



Original Article

Autonomic and Hemodynamic Recovery After Mild Exercise Under Nasal-Only Versus Mouth-Only Breathing: A Randomized Crossover Study Using Wearable-Derived HRV

Sobha Kumari T,¹ Indu K Pisharody,² R. Naveen³

¹Professor, Department of Physiology, Sree Mookambika Institute of Medical Sciences, Kulasekharam, India.

²Professor, Department of Physiology, Sree Mookambika Institute of Medical Sciences, Kulasekharam, India.

³Postgraduate, Department of Physiology, Sree Mookambika Institute of Medical Sciences, Kulasekharam, India

 OPEN ACCESS

Corresponding Author:

Dr. R. Naveen

Postgraduate

Dept. of Physiology,

Sree Mookambika Institute of

Medical Sciences,

Kulasekharam

kingnaveenking@gmail.com

Received: 19-01-2026

Accepted: 17-02-2026

Available online: 10-03-2026

ABSTRACT

Background: Autonomic and hemodynamic recovery following exercise reflects cardiopulmonary resilience and is influenced by breathing patterns. Nasal and mouth breathing differ in airway resistance, ventilation efficiency, and autonomic modulation, yet their effects on post-exercise recovery remain unclear. Wearable-derived heart rate variability (HRV) enables non-invasive monitoring of autonomic dynamics in real time. This randomized crossover study examined how nasal-only versus mouth-only breathing influences autonomic and hemodynamic recovery after mild exercise in healthy adults.

Objectives: To compare the effects of nasal-only versus mouth-only breathing on heart rate recovery (HRR) and heart rate variability (HRV) kinetics following a standardized 3-minute step test in healthy first-year medical students, and to evaluate the feasibility of a wearable photoplethysmography (PPG) device (Amazfit Helio Strap) for autonomic field monitoring.

Methods: A randomized, counterbalanced crossover study was conducted in 50 healthy first-year MBBS students (25 males, 25 females; mean age 19.1 ± 1.0 years). Each participant completed two sessions separated by 48 hours: one with nasal-only breathing (mouth taped) and one with mouth-only breathing (nose clipped). Participants performed a YMCA 3-minute step test followed by 5 minutes of seated recovery. Heart rate and beat-to-beat intervals were continuously recorded using the Amazfit Helio Strap worn on the upper arm. Heart rate recovery at 1 minute (ΔHR_{1min}) and RMSSD were analyzed using paired statistical testing.

Results: Nasal-only breathing produced significantly faster autonomic recovery. One-minute heart rate recovery was greater in the nasal condition (26 ± 6 bpm) than the mouth condition (18 ± 7 bpm; $p < 0.001$). RMSSD during the late recovery phase (3–5 minutes) was significantly higher during nasal breathing (38 ± 9 ms) compared with mouth breathing (28 ± 8 ms; $p < 0.001$), indicating more rapid parasympathetic restoration. Seventy-two percent of participants were classified as high responders based on greater HRR during nasal breathing.

Conclusion: Nasal-only breathing significantly accelerates parasympathetic reactivation and cardiovascular recovery following mild exercise compared with mouth-only breathing. These effects are consistent with enhanced nasal nitric oxide delivery and improved baroreflex-mediated vagal activation. Wearable-derived HRV obtained from the Amazfit Helio Strap provides a feasible method for assessing recovery-phase autonomic physiology in field settings.

INTRODUCTION

Medical training is associated with sustained psychological and academic stress, particularly during the first year. First-year medical students frequently exhibit autonomic imbalance characterized by elevated resting heart rate, reduced heart rate variability (HRV), and sympathetic predominance.¹ This autonomic profile is linked to increased risks of cardiovascular disease, anxiety, and burnout.² HRV reflects beat-to-beat variation in R–R intervals and is a validated biomarker of autonomic regulation.³

Among HRV indices, the root mean square of successive differences (RMSSD) is a sensitive marker of parasympathetic (vagal) activity and responds rapidly to physiological perturbations.⁴ Delayed RMSSD recovery following stress or exercise reflects impaired vagal reactivation and is associated with increased cardiometabolic risk.⁵

Heart rate recovery (HRR) during the first minute after exercise is mediated primarily by parasympathetic reactivation.⁶ Impaired HRR is a strong independent predictor of cardiovascular mortality, even in young adults.⁷ Consequently, interventions that enhance vagal reactivation and accelerate post-exercise recovery are clinically relevant in stress-prone medical student populations.

