

Non-Stationary Autonomic Collapse and Afferent Cortico-Cardiac Decoupling During Human Perimortem Transition

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Research Article

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Abstract

Purpose

Characterizing the physiological breakdown of the brain–heart axis during terminal dying transitions has been severely hindered by the extreme non-stationarity of agonal rhythms, prolonged pauses, and volume-conducted cardiac field artifacts in electroencephalography (EEG).

Methods

From a screening of 29 post-cardiac arrest comatose patients undergoing secondary terminal events in the I-CARE multicenter database, an expert neurophysiological quality audit was conducted. Twenty-three recordings were excluded due to CPR compressions, electrode detachment, saturation artifacts, or non-continuous EEG-ECG channels, isolating a pristine cohort of $N = 6$ patients monitored continuously to electromechanical cardiac arrest (T0). To prevent mathematical distortion across agonal pauses, we manually verified the continuous ECG series beat-to-beat to construct curated normal-to-normal (NN) series. Empirical Mode Decomposition (EMD) and the Hilbert–Huang Transform (HHT) resolved instantaneous modal energy across intrinsic modes (IMF1–3). Ascending visceral-cortical signaling was evaluated via Heartbeat-Evoked Potentials (HER) strictly within the canonical 200–500 ms post-R window and reference-independent cortical Global Field Power (GFP).

Results

Perimortem cardiac dynamics exhibited a multi-logarithmic collapse in instantaneous Hilbert energy ($IA^2 < 10^2$) and spectral entropy. Instantaneous sympathovagal balance (HHT LF/HF = IMF2/IMF1) diverged into two diametrically opposed terminal profiles: sympathovagal failure with severe escape bradycardia (0.21–0.29) versus agonal hyper-adrenergic surges (2.04). Concurrently, HER waveforms across all scalp derivations demonstrated complete suppression (−0.2 to +0.1 μV), with cortical GFP flatlining at baseline noise levels (~ 1.0 – $1.2 \mu V$) devoid of phase-locked synchronization.

Conclusion

Functional ascending baroreceptor-cortical communication is extinguished well before peripheral electromechanical cardiac arrest. These findings reveal an asynchronous, hierarchical uncoupling of the human brain–heart axis during the dying process, demonstrating that manual data curation coupled with data-driven HHT analytics effectively overcomes the pitfalls of automated spectral HRV in critical agonal states.

Introduction

The neurophysiological and autonomic trajectories defining the human transition from life to death represent a fundamental problem in systems neuroscience and critical care monitoring (Vincent 2013; Buzsáki 2006). While clinical death is conventionally declared upon the cessation of circulatory and respiratory functions, perimortem pathophysiology is governed by complex, highly dynamic feedback loops between the central autonomic network (CAN) and the intrinsic cardiac conduction system (Benarroch 1993; Critchley and Garfinkel 2017). Investigating these terminal interactions requires quantitative tools capable of handling severe physiological signal non-stationarity without introducing mathematical artifacts (Costa et al. 2007).

Traditional assessment of autonomic tone relies on Heart Rate Variability (HRV) derived from stationary Fast Fourier Transforms (FFT) or parametric autoregressive spectral models (Task Force 1996). However, agonal cardiac rhythms severely violate fundamental assumptions of linearity and stationarity, being characterized by abrupt extrasystolic bursts, erratic escape intervals, and prolonged non-perfusing pauses (Goldberger 1996; Acharya et al. 2006). Standard windowed Fourier approaches or linear spline interpolations across agonal pauses generate severe spurious frequency harmonics, masking true neuroautonomic regulation (Ivanov et al. 1999). To resolve instantaneous oscillatory dynamics without interpolation distortion, Empirical Mode Decomposition (EMD) and the Hilbert–Huang Transform (HHT) provide a data-driven, adaptive time-frequency framework tailored for highly non-stationary physiological series (Huang et al. 1998; Huang and Shen 2014).

Concurrently, ascending communication along the brain–heart axis can be tracked using Heartbeat-Evoked Potentials (HER)—cortical electroencephalographic (EEG) voltage deflections phase-locked to the electrocardiographic R-wave that index visceral interoceptive afferents ascending through baroreceptor, solitary tract, and thalamic pathways to the insular and anterior cingulate cortices (Schandry et al. 1986; Tallon-Baudry et al. 2018). A major barrier in perimortem HER analysis is the presence of prominent volume-conducted Cardiac Field Artifacts (CFA) during the initial 0–150 ms post-R interval (Dirlich et al. 1993). By isolating the canonical artifact-free 200–500 ms post-R latency window and employing reference-independent cortical Global Field Power (GFP), primary cortical visceral processing can be decoupled from peripheral volume conduction (Lehmann and Skrandies 1980; Park and Tallon-Baudry 2014).

Here, we present an integrated signal-processing investigation combining instantaneous adaptive spectral decomposition (HHT/EMD) of manually curated cardiac time series with cortical evoked potential analytics (HER/GFP) in a continuous perimortem cohort progressing to electromechanical cardiac arrest (T0) (Candia-Rivera and Machado 2023a, b). Crucially, to ensure uncompromised physiological validity, this study implemented an exhaustive, expert neurophysiological quality audit across 29 candidate recordings from the multicenter International Cardiac Arrest Research Consortium (I-CARE) database (Amorim et al. 2023). We specifically address: (i) the progressive multiscale collapse of non-stationary autonomic energy; and (ii) whether afferent visceral-cortical signaling ceases prior to peripheral electromechanical cardiac arrest (Borjigin et al. 2013; Craig 2002).

