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Immediate Autonomic Modulation Following Targeted Pranayama in Obese and Normal-Weight Females: A Two-Arm Pre-Post Pilot Investigation

Swetha .B.S [1] ,Uthara .K [1], Sureshbabu Venkatasamy [2]

[1]CRRI JSS Institute of Naturopathy and Yogic Sciences, Coimbatore, Tamil Nadu, India
[2]Research Officer, JSS Institute of Naturopathy and Yogic Sciences, Coimbatore, Tamil Nadu, India

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Abstract:

Background: *Dysautonomia and sympathetic overactivity establish a pathological mechanism in obesity, predisposing individuals to cardiovascular pathology. While prolonged interventions demonstrate robust benefits, the acute physiological responses to abbreviated yogic breathing techniques for obesity remain inadequately quantified across differing body mass index (BMI) strata.*

Methods: *A two-arm comparative pre-post pilot study evaluated eleven healthy females (aged 18–20 years; no comorbidities, co-infections, or recent hospitalisations). Participants were stratified into obese (n=6; BMI ≥ 30 kg/m^2) and normal-weight (n=5; BMI 21–24 kg/m^2) cohorts. The 15-minute intervention comprised Thoracic Mobilisations (3 minutes), Nadishuddhi Pranayama (4 minutes), Bhramari Pranayama (4 minutes), and Vibhagiya Pranayama (4 minutes). Autonomic and haemodynamic parameters were recorded at baseline and immediately post-intervention.*

Results: *The intervention elicited highly significant immediate reductions in both systolic and diastolic blood pressure within the obese cohort. The normal-weight group yielded significant reductions in systolic blood pressure and respiratory rate, but no significant changes in diastolic pressure. A unique physiological synthesis emerged: obese participants exhibited a marked, statistically significant increase in breath-holding time, indicating a differential respiratory capacity response to the intervention based on BMI.*

Conclusion: *Targeted yogic breathing interventions establish immediate haemodynamic stabilisation and autonomic modulation. The distinct physiological shifts between obese and normal-weight cohorts confirm these techniques function as precise, evidence-based applications for autonomic regulation. Future research needs to investigate longitudinal adaptations across broader clinical demographics.*

Keywords: Yogic breathing techniques, obesity, dysautonomia, haemodynamics, Pranayama, breath- holding time, autonomic regulation.

Introduction

Obesity establishes a profound pathological continuum characterised by severe dysautonomia, specifically manifesting as chronic sympathetic nervous system overactivity [1]. This persistent autonomic imbalance acts as a primary physiological driver of maladaptive haemodynamic responses, directly accelerating the trajectory towards cardiovascular morbidity. Pathophysiologically, excess adiposity induces hyperleptinemia, upregulates the renin-angiotensin-aldosterone system, and impairs baroreflex sensitivity, all of which culminate in a state of elevated sympathetic outflow [2]. Consequently, individuals with obesity demonstrate persistent sympathetic hyperactivation and significantly reduced heart rate variability (HRV) when compared to normal-weight cohorts, escalating their baseline cardiovascular risk profile [3]. The chronic elevation of sympathetic tone disrupts essential vagal modulation, resulting in sustained endothelial dysfunction, elevated systemic vascular resistance, and the early onset of hypertension. Therefore, rapidly identifying and mitigating this obesity-induced autonomic dysfunction remains an urgent, primary target for evidence-based cardiovascular risk reduction.

Non-pharmacological, naturopathic interventions, particularly structured yogic practices, exert significant and quantifiable modulatory effects on the human autonomic nervous system. Extensive longitudinal data validate the robust, systemic cardiometabolic benefits of sustained yoga interventions over months or years [4].

However, a critical scientific gap persists within the current literature regarding the immediate, acute physiological responses to targeted, sequential pranayama (yogic breathing) techniques. While current research adequately details the cumulative structural adaptations of long-term practice, it fails to quantify the precise, immediate autonomic and haemodynamic shifts occurring during and directly following a single, brief breathing session. The acute physiological responses to sequential, targeted pranayama remain inadequately quantified. Understanding these acute transient mechanisms provides the essential evidence-based foundation required for optimizing short-duration, high-yield therapeutic breathing protocols.

