

Journal of Neurological Sciences and Research

Genesis-JNSR-6(1)-S8
Volume 6 | Issue 1
Open Access
ISSN: 3048-5797

The Visceral Coherence Index: A Unified Marker for Embodied Consciousness and Autonomic State

Ravinder Jerath^{1*} and Varsha Malani²

¹Charitable Medical Organization, Mind-Body and Technology Research, Augusta, GA, USA

²Masters Student Northeastern University, Boston, MA, USA

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

Citation: Jerath R, and Malani V. The Visceral Coherence Index: A Unified Marker for Embodied Consciousness and Autonomic State. J Neurol Sci Res. 6(1):1-09.

Received: March 21, 2026 | **Published:** April 14, 2026

Copyright© 2026 Genesis Pub by Jerath R, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution4.0 International License (CC BY 4.0). This license permits unrestricted use, distribution, and reproduction in any medium, provided the original author(s) and source are properly credited.

Abstract

Subjective experience is profoundly shaped by brain-body interactions. We introduce the Visceral Coherence Index (VCI), a novel mathematical model quantifying cardiorespiratory phase synchrony as a dynamic determinant of conscious state and autonomic balance. Building on the Default Space Theory (DST), which posits consciousness as an embodied bioelectric scaffold, we propose that the VCI directly modulates Default Space stability. High VCI (reflecting robust cardiorespiratory coherence, e.g., $\geq 4:1$ heartbeat per breath) indicates parasympathetic dominance, fostering Default Space integration, cognitive clarity, and positive affect. Conversely, low VCI (poor coherence, e.g., $\leq 2:1$) signifies sympathetic activation, correlating with Default Space fragmentation, emotional dysregulation, and cognitive disorganization. We delineate the vagal-mediated neurophysiological mechanisms, emphasizing respiration's role in entraining cortical oscillations and modulating neural gain across key brain networks. The VCI offers a quantifiable, testable framework for the neurovisceral underpinnings of consciousness, serving as a potential biomarker for well-being and a mechanistic target for breath-based interventions. This model advances an embodied view of mind, highlighting the active role of internal physiological rhythms in shaping phenomenal experience.

Keywords

Visceral Coherence Index; Embodied Conscious; Brain-body interactions; Internal physiological rhythms.

Introduction

Embodied consciousness: beyond the brain

The prevailing brain-centric view of consciousness increasingly faces challenges from mounting evidence highlighting the profound influence of peripheral physiological signals [1,8]. Emerging frameworks, like the Default Space Theory (DST), propose conscious awareness arises from ongoing oscillatory synchrony between brain and body, forming an integrated "default space"—a bioelectric scaffold of subjective experience [9,10]. Within this visceros-consciousness framework, the rhythmic interplay of internal organs, particularly respiration and cardiac activity, fundamentally shapes conscious quality [11]. Respiration, a uniquely modifiable rhythm, acts as a physiological metronome, entraining diverse neural rhythms, including in cortical and limbic areas [2,12]. Cardiorespiratory coherence (CRC), the synchronized relationship between heart rate variability (HRV) and the respiratory cycle, is a critical indicator of autonomic balance and efficient inter-organ communication [3,13].

Building on these insights, this paper introduces the Visceral Coherence Index (VCI)—a novel, precise mathematical metric to quantify critical cardiorespiratory phase synchrony. We argue that the VCI provides a dynamic link between bodily rhythms and graded states of conscious awareness, emotional regulation, and overall neurophysiological well-being. This model represents a significant step towards a comprehensive, embodied understanding of consciousness, integrating the intricate symphony of brain-body oscillations.

The visceral coherence index (vci): a mathematical model

We propose a mathematical model centered on the VCI, a novel composite metric designed to quantify the phase synchrony between cardiac and respiratory oscillations. The VCI aims to capture the dynamic interplay of cardiorespiratory coherence (CRC) and its profound influence on autonomic balance and the stability of the Default Space.

