Defining and testing autonomic states

A commentary on Kram and MacDuffie

Boris Živný, MD
SPP Institute, NeuroCentrum Clinic
30 October 2026

Correspondence: Boris Živný, MD
NeuroCentrum Clinic, Říčanská 1177, 252 42 Jesenice, Czech Republic
zivny@neurocentrum.cz
Website: https://spp.institute

Abstract

Kram and MacDuffie propose that neuromodulatory conditions help distinguish defensive states and move the polyvagal controversy toward resolution. Evaluating that proposal requires an accurate account of the theory being extended and a distinction between plausible mechanisms and demonstrated state markers. Published polyvagal accounts already discuss concurrent autonomic influences, candidate circuits for neuroception, and opioid-related mechanisms. These precedents qualify claims of novelty without establishing the validity of either framework. Further distinctions concern observed presentation versus inferred mechanism, conditioned freezing versus tonic immobility, and regional nerve composition versus a localized anatomical boundary. I approach this discussion from a separate line of work on the bodily conditions that generate afferent input and the central processes that weight it. The Somato-Psychic Pathway, Persistent Afferent Bias, and Axial Organ frameworks offer hypotheses for this purpose. Their possible convergence with PVT should be examined through independently defined outcomes and prospective tests that distinguish competing explanations.

The contribution and its evidential scope

Kram and MacDuffie (2026) seek to move the polyvagal controversy beyond its present disagreements by distinguishing autonomic states through neuromodulatory conditions as well as recruited circuitry. Their discussion recognizes the importance of afferent signaling and the limits of interpreting respiratory sinus arrhythmia without respiratory information. These questions warrant investigation. My concern is how the account represents the theory it proposes to extend, how it moves from synthesis to claims of physiological identification, and how the sources of afferent input can be specified more fully.

This commentary addresses the PDF marked Version 1.2, accessed through the ResearchGate publication record on 29 October 2026; the cited DOI carries the separate identifier v3. It examines selected claims about autonomic states and their testability. A new mechanism may explain particular observations without settling the broader controversy. Individual anatomical, evolutionary, and measurement claims of Polyvagal Theory (PVT) still require assessment on their own evidence. An expanded model should identify which earlier claims it retains, modifies, or no longer requires.

The criticisms also need to be represented from their original sources. Grossman (2023) challenges five physiological premises of PVT and the interpretation of respiratory sinus arrhythmia (RSA) as an index of vagal tone. Grossman et al. (2026) address RSA, brainstem vagal nuclei, vagal evolution, and

mammalian sociality. An opioid hypothesis could bear on a particular state mechanism without resolving all these objections. The scope of any proposed reconciliation should therefore be explicit.

Identify the theoretical claims under examination

A proposal to reconcile competing accounts needs to specify the propositions being reconciled. The history of a theory, its current formulation, and its clinical teaching models may differ. A correction to a simplified presentation does not necessarily revise the published theory, while a later clarification does not establish that an earlier formulation was accurate. These distinctions allow disagreement to be examined without assuming that either the theory or its critics constitute a single unchanging position.

The preprint's account of a one-system/one-state hierarchy (p. 27) needs to be considered alongside published descriptions of concurrent autonomic influences. Porges (2026b, section 5.5) explicitly distinguishes changes in regulatory dominance from exclusive activation or complete suppression of individual pathways. This does not establish the empirical adequacy of that formulation. It does mean that coactivation alone cannot refute it. If the objection concerns an earlier model, the relevant text and the historical scope of the objection should be identified.

Similarly, the characterization of PVT as predicting profound bradycardia across ordinary freezing and dissociation (p. 12) requires qualification. Porges (2023, section 13) explicitly states that bradycardia is not a reliable index of every life-threat response and discusses variation in the visceral expression of defensive regulation. This qualification leaves specific cardiac claims open to testing. Grossman et al. (2026, p. 104) also contest the attribution of marked cardiac slowing to dorsal vagal pathways. Qualifying the frequency of bradycardia leaves that anatomical objection to be evaluated separately.

The assertion that neuroception has never received an anatomical account (p. 25) is too broad. Porges (2003, pp. 39–45) proposed circuitry involving temporal cortical regions, the amygdala, and the periaqueductal gray, with diagrams linking these structures to defensive and social responses. The existence of a proposed circuit does not amount to a validated anatomical identification of neuroception. It nevertheless differs from having no anatomical proposal. A critique could examine the specificity and evidence for these candidate circuits directly.

