AUTONOMIC INTERPRETATION ENGINE (AIE™) CLINICAL ANALYSIS

1. Domain Integrity Analysis

- Cardiovagal (parasympathetic) integrity:
    - Resting HRV shows reduced time-domain variability (SDNN 25.62 ms with reference >40; pNN50 1%).
    - Respiratory sinus arrhythmia during deep breathing appears preserved by ratio criterion (E/I ratio 1.33 with reference >1.2) with higher variability (SDNN 68.89 ms).
    - Orthostatic cardiovagal timing index is reduced (K30/15 ratio 1.03 with reference >1.1).
- Adrenergic (sympathetic/vascular) integrity:
    - Resting SNS Power is slightly below the stated reference band (SNS Power 0.875 bpm²/Hz with reference 0.9–3.5) with SNS/PNS 0.742 (reference 0.6–1.3).
    - Valsalva global ratios meet stated adequacy thresholds (Valsalva ratio 1.25 with reference >1.2; BP ratio 1.34 with reference >1.1).
    - Valsalva SNS activation index is at the high/borderline-high range by provided device values (xSNS 7.27 with norms x2–7; SNS Power 6.362 bpm²/Hz with reference 1.75–6.12; SNS/PNS 1.95).
    - Standing blood pressure values provided (e.g., 109/79 mmHg) do not by themselves characterize dynamic vascular tone; beat-to-beat BP recovery dynamics are not numerically provided.
    - Valsalva Phase Dynamics (Qualitative, AIE-Derived)
        - Phase I: A brief blood pressure upstroke is visible on the plot with a concurrent modest heart-rate deflection. Waveform-derived (qualitative).
        - Phase II (late): A compensatory blood pressure recovery is visible on the waveform with relative heart-rate elevation during the strain interval. Waveform-derived (qualitative).
        - Phase IV: An overshoot/rebound in blood pressure is visible on the plot after release with heart-rate returning downward toward baseline. Waveform-derived (qualitative).
- Sudomotor integrity (sympathetic cholinergic):
    - Electrochemical skin conductance-type response is reduced in hands (Hands bioelectrical conductivity response 28.31 µS with reference >50) with normal hand asymmetry (0% with reference <20).
    - Feet show borderline/high asymmetry (Feet asymmetry 22% with reference <20) with feet conductivity response meeting the stated threshold (52.33 µS with reference >50).
    - QSART-type reflex output is regionally reduced in feet (Feet sweat activity reflex 46.01 with reference >50) with preserved hand reflex (58.14 with reference >50) and a preserved combined metric (Sudomotor Axon Reflex 52.95 with reference >50).

    - Device composite “risk” indicator is above the stated normal boundary (Risk for sudomotor autonomic neuropathy 57.66 with reference <50), representing a device-level aggregate flag rather than a physiologic diagnosis.
- Baseline cardiopulmonary vitals (context for testing):
    - Resting HR 80.15 bpm; SpO2 97%.
    - Resting BP 123/89 mmHg; deep breathing BP 108/84 mmHg; Valsalva BP 117/89 mmHg; standing BP 109/79 mmHg.

2. Cross-Domain Interaction Analysis

- Task-dependent dissociation is present: cardiovagal modulation is preserved during paced deep breathing (E/I 1.33; SDNN 68.89 ms) while resting and standing variability indices are reduced (resting SDNN 25.62 ms; standing SDNN 23.53 ms; K30/15 1.03).
- Adrenergic reactivity appears stronger during Valsalva strain/release than at baseline by provided sympathetic indices (Valsalva SNS Power 6.362 bpm²/Hz; xSNS 7.27) with baseline SNS Power slightly low (0.875 bpm²/Hz).
- Sudomotor findings are regionally heterogeneous: hands show low conductivity response while QSART hand reflex is preserved; feet show reduced QSART reflex with elevated asymmetry on conductivity metrics, suggesting non-uniform peripheral autonomic effector performance across sites.

3. Mechanistic Failure-Mode Classification

- parasympathetic_failure
    - Supported by reduced resting HRV time-domain metrics (SDNN 25.62 ms; pNN50 1%) and reduced orthostatic cardiovagal timing index (K30/15 1.03), with preservation during paced breathing suggesting state-dependent limitation rather than uniform absence.
- baroreflex_timing_dysfunction
    - Supported by reduced K30/15 ratio (1.03 with reference >1.1), indicating impaired/lagged short-latency vagal modulation during postural transition.
- sympathetic_underactivation
    - Supported at baseline only by slightly low resting SNS Power (0.875 bpm²/Hz with reference 0.9–3.5) despite normal resting SNS/PNS ratio (0.742).
- sympathetic_overactivation
    - Supported during Valsalva by elevated/borderline-high sympathetic indices (xSNS 7.27 vs norms x2–7; SNS Power 6.362 bpm²/Hz vs reference 1.75–6.12) with high LF/HF reported in the device summary (LF/HF 30.67).
- peripheral_autonomic_neuropathy
    - Mechanistically suggested by distal sudomotor reduction (feet QSART reflex 46.01 with reference >50) and elevated device risk flag (57.66 with reference <50), with preserved global QSART aggregate (52.95) indicating partial/patchy involvement rather than diffuse failure.

- central_autonomic_dysregulation
    - Considered (not confirmed) due to task-dependent variability (rest vs paced breathing vs standing) without uniformly concordant impairment across all domains.

