Breathwork Compendium

Volume 2 — When Breathing Becomes a Training Stimulus

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Part 0 — Key Points at a Glance

If you only have five minutes: This page is enough for an overview. Volume 2 builds on Volume 1 and assumes its foundations — physiology, safety rules, the Evidence Rating and Safety symbols — are already familiar. The character is different here: Volume 1 mainly answers which breathing techniques suit everyday life. Volume 2 asks more why athletes, physicians, or divers deliberately use breathing as a training stimulus — less how-to, more context.

Evidence, Safety, and Suitability: The Three Ratings

Part 1 introduced two rating dimensions: evidence and safety. Volume 2 adds a third: suitability. Unlike evidence and safety, suitability isn't a scale of effect or risk — it answers a different question: for whom is a technique the right choice, evidence and safety notwithstanding? The three dimensions are deliberately marked in different ways — evidence as a rating light (🟢🟡🔴), safety with its own symbols (☑ ⚠ ⛔), suitability without a symbol as a plain text label. Color is always paired with text, so the classification also works for color blindness and in black-and-white print.

Evidence and Safety

Both systems carry over unchanged and aren't re-derived here. Evidence Rating: 🟢 Established, 🟡 Mixed, 🔴 Speculative — rates how well an effect is supported. Safety: ☑ No restriction, ⚠ Individual, ⛔ Caution — rates the level of caution warranted for certain techniques or pre-existing conditions.

Suitability

Rates for whom a technique makes sense, evidence and safety notwithstanding — new in Volume 2. Not a substitute for an individual medical assessment.

General — generally applicable without restriction for healthy individuals.

Individual — suitability depends heavily on the person, e.g., responders/non-responders to altitude training.

Not without professional guidance — sensible only under medical or physiotherapeutic supervision, e.g., inspiratory muscle training in Post-Exertional Malaise.

Five Key Messages

1. **Inspiratory muscle strength is trainable** — strength gains in the respiratory muscles through targeted training are well documented; the transfer to overall athletic performance, however, is inconsistent (Part 2).

2. **Strain breathing is a tool, not an automatism** — the Valsalva maneuver can increase trunk stability during strength training, but comes with brief, sometimes substantial blood pressure spikes (Part 3).

3. **Hypocapnia is not hypoxia** — too much breathing (CO_2 deficiency) and too little oxygen are two different physiological mechanisms with different risks. Confusing them is dangerous (Part 1, Part 5, Part 6).

4. **Relieving symptoms is not treating the underlying condition** — breathing therapy can complement treatment for asthma, COPD, and long COVID, but doesn't replace medical treatment. Post-Exertional Malaise calls for particular caution (Part 7).

5. **Many popular claims about breathing, digestion, and longevity are insufficiently supported** — from “boosting the immune system” to “extending lifespan”: the current evidence doesn't support these claims in their widely circulated form (Part 9).

Part 1 — Safety First: When Breathing Becomes a Training Stimulus

The Three Absolute Rules Still Apply 🟢

The three absolute rules from Volume 1 still apply unchanged — and even more strictly for the higher intensities in this volume:

1. **Never in or near water.** Not even in the bathtub, not even sitting on the pool edge with your feet in the water.
2. **Never while driving or operating machinery.**
3. **Never push through dizziness.** Dizziness is the signal to stop immediately and resume normal breathing – not to "push through" the exercise.

The Four Levers: What's Stressed Differently, Starting in Part 2 🟢

Every technique in this volume is assigned to one primary physiological lever; additional mechanisms may be involved but aren't the organizing principle. This assignment determines the chapter boundaries: each chapter covers one primary lever.

1. **Resistance** — the respiratory muscles must work against increased resistance — a training stimulus for respiratory muscle strength (Part 2).
2. **Pressure** — deliberately generated intrathoracic and intra-abdominal pressure stabilizes the trunk but briefly strains the cardiovascular system (Part 3).
3. **CO_2 depletion** — hyperventilation lowers CO_2 levels (hypocapnia) and thereby alters cerebral blood flow and perception — independent of oxygen levels (Part 5).
4. **O_2 depletion / hypoxia exposure** — breath-holding, altitude exposure, or hypoxia devices lower oxygen availability (hypoxia) and trigger a metabolic adaptation stimulus (Part 6).

Hypocapnia ≠ Hypoxia — the Most Important Distinction in This Volume 🟢

The most common mistake with intense breathing techniques is equating “breathing hard” with “taking in a lot of oxygen.” These are two different physiological states with different mechanisms and risks.

CO_2 Drop (Hypocapnia) from Hyperventilation

Increased breathing lowers blood CO_2 levels. Via hypocapnic vasoconstriction, this reduces cerebral blood flow, along with tingling and dizziness (Volume 1, Part 1). Crucially: oxygen saturation typically rises only slightly as a result in healthy people at rest — it's already close to its physiological maximum without hyperventilation. A pulse oximeter (SpO_2) doesn't show this distinction: it measures only oxygen saturation, not CO_2 levels — an unremarkable SpO_2 reading therefore doesn't rule out pronounced hyperventilation.

O_2 Drop (Hypoxia) from Breath-Holding, Altitude, or Devices

Here, the actual oxygen content in the blood drops. Tissue must activate real adaptive responses, such as the production of red blood cells (erythropoiesis, Part 6). This adaptation is the actual training stimulus in altitude training.

Why the Combination Is Dangerous

If you hyperventilate before holding your breath, the CO_2 level drops artificially. This delays the natural urge to breathe while blood oxygen levels keep dropping unchecked — the body notices the oxygen shortage too late as a result. This exact mechanism underlies shallow water blackout (Volume 1, Part 1: Why the Water Rule Applies). It applies not only in water, but to any activity that depends on uninterrupted attention.

Both mechanisms compared directly:

	CO_2 Depletion (Hypocapnia)	O_2 Depletion (Hypoxia)
Trigger	Hyperventilation	Breath-holding, altitude, hypoxia device
Primary Effect	Cerebral vasoconstriction	Cellular oxygen deprivation
Subjective Signal	Dizziness, tingling, tightness	Inconsistent — weaker and slower than the CO_2 stimulus, can be absent with a rapid drop
Greatest Danger	Combination with water (shallow water blackout)	Lack of warning signal with sudden drop, especially after prior hyperventilation
Normalization	Calm the breathing — CO_2 rises again on its own	Oxygen supply: fresh air, stop exertion/breath-hold

Extended Safety and Caution Matrix 🟡

This overview is not a medical contraindication list. It's meant to help orient which techniques from this volume require special caution. If you have a known condition or are pregnant, individual suitability should be clarified with a medical professional. It extends the matrix from Volume 1, Part 1 with the three central techniques of this volume (Part 2, Part 3, Part 6); the five rows there remain unchanged.

Legend: ⛔ Caution = clarify individually with a doctor · ⚠️ Individual = situation-dependent · ☑️ No restriction = no known specific restriction

Technique	Cardiovascular Risks	Pregnancy	Acute Respiratory Illness	Epilepsy (known/ suspected)
Strain Breathing / Valsalva Maneuver	⛔ Caution	⛔ Caution	⚠️ Individual	☑️ No restriction
Inspiratory Muscle Training (IMT, high intensity)	⚠️ Individual	⚠️ Individual	⛔ Caution	☑️ No restriction
Hypoxia Exposure (altitude, apnea, device)	⛔ Caution	⛔ Caution	⛔ Caution	⚠️ Individual

Note: strain breathing with a pronounced pressure component (Valsalva maneuver) can raise intraocular pressure; medical consultation is advisable for glaucoma — analogous to the corresponding note in Volume 1, Part 1, which remained without its own matrix row there since the base compendium doesn't cover strain breathing in depth.

A note on methodology: the classifications in this extended matrix are a first, deliberately cautious draft. They are checked against primary sources in the respective specialist chapters (Part 2, Part 3, Part 6) and refined as needed.

An example of suitability in practice: inspiratory muscle training is an established form of training for healthy athletes — **General** (Part 2). In Post-Exertional Malaise associated with long COVID or ME/CFS, the same load can worsen symptoms — here, depending on severity, **Individual** to **Not without professional guidance** applies (Part 7). Evidence and safety alone therefore say nothing yet about whether a technique is the right choice for a particular person.

With this framework in place, the four levers follow in detail — starting with the trainable respiratory muscle (Part 2).

Part 2 — Inspiratory Muscle Training (IMT)

A quick reminder from Volume 1: breathing is a chain of four stations — air flows in through the nose, oxygen diffuses into the blood in the alveoli, the blood transports it to the tissues, where it's consumed in the mitochondria to generate energy. In a healthy person, oxygen transport usually isn't the bottleneck — CO_2 and pH regulation is (Volume 1, Part 2).

