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REVIEW ARTICLE

Breath of Life: *Pranayama* in Hypertension Management—A Narrative Review of Evidence and Emerging Mechanisms

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Abstract

Background: Hypertension remains the largest modifiable contributor to premature cardiovascular death worldwide, yet detection and control remain poor even where drug treatment is available. Regulated yogic breathing (*pranayama*) is widely promoted as an adjunctive measure, and a substantial body of trials now examines its effects on blood pressure (BP) and autonomic function. **Objective:** To synthesise clinical and mechanistic evidence on *pranayama* in hypertension, to situate the practice within its classical Yoga and Ayurvedic framework, and to identify the methodological gaps that currently constrain clinical recommendations. **Evidence synthesis:** Controlled trials in hypertensive adults report BP reductions of approximately 5–8 mmHg systolic and 2–5 mmHg diastolic with supervised *pranayama* programmes lasting four weeks to three months; pooled estimates are most consistent against usual care or wait-list comparison. Meta-analyses of breathing exercises and of *pranayama* specifically show statistically significant but modest reductions in BP and heart rate, with yoga trials of similar magnitude; certainty of evidence is gradeable only as very low. Mechanistic studies consistently demonstrate increased baroreflex sensitivity, vagal predominance and reduced sympathetic activity during slow breathing at about six breaths per minute, alongside preliminary signals of reduced oxidative stress and improved sleep. Device-guided slow breathing, in contrast, has not convincingly shown a specific antihypertensive effect in blinded, active-controlled trials. Protocol heterogeneity, small samples, short follow-up and scarce ambulatory BP data remain the principal limitations. **Conclusion:** *Pranayama* is best understood as a low-cost, generally well-tolerated adjunct within guideline-directed hypertension care rather than a substitute for pharmacotherapy. Adequately powered trials with ambulatory endpoints and active comparators are needed.

Keywords: *Pranayama*; Hypertension; Blood Pressure; Yoga; Autonomic Regulation; Heart-Rate Variability; Complementary Medicine

Introduction

Hypertension remains the most consequential modifiable risk factor in cardiovascular medicine. In 2024, an estimated 1.4 billion adults aged 30–79 years were living with raised blood pressure—roughly one-third of the world's population in that age band—while about 600 million of them (44%) were unaware of the diagnosis and only around 320 million (23%) had it controlled.¹ Pooled analyses of 1201 population-representative studies covering more than 104 million participants indicate that detection, treatment and control improved only modestly between 1990 and 2019, and that performance in most low- and middle-income countries remains well below what the available interventions could achieve.² Sustained elevation of arterial pressure accelerates atherogenesis, promotes left ventricular hypertrophy and heart failure, and is a dominant driver of stroke, ischaemic heart disease, chronic kidney disease and premature death.^{3,4}

Contemporary guidance therefore places lifestyle modification at the foundation of care for every person with elevated BP, whether or not pharmacotherapy is indicated, and treats non-pharmacological measures as cumulative rather than alternative to drug treatment. The Indian literature tells a particular story: hypertension is arguably the most important chronic disease risk factor in the country, control rates are low, and the burden is increasingly concentrated in younger adults and rural populations, which makes inexpensive, scalable, self-administered adjuncts attractive.^{5,6} It is in this space that breathing-based practices have attracted attention.

Pranayama is not a single technique but a family of regulated breathing practices embedded within the eight-limbed framework of classical Yoga. *Patanjali's Yoga Sutras* define *Pranayama* as the regulation of the incoming and outgoing flow of breath once posture has been steadied, while the *Hatha Yoga Pradipika* devotes

its second chapter to the purification of the *Nadis*, to specific breathing methods, and to an explicit warning that improper practice generates disease even though correct practice is held to remove it.⁷ That classical caution has a modern counterpart: any recommendation for *Pranayama* in a hypertensive population must be accompanied by clarity about technique, dose and contraindications.

Modern scientific interest in breath regulation and cardiovascular control has its own lineage, beginning with observations that paced slow breathing alters autonomic outflow and baroreflex gain in healthy volunteers, and extending through controlled studies in heart failure and essential hypertension. Reviews of *Pranayama* as a discrete intervention across health conditions have mapped a heterogeneous literature in which respiratory, autonomic and psychological outcomes are measured in different ways.⁸ In parallel, the American Heart Association has issued a scientific statement on meditation and cardiovascular risk reduction⁹, signalling that mind–body practices are no longer marginal to cardiology discourse.