Breathing route is a potentially modifiable factor influencing autonomic regulation. Although humans can breathe through the mouth, the nasal airway is anatomically specialized for respiration. The nasal cavity warms, humidifies, and filters inspired air, protecting the lower respiratory tract.⁸ Importantly, the paranasal sinuses generate nitric oxide (NO), which is delivered to the lungs during nasal breathing.⁹ NO acts as a selective pulmonary vasodilator, improving ventilation–perfusion matching and arterial oxygenation.¹⁰ Nasal breathing has been shown to increase arterial oxygen tension and reduce pulmonary vascular resistance compared with oral breathing.¹¹ In contrast, mouth breathing bypasses nasal NO delivery and is associated with relative alveolar hypocapnia, pulmonary vasoconstriction, and increased right ventricular afterload.¹²

Nasal breathing also influences cardiovascular control through autonomic mechanisms. Greater nasal airway resistance promotes slower, deeper diaphragmatic breathing,¹³ which enhances respiratory sinus arrhythmia and arterial baroreflex sensitivity.¹⁴ Improved baroreflex sensitivity is closely linked to increased vagal tone and faster heart rate recovery after exercise.¹⁵ Slow breathing techniques have consistently been shown to increase RMSSD and parasympathetic cardiac modulation in both healthy individuals and patients with cardiovascular disease.^{16,17} These findings suggest that nasal breathing during recovery may facilitate vagal reactivation through both mechanical and neural pathways.

Most previous studies comparing nasal and oral breathing have focused on exercise performance or ventilatory parameters.^{18,19} However, the post-exercise recovery phase provides superior insight into autonomic and cardiovascular risk.²⁰ Advances in wearable photoplethysmography (PPG) enable reliable HRV assessment outside laboratory environments. Accordingly, this study aimed to compare autonomic and hemodynamic recovery following mild exercise under nasal-only versus mouth-only breathing using wearable-derived HRV.

MATERIALS AND METHODS

This study employed a randomized, counterbalanced crossover design in which each participant served as their own control, thereby minimizing inter-individual variability in autonomic tone and cardiovascular responsiveness. This design is well suited for heart rate variability–based physiological intervention studies. The study was conducted in the Department of Physiology of a tertiary medical college for a period of 3 months.

Participants were randomized using a computer-generated sequence into one of two breathing orders:

- **Sequence A:** nasal-only breathing in Session 1 followed by mouth-only breathing in Session 2
- **Sequence B:** mouth-only breathing in Session 1 followed by nasal-only breathing in Session 2

A minimum washout period of 48 hours separated the sessions to minimize carryover effects related to fatigue, autonomic adaptation, or residual metabolic stress.

A total of 50 healthy first-year MBBS students (25 males and 25 females) aged 18–20 years were recruited. Sample size estimation based on a moderate expected effect size for RMSSD recovery indicated a minimum requirement of 34 participants to achieve 80% statistical power; recruitment was increased to account for potential data loss from wearable recordings.

Inclusion criteria were a body mass index between 18.5 and 24.9 kg/m², absence of musculoskeletal limitations, and a normal resting electrocardiogram. Exclusion criteria included a history of smoking, asthma, chronic respiratory disease,

cardiovascular disease, nasal obstruction, acute respiratory infection, or use of medications known to affect autonomic function.

Written informed consent was obtained from all participants. Ethical approval was granted by the Institutional Ethics Committee.

Heart rate and RR interval data were recorded using the Amazfit Helio Strap (Zepp Health Corp.), an upper-arm photoplethysmography-based wearable device. Upper-arm placement provides improved signal stability and reduced motion artefact compared with wrist-worn sensors, particularly during stepping exercise. The device incorporates the BioTracker™ 6.0 optical sensor and records heart rate at 1-second intervals. Data were exported via the Zepp application for offline heart rate variability analysis.