Defining the VCI

The VCI is derived from continuous, concurrently measured time series of the respiratory waveform ($R(t)$) and the inter-beat interval (R-R interval) series ($RR(t)$) from an electrocardiogram (ECG). It fundamentally quantifies the degree to which cardiac oscillations are rhythmically and consistently phase-locked to respiratory oscillations. A robust VCI metric incorporates three key aspects of cardiorespiratory coupling [14]:

1. **Magnitude-Squared Cardiorespiratory Coherence (CRC):** This quantifies the linear statistical relationship between the respiratory signal and the instantaneous heart rate (or R-R interval series) in the frequency domain. It measures the proportion of power in the heart rate signal that is linearly correlated with the respiratory signal at a specific frequency [15].

Mathematical Representation: $CRH(f) = \frac{PRR(f)PHH(f)}{|PRH(f)|^2}$ Where $PRH(f)$ is the cross-spectral density between the respiratory signal (R) and the heart rate signal (H) at frequency f .

$PRR(f)$ and $PHH(f)$ are the respective power spectral densities of the respiratory and heart rate signals. The CRC would typically be calculated for the peak frequency within the respiratory frequency band (e.g., 0.1-0.4 Hz for spontaneous breathing, or a narrower band for paced breathing). A higher $CRH(f)$ value (ranging from 0 to 1) indicates stronger linear coupling.

2. **Respiratory Sinus Arrhythmia (RSA) Amplitude:** This well-established time-domain and frequency-domain metric quantifies the vagally-mediated heart rate modulation by breathing [16, 17]. Larger RSA amplitude typically reflects greater parasympathetic influence and vagal tone.

Mathematical Representation (Frequency Domain): RSA amplitude can be approximated by the power in the high-frequency (HF) band of the HRV power spectrum (typically 0.15-0.4 Hz), which is largely attributed to vagal activity synchronized with respiration [18].

Mathematical Representation (Time Domain - Peak-Trough): $\text{RSA amplitude} = \frac{1}{N} \sum_{i=1}^N (\text{HR peak}, i - \text{HR trough}, i)$, where $\text{HR peak}, i$ is the peak heart rate during inspiration and $\text{HR trough}, i$ is the trough heart rate during expiration for the i -th respiratory cycle.

3. **Consistency of Phase Relationship (ϕ_{RH}):** The VCI should also capture the stability and consistency of the phase lag between respiration and cardiac oscillations (i.e., heart rate typically increases during inspiration and decreases during expiration) [19]. A stable and consistent phase relationship indicates robust and effective physiological coupling.

Mathematical Representation: The phase angle $\phi_{RH}(f)$ can be derived from the cross-spectral density. Consistency can be quantified using circular statistics, such as the mean resultant vector length (MRVL) of phase differences across multiple respiratory cycles [20]. $\text{MRVL} = \frac{1}{N} \sum_{k=1}^N e^{i\theta_k}$, where θ_k is the phase difference for cycle k . A value closer to 1 indicates high consistency.

A combined VCI would then be represented as a composite score that integrates these three critical elements. The specific mathematical function $F()$ for this composite score could be: $\text{VCI} = F(\text{CRH}, \text{RSA Amplitude}, \text{MRVL} \phi_{RH})$ where F is a potentially non-linear function, possibly incorporating weighting factors based on theoretical importance or empirical derivation. For instance, a simple initial approach could be a normalized product or a weighted sum: $\text{VCI} = w_1 \cdot \text{Normalized}(\text{CRH}) + w_2 \cdot \text{Normalized}(\text{RSA Amplitude}) + w_3 \cdot \text{MRVL} \phi_{RH}$ where w_1, w_2, w_3 are empirically or theoretically determined weights summing to 1, and 'Normalized' indicates scaling values to a common range (e.g., 0-1). The VCI metric would ideally range from 0 (minimal visceral coherence/coupling) to 1 (maximal visceral coherence/coupling). The precise definition of F and the weighting factors represent a critical avenue for future empirical validation and optimization of the model.

