A Unified Probabilistic Framework for Non-Stationary Heart Rate Variability Analysis

Modeling the Dynamic Evolution of Autonomic Control

Matías Castillo-Aguilar

Introduction

The rhythm of the human heart, far from being a simple, constant beat, is a complex and dynamic signal reflecting the continuous interplay between an organism and its internal and external environments. The precise timing between successive heartbeats, measured as the R-R interval (RRi), is a primary non-invasive proxy for autonomic nervous system (ANS) activity. The analysis of variations in this interval, a discipline known as heart rate variability (HRV), has become a cornerstone for assessing cardiovascular health, stress responses, and overall physiological state. This variability arises from the coordinated actions of multiple regulatory systems, including the sympathetic and parasympathetic branches of the ANS, baroreflexes, and thermoregulatory mechanisms.

Despite its utility, a significant limitation of traditional HRV analysis is its reliance on time-domain or frequency-domain metrics derived from static, short-term data windows. While such methods may offer insights into a snapshot of a physiological state under controlled conditions, they are fundamentally ill-suited to capture the dynamic, non-stationary nature of RRi signals during physiological transitions or stress, such as exercise, cognitive tasks, or pharmacologic interventions. The implicit assumption of stationarity in these fixed-window approaches can obscure subtle, yet physiologically critical, shifts in heart rate and its underlying variability. Furthermore, these conventional analyses often fail to establish a direct mechanistic link between observed changes in HRV and the underlying physiological processes responsible for them. For instance, a generalized decrease in HRV may be interpreted as a withdrawal of parasympathetic tone but could also stem from a complex shift in the balance of different frequency components of autonomic modulation. Disentangling these potential causes necessitates a more sophisticated, unified modeling paradigm.

Attempts to address these limitations have been made using a variety of modeling approaches, but these have often fallen short. For instance, state-space models have shown promise in tracking the evolution of cardiac dynamics, but their complexity can make parameter estimation difficult and their physiolog-

ical interpretability limited. While time-frequency analysis methods, such as wavelets, can successfully visualize the time-varying nature of spectral content, they typically do not provide a generative model for testing specific physiological hypotheses. Moreover, they often lack a principled way to separate structured physiological variability from unstructured noise. Other efforts have focused on non-linear dynamics and fractal analysis, but these models are often phenomenological, describing the properties of the signal without offering a clear, mechanistic link to the underlying physiology. Consequently, no existing model provides a unified, probabilistic framework that can simultaneously capture and mechanistically interpret changes in both the mean RRi and its multi-component variability.

To advance the field beyond these constraints, a new generation of statistical models is clearly needed. These models must transcend simple descriptive statistics to provide a unified framework that simultaneously captures the time-varying nature of both the mean heart rate and the dynamic evolution of its multi-timescale variability. Such a framework should explicitly address the non-stationarity inherent to physiological signals, obviating the need for arbitrary, fixed-length analysis windows. It must be capable of decomposing the signal into its distinct components, separating the gross, underlying trends in heart rate from the structured, oscillatory variability that represents physiological regulation. Crucially, the model's parameters should have a direct, interpretable link to specific physiological processes, such as autonomic tone, sympathetic-parasympathetic balance, and the dynamics of recovery. As a probabilistic framework, it should also provide a principled means of quantifying the uncertainty associated with all parameter estimates, moving beyond a reliance on point estimates to offer a more complete picture of the physiological state.

This paper presents a novel probabilistic framework for analyzing non-stationary RRi signals. Our approach directly confronts these challenges by formulating the RRi signal as a continuous stochastic process. This model decomposes the signal into a deterministic mean trajectory and a time-varying total standard deviation, both of which are constructed mechanistically. The model's key innovations include a mechanistic model for the mean RRi that uses a flexible double-logistic function to allow for the direct estimation of physiologically salient parameters, such as the magnitude of heart rate change and the timing of response and recovery. A core innovation is the explicit decomposition of the total signal variance into components representing structured, oscillatory variability and unstructured, residual noise, which is critical for understanding the sources of change in HRV. We generalize the traditional SDNN metric into a time-varying trajectory, which is also modeled using a logistic function analogous to that for the mean RRi, thereby capturing the dynamic suppression and recovery of total variability in a parsimonious manner. A crucial mathematical inversion within the framework ensures that the amplitude of the synthesized structured signal exactly matches the target SDNN trajectory at every time point, effectively decoupling the total magnitude of variability from its spectral composition and enabling more granular analysis. Finally, the model captures