Two problems complicate interpretation. First, the terminology is loose: “slow breathing” in biomedical usage often denotes a non-traditional device-guided protocol, whereas *Pranayama* carries specific technical and philosophical content. Second, many trials deliver *Pranayama* as one component of a multi-component yoga programme, so its independent contribution cannot be isolated.^{8,10} Conversely, systematic reviews of structured *Pranayama* alone are few, small and clinically heterogeneous.¹⁰ Device-guided slow breathing was once endorsed as a BP-lowering technology but has not weathered blinded, active-controlled scrutiny.^{11,12} The present review therefore deliberately focuses on *Pranayama* and closely related yogic breathing practices in hypertension, while using the device-guided literature as an instructive contrast.

Aims and Objectives

This narrative review aims to examine, critically and with explicit attention to the quality of the underlying evidence:

- the effects of *Pranayama* on systolic and diastolic blood pressure in hypertensive and prehypertensive adults;

- its effects on heart rate, heart-rate variability and autonomic balance;
- the putative cardiovascular mechanisms, particularly baroreflex and respiratory–cardiovascular coupling;
- the evidence from controlled trials, systematic reviews and meta-analyses, including their limitations;
- the relevance of classical Yogic and Ayurvedic concepts to contemporary understanding;
- safety considerations, practical prescription parameters, and the priorities for future research.

Methodological Approach

This is a narrative review; it does not claim the rigour of a systematic review and no PRISMA-compliant selection process was applied. Literature was identified through searches of PubMed/MEDLINE, Scopus, Web of Science, the Cochrane Library and the AYUSH Research Portal, with bibliographic details of every cited record verified against its indexed PubMed entry. Classical material was drawn from published English translations of the *Hatha Yoga Pradipika* rather than from secondary summaries.⁷ Search strings combined terms for the intervention and the condition: “*Pranayama* AND hypertension”, “*Pranayama* AND blood pressure”, “yogic breathing AND hypertension”, “slow breathing AND blood pressure”, “yoga AND hypertension”, “*Pranayama* AND heart rate variability”, “baroreflex AND slow breathing”, “breathing exercise AND blood pressure”, and the Ayurvedic terms “*Raktagata Vata*” and “hypertension AND Ayurveda”. Searches were run from database inception to September 2026, without language restriction.

Priority was given to randomised controlled trials (RCTs), systematic reviews and meta-analyses, controlled clinical studies in hypertensive populations, human physiological and mechanistic studies, and authoritative guidelines. Case reports, editorials without primary data, commercial wellness material and non-peer-reviewed sources were excluded, as were records whose bibliographic details could not be independently confirmed. Where a trial measured BP acutely (immediately after a single practice session) this is stated explicitly, since acute and training effects answer different questions.

Traditional and Conceptual Background

In the Ayurvedic literature, hypertension has no single classical designation; a set of reasoned parallels has been proposed, including *Raktaḡata Vata* (vitiated *Vata* lodged in the blood tissue), *Siraḡata Vata* and *Avṛta Vata*. Menon and Shukla, reviewing this literature comprehensively, argued that hypertension is better understood as a *Prasara-Avastha*—a stage in which vitiated *Doṣas*, principally *Vyana Vata*, *Praṇa Vata*, *Sadhaka Pitta* and *Avalambaka Kapha*, spread along with *Rakta* from their sites of origin—than as a discrete disease entity, and they mapped this onto the ṣaṭ-*Kriyakala* (six stages of *Doṣic* imbalance) and onto the concept of *Avaraṇa*, the occlusion of normal *Vata* function.¹³ The framing is conceptually interesting because it treats asymptomatic early hypertension as latent pathogenesis rather than as health.