Volume 2 asks a different question: if the respiratory muscles themselves — like any other skeletal muscle — can fatigue, can they also be trained deliberately? And if so, what's it good for?

What Is Inspiratory Muscle Training? 🟢

Inspiratory Muscle Training (IMT) specifically trains the muscles responsible for inhalation — primarily the diaphragm and the external intercostal muscles. The principle: a device generates a defined resistance during inhalation that the respiratory muscles must work against — repeatedly, over weeks, like classic strength training.

Respiratory muscle strength is usually measured via maximal inspiratory pressure (MIP, also PImax): after fully exhaling, you inhale as forcefully as possible against a closed system, and the resulting negative pressure in cmH_2O is the measure. In young healthy adults, MIP is typically in the rough range of about −80 to −120 cmH_2O as a general benchmark; values less negative than about −60 cmH_2O (e.g., −50, −40 cmH_2O) are considered a sign of clinically relevant respiratory muscle weakness (Evans & Whitelaw, 2009). The counterpart for the expiratory muscles is maximal expiratory pressure (MEP).

Devices: Dosing Resistance Precisely 🟢

Two device types dominate the literature. Pressure-threshold devices only open a valve once a set pressure is exceeded — independent of individual breathing behavior, making them precisely reproducible. Flow-resistive devices instead narrow an opening through which you breathe; the pressure actually generated here also depends on how fast someone inhales, which makes dosing less exact. In comparison studies, flow-resistive systems with variable resistance were perceived as more comfortable and produced larger volume and flow responses — which system is superior for which purpose is considered unresolved in the literature overall.

Why Respiratory Muscles Fatigue at All: The Metaboreflex 🟢

The reason inspiratory muscle training has a plausible rationale: when the respiratory muscles fatigue — for instance during intense endurance exercise — metabolic byproducts accumulate in them. Via specialized nerve fibers (type IV afferents), this triggers a sympathetically mediated reflex response: blood vessels in the working limb muscles constrict, redirecting blood flow in favor of the respiratory muscles. This protects breathing in the short term, but can additionally fatigue the leg muscles and limit performance — the so-called respiratory muscle metaboreflex.

A small controlled study (Witt et al., 2007; n=8 training group at 50% MIP, n=8 placebo control at 10% MIP, 5 weeks) demonstrates the mechanism concretely: after training, the heart rate increase during a standardized breathing load was attenuated from a previous roughly 35–41% to about 27%, and the increase in mean arterial pressure from 17–21% to only about 4% — with the control group unchanged. Interpretation: trained respiratory muscles fatigue more slowly and need to rely on the metaboreflex less often. Important for context: this is a very small sample — suitable as evidence of the mechanism, not as proof of a performance effect of a particular magnitude.

Training Protocols: Plenty of Disagreement 🟡

There is no standardized IMT protocol. Published studies train at intensities between 30 and 80% of individual MIP, most commonly around 50–60%, usually over 6–12 weeks, with 3–7 training days per week. Some protocols follow classic strength-training logic (few repetitions at high load), others follow endurance logic (many repetitions at low load) — both approaches can be made roughly comparable via total training load (repetitions × sets × sessions × %MIP), without this yielding an optimal protocol.

Evidence in Healthy Adults and Athletes 🟡

In healthy adults, the strength gain of the respiratory muscles themselves is well documented and undisputed 🟢. More interesting — and considerably less clear-cut — is the question of whether this transfers to athletic performance.

A systematic review of endurance sports (Shei, 2018) reports improvements across several disciplines: in cycling time trials, in time to exhaustion while running (+42% in one individual study), in swimming (+3 to +7% depending on distance), and in VO_2max (+11 to +14% across several studies). A rowing study reported in Shei (2018), however, found no effect — the authors suspect too weak a training stimulus.

A population difference in swimming is particularly revealing: elite swimmers with 10–12 training sessions per week showed no additional benefit from IMT, while sub-elite athletes did benefit. The obvious explanation: a very high regular training volume already delivers an equivalent stimulus to the respiratory muscles — IMT then has little to add.

A caveat: the studies vary considerably in blinding quality, and a placebo effect (less perceived breathlessness, not necessarily more performance) can't be cleanly separated from a genuine physiological effect in many designs.

Evidence in COPD 🟢

In COPD, IMT is one of the most thoroughly studied topics in this chapter — and at the same time provides the clearest example of how strongly the result depends on the chosen measurement method.

The current Cochrane review (Ammous et al., 2023) finds a 35.7 m improvement in the 6-minute walk distance (95% confidence interval 25.7–45.7 m; 16 studies, 501 participants) with moderate evidence certainty. An earlier meta-analysis (Beaumont et al., 2018), which instead evaluated the shuttle or 12-minute walk test, found no statistically significant effect (the confidence intervals each extend well into negative territory). This isn't a contradiction that resolves easily — different walk tests, different included studies, different results.

For clinical context: how large a noticeable improvement in walk distance needs to be is itself disputed — estimates of the minimal clinically important difference (MCID) range from about 30 m to 54–80 m, depending on the calculation method. The Cochrane effect thus sits at the lower end of some estimates and below others. A definitive statement of “clinically relevant” or “not relevant” would be an overinterpretation in either direction 🟡.

Respiratory muscle strength itself increases consistently — a meta-analysis of 16 studies (Han et al., 2024) finds a standardized effect size of 0.86 versus control for the subset of 10 studies reporting MIP 🟢. Dyspnea and quality of life also improve, though with lower to very low evidence certainty. The Cochrane review also notes that the added benefit of IMT as a supplement to an existing physical rehabilitation program is questionable — the effect is more clearly documented when IMT is used alone, for instance in people who can't (yet) do regular exercise training.

Evidence in Asthma

In asthma, a similar pattern emerges as in COPD: several meta-analyses, differing results — here with the added complication of a difference between children and adults.

In adults, a current meta-analysis (Lista-Paz et al., 2022) finds no significant effect on lung function (FEV1), but does find one for exertional dyspnea. Another source, not accessible in full text within this research, reports instead an FEV1 increase of about 3.3 percentage points and an MIP increase of about 22 cmH_2O. As long as this second figure can't be verified against the primary source, it remains unconfirmed here — not refuted, but also not citable.

In children (4–18 years), a meta-analysis of six randomized studies (Xiang et al., 2024) finds significant improvements in MIP (+25 cmH_2O), FEV1 (+5 percentage points), and asthma control — though with very high statistical heterogeneity between studies (I^2 around 92–98%), extremely wide confidence intervals, and the authors' explicit note that none of the included studies recorded training adherence.

The honest summary: in asthma, IMT can improve respiratory muscle strength and probably exertional dyspnea. Whether it measurably improves lung function itself is inconsistent between studies — and in children, further complicated by high study heterogeneity.

A Word on Review Quality Itself

Before citing individual figures from this chapter in isolation: an overview that assessed 23 systematic reviews on IMT in chronic respiratory diseases (Torres-Castro et al., 2025) arrives at a sobering finding. Only 2 of the 23 reviews were rated as methodologically high quality, 14 as critically low quality. The authors also document that different reviews addressing identical questions — such as dyspnea in asthma — arrive at contradictory statements about evidence certainty.

This doesn't mean IMT is ineffective. It means that the quantity of available literature on this topic shouldn't be confused with the quality of that literature — a distinction that applies to practically every topic in this compendium, but is documented especially clearly for IMT.

Suitability: Who Benefits from IMT?

General: Healthy adults with moderate to normal training volume looking for a low-effort addition to their existing training.

Individual: Athletes with already very high training volume (see elite swimmers above) — here, additional benefit is uncertain. Likewise people with COPD or asthma using it as a supplement to existing therapy — the benefit is real, but its magnitude varies considerably between studies.

Not without professional guidance: With acute respiratory illness (see matrix, Part 1), training should wait until stabilization. With long COVID or ME/CFS involving Post-Exertional Malaise, fundamental caution is warranted, since IMT represents a real physical load that can worsen PEM symptoms (explored further in Part 7).

Conclusion: Four Questions, Four Answers

Does IMT make the respiratory muscles stronger? Yes — well documented, across all populations studied 🟢.

Does IMT improve exercise capacity? In COPD, probably, depending on the measurement method used 🟡. In asthma, mainly for perceived breathlessness, less clearly for lung function itself 🟡.

Does IMT improve athletic performance? In non-elite athletes across several disciplines, yes — questionable at already very high training volumes 🟡.

Is IMT suitable for everyone? No — see Suitability above.

Sources: Ammous et al. (2023), Cochrane Database of Systematic Reviews. Beaumont et al. (2018), The Clinical Respiratory Journal. Han et al. (2024), Life. Lista-Paz et al. (2022), Annals of Physical and Rehabilitation Medicine. Xiang et al. (2024), Frontiers in Pediatrics. Witt et al. (2007), The Journal of Physiology. Shei (2018), Journal of Strength and Conditioning Research. Torres-Castro et al. (2025), Frontiers in Sports and Active Living. Evans & Whitelaw (2009), Respiratory Care.