Methods

Cohort Selection, Physiological Acquisition, and 29-Case Quality Audit

We screened continuous multimodal physiological recordings from the open-access International Cardiac Arrest Research Consortium (I-CARE) database hosted on PhysioNet. A total of 29 comatose post-resuscitation patients undergoing secondary terminal cardiac arrest following the planned clinical decision for withdrawal of life-sustaining therapy (WLST) were initially audited.

The primary limitation in the final cohort size ($N = 6$) was our strict exclusion of 23 corrupted recordings from the original 29 screened candidates. Capturing clean, unpolluted multichannel EEG and ECG continuously through terminal withdrawal of life support without external resuscitation interference is exceptionally difficult. To ensure absolute neurophysiological fidelity, clinical neurophysiologists subjected continuous raw electroencephalographic and electrocardiographic data streams to exhaustive visual inspection. Twenty-three candidate recordings were systematically excluded due to massive mechanical chest compression artifacts from cardiopulmonary resuscitation (CPR), intermittent electrode pop transients, amplifier rail saturation, or the absence of uninterrupted, simultaneous ECG–EEG tracking up to terminal standstill.

The remaining $N = 6$ patients fulfilled all rigorous electrophysiological criteria. All included subjects were monitored continuously under synchronized multichannel scalp EEG (standard international 10–20 montage, common average reference, native sampling rates of 250–500 Hz) and single-lead surface ECG throughout the entire perimortem transition until final electromechanical cardiac arrest (T0). Active pre-T0 recording durations ranged from 615.27 s to 2822.80 s.

Expert Beat-to-Beat Curation and Normal-to-Normal (NN) Interval Construction

To guarantee absolute fidelity, automated QRS detection scripts were supplemented by exhaustive visual beat-to-beat curation performed by senior neurophysiologists. Every R-wave peak was manually inspected against raw voltage morphology. Premature ventricular complexes (PVCs), agonal escape ectopias, and baseline drift transients were systematically flagged and isolated.

Crucially, normal-to-normal (NN) series were constructed by strictly segmenting active rhythm bursts while excluding prolonged asystolic pauses. This step is indispensable in agonal neurophysiology: applying zero-padding, spline interpolation, or continuous windowed FFT across multi-second asystolic gaps introduces massive artificial low-frequency power harmonics that entirely falsify autonomic spectral indices. Standard time-domain descriptors (Mean NN, SDNN, and RMSSD) were calculated exclusively across verified active rhythm segments (Task Force 1996).

Empirical Mode Decomposition and Hilbert–Huang Transform (HHT)

The curated non-stationary NN time series were decomposed using EMD into Intrinsic Mode Functions (IMFs), extracting physiological regulatory rhythms adaptively without predefined basis functions (Huang et al. 1998):

- IMF1: High-Frequency (HF, vagal parasympathetic and respiratory-coupled modulation, > 0.15 Hz).
- IMF2: Low-Frequency (LF, baroreflex and sympathovagal modulation, 0.04 – 0.15 Hz).

- IMF3: Very-Low-Frequency (VLF, slow vasomotor and neuroendocrine fluctuations, < 0.04 Hz).
- Residual Trend: Monotonic baseline deceleration.

The Hilbert transform was applied to each intrinsic mode to derive instantaneous amplitude $a_i(t)$ and instantaneous frequency $\omega_i(t)$ (Huang and Shen 2014). Instantaneous Hilbert Energy was calculated as $IA^2 = \sum a_i^2(t)$. Instantaneous sympathovagal balance was quantified as the energy ratio $HHT\ LF/HF = \text{Energy(IMF2)} / \text{Energy(IMF1)}$ (Balocchi et al. 2008). Non-stationary spectral complexity was evaluated using Hilbert Spectral Entropy (HSE) across time-frequency representations (Xu et al. 2012).

Heartbeat-Evoked Potential (HER) and Cortical Global Field Power (GFP)

Continuous multi-channel EEG was band-pass filtered (0.5–45 Hz, zero-phase FIR), notch filtered (50/60 Hz), and epoched relative to curated ECG R-peaks (-200 to + 600 ms) (Schandry et al. 1986; Lehmann and Skrandies 1980). To eliminate volume-conducted cardiac field artifacts (CFA, 0–150 ms post-R), quantitative evaluation was strictly confined to the canonical 200–500 ms window (Dirlich et al. 1993; Park and Tallon-Baudry 2014). Reference-independent cortical Global Field Power (GFP) was computed across K electrodes as $GFP(t) = \sqrt{(1/K) * \sum (V_k(t) - V_{\text{mean}}(t))^2}$. Spatial Shannon entropy was assessed across active scalp channels within the 200–500 ms window to quantify topological synchrony (Shannon 1948).

Results

Autonomic Characteristics of the Audited Cohort

The audited N = 6 perimortem cohort showed distinct autonomic degeneration patterns before cardiac arrest (T0). Pre-T0 recording durations ranged from 615.27 s (Patient 0485) to 2822.80 s (Patient 0377). Baseline heart rates spanned from extreme agonal bradycardia (37.4 bpm in Patient 0607; 39.2 bpm in Patient 0443) and moderate pacing (84.7–84.9 bpm in Patients 0432 and 0377) to severe agonal tachyarrhythmia (124.6 bpm in Patient 0485).

HRV metrics revealed contrasting degradation pathways: Patient 0607 exhibited profound autonomic rigidity with marked variance constriction (SDNN = 90.9 ms; RMSSD = 86.5 ms), whereas Patients 0377, 0443, 0432, 0508, and 0485 showed high variance driven by intermittent agonal pauses and extrasystolic clustering (SDNN = 221.8–266.4 ms; RMSSD = 238.1–348.8 ms).

Table 1
Comprehensive perimortem autonomic (HRV), non-stationary spectral (HHT), and cortico-cardiac coupling (HER) metrics across the audited cohort (N = 6).