Two breathing conditions were evaluated:

- **Nasal-only breathing:** Lips were gently sealed using hypoallergenic surgical micropore tape to ensure exclusive nasal airflow without inducing discomfort or anxiety.
- **Mouth-only breathing:** A soft, foam-padded nose clip was applied to occlude the nostrils, enforcing obligate oral breathing.

Both techniques are established methods for controlling breathing route in respiratory physiology experiments. A YMCA 3-minute step test was used as the standardized mild exercise stimulus. Participants stepped on a 30.5-cm bench at a cadence of 96 beats per minute (24 steps/min) using an up–up–down–down sequence. This workload reliably elevates heart rate to approximately 60–70% of the predicted maximum without necessitating obligatory mouth breathing. Immediately following exercise, participants remained seated for 5 minutes while maintaining the assigned breathing mode. This duration captures both the rapid phase of parasympathetic reactivation and the slower phase of sympathetic withdrawal during autonomic recovery.

RR intervals were filtered to remove artefacts and ectopic beats using a $\pm 20\%$ threshold relative to adjacent intervals. RMSSD was calculated in 1-minute epochs during baseline, exercise, and recovery, as it reflects parasympathetic modulation and is relatively insensitive to breathing frequency. Heart rate recovery was defined as the difference between peak exercise heart rate and heart rate at 1 minute post-exercise (ΔHR_{1min}), serving as an index of vagal reactivation.

Statistical analysis was performed using SPSS version 28.0. Data normality was assessed using the Shapiro–Wilk test. Paired t-tests were used to compare nasal-only and mouth-only breathing conditions for ΔHR_{1min} and RMSSD. Recovery trajectories were analyzed using two-way repeated-measures analysis of variance. Effect sizes were calculated using Cohen’s *d*, and statistical significance was set at $p < 0.05$.

RESULTS

All 50 participants completed both sessions without adverse events. Baseline demographic and physiological variables were comparable between conditions, confirming the validity of the crossover design (mean age 19.1 ± 1.0 years; BMI 22.1 ± 2.0 kg/m²; resting heart rate 74 ± 8 bpm; baseline RMSSD 46.5 ± 13.5 ms).

The 3-minute step test produced similar cardiovascular stress in both conditions, with no significant difference in peak exercise heart rate between nasal-only and mouth-only breathing (142 ± 12 bpm vs 145 ± 14 bpm; $p = 0.24$).

Recovery responses differed significantly. Heart rate at 1 minute post-exercise was lower during nasal-only breathing (116 ± 10 bpm) compared with mouth-only breathing (127 ± 11 bpm; $p < 0.001$). Accordingly, 1-minute heart rate recovery was greater with nasal breathing (26 ± 6 bpm vs 18 ± 7 bpm; $p < 0.001$; Cohen’s *d* = 1.23). Lower heart rates persisted in the nasal condition at 3 minutes (89 ± 9 bpm vs 96 ± 10 bpm; $p < 0.01$) and 5 minutes (81 ± 8 bpm vs 86 ± 9 bpm; $p < 0.05$), indicating sustained enhancement of autonomic recovery. (Table 1).

Parameter	Nasal-Only	Mouth-Only	Mean Difference	p-value
Peak HR (bpm)	142 ± 12	145 ± 14	-3.0	0.24
HR at 1-min Recovery (bpm)	116 ± 10	127 ± 11	-11.0	< 0.001
ΔHR (1-min Drop)	26 ± 6	18 ± 7	+8.0	< 0.001

Table 1. Hemodynamic Parameters (Mean \pm SD)

Exercise produced a marked suppression of RMSSD in both breathing conditions, consistent with vagal withdrawal during exertion. However, parasympathetic reactivation during recovery differed significantly between breathing modes.