the dynamic allocation of power across different physiological frequency bands by modeling their proportions as a function of a smooth master controller, which allows for a detailed analysis of shifts in spectral balance during physiological transitions.

The primary objective of this study is to introduce and validate this novel probabilistic model for the analysis of non-stationary RRi signals. We aim to demonstrate that this framework provides a more robust, informative, and physiologically interpretable analysis of heart rate dynamics than traditional methods. We hypothesize that the model will accurately and robustly capture the complex, time-varying dynamics of both the mean RRi and its variability during transient physiological perturbations. We further propose that the model's parameters will yield enhanced mechanistic nuance into autonomic control and cardiovascular regulation, thereby serving as a superior tool for both clinical research and basic physiological investigation. Through this work, we seek to establish a new standard for the analysis of time-varying physiological signals.

Methods

Model Formulation

We present a comprehensive generative model for the R–R interval (RRi) signal, observed at a discrete set of time points $\{t_i\}_{i=1}^{N}$. The model's architecture is designed to deconstruct the signal into its constituent physiological components, providing a mechanistic account of the processes that shape heart rate dynamics. This is achieved by conceptualizing the signal as a probabilistic process controlled by a time-varying mean, $\mu(t_i)$, and a partitioned, time-varying standard deviation. This sophisticated approach moves beyond simple curve-fitting to formalize a theory of autonomic control and its temporal evolution.

The cornerstone of the model is the partitioning of the total signal variance into two distinct, time-dependent components: a structured variance, $\sigma^2_{\text{struct}}(t_i)$, which captures the physiologically meaningful, oscillatory patterns of heart rate variability (HRV), and a residual variance, $\sigma^2_{\text{resid}}(t_i)$, which accounts for unstructured, moment-to-moment fluctuations or measurement noise. The complete observation model, which integrates these components, is defined by the Normal likelihood in Equation 1.

$$\text{RRi}(t_i) \sim \mathcal{N}(\mu(t_i), \sigma_{\text{resid}}(t_i)) \tag{1}$$

The core of the model lies in the construction of the mean and variance components from a shared set of underlying dynamic functions. The mean trajectory, $\mu(t_i)$, is a superposition of a smoothly varying baseline trend and the synthesized structured signal itself, as shown in Equation 2.

$$\mu(t_i) = \underbrace{\mathrm{RR}(t_i)}_{\substack{\text{Gross} \\ \text{RRi Trend}}} + \underbrace{A(t_i) \cdot \sum_{j=1}^{J} p_j(t_i) \cdot S_j(t_i)}_{\text{Structured Variability Signal}} \tag{2}$$

Here, $\mathrm{RR}(t_i)$ represents the gross, underlying heart period trajectory. The structured variability signal is synthesized from a set of dynamically evolving spectral oscillators, $S_j(t_i)$, which represent activity in different physiological frequency bands ($j = 1, 2, 3$ for VLF, LF, and HF, respectively). These oscillators are weighted by time-varying proportions, $p_j(t_i)$, and their overall magnitude is controlled by a scaling amplitude, $A(t_i)$, which is deterministically calculated to ensure the signal's variance matches the target structured variance, $\sigma^2_{\text{struct}}(t_i)$.

