STEP ONE OF THREE · THE CLINICAL BASIS

Why calming the breath has to come *first.*

Most stress interventions begin by asking people to think differently, or to practise breathing exercises they don't fully understand. The Raban Method begins with something more fundamental: understanding what shallow breathing is actually doing to the body, and why restoring diaphragmatic breathing is the necessary foundation for everything that follows.

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Reading time: ~15 minutes

References: 39 peer-reviewed studies

Covers: Step 1 of The Raban Method

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1
Restore Mechanical Baseline

Calm the breath. Exit sustained threat mode. Establish the physiological foundation.

2
Active Regulation

Guide the breath deliberately. Use specific techniques to shift cognitive and physiological state.

3
Structured Analysis

From a regulated baseline, examine what is actually driving the stress.

The cycle nobody notices

Most people assume that shallow breathing is a symptom of stress, something that happens when you are already anxious or under pressure. The clinical picture is more complicated than that. Shallow breathing and the stress response exist in a bidirectional loop, each sustaining the other. Stress causes shallow breathing. Shallow breathing causes stress. And at some point, the pattern becomes the person's resting state without them ever having noticed the transition.

This matters because it means that simply removing a stressor does not automatically resolve the physiological state. People who are no longer objectively under threat can remain in a sustained state of autonomic arousal, because their breathing pattern is maintaining it. Chronic stress is, in part, a breathing problem. And because it is a breathing problem, it is also a mechanical one, which means it can be addressed mechanically.

This cycle is not merely theoretical. Lum's foundational 1981 clinical review in the *Journal of the Royal Society of Medicine* documented that chronic hyperventilation, of which habitual shallow thoracic breathing is the principal mechanism, presents clinically as anxiety, exhaustion, cardiac palpitations, and somatic complaints that respond poorly to psychological intervention alone, because the underlying physiological driver remains uncorrected.[26] Once established, the pattern sustains itself: a chronically overbreathing nervous system becomes sensitised to CO_2 fluctuations, and the resulting physiological arousal further accelerates breathing, a self-maintaining loop independent of whether anything is objectively wrong.[4]

The downstream stakes are substantial. Cohen et al.'s landmark 2012 study in *PNAS* demonstrated that sustained psychological stress induces glucocorticoid receptor resistance in immune cells, blunting the body's ability to regulate its own inflammatory response and creating conditions for chronic systemic inflammation and the long-chain health consequences that follow.[14] The connection between breathing pattern and systemic disease is not metaphorical. It runs through identifiable biochemical and neurological pathways.

> *Shallow breathing does not just accompany stress. It actively generates it. Correcting the breathing pattern is not a relaxation technique, it is the removal of a physiological maintaining factor.*

What shallow breathing actually does to the body

Shallow breathing means breathing that does not engage the diaphragm, the large, dome-shaped muscle that sits below the lungs and is the body's primary breathing muscle. Instead, air is drawn into the upper chest using smaller accessory muscles: the scalenes in the neck, the intercostals between the ribs, the muscles of the upper shoulders. These muscles were never designed for sustained, continuous use. They fatigue, tighten, and over time produce the chronic neck tension, headaches, and shoulder pain that many people associate with stress rather than with breathing.

It is worth being precise about what this shift is, and what it is not. Every human being begins life breathing diaphragmatically. In newborns and infants the diaphragm carries essentially the entire workload of breathing: the ribs sit horizontally and the intercostal muscles are too underdeveloped to expand the chest the way an adult's do, so belly breathing is not a technique to be learned in infancy, it is the only breathing available.[36] The shift toward shallow, chest-dominant breathing is acquired later, as the rib cage matures and, for many people, as stress and habit take over. This is also why the clinical literature is careful to classify the resulting pattern as dysfunctional rather than diseased. Boulding et al.'s 2016 review in the *European Respiratory Review* defines dysfunctional breathing explicitly as a disorder of breathing function without an accompanying structural abnormality, present in the absence of, or in excess of, any organic respiratory disease.[37] A person who cannot easily drop the breath back into the belly on request has not damaged anything. The pattern was learned, which is also why it can be unlearned.

