

A Nervous System First Methodology: Regulation as a Trained Skill

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Abstract

This white paper sets out a methodology that treats autonomic regulation as the primary training target in human performance work, with voluntary slow breathing as the most accessible input to it. The argument is structural rather than symptomatic: sleep quality, attentional stability, pain sensitivity and recovery capacity each have their own literature, and interventions aimed at each in isolation address outputs rather than the regulatory state they share. The paper sets out a practice model built on one respiratory input directed toward three aims, reset after a demand, recovery toward rest, and deliberate activation before demand, distributed across a working day as brief repeated practice attached to existing daily cues. Evidence is reported with its actual strength. A systematic review and meta-analysis of 223 studies found vagally mediated heart rate variability increased during slow breathing, after a single session, and following multi-session training. Resonance breathing near six breaths per minute has a well-described baroreflex mechanism, though its originating authors concede the controlled clinical evidence is limited. The paper also corrects two claims commonly made in this field, including in the author's own prior work: heart rate variability driven by paced breathing is not a measure of vagal tone, and there is no evidence that brief frequent practice produces greater physiological adaptation than longer infrequent practice. The defensible case for brief daily practice is that no study has shown longer or less frequent sessions to do better, and that brief practice fits more easily into a working day; whether it is also easier to sustain has not been shown.

Keywords

autonomic regulation, slow breathing, resonance frequency breathing, heart rate variability, respiratory heart rate variability, practice dose, habit formation, human performance methodology, self-regulation training, nervous system regulation

Introduction

A person with poor sleep, afternoon fog, a short fuse and recurring tension has four problems on their calendar and four categories of product waiting for them. Each of those complaints has its own literature, its own specialists and its own interventions. Treated separately, each consumes attention and each produces a modest result.

The methodology described here starts from a different organizing assumption: those complaints share a regulatory substrate, and the regulatory substrate is the more efficient target. Autonomic and neuroendocrine stress responses are coordinated through limbic, hypothalamic and brainstem circuitry rather than generated independently per symptom (Ulrich-Lai and Herman, 2009). That does not mean every complaint reduces to autonomic state, and this paper does not claim it does. It means that a person whose regulation is poor is paying a cost that shows up in several places at once, and that addressing regulation is a reasonable first move rather than a last resort.

The practical question is then what input to use. Among the levers available to a person in the middle of a working day, without equipment and without leaving the situation, voluntary slow breathing has the clearest measured association with autonomic change. A systematic review and meta-analysis of 223 studies found vagally mediated heart rate variability increased during slow breathing, immediately after a single session, and following multi-session interventions (Laborde et al., 2022). The mechanism most strongly supported involves baroreflex resonance, with maximum heart rate oscillation near 0.1 Hz, roughly six breaths per minute (Lehrer and Gevirtz, 2014).

This paper is also, in part, a correction. Two claims are widely made in this field, including in the author's own earlier work, that the evidence does not support. The first is that heart rate variability produced by paced breathing can be read as a measure of vagal tone. An international expert recommendation published in 2025 states directly that respiratory heart rate variability amplitude should not be misconstrued as a measure of vagal tone (Menuet et al., 2025), and the methodological basis for that caution was established nearly two decades earlier (Grossman and Taylor, 2007). The second is that brief frequent practice produces greater physiological adaptation than longer infrequent practice. The one randomized trial that tested distribution with total practice time held constant, with 351 participants, found no significant difference (Riordan et al., 2024).

Stating both corrections is deliberate. A methodology that depends on claims its own field has retired is a liability to the person using it. The case for this methodology does not require either claim, and the paper sets out what it does require.

The framework draws on the author's applied work with executives, athletes and military personnel and on the author's book *Stress: Our Human Superpower*. Claims from that practice are labeled as practitioner observation.

The Problem With the Current View

Treating outputs one at a time. The dominant approach to the complaints described above is parallel and specialized. A sleep intervention for the sleep, an attention tool for the fog, a physical intervention for the tension. Each may help. The cost is that a person running four interventions is

running four maintenance projects, and none of them addresses whatever is common to the four. The methodology argued here does not claim that the specialized interventions are wrong. It claims that regulation is the more economical place to start, and that it is the step most often skipped.

Mistaking understanding for capability. Reading about a physiological mechanism does not install the response. This is unremarkable in physical training and routinely forgotten in regulation work. The relevant evidence is that multi-session practice, not single exposure, is what produces post-intervention autonomic change: of the 223 studies in the largest review, 49 examined multi-session interventions and found increases in vagally mediated heart rate variability persisting beyond the session itself (Laborde et al., 2022). Practice is the active ingredient.

Assuming the practice has to be long. The standard protocol in heart rate variability biofeedback research is 10 to 20 minutes of paced breathing per session, typically a weekly supervised session plus daily home practice of around 20 minutes over two to eight weeks (Lalanza et al., 2023). For most working adults that is not a schedule they will keep, and adherence data bear this out: in one pilot with healthcare workers, home practice adherence reached only 62 percent of the target (Castro Ribeiro et al., 2023).

The honest position on session length is narrower than the one often advertised, including in the author's prior work. A meta-analysis of 58 heart rate variability biofeedback studies found that neither the number of sessions nor the number of weeks moderated outcomes (Lehrer et al., 2020). A dose-response meta-regression across 203 randomized trials of mindfulness-based programs with 15,971 participants found no evidence that larger doses outperformed smaller ones for depression, anxiety or stress (Strohmaier, 2020). These are null findings. They support the claim that brief practice is sufficient. They do not support the claim that brief practice is superior, and the paper does not make that claim.

