

# Geometry of Critical Transition: A New Language for Clinical Cardiology

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## Abstract

Standard clinical markers measure current state but do not estimate system resilience to phase transition. Patients with identical LVEF, heart rate, and blood pressure may be at fundamentally different distances from the decompensation threshold. We apply the geometric framework of Vector Interaction Theory (VIT) to describe the cardiovascular system as a geometric object in multidimensional phase space. In this view, reduced heart rate variability, loss of fractal complexity, and paradoxical patient stability are not isolated phenomena but manifestations of phase volume contraction, fragility accumulation, and approaching a critical threshold. We formulate testable predictions using open PhysioNet data and discuss why aggressive arrhythmia suppression (similar to CAST) may be more dangerous than the symptom itself.

## 1 Introduction: What is Vector Interaction Theory

VIT is a framework describing the behavior of any complex system near a critical transition [1]. Unlike statistical models (estimating probabilities) or phenomenological scales (assessing severity), VIT operates with phase space geometry. The system state at time  $t$  is described by a vector  $V(t)$  in the space of independent parameters  $I_k$  (for cardiology: heart rate, variability, ejection fraction, sympathovagal balance). The vector has length  $\|V\|$  and direction. A critical transition occurs when the misalignment angle  $\alpha$  between the system vector and the external intervention vector reaches a threshold  $\theta$ .

Key VIT concepts with direct analogs in cardiovascular physiology:

- **Phase volume**  $\text{Vol}_{\text{phase}} \propto \sqrt{\det(\text{cov}(V_t))}$  measures the set of states accessible to the system. In cardiology this is heart rate variability in all its dimensions [2]. Phase volume contraction means loss of flexibility: the system can respond to perturbations but has no margin for maneuver.
- **Misalignment angle**  $\alpha$  measures how well an external intervention (drug, exercise, stress) aligns with the system's intrinsic dynamics. Low  $\alpha$  means treatment works with the system; high  $\alpha$  means treatment works against the system, depleting reserve.

- **Fragility accumulation**  $P_{frag}$  is the integral of phase volume contraction under external interventions. In cardiology, an analog is the decline in heart rate variability after repeated hospitalizations, decompensation episodes, or aggressive dose titration. When fragility reaches a threshold, even a small perturbation triggers breakdown.
- **Critical slowing down** is a transition precursor seen as increased return time to baseline after a perturbation [4]. In HRV this appears as reduced short-term variability (RMSSD < 15-20 ms) with preserved mean heart rate.

Why is VIT applicable in cardiology? The cardiovascular system meets the criteria of a complex dynamical system with phase transitions: it has many interacting elements (sinus node, autonomic nervous system, contractile myocardium, vascular bed) and can undergo abrupt qualitative changes (fibrillation, asystole, decompensation).

## 2 Problem: System Collapse Where It Was “Stable”

Central claim: clinical “stability” is an observed parameter, not a characteristic of system resilience. A patient with compensated heart failure and a patient on the verge of decompensation may have identical LVEF, heart rate, and blood pressure but be at very different distances from the transition threshold.

Standard clinical practice focuses on average values: ejection fraction, resting heart rate, BNP level. However, these values describe only the position of the system in parameter space, not its geometry. Two systems can occupy the same point in space but have different margins for maneuver and different phase volumes. A system with narrow phase volume loses the ability to respond to perturbations without crossing the threshold.

In VIT terms, the system state is described by a vector  $V = (w_1 I_1, w_2 I_2, \dots, w_n I_n)$  in  $n$ -dimensional space. Phase volume  $\text{Vol}_{phase} \propto \sqrt{\det(\text{cov}(V_t))}$  reflects the space of available states. A healthy system occupies a volume and can shift in response to load without reaching the boundary. Phase volume contraction while means remain stable is hidden loss of resilience not captured by routine measurements.

- **Clinical paradox:** sudden death in “stable” coronary artery disease [10], decompensation after “planned” dose adjustment are not anomalies but predictable events in a system that has lost phase volume.
- **Marker:** SDNN and RMSSD measure rhythm dispersion but not phase space geometry. Reduced variability with stable means signals phase volume contraction.