Part 3 — Strain Breathing, Bracing, and the Valsalva Maneuver

Volume 1 deliberately left strain breathing out: it isn't part of the base compendium and was only mentioned there with a note on intraocular pressure. This chapter makes up for that — with the second lever from Part 1: pressure.

Four Terms That Are Often Confused 🟢

“Holding your breath while lifting” isn't a single technique, but an umbrella term for at least four different things:

Breath-holding (apnea): simply holding your breath, without active straining — the base state from which the other three techniques develop.

Bracing: voluntary bracing of the trunk muscles to stabilize the spine. Bracing can be performed with or without breath-holding — conceptually independent of the other three techniques, even though it's often combined with them in practice.

Valsalva maneuver: forced exhalation against a closed glottis. The air can't escape, so high intrathoracic and intra-abdominal pressure build up simultaneously.

Forced exhalation with an open glottis: exhaling under tension during the concentric phase of a movement, but without the glottic closure of the Valsalva maneuver — resulting in lower pressure buildup in the chest.

The difference between an open and closed glottis is the decisive variable in this chapter: it separates the technique with the highest pressure buildup (and highest cardiovascular cost) from an alternative with lower pressure.

The Mechanism: Pressure as a Tool 🟢

The idea behind strain breathing in strength training: elevated intra-abdominal pressure (IAP) temporarily turns the trunk into a more stable, pressure-stiffened cylinder. This is meant to relieve the spine under heavy loads and improve force transfer from the leg muscles to the load.

How Much Pressure? Strongly Dependent on the Exercise 🟢

“Strain breathing raises pressure” isn't a fixed quantity — it depends heavily on which exercise is performed. A systematic review of 16 studies (Blazek et al., 2019) shows this clearly: in the squat, intra-abdominal pressure rises above 200 mmHg — in the bench press, by contrast, only to an average of 79 mmHg. Deadlifts, leg press, and cable rows fall in between at 161–176 mmHg. The authors recommend bench press and rowing as entry-level exercises for beginners and people with high blood pressure, while progressive habituation makes sense for squats, deadlifts, and similar exercises with high axial load.

The Price: Blood Pressure Spikes 🟢

The classic reference study comes from MacDougall et al. (1985): in five experienced bodybuilders, blood pressure was measured directly via a catheter in the brachial artery during exercises to muscle failure. In the bilateral leg press, blood pressure reached a group average of 320/250 mmHg — in one subject, 480/350 mmHg was measured. For comparison: a blood pressure of about 180/120 mmHg or above is already considered a hypertensive emergency in medicine if sustained. However, these extreme values occur only briefly, during the concentric phase of a maximal lifting attempt — not as a sustained state.
That Valsalva systematically produces higher blood pressure responses than controlled breathing has been replicated multiple times — this pattern already showed up in the biceps curl and knee extension (Linsenbardt, Thomas & Madsen, 1992). A randomized comparison study with squats (Hummelmann et al., 2026) found systolic blood pressure 16–23% higher during breath-holding than during controlled breathing, plus a 20–30% higher cardiovascular load (rate-pressure product). Under bodyweight-only loading, blood pressure under breath-holding reached 149% of the resting value, versus only 122% under controlled breathing.

Performance ≠ Safety: What a Study Really Shows 🟡

This is the most interesting — and most misunderstood — finding in this chapter. A study by Ikeda et al. (2009) tested maximal isometric force of knee, shoulder, and elbow movements in ten subjects under four breathing conditions: normal breathing, forced inhalation, forced exhalation, and the Valsalva maneuver. Result: forced exhalation significantly increased maximal force in several of the tested movements — and didn't differ from force under Valsalva in any of the tested muscle groups. Important up front: this applies to isometric single-joint movements, not automatically to dynamic compound exercises like squats or deadlifts — the caveat on that follows in the next paragraph.
Together with the blood pressure data from the previous section (MacDougall et al., 1985; Linsenbardt, Thomas & Madsen, 1992; Hummelmann et al., 2026), this yields a strong argument: for isometric single-joint movements, forced exhalation with an open glottis appears to match Valsalva's strength benefit without producing the same rise in blood pressure.
Two caveats necessarily belong here. First, the strength finding and the blood pressure finding come from different studies with different exercises — no single study measured both at once. Second, and more importantly: Ikeda et al. tested isometric single-joint movements, not complex dynamic compound exercises like squats or deadlifts under maximal load, where spinal stabilization via intra-abdominal pressure plays a different, more complex role than in an isolated joint movement. Whether forced exhalation also provides the same stability as Valsalva in a maximal squat is not answered by this study 🟡.

Intraocular Pressure and Glaucoma 🟡

A study of 30 healthy men (Vieira et al., 2006) measured intraocular pressure during bench press at 80% of maximum weight: under breath-holding, pressure rose in 90% of subjects, by an average of 4.3 mmHg. Under normal breathing, it rose in 62% of subjects, by an average of 2.2 mmHg. A methodological limitation: the order was apparently fixed — breath-holding first, then normal breathing — a fatigue effect can't be ruled out. The finding nonetheless supports the note already made in Part 1: for glaucoma, medical consultation is advisable before pronounced strain breathing.

Venous Return Under Combined Loading 🟡

A small study of eight elite athletes (Hughes et al., 1989) shows a counterintuitive mechanism: Valsalva alone reduced cardiac stroke volume by 68%. Combined with additional isometric contraction (handgrip), stroke volume instead dropped by only 42% — so the combination had a more stabilizing effect than Valsalva alone, presumably because additional vascular tone better maintains venous return. With only eight subjects, this is a mechanistic finding, not evidence for a general safety recommendation.

Cerebrovascular Risk: What the Evidence Shows 🟡

The concern often circulates that Valsalva during strength training could trigger a stroke or the rupture of a brain aneurysm. The literature reviewed in this research round finds no robust causal link for this — documented case reports are extremely rare, and an often-cited specific case count couldn't be traced to any findable primary source and is therefore not mentioned here. For people with known vascular conditions, caution remains sensible — not because a causal link has been proven, but because the evidence simply isn't sufficient for a confident all-clear in this group 🟡.

Suitability: Who Should Avoid or Modify Valsalva?

General: Healthy people without pre-existing cardiovascular conditions who train heavy — brief Valsalva during individual maximal attempts is considered a well-controllable risk.

Individual: People with high blood pressure or glaucoma — here, the data support deliberately choosing gentler exercise alternatives (bench press/rowing instead of squats/deadlifts, see above) or forced exhalation instead of Valsalva where possible.

Not without professional guidance: Known cardiovascular conditions, uncontrolled high blood pressure, or diagnosed glaucoma — consult a doctor before intensive strain-breathing training (see matrix, Part 1).

Conclusion: Practical Takeaways

Does strain breathing increase strength? Yes, plausibly and with a mechanistic basis — how much depends heavily on the exercise and movement type 🟢.
Is Valsalva necessary for this? For isometric single-joint movements, apparently not — forced exhalation achieves the same strength. For complex compound exercises under maximal load, this hasn't been studied 🟡.
Is strain breathing dangerous? It briefly raises blood pressure and intraocular pressure, sometimes substantially — for most healthy people, that's a controllable, not a proven dangerous risk. This assessment changes with pre-existing cardiovascular conditions or glaucoma 🟡.
Who does this apply to? See Suitability above — the answer isn't uniform.

Sources: Blazek et al. (2019), Biology of Sport. MacDougall et al. (1985), Journal of Applied Physiology. Linsenbardt, Thomas & Madsen (1992), British Journal of Sports Medicine. Hummelmann et al. (2026), Physiological Reports. Ikeda et al. (2009), Journal of Strength and Conditioning Research. Vieira et al. (2006), Archives of Ophthalmology. Hughes et al. (1989), European Heart Journal.

Part 4 — Breathing Under Exertion

Volume 1 answered this briefly: “Under exertion, breathing is rarely the limiting factor.” For everyday life, that's true — and sufficient as an answer there. But anyone who trains seriously or competes asks the same question more precisely: rarely doesn't mean never, and “not limiting” doesn't mean breathing technique plays no role. This chapter isn't a new lever, but a bridge chapter: it shows where resistance (Part 2) and pressure (Part 3) concretely converge in sport — and where breathing actually does become the limit.

When Does Breathing Become Limiting? The EIAH Exception 🟢

Volume 1 mentions the exception only as a side note: exercise-induced arterial hypoxemia (EIAH) in elite endurance athletes. For Part 4, that's exactly the relevant point. Under the classification by Dempsey & Wagner (1999), a drop in arterial oxygen saturation to 95–93% counts as mild, 93–88% as moderate, and below 88% as severe. An estimated 40–50% of highly trained endurance athletes show a relevant EIAH during exercise near VO_2max — in untrained or moderately trained individuals, the phenomenon practically doesn't occur.