The hierarchy also retains substantive commitments. Porges (2025, section 17.4) describes sequential recruitment under threat as a testable prediction. Consequently, clarification about coactivation should not make the sequence immune to contrary observations. Testing it requires a defined transition, temporal resolution, and measures that distinguish the proposed regulatory influences. The properties of a ladder metaphor, invoked on page 30 of the preprint, cannot by themselves establish either the prediction or its failure.

Specify what the neurochemical proposal adds

The preprint itself acknowledges earlier neurochemical accounts within PVT, including the proposed distinction between immobilization under different conditions (p. 13). The narrower question is what its opioid hypothesis adds. Porges (2003, p. 41) already discussed opioid-mediated analgesia associated with the ventrolateral periaqueductal gray, alongside immobility and its connections with the dorsal vagal complex. Grossman et al. (2026, Figure 1) likewise discuss peripheral neuropeptide influences on vagal transmission. These antecedents do not establish Kram and MacDuffie's proposed opioid

requirement for shutdown. They do require a precise account of what the proposed conjunction adds to the mechanisms already discussed by both sides.

Nazarloo et al. (2026), whom the preprint also cites, discuss interactions among oxytocin, vasopressin, corticotropin-releasing hormone, and urocortins in autonomic and cardiovascular regulation. Their review supports examining a broader neuromodulatory context; it does not establish a specific chemical signature for each clinical presentation. The preprint names mu and kappa opioid mechanisms and a ventrolateral periaqueductal location (p. 16). The experimental task is to translate these candidates into measurements or perturbations with a specified relationship to the observed state transition. A claim of necessity requires a prediction about what happens when the proposed mechanism is absent or perturbed. Evidence of an opioid-associated effect on analgesia or dissociation would leave the proposed cardiorespiratory conjunction unresolved.

Define the state independently of its proposed mechanism

The preprint acknowledges that the proposed cardiorespiratory and opioid conjunction has not been measured together in a living human (p. 16). Endnote 30 further distinguishes clinical observations from mechanistic evidence and states that the respiratory regularities were recognized retrospectively rather than recorded prospectively under a protocol. These qualifications are valuable. They should also govern the stronger formulations in the main text, particularly assertions that a specified opioid condition is required for a given state.

A test must avoid identifying shutdown by presumed opioid effects and then using that classification to establish opioid involvement. Observable inclusion criteria, the reliability of state classification, and evidence for the proposed mechanism are separate questions. Neither an observer's confidence nor a coherent physiological interpretation substitutes for independent validation. The claim that an observer can reliably discriminate states requires explicit assessment of agreement between observers, including observers who were not trained by the model's developers.

The distinction between apnea and breathing of very low amplitude is particularly consequential. Endnote 30 acknowledges that this distinction has not yet been resolved instrumentally. A classification based on apparent absence of breathing therefore remains provisional. Likewise, an absence of visible respiratory struggle does not, by itself, demonstrate absence of inspiratory drive. The narrower clinical series also consists of selected adults and excludes autism and several other presentations. Extrapolation to children or excluded populations requires separate evidence.

Keep freezing and tonic immobility distinct

The respiratory argument needs a specific correction. The experiments by Bagur et al. (2021) and Moberly et al. (2018) concern conditioned-fear freezing in mice. They report respiration-related patterns during that behavior. The preprint uses these findings to describe the respiratory signature of tonic immobility as established (p. 18), while elsewhere treating freezing and tonic immobility as distinct defensive states.

The studies can support a hypothesis about respiratory organization during defensive immobility. Their findings do not directly establish the proposed respiratory signature of human tonic immobility. The manuscript needs either an explicit argument for construct equivalence or separate evidence from a preparation that operationalizes tonic immobility. This problem concerns the identity of the behavior as

well as translation across species. It would remain even if absolute respiratory frequencies were omitted. More cautious wording would preserve the prediction while making its evidential basis clear.

Separate regional anatomy from a localized boundary

Havton et al. (2021) provide direct human evidence that cervical and abdominal vagus samples differ in their myelinated fiber populations. That result constrains anatomical models. However, comparing two sampled regions does not locate a sharp discontinuity precisely at the diaphragm. Nor do those measurements alone identify the functional contribution of each fiber population or demonstrate a corresponding division between psychological or autonomic states.

I would therefore describe the finding as a measured regional difference in vagal composition. A stronger claim about a diaphragmatic boundary requires appropriately localized sampling. A further claim about perceptual resolution requires functional evidence. Keeping these inferences separate preserves the anatomical result without treating it as confirmation of the broader explanatory model.