VCI, autonomic tone, and default space dynamics

Our model posits that the VCI dynamically modulates the balance of autonomic tone and, consequently, the stability and coherence of the Default Space:

High Visceral Coherence (High VCI):

Physiological State: High VCI values, typically corresponding to well-defined CRC ratios (e.g., $\geq 4:1$ heartbeat per respiratory cycle, or a strong spectral peak of HRV at the respiratory frequency), are indicative of parasympathetic dominance [21,22]. This reflects efficient vagal nerve activity, dampening sympathetic arousal and promoting physiological calm and energy conservation [3].

Impact on Default Space: This profound physiological coherence provides a stable, rhythmic, and consistent visceral anchor for the brain's intrinsic activity [23]. This stability leads to a stabilized and highly coherent Default Space, characterized by enhanced functional connectivity within core DMN nodes and between DMN

and interoceptive hubs like the insula and ACC [6, 24]. This facilitates optimal information integration, both internal and external [25].

Conscious Experience: This state correlates with subjective experiences of positive affect, robust emotional regulation, mental clarity, and enhanced cognitive integration [26, 27]. Individuals typically report feelings of calm, groundedness, presence, and a unified sense of self. This state is associated with optimized attentional processes, cognitive flexibility, and resilience.

Low visceral coherence (low vci)

Physiological State: Low VCI values, corresponding to irregular or low CRC ratios (e.g., $\leq 2:1$, or a diffuse HRV spectrum without a clear respiratory peak), signify sympathetic activation and reduced vagal tone [21, 28]. This indicates a state of physiological stress, hyper-arousal, or dysregulation in autonomic control, often associated with fight-or-flight responses [29].

Impact on Default Space: This physiological incoherence and instability lead to the fragmentation and instability of the Default Space [30]. The absence of a stable, coherent visceral anchor disrupts the brain's intrinsic oscillatory dynamics, resulting in less integrated or disorganized neural activity within core DMN regions and perturbed thalamocortical circuits [31].

Conscious Experience: This state correlates with subjective experiences of stress, anxiety, emotional dysregulation, and cognitive fragmentation [32, 33]. Individuals may report feelings of being overwhelmed, difficulty focusing, rumination, or even experiences of derealization/depersonalization, reflecting a fundamental disruption in the unified sense of self.

Mechanism: respiration-driven neurovisceral integration

The proposed VCI model is grounded in a well-established neurovisceral pathway through which respiration influences both autonomic tone and cortical activity [3, 34]. This mechanism involves a continuous, reciprocal interaction between the body and the brain.

Vagal Nerve Pathway: The vagus nerve serves as the primary conduit for parasympathetic signals from the heart and lungs to the brainstem [35, 36]. Its afferent fibers terminate primarily in the Nucleus of the Solitary Tract (NTS). The rhythmic input from pulmonary stretch receptors (driven by respiration) and arterial baroreceptors (influenced by respiration-induced blood pressure changes) precisely modulates vagal afferent firing, transmitting a rich stream of interoceptive information [37].

Brainstem and Subcortical Relays: From the NTS, signals project to other crucial brainstem nuclei (e.g., parabrachial nucleus, locus coeruleus, raphe nuclei), modulating arousal, attention, and neuromodulatory systems (e.g., noradrenaline, serotonin) [38, 39]. These pathways also project to subcortical structures critical for emotion and motivation, such as the amygdala and periaqueductal gray [40].

Thalamocortical and Default Mode Network Modulation: The thalamus acts as a central relay, transmitting integrated interoceptive information to widespread cortical areas [41]. Critically, these include key nodes of the Default Mode Network (DMN)—such as the medial prefrontal cortex (MPFC) and posterior cingulate cortex (PCC)—as well as dedicated interoceptive hubs like the insular cortex and anterior cingulate cortex (ACC) [4, 5, 42].