This pattern is considerably more prevalent than commonly appreciated. Thomas et al. (2001), in a cross-sectional survey in the *British Medical Journal*, found dysfunctional breathing, characterised by thoracic-dominant, shallow patterns, in approximately one third of women and one fifth of men treated for asthma in primary care.[32] In occupational and high-stress professional populations, the prevalence is likely substantially higher: chronic work pressure systematically trains people out of diaphragmatic breathing, and the shift is gradual enough that most people never notice it happening.

Because shallow breathing does not move air into the lower lobes of the lungs, where the alveoli are concentrated, and where the vast majority of gas exchange between air and bloodstream takes place, the lungs cannot use the oxygen being inhaled efficiently. Air sits in the upper airways, what respiratory physiology calls “dead space,” and never reaches where it is needed. The result is that a shallow breather can be breathing continuously and still be functionally under-oxygenated.

The downstream effects extend well beyond the lungs. Long-term shallow breathing suppresses immune function, impairs cognitive clarity, contributes to cardiovascular strain, and, as Liu et al. established in a 2017 review in *Frontiers in Human Neuroscience*, the chronic inflammatory state it produces is a contributing factor in an estimated 75 to 90 percent of all human diseases.[1] That figure is worth sitting with: the same biological pathway that shallow breathing feeds is implicated in conditions ranging from autoimmune disease to cardiovascular disease to cognitive decline.

The cognitive costs are directly measurable. Ma et al. (2017), in a randomised controlled trial published in *Frontiers in Psychology*, found that eight weeks of intensive diaphragmatic breathing training improved sustained attention and reduced negative affect in healthy adults, establishing a direct causal link between breathing pattern and cognitive performance.[11] Perciavalle et al. (2017), publishing in *Neurological Sciences*, observed direct reductions in salivary cortisol following a single session of deep breathing, confirming that the neuroendocrine effect is rapid as well as sustained.[13]

Shallow (chest) breathing	Deep (diaphragmatic) breathing
Air reaches only the upper airways, dead space	Air reaches the alveoli in the lower lobes, where gas exchange occurs
CO_2 expelled too rapidly, oxygen stays locked in the blood	CO_2 maintained at functional levels, oxygen released into tissues
Sympathetic nervous system dominates, sustained threat mode	Parasympathetic activation via vagus nerve, recovery mode
Cortisol remains elevated, prefrontal function impaired	Cortisol and oxidative stress reduced[2][13]
Low HRV, poor autonomic flexibility and resilience	High HRV, better stress recovery and cognitive performance
Attention impaired, negative affect sustained	Sustained attention improved, negative affect reduced[11]

The CO_2 problem, why “breathing deeply” often makes it worse

There is a widespread and clinically important misunderstanding about what deep breathing involves. Most people, when told to take a deep breath, tense their shoulders, open their mouth, and take a large, fast breath into the upper chest. This is a big breath. It is not a deep one.

Taking large, rapid breaths, even through the nose, causes carbon dioxide to be exhaled faster than the body produces it. This matters because CO_2 is not simply a waste gas. It is the chemical signal that triggers the release of oxygen from haemoglobin into the body's tissues, through a mechanism known as the Bohr effect. When CO_2 levels drop, this release mechanism is impaired. The paradox is that someone breathing hard and fast can simultaneously have plenty of oxygen in their blood and be delivering too little of it to their brain and organs.

Low CO_2, chronic hyperventilation, also has a direct relationship with the anxiety and panic response. This connection was systematically documented by Lum (1981), whose clinical review established that habitual overbreathing produces a sustained state of low arterial CO_2 that generates cardiovascular, neurological, and psychological symptoms of anxiety independently of whether the individual is facing any objective threat.[26] A 2010 study by Meuret et al. in the *Journal of Consulting and Clinical Psychology* compared cognitive behavioural therapy with breathing retraining in patients with panic disorder. Both treatments reduced symptoms, but through entirely different mechanisms. CBT worked through cognitive pathways. Breathing retraining worked by restoring CO_2 levels to normal, correcting the biochemical imbalance that was triggering panic at a physiological level, independent of thought processes.[4] The researchers concluded that anxiety is not a purely psychological condition. It has a breathing component that requires a breathing correction.