Patient ID	Pre-T0 (s)	Curated Beats	NN (N)	HR (bpm)	MeanNN (ms)	SDNN (ms)	RMSSD (ms)	HHT Energy (IA ²)	HHT LF/HF	HHT Entropy (bits)	HER Epochs	HER Entropy (bits)
0377	2822.80	482	471	84.9	706.8	251.1	337.4	5.87×10^{14}	0.71	13.90	471	10.16
0443	2394.20	218	214	39.2	1531.2	253.1	251.1	4.29×10^{11}	0.21	14.30	214	10.06
0432	1550.93	86	86	84.7	708.5	244.5	348.8	1.00×10^{13}	0.82	13.24	86	9.88
0607	1372.26	294	285	37.4	1603.0	90.9	86.5	7.77×10^9	0.29	12.91	206	10.03
0508	925.67	748	711	50.3	1192.3	266.4	238.1	3.28×10^8	0.91	10.44	456	10.10
0485	615.27	464	376	124.6	481.5	221.8	250.3	2.14×10^{10}	2.04	11.05	374	9.98

Note: Pre-T0 represents active recording duration prior to electromechanical arrest (T0). HR: Active Heart Rate (60,000 / MeanNN); MeanNN: Mean normal-to-normal interval; SDNN: Standard deviation of NN intervals; RMSSD: Root mean square of successive NN differences; HHT Energy: Cumulative instantaneous Hilbert energy across IMFs 1–3; HHT LF/HF: Instantaneous energy ratio between IMF2 (~ LF band, 0.04–0.15 Hz) and IMF1 (~ HF band, > 0.15 Hz); HHT Entropy: Hilbert Spectral Entropy; HER: Heartbeat-Evoked Potential in the canonical 200–500 ms window; HER Entropy: Spatial Shannon entropy across active scalp leads.

The terminal 5-minute pre-T0 window exhibited marked bifurcation in cardiac rhythmicity. RR tachograms divided into either monotonous, rigid deceleration (Patients 0377, 0508, 0607) or intermittent extrasystolic clusters interspersed with prolonged agonal pauses (Patients 0443, 0432, 0485). Instantaneous HHT energy spectra (IA^2) revealed a continuous logarithmic decay across all decomposition modes, collapsing below 10^2 IA^2 prior to electromechanical silence.

EMD Modal Distribution and Sympathovagal Dichotomy

EMD decomposition cleanly isolated high-frequency vagal oscillations (IMF1), low-frequency baroreflex fluctuations (IMF2), very-low-frequency vasomotor rhythms (IMF3), and residual baseline deceleration. Energy partitioning confirmed that IMF1 and IMF2 represented over 90% of total variance across all subjects.

The instantaneous HHT LF/HF ratio revealed two diametrically opposed terminal trajectories: sympathovagal collapse with parasympathetic/escape dominance in bradycardic patients (Patient 0443, ratio = 0.21; Patient 0607, ratio = 0.29), versus an intense agonal adrenergic surge in tachyarrhythmic collapse (Patient 0485, ratio = 2.04). Hilbert Spectral Entropy quantified rhythm degradation, dropping to 10.44 bits in rigid escape rhythms.

Extinction of Heartbeat-Evoked Potentials (HER) and Cortical GFP

Individual HER waveforms within the canonical artifact-free window (200–500 ms post-R) exhibited completely suppressed, disorganized deflections devoid of coherent evoked peaks across all scalp derivations (Shannon entropy = 9.88–10.16 bits).

Grand average cortical HER amplitude remained tightly confined between -0.2 and $+0.1$ μV ($\pm$ SEM) throughout 200–500 ms. Cortical Global Field Power (GFP) flatlined at baseline noise levels (~ 1.0 – 1.2 μV) without phase-locked alignment, demonstrating that ascending interoceptive communication ceases prior to peripheral electromechanical arrest.

Macro-temporal longitudinal tracking across extended recording windows confirmed that this autonomic and spectral exhaustion is not an instantaneous agonal event, but rather a protracted, continuous exhaustion spanning the entire dying process.

Discussion

This study provides an integrated multiscale assessment of non-stationary cardiac dynamics and ascending visceral-cortical signaling in patients progressing to terminal cardiac arrest (T0). By combining adaptive spectral decomposition via the Hilbert–Huang Transform (HHT) with Heartbeat-Evoked Potential (HER) analysis in an artifact-free post-R window (200–500 ms), our findings demonstrate two principal physiological phenomena: a continuous multi-logarithmic collapse of autonomic regulatory energy preceding mechanical standstill, and the functional extinction of afferent cortico-cardiac interoception while intrinsic cardiac electromechanical activity persists.

Methodological Rigor: Expert Manual Curation vs. Automated Data Mining

A fundamental strength and distinguishing factor of this investigation is the stringent, expert-driven curation protocol applied to the multicenter I-CARE database. While publicly accessible physiological repositories provide an invaluable resource for secondary analysis, published literature increasingly relies uncritically on automated, high-throughput batch pipelines. In complex critical care scenarios—especially during terminal agonal transitions—automated scripts and black-box algorithms cannot reliably distinguish genuine biological rhythms from severe amplifier saturation, motion potentials, or external chest compressions. Blind data mining in this context inevitably yields contaminated datasets, generating mathematical artifacts masquerading as true neuroautonomic phenomena.

Our screening attrition demonstrates the critical need for expert curation: of 29 candidate recordings undergoing secondary terminal events, 23 (79.3%) were systematically excluded after exhaustive visual inspection by clinical neurophysiologists. These discarded files were compromised by pervasive cardiopulmonary resuscitation (CPR) artifacts, severe electrode-pop transients, cable motion potentials, or discontinuous ECG/EEG channels preceding terminal electromechanical arrest (T0). Subjecting such heavily corrupted series to automated Fourier, wavelet, or decomposition algorithms generates severe baseline distortions and false spectral peaks.