## 3 Geometry Without Formulas: What “Loss of Flexibility” Means

A healthy heart is not a metronome but a system with rich phase space. Pathology and iatrogenic effects narrow this space long before mean values change.

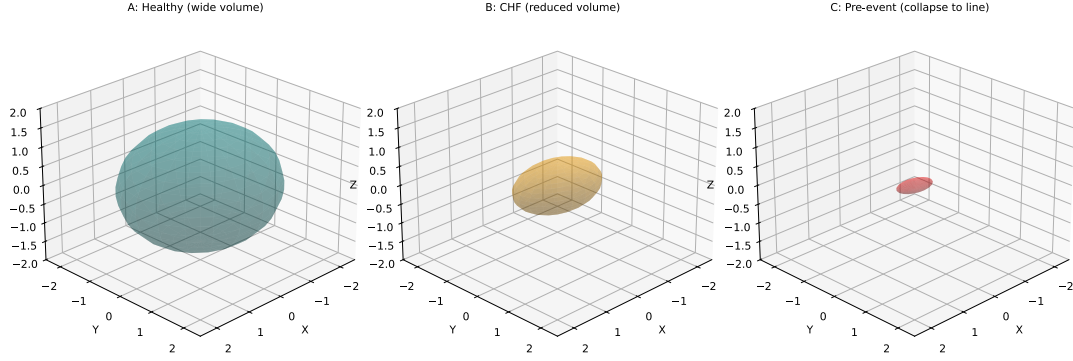


Figure 1: Phase volume contraction. A: healthy (wide volume, high adaptability). B: heart failure (reduced volume, lost flexibility). C: pre-event (collapse to line, critical slowing down).

### 3.1 Phase Volume as Margin for Maneuver

In VIT, phase volume  $\text{Vol}_{\text{phase}} \propto \sqrt{\det(\text{cov}(V_t))}$  is proportional to the product of variances of key parameters. Reduction of  $\text{Vol}_{\text{phase}}$  while means remain stable is loss of flexibility. In clinical practice, reduced heart rate variability is not “noise” but the use of phase volume. Declining variability signals that the system spends most time near a single point, with no maneuver margin [2, 3].

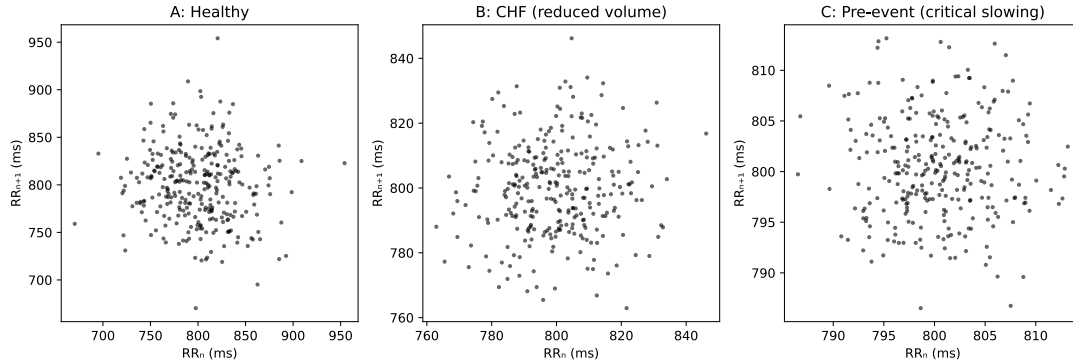


Figure 2: Poincaré plots of RR intervals. A: healthy (wide scatter). B: heart failure (narrowed scatter, reduced SD1). C: pre-event (collapse to line, critical slowing down).

### 3.2 Misalignment Angle $\alpha$ as a Measure of Treatment Efficacy

The angle  $\alpha$  between the external intervention vector and the system’s intrinsic dynamics determines treatment efficacy. When  $\alpha \rightarrow 0$ , the intervention aligns with the system’s natural motion: minimal cost, maximal effect. When  $\alpha \rightarrow \pi/2$ , the intervention is neutral. When  $\alpha \rightarrow \pi$ , the intervention opposes system dynamics: resources are spent on compensation, treatment becomes a source of fragility.