Pranayama functions as a precise somatosensory intervention that directly dictates autonomic nervous system firing rates and cardiovascular regulation. Slow, controlled yogic breathing unequivocally enhances parasympathetic vagal efferent activity and systematically subdues sympathetic dominance [5] . Physiologically, the voluntary modulation of tidal volume and respiratory frequency during these techniques amplifies the Hering-Breuer inflation reflex. This alters pulmonary stretch receptor firing rates, which subsequently send inhibitory impulses to the medullary cardiovascular centres [6] . This central integration immediately lowers the pulse rate, reduces both systolic and diastolic blood pressure, and optimises peripheral oxygen saturation (SpO2) through enhanced ventilation-perfusion matching. Furthermore, specific yogic breathing practices measurably extend breath-holding time by improving respiratory muscle efficiency, expanding vital capacity, and enhancing carbon dioxide tolerance at the central chemoreceptor level [7] .

The diverse baseline autonomic profiles of obese versus normal-weight individuals dictate distinct physiological trajectories and adaptations during paced breathing. Obese individuals operate from a baseline of heightened sympathetic arousal and restricted thoracic mobility, whereas normal-weight individuals typically exhibit a more balanced autonomic tone and functional respiratory mechanics. A carefully structured, sequential protocol systematically leverages and corrects these profiles. Thoracic mobilisations mechanically expand the rib cage and immediately improve chest wall compliance, counteracting the restrictive mechanical load of adipose tissue. Nadi Shuddhi (alternate nostril breathing) forces bilateral hemispheric engagement, dictating interhemispheric balance and directly reducing sympathetic outflow [8]. Bhramari(humming bee breath) induces profound parasympathetic dominance; the controlled vocalisation and specific facial mudras stimulate pharyngeal, laryngeal, and auricular branches of the vagus nerve [9]. Finally, Yogic Breathing, a sequential abdominal, intercostal, and clavicular breathing maximises alveolar ventilation and enforces a systemic state of parasympathetic activation across both cohorts.

To address the identified literature gap and quantify these mechanisms, this pilot investigation evaluated the short-term, immediate effects of a 15-minute sequential pranayama protocol on specific cardiovascular and pulmonary functions. We employed a rigorous two-arm, comparative pre-post pilot design. We recruited healthy female university students aged 18 to 20 years, enforcing strict exclusion criteria to eliminate confounding comorbidities, co-infections, or recent hospitalisations. We stratified the participants into an obese cohort (BMI 30 kg/m^2 - 35 kg/m^2) and a normal-weight cohort (BMI 20 - 24 kg/m^2) following established World Health Organization criteria. The primary objective was to quantify the acute changes in systolic blood pressure, diastolic blood pressure, respiratory rate, pulse rate, breath-holding time, and SpO2 immediately before and after the intervention. We hypothesised that the sequential yogic breathing techniques elicit immediate, positive, and quantifiable effects on autonomic variables in both the obese and normal-weight groups, thereby rejecting the null hypothesis of zero physiological effect.

Quantifying these acute physiological responses yields critical, objective insights into the real-time efficacy of yogic breathing as a primary autonomic modulator. Establishing the immediate haemodynamic benefits validates the utility of brief, targeted breathing protocols in high-stress or acute clinical settings where immediate autonomic correction is necessary. This pilot investigation provides the foundational, quantitative data necessary to power larger, randomised controlled clinical trials. Ultimately, the precise characterisation of these acute shifts

informs the direct integration of sequential pranayama into evidence-based, forward-thinking cardiovascular risk management strategies.

Aim of the Study is to evaluate the **acute effects of a 15-minute sequential pranayama intervention on haemodynamic and autonomic parameters** in normal-weight and obese young females, and to determine whether the intervention produces differential intra-group changes and inter-group standardisation of physiological responses.

2. Methodology

2.1 Study Design and Ethical Considerations We employed a rigorous two-arm, comparative pre-post pilot design to evaluate the acute physiological responses to a targeted yogic intervention. All experimental protocols strictly adhered to the ethical principles outlined in the Declaration of Helsinki for medical research involving human subjects. To fulfil absolute confidentiality requirements, we securely anonymised all participant identities and physiological data prior to analysis, ensuring no identifiable information appeared in the study records or subsequent reports.