The physiological bridge is straightforward. *Vyana Vata* is described as propelling blood from the heart and distributing it through the body; the *hṛdaya* is its seat, and *Praṇa Vata* governs respiration. Read alongside modern physiology, this describes a circulation–respiration axis whose disturbance manifests as disturbed pressure and flow. Reviews of integrative Ayurveda–Yoga approaches to hypertension have used exactly this logic to justify combining *Sodhana* procedures, herbal formulations and breathing practices.¹⁴ Two cautions are warranted. First, this hermeneutic exercise does not by itself establish efficacy; the parallel is explanatory, not evidential. Second, it should not obscure the fact that the strongest evidence for *Pranayama* comes from physiological and clinical studies conducted with modern methodology, and that the rigorous clinical evidence for Ayurvedic pharmacological interventions in hypertension is itself modest and heterogeneous.³⁵

Evidence from Controlled Trials

Trials with *Pranayama* as the principal intervention

The most instructive trial for mechanistic reasoning is that of Mourya and colleagues, who randomised 60 patients with stage 1 essential hypertension into slow-breathing, fast-breathing and control groups for three months. BP declined longitudinally in both intervention arms, but only the slow-breathing group showed significant improvement in the standing-to-lying ratio,

the 30:15 ratio, the expiratory-to-inspiratory heart-rate ratio, and the pressor responses to handgrip and cold pressor testing.¹⁵ The finding that fast breathing did not reproduce these autonomic gains anticipated a conclusion reached repeatedly since: slow, not merely forceful, breathing carries the autonomic benefit.

Two randomised trials in hypertensive adults have tested specific cooling *Pranayama* s. Shetty and colleagues randomised 60 hypertensive participants aged 25–65 years to *Sheetali* and *Sheetkari Pranayama*, practised for 10 minutes daily, or to a wait-list control; BP, autonomic and respiratory function were compared before and after the intervention.¹⁶ In a larger three-month trial, 100 patients with primary hypertension were allocated to *Sheetali Pranayama* or to no *Pranayama*; the intervention group showed significant reductions in BP variables and short-term HRV indices consistent with parasympathetic dominance.¹⁷ A comparative randomised study of 100 hypertensive patients at a naturopathy hospital examined 20-minute sessions of Nadi Shodhana versus Bhramari *Pranayama*, with HRV and auditory reaction time as the primary outcomes, addressing the practically important question of whether different *Pranayama* s are interchangeable.¹⁸ An RCT of 70 patients with essential hypertension evaluated the immediate effect of bee-humming breathing (*Bhramari*) on BP parameters and HRV.¹⁹

Acute studies add a dimension that training studies cannot. In a carefully designed experiment in 39 healthy volunteers, five minutes of *Bhastrika* at six breaths per minute reduced both systolic and diastolic pressure with a slight fall in heart rate; when ten participants repeated the exercise after oral hyoscine-N-butylbromide, a parasympathetic blocker, the BP response was abolished.²⁰ This pharmacological dissection is among the strongest available evidence that the acute hypotensive response to slow *Pranayama* is vagally mediated rather than a measurement artefact.

Trials of yoga programmes containing *Pranayama*

Because *Pranayama* is usually taught within a broader yoga protocol, several RCTs speak to its clinical context rather than to its isolated effect. A randomised trial in 57 elderly men with grade I hypertension compared three months of supervised yoga with supervised brisk walking

and found significant reductions in serum malondialdehyde alongside increases in superoxide dismutase, reduced glutathione and vitamin C in the yoga arm, with the opposite trend for oxidative markers in the walking arm.²¹ An RCT of 80 patients with diagnosed hypertension tested two months of Om chanting combined with Yoga Nidra, five days per week, and reported significantly greater reductions in systolic and diastolic BP and LDL cholesterol, and a rise in HDL, than in the control group, with no adverse events recorded during the trial.²² More recently, a randomised trial in 105 elderly hypertensive participants compared 24 weeks of standard treatment with standard treatment plus home-based, mobile-guided *Pranayama* and *Yoga Nidra*; the intervention arm showed a statistically significant reduction of 4.7 mmHg in systolic BP and

improved sleep quality.²³

Ambulatory BP outcomes, which are less vulnerable to measurement artefact, remain rare. Hagins and colleagues randomised 84 adults with prehypertension or stage 1 hypertension to yoga or to a non-aerobic active control and measured 24-hour ambulatory BP. Within the yoga group, 24-hour diastolic pressure fell by 3.93 mmHg, night-time diastolic pressure by 4.7 mmHg and mean arterial pressure by 4.23 mmHg, with no significant within-group changes in the control arm; between-group comparison reached significance only for night-time diastolic pressure.²⁴ The authors candidly noted that the study was underpowered for between-group inference—an honest characterisation of much of this field.

