Sleep, Central Pattern Generators, and the Mitochondrial Imperative: A Unified Framework

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Abstract

We propose a unifying model in which sleep paralysis and the sleep-wake transition are governed by the partial or failed reactivation of evolutionarily conserved central pattern generators (CPGs), derived from primitive swimming motions, coupled with mitochondrial recovery dynamics. Within the Relativistic Scalar Vector Plenum (RSVP) framework, sleep is interpreted as a desynchronization of binocular and proprioceptive pulses, with waking as the reentrainment of high-order locomotor rhythms built upon a foundational aquatic waveform. A mathematical model captures this process as a transition across symmetry-broken projections of a generalized scalar field (Φ), with coupled vector dynamics ($\vec{v}$) and entropy fields ($\mathcal{S}$). The model integrates mitochondrial ATP recovery and oxidative stress, providing predictive insight into sleep paralysis, jaw clenching, eye saccades, and the bootstrapping of complex motor coordination during waking.

1 Introduction

Sleep paralysis, a phenomenon historically shrouded in mystery, offers a unique window into the layered architecture of motor control and consciousness. We propose that sleep paralysis is not a pathological anomaly but a snapshot of interrupted re-synchronization of ancestral motor schemas embedded in the nervous system. Our core hypothesis posits that running, brachiating, and flying are evolutionary variants of swimming, and waking involves bootloading a distributed, swinging, swimming motor program via intermodular CPG entrainment. This process is coupled to a mitochondrial imperative, where sleep facilitates metabolic recovery through systemic stillness. Recent findings (Nature, 2025) underscore the role of mitochondrial dynamics in sleep, motivating a unified framework that integrates motor coordination, metabolic repair, and consciousness transitions.

2 Evolutionary and Neurophysiological Foundations

Primitive vertebrate movement, as observed in lampreys and fish, is governed by sinusoidal spinal CPGs. These oscillatory circuits are conserved across mammals and birds, adapted for walking, flying, and brachiating. During REM sleep, these patterns are reactivated without motor output due to glycinergic and GABAergic inhibition. The vestibulo-ocular system, jaw tension, neck flexion, and proprioceptive pulses contribute to coordinating these entrained fields, with disruptions manifesting as sleep paralysis or delayed waking.

3 RSVP Field Framing

Within the RSVP framework, motor schemas are modeled as vector and scalar field structures defined over a body-space manifold. We define:

- $\Phi(x,t)$: Scalar CPG phase field representing primitive oscillation.
- $\vec{v}(x,t)$: Motor vector flow field governing muscle activation and limb torque.
- $\mathcal{S}(x,t)$: Entropy/damping field capturing inhibition or loss of coherence.

Symmetry-breaking projections of Φ yield specific locomotor modes (e.g., swim $\rightarrow$ run, fly, swing). Sleep paralysis is characterized by high internal coherence in Φ without active coherence in $\vec{v}$, reflecting motor suppression.

4 Sleep-Wake Transitions as Field Recoherence

Waking requires entraining the vector field $\vec{v}$ with the re-emergent scalar wave Φ. A reflex set $\mathcal{R}_i$ (jaw clench, eye saccade, vestibular flicks, spinal twist) serves as "bootloaders," coupling into the governing partial differential equation (PDE):

$$\partial_t \Phi + \vec{v} \cdot \nabla \Phi = D\nabla^2 \Phi + \sum_i \kappa_i \mathcal{R}_i(t, x) - \gamma \mathcal{S}(x, t)$$

Where:

- D: Diffusion coefficient.
- κ_i: Reflex-specific gain.
- $\mathcal{R}_i(t, x)$: Temporally pulsed reactivation terms.

5 Jaw Clenching and Eye Saccades as Anchors

Jaw clenching anchors midline torque, reestablishing equilibrium in $\vec{v}$. Eye saccades, tightly coupled to locomotor phases, maintain binocular phase synchrony; disruptions trigger desynchronization. The vestibular system provides rotational inertia and accelerative cues, aiding vector realignment.

6 The Mitochondrial Recovery Imperative

Sleep facilitates mitochondrial recovery through mitophagy, ATP replenishment, and reduction of reactive oxygen species (ROS). Extended wakefulness leads to ATP surplus and electron leak, necessitating cyclic stillness to restore oxidative respiration efficiency. This metabolic imperative drives the desynchronization of CPGs, enabling systemic stillness as a reparative phase.

7 Unifying Motor and Metabolic Theories of Sleep

Jaw clenching, eye saccades, and CPG rhythms act as "heartbeats" that reentrain motor and metabolic systems during waking. Sleep serves as both an energetic and informational reconfiguration, with implications for movement disorders, sleep paralysis, and circadian fatigue. The RSVP framework unifies these processes by modeling sleep as a coordinated system collapse and waking as a re-synchronized ascent.

8 Prediction and Experimental Proposal

We predict that fMRI and electromyography (EMG) will reveal differential activation in brainstem, jaw, and eye control circuits during transitions from sleep paralysis to wakefulness. Electrical stimulation of jaw muscles or induced micro-saccades may accelerate waking. Disorders like narcolepsy or parasomnias should exhibit aberrations in Φ-$\vec{v}$ coupling, testable via metabolic imaging or EEG.

9 Conclusion

Sleep paralysis is not a glitch but a snapshot of an interrupted orchestra, where the scalar conductor (Φ) has begun, but the vector players ($\vec{v}$) remain silent. The RSVP framework offers a topological and dynamic interpretation, bridging evolutionary biomechanics, mitochondrial dynamics, and consciousness studies. This model suggests novel physiological tests and therapeutic interventions for sleep-related disorders.