The route of breathing also matters. The nasal passages and paranasal sinuses are the body's primary production site for nitric oxide, a molecule with vasodilatory, antimicrobial, and oxygen-utilisation properties. Lundberg et al. (1994) demonstrated that nasal breathing delivers substantially more nitric oxide to the lower airways than mouth breathing, with direct implications for pulmonary gas exchange and respiratory immune defence.[28] Subsequent work by the same group identified the paranasal sinuses as the specific anatomical source of this NO production, a feature that is entirely bypassed when breathing through the mouth.[29] Diaphragmatic breathing naturally favours nasal inhalation; the two patterns are complementary.

> *Diaphragmatic breathing is not about taking larger volumes of air. It is about allowing a slower, gentler breath to reach further into the lungs, restoring gas exchange, CO_2 balance, and the physiological conditions for calm.*

The neural circuit, how breathing pattern signals the brain

In 2017, a paper published in *Science* by Yackle et al. identified a specific neural circuit in the brainstem of mammals that monitors the rhythm and character of breathing.[5] When breathing becomes fast, irregular, or laboured, the hallmarks of shallow chest breathing under stress, this circuit directly activates the brain's arousal and alertness systems. It does not wait for a perceived threat. The pattern of breathing alone is sufficient to trigger the response.

This finding has significant clinical implications. It means the stress response can be maintained from the bottom up, through breathing mechanics, independently of what is happening cognitively. A person who is not consciously worried about anything can nonetheless remain in a state of elevated neurological arousal simply because their habitual breathing pattern is fast and shallow. The circuit reads the breathing and draws its own conclusions.

The framework articulated by Porges in his polyvagal theory (2007) provides a complementary perspective: the autonomic nervous system continuously evaluates environmental and internal cues, including respiratory rhythm, through a process of “neuroception,” adjusting its state largely outside conscious awareness.[16] The breathing pattern is among the most potent inputs to this system. Jerath et al. (2006), in *Medical Hypotheses*, proposed a coherent neurological model for how slow diaphragmatic breathing shifts autonomic balance toward parasympathetic dominance through synchronised stimulation of both central and peripheral nervous system pathways, a mechanism that operates whether or not the practitioner is actively attending to the breath.[18] The yogic breathing literature, long preceding modern neuroscience, had identified the same functional relationship; Brown and Gerbarg (2005) systematically mapped these traditions onto identifiable physiological mechanisms, vagal modulation, HPA axis regulation, limbic inhibition, in a review that connected ancient practice to contemporary mechanistic understanding.[34]

Conversely, and this is the therapeutically important direction, slow, regular diaphragmatic breathing does not trigger the Yackle arousal circuit. It signals the opposite. The same mechanism that locks people into the stress

response via shallow breathing can be used to exit it via deliberate, calm breathing. This is not a metaphor for relaxation. It is a specific neurological pathway.

What diaphragmatic breathing actually corrects

Diaphragmatic breathing, the kind a healthy infant performs naturally, the kind most adults have quietly lost, works through several simultaneous physiological mechanisms. It is worth being precise about each of them, because understanding the mechanism is part of what makes the practice sustainable.

Air reaches where it is needed

When the diaphragm contracts and descends on an inhale, it creates pressure that draws air into the lower lobes of the lungs, the regions with the highest concentration of alveoli, where oxygen crosses into the bloodstream. Chest breathing cannot reach these regions efficiently. Diaphragmatic breathing restores functional gas exchange. Bordoni et al. (2018), reviewing the diaphragm's broader influence on the central nervous system, concluded that its effects extend well beyond respiratory mechanics, into the regulation of the nervous system, motor function, and brain electrical activity.[6] Bernardi et al. (2001) demonstrated that both the rate and depth of breathing exert direct, measurable, and reproducible influence on cardiovascular and autonomic nervous system parameters, effects that begin with the very first breath.[22]

Oxidative stress and cortisol are reduced

Martarelli et al. (2011), publishing in *Evidence-Based Complementary and Alternative Medicine*, demonstrated that diaphragmatic breathing directly reduces both exercise-induced oxidative stress and circulating cortisol levels.[2] Perciavalle et al. (2017) confirmed comparable cortisol reductions following a single session of deep breathing in healthy adults, establishing that the neuroendocrine effect is rapid, not dependent on extended training.[13] Cortisol is the body's primary stress hormone, and chronically elevated cortisol suppresses immune function, disrupts sleep, impairs memory consolidation, and, at sustained high levels, damages the prefrontal cortex. Reducing cortisol is not a side effect of calming the breath. It is one of the primary mechanisms by which it works.