The mechanism: at very high cardiac output, the blood's contact time in the pulmonary capillaries shortens, which can leave hemoglobin's oxygen loading incomplete — amplified in athletes with relatively small lung capacity relative to cardiac output. Two converging studies (Powers et al., 1993; Harms et al., 2000) quantify the consequence: above a threshold of 4% SaO_2 drop in men and 3% in women, each additional percentage point of saturation drop costs about one percent of VO_2max.

The context nonetheless stays narrow: this affects a minority even within the endurance elite, not athletes in general — and for most sports with shorter exertion duration or without maximal aerobic exhaustion, EIAH isn't a relevant topic.

Breath-Stride Coupling While Running 🟡

Runners often couple breathing and stride rhythm spontaneously — a phenomenon called locomotor-respiratory coupling (LRC). A study with natural arm swing (Daley, Bramble & Carrier, 2013) found a non-random distribution of breath transitions across the stride cycle in 13 of 14 subjects, most commonly at a 2:1 ratio (two strides per breath). Crucially: breath transitions in "preferred" phases of the stride cycle occurred twice as fast as in "avoided" phases — a mechanical effect, not a purely voluntarily chosen rhythm. Foot-strike impact appears to play the decisive role here: the body favors coupling patterns that minimize the conflict between impact forces and respiratory mechanics.

For practice, this means: a deliberately imposed breathing rhythm while running isn't an arbitrary invention, but follows a physiologically comprehensible logic. Whether a deliberately trained rhythm actually delivers a performance benefit isn't shown by this, though — the study documents the coupling itself, not a benefit from deliberately intervening in it 🟡.

Nasal Breathing in Competition: What a Recent Study Shows 🔴

Volume 1 explains why mouth breathing at high intensity becomes a physiological necessity, not a bad habit (Volume 1, Part 4). In competitive sport, the claim nonetheless circulates that targeted nasal breathing training improves competitive performance. A recent randomized crossover study (Bergqvist et al., 2025) tested this directly in twelve well-trained cyclists and triathletes: oral, oronasal, and oronasal with a decongested nose, each in a maximal graded exercise test. Result: no statistically significant difference in VO_2max or maximal power between the three conditions. Time to exhaustion also didn't differ significantly. Notably, in the opposite direction: purely oral breathing even produced lower post-exercise lactate values than oronasal breathing.

This contrasts with older, methodologically weaker studies reporting an efficiency gain from nasal breathing. The most recent and methodologically cleanest study available here finds no performance advantage 🔴 — one of the places in this compendium where a popular training claim doesn't hold up under direct testing.

Swimming: Breathing Side and Symmetry

Unilateral breathing to the preferred side produces a measurable asymmetry in hip rotation during front crawl. A study with 18 elite competitive swimmers (Barden & Barber, 2022) quantifies this: with unilateral breathing, hip rotation toward the breathing side was 54.2°, versus only 44.6° to the opposite side — a significant difference of almost 10°. Bilateral breathing (alternating every three strokes) reduced the asymmetry to 51.6° versus 46.9°, but didn't eliminate it entirely. Only a snorkel condition with no breaths at all produced a symmetric pattern with no significant side difference.

The nuanced takeaway: bilateral breathing measurably improves symmetry — the common equation “breathing bilaterally = swimming symmetrically” is nonetheless a simplification.

Side Stitches: More Than Just Chance

Exercise-related transient abdominal pain (ETAP), colloquially known as a side stitch, affects 61% of people at least once a year, according to a survey of 965 athletes across six sports (Morton & Callister, 2014) — with clear differences between sports: swimming 75%, running 69%, cycling only 32%. At a single marathon, around 27% of participants are affected. Among those affected, 76% reduce intensity, and 12% stop the activity entirely.

Several mechanisms have been investigated and partly disproven: diaphragmatic ischemia is now regarded as a largely disproven explanation, and abdominal muscle cramping has been ruled out by electromyographic studies. The best current fit is an explanation involving irritation of the parietal peritoneum. Known triggers: eating shortly before exertion (under two hours), strongly hypertonic drinks, a hunched trunk posture. Trunk strength, better posture, and a higher training status all have a mitigating effect.

Conclusion: When Does Breathing Become the Limit?

For most people: practically never — the cardiovascular system, muscles, or metabolism become limiting first (Volume 1).

For the endurance elite near VO_2max: actually relevant in 40–50%, via the EIAH mechanism 🟢.

For runners: breathing couples mechanically to the stride rhythm — but this doesn't automatically demonstrate a performance benefit from deliberately controlling it 🟡.

For nasal breathing training in competition: the most recent evidence speaks against a performance advantage 🔴.

For swimming and side stitches: both are real and measurable, but more nuanced than the common rules of thumb suggest 🟡.

Sources: Dempsey & Wagner (1999), Journal of Applied Physiology. Powers, Martin & Dodd (1993), Sports Medicine. Harms et al. (2000), Journal of Applied Physiology. Daley, Bramble & Carrier (2013), PLoS One. Bergqvist et al. (2025), Frontiers in Physiology. Barden & Barber (2022), Sensors. Morton & Callister (2014), Sports Medicine.

Part 5 — Too Much Breathing: Hyperventilation as a Method

This chapter covers the third lever from Part 1: CO_2 depletion through deliberate hyperventilation. The 2014 Kox study (the “Wim Hof study”) isn't revisited here — it's already fully addressed in Volume 1, Part 7, Category C (suppression rather than strengthening of the immune system, three training components, only 24 men). This chapter covers what's missing there: the broader category of “high-ventilation breathwork” (HVB), which includes not only Wim Hof-style cycles but also Holotropic Breathwork, rebirthing, and Sudarshan Kriya Yoga.

Is “High-Ventilation Breathwork” Even a Category? 🟡

A recent review (Fincham et al., 2023) frames HVB as an umbrella term for practices with deliberately increased ventilation — and in doing so names the problem itself: the effect is attributed to several mechanisms operating simultaneously (sympathetic activation, altered cerebral blood flow, alkalosis, neuronal excitability, a mismatch in interoceptive predictions), while the individual practices differ substantially in breathing pattern, duration, intensity, breath retentions, group setting, and psychological framing. The answer to this chapter's guiding question: HVB has a genuine shared physiological denominator — increased ventilation — but isn't a uniform intervention. As the following sections show, that's true not only for the practice itself, but also for the quality of the evidence behind it.

The Mechanism in Detail: What Hypocapnia Does in the Brain 🟢

Part 1 laid the groundwork: hyperventilation lowers CO_2 levels and, via hypocapnic vasoconstriction, narrows the cerebral blood vessels. What remained open there: hypocapnia additionally releases excitatory neurotransmitters (including glutamate) and thereby directly increases neuronal excitability — independent of blood flow. A study on corticospinal excitability (Hartley et al., 2016) shows this explicitly: the increased excitability depends specifically on the CO_2 level, not on the accompanying reduced cerebral blood flow. Both effects run through separate mechanisms — an important refinement of the common simplification that equates the two.

This increased excitability also explains the characteristic tingling and cramping seen with pronounced hyperventilation: the rising blood pH causes more calcium to bind to albumin, which lowers the proportion of biologically active, ionized calcium — while the total blood calcium level stays unchanged. Below a CO_2 partial pressure of about 20 mmHg (normal value roughly 40 mmHg), muscle cramps typically occur; below 15 mmHg, whole-body tetany from skeletal muscle contraction. These symptoms are unpleasant but reversible and normalize on their own as breathing returns to normal.

Capnography Aside: What a Wearable Actually Measures

Capnography measures end-tidal CO_2 ($etCO_2$, normal range 35–45 mmHg) — the concentration at the end of exhalation, as an approximation of the arterial CO_2 level. It's clinically established, for instance for ventilation monitoring. For consumers, the key distinction is from the pulse oximeter: an SpO_2 sensor measures only oxygen saturation, not CO_2 level. The two measurements are independent of each other — a wearable that only tracks SpO_2 fundamentally can't say anything about hypocapnia (as already noted in Part 1). This technical limitation is a recurring theme for evaluating apps and sensors in Part 9.