Clinically: aggressive rhythm control in vagal atrial fibrillation, forced diuresis in hypovolemic edema, antiarrhythmic drugs for compensatory tachycardia are all high- $\alpha$  regimens.  $\alpha$  is not directly observed but can be estimated indirectly: if increasing doses are required to maintain effect,  $\alpha$  is increasing.

## 4 Three Regimes Described by the Model

VIT distinguishes three regimes along the path to breakdown. Each has a signature pattern in HRV data.

### 4.1 Early Instability: System “Hunts”

Phase volume is preserved but the trajectory shows growing oscillations around the attractor. The system takes longer than before to return to equilibrium after each perturbation.

**Clinical equivalent:** prolonged return to baseline rhythm after exercise or orthostasis. In HRV, increased low-frequency component (LF) with preserved high-frequency component (HF); resting LF/HF > 2.5-3.0.

**Window of reversibility:** this is an early signal where low- $\alpha$  intervention may restore phase volume.

### 4.2 Fragility Accumulation During Treatment: Iatrogenic Phase Volume Contraction

Each high- $\alpha$  intervention reduces phase volume. If treatment suppresses symptoms without restoring dynamics, the system loses degrees of freedom. In VIT, fragility  $P_{frag}$  is proportional to  $1/Vol_{phase}$ . Accumulation of several high- $\alpha$  interventions leads to non-linear fragility growth, a “stability trap”: the patient appears compensated, but each subsequent perturbation is closer to the threshold [4].

**Marker:** progressive decline in SDNN while LVEF, heart rate, and blood pressure remain stable. Loss of nonlinear characteristics, decreased DFA  $\alpha_1$  below 0.75 or increased above 1.5 (both deviations are pathological) [5, 6].

**Clinically:** the patient is “well compensated”, but any deviation (infection, stress, missed dose) leads to disproportionately severe decompensation.

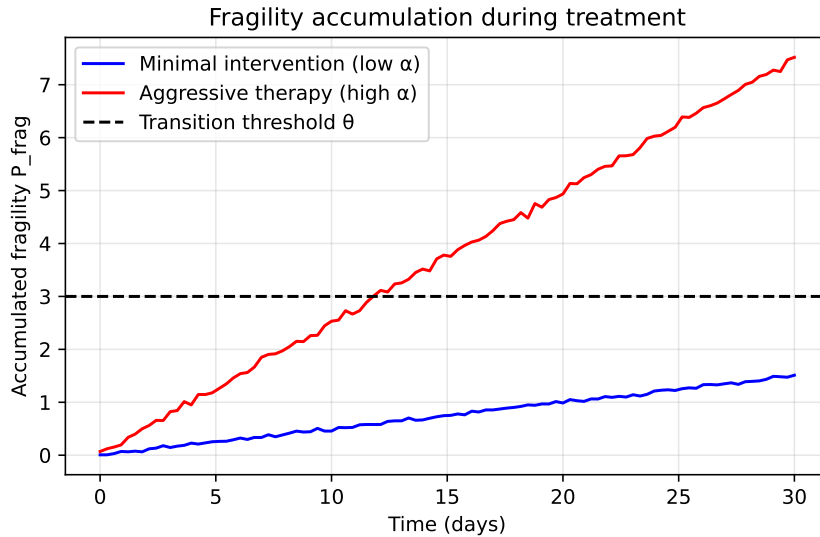


Figure 3: Fragility accumulation during treatment. Blue: minimal intervention (low  $\alpha$ ). Red: aggressive therapy (high  $\alpha$ ). Dashed line: transition threshold  $\theta$ .

### 4.3 Approaching the Threshold: Critical Slowing Down

As the system approaches a phase transition, the return time to the attractor after a perturbation tends to infinity, a universal precursor of transition in dynamical systems theory [4].

In VIT, the parameter  $W_k \rightarrow 1$  means the system constantly spends its reserve to stay near the edge, with no reserve left.

**Marker:** abrupt decline in short-term variability (RMSSD < 15-20 ms in patients with previous values above 30). On the Poincaré plot, the point cloud collapses to a straight line [3, 8].