Table 1. Selected Controlled Trials of *Pranayama* or Yogic Breathing in Hypertension

Study (year)	Design and population	Intervention and dose	Duration	Principal Findings
Mourya et al. (2009) ¹⁵	RCT, 3 groups; 60 adults, stage 1 essential hypertension	Slow-breathing vs fast-breathing vs control	3 months	BP fell in both intervention arms; significant autonomic reflex improvements (S/L, 30:15, E/I ratios, handgrip, cold pressor) only with slow breathing
Shetty et al. (2017) ¹⁶	RCT; 60 hypertensive adults (25–65 y)	Sheetali + Sheetkari, 10 min/day vs wait-list	Trial-defined (before/after)	Changes in BP, autonomic and respiratory function in the intervention group
Thanalakshmi et al. (2020) ¹⁷	RCT; 100 patients with primary hypertension	Sheetali <i>Pranayama</i> vs no <i>Pranayama</i>	3 months	Significant reduction in BP variables; HRV time- and frequency-domain indices shifted toward parasympathetic dominance
Upadhyay et al. (2023) ¹⁸	Randomised comparative trial; 100 hypertensive inpatients	Nadi Shodhana vs Bhramari, 20 min	Single session (immediate effects)	Comparative HRV, auditory reaction time and BP outcomes after each practice
Ghati et al. (2021) ¹⁹	RCT; 70 patients with essential hypertension	Bee-humming (<i>Bhramari</i>) breathing	Single session (immediate effects)	Immediate effects on BP parameters and HRV
Pramanik et al. (2009) ²⁰	Experimental study; 39 volunteers (n = 10 in blocker substudy)	Slow <i>Bhastrika</i> , 6 breaths/min, 5 min; repeated after hyoscine-N-butylbromide	Acute	Both SBP and DBP fell with slight HR reduction; response abolished by parasympathetic blockade
Patil et al. (2014) ²¹	Open parallel-arm RCT; 57 elderly men, grade I hypertension	Yoga vs supervised brisk walking	3 months	Reduced malondialdehyde; increased SOD, GSH and vitamin C in the yoga arm
Anjana et al. (2022) ²²	RCT; 80 patients with diagnosed hypertension	Om chanting + <i>Yoga Nidra</i> , 5 days/week	2 months	Greater reduction in SBP, DBP and LDL and higher HDL than control; no adverse events reported
Akshita et al. (2025) ²³	RCT; 105 elderly hypertensive participants	Standard care ± mobile-guided <i>Pranayama</i> + <i>Yoga Nidra</i> at home	24 weeks	Significant SBP reduction (4.7 mmHg) and improved sleep quality versus control
Hagins et al. (2014) ²⁴	RCT with active control; 84 adults, prehypertension/stage 1 hypertension	Yoga vs non-aerobic active control; 24-h ambulatory BP	Trial-defined	Within-group reductions in 24-h diastolic, night diastolic and MAP; only night diastolic differed between groups (P = 0.038)

Evidence from Systematic Reviews and Meta-analyses - Pooled analyses converge on a modest, statistically significant signal. Garg and colleagues

pooled 15 trials of breathing exercises in hypertension and reported reductions of 7.06 mmHg in systolic BP (95% CI −10.20 to −3.92), 3.43 mmHg in diastolic BP

(95% CI -4.89 to -1.97) and 2.41 beats per minute in heart rate (95% CI -4.53 to -0.30) [25]. The most specific synthesis—seven RCTs, 683 participants, restricted to *Pranayama* in hypertension—found a significant reduction in heart rate (standardised mean difference -0.43; 95% CI -0.52 to -0.34; $I^2 = 0\%$) and a significant systolic BP reduction under a fixed-effects model (SMD -0.72), while diastolic BP and HRV improvements were described as modest.²⁶ An earlier meta-analysis of 17 slow-breathing trials, using an explicit threshold definition of ≤ 10 breaths per minute for ≥ 5 minutes on ≥ 3 days per week over ≥ 4 weeks, reported an overall systolic reduction of 5.62 mmHg (95% CI -7.86 to -3.38), and explicitly separated device-guided from non-device-guided (*Pranayama* - type) interventions.²⁷