Specify the conditions that generate afferent input

The starting point of my own work is the clinical relationship between somatic structural-functional conditions, persistent afferent input, and the development of regulation. The preprint already gives afference an important role. The additional question is what generates, sustains, and changes the input available for central regulation. A model can describe circuitry and neuromodulation precisely while leaving persistent peripheral conditions underspecified. Somatic and respiratory-postural inputs should therefore be considered alongside visceral vagal signaling. The distinction concerns the origin and persistence of input, its central weighting, and the resulting regulatory response.

In the Somato-Psychic Pathway framework, I propose a developmental relationship between somatic structural-functional integrity, autonomic regulation, and the emergence of mental capacities (Živný, 2026b). The framework allows reciprocal regulation and does not establish a universal causal sequence for individual symptoms. Its relevance here is the need to distinguish the conditions under which regulation develops from the mechanisms through which a defensive response is expressed at a particular moment.

Persistent Afferent Bias develops a more specific hypothesis: persistent altered afferent conditions and their state-dependent weighting may contribute to sustained regulatory changes (Živný, 2026a). Applied to the present discussion, it asks whether some recurring defensive presentations are influenced by ongoing bodily input as well as by threat-related context and neuromodulation. This is a testable possibility, not an established explanation of shutdown or dissociation. It requires measurements that can distinguish changes in peripheral input from changes in central processing and from changes in autonomic output.

The Axial Organ position paper supplies a proposed anatomical and functional domain for investigating axial and respiratory-postural contributions (Živný & Živná, 2026). It distinguishes that domain from the central nervous system that regulates it and from Persistent Afferent Bias. Its definitions may help specify observations and candidate inputs; the position paper does not demonstrate a causal pathway to a particular defensive state.

SPP and PAB were developed from these somatic and developmental questions. PVT approaches related problems through the proposed functional organization and recruitment of autonomic pathways (Porges,

2025). These are distinct explanatory starting points. Their possible convergence does not establish a common causal model, and SPP or PAB hypotheses can be assessed independently of disputed PVT claims. Compatibility should be examined proposition by proposition, using observations that could also distinguish the frameworks.

The distinction also matters when interpreting intervention effects. A change following bodily, attentional, or neuromodulatory intervention does not by itself identify which component mediated the change. Porges's (2026a) recent account of mammalian regulatory architecture similarly distinguishes routes of initial engagement from their proposed effects on broader regulatory organization. Convergence of outcomes can motivate a mechanistic question, but cannot answer it without measurements that discriminate the alternatives.

A prospective route to discrimination

A first study should ask whether the proposed presentations can be identified reproducibly and whether their predicted respiratory differences occur. Definitions and predictions should be specified before data collection. Where respiration is the outcome under test, the classification procedure should not make the predicted respiratory pattern a necessary inclusion criterion. Independent ratings, masked where feasible to the physiological recordings and theoretical assignment, would help assess whether the distinctions survive outside the originating clinical setting.

Synchronized recordings could combine ECG with direct assessment of respiration, motor behavior, and other autonomic measures selected for the research question. If the claim concerns apnea, measurement must distinguish absent airflow from low-amplitude breathing and respiratory effort. Posture, voluntary breath control, medication, task conditions, and recording artifacts should be documented. These design choices would make the predicted respiratory contrast interpretable before assigning it to a specific circuit or neuromodulatory mechanism.

A subsequent mechanistic study could test whether information about neuromodulation improves prediction beyond the observable presentation and context. The same standard applies to an afferent account: measures of candidate bodily inputs and their changes should add explanatory or predictive value beyond simpler models. Changes in pain, respiratory mechanics, attention, expectancy, or arousal are competing explanations that need to be separated where relevant. An association with one of these variables would justify further investigation, while leaving necessity and sufficiency unresolved.

Kram and MacDuffie's proposal can contribute to the discussion if its candidate mechanisms are stated as hypotheses with identifiable alternatives. Accurate representation of the published theory, consistent definitions of behavior, and proportionate anatomical inferences are necessary for that comparison. Specifying the conditions that generate and sustain afferent input would add a further set of testable questions. None of these frameworks, including my own, should receive exemption from the same evidential requirements.

Author perspective

I developed the Somato-Psychic Pathway and Persistent Afferent Bias frameworks and coauthored the Axial Organ position paper cited here. These works are included because they address the afferent and developmental questions discussed above.