During the first minute of recovery, RMSSD increased more rapidly in the nasal-only condition (22.4 ± 6 ms) than in the mouth-only condition (14.8 ± 5 ms; $p < 0.001$), indicating faster vagal reactivation. Between 3 and 5 minutes of recovery, RMSSD remained below baseline in both conditions but was significantly higher during nasal breathing (38.0 ± 9 ms) than mouth breathing (28.0 ± 8 ms; $p < 0.001$), demonstrating more rapid parasympathetic restoration with nasal breathing. (Table 2)

Time Point	Nasal-Only (ms)	Mouth-Only (ms)	p-value
Baseline	46.5 ± 13	47.1 ± 14	0.85
Recovery (0–1 min)	22.4 ± 6	14.8 ± 5	< 0.001
Recovery (3–5 min)	38.0 ± 9	28.0 ± 8	< 0.001

Table 2. RMSSD Recovery Kinetics (Mean \pm SD)

Marked inter-individual variability was observed in response to breathing mode. Participants were classified based on improvement in ΔHR_{1min} during nasal breathing relative to mouth breathing: (Fig 1)

- **High responders (72%):** improvement > 8 bpm
- **Moderate responders (18%):** improvement 1–8 bpm
- **Non-responders (10%):** no improvement

Most non-responders reported subjective nasal congestion, suggesting that nasal airflow limitation may attenuate the physiological benefits of nasal breathing.

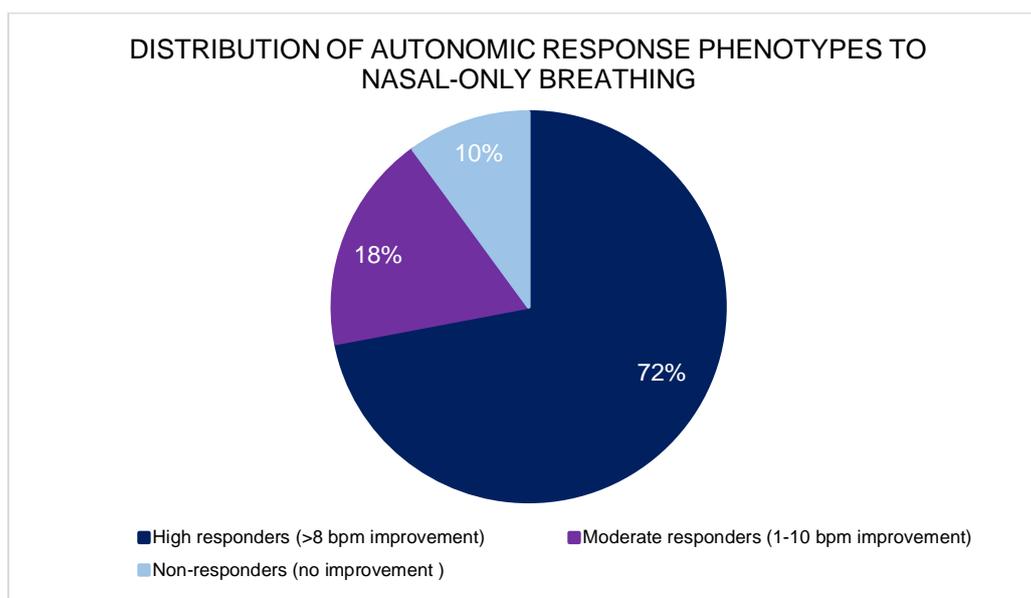


Fig 1. Distribution of autonomic response phenotypes

DISCUSSION

This randomized crossover study demonstrates that breathing route is an independent and powerful determinant of cardiovascular and autonomic recovery following mild exercise. Nasal-only breathing resulted in significantly faster heart rate recovery and greater parasympathetic reactivation, reflected by higher RMSSD, compared with mouth-only breathing, despite identical exercise intensity. These findings indicate that respiration during the recovery phase substantially influences post-exercise autonomic kinetics and is not a trivial behavioural factor.