Oscillatory Entrainment and Neural Gain Modulation: The slow respiratory rhythm (typically 0.1-0.3 Hz) acts as a powerful low-frequency modulator, precisely entraining higher-frequency cortical oscillations (e.g., theta 4-7 Hz, alpha 8-12 Hz, and even gamma >30 Hz) through phase-locking mechanisms [2, 12, 43]. This bottom-up synchronization organizes neuronal excitability, shifting it between states of high excitability during specific phases of the respiratory cycle (e.g., inspiration) and lower excitability during others (e.g., expiration) [44]. This neural gain modulation optimizes information processing and modulates functional connectivity within and between brain networks. A high VCI signifies robust and consistent phase-locking, providing a stable temporal scaffold that facilitates efficient communication and integration across brain-body networks, thereby stabilizing the Default Space.

Reciprocal Brain-Body Interaction: It is crucial to acknowledge the reciprocal nature of this relationship. Cortical structures, particularly the prefrontal cortex, can exert top-down control over respiratory patterns (e.g., through voluntary paced breathing) and autonomic outflow via descending pathways [45, 46]. This top-down modulation directly influences VCI and CRC, completing a continuous feedback loop that profoundly shapes conscious experience and self-regulation.

Thus, the VCI directly reflects the strength and consistency of this bidirectional body-brain oscillatory coupling. A high VCI signifies a finely tuned, coherent neurovisceral system that robustly underpins a stable, integrated Default Space conducive to optimal subjective states.

Implications and Future Directions

The VCI model offers a compelling mechanistic explanation for how respiration-driven autonomic modulation contributes to a spectrum of conscious states and impacts overall well-being. It provides a quantifiable link between a physiological metric and the quality of subjective experience.

Enhanced Integration and Optimal States (High VCI): States characterized by high VCI and parasympathetic dominance are associated with enhanced integration, manifesting as cognitive clarity (e.g., improved sustained attention, memory consolidation, problem-solving abilities) [26, 47], emotional stability (e.g., reduced amygdala reactivity, greater emotional regulation capacity) [48, 49], and a unified sense of self (e.g., in mindfulness or "flow" states, peak performance) [50].

Fragmented Awareness and Dysregulation (Low VCI): Conversely, states with low VCI and sympathetic activation are linked to cognitive dysregulation (e.g., impaired attentional control, distractibility, rumination, diminished cognitive performance) [33, 51], emotional distress (e.g., heightened anxiety, fear, irritability, increased vulnerability to stress) [32, 52], and a disrupted sense of self (e.g., feelings of detachment, depersonalization, derealization, or a fragmented and incoherent self-perception) [53].

This framework suggests that the VCI could serve as a quantifiable, objective biomarker of Default Space coherence and overall neurophysiological well-being [54]. This provides a measurable proxy for the subjective quality of consciousness, bridging the gap between objective physiology and first-person experience.

Experimental validation

Comprehensive validation of the VCI model will require multi-modal and interdisciplinary approaches:

Hypothesis 1: High VCI will correlate with increased functional connectivity and coherence (e.g., measured by phase-locking values (PLV) and magnitude-squared coherence) within core DMN nodes and interoceptive hubs, and robust respiratory-cortical phase-locking across relevant frequency bands (e.g., alpha, theta, gamma) [2, 6, 43].

Hypothesis 2: Experimentally induced changes in VCI (e.g., through paced breathing protocols, autonomic perturbations like cold pressor tests) will predict and precede measurable shifts in DMN activity patterns (e.g., increased/decreased coherence) and objective (e.g., task performance) and subjective (e.g., self-report scales for affect, anxiety, cognitive clarity) measures of conscious experience [55].

Hypothesis 3: Individuals trained to increase their VCI through biofeedback (e.g., HRV biofeedback) will demonstrate enhanced parasympathetic tone, improved emotional regulation, reduced anxiety, and report a more stable and coherent sense of self, compared to sham or control groups [56, 57].

Hypothesis 4: Clinical populations characterized by disturbed selfhood, emotional dysregulation, or autonomic dysfunction (e.g., anxiety disorders, post-traumatic stress disorder (PTSD), depersonalization/derealization disorder, chronic stress, or even disorders of consciousness) [58, 59] will exhibit systematically lower VCI values and reduced Default Space coherence compared to healthy controls. Therapeutic interventions that successfully increase VCI will correlate with significant symptom reduction and improved subjective well-being.