**Clinically:** “the patient has become too stable”. This paradoxical sign is intuitively recognized by experienced cardiologists but lacks a formal language. In the literature, critical slowing down in HRV has been documented before ventricular fibrillation and in heart failure patients with preserved sinus rhythm [7].

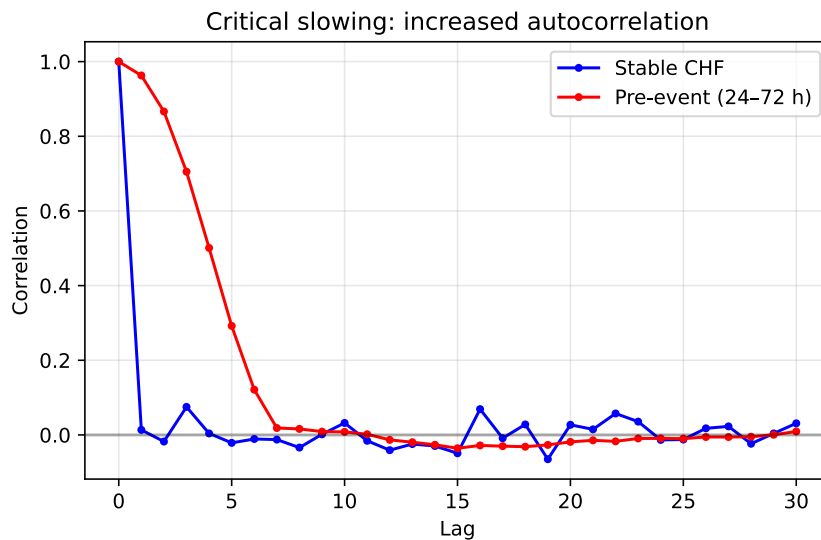


Figure 4: Critical slowing down: increased autocorrelation before transition. Blue: stable heart failure. Red: pre-event (24-72 hours before the event); note slower decay of autocorrelation indicating increased memory in the system — a hallmark of critical slowing down.

## 5 Minimal Intervention: Why Aggressive Suppression Sometimes Worsens Outcomes

The law of anthropogenic resonance: high- $\alpha$  intervention is not simply ineffective, it actively accelerates phase volume contraction.

Arrhythmia is in most cases not the cause of instability but a symptom that the system is searching for a new attractor. Suppressing arrhythmia without changing the geometry of phase space removes the signal while the cause remains.

The classic example is the CAST trial (1989) [4]. Class Ic antiarrhythmics (encainide, flecainide) effectively suppressed ventricular arrhythmias but increased mortality: arrhythmic death 4.5% on active treatment versus 1.2% on placebo, total mortality 7.7%

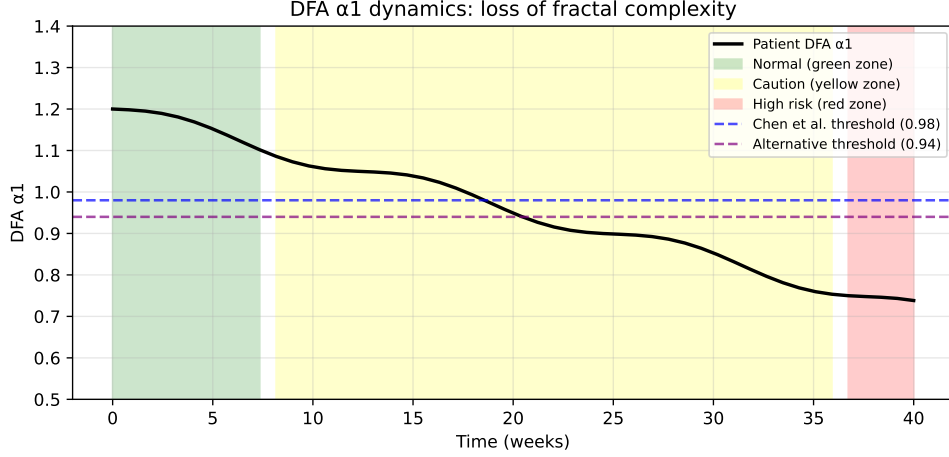


Figure 5: DFA  $\alpha_1$  dynamics with risk zones. Decline below the thresholds indicates loss of fractal complexity and increasing risk.

versus 3.0%. From the VIT perspective, these drugs acted with high  $\alpha$ , accumulated fragility  $P_{frag}$ , and accelerated the exit beyond the threshold, reducing phase volume.