2.2 Participants and Setting We recruited eleven healthy female university students, aged 18 to 20 years, from the Residential academic community. To isolate the physiological variables and eliminate pathophysiological confounders, we established strict inclusion criteria. Participants required a verified absence of any pre-existing comorbidities, active co-infections, or recent hospitalisations. Following initial screening, we stratified the participants into two distinct cohorts based on their body mass index (BMI), strictly adhering to established World Health Organization (WHO) classification standards for the Indian population. The final sample comprised an obese cohort (n=6; BMI 30 - 35kg/m^2) and a normal-weight cohort (n=5; BMI 21-24 kg/m^2). We conducted all interventions and data collection in a quiet, controlled environment to minimise external sensory stimuli and baseline sympathetic arousal.

2.3 Intervention Protocol We administered a continuous 15-minute pranayama session to all participants, as mentioned in the intervention protocol (Table 1). Prior to the intervention, an experienced yoga instructor briefed the subjects on the precise procedures and provided continuous, direct guidance throughout the session. Participants assumed a comfortable, upright seated posture on a yoga mat with their eyes closed to promote interoceptive awareness. The protocol mandated the following sequential, meticulously timed phases:

TABLE 1:

INTERVENTIONS	DURATION	PROCEDURE
THORACIC MOBILISATION	3 MINS	Participants executed a standardised sequence of biomechanical movements to mechanically optimise chest wall expansion and compliance. This included hands in- and-out stretches, hands up-and-down stretches, shoulder rotation exercises, and Marjari asana (cat and cow stretch), all performed at a slow, steady, and synchronised pace.
NADI SHUDDHI PRANAYAMA	4 MINS	Participants engaged in alternate nostril breathing to enforce interhemispheric autonomic balance. Using the right hand formed in Nasigra mudra, subjects alternately occluded the right and left nostrils. They maintained a precise 1:1 inhalation-to- exhalation ratio and explicitly avoided any breath retention (Kumbhaka).

BHRAMARI PRANAYAMA	4MINS	Following a deep bilateral inhalation, participants executed a prolonged exhalation while producing a soft, high-pitched humming sound. They maintained closed mouths to maximise internal acoustic resonance and directly stimulate the vagus nerve.
SEQUENTIAL YOGIC BREATHING	4MINS	The protocol concluded with deep, conscious respiratory regulation. Participants performed sequential abdominal, intercostal, and clavicular breathing, culminating in a controlled, extended exhalation to definitively establish systemic parasympathetic dominance

INTERVENTION TABLE

2.4 Outcome Measures and Data Collection To quantify the immediate autonomic and haemodynamic shifts, we recorded specific physiological parameters at two distinct time points: immediately prior to the intervention (baseline) and immediately following the cessation of the 15-minute protocol. We evaluated the following primary outcome variables using standardised, non-invasive clinical instrumentation: Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Pulse Rate (PR), Respiratory Rate (RR), Breath-Holding Time (BHT) and Peripheral Oxygen Saturation (SpO2).

3. Results

3.1 Intra-Group Haemodynamic and Autonomic Responses The statistical analysis establishes clear, variable-specific physiological adaptations within both cohorts following the 15-minute sequential pranayama intervention.

In the normal-weight cohort (Group 1, n=5), paired samples t-tests reveal statistically significant reductions in Respiratory Rate (Student's $t=3.586$, $p=0.023$) and Systolic Blood Pressure (Student's $t=4.472$, $p=0.011$). Consequently, we reject the null hypothesis for these specific parameters, confirming the immediate efficacy of the intervention in enhancing baseline parasympathetic tone in healthy individuals. Variables including Pulse Rate ($p=0.578$), Diastolic Blood Pressure ($p=0.587$), and Breath-Holding Time ($p=0.542$) showed no statistically significant change; thus, we retain the null hypothesis for these measures in the normal-weight group.

The obese cohort (Group 2, n=6) demonstrates a broader and more pronounced autonomic correction. Paired samples t-tests confirm highly significant reductions in both Systolic Blood Pressure (Student's $t=5.078$, $p=0.004$) and Diastolic Blood Pressure (Student's $t=4.392$, $p=0.007$). Furthermore, this group achieved a drastic improvement in Breath-Holding Time (Student's $t=-7.311$, $p<0.001$). We emphatically reject the null hypothesis for SBP, DBP, and BHT in the obese cohort. The reduction in Respiratory Rate approached significance (Student's $t=2.335$, $p=0.067$), while Pulse Rate remained unchanged ($p=0.594$).