A pivotal feature is the a dynamic trajectory for the total signal variability, denoted $\mathrm{SDNN}(t_i)$, which is then partitioned into its structured and residual components. This partition is controlled by a new parameter, w, which represents the fraction of total variance attributable to the structured component, as defined in Equation 3.

$$\begin{aligned} \sigma^2_{\text{struct}}(t_i) &= w \cdot \mathrm{SDNN}(t_i)^2 \\ \sigma^2_{\text{resid}}(t_i) &= (1 - w) \cdot \mathrm{SDNN}(t_i)^2 \end{aligned} \tag{3}$$

This formulation allows the model to simultaneously learn not only how the total amount of HRV changes over time (via $\mathrm{SDNN}(t_i)$), but also how the nature of that variability evolves—that is, the balance between predictable, oscillatory patterns and unpredictable noise (via w). This unified analysis facilitates a deeper understanding of autonomic regulation by capturing phenomena across both time and frequency domains within a single, coherent inferential framework.

Baseline Heart Period and Total Variability Trajectories

The model posits that the primary trends in both the mean heart period and its total variability are driven by a common underlying physiological response to a stimulus. To capture this, both the $\mathrm{RR}(t_i)$ and $\mathrm{SDNN}(t_i)$ trajectories are parameterized using the same flexible double-logistic functional form.

The baseline heart period, $\mathrm{RR}(t_i)$, which quantifies the gross, underlying variations in the mean R–R interval, is defined in Equation 4.

$$\mathrm{RR}(t_i) = \underbrace{\alpha_r}_{\text{Resting RRi}} - \underbrace{\beta_r \cdot \mathcal{D}_1(t_i)}_{\substack{\text{Perturbation-induced} \\ \text{RRi Drop}}} + \underbrace{c_r \beta_r \cdot \mathcal{D}_2(t_i)}_{\substack{\text{Post-perturbation} \\ \text{RRi Recovery}}} \tag{4}$$

In this formulation, α_r represents the initial, stable heart period. The parameter β_r signifies the magnitude of the decline in RRi induced by the perturbation.

The fractional recovery amplitude is denoted by c_r, where $c_r > 1$ indicates an overshoot.

Concurrently, the total instantaneous variability, $\text{SDNN}(t_i)$, follows a parallel dynamic trajectory, as defined in Equation 5.

$$\text{SDNN}(t_i) = \underbrace{\alpha_s}_{\substack{\text{Resting SDNN}}} - \underbrace{\beta_s \cdot \mathcal{D}_1(t_i)}_{\substack{\text{Perturbation-induced} \\ \text{SDNN Drop}}} + \underbrace{c_s\beta_s \cdot \mathcal{D}_2(t_i)}_{\substack{\text{Post-perturbation} \\ \text{SDNN Recovery}}} \tag{5}$$

Here, α_s, β_s, and c_s are analogous to their counterparts in the baseline model, representing the resting total SDNN, the magnitude of its suppression, and its fractional recovery, respectively. The dynamics for both trajectories are driven by a shared pair of logistic transition functions, $\mathcal{D}_1(t_i)$ and $\mathcal{D}_2(t_i)$, defined in Equation 6.

$$\begin{aligned} \mathcal{D}_1(t_i) &= \left(1 + e^{-\lambda(t_i - \tau)}\right)^{-1} \\ \mathcal{D}_2(t_i) &= \left(1 + e^{-\phi(t_i - \tau - \delta)}\right)^{-1} \end{aligned} \tag{6}$$

The shared timing parameters enforce a strong physiological coupling: τ is the inflection point of the initial response, with rate λ. The second transition, representing recovery, is offset by a delay δ and proceeds at a rate ϕ. This shared structure ensures that changes in the magnitude of variability are temporally synchronized with changes in the mean heart period, while still allowing their relative magnitudes and recovery profiles to differ.

Generative Model for the Structured Signal

The structured component of the signal is where the model's spectral properties are defined. Its construction involves three key elements: the dynamic spectral proportions, the latent spectral oscillators, and the deterministic amplitude inversion that ties them to the target variance.