The principle of minimal intervention favors interventions that expand phase volume rather than fix the system at a point. Low- $\alpha$  examples include moderate aerobic exercise in heart failure patients, vagal stimulation, and rhythmic breathing.

## 6 What Can Be Tested: Data and Simulation

The model makes testable predictions using open data. Available datasets include:

- MIT-BIH Arrhythmia Database: 47 recordings, 360 Hz, arrhythmia annotations
- BIDMC Congestive Heart Failure Database: 15 heart failure patients, about 20 hours each, 250 Hz
- Normal Sinus Rhythm RR Interval Database: 54 healthy adults

### 6.1 Implementation Algorithm

To reproduce the calculations, follow this protocol:

1. **Time-delay embedding (state space reconstruction):** From the RR interval time series  $RR_i$ , create a sequence of vectors

$$V_i = [RR_i, RR_{i+1}, RR_{i+2}]$$

using a window of three consecutive beats (embedding dimension  $m = 3$ ). Other embedding parameters (delay = 1) are fixed.

2. **Covariance matrix over the observation window:** For a fixed time window (e.g., 5 minutes), collect all vectors  $\{V_i\}$  into a matrix  $V$  (each column is one state vector). Compute the covariance matrix

$$C = \text{cov}(V)$$

which captures the spread of the system in phase space.

3. **Phase volume estimation:** The effective volume occupied by the system in phase space is

$$\text{Vol}_{\text{phase}} = \sqrt{\det(C)}.$$

This scalar quantifies the system’s complexity and flexibility. A decreasing  $\text{Vol}_{\text{phase}}$  while mean values remain stable signals hidden fragility.

4. **Mean state vector:** Compute the average system state as the row-wise mean of matrix  $V$ :

$$\bar{V} = \frac{1}{N} \sum_{i=1}^N V_i.$$

5. **Misalignment angle  $\alpha$ :** Let  $V_{\text{norm}}$  be a reference vector for a healthy or baseline state (e.g., from the patient’s own stable period). Then

$$\cos \alpha = \frac{\bar{V} \cdot V_{\text{norm}}}{\|\bar{V}\| \|V_{\text{norm}}\|}, \quad \alpha \in [0, \pi].$$

Low  $\alpha$  means the current dynamics align with the healthy pattern; high  $\alpha$  signals that external interventions are working against the system’s natural dynamics.

## 6.2 Dimensionless Fragility Index $P_{\text{frag}}$

Instead of speaking qualitatively about fragility, we define a computable index

$$P_{\text{frag}} = 1 - \left( \frac{\text{Vol}_{\text{observed}}}{\text{Vol}_{\text{baseline}}} \cdot \cos \alpha \right),$$

with  $0 \leq P_{\text{frag}} \leq 1$ . To ensure  $P_{\text{frag}} \in [0, 1]$ , values are clipped:  $P_{\text{frag}} = \max(0, \min(1, 1 - (\text{Vol}_{\text{observed}} / \text{Vol}_{\text{baseline}} \cdot \cos \alpha)))$ .  $P_{\text{frag}} \rightarrow 0$  when the system is flexible and interventions are well aligned.  $P_{\text{frag}} \rightarrow 1$  when phase volume collapses or misalignment is large.

A rising  $P_{\text{frag}}$  during treatment (as seen in the CAST trial) indicates that therapy increases fragility even when symptoms are suppressed.