Depending on contested theory. A good deal of popular nervous system material rests on polyvagal theory. Its standing is addressed directly in the Supporting Evidence section, because a methodology built on it inherits a problem that has become acute. The short version is that the theory remains influential in clinical and somatic practice while its core neurophysiological and evolutionary premises have been rejected by the relevant specialist communities, most comprehensively in a 39-author international evaluation (Grossman et al., 2026). The methodology described here does not require it.

The Framework

1. Regulation is the upstream target

The methodology treats autonomic regulatory capacity as the training object, and the specific complaints that bring people in as downstream indicators. The justification is coordination rather than reduction: the systems involved are centrally regulated rather than independent (Ulrich-Lai

and Herman, 2009), so a person with poor regulation is likely to show costs in more than one place.

The claim stays bounded. This is a sequencing argument, not a theory of everything. It does not assert that sleep, pain and attention are the same problem, and it does not displace specialized care where that is indicated.

2. One input, three aims

Rather than a catalogue of techniques matched to situations, the methodology uses one respiratory input directed at three aims. The engine is a slow nasal cadence with the exhale longer than the inhale, run near resonance frequency, around six breaths per minute, where baroreflex-mediated heart rate oscillation is largest (Lehrer and Gevirtz, 2014).

Aim	When it is used	What it is for
Reset	Immediately after a demand ends	Close out activation before the next demand begins
Recover	Approaching rest and sleep	Move the system toward the low arousal state that deep rest requires
Activate	Before a demand begins	Enter the demand deliberately rather than reactively

The practical value of one engine with three aims is retrieval under pressure. A single cadence with a known rhythm is more likely to be available in a difficult moment than a library of protocols requiring selection. This is a practitioner rationale rather than a research finding.

3. Practice is brief, repeated and attached to existing cues

The methodology places short practice at four points in the day that already exist: on waking, at a midday transition, immediately after a demanding event, and before sleep. Total daily time is roughly seven to ten minutes.

The reasoning is practical rather than physiological. Habit formation research indicates that automaticity develops through repetition of a behavior anchored to a consistent existing context. In the study most often cited on this point, the median time to reach 95 percent of maximum automaticity was 66 days, with a range from 18 to 254 days, and missing a single opportunity did not materially disrupt the trajectory (Lally et al., 2010). That median was computed across the 39 of 82 participants whose data fitted the model, so it describes fewer than half the sample. Specifying in advance when and where a behavior will occur produces a substantial improvement in follow-through, with a pooled effect of d equals 0.65 across 94 independent tests (Gollwitzer and Sheeran, 2006).

Brief daily practice is sufficient to produce measurable change in mood and respiratory rate. A randomized trial with 108 enrolled participants, in which each person practiced one of three breathing techniques for five minutes once daily for one month, found greater improvement in

positive affect and a greater reduction in respiratory rate than an equivalent period of mindfulness meditation, with exhale-emphasized cyclic sighing strongest, and no significant change in heart rate variability across the month (Yilmaz Balban et al., 2023). A four-week protocol of 10 to 20 minute sessions twice daily, five days a week, produced a significant increase in low frequency heart rate variability with a small effect, d equals 0.33 (Dolgilevica, Grunfeld and Derakshan, 2025).

What the evidence does not support, and what the author's earlier work asserted, is that this distribution produces more adaptation than longer sessions. The single randomized trial testing distribution with total time held constant, 351 participants comparing 20 minutes once daily against two 10 minute sessions, found no difference on any measure, with p values from .158 to .729 (Riordan et al., 2024). The defensible claim is that no difference in measured outcome has been detected between the schedules. Whether brief practice is easier to sustain is plausible but untested; in the one trial that measured practice, days and minutes were similar across schedules.

4. Measurement is feedback, within strict limits

The methodology uses heart rate variability trends as week-over-week feedback for an individual against their own baseline. Two constraints govern how that can be described.

First, a breathing-driven variability figure is not a measure of vagal tone. Respiratory heart rate variability amplitude depends on respiratory rate and tidal volume, so changing how a person breathes alters the measure independently of any change in autonomic outflow (Grossman and Taylor, 2007), and an international expert recommendation states plainly that this amplitude should not be misconstrued as a measure of vagal tone (Menuet et al., 2025). Second, measurement conventions are not interchangeable across recording durations, and age, sex, posture, respiration and health all shift baselines (Shaffer and Ginsberg, 2017).

What remains defensible is narrow and still useful: a non-medical trend for one person against their own prior values, interpreted as a signal about that person's pattern over weeks, never as a diagnostic value or a comparison between people.

Supporting Evidence

Slow breathing changes heart rate variability indices

The largest synthesis available is a systematic review and meta-analysis of 223 studies, analysed in three categories: 172 measuring during the breathing session, 16 immediately after a single session, and 49 following multi-session interventions, with some studies contributing to more than one category (Laborde et al., 2022). Vagally mediated indices increased significantly at all three time points. For RMSSD the pooled effects were Hedges g equals 0.530, 95 percent confidence interval 0.430 to 0.620, during practice; g equals 0.140, confidence interval 0.030 to 0.240, immediately

after a single session; and g equals 0.320, confidence interval 0.080 to 0.560, after multi-session interventions. Low frequency absolute power during practice was larger again, g equals 1.490, confidence interval 1.280 to 1.690.

One interpretive caution belongs with that result and is rarely stated. Low frequency power rising during slow breathing is partly what would be predicted mechanically, because breathing at roughly 0.1 Hz moves respiratory activity into the low frequency band. Since respiratory heart rate variability amplitude is itself a function of respiratory rate and tidal volume (Grossman and Taylor, 2007), the finding that slow breathing raises these indices is partly definitional and does not on its own establish an increase in autonomic outflow. Two further caveats come from the review itself. Heterogeneity was high in the during-practice analyses, with I^2 of 81 percent for RMSSD and 93 percent for low frequency power. And overall risk of bias was rated high in 215 of the 223 included studies, with some concerns in 5 and low risk in 1, with no certainty-of-evidence assessment performed. The direction of effect is consistent across a very large literature, and the quality of that literature is mostly poor.