That separation matters, because the device-guided literature is where enthusiasm has most clearly outrun evidence. Mahtani and colleagues pooled eight trials of a commercial paced-breathing device in 494 patients and found significant reductions in systolic (3.67 mmHg; 95% CI -5.99 to -1.39) and diastolic (2.51 mmHg; 95% CI -4.15 to -0.87) pressure; when the five trials sponsored by or involving the device manufacturer were excluded, no overall effect on BP remained.¹¹ An individual-patient-data meta-analysis confined to blinded, active-controlled trials was likewise unable to substantiate a specific antihypertensive effect of device-guided slow breathing.¹² A more recent systematic review of 22 studies involving 17,214 participants found that device-guided slow breathing did not significantly reduce BP compared with usual care (systolic mean difference -2.13 mmHg; 95% CI -12.71 to 8.44), with high heterogeneity; non-device-guided slow breathing could be assessed only qualitatively because just three studies were available.²⁸ The lesson is not that breathing is irrelevant, but that unblinded designs and device-specific ritual effects can manufacture apparent benefit. *Pranayama* practised without apparatus fares better in pooled analyses. A meta-analysis of alternate-nostril breathing reviewed 14 eligible studies and pooled six RCTs (525 participants), finding significant reductions in both systolic and diastolic pressure for the practice alone and when embedded in yoga programmes.²⁹ Yoga meta-analyses describe similar magnitudes. Geiger and colleagues, pooling 30 RCTs with 2283 participants,

reported reductions of 7.95 mmHg systolic (95% CI -10.24 to -5.66) and 4.93 mmHg diastolic (95% CI -6.25 to -3.60) versus wait-list control, but only -4.16 mmHg systolic (95% CI -10.76 to 2.44) versus active control—a difference that did not reach significance—and graded the certainty of evidence as very low.³⁰ Wu and colleagues found that BP reductions in yoga trials were roughly twice as large in samples with hypertension (approximately 6/3 mmHg) as in normotensive samples.³¹ In prehypertension, a meta-analysis of eight RCTs found significant reductions in both systolic (SMD -0.62; 95% CI -0.83 to -0.41) and diastolic pressure (SMD -0.81; 95% CI -1.39 to -0.22).³² Nalbant and colleagues analysed not merely whether yoga works but what an effective intervention looks like: across 34 RCTs and 38 interventions, yoga reduced systolic BP by 6.49 mmHg (95% CI -8.94 to -4.04) and diastolic BP by 2.78 mmHg (95% CI -4.11 to -1.45), and the interventions that improved BP typically contained all three components—postures, *Pranayama*, and meditation or relaxation—with comparable time allocated to each, whereas ineffective interventions emphasised postures and devoted longer periods to them.³³ A recent network meta-analysis of mind-body therapies in essential hypertension ranked Bhramari *Pranayama* among the better-performing interventions for diastolic pressure reduction.³⁴ Finally, for completeness, meta-analytic evidence for Ayurvedic pharmacological and *Panchakarma* interventions in essential hypertension shows good safety but no significant BP advantage over either placebo or standard antihypertensives, with high heterogeneity ($I^2 > 90\%$)³⁵—a reminder that the traditional-system rationale is shared by practices with quite different evidential standing.

Mechanisms: What Slow Breathing Does to the Circulation

Baroreflex gain and respiratory-cardiovascular resonance

The most reproducible mechanistic finding is an increase in arterial baroreflex sensitivity during slow breathing. Joseph and colleagues studied 20 patients with essential hypertension and 26 controls during spontaneous breathing and controlled breathing at 6 and 15 breaths per minute. At six breaths per minute, systolic pressure fell from 149.7 ± 3.7 to 141.1 ± 4 mmHg

Table 2. Systematic Reviews and Meta-Analyses of Breathing Practices and Yoga in Hypertension