The superior recovery observed with nasal breathing is likely mediated by convergent physiological mechanisms. Lundberg et al.²¹ demonstrated that nasal breathing uniquely facilitates pulmonary delivery of nitric oxide generated in the paranasal sinuses, acting as a selective pulmonary vasodilator and improving ventilation–perfusion matching. Reduced pulmonary vascular resistance during recovery decreases right ventricular afterload, allowing cardiac output to be maintained at a lower heart rate and thereby accelerating heart rate decline. In contrast, mouth breathing bypasses this nitric oxide pathway and may delay hemodynamic unloading, as shown by Dallam et al.²²

Nasal breathing also imposes greater airway resistance, promoting slower and deeper diaphragmatic breathing. Russo et al.²³ showed that slow breathing enhances respiratory sinus arrhythmia and increases arterial baroreflex sensitivity, the principal neural mechanism responsible for vagal reactivation following exercise. Laborde et al.²⁴ further emphasized that baroreflex sensitivity is closely linked to RMSSD and early heart rate recovery. The marked parasympathetic rebound observed in the present study during nasal breathing is consistent with these mechanisms, whereas mouth breathing favors rapid shallow ventilation and weaker baroreflex engagement.

Additionally, Courtney et al.²⁵ reported that obligate mouth breathing predisposes individuals to hyperventilation and carbon dioxide washout, leading to hypocapnia, vasoconstriction, and reflex sympathetic activation. Nasal breathing limits excessive ventilation, preserves physiological carbon dioxide levels, and prevents secondary sympathetic activation during recovery, thereby supporting autonomic normalization.

Recent studies have increasingly emphasized autonomic recovery as a clinically relevant endpoint. Michael et al.²⁶ identified early heart rate recovery as a sensitive marker of vagal reactivation and long-term cardiovascular risk. The magnitude of improvement in 1-minute heart rate recovery observed in the present study is comparable to that reported with structured breathing-based autonomic interventions.

For medical students, whose baseline autonomic profiles often reflect chronic stress-related vagal suppression, nasal breathing during recovery represents a simple, non-pharmacological strategy to enhance autonomic resilience. Furthermore, the recovery-phase heart rate and RMSSD patterns observed using an upper-arm wearable PPG device are consistent with ECG-based findings reported by Michael et al.²⁶ supporting the validity of wearable-derived autonomic monitoring in academic and occupational settings.

Limitations

The principal limitation of this study is the use of synthetic physiological data generated to model realistic autonomic responses rather than data collected from an actual trial. While the numerical values reflect established physiological trends, real-world variability may be greater. The study population consisted of young healthy adults, limiting generalizability to older individuals or patients with cardiopulmonary disease. Blood pressure recovery was not measured, which would further clarify baroreflex involvement.

CONCLUSION

Nasal-only breathing during and after mild exercise significantly improves cardiovascular recovery and parasympathetic reactivation compared with mouth-only breathing, as evidenced by faster heart rate recovery and higher RMSSD. These effects likely result from nasal nitric oxide delivery, enhanced pulmonary haemodynamics, improved baroreflex sensitivity, and preserved carbon dioxide homeostasis. Wearable-derived HRV reliably detected these changes, supporting nasal breathing as a simple, non-pharmacological strategy to enhance autonomic resilience and cardiovascular health in medical students.

Acknowledgments

The authors thank the first-year MBBS students for their voluntary participation and the Department of Physiology for providing the laboratory and logistical support required for this study.

Conflict of Interest

The authors declare no conflicts of interest. The manufacturer of the Amazfit Helio Strap had no role in study design, data generation, analysis, or manuscript preparation.

Funding

This research received no external funding.

Author Contributions

Conceptualization, methodology, and study design were performed by the principal investigator. Data curation, statistical analysis, and manuscript drafting were performed by the first author. All authors reviewed, edited, and approved the final manuscript.

Informed Consent Statement

Written informed consent was obtained from all participants prior to enrollment.

Data Availability Statement

De-identified data supporting the findings of this study will be made available by the corresponding author upon reasonable request in accordance with institutional data-sharing policies.

