

Deep Slow Breathing and Cardiovascular Changes After Moderate Intensity Training in Runners

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Abstract

Background: Blood pressure and heart rate change after moderate intensity exercise, and controlled breathing may influence these cardiovascular responses. Evidence in recreational runners remains limited.

Objective: To compare pre and post intervention changes in systolic blood pressure, diastolic blood pressure, and heart rate between runners receiving deep slow breathing and normal breathing.

Methods: This nonrandomized quasi experimental two group pretest posttest study included 20 recreational runners, with 10 participants in each group. The reported deep slow breathing protocol used a 4 4 8 pattern for 5 minutes and was delivered across 8 sessions over 4 weeks. Within group changes were analyzed using paired tests and between group differences in change scores using independent tests, with nonparametric alternatives when assumptions were not met.

Results: Systolic blood pressure decreased by 6.10 ± 3.98 mmHg in the intervention group and 1.70 ± 2.94 mmHg in controls, with a between group mean difference of -4.00 mmHg (95% CI, -7.69 to -1.11 ; $p=0.012$). Heart rate decreased by 9.30 ± 7.13 and 3.10 ± 4.31 beats/min, respectively, with a mean difference of -6.20 beats/min (95% CI, -11.74 to -0.66 ; $p=0.030$). The between group difference in diastolic blood pressure was not statistically significant (-2.40 mmHg; 95% CI, -5.62 to 0.82 ; $p=0.135$).

Conclusion: Deep slow breathing was associated with greater reductions in systolic blood pressure and heart rate than normal breathing. Because allocation was nonrandomized and the source documents contain unresolved inconsistency regarding the exact measurement timeline, the findings should not be interpreted as definitive causal evidence or as a quantified measure of recovery kinetics.

Keywords

Breathing Exercises; Blood Pressure; Heart Rate; Exercise; Runners

Introduction

Running is a widely accessible form of physical activity and is commonly undertaken in community settings. Cardiovascular responses to exercise and the period immediately surrounding exercise are relevant to exercise monitoring because heart rate and blood pressure are influenced by exercise intensity, training status, age, sex, previous exercise exposure, environmental conditions, and the timing of measurement.¹⁻⁴ The practical appeal of strategies that may support cardiovascular regulation has therefore stimulated interest in simple interventions that can be applied without specialized equipment.

Deep slow breathing is a controlled breathing approach characterized by deliberate regulation of the respiratory cycle and, in many protocols, a relatively prolonged expiratory phase. Slow paced breathing has been associated with changes in cardiovascular and autonomic measures in healthy populations, although the magnitude and mechanism of these responses vary across protocols and study settings.⁵⁻⁷ Physiological explanations involving respiratory cardiac coupling, vagal modulation, heart rate variability, and baroreflex mechanisms are biologically plausible, but these mechanisms should not be treated as direct findings unless they are measured in the study itself.

Previous work has examined brief breathing interventions and protocols involving approximately six breaths per minute or the 4 4 8 pattern. The available evidence, however, differs substantially in participant characteristics, exercise exposure, breathing dose, comparator conditions, and outcome timing.^{4,6,7} Findings from healthy adults who did not complete a structured exercise stimulus cannot automatically be generalized to recreational runners after moderate intensity training. In addition, a study based on only two measurements cannot establish the time course of cardiovascular recovery or quantify a recovery slope.

The present study addressed this practical context by comparing cardiovascular changes in recreational runners assigned to guided deep slow breathing or normal breathing after participation in moderate intensity training. The intervention was reported as a 4 4 8 breathing pattern performed for 5 minutes across 8 sessions over 4 weeks. The source documents also describe a session sequence involving moderate intensity training, a 5 minute rest period, pretest measurement, breathing intervention or normal breathing, and posttest measurement. Because these descriptions conflict with other statements indicating program level pretest and posttest assessment, the exact relationship between the 8 session program and the analyzed measurements cannot be resolved from the available documents. The present finalization therefore preserves the reported data, avoids claiming a quantified recovery rate, and interprets the principal outcome as the observed between group difference in pre and post change under the reported study conditions.

The study objective was to determine whether changes in systolic blood pressure, diastolic blood pressure, and heart rate differed between the deep slow breathing and normal breathing groups. Given the nonrandomized allocation and absence of a reported adjusted analysis, causal interpretation was deliberately limited.

Methods

This quantitative study used a nonrandomized quasi experimental two group pretest posttest design. The intervention group received guided deep slow breathing, whereas the control group performed normal breathing. The study was conducted in the Fakerunners Bandung community from April to May 2026. The intervention program was reported to include 8 sessions over 4 weeks.