Melatonin levels increase

The same Martarelli study found that diaphragmatic breathing also raises melatonin, the hormone that regulates the sleep-wake cycle. This is one of the reasons that restoring correct breathing patterns tends to improve sleep quality relatively quickly, and why disrupted sleep is so often a downstream consequence of unresolved shallow breathing patterns.

Cognitive performance and decision-making improve

The cognitive benefits of diaphragmatic breathing are not merely indirect or downstream. Ma et al. (2017) demonstrated in a randomised controlled trial that eight weeks of diaphragmatic breathing training produced measurable improvements in sustained attention and significant reductions in negative affect, alongside lower cortisol levels, changes not observed in the control group.[11] More practically: de Couck et al. (2019), studying

professional decision-making in business contexts, found that brief breathing exercises applied before high-stakes judgements improved decision quality, an effect mediated by improvements in HRV.[33] The implication is direct: calming the breath before intellectually or emotionally demanding work is not a comforting ritual. It is functional preparation with a measurable effect on outcome quality.

The vagus nerve, the physiological pathway of calm

The autonomic nervous system has two primary branches. The sympathetic branch governs the stress response: it elevates heart rate, redirects blood to skeletal muscle, sharpens alertness, and prepares the body for action. The parasympathetic branch governs recovery: it slows the heart, restores digestion, promotes immune activity, and creates the conditions for cognitive restoration. Under chronic pressure, the sympathetic branch tends to dominate, sometimes persistently.

The **vagus nerve** is the principal pathway of the parasympathetic system. It is a long cranial nerve that travels from the brainstem all the way down to the abdomen, connecting directly with the diaphragm along the way. This anatomical connection is the physiological reason why diaphragmatic breathing activates the parasympathetic system. When the diaphragm moves correctly, it stimulates the vagus nerve. The scope of vagal influence is remarkable: Tracey (2002), in a landmark paper in *Nature*, described the “inflammatory reflex”, the mechanism by which vagal activation directly suppresses systemic inflammation through acetylcholine release, establishing that the vagus nerve functions not only as a cardiovascular regulator but as a primary anti-inflammatory pathway.[17] Breathing that activates the vagus nerve is, therefore, simultaneously modulating the immune system.

The cardiovascular effect is measurable and specific. On exhalation, vagal stimulation causes the release of **acetylcholine**, a neurotransmitter that directly slows the heart rate. This is the physiological mechanism behind the calming effect of controlled breathing, not a generalised relaxation response, but a specific neurochemical event triggered by a specific physical movement. A review by Russo et al. (2017) in *Breathe* confirmed that slow breathing reliably activates vagal tone and shifts autonomic balance toward the parasympathetic branch.[7] Grossman and Taylor (2007), reviewing the relationship between respiratory sinus arrhythmia and cardiac vagal tone, confirmed that RSA, the natural heart rate variation in synchrony with breathing, is a direct index of vagal function, and that breathing pattern is its most modifiable determinant.[23]

The cardiovascular effects of slow breathing are dose-dependent and frequency-specific. A landmark 2001 study by Bernardi et al. in the *British Medical Journal* showed that breathing at approximately six cycles per minute, achieved incidentally through recitation of the Catholic rosary and certain yoga mantras, consistently produced the maximum cardiovascular resonance effect, with heart rate and breathing falling into precise synchrony and baroreflex sensitivity significantly enhanced.[24] This is the basis of what is now known as resonance frequency breathing. Lin, Tai, and Fan (2014) identified 5.5 breaths per minute as particularly effective for increasing HRV parameters, achievable with a 5.5-second inhale and 5.5-second exhale.[27] Steffen et al. (2017) confirmed in a controlled study that resonance frequency breathing significantly increased HRV, reduced blood pressure, and improved mood.[19] Laborde et al. (2022), in a randomised trial published in