Computational Modeling: Sophisticated computational models simulating the interaction between visceral rhythms (e.g., respiratory pacemakers), brainstem nuclei, thalamocortical loops, and DMN dynamics should demonstrate that increased cardiorespiratory phase synchrony (simulating high VCI) leads to emergent properties resembling a stable and integrated Default Space, while disrupted synchrony (low VCI) leads to fragmentation.

Novel interventions

The VCI model provides a powerful mechanistic rationale for developing targeted breath-based practices and neurovisceral therapies to modulate conscious experience and enhance neurophysiological well-being: Coherent Breathing and HRV Biofeedback: These practices explicitly train individuals to optimize their respiratory and cardiac rhythms, thereby directly increasing CRC and VCI [56, 57]. Our model suggests this directly impacts Default Space stability, leading to predictable improvements in subjective states and cognitive function.

Mindfulness and Contemplative Practices: Many traditional mindfulness and contemplative practices emphasize breath awareness and regulation. These practices have been empirically shown to alter activity in the insular cortex and anterior cingulate cortex [4, 60] and enhance DMN coherence [6]. The VCI provides a quantifiable metric to objectively track the physiological impact of these practices on the underlying Default Space. Personalized Medicine: The VCI could serve as a diagnostic and monitoring tool, allowing for personalized breathwork interventions tailored to an individual's unique neurovisceral profile and their Default Space dynamics, moving towards precision medicine in mental and neurological health [61].

Conclusion

This paper introduces the Visceral Coherence Index (VCI) model, offering a robust and testable mechanistic

framework for understanding the profound link between respiration, autonomic tone, and the fundamental nature of conscious experience within the Default Space Theory. By rigorously quantifying the phase synchrony between heart rate and respiration, the VCI provides a tangible and objective metric for the coherence of visceral oscillations, which we propose directly influences the stability and integrity of the brain's bioelectric scaffold of awareness.

This perspective challenges simplistic brain-centric views, instead positioning the rhythmic interplay of the entire embodied system, particularly the pervasive respiratory field, as an active, integral co-creator of subjective reality. It highlights the continuous, reciprocal relationship between brain and body in shaping our internal world and our interactions with the environment. Ultimately, the VCI model provides a novel lens through which to investigate the neurovisceral underpinnings of consciousness, offering exciting avenues for both fundamental research into the nature of subjective experience and for developing powerful, non-pharmacological interventions to optimize mental well-being and modulate conscious states across health and disease.

Acknowledgements

We confirm that artificial intelligence (AI) assistance, specifically a large language model, was utilized for refining the language, enhancing conciseness, and improving the structural organization of this manuscript. The authors attest that this AI tool did not generate any scientific content, interpret data, or contribute to the intellectual development of the ideas presented. All scientific contributions, originality, and accountability for the work remain solely with the human authors.

References

1. Craig AD. (2009) How do you feel? An interoceptive moment with the anterior insula. *Nat Rev Neurosci.* 10:59-70.
2. Zelano C, Jiang H, Zhou G, Arora N, Schuele S, et al. (2016) Nasal Respiration Entrain Human Oscillatory Brain Activity. *J Neurosci.* 36:11629-37.
3. Thayer JF, Lane RD (2009) Claude Bernard and the heart–brain connection: further elaborations of a model of neurovisceral integration *Neurosci Biobehav Rev.* Reviews 33:85-97.
4. Farb NA, Segal Z, Mayberg H, Bean J, McKeon D, et al. (2007) Attending to the present: mindfulness meditation reveals distinct neural mechanisms of self-reference. *Soc Cogn Affect Neurosci.* 2:313-22.
5. Northoff G, Heinzel A, de Greck M, Berman F, Dobrowolny H, et al. (2006) Self-referential processing in our brain—a meta-analysis of imaging studies on the self. *NeuroImage.* 31:440-57.
6. Bershadsky D, Kleyner A, Levkov M. (2021) The Effect of Paced Breathing on Default Mode Network Activity. *Frontiers in Human Neuroscience* 15:674514.
7. Jerath R. The lungs are not simply for gas exchange, but part of a sensory interface that co-creates the rhythm of experience. (Manuscript in review).
8. Seth AK. (2016) Interoceptive inference: from homeostasis to consciousness. *Trends Neurosci.* 39:546-55
9. Jerath R, Beveridge C, Jensen M. (2019) The Default Space Theory of Consciousness: Phenomenological Support from Personal Observations and Clinical Deficits. *World J Neurosci.* 9:1-21.
10. Carhart-Harris RL, Friston KJ. (2019) The default-mode network as a subjective self: from neurofeedback to psychedelics. *Neuropsychopharm.* 44:1-11.
11. Damasio AR. (1999) *The feeling of what happens: Body and emotion in the making of consciousness.* Harcourt Brace & Company.