The source population comprised approximately 150 to 200 active community runners, with average attendance of approximately 40 runners at a community training session. Participants were recruited through nonprobability purposive sampling. Eligibility criteria were active membership in Fakerunners Bandung, age 20 to 55 years, regular running experience for more than 6 months, participation in moderate intensity running training, physical ability to participate, willingness to provide written informed consent, and ability to follow study instructions. Exclusion criteria were injury or illness during the study, a history of cardiovascular disease or severe respiratory disease, failure to complete study procedures, or withdrawal of consent. Twenty participants were included, with 10 participants in the deep slow breathing group and 10 in the normal breathing group. The exact mechanism by which eligible participants were assigned to the two groups, the person responsible for allocation, and any balancing procedure were not documented in the available source material. Accordingly, selection bias and confounding cannot be considered fully controlled.

No a priori sample size calculation was reported in the source manuscript. The study therefore cannot be presented as having established statistical power for a prespecified primary outcome. The small sample is retained as an important limitation.

The deep slow breathing protocol used a 4 4 8 pattern consisting of 4 seconds of inspiration, 4 seconds of breath holding, and 8 seconds of expiration. The intervention was reported as lasting 5 minutes per session for 8 sessions over 4 weeks. Sessions were performed while seated in a relaxed position in a quiet environment. The control group performed normal breathing. The available documents do not provide a detailed instruction script, confirm whether diaphragmatic breathing was required, document external monitoring of respiratory frequency, or describe an adherence assessment. The source manuscript also states that the 16 second cycle was approximately six breaths per minute, although the cycle duration corresponds mathematically to approximately 3.75 cycles per minute. No unsupported correction to the protocol has been made; the breathing pattern is therefore reported exactly as documented.

Participants completed the community moderate intensity training session with a reported target heart rate of 64% to 76% of maximum heart rate. Heart rate was monitored using a smartwatch connected to the Strava application. The formula used to estimate maximum heart rate, training duration, session structure, method used to confirm achievement of the target range, actual heart rate achieved, and adherence criteria were not reported in the available documents.

The outcomes were systolic blood pressure, diastolic blood pressure, and heart rate. Blood pressure was measured using a calibrated digital sphygmomanometer. Participants were seated with their feet on the floor and the arm positioned at heart level, and they were instructed not to speak during measurement. Heart rate was measured using fingertip pulse oximetry while participants remained seated and still, with the value recorded after the display became stable. Device model, number of readings, interval between readings, measurement arm, detailed calibration procedure, and the rule used to determine the final value were not reported. The original manuscript included reliability coefficients for the devices, but the underlying sources were not available for verification; those unsupported coefficients were therefore not retained as established evidence in this final version.

The reported procedural sequence was completion of moderate intensity training, a 5 minute rest period, pretest measurement, 5 minutes of deep slow breathing or normal breathing, and posttest measurement. Elsewhere, however, the source manuscript states that measurement occurred before the intervention and after completion of 8 sessions over 4 weeks. The available documents do not establish whether the analyzed values represented a single session, the final session, or a program level assessment. This unresolved factual inconsistency is preserved explicitly rather than resolved by assumption.

Data were analyzed using SPSS. Normality was assessed using the Shapiro Wilk test and homogeneity using Levene's test. Within group comparisons used the paired samples t test when parametric assumptions were met or the Wilcoxon signed rank test otherwise. Between group comparisons used the independent samples t test when assumptions were met or the Mann Whitney U test otherwise. Statistical significance was set at $p < 0.05$. The reported between group analysis used change scores. The available documents do not provide the actual assumption test results for each outcome or the rationale for selecting change score analysis instead of an adjusted baseline model. No ANCOVA or other adjusted analysis was added because individual level data were unavailable. Standardized effect sizes were also not calculated because the underlying raw data were not available.

All 20 participants completed the reported procedures, with no missing outcome data and no reported adverse events. The study was approved by the Health Research Ethics Committee of Universitas Binawan under Ethical Approval No. 093/KEPK-UBN/IV/2026. Written informed consent was obtained from participants.

Results

Twenty participants were included in the final analysis, with 10 participants in each group. All participants completed the reported study procedures, and no dropout or missing outcome data were reported. The source documents do not provide the number of individuals approached, screened, excluded, or the reasons for exclusion; therefore, a detailed participant flow diagram cannot be constructed without fabricating information.

Baseline characteristics are summarized in Table 1. The mean age was 36.80 ± 10.74 years in the deep slow breathing group and 34.50 ± 7.86 years in the control group. Sex distribution was identical across groups. The largest age category was 27 to 33 years, representing 45% of the total sample. The previously inconsistent entry for the control group in the 41 to 47 year category has been corrected from 0 (10%) to 0 (0%).