A narrative review of the physiological effects of slow breathing in healthy humans attributes the observed respiratory, cardiovascular and autonomic changes to diaphragmatic engagement and a shift in sympathovagal balance, while calling for expanded clinical research (Russo, Santarelli and O'Rourke, 2017). It is a useful summary and weak as evidence.

The resonance mechanism is well described and clinically underproven

The best-supported mechanism for paced breathing effects is baroreflex resonance. Maximum heart rate oscillation amplitude occurs near 0.1 Hz, around six breaths per minute, with some data placing it closer to 5.5 breaths per minute, at which point heart rate and blood pressure oscillate approximately 180 degrees out of phase (Lehrer and Gevirtz, 2014). The characteristics of that resonance were examined directly across 56 participants, 32 with asthma and 24 healthy adults, finding that an individual's resonant frequency relates inversely to height, is lower in men than in women, is unrelated to age, weight or the presence of asthma, and stays constant across ten sessions of biofeedback training (Vaschillo, Vaschillo and Lehrer, 2006).

The same authors who describe the mechanism are candid about the evidence for outcomes, noting that few areas have extensive support from controlled studies while describing the overall picture as promising (Lehrer and Gevirtz, 2014). Respiratory sinus arrhythmia, the rhythm underlying the effect, has been argued to serve a function in matching ventilation and perfusion rather than being an incidental byproduct (Yasuma and Hayano, 2004), though that functional account has itself been contested.

An international expert recommendation published in 2025 proposes replacing the term respiratory sinus arrhythmia with respiratory heart rate variability, since arrhythmia wrongly implies pathology, and states explicitly that the amplitude of this variability should not be misconstrued as a measure of vagal tone (Menuet et al., 2025).

Mechanistic sources frequently cited in this field, with their actual weight

Several papers routinely cited as establishing how breathing influences brain state carry less weight than their reputation suggests, and this paper states that rather than borrowing the authority.

A study identifying a neuronal subpopulation in the preBötzinger complex whose ablation shifted animals toward calm states while leaving breathing intact is a mouse study (Yackle et al., 2017). It demonstrates a brainstem circuit linking respiratory rhythm to arousal in mice. It does not establish that voluntary slow breathing in humans operates through that circuit.

A proposal that slow deep breathing recruits slowly adapting pulmonary stretch receptors in a pattern that drives central relaxation is explicitly labeled a hypothesis by its authors and presents no new data (Noble and Hochman, 2019). A study proposing the locus coeruleus as a synchronizing node between respiration and attention combines data from 14 participants with a mathematical model, in a region where brain imaging is technically difficult (Melnychuk et al., 2018).

Each is citable with its limitation stated. None can carry a primary claim.

Polyvagal theory: influential in practice, rejected on mechanism

This section is included because polyvagal theory appears widely in nervous system material aimed at general audiences, including in the author's earlier work, and readers deserve an accurate account of its standing.

The theory proposes a hierarchical account of autonomic states distinguishing a myelinated ventral vagal system supporting social engagement from an older unmyelinated dorsal system (Porges, 2007; Porges, 2025). Its core premises have been challenged across four independent lines.

On measurement, respiratory sinus arrhythmia indexes cardiac vagal control only under restricted conditions, and respiratory parameters confound the relationship between it and vagal tone (Grossman and Taylor, 2007). On comparative anatomy, electron micrographs of myelinated fibers in the cardiac vagal branches of reptiles contradict the claim that a myelinated vagus is a mammalian innovation, and dual vagal motor nuclei and respiratory heart rate variability have been documented in reptiles, amphibians, birds and lungfish (Taylor, Wang and Leite, 2022). On neuroanatomy, a review of vagal functional anatomy concluded that the theory's basic phylogenetic and functional-anatomical tenets do not withstand closer scrutiny, while allowing that it may retain heuristic value (Neuhuber and Berthoud, 2022). A premise-by-premise critique argued that equating respiratory sinus arrhythmia with general vagal tone is a category error (Grossman, 2023).

In 2026 a 39-author international evaluation, including autonomic physiologists, cardiologists, comparative biologists and neuroanatomists, concluded that the theory is untenable on existing neurophysiological and evolutionary evidence, and noted that psychological applications built on those foundations lack supporting evidence (Grossman et al., 2026).

The methodology described in this paper does not depend on polyvagal theory. Its mechanistic account rests on baroreflex resonance and on measured changes in heart rate variability indices, both of which stand independently. Practitioners using polyvagal language should treat it as a clinical heuristic, as Neuhuber and Berthoud allow, and not as a physiological explanation.

Nasal breathing: plausible, with thin evidence

The methodology specifies nasal breathing. The supporting evidence is limited to small acute studies.

A randomized-order crossover with 20 young adults found five minutes of nasal-only breathing produced lower mean and diastolic blood pressure and a higher high frequency contribution to heart rate variability than oral breathing, with moderate effect sizes and p values at or near 0.04 for the variability findings (Watso et al., 2023). A crossover study of 45 young men found nasal breathing increased pulse propagation time and brachial artery diameter, though blood pressure, heart rate and autonomic measures were not assessed (Snoeck, Paillaugue and Provyn, 2026). Intracranial recordings in seven surgical patients, with behavioral testing in 107 healthy participants, found that nasal airflow entrains limbic oscillations and that respiratory phase modulates performance on specific emotional and memory tasks (Zelano et al., 2016). That finding concerns the phase of the breath, not a general cognitive benefit of nasal breathing.

