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# Breath–Gut Synchrony: A Default Space Theory Framework for Irritable Bowel Syndrome

**Ravinder Jerath[1*] and Varsha Malani[2]**
[1]Charitable Medical Organization, Mind-Body and Technology Research, Augusta, GA, USA
[2]Masters Student Northeastern University, Boston, MA, USA

[*]**Corresponding author:** Ravinder Jerath, Professor in the pain diploma program Central University of Venezuela

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## Abstract

Irritable bowel syndrome (IBS) is a prevalent disorder of gut–brain interaction marked by abdominal pain, bloating, and altered bowel habits, with substantial psychosocial and healthcare impact. Contemporary models implicate bidirectional CNS–gut communication mediated by autonomic, neuroendocrine, and immune pathways, alongside visceral hypersensitivity and stress reactivity. This article advances a unifying hypothesis: stress-induced alterations in breathing patterns entrain gastrointestinal activity via vagal pathways, establishing a self-reinforcing "breath–gut synchrony" loop that drives maladaptive neuroplasticity in pain and emotion circuits. Anchored in Default Space Theory (DST), the framework posits IBS as a breath-modulated visceral dysrhythmia embedded within an altered internal neural space integrating interoceptive signals with self-in-space representation. We synthesize evidence on autonomic imbalance and reduced vagal tone in IBS, brain–gastric phase coupling, and respiratory modulation of HRV, and derive testable predictions and clinical strategies prioritizing slow, deep breathing (SDB) and HRV-biofeedback as foundational therapies. This perspective reconciles peripheral and central mechanisms, proposes mechanistic biomarkers, and outlines research methods to validate the model, with implications for precision non-pharmacologic care in IBS.

## Keywords

Breath–Gut Synchrony; Irritable Bowel Syndrome; Neuroendocrine; Immune pathways.

## Introduction

IBS affects roughly 5–10% of the global population and is defined by recurrent abdominal pain associated with defecation and changes in stool frequency or form in the absence of structural disease.

Neurogastroenterology has shifted from a bowel-centric view to a gut–brain interaction framework that integrates autonomic dysregulation, HPA axis activation, immune signaling, and psychosocial factors that modulate symptom expression and chronicity. A key convergence is autonomic imbalance—relative sympathetic overactivity with parasympathetic (vagal) withdrawal—linked to visceral hypersensitivity, anxiety, and dysmotility in IBS. Respiration is a central gate for autonomic control: under psychological stress, breathing becomes rapid, shallow, and thoracic, reducing respiratory sinus arrhythmia (RSA) and vagal afferent signaling, thereby sustaining sympathetic dominance and heightened gut sensitivity. Building on these observations and evidence that gastric slow waves phase-couple with distributed brain networks, this perspective proposes that stress-breathing changes entrain gastrointestinal excitability through the vagus, creating a self-sustaining breath–gut loop that consolidates maladaptive central sensitization in IBS. Within DST, these coherent but pathological interoceptive rhythms distort the internal spatial representation of self, biasing perception toward pain and threat and providing a mechanistic basis for symptom persistence.

## Conceptual Framework

- Breath–gut synchrony: stress perturbs breathing mechanics (rate increase, shallow thoracic pattern), weakening vagal afferent bursts and lowering HRV, which reduces parasympathetic gating of visceral input and promotes sympathetic drive to the gut, amplifying motility dysrhythmia and nociception.
- Vagal pathways: deep inhalation activates pulmonary stretch receptors, increasing vagal afferent traffic to the NTS and higher centers, enhancing parasympathetic tone and anti-inflammatory cholinergic signaling; shallow rapid breathing yields the opposite profile and favors dysrhythmia and hypersensitivity.
- Brain–gastric coupling: gastric pacemaker activity (~0.05 Hz) can synchronize with cortical networks; vagal imbalance disrupts this coupling, increasing noisy afferent inflow and central gain on visceral signals, reinforcing hypervigilance and pain.
- DST embedding: persistent dysregulated respiratory–visceral rhythms become the dominant interoceptive scaffold of the default internal space, altering thalamocortical dynamics and salience attribution, thereby stabilizing IBS as a disorder of internal state rather than isolated bowel dysfunction.