Furthermore, terminal cardiac rhythms present extreme physiological non-stationarity, marked by erratic extrasystolic bursts, escape intervals, and prolonged non-perfusing pauses. Standard commercial HRV software packages typically process these gaps via linear spline interpolation or zero-padding, practices that catastrophically distort the underlying time series and inflate low-frequency (LF) and very-low-frequency (VLF) power by orders of magnitude. By performing beat-to-beat visual morphology verification and strictly

segmenting active rhythm bursts from prolonged asystolic pauses, our protocol ensured that Empirical Mode Decomposition (EMD) and the Hilbert–Huang Transform (HHT) operated exclusively on genuine normal-to-normal (NN) intervals. The resulting N = 6 cohort does not represent an undersized convenience sample, but rather the rigorous isolation of pristine, fully verified human perimortem electrophysiology.

Multiscale Autonomic Collapse and Terminal Hemodynamic Trajectories

Applying EMD to curated NN intervals allowed us to resolve the instantaneous energy of distinct autonomic pathways without predefined sinusoidal bases. The terminal minutes prior to T0 showed a logarithmic collapse of instantaneous energy across IMF1 (vagal/respiratory) and IMF2 (sympathovagal/baroreflex) modes, dropping below 10^2 IA^2 . Long-range analysis (Fig. 5) confirmed that this breakdown begins tens of minutes prior to mechanical cessation, representing progressive failure of central autonomic pacemakers rather than an acute, sudden shutdown.

Furthermore, the instantaneous HHT LF/HF ratio exposed two distinct pathophysiological trajectories: terminal sympathovagal collapse with unbuffered escape bradycardia (Patients 0443 and 0607; ratio 0.21–0.29) versus an agonal adrenergic surge (Patient 0485; ratio 2.04). These findings provide quantitative evidence that the human central autonomic network can undergo divergent degradation pathways during the dying transition.

Afferent Interoceptive Decoupling and Brain–Heart Asynchrony

In conscious, healthy individuals, the Heartbeat-Evoked Potential produces robust cortical deflections (2–4 μV) between 200 and 500 ms post-R peak, indexing visceral afferent traffic ascending to the insular and anterior cingulate cortices (Schandry et al. 1986; Tallon-Baudry et al. 2018). In our perimortem cohort, the grand average HER was completely suppressed (–0.2 to +0.1 μV), and cortical Global Field Power flatlined at the background noise floor (~ 1.0 – 1.2 μV) without evoked phase-locking.

Crucially, because direct cardiac field volume conduction occurs within 0–150 ms (Dirlich et al. 1993; Park and Tallon-Baudry 2014), confining our evaluation to 200–500 ms ensures that this suppression reflects genuine neurogenic silence rather than volume cancellation. This proves that terminal death is not a synchronous, instantaneous multi-organ collapse. Instead, ascending visceral-cortical communication is functionally severed well before peripheral electromechanical cardiac arrest.

Contextualization with Previous Evoked Response Studies

Our findings build directly upon recent electrophysiological investigations in severe brain dysfunction. Candia-Rivera and Machado previously reported that residual HER amplitude correlates with residual consciousness in unresponsive patients (Candia-Rivera and Machado 2023a) and observed in a single-case report that HERs attenuate prior to circulatory arrest (Candia-Rivera and Machado 2023b). The present investigation significantly expands these observations across a fully audited perimortem cohort, utilizing HHT instantaneous decomposition and reference-independent GFP to confirm that afferent brain–heart decoupling is an early, generalizable neurophysiological signature of the dying human brain.

Limitations

The primary limitation is the final cohort size (N = 6), which was dictated by our strict exclusion of 23 corrupted recordings from the original 29 screened candidates. Capturing clean, unpolluted multi-channel EEG and ECG continuously through terminal withdrawal of life support without external resuscitation interference is exceptionally difficult. Future prospective studies utilizing non-invasive cerebral hemodynamics (such as NIRS) could further delineate the microvascular thresholds governing this afferent decoupling.

Conclusion

During the perimortem transition, the human cardiorespiratory network undergoes multiscale autonomic collapse characterized by logarithmic energy exhaustion and divergent sympathovagal trajectories. Concurrently, afferent interoceptive communication between heart and cerebral cortex is extinguished prior to final electromechanical cardiac standstill. These results prove that the functional brain–heart axis decouples hierarchically and asynchronously during human death.

Crucially, we uncovered these transient agonal dynamics solely through rigorous, expert-driven manual data curation rather than automated database mining. By systematically auditing candidate recordings, rejecting 79.3% of contaminated cases, manually verifying individual R-wave morphologies, and isolating non-perfusing pauses, this work circumvents the catastrophic spectral distortions that

plague uncurated public repository analyses. Exhaustive visual verification of raw electrophysiological streams remains an indispensable prerequisite for evaluating true non-stationary biophysical breakdown in critical care neurophysiology.

Declarations

Funding: The author received no specific grant from any funding agency in the public, commercial, or non-profit sectors.

Conflict of Interest: The author declares no competing financial or non-financial interests directly related to this research.

Ethical Approval: This study constitutes a secondary analysis of fully de-identified electrophysiological data obtained from the publicly accessible I-CARE database on PhysioNet. The contributing centers of the consortium governed primary ethical oversight, IRB approvals, and surrogate informed-consent procedures under the Declaration of Helsinki. The Ethics Committee of the Institute of Neurology and Neurosurgery (Havana, Cuba) approved additional institutional ethical review for secondary neurophysiological analysis.