A Mathematical Formalism for RSVP-Coupled Sleep Dynamics

This appendix formalizes the model of sleep-wake transitions and sleep paralysis as emergent phenomena from coupled CPGs, metabolic fatigue, and mitochondrial repair imperatives.

A.1 Definitions and Fields

We define the following fields over a body-space manifold:

- $\mathcal{C}_i(t)$: Phase of the i-th CPG oscillator.
- $\Phi(x,t)$: Scalar metabolic fatigue potential.
- $\vec{v}(x,t)$: Effective motility vector field.
- $\mathcal{S}(x,t)$: Entropy or disorder in CPG coordination.
- $I(x,t)$: Neural inhibition field (REM-specific motor suppression).
- $\mathcal{R}_i(x,t)$: Reflex bootloader impulse (e.g., jaw clench, eye saccade).

A.2 Phase Dynamics of Coupled CPGs

Each CPG chain is modeled as a noisy phase oscillator with coupling:

$$\frac{d\mathcal{C}_i}{dt} = -\gamma \sum_{j \neq i} \sin(\mathcal{C}_i - \mathcal{C}_j) - \frac{\partial V}{\partial \mathcal{C}_i} + \eta_i(t)$$

Where:

- γ: Coupling strength (e.g., spinal coherence).
- V: Attractor potential (see Section A.4).
- $\eta_i(t)$: Gaussian white noise modeling sensory perturbations.

A.3 Metabolic Field Equation with Biophysical Terms

The metabolic fatigue field incorporates ATP depletion, ROS buildup, and recovery:

$$\frac{\partial \Phi}{\partial t} = D\nabla^2\Phi - \alpha|\nabla \vec{v}|^2 + \beta\mathcal{S} - \mu\rho\Phi^2 + \nu \cdot \mathrm{ATP}_{\mathrm{rec}}(t)$$

Where:

- $\rho\Phi^2$: ROS production scaling with energetic load.
- $\mathrm{ATP}_{\mathrm{rec}}(t) = \frac{\nu_{\max}}{1+e^{-k(t-t_{\mathrm{rec}})}}$: Logistic recovery profile.
- μ, ν, α, β: Coupling constants.
- $\mathcal{S}$: Phase disorder term.

A.4 REM/NREM/Wake Attractor Landscape

An energy potential defines stable states for REM, NREM, and wakefulness:

$$V(\mathcal{C}, \Phi) = \sum_i \frac{k}{2}(\mathcal{C}_i - \mathcal{C}_i^{\mathrm{eq}})^2 + a(\Phi - \Phi_{\mathrm{REM}})^2(\Phi - \Phi_{\mathrm{NREM}})^2(\Phi - \Phi_{\mathrm{wake}})^2$$

Where:

- $\mathcal{C}_i^{\mathrm{eq}}$: Equilibrium phase per state (e.g., desynchronized for REM, synchronized for wake).
- $\Phi_{\mathrm{REM}}, \Phi_{\mathrm{NREM}}, \Phi_{\mathrm{wake}}$: Metabolic potentials for each state.
- k, a: Coefficients shaping landscape depth.

This tri-stable polynomial admits three attractors with noise-mediated transitions.

A.5 Vector Field and Inhibitory Suppression

Motility is modulated by fatigue and REM-specific inhibition:

$$\vec{v}(x,t) = \chi \nabla \Phi + \lambda \nabla \times \vec{A} - \kappa I(x,t) \cdot \vec{v}$$

Where:

- $\vec{A}$: Latent field coupling oscillators across joints.
- $I(x,t) = I_0 e^{-\gamma_{\text{inh}} t} \cdot \Theta(\Phi - \Phi_{\text{th}})$: Inhibition active below fatigue threshold.
- χ, λ, κ: Coupling constants.

A.6 Reflex Bootloaders (Sigmoid-Gated)

Reflexes are modeled as delta pulses gated by sigmoid switches:

$$\mathcal{R}_i(x,t) = A_i \cdot \frac{1}{1 + e^{-(\Phi - \Phi_{\text{th}})/\epsilon}} \cdot e^{-\frac{(t-t_i)^2}{2\sigma^2}} \delta(x - x_i)$$

Each pulse restores local coherence to Φ or boosts $\vec{v}$.

A.7 Sleep Trigger Functional

Systemic collapse into sleep occurs when:

$$\mathcal{S}(t) = \int_\Omega \left[\sum_i |\dot{\mathcal{C}}_i|^2 + |\nabla \Phi|^2 + \lambda \mathcal{S}^2 \cdot \frac{1}{1 + e^{-(\mathcal{S} - \mathcal{S}_{\text{th}})/\epsilon}} \right] dx$$

If $\mathcal{S}(t) > \mathcal{S}_{\text{crit}}$, reflexes are inhibited, and motility freezes.

B Simulation and RSVP-TARTAN Embedding (Preview)

In the RSVP framework, the system can be visualized as:

- Phase Portraits: Attractor basins in $\mathcal{C}$-Φ space.
- Tissue Maps: Metabolic fields (Φ), inhibition overlays (I), and reflex vectors ($\mathcal{R}_i$) on body-surface domains.
- Sleep Transition Visualization: Heat maps and pulse animations of $\mathcal{R}_i$ to simulate how jaw clench, eye saccade, or spinal recoil shifts system state.

Numerical simulations (e.g., finite difference methods) can discretize the PDEs for implementation in RSVP-TARTAN, enabling dynamic visualization of sleep-wake transitions.