4. Compensation vs. Failure Analysis

- Preserved/compensated elements:
    - Cardiovagal reserve can be recruited under paced breathing (E/I ratio 1.33; SDNN 68.89 ms), indicating preserved efferent vagal responsiveness in at least one standardized context.
    - Global Valsalva adequacy by ratios is preserved (Valsalva ratio 1.25; BP ratio 1.34), consistent with maintained baroreflex-mediated responsiveness during strain/release as a whole.
- Limited/failed elements:
    - Resting autonomic flexibility is constrained (resting SDNN 25.62 ms; pNN50 1%), suggesting reduced baseline variability.
    - Orthostatic short-latency vagal timing is reduced (K30/15 1.03), indicating limited rapid cardiovagal reflex adjustment on standing.
    - Sudomotor output is regionally limited (hands conductivity response 28.31 μS; feet sweat activity reflex 46.01) with site-to-site heterogeneity.

5. Phenotype Synthesis

- Primary phenotype
    - State-dependent reduced cardiovagal reserve with orthostatic timing limitation: preserved paced-breathing cardiovagal modulation (E/I 1.33) alongside reduced resting/standing variability (resting SDNN 25.62 ms; standing SDNN 23.53 ms) and reduced K30/15 (1.03).
- Secondary phenotype
    - Regional sudomotor heterogeneity with distal reduction: low hands conductivity response (28.31 μS) with reduced feet QSART reflex (46.01) and elevated feet asymmetry (22%), despite preserved aggregate QSART metric (52.95).

6. Autonomic Pattern Recognition (Non-Diagnostic)

- Reduced Autonomic Reserve Pattern
- Baroreflex Timing Dysfunction Pattern
- Task-Dependent Sympathetic Dysregulation Pattern
- Regional Autonomic Heterogeneity Pattern
- Peripheral Autonomic Neuropathic Pattern
- Mixed Autonomic Dysregulation Pattern

7. Mechanism-Directed Strategy Mapping

- Mechanistic framing only:
    - If symptoms are orthostatic, focus on strategies that improve baroreflex loading and reduce rapid postural hemodynamic transients (e.g., slower posture transitions; mechanical counter-pressure concepts), recognizing that the measured K30/15 ratio suggests limited rapid cardiovagal timing.
    - If physiologic stress reactivity is a concern, emphasize approaches that reduce excessive strain-phase sympathetic surges and improve autonomic flexibility (consistent with Valsalva sympathetic indices reaching borderline-high range).
    - For regional sudomotor limitation, prioritize strategies that reduce thermal/heat load and support peripheral heat dissipation efficiency, given reduced sweat reflex in feet and reduced hand conductance response.
    - Consider reconciliation of baseline-state factors that can suppress resting HRV (sleep quality, acute stress, testing environment, stimulants) as mechanistic confounders, since paced breathing performance is preserved while baseline is reduced.

8. Uncertainty Statement

- Missing data (limits physiologic specificity):
    - No explicit beat-to-beat standing BP time-series or orthostatic BP nadir/recovery metrics; only discrete standing BP values are provided.
    - No explicit Valsalva phase amplitude metrics (e.g., phase II drop magnitude, phase IV overshoot magnitude); qualitative waveform is visible but not numerically labeled for phases.
    - No symptom report, medication list, caffeine/nicotine status, hydration status at time of test, or temperature/skin preparation conditions for sudomotor testing.
    - No explicit respiratory rate/target adherence data beyond test labels (deep breathing, Valsalva).
- Ambiguous or unmappable elements (not interpreted beyond listing):
    - "X 749 ms / 764 ms / 756 ms / 645 ms" is not explicitly defined in the provided text (e.g., mean RR vs another timing metric) and is not used for mechanistic conclusions.
    - "Hydration 81.06 (55–70)" and concurrent low total body water percentage (56.9% with reference 59–69) are directionally discordant across listed BIA indices without clarifying definitions; no mechanistic conclusions are drawn from BIA hydration fields.
- Confounding limitations:
    - Artifact counts are low (generally 0–1) but still present in several segments; potential minor influence on HRV metrics cannot be excluded.
    - Sudomotor measures show cross-modality discordance (e.g., low hand conductivity response with preserved hand QSART reflex), which can reflect measurement-context differences rather than a single unified effector deficit.

9. Summary Statement

- Mechanistic summary:
    - The data show reduced baseline and standing HRV (resting SDNN 25.62 ms; standing SDNN 23.53 ms) with preserved paced-breathing cardiovagal modulation (E/I ratio 1.33) and preserved global Valsalva ratios (Valsalva ratio 1.25; BP ratio 1.34), alongside regionally heterogeneous sudomotor findings (hands conductivity response 28.31 μS; feet sweat activity reflex 46.01).
- Overall physiologic status:
    - Overall, the dominant physiology suggests constrained resting autonomic flexibility with task-dependent recruitment and a reduced rapid cardiovagal timing response to standing. In simpler terms, resting/standing regulation looks less adaptive, but the system can still respond appropriately during paced breathing and Valsalva.
- What is functioning well:
    - Cardiovagal responsiveness during paced deep breathing and global Valsalva ratio adequacy are preserved (E/I 1.33; Valsalva ratio 1.25; BP ratio 1.34).
- What is limited:
    - Resting and orthostatic autonomic variability/timing are limited (resting SDNN 25.62 ms; K30/15 1.03) with evidence of regional sudomotor reduction (feet sweat activity reflex 46.01; hands conductivity response 28.31 μS).
- Dominant Physiology Translation (Non-Diagnostic):
    - AIE™ Translation Mapping (clinician interpretive aid): This pattern of reduced baseline autonomic reserve with orthostatic cardiovagal timing limitation and regional sudomotor heterogeneity is most commonly associated, in appropriate clinical contexts, with orthostatic intolerance physiology, stress/reactivity physiology, and patchy peripheral autonomic effector impairment categories.

This analysis provides clinical decision support only. It does not establish diagnoses, prescribe treatments, or replace licensed clinician judgment. Please correlate these mechanistic patterns with the patient's symptoms, medications, and examination findings in consultation with the treating clinician for individualized guidance.