Sudarshan Kriya Yoga: The Best-Documented HVB Practice 🟢

Within the HVB category, Sudarshan Kriya Yoga (SKY) is by far the practice with the most solid evidence base. A randomized controlled trial in 21 Iraq/Afghanistan veterans with PTSD (Seppälä et al., 2014) found, after a one-week intervention (21 hours of SKY spread over 7 days), an effect size of $d = 1.16$ for the reduction in PTSD symptoms versus the waitlist control group — a large effect. The reduction in startle response correlated closely with the reduction in hyperarousal symptoms, both directly after the intervention ($r = 0.93$) and after one year ($r = 0.77$) — an indication of a lasting effect, even without continued practice. A later, larger study in 85 veterans (Bayley et al., 2022) compared SKY directly with an established standard therapy (Cognitive Processing Therapy) and found SKY non-inferior to it — notable because here SKY is competing against an active treatment, not just against doing nothing 🟢.

Holotropic Breathwork: The Least-Documented HVB Practice 🔴

At the other end of the evidence spectrum is Holotropic Breathwork. The research base specifically for this technique is thin: solid empirical studies can be counted on one hand, and reported effects on addiction, asthma, anxiety, or depression come predominantly from uncontrolled or retrospective designs without a comparison group. This isn't evidence that the technique doesn't work — it's evidence that there's simply barely any robust data 🔴. Providers of Holotropic Breathwork courses themselves list an extensive set of contraindications: pregnancy, cardiovascular disease, uncontrolled high blood pressure, glaucoma, retinal detachment, epilepsy, severe psychiatric conditions — matching exactly the caution list already established in Part 1.

Breathwork in General: A Meta-Analysis With a Caveat 🟡

A meta-analysis of 12 randomized controlled trials with 785 participants (Fincham et al., 2023 — a different publication than the review above, same lead author) finds significant improvements in stress, anxiety, and depression from “breathwork” versus control groups. Important for context: this meta-analysis bundles together very different techniques — not just HVB, but also the slow breathing techniques from Part 6 of Volume 1. The umbrella term “breathwork” thus mixes categories here that would actually need to be evaluated separately — exactly the problem this chapter identified for HVB on a small scale repeats itself on an even broader level.

On safety: most included studies reported no adverse effects. For fast breathing specifically, a subgroup showed hot flashes, breathlessness, or sweating, though no one discontinued because of it; at the same time, cortisol spikes during the exercise were higher than in control groups, with faster recovery afterward. The authors themselves caution: only around 20% of the included studies actively monitored for side effects at all — the safety data specifically for fast breathing is insufficiently captured systematically 🟡.

Suitability: Who Does HVB Make Sense For?

General: Healthy adults looking for a structured, guided practice with a solid evidence base (e.g., Sudarshan Kriya Yoga) — while following the safety rules from Part 1.

Individual: People considering Holotropic Breathwork or similarly under-researched practices — the evidence doesn't permit a reliable benefit-risk assessment, regardless of individual tolerance.

Not without professional guidance: Epilepsy, pregnancy, uncontrolled high blood pressure, glaucoma, retinal conditions, severe cardiovascular or psychiatric pre-existing conditions (see matrix, Part 1) — and without exception: never in or near water, never while driving.

Conclusion: One Category, Two Evidence Worlds

Is HVB a uniform technique? Physiologically: a shared mechanism. Practically and evidence-wise: no — the individual practices differ substantially, both in procedure and in the strength of evidence.

What's best documented? Sudarshan Kriya Yoga, with robust RCTs extending to a direct comparison with standard therapy for PTSD.

What's least documented? Holotropic Breathwork — plausible reports, but barely any controlled studies.

Is HVB safe? For most healthy people who follow the safety rules from Part 1, presumably yes — but the safety data specifically for fast breathing is, by the researchers' own assessment, still insufficient.

Sources: Fincham et al. (2023), Neuroscience and Biobehavioral Reviews (HVB review). Fincham, Strauss, Montero-Marin & Cavanagh (2023), Scientific Reports (breathwork meta-analysis). Hartley et al. (2016), The Journal of Physiology. Seppälä et al. (2014), Journal of Traumatic Stress. Bayley et al. (2022), BMJ Open.

Part 6 — Too Little Oxygen: Apnea, Altitude, and Hypoxia

> **Important note before this chapter: the following pages explain the physiology of oxygen deprivation — through breath-holding, high altitude, or specialized devices. This is an explanation of mechanisms and risks, not instructions for freediving, apnea training, or unsupervised hypoxia training. Volume 1 deliberately excluded apnea diving because unsupervised self-study is life-threatening — that remains unchanged here. Anyone who wants to freedive or train deliberately with hypoxia needs qualified instruction with on-site supervision, not a compendium.**

This chapter covers the fourth lever from Part 1: O_2 depletion / hypoxia exposure. The contrast with Part 5 is the central point of this entire volume: hypocapnia (too little CO_2) and hypoxia (too little O_2) are different mechanisms with different risks — this is about genuine oxygen deprivation, not increased breathing.

The Mechanism: The Dive Reflex 🟢

When the face is submerged in water, the body automatically triggers the dive reflex (mammalian dive reflex): heart rate drops (bradycardia), blood flow is redirected away from the extremities toward the brain and heart, and the spleen contracts to release additional red blood cells into circulation. This reflex is an evolutionary holdover that lowers oxygen consumption when supply is restricted — but it doesn't change the fact that the oxygen reserve in the blood is finite.

Shallow Water Blackout: The Mechanism That Matters 🟢

Volume 1 already explained the mechanism: hyperventilation before breath-holding artificially lowers the CO_2 level and thereby delays the natural urge to breathe, while blood oxygen content keeps dropping — this can allow a critical hypoxia to be reached before a strong urge to breathe forces a stop. Unconsciousness can occur here without reliable warning; case reports cite oxygen saturations in the range of roughly 50–60% at the point of loss of consciousness, though a single fixed threshold applicable to everyone can't be given. Because the affected person is already severely oxygen-deprived at the moment of unconsciousness, brain damage can set in faster here than with “ordinary” drowning. Precise-looking percentage figures for the share of global drowning deaths circulate on the internet — in this research, they couldn't be traced to any robust primary source and are therefore deliberately not cited here. The mechanism itself is well documented regardless, and reason enough for the absolute water rule from Part 1.

Altitude Training: Live-High-Train-Low 🟢

The foundational study of what's now the established live-high-train-low concept (Levine & Stray-Gundersen, 1997) had trained runners live at about 2,500 m for four weeks, while the more intense training sessions took place at about 1,250 m. Result, all figures from the same study: an improvement in sea-level competitive performance of about 1.5% on average, with a range from no improvement up to 6%. Mechanistically, this is driven by an increase in red blood cell mass of about 8%, which leads to a VO_2max improvement of about 5%. The effect persists for at least three weeks after returning to sea level.
For context: this is a real but modest effect in already highly trained athletes — not a miracle cure, but a small additional improvement on top 🟢.

Non-Responders: Not a Fixed Trait 🟡

Not everyone benefits equally from altitude training — that's well known. Less well known: being classified as a “non-responder” isn't a fixed trait. Recent research shows that the response to altitude exposure depends on iron status, altitude dose, baseline hemoglobin mass, training load, and recovery. Iron status in particular is decisive: since red blood cell formation depends on iron, an iron deficiency directly blocks the adaptation to altitude — regardless of how “well” someone might fundamentally respond to hypoxia 🟢.

Normobaric or Hypobaric: An Unresolved Expert Debate 🟡

Is it physiologically equivalent whether hypoxia is produced by a reduced oxygen fraction at normal air pressure (normobaric, e.g., an altitude tent) or by actually reduced air pressure (hypobaric, as at real altitude)? This question isn't conclusively settled in the specialist literature — disputed enough that one journal has set up a dedicated pro/con debate format for it. Differences have been reported in, among other things, minute ventilation and nitric oxide levels; other studies find comparable effects on red blood cell mass. What matters most in practice is that normobaric setups are more readily available, since athletes can stay in their familiar environment 🟡.

Intermittent Hypoxic Training (IHT): Less Than Expected 🔴

A recent meta-analysis (Dorelli et al., 2025) evaluated 35 studies with 524 participants and found no significant difference between aerobic IHT and training performed under normoxic conditions — neither for VO_2max nor for peak performance. Notably, the authors demonstrate a statistically detectable publication bias — studies with larger effects were preferentially published after 2010. This may explain why IHT enjoys a better reputation in popular literature than the overall evidence supports 🔴. One caveat: high-intensity exercise forms performed under hypoxia (instead of purely aerobic training) show more promising results in other reviews not examined in detail here — the training stimulus itself appears to matter more than hypoxia alone.

IHHT: What the Evidence Shows — And What It Doesn't 🟡

Intermittent Hypoxic-Hyperoxic Training (IHHT) deliberately alternates between phases of reduced and increased oxygen supply. A controlled study in 145 long-COVID patients (Doehner et al., 2024) found marked improvements in stair-climbing performance and 6-minute walk distance versus the control group, plus improvements in hand grip strength, fine motor skills, breathlessness, and quality of life — with good tolerability and no reported adverse events. Important caveat: the study wasn't randomized; assignment was based on clinical judgment — expectation or selection effects therefore can't be ruled out 🟡.
What IHHT doesn't demonstrate, by contrast: commercial providers widely market the method with mitochondrial “biohacking” and longevity claims. For the claim that IHHT extends human lifespan, this research found no peer-reviewed evidence in humans — the available data concern concrete clinical endpoints like exercise capacity, not life expectancy. This claim is examined in more depth in Part 9 🔴.