References

Bagur, S., Lefort, J. M., Lacroix, M. M., de Lavilléon, G., Herry, C., Chouvaeff, M., Billand, C., Geoffroy, H., & Benchenane, K. (2021). Breathing-driven prefrontal oscillations regulate maintenance of conditioned-fear evoked freezing independently of initiation. *Nature Communications, 12*, Article 2605. https://doi.org/10.1038/s41467-021-22798-6

Grossman, P. (2023). Fundamental challenges and likely refutations of the five basic premises of the polyvagal theory. *Biological Psychology, 180*, Article 108589. https://doi.org/10.1016/j.biopsycho.2023.108589

Grossman, P., Ackland, G. L., Allen, A. M., Berntson, G. G., Booth, L. C., Burghardt, G. M., Buron, J., Dinets, V., Doody, J. S., Dutschmann, M., Farmer, D. G. S., Fisher, J. P., Gourine, A. V., Joyner, M. J., Karemaker, J. M., Khalsa, S. S., Lakatta, E. G., Leite, C. A. C., Macefield, V. G., . . . Zucker, I. H. (2026). Why the polyvagal theory is untenable: An international expert evaluation of the polyvagal theory and commentary upon Porges, S. W. (2025). Polyvagal theory: Current status, clinical applications, and future directions. Clin. Neuropsychiatry, 22(3), 169–184. *Clinical Neuropsychiatry, 23*(1), 100–112. https://doi.org/10.36131/cnfioritieditore20260110

Havton, L. A., Biscola, N. P., Stern, E., Mihaylov, P. V., Kubal, C. A., Wo, J. M., Gupta, A., Baronowsky, E., Ward, M. P., Jaffey, D. M., & Powley, T. L. (2021). Human organ donor-derived vagus nerve biopsies allow for well-preserved ultrastructure and high-resolution mapping of myelinated and unmyelinated fibers. *Scientific Reports, 11*, Article 23831. https://doi.org/10.1038/s41598-021-03248-1

Kram, G., & MacDuffie, H. (2026). *Breaking the stasis in the polyvagal controversy: Two category errors, a missing variable, and a measured boundary* [Preprint]. PsyArXiv. https://doi.org/10.31234/osf.io/yehuc_v3

Moberly, A. H., Schreck, M., Bhattarai, J. P., Zweifel, L. S., Luo, W., & Ma, M. (2018). Olfactory inputs modulate respiration-related rhythmic activity in the prefrontal cortex and freezing behavior. *Nature Communications, 9*, Article 1528. https://doi.org/10.1038/s41467-018-03988-1

Nazarloo, H. P., Porges, S. W., Davis, J. M., & Carter, C. S. (2026). From molecules to meaning: Integrating neuropeptides, sociostasis, and hormesis in the brain–heart axis. *Current Issues in Molecular Biology, 48*(4), Article 386. https://doi.org/10.3390/cimb48040386

Porges, S. W. (2003). Social engagement and attachment: A phylogenetic perspective. *Annals of the New York Academy of Sciences, 1008*, 31–47. https://doi.org/10.1196/annals.1301.004

Porges, S. W. (2023). The vagal paradox: A polyvagal solution. *Comprehensive Psychoneuroendocrinology, 16*, Article 100200. https://doi.org/10.1016/j.cpnec.2023.100200

Porges, S. W. (2025). Polyvagal theory: A journey from physiological observation to neural innervation and clinical insight. *Frontiers in Behavioral Neuroscience, 19*, Article 1659083. https://doi.org/10.3389/fnbeh.2025.1659083

Porges, S. W. (2026a). The mammalian regulatory architecture: Biobehavioral portals and the organization of physiological state. *Frontiers in Neuroscience, 20*, Article 1923279. https://doi.org/10.3389/fnins.2026.1923279

Porges, S. W. (2026b). When a critique becomes untenable: A scholarly response to Grossman et al.'s evaluation of Polyvagal Theory. *Clinical Neuropsychiatry*, *23*(1), 113–128. https://doi.org/10.36131/cnfioritieditore20260111

Živný, B. (2026a). Persistent Afferent Bias: A mechanistic link between somatic disruption and autonomic dysregulation. *Frontiers in Integrative Neuroscience, 20*, Article 1848233. https://doi.org/10.3389/fnint.2026.1848233

Živný, B. (2026b). Somato-Psychic Pathway: A universal developmental trajectory linking somatic structural–functional integrity, autonomic regulation, and the emergence of mind. *Frontiers in Integrative Neuroscience*, *20*, Article 1771123. https://doi.org/10.3389/fnint.2026.1771123

Živný, B., & Živná, K. (2026). *Axial organ (AxO): A position paper on definition, boundaries, and functional organization* (SPP Institute Position Papers, No. 2). NeuroCentrum. https://doi.org/10.5281/zenodo.22737933