Study (year)	Studies / participants	Intervention	Pooled effect	Notes
Garg et al. (2024) ²⁵	15 studies	Breathing exercises	SBP -7.06 mmHg (-10.20 to -3.92); DBP -3.43 mmHg (-4.89 to -1.97); HR -2.41 bpm (-4.53 to -0.30)	Modest but significant effects
Chidambaram et al. (2026) ²⁶	7 RCTs / 683	<i>Pranayama</i>	HR SMD -0.43 (-0.52 to -0.34; I ² = 0%); SBP SMD -0.72 (fixed effects)	DBP and HRV changes modest; authors call for larger trials
Chaddha et al. (2019) ²⁷	17 studies	Slow breathing (≤10/min, ≥4 wk)	SBP -5.62 mmHg (-7.86 to -3.38)	Device-guided and non-device-guided distinguished
Mahtani et al. (2012) ¹¹	8 trials / 494	Device-guided breathing	SBP -3.67 mmHg (-5.99 to -1.39); DBP -2.51 mmHg (-4.15 to -0.87)	No overall effect after excluding manufacturer-linked trials
Landman et al. (2014) ¹²	Individual patient data, blinded active-controlled trials	Device-guided breathing	No evidence of a specific antihypertensive effect	Design-strength contrast to unblinded trials
Gonçalves et al. (2022) ²⁸	22 studies / 17,214	Device-guided vs non-device-guided	SBP MD -2.13 mmHg (-12.71 to 8.44) for device-guided	Non-device-guided assessable only qualitatively (3 studies)
Nam et al. (2024) ²⁹	14 studies; 6 RCTs pooled / 525	Alternate-nostril breathing	Significant reductions in SBP and DBP	Benefit for practice alone and within yoga programmes
Geiger et al. (2025) ³⁰	30 RCTs / 2283	Yoga (incl. <i>Pranayama</i>)	SBP -7.95 mmHg; DBP -4.93 mmHg vs wait-list; SBP -4.16 mmHg (-10.76 to 2.44) vs active control	Certainty of evidence graded very low
Wu et al. (2019) ³¹	Yoga trials (systolic/diastolic endpoints)	Yoga as antihypertensive lifestyle therapy	Approximately 6/3 mmHg in hypertensive samples	Effect roughly double that in normotensive samples
Khandekar et al. (2021) ³²	8 RCTs	Yoga in prehypertension	SBP SMD -0.62 (-0.83 to -0.41); DBP SMD -0.81 (-1.39 to -0.22)	Secondary outcomes also improved
Cui et al. (2026) ³⁴	Network meta-analysis of RCTs	Mind-body therapies in essential hypertension	Comparative ranking	<i>Bhramari Pranayama</i> ranked favourably for diastolic BP
Deshmukh et al. (2025) ³⁵	44 studies; 10 RCTs / 524 in meta-analysis	Ayurvedic interventions	SBP -2.63 mmHg (-6.04 to 0.79) vs placebo; -0.22 (-0.82 to 0.38) vs antihypertensives	Good safety; no significant BP advantage; I ² > 90%

and diastolic from 82.7 ± 3 to 77.8 ± 3.7 mmHg in the hypertensive group, while baroreflex sensitivity rose from 5.8 ± 0.7 to 10.3 ± 2.0 ms/mmHg; at 15 breaths per minute only systolic pressure fell and baroreflex gain was unchanged.³⁶ Notably, hypertensives displayed faster spontaneous breathing rates and lower end-tidal CO₂ at baseline, suggesting mild chronic hyperventilation and chemoreflex activation that slow breathing appears to reverse without inducing hypoventilation. Comparative work in chronic heart failure had already shown the same qualitative phenomenon at a slower breathing rate³⁷, and a cross-sectional study in yoga practitioners found that slow breathing at 0.1 Hz was accompanied by lower heart rate, higher pNN50, reduced systolic BP variability and higher sequence and spectral baroreflex indices than in yoga-naïve controls.³⁸ The physiological basis for these observations is now reasonably well described. When

breathing is paced near 0.1 Hz—approximately six breaths per minute—respiratory and baroreflex oscillations come into phase, and heart-rate, BP and ventilatory oscillations amplify at that frequency, an effect variously described as resonance or coherence.^{39,40} Training at individually determined resonant frequency increases cardiac variability substantially, and this increase is the mediating mechanism proposed for the clinical effects of HRV biofeedback⁴¹, an intervention that is functionally a secular cousin of *Pranayama*.

