123–146. https://doi.org/10.1016/S0167-8760(01)00162-3
47. Shaffer, F.; Ginsberg, J.P. An overview of heart rate variability metrics and norms. Front. Public Health 2017, 5, 258. https://doi.org/10.3389/fpubh.2017.00258
48. Appelhans, B.M.; Luecken, L.J. Heart rate variability as an index of regulated emotional responding. Rev. Gen. Psychol. 2006, 10, 229–240. https://doi.org/10.1037/1089-2680.10.3.229
49. Buckner, R.L.; Andrews-Hanna, J.R.; Schacter, D.L. The brain's default network: Anatomy, function, and relevance to disease. Ann. N. Y. Acad. Sci. 2008, 1124, 1–38. https://doi.org/10.1196/annals.1440.011
50. Raichle, M.E. The brain's default mode network. Annu. Rev. Neurosci. 2015, 38, 433–447. https://doi.org/10.1146/annurev-neuro-071013-014030
51. Spreng, R.N.; Mar, R.A.; Kim, A.S.N. The common neural basis of autobiographical memory, prospection, navigation, theory of mind, and the default mode: A quantitative meta-analysis. J. Cogn. Neurosci. 2009, 21, 489–510. https://doi.org/10.1162/jocn.2008.21029
52. Schacter, D.L.; Addis, D.R.; Buckner, R.L. Remembering the past to imagine the future: The prospective brain. Nat. Rev. Neurosci. 2007, 8, 657–661. https://doi.org/10.1038/nrn2213
53. Hasselmo, M.E.; Stern, C.E. Theta rhythm and the encoding and retrieval of space and time. Neuroimage 2014, 85, 656–666. https://doi.org/10.1016/j.neuroimage.2013.06.022
54. Buzsáki, G.; Moser, E.I. Memory, navigation and theta rhythm in the hippocampal-entorhinal system. Nat. Neurosci. 2013, 16, 130–138. https://doi.org/10.1038/nn.3304
55. Steriade, M.; Timofeev, I.; Grenier, F. Natural waking and sleep states: A view from inside neocortical neurons. J. Neurophysiol. 2001, 85, 1969–1985. https://doi.org/10.1152/jn.2001.85.5.1969
56. Brown, E.N.; Lydic, R.; Schiff, N.D. General anesthesia, sleep, and coma. N. Engl. J. Med. 2010, 363, 2638–2650. https://doi.org/10.1056/NEJMra0808281
57. Mashour, G.A.; Hudetz, A.G. Neural correlates of unconsciousness in large-scale brain networks. Trends Neurosci. 2018, 41, 150–160. https://doi.org/10.1016/j.tins.2018.01.003
58. Alkire, M.T.; Hudetz, A.G.; Tononi, G. Consciousness and anesthesia. Science 2008, 322, 876–880. https://doi.org/10.1126/science.1149213
59. Carhart-Harris, R.L.; Friston, K.J. REBUS and the anarchic brain: Toward a unified model of the brain action of psychedelics. Pharmacol. Rev. 2019, 71, 316–344. https://doi.org/10.1124/pr.118.017160
60. Carhart-Harris, R.L.; Leech, R.; Hellyer, P.J.; Shanahan, M.; Feilding, A.; Tagliazucchi, E.; Chialvo, D.R.; Nutt, D. The entropic brain: A theory of conscious states informed by neuroimaging research with psychedelic drugs. Front. Hum. Neurosci. 2014, 8, 20. https://doi.org/10.3389/fnhum.2014.00020
61. Sitt, J.D.; King, J.-R.; El Karoui, I.; Rohaut, B.; Faugeras, F.; Gramfort, A.; Cohen, L.; Sigman, M.; Dehaene, S.; Naccache, L. Large scale screening of neural signatures of consciousness in patients in a vegetative or minimally conscious state. Brain 2014, 137, 2258–2270. https://doi.org/10.1093/brain/awu141
62. Engemann, D.A.; Raimondo, F.; King, J.-R.; Rohaut, B.; Louppe, G.; Faugeras, F.; Annen, J.; Cassol, H.; Gosseries, O.; Fernandez-Slezak, D.; et al. Robust EEG-based cross-site and cross-protocol classification of states of consciousness. Brain 2018, 141, 3179–3192. https://doi.org/10.1093/brain/awy251
63. Schartner, M.M.; Carhart-Harris, R.L.; Barrett, A.B.; Seth, A.K.; Muthukumaraswamy, S.D. Increased spontaneous MEG signal diversity for psychoactive doses of ketamine, LSD and psilocybin. Sci. Rep. 2017, 7, 46421. https://doi.org/10.1038/srep46421
64. Lempel, A.; Ziv, J. On the complexity of finite sequences. IEEE Trans. Inf. Theory 1976, 22, 75–81.

https://doi.org/10.1109/TIT.1976.1055501
65. Sarasso, S.; Boly, M.; Napolitani, M.; Gosseries, O.; Charland-Verville, V.; Casarotto, S.; Rosanova, M.; Casali, A.G.; Brichant, J.-F.; Boveroux, P.; et al. Consciousness and complexity during unresponsiveness induced by propofol, xenon, and ketamine. Curr. Biol. 2015, 25, 3099–3105. https://doi.org/10.1016/j.cub.2015.10.014
66. Comolatti, R.; Pigorini, A.; Casarotto, S.; Fecchio, M.; Faria, G.; Sarasso, S.; Rosanova, M.; Gosseries, O.; Boly, M.; Bodart, O.; et al. A fast and general method to empirically estimate the complexity of brain responses to transcranial and intracranial stimulations. Brain Stimul. 2019, 12, 1280–1289. https://doi.org/10.1016/j.brs.2019.05.013
67. Thayer, J.F.; Åhs, F.; Fredrikson, M.; Sollers, J.J.; Wager, T.D. A meta-analysis of heart rate variability and neuroimaging studies: Implications for heart rate variability as a marker of stress and health. Neurosci. Biobehav. Rev. 2012, 36, 747–756. https://doi.org/10.1016/j.neubiorev.2011.11.009
68. Schiff, N.D. Recovery of consciousness after brain injury: A mesocircuit hypothesis. Trends Neurosci. 2010, 33, 1–9. https://doi.org/10.1016/j.tins.2009.11.002