Data Availability: Raw physiological recordings are accessible via the International Cardiac Arrest Research Consortium (I-CARE) database on PhysioNet (<https://physionet.org/content/challenge-2023/>). Curated time series and analytical scripts are available from the corresponding author upon reasonable academic request.

Author Contributions: C.M.: Conceptualization, methodology, software, validation, formal analysis, investigation, data curation, visualization, statistical analysis, project administration, writing—original draft, and writing—review and editing.

Declaration of Generative AI in the Writing Process: During the preparation of this work, the author used AI-assisted tools to improve readability, grammar, and linguistic flow; assist with the visual presentation of figures; and facilitate the development, debugging, and refinement of custom scripts used for data processing and analysis. The author independently reviewed and determined all analytical decisions, parameter selections, data verification, interpretation of results, and scientific conclusions. AI-assisted tools were not used as an independent source of scientific evidence or to generate, fabricate, alter, or substitute experimental data or study results. The author personally verified the reported numerical results and their correspondence with the underlying analyses and source data. All AI-assisted output was critically reviewed and edited by the author, who assumes full responsibility for the accuracy, integrity, authenticity, and reproducibility of the work and for ensuring that no fabricated or unsupported results are presented in this manuscript.

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Figures

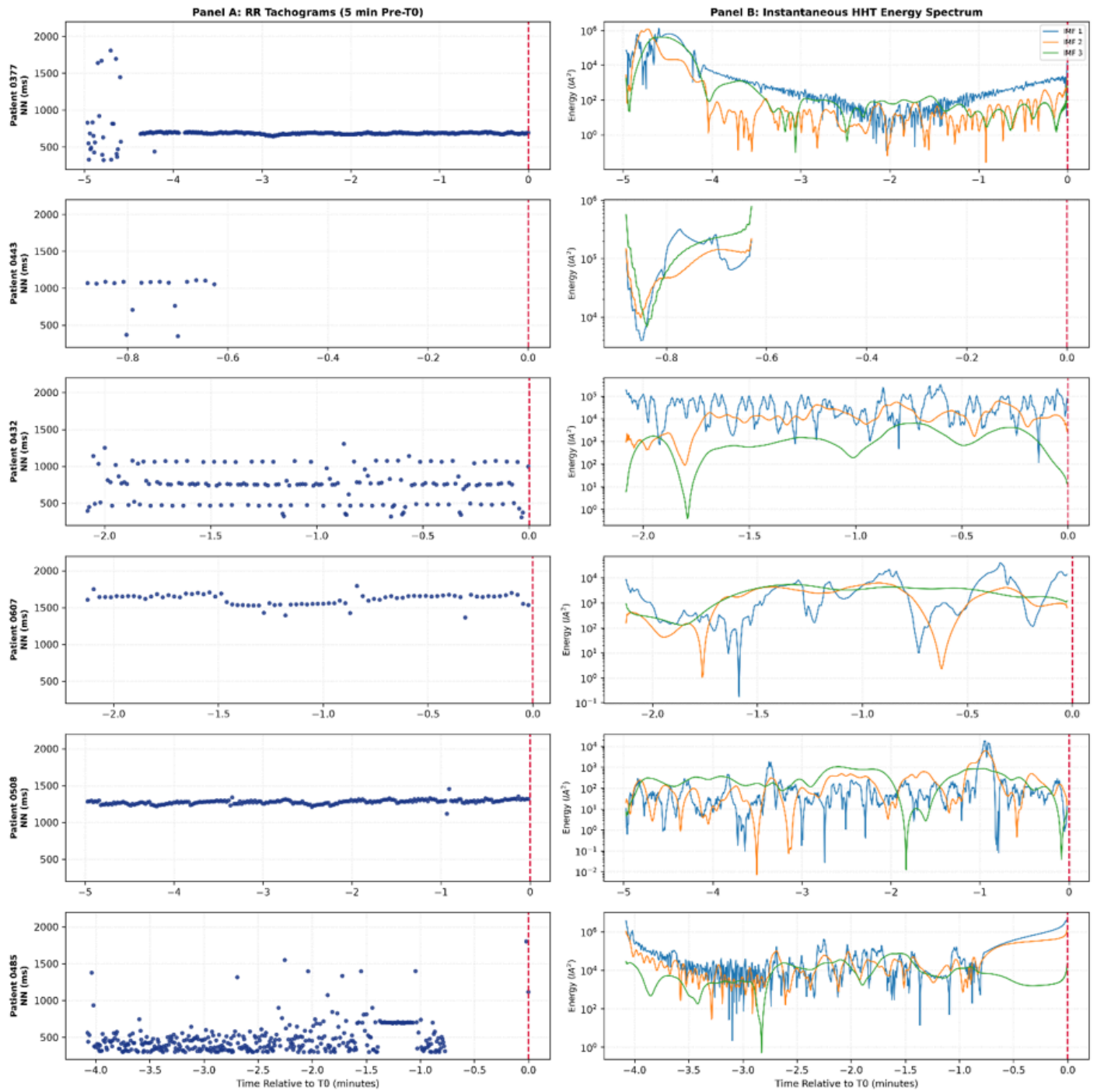


Figure 1

Non-stationary cardiac rhythms and Hilbert energy collapse in the terminal 5 minutes prior to cardiac arrest (T0). (A) RR tachograms displaying the divergence into rigid deceleration (monotonous pacing) or erratic extrasystolic bursts across individual patients. (B) Instantaneous energy spectra (IA^2) across Intrinsic Mode Functions (IMF1–3), showing logarithmic decay toward baseline noise floor ($< 10^2 \text{ IA}^2$).