Autonomic balance and heart-rate variability

HRV studies of *Pranayama* are numerous but small, and their designs vary. A component-analysis study examined which elements of *Pranayama* drive HRV changes, addressing the practical question of whether technique, pace or duration matters most.⁴² *Bhramari Pranayama* was associated with HRV changes both during and after practice⁴³, and *Nadi Shuddhi* produced

immediate HRV shifts consistent with increased vagal modulation in young adults.⁴⁴ These acute observations align with the pharmacological dissection described earlier²⁰ and with the trial-level finding that slow *Pranayama*, rather than fast kapalabhati-type techniques, improves autonomic reactivity in hypertension.^{15,17}

The clinical relevance of these indices is not trivial. In the ATRAMI study of patients after myocardial infarction, depressed baroreflex sensitivity and reduced HRV independently predicted total cardiac mortality.⁴⁵ Interventions that durably raise baroreflex gain therefore deserve serious consideration rather than being dismissed as relaxation effects, although—importantly—no *Pranayama* trial has yet demonstrated hard-outcome benefit, and autonomic markers are surrogate endpoints.

Adjunctive pathways: oxidative stress, sleep and stress physiology

Beyond the autonomic axis, several smaller studies point to pathways of potential interest. Three months of yoga in elderly men with grade I hypertension reduced serum malondialdehyde and raised superoxide dismutase, glutathione and vitamin C relative to brisk walking, suggesting an antioxidant effect not attributable to physical activity per se.²¹ Trials combining *Pranayama* with relaxation techniques have reported improvements in lipid fractions²² and in sleep quality among elderly hypertensives²³. Because short sleep duration and poor sleep quality are independent determinants of BP and of non-dipping status, the sleep finding may be more than incidental. Meditation-based programmes, which overlap with *Pranayama* in practice and in mechanism, have been systematically reviewed for psychological and physiological outcomes⁹, and stress reduction is a plausible mediator of at least part of the observed BP response.

Practical Prescription: Which Breathing, How Much, and For Whom?

The evidence does not support a single canonical protocol. The inclusion criteria used in the most explicit meta-analysis of slow breathing—a rate of ≤ 10 breaths per minute sustained for at least five minutes, on at least three days per week, for at least four weeks—represent a reasonable minimum dose framework.²⁷ Physiological reasoning favours approximately six breaths per minute

(0.1 Hz), the frequency at which baroreflex resonance is maximal.^{36,39,40} Trial protocols have used 10 minutes daily of a cooling *Pranayama*¹⁶, 20 minutes of Nadi Shodhana or Bhramari¹⁸, three months of supervised slow-breathing practice^{15,17}, and 24 weeks of home-based mobile-guided practice in the elderly.²³ Programmes combining postures, *Pranayama* and meditation, with comparable time given to each, have outperformed posture-dominant protocols.³³

For clinical translation, three practical points follow. *Pranayama* is best introduced as a supervised, technique-specific practice before it is transferred to unsupervised home use, since both trial evidence²³ and classical warning⁷ emphasise correct technique. It should be scheduled rather than opportunistic, because dose appears to matter. And it should be framed to patients as an addition to established therapy, with BP monitoring rather than subjective wellbeing as the arbiter of benefit.

Comparison with Device-Guided Breathing and Other Mind–Body Approaches

The contrast between *Pranayama* and device-guided paced breathing is informative for both clinical and epistemological reasons. Device-guided breathing was sufficiently well regarded that it was cited as a recommended non-pharmacological option in earlier cardiovascular guidance¹², yet pooled analyses that excluded manufacturer-linked trials¹¹ or restricted inclusion to blinded active-controlled designs¹² failed to confirm a specific effect. Non-device-guided slow breathing, by contrast, showed a pooled systolic reduction of 5.62 mmHg across 17 trials²⁷, although the most recent systematic review could analyse it only qualitatively because so few trials exist.²⁸ The unresolved question—whether the active ingredient is the breathing pattern itself, attentional focus, the expectation of benefit, or the regularity of a daily practice—applies equally to *Pranayama* and is the central methodological challenge for the field. Comparisons with meditation and yoga are similarly hedged: pooled analyses of yoga show consistent but modest reductions whose certainty is graded very low and which attenuate markedly against active controls^{30,31}, and evidence for Ayurvedic pharmacological management in hypertension is weaker still.³⁵