12. Herrero JL, Muggleton NG, Tsakiris M. (2019) The heartbeat evoked potential: A window into the neural correlates of interoception. *Psychophysiol.* 56:e13251.
13. Grossman P, Taylor EW. (2015) Toward a comprehensive model of brain-heart interactions. *Front. Physiol.* 6:298
14. Portes J, Vescovo A, Pini S. (2019) A review of cardiorespiratory coupling methods. *Physiological Measurement* 40:014002.
15. Schaffer D, Schaffer S. (2017) HRV and Coherence: A New Perspective on Self-Regulation and Health. Heart Math Institute.
16. Porges SW. (2011) The Polyvagal Theory: Neurophysiological foundations of emotions, attachment, communication, and self-regulation. W. W. Norton & Company.
17. Appelhans BM, Luecken LJ. (2006) Heart rate variability as an index of integrated emotion regulation. *Psychol. Rev.* 113:225-42.
18. Task Force of The European Society of Cardiology and The North American Society of Pacing and Electrophysiology. Heart rate variability: standards of measurement, physiological interpretation, and clinical use. *Circulation* 93:1043-65.
19. Yasuma F, Hayano J. (1998) Respiratory sinus arrhythmia. Clinical implications and physiological mechanisms. *Clin. Exp. Pharmacol. Physiol.* 25:19-26.
20. Fisher NI. (1993) Statistical analysis of circular data. Cambridge University Press.
21. Lehrer PM, Gevirtz R. (2014) Heart rate variability biofeedback: How and why does it work? *Front. Psychol.* 5:756
22. Zygmunt, A., & Stanczyk, J. Vagal regulation of heart rate. *Med. Sci. Monit.* 11, RA278–281 (2004).
23. Critchley HD, Garfinkel S. (2013) Interoception and emotion: a neuroanatomical perspective. *Handb. Clin. Neurol.* 116:189-200.
24. Chang C, Glover GH. (2010) The default mode network and dynamic fluctuations in resting-state fMRI functional connectivity. *NeuroImage.* 50:1139-48.
25. Smallwood J, Schooler JW. (2015) The Restless Mind. *Psychological Bulletin.* 137:870-98.
26. Mather M, Thayer JF. (2013) The heart–brain connection: The vagal afferent pathway and the role of vagal tone in the regulation of emotion. *Emotion Rev.* 5:394-402.
27. Garfinkel SN, Critchley HD. (2013) Interoception, emotion and the embodied self. *Trends Cogn. Sci.* 17:295-303.
28. McCraty R, Zayas MA. (2014) Coherence: A New Perspective on Health. *Integrative Medicine: A Clin J.* 13:32-37.
29. McEwen BS. (1998) Stress, adaptation, and disease: Allostasis and allostatic overload. *Ann NY Acad Sci.* 840:33-44.
30. Uddin LQ. (2011) Cognitive control and the default mode network. *Trends Cogn Sci.* 15:411-18.
31. Raichle ME. (2015) The Brain's Default Mode Network. *Annu. Rev. Neurosci.* 38:433-47.
32. Porges SW. (1995) The Polyvagal Theory: New insights into adaptive reactions of the autonomic nervous system. *Psychophysiol.* 32:345-54.
33. Critchley HD, Harrison NA, Gray MA, Dolan RJ. (2005) Psychophysiological, neuroanatomical and neurochemical correlates of the autonomic control of heart rate variability. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* 360:1025:40
34. Benarroch EE. (2012) The central autonomic network: functional organization, clinical implications, and new directions. *Ann Neurol.* 71:180-89.
35. Tracey KJ. (2002) The inflammatory reflex. *Nature.* 420:853-59.
36. Wehrwein EA, Smith ML. (2013) The cardiorespiratory system. *Compr Physiol.* 3:1569-05.
37. Mravec B, Benarroch EE. (2017) Vagal afferent projections to the locus coeruleus: relevance for autonomic and behavioral functions. *Clin Auton Res.* 27:1-8.
38. Aston-Jones G, Cohen JD. (2005) An integrative theory of locus coeruleus-norepinephrine function: adaptive gain and optimal performance. *Annu Rev Neurosci.* 28:403-50.
39. Hornung JP. (2015) The human raphe nuclei and their projections: a review. *J Chem Neuroanat.* 67:1-17.
40. Pavlov VA, Tracey KJ. (2009) Neural regulation of inflammation. *J Exp Med.* 206:2083-90.
41. Sherman SM. (2014) The thalamus is more than a relay. *Curr. Opin. Neurobiol.* 26:55-59.