One result runs the other way and belongs in any honest account. A randomized crossover in 12 healthy adults undergoing maximal cardiopulmonary exercise testing found that enforced nasal-only breathing reduced peak oxygen uptake by roughly 16 percent and ventilation by more than a third, with markedly elevated breathlessness, while differences were minor up to about 75 percent of maximal workload (Mapelli et al., 2025).

The practical reading is specific: nasal breathing is appropriate for regulation and submaximal work, and should not be enforced at maximal effort. No chronic outcome evidence in healthy adults supports anything stronger.

Practice dose: brief is sufficient, not superior

A meta-analysis of 58 heart rate variability biofeedback studies found a small to moderate pooled effect favoring the intervention and, importantly for this methodology, no significant moderation by number of sessions or number of weeks (Lehrer et al., 2020). A dose-response meta-regression across 203 randomized trials of mindfulness-based programs with 15,971 participants found no evidence that larger doses produced better outcomes for depression, anxiety or stress (Strohmaier, 2020).

On session length specifically, a three-arm randomized experiment with 71 novices, holding total time constant at 25 minutes, found that four 5 minute practices outperformed four 20 minute practices on trait mindfulness and stress (Strohmaier, Jones and Cane, 2021). Both arms practised

four times over two weeks at identical frequency, so the study varies duration and not frequency.

On frequency, a randomized trial with 351 undergraduates carrying clinically elevated depression or anxiety compared 20 minutes of daily app-delivered meditation in a single session against two 10 minute sessions over two weeks, with total daily time held constant. Both conditions improved on every outcome, with absolute effect sizes from 0.12 to 0.63, and there were no differences between the two schedules on any measure, with p values from .158 to .729 (Riordan et al., 2024). The population limits how far this carries: these were distressed beginners practising meditation, not healthy adults practising paced breathing. A 2026 review of the dose-response question notes that despite more than 2,500 randomized trials in this area, only four studies have experimentally manipulated session duration and only one has manipulated frequency (Bowles et al., 2026).

A direct comparison of brief daily practice against a long infrequent session, at the ratio this methodology implies, has not been conducted. The claim made here is therefore no detected difference, plus a plausible practical advantage, rather than physiological superiority.

For completeness, protocols that have produced measured autonomic change used short, near-daily sessions. A four-week remote protocol at six breaths per minute, 10 to 15 minutes rising to 20 minutes, twice daily, five days per week, produced a significant increase in low frequency heart rate variability with d equals 0.33, alongside sleep and anxiety improvements (Dolgilevica, Grunfeld and Derakshan, 2025). A pilot with 21 healthcare workers combining weekly 45 to 60 minute sessions with daily home practice of 10 to 20 minutes found increases in resting SDNN and reductions in respiratory rate, with home adherence at 62 percent and no control group (Castro Ribeiro et al., 2023). A randomized trial with 108 enrolled participants, comparing three breathing techniques, each practiced for five minutes once daily, against mindfulness meditation over one month, found greater improvement in positive affect for breathwork, 1.91 points on the PANAS positive scale against 1.22 for meditation, and a greater reduction in respiratory rate, with exhale-emphasized cyclic sighing strongest (Yilmaz Balban et al., 2023). That same trial found no significant change in heart rate variability or resting heart rate across the month, which is a result this methodology has to account for rather than set aside, and it is taken up in the Limitations section.

Standard practice in this literature is 10 to 20 minutes per session over two to eight weeks, and nearly two thirds of reviewed studies did not report protocol detail sufficient for replication (Lalanza et al., 2023). The field does not have a well-characterized minimum effective dose.

Practical Implications

The implementation model

Four practice points attached to moments that already occur in a working day. Total time is roughly seven to ten minutes.

Anchor	Timing	Aim	Approximate duration
Morning	On waking, before the first demand	Establish respiratory control for the day	2 minutes
Midday	At a transition between work blocks	Clear accumulated activation	90 seconds
Post-demand	Immediately after a meeting, conflict or hard effort	Close out the activation that demand opened	1 to 2 minutes
Evening	10 to 15 minutes before sleep	Move the system toward rest	3 to 5 minutes

The cadence is a slow nasal pattern with the exhale longer than the inhale, run near six breaths per minute where the resonance effect is largest (Lehrer and Gevirtz, 2014). The specific ratios used in the author's practice are a practitioner choice rather than an evidence-derived prescription.

What to expect, and in what order

Mood, self-reported stress and respiratory rate are the outcomes with the strongest measured support in this literature (Yilmaz Balban et al., 2023; Strohmaier, 2020). Resting autonomic change has been observed over four to five week protocols with small effects, d equals 0.33 in one randomized trial (Dolgilevica, Grunfeld and Derakshan, 2025), while a one month trial of brief daily breathing found no significant change in heart rate variability at all (Yilmaz Balban et al., 2023). Set expectations accordingly: mood and breathing rate move first and most reliably, and autonomic change is small, slower and not guaranteed. Anyone promising rapid or large physiological change is going beyond the evidence.

How to make it stick

The adherence mechanics matter more than the protocol details, because an unperformed protocol has no effect. Two findings are directly applicable. Specifying in advance when and where a practice will occur substantially improves follow-through, d equals 0.65 across 94 tests (Gollwitzer and Sheeran, 2006). Automaticity develops through repetition in a consistent context, taking a median of 66 days to reach 95 percent of maximum in the study most cited on this, with a range from 18 to 254 days, and missing a single opportunity does not materially disrupt the trajectory (Lally et al., 2010).

The second point deserves emphasis in practice, because the belief that one missed day ruins a streak is a common reason people abandon a practice entirely.

What to measure and how to describe it

Use heart rate variability trends as a non-medical week-over-week signal for one person against their own baseline. Three constraints follow from the evidence.