Table 1. Baseline characteristics of participants

Characteristic	DSB (n=10)	Control (n=10)	Total (n=20)
Age, years, mean $\pm$ SD	36.80 $\pm$ 10.74	34.50 $\pm$ 7.86	35.65 $\pm$ 9.24
20–26 years, n (%)	1 (10%)	1 (10%)	2 (10%)
27–33 years, n (%)	4 (40%)	5 (50%)	9 (45%)
34–40 years, n (%)	1 (10%)	3 (30%)	4 (20%)
41–47 years, n (%)	2 (20%)	0 (0%)	2 (10%)
48–54 years, n (%)	2 (20%)	1 (10%)	3 (15%)
Male, n (%)	5 (50%)	5 (50%)	10 (50%)
Female, n (%)	5 (50%)	5 (50%)	10 (50%)
Passive smoker, n (%)	10 (100%)	10 (100%)	20 (100%)
Active smoker, n (%)	0 (0%)	0 (0%)	0 (0%)

DSB, deep slow breathing. Running experience categories were reported in the source manuscript but are omitted from this table because the documented category counts in the available text exceed the group denominators and require verification against the primary dataset.

Within group cardiovascular changes are presented in Table 2. In the deep slow breathing group, systolic blood pressure changed from 113.90±9.35 to 107.80±8.36 mmHg, diastolic blood pressure from 74.80±6.68 to 69.20±4.10 mmHg, and heart rate from 88.20±9.34 to 78.90±10.63 beats/min. In the control group, the corresponding changes were smaller for systolic blood pressure and heart rate.

Table 2. Within group cardiovascular outcomes before and after the reported intervention assessment

Outcome	Group	Pretest	Posttest	Change; p value
Systolic BP, mmHg	DSB	113.90±9.35	107.80±8.36	-6.10±3.98; p=0.001
Systolic BP, mmHg	Control	113.70±8.69	112.00±9.13	-1.70±2.94; p=0.101
Diastolic BP, mmHg	DSB	74.80±6.68	69.20±4.10	-5.60±3.86; p=0.001
Diastolic BP, mmHg	Control	79.50±8.46	76.30±9.33	-3.20±2.94; p=0.005
Heart rate, beats/min	DSB	88.20±9.34	78.90±10.63	-9.30±7.13; p=0.003
Heart rate, beats/min	Control	93.80±7.73	90.70±9.64	-3.10±4.31; p=0.049

BP, blood pressure; DSB, deep slow breathing. The exact timing represented by the reported pretest and posttest values requires factual confirmation from the authors.

Between group comparisons of change scores are shown in Table 3. The between group difference favored deep slow breathing for systolic blood pressure and heart rate. The confidence intervals for these two outcomes did not cross zero. The difference for diastolic blood pressure was not statistically significant.

Table 3. Between group differences in change scores

Outcome	Mean difference in change	95% CI	p value
Systolic BP, mmHg	-4.00	-7.69 to -1.11	0.012
Diastolic BP, mmHg	-2.40	-5.62 to 0.82	0.135
Heart rate, beats/min	-6.20	-11.74 to -0.66	0.030

Negative values indicate a greater reduction in the deep slow breathing group relative to the control group. Standardized effect sizes were not available from the source data and were not calculated without individual level observations. No subgroup or sensitivity analyses were reported. No adverse events were reported during the intervention.

Discussion

The principal finding was that the deep slow breathing group showed greater reductions in systolic blood pressure and heart rate than the normal breathing group, whereas the between group difference in diastolic blood pressure was not statistically significant. These findings are consistent with the possibility that controlled slow breathing influences selected cardiovascular outcomes, but the present design does not establish a definitive causal effect.

The greater reduction in systolic blood pressure is broadly compatible with previous reports that slow or controlled breathing can be associated with lower blood pressure in healthy adults.^{4,6,7} Direct comparison should nevertheless be cautious because prior studies differ in participant populations, breathing protocols, intervention duration, exercise exposure, and measurement timing. The present study extends this literature to a community running context, but it cannot determine whether the observed difference reflects an acute response, a cumulative response across the reported program, or a combination of both because the measurement timeline remains inconsistent in the source documentation.

The absence of a statistically significant between group difference in diastolic blood pressure should also be interpreted in the context of the within group reductions observed in both groups. This pattern is compatible with natural post exercise variation and other time dependent changes that may occur independently of the breathing intervention. Diastolic pressure is influenced by peripheral vascular resistance and arterial properties, and brief interventions may not produce a consistent additional effect under all conditions.^{4,8} Because no minimal clinically important difference or prespecified clinical benchmark was reported, statistical significance should not be equated with clinical importance.

Heart rate decreased more in the deep slow breathing group than in the control group. Previous literature suggests that slow breathing may influence cardiovascular regulation through respiratory cardiac interactions and autonomic mechanisms.⁹ However, this study did not measure heart rate variability, baroreflex sensitivity, respiratory rate during the intervention, or another direct autonomic marker. Increased parasympathetic activity or enhanced baroreflex function must therefore remain explanatory hypotheses derived from external literature rather than mechanisms demonstrated by the present data.