## 6.3 Technical Constraints for Reliable Application

To avoid artefacts, the following data quality criteria must be met:

- **Sampling rate:** At least 250 Hz, preferably 500–1000 Hz. Lower rates (e.g., 125 Hz) introduce R-peak misalignment errors that artificially inflate covariance and give false phase volumes.
- **Ectopy removal and interpolation:** Ventricular ectopic beats must be not only removed but replaced by interpolated values (e.g., cubic splines). Simply deleting them creates holes that artificially enlarge  $\det(C)$  and distort phase volume estimation.
- **Window length:** For short-term analysis, use a fixed window of exactly 300 s (5 min). This follows HRV standards and allows direct comparison with classical time- and frequency-domain indices.

## 6.4 Testable Predictions Revisited

With the computable indices given above, the model yields three concrete predictions:

1. In heart failure patients, a decreasing DFA  $\alpha_1$  (threshold around 0.73 according to Mizobuchi et al. [5]) should precede clinical decompensation by a measurable interval. This is equivalent to a rise in  $P_{frag}$  weeks before symptoms.
2. Patients with subsequent sudden death should show increasing Poincaré plot monotonicity (decreasing SD1/SD2 ratio) and rising  $P_{frag}$ , as suggested by ultra-short-term HRV analysis [7].
3. Postoperative atrial fibrillation after CABG can be predicted by reduced DFA Alpha 1 (AUC=0.725) [9].

*Simulation methodology:* Phase portrait reconstruction from RR intervals using time-delay embedding (Takens method), estimation of phase volume as the volume of the minimal enclosing ellipsoid, tracking volume dynamics as a leading indicator.

## 7 Limitations of the Model

VIT describes the geometry of the state but not the underlying mechanism. The model is not a ready diagnostic tool but rather offers a language and metrics that require prospective validation.

Resuscitation is outside the scope. Once the system has crossed the threshold, the model describes the “after” state as a new attractor. Returning to the original state requires forced external management, not minimal intervention.

The model does not account for individual differences in baseline phase volume (athlete versus sedentary patient). Personalized baselines are needed, not population norms.

Additional limitations include: dependence on recording quality (minimum 250 Hz, artifact-free RR series); the three-dimensional embedding ( $m = 3$ ) used here is a simplification — optimal embedding dimension may vary by patient and condition; and the  $P_{frag}$  index, while computable, has not been validated against clinical endpoints in a prospective cohort.

A predictable reviewer question: “Where are the randomized data?” The honest answer is that the model is at the hypothesis-generating stage. This is the purpose of the work.

## 8 Conclusion

Standard clinical markers successfully detect changes that have already occurred but do not measure the distance to the threshold. VIT offers an additional lens: phase volume, misalignment angle, fragility accumulation, and critical slowing down. These concepts have quantitative analogs in HRV metrics (DFA  $\alpha_1$ , SD1/SD2, reduced variability with stable means) and can be tested on open data.

CAST remains a warning: symptom suppression without increasing the system’s phase volume may be more dangerous than the symptom itself [4]. The principle of minimal intervention (low  $\alpha$ , phase volume expansion) is not a rhetorical figure but a geometric requirement for therapy.



This work does not offer a ready clinical algorithm. It offers a language for formulating testable hypotheses and translating clinical observations (“the patient has become too stable”) into the geometry of phase transitions.

## A Data and Methods

Table 1: Threshold values of DFA  $\alpha_1$  for cardiac outcomes

Outcome	DFA $\alpha_1$ threshold	Sensitivity	Specificity	Source
Heart failure (HFpEF)	$0.73 \pm 0.27$	NR	NR	[5]
Healthy controls	$1.01 \pm 0.20$	NR	NR	[5]
Sudden cardiac death (general population)	DFA2 $\alpha_1$ significant predictor	NR	NR	[7]
POAF post-CABG	DFA Alpha 1 lower in AF	AUC=0.725	NR	[9]
Heart failure diagnosis	DFA $\alpha_1$ reduced vs controls	AUC=0.719	NR	[6]

Table 2: Poincaré metrics in normal subjects, heart failure, and post-myocardial infarction patients

Group	SD1 (ms)	SD2 (ms)	SD1/SD2	Source
Healthy adults (40-70 years)	21-37	50-75	0.32-0.48	[2]
Advanced heart failure	8-15	25-35	0.25-0.40	[3]
Post-myocardial infarction	14-22	38-52	0.30-0.45	[8]

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