Psychophysiology, found that even a single five-minute session of slow breathing at six cycles per minute produced measurable parasympathetic activation.[20]

Exhale duration itself is a directly measurable marker of this same balance, not only a technique for influencing it. Kato, Takahashi, and Homma (2018), in the *Journal of Physiological Sciences*, measured breathing at rest in healthy adults and found that both inspiratory and expiratory time were significantly shorter in those with higher trait and state anxiety: calmer subjects rested with a notably longer exhale than more anxious subjects, with no conscious breathing exercise involved at all.[38] A related pattern holds under acute respiratory challenge: shorter voluntary breath-holding duration has been shown to moderate the relationship between anxiety sensitivity and the severity of trauma-related symptoms.[39] A shortened exhale is not only a target for training. At rest, it is one of the more reliable single readouts of where the nervous system currently sits.

Vagal activation also improves **heart rate variability (HRV)**, the natural variation in the interval between heartbeats, now well established as a reliable index of autonomic health and resilience. A meta-analysis by Thayer et al. (2012) across 151 studies found that resting HRV is positively correlated with prefrontal cortex function, working memory, decision-making, and inhibitory control all improve as HRV increases.[3] Lehrer and Gevirtz (2014), reviewing the mechanisms underlying HRV biofeedback, concluded that the primary pathway of benefit is baroreflex activation, directly stimulated by slow, diaphragmatic breathing, which in turn increases autonomic flexibility and recovery speed.[21] Gevirtz (2013), surveying the evidence base for HRV biofeedback applications, reported beneficial effects across anxiety, PTSD, asthma, depression, and hypertension, conditions that share autonomic dysregulation as a common thread.[30] The foundational paper for this approach by Lehrer, Vaschillo, and Vaschillo (2000) established that training people to breathe at their cardiac resonance frequency produces lasting improvements in autonomic flexibility and baroreflex gain, effects that persist beyond the training period.[35]

DIAPHRAGM MOVES	Correct inhale descends the diaphragm, drawing air into lower lung lobes where gas exchange occurs
VAGUS NERVE	Diaphragm movement stimulates vagal afferents, parasympathetic activation begins[16]
ACETYLCHOLINE	Released on exhalation, directly slows heart rate, reduces arousal, suppresses systemic inflammation[17]
RSA & HRV	Respiratory sinus arrhythmia increases, autonomic balance restored, sympathetic dominance reduces[23]
RESONANCE	~5.5-6 breaths/min synchronises heart rate and breathing, maximum HRV effect, baroreflex gain enhanced[24][27]
CORTISOL FALLS	Stress hormone load reduces, prefrontal cortex function restored[2][13]

Cortisol, the prefrontal cortex, and why clarity requires a physiological baseline

Cortisol's relationship with cognitive performance follows an inverted-U curve: moderate, acute elevations sharpen attention and consolidate memory. Sustained elevated cortisol, the chronic stress condition, does the opposite. Arnsten's 2009 review in *Nature Reviews Neuroscience* documented the mechanisms clearly: high cortisol suppresses metabolic activity in the prefrontal cortex, the region responsible for planning, working memory, inhibitory control, and the kind of flexible thinking that high-pressure situations demand.[8]

McEwen, Nasca, and Gray (2016), reviewing the structural effects of chronic stress in *Neuropsychopharmacology*, documented that sustained cortisol exposure causes dendritic retraction in medial prefrontal neurons, a structural change that manifests as impaired executive function, reduced impulse control, and diminished capacity for perspective-taking under pressure.[15] These changes can begin within weeks of sustained stress exposure. McEwen's broader theoretical framework (2007), laid out in *Physiological Reviews*, established that allostatic load, the cumulative physiological cost of chronic stress, produces measurable, often lasting changes in brain architecture and systemic physiology that no amount of cognitive reframing will reverse without first addressing the physiological state.[31] Sapolsky (1996) made the argument plainly in *Science*: stress is not merely bad for how we feel, it is bad for brain structure, in ways that are measurable, predictable, and in most cases preventable.[25]