3.2 Inter-Group Comparative Analysis Independent samples t-tests comparing the post-intervention parameters between the normal-weight and obese cohorts reveal a crucial standardising effect of the practice. Post-intervention measurements for Pulse Rate ($p=0.958$), Respiratory Rate ($p=0.394$), Systolic Blood Pressure ($p=0.118$), Diastolic Blood Pressure ($p=0.117$), and SpO2 ($p=0.900$) show no statistically significant differences between the two disparate groups. The sole exception is post-intervention Breath-Holding Time, which exhibits a statistically significant inter-group difference (Welch's $t=-2.311$, $p=0.048$).

4. Discussion

The data from this pilot investigation unequivocally validate the acute physiological efficacy of a 15-

minute sequential pranayama protocol in modulating cardiovascular and autonomic parameters. This reveals that targeted yogic breathing functions as a rapid, potent, and non-pharmacological mechanism to override sympathetic dominance, particularly in individuals with obesity-induced dysautonomia.

In the normal-weight cohort, the significant immediate reductions in Respiratory Rate and Systolic Blood Pressure align precisely with the anticipated physiological cascade of slow-paced breathing. The controlled expansion of tidal volume activates the Hering-Breuer reflex, which suppresses medullary sympathetic outflow and enhances vagal efferent discharge [10]. This central integration directly reduces myocardial work and systemic vascular resistance, manifesting as the recorded drop in systolic pressure. Because healthy individuals already possess functional autonomic flexibility, their diastolic pressure and breath-holding times require less immediate correction, explaining the retention of the null hypothesis for those specific variables.

Conversely, the obese cohort exhibited profound, widespread haemodynamic corrections. Obesity inherently drives a state of chronic sympathetic overdrive, elevated systemic vascular resistance, and restricted thoracic excursion [2]. The significant post-intervention reductions in *both* systolic and diastolic blood pressures prove that the sequence of Nadi Shuddhi, Bhramari, and controlled yogic breathing directly dismantles this pathological sympathetic tone. Bhramari pranayama, specifically, utilises laryngeal vibration to directly stimulate the auricular and pharyngeal branches of the vagus nerve, forcing a systemic parasympathetic override that rapidly lowers diastolic pressure by inducing peripheral vasodilation [3].

Furthermore, the highly significant increase in Breath-Holding Time among the obese participants underscores the intervention's mechanical and chemoreceptor efficacy. Obesity mechanically restricts diaphragmatic excursion; the preparatory thoracic mobilisations and deep yogic breathing overcome this physical impedance, recruiting previously under-ventilated alveoli. This improves central carbon dioxide tolerance and drastically enhances respiratory efficiency within a single 15-minute session [4].

Crucially, the independent samples t-test results expose the most compelling clinical application of this protocol. Following the intervention, the obese and normal-weight groups exhibited no statistically significant differences in their post-intervention pulse rate, respiratory rate, blood pressures, or oxygen saturation. This establishes that a brief, targeted pranayama session effectively normalises the acute haemodynamic profile of an obese individual, temporarily aligning their autonomic outputs with those of a healthy, normal-weight counterpart. The isolated inter-group difference remaining in Breath-Holding Time reflects the baseline anatomical limitations of adiposity on total lung capacity, which requires longitudinal, rather than acute, intervention to fully resolve [11].

These findings dictate a forward-thinking shift in naturopathic and allopathic cardiovascular risk management. We definitively reject the premise that yogic interventions require long-term practice to yield measurable cardiovascular outcomes. Acute, sequential pranayama delivers immediate, quantifiable corrections to obesity-induced dysautonomia. Integrating these brief, evidence-based breathing protocols into primary care and acute clinical settings provides an instantaneous, cost-effective tool to interrupt the pathogenesis of cardiovascular morbidity at the autonomic level.