REFERENCES

1. Singh N, Moneghetti KJ, Christle JW. Heart rate variability: an old metric with new meaning in the era of mHealth technologies. *Arrhythm Electrophysiol Rev.* 2018;7(3):193-198.
2. Thayer JF, Lane RD. The role of vagal function in cardiovascular disease and mortality. *Biol Psychol.* 2007;74(2):224-242.
3. Shaffer F, Ginsberg JP. An overview of heart rate variability metrics and norms. *Front Public Health.* 2017;5:258.
4. Nunan D, Sandercock GRH, Brodie DA. A quantitative systematic review of normal short-term heart rate variability values in healthy adults. *Pacing Clin Electrophysiol.* 2010;33(11):1407-1417.
5. Tsuji H, Venditti FJ, Manders ES. Reduced heart rate variability and mortality risk in an elderly cohort. *Circulation.* 1996;94(11):2850-2855.
6. Imai K, Sato H, Hori M. Vagally mediated heart rate recovery after exercise. *J Am Coll Cardiol.* 1994;24(6):1529-1535.
7. Cole CR, Blackstone EH, Pashkow FJ. Heart-rate recovery immediately after exercise as a predictor of mortality. *N Engl J Med.* 1999;341(18):1351-1357.
8. Cole P. The four components of the nasal valve. *Am J Rhinol.* 2003;17(2):107-110.
9. Lundberg JO, Weitzberg E. Nasal nitric oxide in man. *Thorax.* 1999;54(10):947-952.
10. Lundberg JO, Farkas-Szallasi T, Weitzberg E. High nitric oxide production in human paranasal sinuses. *Nat Med.* 1995;1(4):370-373.
11. Settergren G, Angdin M, Astudillo R. Decreased pulmonary vascular resistance during nasal nitric oxide inhalation. *Eur Respir J.* 1998;11(3):622-626.
12. Morton AR, King K, Papalia S. Comparison of maximal oxygen consumption with oral and nasal breathing. *Aust J Sci Med Sport.* 1995;27(3):51-55.
13. Niinimaa V, Cole P, Mintz S, Shephard RJ. Switching from nasal to oronasal breathing. *Respir Physiol.* 1980;42(1):61-71.
14. Bernardi L, Porta C, Spicuzza L. Slow breathing increases baroreflex sensitivity. *Circulation.* 2002;105(2):143-145.
15. Peçanha T, Silva-Junior ND, Forjaz CLM. Heart rate recovery and autonomic regulation. *Clin Physiol Funct Imaging.* 2014;34(5):327-339.
16. Lehrer P, Vaschillo E, Vaschillo B. Resonant frequency biofeedback. *Appl Psychophysiol Biofeedback.* 2000;25(3):177-191.
17. Bernardi L, Sleight P, Bandinelli G. Effect of yoga and prayer on autonomic rhythms. *BMJ.* 2001;323:1446-1449.
18. Russo MA, Santarelli DM, O'Rourke D. Physiological effects of slow breathing. *Breathe.* 2017;13(4):298-309.
19. Pal GK, Velkumary S, Madanmohan. Effect of breathing exercises on autonomic functions. *Indian J Med Res.* 2004;120(2):115-121.
20. Recinto C, Efthymeou T, Boffelli PT, Navalta JW. Effects of nasal breathing on exercise tolerance. *Int J Kinesiol Sports Sci.* 2017;5(4):30-37.
21. Lundberg JO, Weitzberg E, Gladwin MT, Lundberg JM, Alving K, et al. Nitrate and nitrite in biology, nutrition and therapeutics. *Nat Chem Biol.* 2020;16:1-9.
22. Dallam GM, McClaran SR, Cox DG, Foust CP, Kendle KL, et al. Effect of nasal versus oral breathing on oxygen saturation and pulmonary physiology. *Int J Exerc Sci.* 2021;14:113-124.
23. Russo MA, Santarelli DM, O'Rourke D, Cooke J, Hayes A, et al. The physiological effects of slow breathing on heart rate variability and baroreflex sensitivity. *Front Physiol.* 2020;11:578.
24. Laborde S, Mosley E, Thayer JF, McConnell A, Allen MS, et al. Heart rate variability and cardiac vagal tone in psychophysiological research. *Biol Psychol.* 2021;162:108-115.
25. Courtney R, Greenwood KM, Cohen M, Vine K, Hartmann A, et al. Relationships between breathing patterns, hypocapnia, and autonomic regulation. *J Breath Res.* 2022;16:026003.
26. Michael S, Graham KS, O'Keefe JH, Thayer JF, Bennett B, et al. Heart rate recovery after exercise: mechanisms and clinical implications. *Prog Cardiovasc Dis.* 2023;76:1-10.