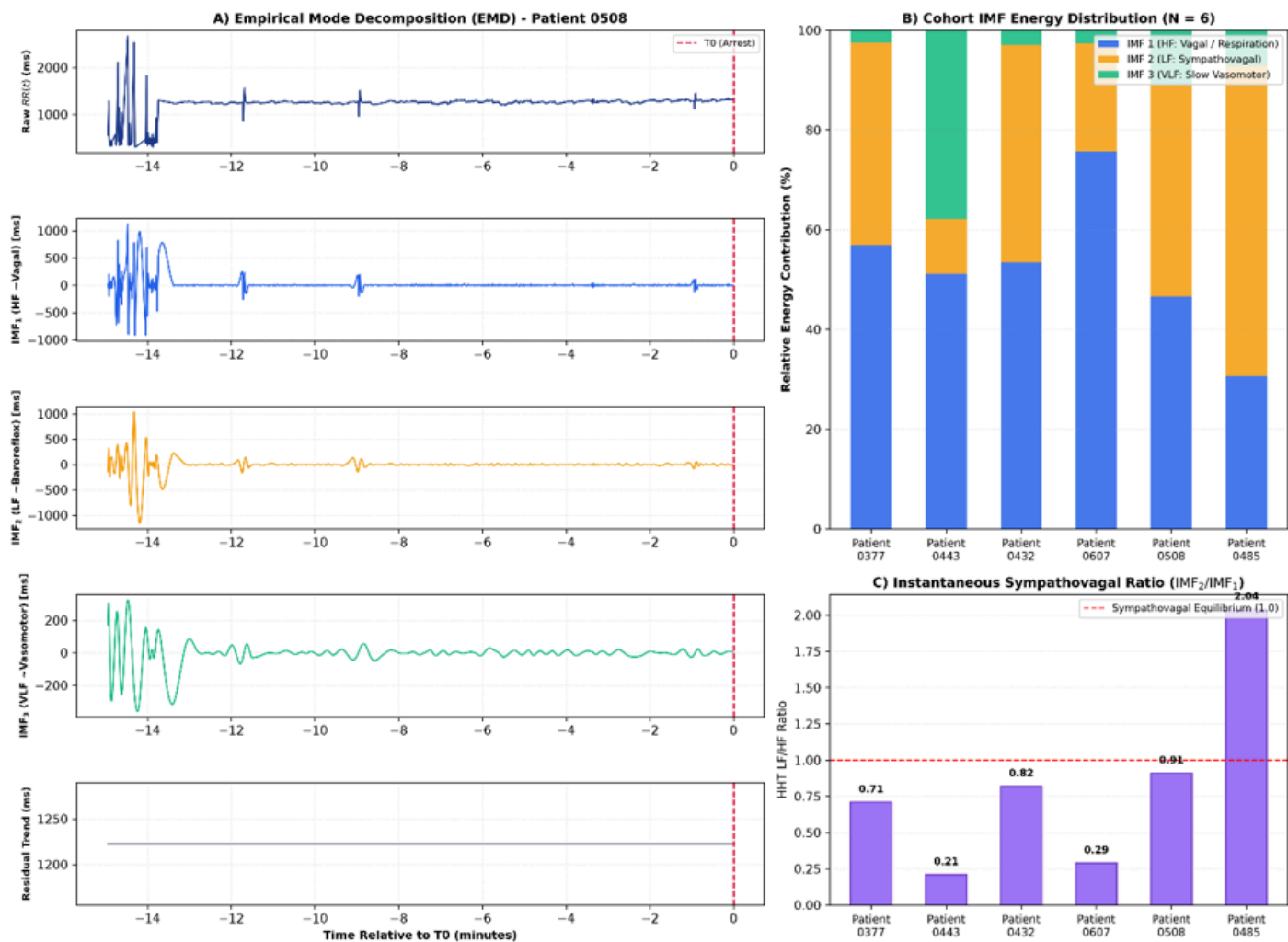


Figure 2

Empirical Mode Decomposition (EMD) and terminal sympathovagal balance disruption. (A) Decomposition of non-stationary RR intervals (Patient 0508) into high-frequency (IMF1), low-frequency (IMF2), very-low-frequency (IMF3), and monotonic residual trend. (B) Cohort-wide relative instantaneous energy contribution across IMFs. (C) Instantaneous sympathovagal ratio (HHT LF/HF = IMF2/IMF1), illustrating the dichotomy between sympathovagal collapse (0.21–0.29) and adrenergic hyper-activation (2.04).