At a Glance: Four Ways to Deplete Oxygen

All four methods produce hypoxia, but in very different ways, with different durations and differently robust evidence:

	Apnea / Freediving	Altitude Training (LHTL)	IHT (Intermittent Hypoxic Training)	IHHT (Intermittent Hypoxic-Hyperoxic Training)
Timescale	Seconds to a few minutes	Days to weeks	Minutes per session	Minutes per session, over several weeks
Main Mechanism	Acute O_2 drop with simultaneous CO_2 rise	Chronic stimulus for erythropoiesis	Repeated hypoxia exposure during training	Alternation between hypoxia and hyperoxia phases
Documented Benefit	No performance benefit in focus here — risk education is the priority	~1.5% performance on average (range 0–6%)	No significant added benefit for aerobic training (Dorelli et al., 2025)	Clinical markers in long COVID, e.g., walk distance and quality of life (Doehner et al., 2024)
Greatest Risk	Unconsciousness without warning (blackout)	Iron deficiency prevents adaptation	Publication bias — advertised benefit exceeds the overall evidence	Non-randomized study evidence; unsupported longevity claims in marketing

What Altitude Training Is Not 🟢

In public discussion, hypoxia, breath-holding, and hyperventilation are often lumped together. Altitude training isn't a breathing technique — it's a training and environmental intervention that acts over weeks, not over individual breaths. It has nothing mechanistically to do with Wim Hof-style hyperventilation (CO_2 depletion, Part 5), except that both ultimately touch the oxygen balance. Anyone who confuses “altitude training” with “breathing fast” or with “holding your breath” is confusing three different physiological processes with three different risk profiles.

Suitability: Which Method Makes Sense for Whom?

General: Altitude training under professional guidance for healthy, already well-trained endurance athletes.
Individual: People with unclear iron status before altitude training (testing advisable); IHT/IHHT outside clinical or well-supervised training environments — benefit is population-dependent and not guaranteed.
Not without professional guidance: Apnea/freediving without exception only with qualified instruction and on-site supervision — never as self-study. Altitude training with pre-existing cardiovascular conditions, anemia, or impaired lung function only after medical evaluation (see matrix, Part 1).

Conclusion: Four Paths, One Mechanism, Very Different Risks

Does altitude training work? Yes, moderately — real, but not a miracle cure 🟢.
Does pure IHT work? The best available meta-analysis argues against it, with a demonstrated publication bias favoring more optimistic older studies 🔴.
Does IHHT work? For concrete clinical applications, yes, promisingly so 🟡. For longevity, there's no evidence in humans whatsoever 🔴.
Is apnea/freediving safe? Only with instruction and supervision. The blackout mechanism is well understood — and precisely for that reason, the water rule from Part 1 isn't one precaution among many, but the single most important statement in this entire chapter.

Sources: Levine & Stray-Gundersen (1997), Journal of Applied Physiology. Dorelli et al. (2025), Scandinavian Journal of Medicine & Science in Sports. Doehner et al. (2024), Journal of Cachexia, Sarcopenia and Muscle (controlled, non-randomized study). Bart, Murray & Lau (2026), StatPearls (Shallow Water Blackout).

Part 7 — Breathing Therapy for Chronic Conditions

> **What this chapter is not: the following sections summarize what research has examined regarding breathing techniques in asthma, COPD, and long COVID. This is a scientific overview, not treatment instructions. It doesn't replace pulmonological diagnosis or therapy, nor individual medical care.**

This section expands on what Volume 1 only announced in Part 11: “Structured breathing rehabilitation for chronic lung diseases or after viral infections belongs under medical and physiotherapeutic guidance.” The throughline is the same as throughout this compendium, but especially important here: a measurable improvement in a marker isn't automatically a clinically relevant benefit — and relieving symptoms isn't the same as treating the underlying condition.

Asthma: What's Already Covered Elsewhere 🟢

Volume 1, Part 5 already covered Buteyko breathing for asthma (Burgess et al., 2011): reduced need for rescue medication, but no improvement in lung function itself. Part 2 of this volume went deeper into the IMT evidence for asthma — with the central finding that adult and pediatric studies contradict each other on lung function outcomes. Neither is repeated here.

COPD: Pursed-Lip Breathing 🟡

Pursed-lip breathing is the longest-established breathing technique for COPD. A systematic review (Roberts et al., 2009; 11 studies, 226 participants) finds moderate evidence: the technique increases oxygen saturation and tidal volume, lowers resting respiratory rate, and shortens recovery time after exertion. Notably honest is the authors' own caveat: the precise mechanism through which breathing exercises work in COPD isn't fully understood — only four of the eleven studies reached moderate quality under GRADE, none reached high quality 🟡. The IMT evidence in COPD was already covered in detail in Part 2, including the controversy there over walk-distance effects depending on the test used.

Long COVID: Strong Markers, Unclear Everyday Benefit 🟡

In long COVID, respiratory muscle training delivers some of the most consistent figures in this entire chapter. A meta-analysis of 7 randomized trials with 449 participants (Correia et al., 2025) finds an improvement in inspiratory muscle strength of 22.7 cmH_2O and a VO_2max improvement of 4.49 ml/kg/min — both figures with narrow confidence intervals and no heterogeneity between studies ($I^2 = 0\%$) 🟢.

The decisive break follows right after: for actual exercise tolerance — what counts in everyday life — the results are inconsistent, one study found no change at all on the Ruffier test, and dyspnea reduction wasn't reported consistently. The authors themselves rate the evidence certainty for all endpoints as low 🟡. This is a textbook example of this compendium's throughline: strong, cleanly measured markers (muscle strength, VO_2max) don't guarantee a noticeable functional benefit.

Post-Exertional Malaise: The Warning Signal

In a subset of people with long COVID and ME/CFS, post-exertional malaise (PEM) occurs: a disproportionate worsening of symptoms after physical or mental exertion, which can begin immediately or with a delay of hours to days, and can last for days, weeks, or months — to an extent not explained by the triggering exertion alone. Biochemical investigations find, among other things, a drop in the purine metabolite hypoxanthine (McGregor et al., 2019) and evidence of disrupted folate/one-carbon metabolism (Baraniuk, 2025) — PEM is thus not a purely subjective complaint, but is associated with measurable metabolic changes.

The most important safety finding in this section: graded exercise therapy (GET) — gradually increasing activity — is currently considered ineffective and potentially harmful in PEM (NICE guideline NG206, 2021). Repeated patient reports document worsening, sometimes permanent, because any increase in exertion can retrigger PEM 🔴⛔. This is why the IMT successes from the previous section can't be uncritically applied to people with pronounced PEM: the cited studies generally don't specifically include PEM patients or screen for PEM.

Suitability: Symptom-Oriented or Unclear

General: Pursed-lip breathing for stable COPD as a complementary, low-risk technique, in consultation with the treating practice.

Individual: IMT for long COVID without PEM — markers improve reliably, but the noticeable benefit varies individually.

Not without professional guidance: Any form of increased exertion — including respiratory muscle training — with existing or suspected PEM. Here it applies: when in doubt, get medical/specialist clearance first before increasing exertion (see Suitability Rating example, Part 1).

Conclusion: A Complement, Not a Replacement

Does breathing therapy help with asthma/COPD/long COVID? For symptoms and markers, often yes, with varying strength of evidence depending on the condition.

Does it treat the underlying condition? No — it complements medical treatment, it doesn't replace it.

Is more training always better? Not with PEM — here, more exertion can mean a real worsening, not something "worth pushing through."

Sources: Roberts et al. (2009), Physical Therapy Reviews. Correia et al. (2025), Systematic Reviews. NICE (2021), Guideline NG206. McGregor et al. (2019), Diagnostics. Baraniuk (2025), International Journal of Molecular Sciences.

Part 8 — Breathing and the Mind

What this chapter is not: the following sections summarize what has been researched regarding breathing techniques for anxiety, stress, and depression. This is a scientific overview, not psychotherapy and not a substitute for it. With a diagnosed anxiety disorder, depression, or trauma-related disorder, no breathing technique replaces appropriate professional treatment.

Volume 1 laid the groundwork: the breathing-anxiety spiral (Part 9) and HRV biofeedback as a path to autonomic regulation (Part 6). Part 5 of this volume already covered the best individual evidence to date for a breathing technique in a psychiatric condition — Sudarshan Kriya Yoga for PTSD. This chapter doesn't repeat that, but asks a different question: when does a breathing technique genuinely help — and when does it itself become part of the problem?