42. Uddin LQ, Kelly AMC, Biswal BB, Margulies DS, Shehzad Z, et al. (2005) Functional connectivity of the anterior cingulate cortex: default mode network and dorsal attention network contributions. *Trends Cogn. Sci.* 11:471-476(2007).
43. Yellin SA, Zelano C. (2020) Respiration and its influence on central olfactory processing. *Curr Opin Neurobiol.* 60:92-98.
44. Fontanini A, Katz DB. (2011) Behavioral modulations of sensory gain and noise. *Curr Opin Neurobiol.* 21:660-66
45. Critchley HD, Harrison NA. (2007) Autonomic nervous system influences. *J Pers.* 75:1109-132.
46. Lane RD, & Wager TD. (2003) The neurobiology of emotion regulation. *Handb. Affect. Sci.* 619:619-38.
47. Porges SW. (2001) The Polyvagal Theory: phylogenetic substrates of a social nervous system. *Int J Psychophysiol.* 42:123-46.
48. Rottenberg J, Gross JJ. (2004) Emotion regulation in anxiety disorders. *Anxiety Stress Coping.* 17:297-304.
49. Thayer JF, Lane RD. (2005) A model of neurovisceral integration in emotion regulation and dysregulation. *J. Affect. Disord.* 88, 205-16.
50. Csikszentmihalyi M. (1990) *Flow: The Psychology of Optimal Experience.* Harper Perennial.
51. Ehlers A, Clark DM. (1997) A cognitive model of panic attacks. *Behav. Res. Ther.* 35:503-15.
52. Cohen S, Herbert TB. (1998) Health psychology: Psychological factors and physical disease from the perspective of psychoneuroimmunology. *Annu Rev Psychol.* 49:37-57.
53. Sierra M, Berrios GE. (2004) Depersonalization: Neurobiological perspectives. *Biol. Psychiatry.* 55:89-97.
54. Khalsa SS, Lapidus RC. (2017) The interoception revolution. *Trends Neurosci.* 40:123-28.
55. Lehrer P, Woolfolk RL. (2007) *Principles and practice of stress management.* Guilford Press.
56. Gevirtz, R. The physiology of heart rate variability biofeedback. *Biofeedback* 37, 22–25 (2009).
57. Goessl VC, Curtiss JE, Hofmann SG. (2017) The effect of heart rate variability biofeedback on autonomic function, anxiety, and stress: A meta-analysis. *Psychol. Med.* 47:769-78.
58. Nagler EM, Thayer JF. (2012) Heart rate variability and emotion: a neurovisceral integration model. *Clin. Psychol. Rev.* 32:500-12.
59. Thoma MV, Ehlert U, Nater UM. (2013) Autonomic nervous system dysregulation in depression and anxiety disorders. *Psychol. Med.* 43:1553-68.