The control group also showed reductions in diastolic blood pressure and heart rate. This observation is important because cardiovascular variables can change over time after exercise as metabolic demand declines.⁸ Without repeated measurements across a defined post exercise interval, the study cannot estimate a recovery curve, recovery slope, or standard heart rate recovery metric. The results should therefore be described as between group differences in observed pre and post change rather than evidence that deep slow breathing accelerated recovery.

Internal validity is limited by the nonrandomized allocation and incomplete documentation of the allocation process. Although age and sex distributions were similar, baseline diastolic blood pressure differed numerically between groups. Potential confounders also include running experience, actual exercise intensity, time between exercise cessation and measurement, environmental conditions, and measurement variability.² The reported analysis did not adjust for these factors, and individual level data were unavailable for an adjusted model. These limitations reduce confidence in causal attribution.

Several additional methodological limitations require emphasis. The sample was small, recruitment occurred in one running community, and no a priori sample size calculation was reported. The detailed protocol for moderate intensity training was incompletely documented, including the maximum heart rate formula, duration, structure, achieved intensity, and adherence criteria. Important measurement details, including device model, repeated reading procedures, intervals, arm selection, and rules for determining final values, were also unavailable. The same researcher performed outcome measurements, but assessor blinding was not reported. Furthermore, the source manuscript acknowledged variation in environmental conditions and measurement timing and occasional technical problems requiring repeated device readings.

The study nevertheless has practical strengths. It examined a simple breathing strategy in a real world community setting, included a comparator group, retained all 20 participants through final analysis, and used a clearly described 4 4 8 breathing pattern

with a reported 5 minute session duration. The protocol required minimal equipment in this study. However, feasibility should not be confused with proof of clinical effectiveness, and no conclusion regarding clinically important benefit can be made without an appropriate benchmark.

Future studies should use random allocation, a prespecified sample size calculation, explicit allocation concealment where feasible, standardized exercise procedures, documented actual exercise intensity, and fully prespecified measurement protocols. Repeated measurements at defined intervals after exercise would allow investigators to distinguish an immediate breathing response from cumulative adaptation and to calculate an appropriate recovery metric. Direct assessment of respiratory frequency, heart rate variability, or baroreflex related measures could also test proposed mechanisms. An adjusted analysis controlling for baseline values and prespecified confounders would further strengthen causal interpretation. Overall, the findings support further investigation of deep slow breathing in recreational runners, while the present results should remain interpreted within the limitations of the available data and reporting.

Conclusion

In this small nonrandomized quasi experimental study, participants assigned to the reported 4 4 8 deep slow breathing intervention showed greater reductions in systolic blood pressure and heart rate than participants who performed normal breathing. The between group difference in diastolic blood pressure was not statistically significant. These results indicate an association between the reported breathing intervention and more favorable pre and post changes in selected cardiovascular outcomes under the study conditions.

The findings should not be interpreted as definitive proof of causality. Nonrandom allocation, potential confounding, the small sample, lack of an adjusted baseline analysis, incomplete standardization of measurement conditions, and missing methodological detail limit internal validity. Most importantly, the available source documents contain unresolved inconsistency regarding whether the analyzed pretest and posttest values represented an acute session response, an assessment after the 8 session program, or another program level comparison. Consequently, the study cannot establish a quantified rate or time course of cardiovascular recovery, and the results should not be described as demonstrating accelerated heart rate recovery.

Deep slow breathing remains a feasible intervention for further evaluation in community running settings. Future adequately powered randomized studies should use a clearly defined intervention timeline, standardized exercise intensity, repeated post exercise measurements, validated measurement procedures, and direct physiological measures when mechanistic interpretation is intended. Such studies would clarify the magnitude, clinical relevance, and temporal characteristics of any cardiovascular effect.

Author Contribution

Valerina Matsy Tauran: Conceptualization; Methodology; Investigation; Writing, original draft; Writing, review and editing; Formal analysis; Data curation; Visualization; Project administration.

Touris Aan Suhadaq: Conceptualization; Methodology; Supervision; Writing, review and editing; Validation.

Lily Nabillah: Conceptualization; Methodology; Supervision; Writing, review and editing; Validation.

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Conflict of Interest Statement

The authors declare no conflict of interest.

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Ethics Statement

This study was approved by the Health Research Ethics Committee of Universitas Binawan under Ethical Approval No. 093/KEPK-UBN/IV/2026. Written informed consent was obtained from all participants before their enrollment in the study. The study was conducted in accordance with the ethical principles for research involving human participants.

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