## Methods (Proposed Research Program)

Because this is a Perspective with testable hypotheses, the following multimodal program is proposed to validate breath–gut synchrony:

### 1 Cross-sectional physiology

- Concurrent polysomnography-grade respiration, ECG-derived HRV, and high-resolution electrogastrography (EGG) in IBS and healthy controls at rest and during validated stressors, with rectal distension to map cardio-respiratory–visceral coupling and vagal indices.
- Primary outcomes: HF-HRV (vagal tone), RSA amplitude, respiratory rate/variability, EGG dominant frequency stability, gastric–cardiac coherence, and symptom provocation scores.

### 2. Neuroimaging of phase coupling


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- Simultaneous fMRI with respiratory bellows, ECG, and EGG to quantify respiratory–gastric–brain phase-amplitude coupling and connectivity within insula, ACC, amygdala, thalamus, and DMN nodes, contrasting IBS vs controls.
- Metrics: coherence, Granger causality, and dynamic functional connectivity states related to respiratory phase and gastric cycle.

3. **Interventional trials**
   - Randomized controlled trials of slow, deep breathing (5–6 breaths/min, diaphragmatic) with or without HRV biofeedback versus sham breathing/education; adjunct arms with taVNS to probe vagal specificity.
   - Endpoints: abdominal pain severity, IBS-SSS, stool metrics, HF-HRV, RSA, EGG stability, fMRI coupling, rectal pain thresholds; durability at 12–24 weeks.
4. **Mechanistic probes**
   - Pharmacologic autonomic blockade (e.g., low-dose propranolol) and posture manipulations to modulate respiratory mechanics and venous return, testing causal links among breathing, vagal tone, and visceral sensation.
   - Exploratory biomarkers: baroreflex sensitivity, inflammatory cytokines, and vagal-mediated anti-inflammatory signatures.

## Results (Anticipated Patterns and Existing Evidence)

- **Autonomic profile:** meta-analytic evidence indicates lower HF-HRV in IBS, consistent with reduced vagal tone and sympathetic predominance, predicting greater symptom severity during stress tasks and rectal distension in IBS cohorts.
- **Respiratory mechanics:** stress-related rapid, shallow breathing is expected to correlate with lower RSA and greater pain ratings, whereas paced SDB should increase HF-HRV, improve baroreflex gain, and raise visceral pain thresholds.
- **Brain–gut coupling:** fMRI–EGG studies should reveal disrupted gastric–cortical synchrony in IBS that normalizes with SDB/HRV biofeedback, particularly within interoceptive and salience hubs (anterior insula, ACC), aligning with reduced hypervigilance and pain.
- **Clinical response:** pilot data in functional GI populations suggest SDB and HRV biofeedback improve pain and rectal sensitivity; taVNS studies report motility and pain benefits, supporting vagal mechanisms consonant with the model.

### Clinical conditions and applications

- **Phenotyping:** incorporate respiration (rate, variability), HF-HRV, and simple EGG into IBS assessments to stratify patients by autonomic–respiratory phenotype, guiding selection for breathing-centric interventions.
- **First-line autonomic therapy:** prescribe SDB (5–6 breaths/min; 10–20 minutes, twice daily) with diaphragmatic emphasis, extended exhalation, and HRV biofeedback where feasible; integrate with psychoeducation, CBT for stress, and graded exposure to reduce interoceptive threat.

- **Adjunctive vagal strategies:** taVNS, yoga/pranayama, mindfulness-based stress reduction, and sleep optimization to raise vagal tone and stabilize breath–gut coupling, alongside diet and pharmacotherapy tailored to bowel habit subtype.
- **Monitoring:** track HF-HRV, RSA, and symptom diaries; consider home wearable HRV and breathing-guided apps; escalate to neuromodulation when biomarkers fail to improve.