Dynamic Frequency Band Proportions

The proportions $p_j(t_i)$ dictate how the total structured variance, $\sigma^2_{\text{struct}}(t_i)$, is allocated across the different frequency bands at each moment. The model captures the evolution of these proportions as a smooth transition between two distinct spectral states: a baseline state ($\vec{\pi}_{\text{base}}$) and a perturbed state ($\vec{\pi}_{\text{pert}}$). The transition is orchestrated by a single master controller function, $C(t_i)$, as shown in the convex combination of Equation 7.

$$\vec{p}(t_i) = (1 - C(t_i)) \cdot \vec{\pi}_{\text{base}} + C(t_i) \cdot \vec{\pi}_{\text{pert}} \tag{7}$$

Here, $\vec{\pi}_{\text{base}}$ and $\vec{\pi}_{\text{pert}}$ are simplex vectors representing the characteristic spectral distributions at rest and during peak perturbation. The master controller,

$C(t_i)$, is itself built from the same logistic building blocks, ensuring the spectral transition is synchronized with the primary physiological response, as defined in Equation 8.

$$C(t_i) = \mathcal{D}_1(t_i) \cdot (1 - c_c \cdot \mathcal{D}_2(t_i)) \tag{8}$$

This function naturally transitions from 0 towards 1, with the parameter c_c allowing for an incomplete spectral recovery.

Latent Multi-Sine Spectral Oscillators

A significant advancement in this model version is the treatment of the spectral oscillators, $S_j(t_i)$, as latent variables to be estimated rather than fixed data. This allows the model to learn the specific phase and amplitude of the underlying spectral components from the data itself. Each oscillator is constructed as a superposition of K_j sinusoids with pre-specified frequencies, but with unknown amplitudes. The signal for band j is given by Equation 9.

$$S_j(t_i) = \sum_{k=1}^{K_j} \left[u_{j,k}^{(\sin)} \sin(2\pi f_{j,k} t_i) + u_{j,k}^{(\cos)} \cos(2\pi f_{j,k} t_i) \right] \tag{9}$$

The coefficients $u_{j,k}^{(\sin)}$ and $u_{j,k}^{(\cos)}$ are the unknown amplitudes for the sine and cosine components at each frequency $f_{j,k}$. To ensure a realistic spectral structure and aid inference, we place a hierarchical prior on these coefficients. This is achieved using a non-centered parameterization, where the coefficients are modeled as draws from a Normal distribution whose standard deviation is frequency-dependent, as defined in Equation 10.

$$u_{j,k}^{(\cdot)} \sim \mathcal{N}(0, \sigma_{u,j} \cdot a_{j,k}) \tag{10}$$

Here, the scale of the prior is determined by two components. First, $a_{j,k}$ imposes a power-law relationship with frequency, $a_{j,k} = f_{j,k}^{-b/2}$, where b is a global spectral exponent. This enforces a $1/f^b$ noise structure, a common characteristic of biological signals. Second, $\sigma_{u,j}$ is a band-specific scaling parameter that allows the model to adjust the overall prior variance for each band (VLF, LF, HF) separately, improving posterior geometry. Finally, after construction, each oscillator signal $S_j(t_i)$ is mean-centered to ensure it represents pure variability.

Deterministic Amplitude Inversion: $A(t_i)$

The final step is to ensure that the synthesized structured signal, $X(t_i) = A(t_i) \sum_{j=1}^{J} p_j(t_i) S_j(t_i)$, has a variance that is equal to the target structured variance, $\sigma^2_{\text{struct}}(t_i)$, at every time point. Because the oscillators $S_j(t_i)$ are not

standardized to unit variance and may be correlated, we must account for their full covariance structure. Let $_S$ be the 3×3 empirical covariance matrix of the three oscillator signals. The variance of the weighted sum is then given by the quadratic form in Equation 11

$$\text{Var}\left[\sum_{j=1}^{J} p_j(t_i) S_j(t_i)\right] = \vec{p}(t_i)^T \, {}_S\vec{p}(t_i) \tag{11}$$

The variance of the complete structured signal is $\text{Var}[X(t_i)] = A(t_i)^2 \cdot (\vec{p}(t_i)^T \, {}_S\vec{p}(t_i))$. To match our target, we set this equal to $\sigma^2_{\text{struct}}(t_i) = w \cdot \text{SDNN}(t_i)^2$. Solving for $A(t_i)$ yields the critical inversion formula in Equation 12.

$$A(t_i) = \frac{\sqrt{w} \cdot \text{SDNN}(t_i)}{\sqrt{\vec{p}(t_i)^T \, {}_S\vec{p}(t_i)}} \tag{12}$$

This inversion is essential. It dynamically adjusts the amplitude of the synthesized spectral signal to ensure its contribution to the total variance is exactly as prescribed by the model's high-level parameters ($\text{SDNN}(t_i)$ and w), thereby connecting the time-domain and frequency-domain components of the model.