Breathwork for Anxiety and Depression: The Meta-Analysis Level

At the meta-analysis level, the evidence is positive: 12 randomized controlled trials with 785 participants (Fincham, Strauss, Montero-Marin & Cavanagh, 2023) find significant improvements in anxiety (effect size $g = -0.32$ across 20 studies) and depression ($g = -0.40$ across 18 studies) from breathing techniques versus control groups.

The Largest Individual Study Contradicts Its Own Meta-Analysis Level

In the same year, the same lead author published the largest parallel-group breathwork study to date: 400 participants, randomized to “coherent breathing” (about 5.5 breaths per minute) or a carefully constructed placebo breathing condition (12 breaths per minute), each about 10 minutes daily over four weeks (Fincham, Strauss & Cavanagh, 2023). Result on the primary endpoint, subjective stress: no significant difference between groups — only a general time effect that both groups showed equally. The author herself draws the conclusion: no measurable added benefit of coherent breathing over a well-constructed placebo breathing exercise.

This isn't a contradiction that can be explained away — it's a textbook example. The smaller studies in the meta-analysis were tested predominantly against waitlists or nonspecific controls, not against an active, equally plausible-seeming alternative condition. As soon as the control condition becomes more demanding, the effect shrinks to zero. This says nothing about whether breathing feels good or whether people report that it helps — but it qualifies the claim that coherent breathing has a specific mechanism of action beyond expectation and attention.

When Does a Breathing Technique Become Avoidance? 🟢

The cognitive-behavioral model of panic disorder explains why breathing technique isn't automatically helpful: people with high anxiety sensitivity interpret harmless physical signals — a racing heart, breathlessness, dizziness — as signs of serious danger. This misinterpretation leads to hypervigilance, anxiety, and avoidance, which in turn reinforces the anxiety response. An established treatment component, interoceptive exposure, therefore deliberately confronts the feared sensations instead of avoiding them.

This is exactly where a calming breathing technique can become a problem: if it's used to immediately end an unpleasant bodily sensation instead of tolerating it, it functions like a safety behavior — the same category as carrying sedatives or constantly checking your own heart rate on a wearable. Safety behaviors relieve anxiety in the short term but maintain it in the long term, because the person never learns that the sensation itself is harmless 🟡. The guiding question therefore isn't “which breathing technique helps against anxiety,” but “is the technique being used for deliberate regulation or to avoid the feared sensation.”

Panic Disorder: One Component Among Several 🟢

Cognitive behavioral therapy for panic disorder typically combines several components: relaxation, breathing retraining, cognitive restructuring, interoceptive and/or in-vivo exposure. Breathing retraining is thus usually one component among several, not the sole treatment — for an isolated efficacy of breathing technique alone in diagnosed panic disorder, there's no robust standalone evidence 🔴.

Suitability: Regulation Yes, Replacement Therapy No

General: Breathing techniques as one of several strategies for everyday stress and anxiety regulation in healthy people without a diagnosed disorder.

Individual: People with pronounced anxiety sensitivity — here, the benefit depends on whether the technique is used for regulation or for avoidance.

Not without professional guidance: Diagnosed panic disorder, depression, or trauma-related disorder — breathing technique can usefully complement appropriate professional treatment, but not replace it.

Conclusion: Helpful, But Not a Cure-All

Does breathwork work against anxiety and depression? At the meta-analysis level, yes — but the most rigorous individual study qualifies this considerably 🟡.
Is every breathing technique equally helpful? No — what matters is whether it's used for regulation or for avoidance.
Does breathing technique replace psychotherapy? No, see the warning box above.

Sources: Fincham, Strauss, Montero-Marin & Cavanagh (2023), Scientific Reports (meta-analysis). Fincham, Strauss & Cavanagh (2023), Scientific Reports (coherent breathing RCT).

Part 9 — What's Often Claimed

Volume 1, Part 11 flagged this topic but left it open: “For claims about a direct connection between specific breathing techniques and digestion [...] or increased life expectancy, the current evidence is too thin for robust recommendations.” This chapter follows through on that — not as a blanket rejection, but as a nuanced examination, one that sometimes turns up a real but overstated effect instead of just “true” or “false.”

Breathing and Digestion: Real, But Narrower Than Claimed 🟡

Unlike most claims in this chapter, this one isn't simply false. A systematic review of 10 randomized trials with 476 participants (Abureesh et al., 2026) finds a moderate improvement in reflux symptoms (GERD) from diaphragmatic breathing as an adjunct to standard treatment, over roughly five weeks of training. An accompanying study in 60 people with reflux-related chronic cough additionally found improvements in anxiety, sleep quality, and depressive symptoms in the breathing training group 🟡.

The mechanism is presumably not the often-claimed “organ massage,” but a vagal-autonomic effect (already covered in Volume 1, Part 6), as well as possibly a mechanical effect on pressure at the lower esophageal sphincter — specific to reflux, not a general “digestive improvement.” This is the most nuanced answer in this chapter: a narrow, specific benefit is regularly inflated in marketing into a general claim of effectiveness.

Longevity and Mitochondrial Marketing 🔴

That mitochondrial function is linked to longevity is real and well-established in basic research. Not supported is the causal chain derived from this in marketing: “Breathing technique X exposes the mitochondria to mild stress → this extends human lifespan.” For exactly this chain, throughout the entire research for this volume — including for IHHT, covered in Part 6 — no peer-reviewed evidence in humans was found 🔴.

Specifically on autophagy (the cellular “clean-up process” often cited as the mechanism of action): a systematic review (Chen et al., 2023) shows that exercise does genuinely influence autophagy in humans — but depending on training type and tissue: acute strength training suppresses it in skeletal muscle, chronic strength training increases it there, and endurance training shows no significant change in the main markers. Notably, breathing isn't mentioned a single time in this entire review. Autophagy marketing for breathing techniques rides on a genuine but, even for exercise and fasting in humans, still uncertain research field — for breathing specifically, there's simply no direct investigation 🔴.

“Detoxification Through Breathing” 🔴

Physiologically, breathing primarily eliminates CO_2 — the metabolic waste product of cellular respiration, not a “toxin” in the colloquial sense. The figure widespread in the wellness sector, that the body eliminates “70% of all toxins” through breathing, repeatedly appears on commercial health websites but couldn't be traced to any independent scientific source 🔴 — the same pattern of precise-sounding but unsupported figures already sorted out several times in this compendium (Part 3, Part 4, Part 6). At most, an indirect effect remains plausible: deepened breathing can support lymphatic circulation via the pumping action of the diaphragm — which is fundamentally different from active “detoxification.”

“Optimizing Oxygen Supply” 🔴

Volume 1 already showed: arterial oxygen saturation in healthy people at rest is practically always in the high normal range (95–100%) — close to its physiological maximum. More breathing can barely raise this value any further. The claim that special breathing techniques could measurably “optimize” the oxygen supply of healthy people at rest thus directly contradicts the physiology already established in the first volume 🔴. The one relevant exception — elite endurance athletes with EIAH near VO_2max — was already covered in Part 4 of this volume and is a narrow special case, not an everyday phenomenon.

“Strengthening the Immune System” 🔴

The most-cited study on Wim Hof-style breathing and immune function (Kox et al., 2014, already addressed in Volume 1, Part 7, Category C) shows a dampened inflammatory response — not a general “strengthening” in the sense of greater immune defense. Whether a suppressed inflammatory response is desirable depends on context. The widespread simplification “this breathing technique strengthens your immune system” implies a direction of effect that the underlying study doesn't actually show 🔴.

Apps and Wearables: What's Measured, What's Extrapolated? 🟡

Consumer wearables mostly measure heart rate via photoplethysmography (PPG) — optical sensors, not ECG electrodes. PPG is prone to motion artifacts, poor skin contact, and ambient light. Validation studies show large differences between devices: the most accurate device tested (Oura Generation 4, heart rate variability) achieved a concordance correlation coefficient (CCC) of 0.99 versus the ECG reference measurement (Dial et al., 2025), while another device underestimated heart rate during activity by 24 to 32 beats per minute. Only about 11% of commercially available consumer wearables have so far been formally validated for at least one biometric measurement (Doherty et al., 2024).

The decisive point: most devices don't display raw heart rate variability, but a derived, company-proprietary score — “stress level,” “Body Battery,” “readiness” — based on models that aren't independently validated. That's not a direct measurement, but an interpretation, even when it's presented as an exact number 🟡. And no mass-market consumer wearable measures CO_2 or end-tidal CO_2 — an unremarkable HRV or SpO_2 reading says nothing about possible hyperventilation (see Part 1 and the capnography aside in Part 5).