Safety, Limitations and Interpretive Caution

Pranayama is generally well tolerated in the trials reviewed here; the largest controlled trial in elderly hypertensives reported no adverse events, and no trial identified in this review reported a serious adverse event attributable to breathing practice.²² This does not make it risk-free. Classical texts explicitly warn that improper practice generates disease⁷, and forced, rapid techniques such as vigorous *bhastrika* or *kapalabhati* are physiologically opposite in effect to the slow practices associated with vagal and baroreflex benefit,^{8,15}. Breath retention and forceful breath-holding that involve Valsalva-like manoeuvres raise intrathoracic pressure and can transiently alter venous return and BP; prudence, though not trial evidence, supports avoiding these in uncontrolled hypertension, in patients with significant carotid or cerebrovascular disease, and in those with proliferative retinopathy. Patients should not reduce or stop antihypertensive medication on the basis of breathing practice, and any programme should be embedded in usual monitoring.

The limitations of the evidence base are substantial and consistent across reviews. Samples are typically small, single-centre and predominantly drawn from one or two countries; blinding of participants and assessors is rarely achieved; follow-up is usually short (frequently eight weeks, occasionally three to six months); and outcome measurement relies heavily on clinic BP, with ambulatory BP used in only a minority of studies.²⁴ Meta-analyses report high statistical heterogeneity^{28,35}, and the certainty of evidence has been formally graded as very low.³⁰ Publication bias is plausible, and several of the most positive early studies of paced breathing were industry-linked.¹¹ Protocols are heterogeneous with respect to technique, pace, duration and the co-intervention of postures and meditation, which limits the possibility of dose–response inference.^{8,10} Guidelines appropriately treat breathing practices as optional supportive measures within comprehensive lifestyle management rather than as antihypertensive therapy in their own right.^{3,4}

Priorities for Future Research

Several design choices would substantially strengthen this field. First, trials should use 24-hour ambulatory BP as the primary endpoint, ideally with home BP

monitoring to capture durability and non-dipping patterns. Second, active comparators are essential: an attention-control breathing task (for example, slow breathing at a rate outside the resonant range) would help separate the specific effect of resonant-frequency breathing from expectancy and relaxation. Third, interventions must be described in replicable detail—technique, rate, session length, frequency, posture, and whether retention is used—since the content analysis by Nalbant and colleagues indicates that protocol composition, not merely the presence of breathing, distinguishes effective from ineffective programmes.³³ Fourth, mechanistic endpoints should be embedded within clinical trials: baroreflex sensitivity, HRV, arterial stiffness, central haemodynamics and markers of oxidative stress and inflammation in the same participants over time. Fifth, dose–response studies are needed to establish whether the resonant frequency confers advantages over other slow rates in hypertensive populations. Finally, pragmatic trials in primary care and in the public health programmes that already deliver hypertension care in low-resource settings—where the appeal of *Pranayama* is greatest—would test scalability, adherence, safety and cost-effectiveness under conditions that resemble real practice, using standardised reporting of adverse events.

Conclusion

Pranayama occupies an unusual position in contemporary hypertension care: a practice with a coherent traditional rationale, reproducible acute autonomic effects, several positive randomised trials and meta-analytic support for modest reductions in blood pressure and heart rate, yet one whose evidence base remains small, heterogeneous and graded very low in certainty. Pooled estimates cluster around 5–8 mmHg systolic and 2–5 mmHg diastolic reduction against usual care, with the specific randomised evidence for *Pranayama* in hypertension centred on heart rate and systolic pressure. Mechanistically, slow breathing near six breaths per minute increases baroreflex sensitivity and shifts autonomic balance toward vagal predominance, which is a plausible route to sustained pressure reduction and is consistent with the prognostic importance of these indices in cardiac populations. What the field lacks is not plausibility but

rigour: adequately powered trials with ambulatory endpoints, active comparators, long follow-up and mechanistic embedding. Until such evidence exists, *Pranayama* is best offered to hypertensive patients as a safe, inexpensive and well-accepted adjunct to lifestyle and pharmacological management—useful, but never a replacement for either.

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Conflicts of Interest

There are no conflicts of interest.

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