Do not describe the measure as vagal tone. An international expert recommendation states that respiratory heart rate variability amplitude should not be interpreted that way (Menuet et al., 2025), and the confound is that the measure varies with respiratory rate and tidal volume independently of autonomic outflow (Grossman and Taylor, 2007). Do not compare values across recording durations or between people, since norms for different durations are not interchangeable and age, sex, posture and health all shift baselines (Shaffer and Ginsberg, 2017). Do not treat a value as diagnostic.

For anyone building products or protocols in this area, these are not stylistic preferences. They are the specific claims that a knowledgeable reviewer, clinician or regulator will test first.

Where nasal breathing applies

Use nasal breathing for regulation practice and submaximal work. Do not enforce it at maximal effort, where it has been shown to reduce peak oxygen uptake substantially and increase breathlessness (Mapelli et al., 2025).

Limitations

This is a white paper. It has not been peer reviewed and reports no original empirical research.

The dose claim in the author's prior work is not supported. The assertion that brief frequent practice produces greater adaptation than longer infrequent practice has not been established. The one randomized trial that tested distribution with total practice time held constant, with 351 participants, found no significant difference (Riordan et al., 2024), and a 2026 review confirms that only four studies have manipulated session duration and one has manipulated frequency across a literature of more than 2,500 trials (Bowles et al., 2026). This paper replaces that claim with a narrower one: no difference has been detected, and brief practice may fit more easily into a working day. Readers who encountered the stronger version in the author's book should treat this section as the correction.

The spacing effect literature cannot be borrowed to fill that gap. Meta-analytic work on distributed practice concerns gaps between trials within or near a session, with the benefit shrinking as intervals lengthen and the smallest effect occurring at intervals greater than one day (Donovan and Radosevich, 1999), and the verbal recall synthesis shows the optimal gap scales non-monotonically with retention interval in a domain with no physiological outcomes (Cepeda et al., 2006). Citing that literature in support of a daily training calendar would be a domain error.

No meta-analysis of distributed practice specific to physiological or self-regulation training was located. That gap is stated rather than papered over.

Heart rate variability findings are partly definitional. Because respiratory heart rate variability amplitude depends on respiratory rate and tidal volume (Grossman and Taylor, 2007), the finding that slow breathing raises these indices does not by itself demonstrate increased autonomic outflow. The quality of that evidence base is also poor: in the largest review, overall risk of bias was rated high in 215 of the 223 included studies, heterogeneity reached I squared of 81 to 93 percent in the during-practice analyses, and no certainty-of-evidence assessment was performed (Laborde et al., 2022).

One trial found no autonomic change from brief daily practice at all. A randomized trial of three breathing techniques, each practiced for five minutes once daily over one month, reported improved mood and reduced respiratory rate but no significant change in heart rate variability or resting heart rate (Yilmaz Balban et al., 2023). This bears directly on the measurement component of the methodology. Brief practice has better evidence for shifting mood and breathing rate than for shifting resting autonomic indices, and anyone using a variability trend as proof that brief practice is working should know that one well-conducted month-long trial did not find that change.

The mechanism is better established than the outcomes. The authors who describe baroreflex resonance also state that few areas have extensive support from controlled studies (Lehrer and Gevirtz, 2014). A plausible mechanism is not an outcome.

Mechanistic citations commonly used in this field are weaker than their reputation. The arousal circuit finding is a mouse study (Yackle et al., 2017), the pulmonary afferent account is explicitly a hypothesis (Noble and Hochman, 2019), and the locus coeruleus work combines 14 participants with a model (Melnichuk et al., 2018).

The nasal breathing evidence is thin. Acute studies with 12 to 45 participants, mostly healthy young adults, exposures of five minutes to a single session, surrogate endpoints, and no chronic outcome evidence in healthy adults. One well-conducted study shows a clear performance decrement when nasal breathing is enforced at maximal exercise (Mapelli et al., 2025).

The upstream regulation argument is a sequencing claim, not a demonstrated causal hierarchy. No study cited here shows that training regulation resolves sleep, attention and pain complaints more efficiently than targeting them directly. That comparison has not been made.

The specific protocol is not evidence-derived. The four anchors, their durations and the breathing ratios used come from the author's applied practice. The supporting research establishes that brief daily practice can produce measurable change; it does not validate this particular configuration.

Practitioner observation is not evidence. Patterns observed in the author's work with executives, athletes and military personnel come from an uncontrolled setting with a self-selected population and no comparison group.

Scope and safety. This paper concerns generally healthy adults. Breathing practices are not appropriate for everyone, and the safety note in the Disclosure section applies.

Conclusion

A methodology is only as durable as the claims holding it up. This paper has set out a nervous system first approach to human performance and has removed two claims that could not bear weight.

What remains is sufficient. Voluntary slow breathing is the most accessible deliberate input to autonomic state available to a working adult, with measured changes in heart rate variability indices during practice, after a single session, and following repeated practice across 223 studies. The mechanism most strongly supported is baroreflex resonance near six breaths per minute. Brief daily practice has produced measurable change in mood and breathing rate. A small change in a resting autonomic index, low frequency heart rate variability, appeared in one trial using longer twice-daily sessions, while the one trial of brief once-daily practice found no change in heart rate variability. Nothing in the literature indicates that longer or less frequent sessions produce more. Practice anchored to existing daily cues is more likely to become automatic, according to habit research, although this has not been tested for breathing practice specifically, and sustained practice is what accumulates.

What has been removed matters as much. Brief frequent practice has not been shown to produce greater adaptation than longer infrequent practice, and the single direct test found no difference. Heart rate variability driven by paced breathing is not a measure of vagal tone, and describing it that way invites exactly the challenge a practitioner cannot answer. Polyvagal theory, whatever its clinical utility as a heuristic, cannot serve as the physiological foundation for this or any methodology, following a 39-author evaluation concluding it is untenable.