Conclusion

A single, structured 15-minute sequence of targeted pranayama definitively rectifies acute autonomic imbalance and suppresses sympathetic overactivity, particularly in individuals presenting with obesity-induced dysautonomia. The intervention drives immediate, statistically significant reductions in systolic and diastolic blood pressures, whilst simultaneously expanding respiratory capacity. Crucially, this brief yogic breathing protocol standardises the acute haemodynamic profile, instantly aligning the cardiovascular outputs of the obese cohort with those of their normal-weight counterparts. These findings validate sequential pranayama as a potent, non-pharmacological, and immediate physiological modulator. The data mandate a forward-thinking integration of naturopathic respiratory interventions into allopathic and preventive clinical settings. Clinicians must utilise these brief, evidence-based breathing protocols as primary, readily accessible tools to rapidly interrupt the pathogenesis of cardiovascular morbidity and enforce acute autonomic correction in vulnerable populations.

Appendices:

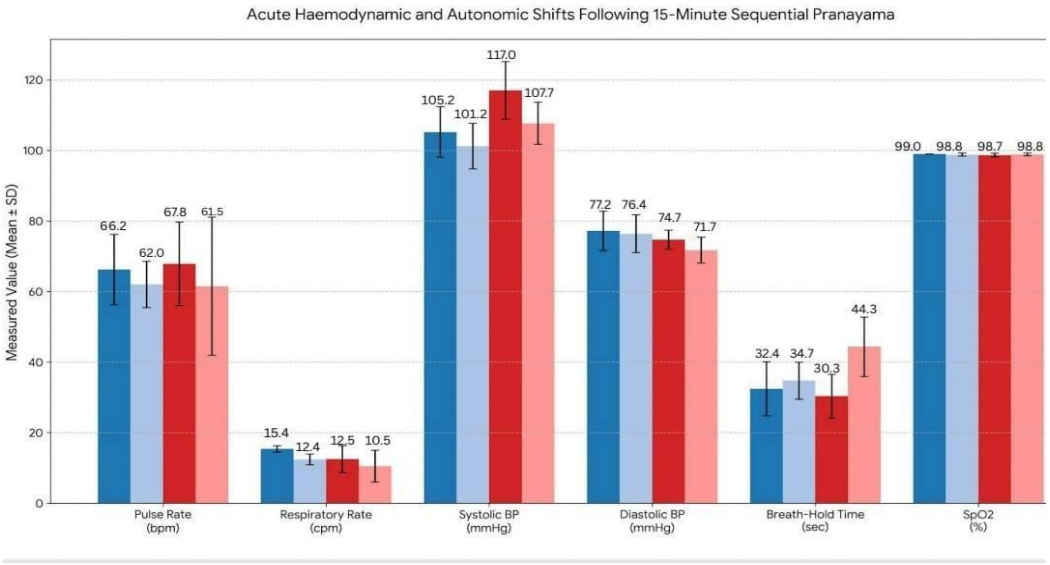

Table –2

Result Table

Variables	Group 1 (Normal-Weight) Pre (Mean ± SD)	Group 1 Post (Mean ± SD)	Group 1 p-value	Group 2 (Obese) Pre (Mean ± SD)	Group 2 Post (Mean ± SD)	Group 2 p-value	Between Group p-value (Post)
PR (bpm)	66.20 ± 10.43	62.00 ± 6.44	0.578	67.83 ± 11.23	61.50 ± 18.25	0.594	0.958
RR (cpm)	15.40 ± 0.89	12.40 ± 1.52	0.023*	12.50 ± 3.89	10.50 ± 4.51	0.067	0.394
SBP (mmHg)	105.20 ± 7.01	101.20 ± 6.46	0.011*	117.00 ± 8.00	107.67 ± 6.09	0.004*	0.118
DBP (mmHg)	77.20 ± 5.67	76.40 ± 6.15	0.587	74.67 ± 2.50	71.67 ± 3.88	0.007*	0.117
BHT (sec)	32.42 ± 7.67	34.72 ± 5.21	0.542	30.33 ± 6.15	44.33 ± 8.52	<.001*	0.048*
SpO2 (%)	99.00 ± 0.00	98.80 ± 0.45	N/A	98.67 ± 0.52	98.83 ± 0.41	N/A	0.900

* Indicates statistical significance ($p < 0.05$).

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