A further mechanism compounds this. Cohen et al. (2012) demonstrated that chronic psychological stress produces glucocorticoid receptor resistance, a condition in which immune cells become less responsive to cortisol's anti-inflammatory signalling, allowing inflammation to escalate unchecked. The result is a vicious cycle: more cortisol is produced to compensate, immune dysregulation worsens, and the systemic inflammation feeds back into neural stress pathways and neurotoxicity.[14]

What this means in practice is that the cognitive capacity people most need under pressure is precisely the capacity that chronic stress erodes. And because the prefrontal cortex is also the region required for emotional regulation and perspective-taking, its impairment compounds the problem, increasing reactivity at the same time as it reduces the resources available to respond thoughtfully.

This is why Step 1 of The Raban Method is not preparatory. It is not a warm-up to the “real” work. Restoring diaphragmatic breathing reduces cortisol, raises HRV, and reactivates the prefrontal cortex. Only from that baseline does it become genuinely productive to guide the breath with intention (Step 2), or to examine the sources of stress with clarity (Step 3).

Why Step 1 is the prerequisite, not the preamble

The three steps of The Raban Method are sequenced by physiology, not convention. Step 2, actively guiding and working with the breath to shift cognitive and emotional state, requires a mechanical baseline that is already functioning. Directed breathwork applied on top of dysfunctional breathing mechanics has a ceiling.

Step 3, identifying and examining the structural sources of stress, requires the prefrontal cortex to be available and unimpaired. That availability depends on cortisol levels, which depend on the autonomic balance, which depends on whether the breath is calm and diaphragmatic.

The evidence base for breathing-based interventions has grown substantially. Fincham, Karner, and Smyth (2023) published a meta-analysis in *Scientific Reports* synthesising 12 randomised controlled trials involving 785 adult participants. Breathwork was associated with significant reductions in self-reported stress, anxiety, and depressive symptoms, with slow-paced diaphragmatic breathing emerging as the modality with the most consistent evidence across outcome measures.[12] Zaccaro et al. (2018), in a systematic review in *Frontiers in Human Neuroscience*, catalogued the psychophysiological correlates of slow breathing across studies, confirming parasympathetic activation, HRV increases, cortisol reduction, and improved emotional regulation as consistent downstream effects.[10]

A 2019 systematic review by Hopper et al. in the *JBI Database of Systematic Reviews* confirmed that diaphragmatic breathing training produces measurable reductions in physiological and psychological stress markers, and that these effects are sustained over time when the mechanical pattern becomes habitual, not just when it is consciously practised.[9] That persistence is the point. Step 1 is not about performing a breathing exercise under controlled conditions. It is about restoring a baseline that the body then maintains.

Lehrer, Vaschillo, and Vaschillo (2000), in the foundational paper on resonance frequency biofeedback in *Applied Psychophysiology and Biofeedback*, established that training the body to breathe at its cardiac resonance frequency produces lasting improvements in autonomic flexibility and the capacity to recover rapidly from stress.[35] The training effect is precisely what Step 1 aims for: not a temporary calming exercise, but the restoration of a breathing pattern that continuously regulates the physiological stress response, independent of whether attention is directed to it.

Most stress interventions attempt Step 3 first, or skip the physiological foundation entirely and rely on cognitive tools alone. The evidence suggests that this is not simply suboptimal. It is asking the system that has been most impaired by stress to do the heaviest lifting. The Raban Method inverts that order, and the inversion is grounded in what the physiology actually requires.

> ***The sequence is not a design preference.*** *It follows from the direction of the causal chain: breathing mechanics govern autonomic tone, autonomic tone governs cortisol, and cortisol governs the availability of the very cognitive resources needed for Steps 2 and 3.*

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All referenced studies are available via PubMed or Google Scholar. This page covers the clinical basis for Step 1 of The Raban Method only. Steps 2 and 3 draw on additional literature in directed breathwork and cognitive stress analysis respectively.