The argument that survives is more modest and more useful than the one it replaces. Regulation is trainable. The input is available without equipment, in the middle of an ordinary day. The adaptation is real and small. The practice that works is the one a person will actually keep.

Two research questions would materially advance this. Whether brief frequent practice differs from longer infrequent practice in physiological adaptation at ratios resembling real-world use, which has not been tested. And whether training regulation upstream is more efficient than targeting downstream complaints directly, which has not been compared. Both are answerable.

Disclosure and Notice

Competing interests. The author founded and operates Fulcrum Performance Lab, LLC, a human performance business, and is the creator of the Morris Method, a commercial methodology. The author is the author of the book *Stress: Our Human Superpower* and has a commercial interest

in Nova Breath, an application that delivers breathing protocols and reports heart rate variability trends. This paper describes a methodology the author sells and corrects claims the author previously published. Readers should weigh those interests in both directions when assessing it.

Status of this document. This is a white paper. It has not been peer reviewed and does not report original empirical research. Nothing in it should be represented as a peer-reviewed finding.

Not medical advice. This paper is educational and addresses wellness and human performance in generally healthy adults. It is not medical advice, does not diagnose or treat any condition, and is not a substitute for care from a qualified professional.

Breathing practice safety. Slow breathing practices are generally well tolerated by healthy adults but are not appropriate for everyone. They should not be performed while driving, operating machinery, or in or near water, or in any situation where lightheadedness would be hazardous. Anyone who is pregnant, or who has a cardiovascular, respiratory, neurological or psychiatric condition, or a history of fainting or seizures, should consult a qualified healthcare professional before beginning any such practice. Practice should stop immediately if faintness, chest discomfort, palpitations or any other concerning symptom occurs.

References

Bibliographic details below were checked against publisher pages, Crossref, OpenAlex or Semantic Scholar records during preparation. The verification column records what was confirmed and notes anything that could not be checked.

Reference	Type and scope	Link	Verification
Bowles NI, Davies JN, Strohmaier S, Galante J, Van Dam NT. Too much or not enough of a good thing? Reconciling differing perspectives on the optimal dose of mindfulness meditation and advancing a dose response research agenda. J Contemplative Stud. 2026.	Review and research agenda	doi 10.57010/CAXS3076	Confirmed on the journal site. Not indexed in PubMed, and no volume, issue or page numbers are assigned. Mindfulness rather than breathing.
Castro Ribeiro T, Sobregrau Sangra P, Garcia Pages E, Badiella L, Lopez Barbeito B, Aguilo S, Aguilo J. Assessing effectiveness of heart rate variability biofeedback to mitigate mental health symptoms: a pilot study. Front Physiol. 2023;14:1147260.	Single-arm pilot, n = 21	doi 10.3389/fphys.2023.1147260	Confirmed on the publisher page, including the protocol and the 62 percent home-practice adherence figure. No control group; all participants female.

Cepeda NJ, Pashler H, Vul E, Wixted JT, Rohrer D. Distributed practice in verbal recall tasks: a review and quantitative synthesis. Psychol Bull. 2006;132(3):354-380.	Quantitative synthesis, 317 experiments	doi 10.1037/0033-2909.132.3.354, PMID 16719566	Confirmed, including the PMID. Verbal recall only; cited here to explain why this literature does not transfer to a training calendar.
Dolgilevica K, Grunfeld E, Derakshan N. Heart rate variability biofeedback training can improve menopausal symptoms and psychological well being in women with a diagnosis of primary breast cancer: a longitudinal randomized controlled trial. Curr Oncol. 2025;32(3):150.	Three-arm RCT, n = 60	doi 10.3390/curroncol32030150, PMID 40136354	Confirmed on the publisher page, including the full protocol and d equals 0.33. Clinical population; cited for protocol and autonomic change, not for generalization.
Donovan JJ, Radosevich DJ. A meta analytic review of the distribution of practice effect: now you see it, now you don't. J Appl Psychol. 1999;84(5):795-805.	Meta-analysis, 63 studies, 112 effects	doi 10.1037/0021-9010.84.5.795	Confirmed, including the rest-interval moderator. Not indexed in PubMed.
Gollwitzer PM, Sheeran P. Implementation intentions and goal achievement: a meta analysis of effects and processes. Adv Exp Soc Psychol. 2006;38:69-119.	Meta-analysis, 94 independent tests	doi 10.1016/S0065-2601(06)38002-1	Bibliographic details confirmed via an institutional repository record. The effect size and test count are as reported in secondary summaries, because the full text could not be accessed. Not indexed in PubMed. Predates current publication bias correction norms.
Grossman P. Fundamental challenges and likely refutations of the five basic premises of the polyvagal theory. Biol Psychol. 2023;180:108589.	Critical review	doi 10.1016/j.biopsycho.2023.108589, PMID 37230290	Confirmed. Single-author critique; its anatomical and comparative claims are independently established in the two sources below.
Grossman P, Taylor EW. Toward understanding respiratory sinus arrhythmia: relations to cardiac vagal tone, evolution and biobehavioral functions. Biol Psychol. 2007;74(2):263-285.	Critical review	doi 10.1016/j.biopsycho.2005.11.014, PMID 17081672	Confirmed. The foundational methodological critique, and directly relevant to any methodology built on heart rate variability.