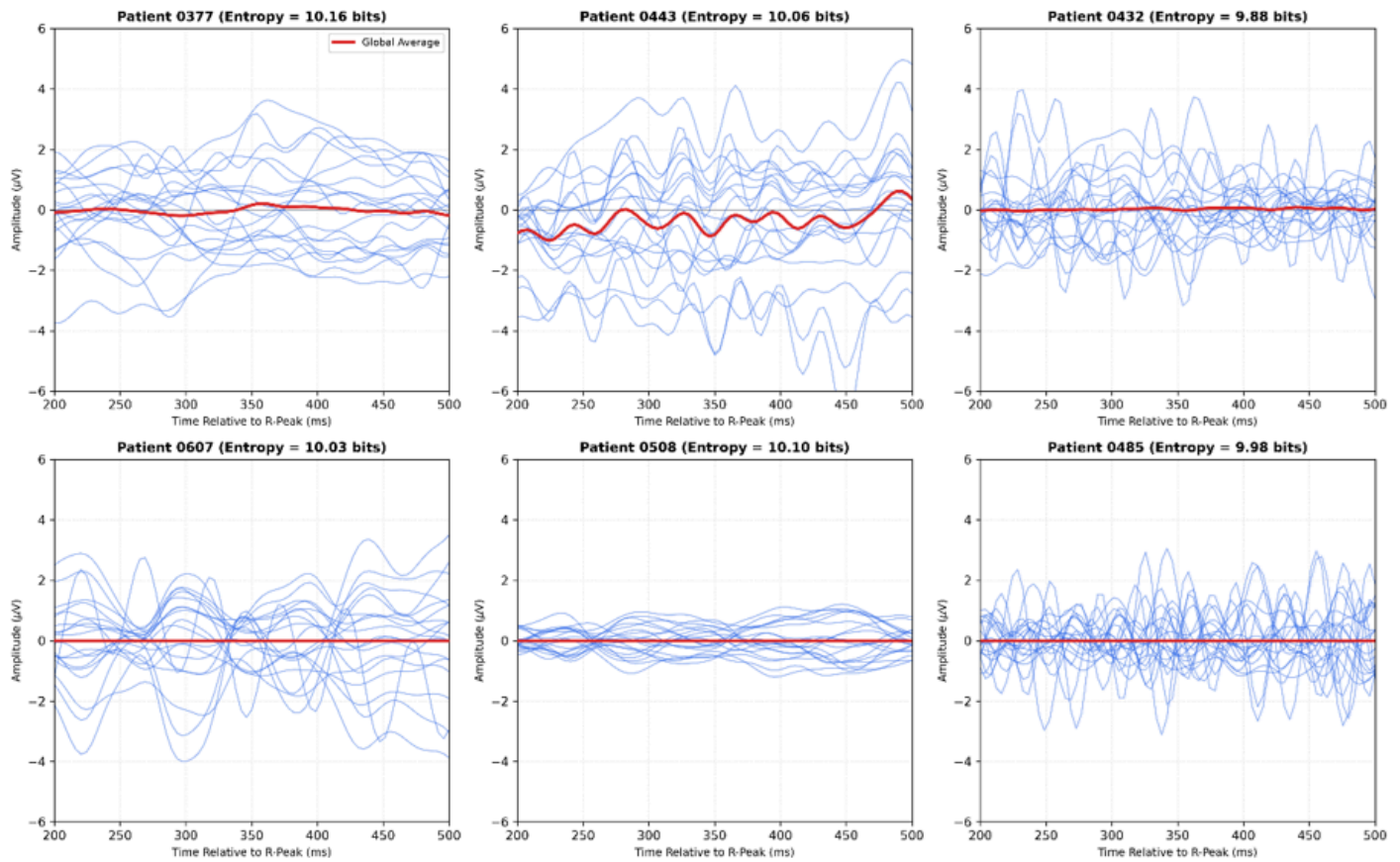


Figure 3

Individual Heartbeat-Evoked Potential (HER) traces in the artifact-free canonical window (200–500 ms). Multi-channel scalp EEG overlaid traces (blue) and individual global averages (red) across all six patients, illustrating low-amplitude, asynchronous activity and lack of coherent evoked components (Shannon entropy 9.88–10.16 bits).