## Discussion

This framework reframes IBS as a systems-level oscillopathy wherein breathing is a controllable pacemaker for gut–brain coherence, synthesizing competing emphases on peripheral motility and central sensitization. By embedding interoceptive rhythms within DST, the model accounts for why symptoms persist beyond local gut pathology, explains variability across stress states and posture, and clarifies how non-pharmacologic autonomic interventions can produce meaningful relief. It yields concrete biomarkers (HF-HRV, RSA, respiratory–gastric coherence) and neuroimaging endpoints (respiration–EGG–fMRI coupling) that can accelerate mechanistic trials and precision therapy. Importantly, it balances correlation and causation: while extant data support each link (stress → breathing → autonomic shift → gut dysrhythmia → central gain), definitive tests require synchronized multimodal recordings and targeted respiratory–vagal interventions in randomized designs. Limitations include heterogeneity of IBS phenotypes, variability in HRV measurement standards, and the need to disentangle direct respiratory effects from generalized relaxation; sham-controlled breathing and phase-specific analyses are therefore essential. The model complements, rather than replaces, microbiome and immune hypotheses by specifying a rapid, reversible control axis that may modulate inflammatory tone and motility through vagal efferents and baroreflex pathways.

### Future directions

- **Mechanistic RCTs:** large, multi-center trials comparing SDB, HRV biofeedback, and taVNS versus sham, with synchronized respiration–HRV–EGG–fMRI endpoints and rectal sensitivity assays to establish causality and dose–response.
- **Biomarker validation:** standardize HF-HRV, RSA, baroreflex measures, and EGG coherence as clinical biomarkers; develop portable sensors for home monitoring and adaptive dosing of breathing regimens.
- **Neurocomputational models:** simulate coupled oscillators (respiration, cardiac, gastric) interacting with thalamocortical loops to predict symptom dynamics and personalize breathing frequencies (resonance breathing).
- **Phenotype-specific protocols:** tailor breathing cadence and duration to IBS subtypes (IBS-C vs IBS-D), circadian patterns, and comorbid anxiety or insomnia; test synergy with diet (low-FODMAP), psychotherapies, and standard medications.
- **Translational links:** extend the framework to related DGBIs and chronic pain syndromes sharing autonomic and interoceptive dysregulation to explore generalizability and shared therapeutic levers.

## Conclusion

IBS can be coherently understood as a breath-modulated visceral dysrhythmia embedded in an altered interoceptive default space, generated by stress-related shifts in breathing that downregulate vagal tone and disrupt gastric–brain synchrony, thereby amplifying pain and dysmotility in a self-reinforcing loop. This systems view reconciles peripheral and central mechanisms, yields measurable biomarkers, and prioritizes slow, deep breathing and HRV-biofeedback as rational, scalable first-line interventions to restore autonomic balance and desynchronize maladaptive loops.

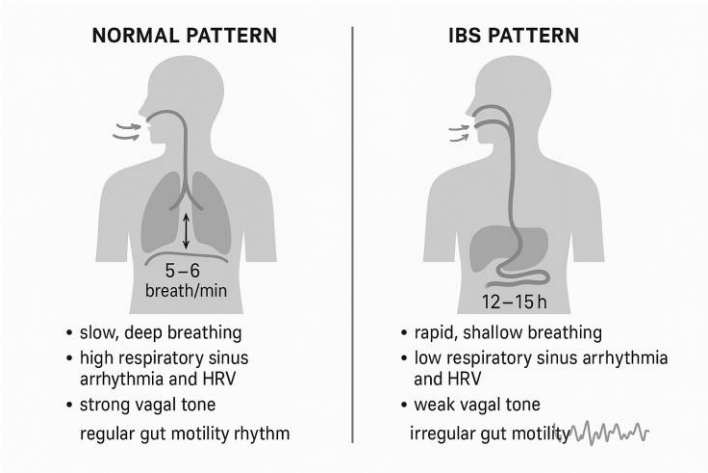


**Figure 1:** Comparison of normal and IBS patterns showing differences in breathing rate, vagal tone, HRV, and gut motility.

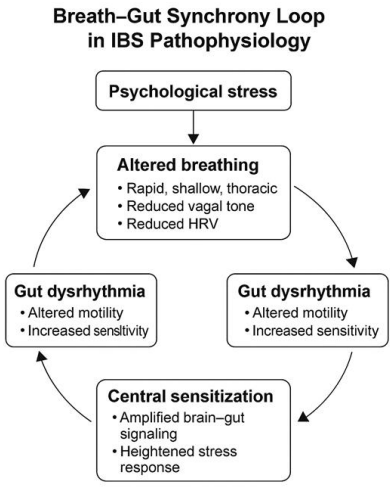


**Figure 2:** Breath–gut synchrony loop in IBS showing how psychological stress, altered breathing, gut dysrhythmia, and central sensitization interact to perpetuate symptoms.

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