Grossman P, Ackland GL, Allen AM, et al. Why the polyvagal theory is untenable: an international expert evaluation of the polyvagal theory. Clin Neuropsychiatry. 2026;23(1):100-112.	Multi-author expert evaluation, 39 authors	doi 10.36131/cnforitieditore20260110, PMID 41768017	Confirmed, including the PMID. Commentary rather than new data, in a modest-impact journal, but the author list is the relevant specialist community.
Laborde S, Allen MS, Borges U, Dosseville F, Hosang TJ, Iskra M, Mosley E, Salvotti C, Spolverato L, Zammit N, Javelle F. Effects of voluntary slow breathing on heart rate and heart rate variability: a systematic review and a meta-analysis. Neurosci Biobehav Rev. 2022;138:104711.	Systematic review and meta-analysis, 223 studies	doi 10.1016/j.neubiorev.2022.104711, PMID 35623448	Confirmed from the accepted author manuscript, including pooled Hedges g values, heterogeneity, and Cochrane RoB2 ratings of high risk in 215 studies, some concerns in 5, and low risk in 1. No certainty-of-evidence assessment was performed. Figures come from the accepted manuscript rather than the published version. PROSPERO CRD42020173255.
Lalanza JF, Lorente S, Bullich R, Garcia C, Losilla JM, Capdevila L. Methods for heart rate variability biofeedback: a systematic review and guidelines. Appl Psychophysiol Biofeedback. 2023;48(3):275-297.	Systematic review, 143 studies	doi 10.1007/s10484-023-09582-6, PMID 36917418	Confirmed on the publisher page, including the finding on reporting quality.
Lally P, van Jaarsveld CHM, Potts HWW, Wardle J. How are habits formed: modelling habit formation in the real world. Eur J Soc Psychol. 2010;40(6):998-1009.	Observational longitudinal, 96 enrolled, 82 analysed	doi 10.1002/ejsp.674	Confirmed. Print issue year is 2010; the online version appeared in 2009, which is why both years circulate. The median of 66 days to 95 percent of asymptote is confirmed as the paper's own figure, computed across the 39 participants whose data fitted the model. Not indexed in PubMed.
Lehrer PM, Gevirtz R. Heart rate variability biofeedback: how and why does it work? Front Psychol. 2014;5:756.	Mechanistic review	doi 10.3389/fpsyg.2014.00756, PMID 25101026	Confirmed from the full text, including the resonance frequency and the authors' own caution that few areas have extensive controlled-study support.

Lehrer P, Kaur K, Sharma A, Shah K, Huseby R, Bhavsar J, Sgobba P, Zhang Y. Heart rate variability biofeedback improves emotional and physical health and performance: a systematic review and meta analysis. <i>Appl Psychophysiol Biofeedback</i> . 2020;45(3):109-129.	Systematic review and meta-analysis, 58 studies from 1,868 records identified	doi 10.1007/s10484-020-09466-z, PMID 32385728	Confirmed on the publisher page, including the null moderation by session count and number of weeks. The abstract reports no numeric pooled effect size, so none is quoted here.
Mapelli M, Salvioni E, Mattavelli I, Grilli G, Zerboni G, Nava A, et al. Nasal vs. oral breathing strategies in healthy individuals during cardiorespiratory exercise testing. <i>PLoS One</i> . 2025;20(7):e0326661.	Randomized crossover, n = 12	doi 10.1371/journal.pone.0326661, PMID 40668801	Confirmed on the publisher page. Small sample; cited for the maximal-effort decrement.
Melnychuk MC, Dockree PM, O'Connell RG, Murphy PR, Balsters JH, Robertson IH. Coupling of respiration and attention via the locus coeruleus: effects of meditation and pranayama. <i>Psychophysiology</i> . 2018;55(9):e13091.	Empirical plus model, n = 14	doi 10.1111/psyp.13091, PMID 29682753	Confirmed from the full text. Exploratory; brainstem imaging at this site is artifact-prone.
Menuet C, Ben-Tal A, Linossier A, et al. Redefining respiratory sinus arrhythmia as respiratory heart rate variability: an international expert recommendation for terminological clarity. <i>Nat Rev Cardiol</i> . 2025;22(12):978-984.	Expert recommendation, 29 authors	doi 10.1038/s41569-025-01160-z, PMID 40328963	Confirmed on the publisher page, including the statement that the amplitude is not a measure of vagal tone, and the full 29-author list via Crossref. Consensus opinion, not primary data.
Neuhuber WL, Berthoud HR. Functional anatomy of the vagus system: how does the polyvagal theory comply? <i>Biol Psychol</i> . 2022;174:108425.	Neuroanatomical review	doi 10.1016/j.biopsycho.2022.108425, PMID 36100134	Confirmed. A corrigendum exists, <i>Biol Psychol</i> 2023;179:108554, correcting one citation without altering the conclusions.
Noble DJ, Hochman S. Hypothesis: pulmonary afferent activity patterns during slow, deep breathing contribute to the neural induction of physiological relaxation. <i>Front Physiol</i> . 2019;10:1176.	Hypothesis paper, no new data	doi 10.3389/fphys.2019.01176, PMID 31572221	Confirmed. Cited only as a proposed mechanism.