Model Parameterization and Priors

To ensure numerical stability and efficient sampling, all model parameters are estimated on an unconstrained real-valued scale ($\mathbb{R}$). Priors are placed on these unconstrained parameters, which are then transformed back to their constrained, physically meaningful scales within the model.

Timing, Rate, and Magnitude Parameters

Parameters constrained to a specific interval are parameterized on the logit scale, while positive parameters are on the log scale. For instance, the timing parameters τ and δ are mapped to the observed time interval and the remaining time, respectively, using the inverse logit transformation, such that $\tau = \text{logit}^{-1}(\tau_{\text{logit}}) \cdot (t_{\max} - t_{\min}) + t_{\min}$ and $\delta = \text{logit}^{-1}(\delta_{\text{logit}}) \cdot (t_{\max} - \tau)$.

The positive rate parameters λ and ϕ are simply log-transformed, e.g., $\lambda = \exp(\lambda_{\log})$. The recovery coefficients (c_r, c_s, c_c) are mapped to the interval $[0, 2]$ using a scaled logit function, $c_i = \text{logit}^{-1}(c_{i,\text{logit}}) \cdot 2$. Similarly, the fraction of structured variance, w, is constrained between 0 and 1 via $w = \text{logit}^{-1}(w_{\text{logit}})$.

The magnitude parameters $(\alpha_r, \beta_r, \alpha_s, \beta_s)$ are scaled relative to data-derived quantities to create dimensionless parameters whose priors are easier to specify. For example, α_r is defined as $\alpha_r = \text{logit}^{-1}(\alpha_{r,\text{logit}}) \cdot 2 \cdot \text{rr\_range} + \text{rr\_min}$, which then scales its corresponding β parameter, $\beta_r = \text{logit}^{-1}(\beta_{r,\text{logit}}) \cdot \alpha_r$. A

similar relationship holds for α_s and β_s, which are scaled by the data's standard deviation. These transformations ensure the sampler explores a valid and well-behaved parameter space.

Spectral and Oscillator Parameters

A key aspect of the model lies in its handling of spectral components. The spectral proportions, $\vec{\pi}_{\text{base}}$ and $\vec{\pi}_{\text{pert}}$, are mapped from the 3-dimensional simplex to 2-dimensional real vectors, $\vec{y} = [y_1, y_2]$, using the additive log-ratio (ALR) transformation, where $y_1 = \log(\pi_1/\pi_3)$ and $y_2 = \log(\pi_2/\pi_3)$. The model estimates the unconstrained vectors $(\vec{y}_{\text{base, log}}, \vec{y}_{\text{pert, log}})$, which are mapped back to the simplex scale via the inverse transformation, where the components are $\pi_1 = e^{y_1}/D$, $\pi_2 = e^{y_2}/D$, and $\pi_3 = 1/D$, with a common denominator $D = 1 + e^{y_1} + e^{y_2}$.

To improve sampling efficiency, the oscillator amplitudes, $u_{j,k}$, are handled with a non-centered parameterization. Instead of estimating the highly correlated u values directly, the model estimates standard normal deviates, $z_{j,k}$. These are then scaled by a hierarchical prior to construct the final amplitudes as $u_{j,k} = z_{j,k} \cdot \sigma_{u,j} \cdot a_{j,k}$, where $a_{j,k} = f_{j,k}^{-b/2}$ is a frequency-dependent component. This technique dramatically reduces posterior correlations between parameters. Weakly informative Normal priors are placed on all unconstrained parameters, allowing the data to drive the final inference.

Results

[...].

Discussion

[...].