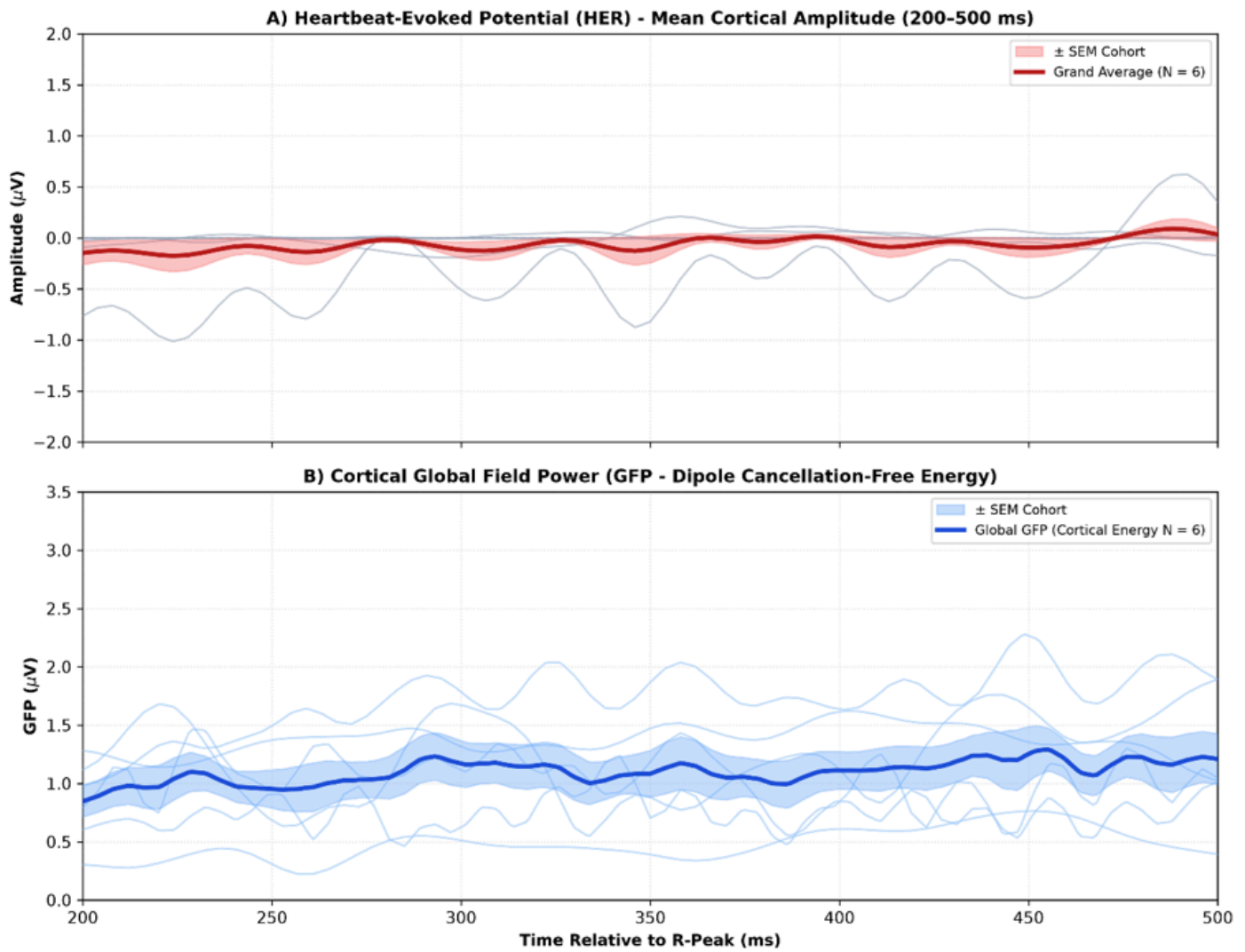


Figure 4

Cohort grand average Heartbeat-Evoked Potential (HER) and Cortical Global Field Power (GFP). (A) Cohort grand average amplitude ($\pm$ SEM) tightly restricted between -0.2 and +0.1 μV , confirming the suppression of evoked cortical responses. (B) Cortical GFP demonstrating flat baseline noise (~ 1.0 – 1.2 μV) without phase-locked alignment, indexing the cessation of ascending baroreceptor-cortical signaling.

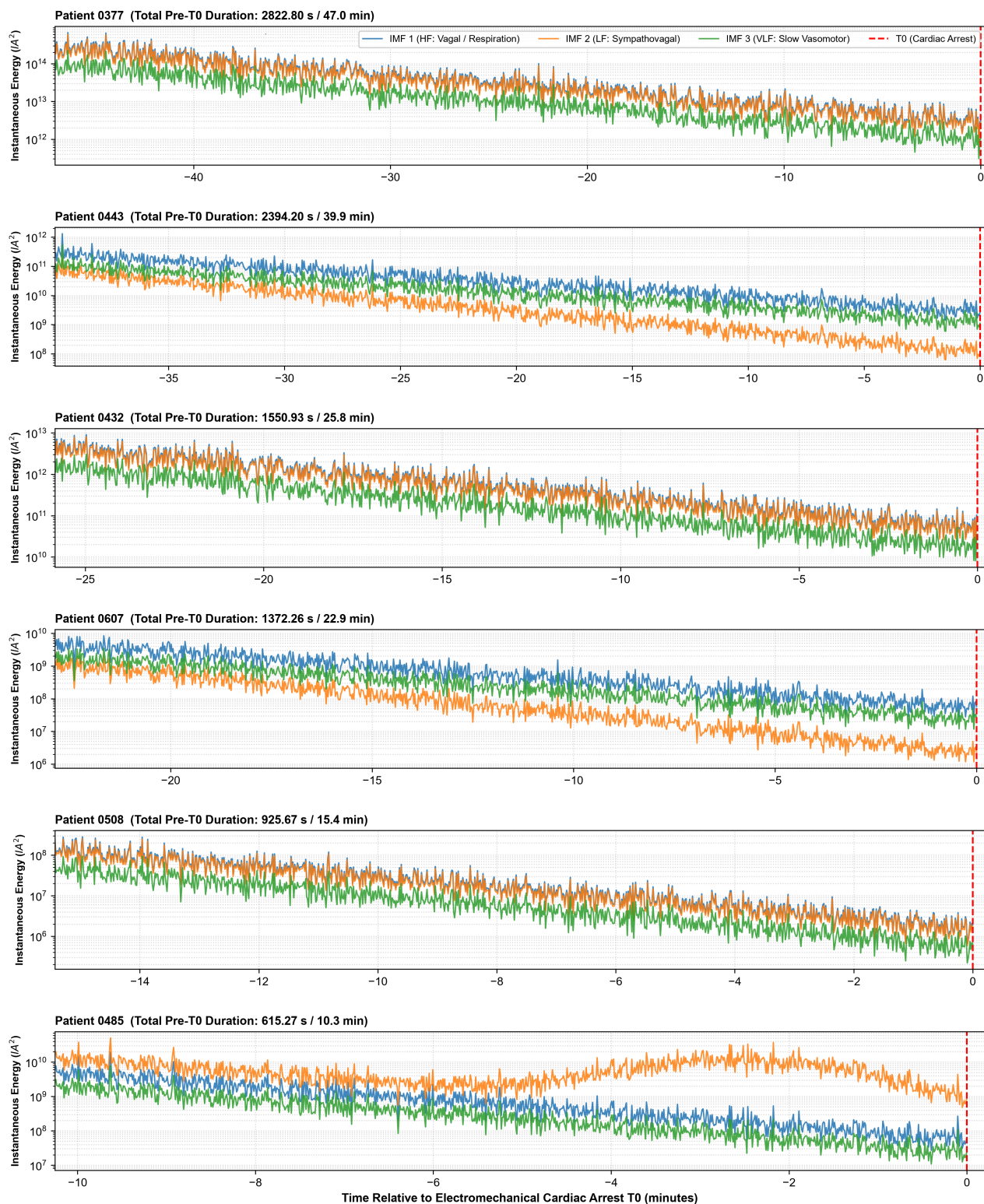


Figure 5

Long-range HHT spectral dynamics across active recording segments. Complete time-course of instantaneous Hilbert energy across all decomposed intrinsic modes (IMF1–3) for each patient from baseline start to final electromechanical cardiac arrest (T0), demonstrating continuous multiscale autonomic exhaustion across extended recording windows.

Supplementary Files

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