Porges SW. The polyvagal perspective. <i>Biol Psychol.</i> 2007;74(2):116-143.	Theoretical paper	doi 10.1016/j.biopsycho.2006.06.009, PMID 17049418	Confirmed. Online first 2006; cite 2007. Cited here as the position under challenge, not as mechanism.
Porges SW. Polyvagal theory: current status, clinical applications, and future directions. <i>Clin Neuropsychiatry.</i> 2025;22(3):169-184.	Review and defense	doi 10.36131/cnfioritieditore20250301, PMID 40735382	Confirmed. Included for balance; this is the paper the 2026 evaluation responds to.
Riordan KM, Simonsson O, Frye C, Vack NJ, Sachs J, Fitch D, Goldman RI, Chiang ES, Dahl CJ, Davidson RJ, Goldberg SB. How often should I meditate? A randomized trial examining the role of meditation frequency when total amount of meditation is held constant. <i>J Couns Psychol.</i> 2024;71(2):104-114.	RCT, n = 351	doi 10.1037/cou0000725, PMID 38376930	Confirmed, including the full author list, both conditions, the effect sizes from 0.12 to 0.63 across outcomes, and p values from .158 to .729 for the frequency comparison. Participants were undergraduates with clinically elevated depression or anxiety, which limits generalization.
Russo MA, Santarelli DM, O'Rourke D. The physiological effects of slow breathing in the healthy human. <i>Breathe (Sheff).</i> 2017;13(4):298-309.	Narrative review	doi 10.1183/20734735.009817, PMID 29209423	Confirmed. Educational journal; not a systematic review.
Shaffer F, Ginsberg JP. An overview of heart rate variability metrics and norms. <i>Front Public Health.</i> 2017;5:258.	Narrative review	doi 10.3389/fpubh.2017.00258, PMID 29034226	Confirmed.
Snoeck T, Paillaugue F, Probyn S. Nasal versus buccal breathing and non-invasive indices of vascular function in healthy young adults: an observational crossover study. <i>Vasc Health Risk Manag.</i> 2026;22:1-9.	Observational crossover, n = 45	doi 10.2147/VHRM.S570629, PMID 42099659	Confirmed on the publisher page. Young men only; no autonomic or blood pressure measures were taken.
Strohmaier S. The relationship between doses of mindfulness based programs and depression, anxiety, stress, and mindfulness: a dose response meta regression of randomized controlled trials. <i>Mindfulness.</i> 2020;11(6):1315-1335.	Meta-regression, 203 RCTs, n = 15,971	doi 10.1007/s12671-020-01319-4	Bibliographic details confirmed on the publisher page. The participant total could not be checked against the full text. Not indexed in PubMed. Observational across studies; supports sufficiency of smaller doses, not superiority.

Strohmaier S, Jones FW, Cane JE. Effects of length of mindfulness practice on mindfulness, depression, anxiety, and stress: a randomized controlled experiment. <i>Mindfulness</i> . 2021;12(1):198-214.	Three-arm RCT, n = 71	doi 10.1007/s12671-020-01512-5	Corrected during verification: this article is <i>Mindfulness</i> 2021;12(1):198-214, not volume 11 as first recorded. Online first October 2020. Not indexed in PubMed. Varies duration only; both arms practised four times over two weeks. Small sample of novices.
Taylor EW, Wang T, Leite CAC. An overview of the phylogeny of cardiorespiratory control in vertebrates with some reflections on the polyvagal theory. <i>Biol Psychol</i> . 2022;172:108382.	Comparative physiology review with primary imaging	doi 10.1016/j.biopsycho.2022.108382, PMID 35777519	Confirmed on the publisher page.
Ulrich-Lai YM, Herman JP. Neural regulation of endocrine and autonomic stress responses. <i>Nat Rev Neurosci</i> . 2009;10(6):397-409.	Review	doi 10.1038/nrn2647, PMID 19469025	Confirmed.
Vaschillo EG, Vaschillo B, Lehrer PM. Characteristics of resonance in heart rate variability stimulated by biofeedback. <i>Appl Psychophysiol Biofeedback</i> . 2006;31(2):129-142.	Experimental study, n = 56 (32 with asthma, 24 healthy)	doi 10.1007/s10484-006-9009-3, PMID 16838124	Abstract confirmed on the publisher page, including the sample and the relationships with height and sex and the stability across ten sessions. The specific numeric resonance frequency sits in the paywalled results, so no single frequency value is attributed to this paper.
Watso JC, Cuba JN, Boutwell SL, Moss JE, Bowerfind AK, Fernandez IM, Cassette JM, May AM, Kirk KF. Acute nasal breathing lowers diastolic blood pressure and increases parasympathetic contributions to heart rate variability in young adults. <i>Am J Physiol Regul Integr Comp Physiol</i> . 2023;325(6):R797-R808.	Within-subject crossover, n = 20	doi 10.1152/ajpregu.00148.2023, PMID 37867476	Confirmed, including effect sizes. Five-minute exposure, narrow age band, and the variability findings sit at p equals 0.04.

Yackle K, Schwarz LA, Kam K, Sorokin JM, Huguenard JR, Feldman JL, Luo L, Krasnow MA. Breathing control center neurons that promote arousal in mice. <i>Science</i> . 2017;355(6332):1411-1415.	Experimental animal study, mice	doi 10.1126/science.aai7984, PMID 28360327	Confirmed. Mouse model, and frequently overcited in this field.
Yasuma F, Hayano J. Respiratory sinus arrhythmia: why does the heartbeat synchronize with respiratory rhythm? <i>Chest</i> . 2004;125(2):683-690.	Narrative review	doi 10.1378/chest.125.2.683, PMID 14769752	Confirmed. The functional account it advances has been contested in later work.
Yilmaz Balban M, Neri E, Kogon MM, Weed L, Nouriani B, Jo B, Holl G, Zeitzer JM, Spiegel D, Huberman AD. Brief structured respiration practices enhance mood and reduce physiological arousal. <i>Cell Rep Med</i> . 2023;4(1):100895.	RCT, 108 enrolled (100 in the physiology analysis), five minutes once daily for one month	doi 10.1016/j.xcrm.2022.100895, PMID 36630953	Confirmed from the full text, including arm sizes and the finding of no significant change in heart rate variability or resting heart rate across the month. Trial registration NCT05304000. The paper reports mean change with standard deviations rather than Cohen's d.
Zelano C, Jiang H, Zhou G, Arora N, Schuele S, Rosenow J, Gottfried JA. Nasal respiration entrains human limbic oscillations and modulates cognitive function. <i>J Neurosci</i> . 2016;36(49):12448-12467.	Intracranial EEG, 7 patients, plus behavioural testing, 107 participants	doi 10.1523/JNEUROSCI.2586-16.2016, PMID 27927961	Confirmed on the publisher page. The cognitive effect concerns respiratory phase, not a general benefit of nasal breathing.