Conclusion: Critical Doesn't Mean Dismissive

What's ultimately real? The GERD-specific effect of diaphragmatic breathing — narrowly limited, but real 🟡.

What's overstated? Almost everything else: longevity, autophagy, detoxification, oxygen optimization, immune strengthening — each a genuine research starting point that's expanded into an unsupported causal chain 🔴.

What do apps and wearables actually deliver? Raw data with varying, often unvalidated accuracy — none of the complex, often-advertised claims about “stress” or “recovery” is a direct measurement 🟡.

Sources: Chen et al. (2023), Autophagy Reports. Kox et al. (2014), PNAS (already in Volume 1). Abureesh et al. (2026), Journal of Clinical Medicine. Dial et al. (2025), Physiological Reports. Doherty et al. (2024), Sports Medicine.

Part 10 — What Remains

Volume 2 asked a different question than Volume 1. Volume 1 asked: which breathing techniques suit everyday life? Volume 2 asked: what happens when breathing is deliberately used as a training stimulus — in sport, medicine, and at physiological limits? The answer was rarely a simple yes or no.

Five Key Messages That Remain

1. **Resistance, pressure, CO_2 depletion, O_2 depletion are four different levers** — no single mechanism explains why IMT, strain breathing, hyperventilation, and altitude training work — each lever has its own effect, its own risks, its own evidence base (Part 1).

2. **A strong marker doesn't guarantee an everyday benefit** — this showed up repeatedly, with IMT and COPD walk distance (Part 2), long-COVID exercise tolerance (Part 7), and the strength-versus-blood-pressure question with Valsalva (Part 3).

3. **Study size and control quality change the outcome** — most clearly with coherent breathing: same lead author, same year, opposite result depending on study design (Part 8).

4. **Suitability is its own question, independent of evidence and safety** — clearest with post-exertional malaise: a well-documented, safe technique can still be the wrong choice for a particular group (Part 1, Part 7).

5. **Many popular claims ride on genuine but misinterpreted research** — longevity, autophagy, detoxification — the underlying research field often really exists, but the specific claim about breathing techniques usually doesn't (Part 9).

What This Volume Deliberately Doesn't Cover

Like Volume 1, this volume can't cover everything either. The following topics were deliberately left out — not because they're unimportant, but because each represents its own distinct subject area with its own body of research:

Breathing and voice, singing: controlling airflow while singing and speaking is a distinct field of voice physiology with its own specialist literature, going beyond the breathing techniques covered in this compendium.

Breathing technique in psychotherapy as a standalone method: Part 8 situated breathing technique as one component within existing forms of therapy (such as CBT for panic disorder). An in-depth presentation of specific breath-centered schools of therapy would exceed the scope of a self-help compendium.

Cardiopulmonary exercise testing and clinical breathing diagnostics: instrumented performance diagnostics (VO_2max measurement, lactate threshold determination) is a diagnostic procedure, not a breathing technique — and was therefore already deliberately excluded from this volume's concept.

In-depth capnography: Part 5 introduced the basics of $etCO_2$ measurement. An in-depth clinical presentation — such as in anesthesia or emergency medicine — goes beyond the scope of this compendium.

Closing Words

Volume 1 ended with a practical ten-minute routine. Volume 2 ends without a comparable set of instructions — for good reason: the levers covered here are training stimuli, not everyday habits. Anyone who wants to start IMT, use strain breathing in strength training, or explore altitude training in more depth will find, in the respective chapters, the physiological foundation, the Suitability Rating as orientation — and, where necessary, the clear reminder that these pages convey knowledge, not professional guidance.

Appendix

Glossary (Supplement to Volume 1)

This list supplements the glossary from Volume 1 with terms newly introduced in Volume 2. For Bohr effect, BOLT, CO_2 tolerance, HRV, hypocapnia, NO, and RSA, see Volume 1.

Bracing: voluntary bracing of the trunk muscles to stabilize the spine. To be distinguished from the Valsalva maneuver, with which it's often confused (Part 3).

Suitability: this volume's third rating dimension, alongside the Evidence Rating and Safety symbols from Volume 1. Doesn't answer “does it work?” or “is it dangerous?”, but “who is it right for?” (**General**, **Individual**, **Not without professional guidance**). See Part 0.

EIAH (exercise-induced arterial hypoxemia): exercise-induced arterial oxygen undersaturation during exertion near VO_2max, affecting an estimated 40–50% of the endurance elite, practically irrelevant in untrained individuals (Part 4).

ETAP (exercise-related transient abdominal pain): the technical term for a side stitch. Best-supported explanation: irritation of the parietal peritoneum, not diaphragmatic ischemia or muscle cramp (Part 4).

GET (graded exercise therapy): a therapeutic approach of gradually increasing activity. Currently considered ineffective and potentially harmful in post-exertional malaise (PEM), since any increase in exertion can retrigger PEM (Part 7).

HVB (high-ventilation breathwork): an umbrella term for breathing techniques with deliberately increased ventilation (including Holotropic Breathwork, rebirthing, Sudarshan Kriya Yoga, cyclic hyperventilation). Physiologically a shared mechanism, practically not a uniform category (Part 5).

IAP / ITP (intra-abdominal / intrathoracic pressure): pressure in the abdominal or chest cavity, which can rise sharply through strain breathing and is thought to contribute to spinal stabilization. Magnitude strongly dependent on the exercise (Part 3).

IHT (intermittent hypoxic training): repeated, brief hypoxia exposure during training. A recent meta-analysis finds no significant added benefit over normoxic training, with a demonstrated publication bias (Part 6).

IHHT (intermittent hypoxic-hyperoxic training): a deliberate alternation between hypoxia and hyperoxia phases. Solid clinical evidence in long COVID, but no human evidence for marketed longevity claims (Part 6, Part 9).

Capnography / etCO_2: measurement of end-tidal CO_2 (normal range 35–45 mmHg). Independent of oxygen saturation (SpO_2) — a pulse oximeter doesn't display CO_2 status (Part 1, Part 5).

LHTL (live-high-train-low): an altitude training protocol: living at moderate altitude, training at lower altitude. Improves competitive performance by about 1.5% on average (range 0–6%) via an increase in red blood cell mass (Part 6).

LRC (locomotor-respiratory coupling): mechanical coupling of breathing and stride rhythm while running, usually at a 2:1 ratio. Influenced by the impact forces of foot contact (Part 4).

Metaboreflex (respiratory muscle metaboreflex): a sympathetically mediated reflex response to fatigued respiratory muscles: blood flow is diverted away from limb muscles in favor of the respiratory muscles. Trainable via IMT (Part 2).

MIP / PImax, MEP: maximal inspiratory or expiratory pressure — the standard measure of respiratory muscle strength, measured against a closed system after full exhalation or inhalation, respectively (Part 2).

Normobaric / hypobaric hypoxia: oxygen reduction at normal air pressure (e.g., an altitude tent) versus through actually reduced air pressure (real altitude). Whether the two are physiologically equivalent isn't conclusively settled in the specialist literature (Part 6).

PEM (post-exertional malaise): a disproportionate worsening of symptoms after physical or mental exertion in long COVID/ME-CFS, with an unpredictable recovery time ranging from days to months. Graded increases in exertion are considered potentially harmful here (Part 7).

Shallow water blackout: sudden underwater unconsciousness from oxygen deprivation, usually after prior hyperventilation: the artificially lowered CO_2 level delays the natural urge to breathe, while oxygen saturation keeps dropping unnoticed — the warning signal fails to appear (Volume 1, Part 1; Part 6).

Dive reflex (mammalian dive reflex): an automatic response to facial contact with cold water: heart rate drops (bradycardia), blood flow is redirected from the extremities to the brain and heart, the spleen releases additional red blood cells — lowers oxygen consumption, but doesn't change the fact that the oxygen reserve in the blood is finite (Part 6).

Valsalva maneuver: forced exhalation against a closed glottis, producing high intrathoracic and intra-abdominal pressure. To be distinguished from bracing and forced exhalation with an open glottis (Part 3).

Evidence, Safety, and Suitability: The Three Ratings Explained

For the full explanation, see Part 0. The Evidence Rating and Safety symbols were carried over unchanged from Volume 1; suitability is new in this volume and answers a different question than the first two (see the glossary entry above).

Extended Safety and Caution Matrix

See the extended safety and caution matrix in Part 1 — it extends the matrix from Volume 1 with strain breathing, respiratory muscle training, and hypoxia exposure.

Bibliography, Organized by Study Type and Source Type

This structure organizes the sources cited in Volume 2 by publication and study type. It doesn't represent an independent assessment of scientific evidence strength — the evidence rating is provided via the ratings in the respective chapter. Sources from Volume 1 (e.g., Kox et al., 2014) are listed there and aren't repeated here.

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