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ORIGINAL RESEARCH

To evaluate the immediate effects of short-duration slow deep breathing on heart rate, blood pressure, respiratory rate, oxygen saturation, and heart rate variability (HRV) in healthy young adults

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ABSTRACT
Aim: The aim of this study was to evaluate the immediate effects of short-duration slow deep breathing on heart rate, blood pressure, respiratory rate, oxygen saturation, and heart rate variability (HRV) in healthy young adults.**Materials and Methods:** This prospective, randomized controlled study included 120 healthy young adults aged 20-25 years. Participants were divided into two groups: the control group (n = 60), who maintained normal breathing for 5 minutes, and the intervention group (n = 60), who performed slow deep breathing exercises for 5 minutes. Baseline and post-intervention measurements of heart rate (HR), blood pressure (BP), respiratory rate (RR), oxygen saturation (SpO2), and HRV were recorded. Statistical analysis was conducted using paired t-tests and independent t-tests.**Results:** The intervention group showed a significant reduction in heart rate (65 ± 7 bpm vs. 75 ± 8 bpm, p < 0.001), systolic BP (115 ± 9 mmHg vs. 120 ± 10 mmHg, p = 0.045), and diastolic BP (75 ± 7 mmHg vs. 80 ± 8 mmHg, p = 0.001). Respiratory rate also decreased significantly (12 ± 1 breaths/min vs. 16 ± 2 breaths/min, p < 0.001), and tidal volume increased (500 ± 40 mL vs. 450 ± 50 mL, p = 0.029). HRV parameters showed improvement, with higher SDNN (70 ± 12 ms vs. 50 ± 10 ms, p < 0.001) and RMSSD (50 ± 9 ms vs. 30 ± 8 ms, p < 0.001).**Conclusion:** Short-duration slow deep breathing significantly lowers heart rate and blood pressure in healthy young adults, promoting parasympathetic activation and enhancing cardiovascular function. This non-pharmacological technique is an effective and accessible tool for managing stress and improving autonomic regulation.

Keywords: Slow deep breathing, heart rate variability, blood pressure, parasympathetic activation, autonomic regulation.

INTRODUCTION

Slow deep breathing (SDB) has emerged as a simple yet highly effective technique for modulating the autonomic nervous system, influencing heart rate (HR), blood pressure (BP), and overall cardiovascular function. Although deep breathing exercises have been practiced for centuries as part of yoga and meditation, their scientific validation has gained prominence only in recent decades. The immediate physiological effects of slow deep breathing on heart rate and blood pressure are of particular interest in the context of promoting cardiovascular health, especially in healthy young adults who are often exposed to stress and other lifestyle factors that may negatively affect their cardiovascular system. This study aims to explore how a brief session of slow deep breathing can impact these two critical parameters: heart rate and blood pressure.[1]The autonomic nervous system (ANS) regulates vital physiological processes, including heart rate and blood pressure, through its two branches—the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). The SNS is responsible for the "fight or flight" response, which increases heart rate and blood pressure, while the PNS promotes a "rest and digest" state, which slows the heart rate and reduces blood pressure. Breathing, particularly slow and controlled breathing, has a direct effect on the balance between these two

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branches of the ANS. By influencing respiratory rate and depth, slow deep breathing enhances parasympathetic activity and reduces sympathetic output, leading to a decrease in heart rate and blood pressure.[2]Slow deep breathing works by engaging the baroreceptor reflex—a feedback mechanism in which stretch receptors in the blood vessels detect changes in blood pressure and trigger responses that stabilize cardiovascular function. When blood pressure rises, baroreceptors in the aorta and carotid arteries send signals to the brain, which then activates the parasympathetic nervous system to slow the heart rate and reduce blood pressure. Conversely, when blood pressure falls, the brain triggers sympathetic activity to raise blood pressure and heart rate. Slow deep breathing amplifies this baroreceptor reflex by increasing the variability of blood pressure and heart rate, resulting in a more responsive and adaptive cardiovascular system. This dynamic regulation of heart rate and blood pressure through breathing is termed respiratory sinus arrhythmia (RSA), a phenomenon in which the heart rate varies with the breathing cycle—speeding up during inhalation and slowing down during exhalation.[3]The practice of slow deep breathing has been shown to reduce heart rate significantly, even with short-term practice. This is especially relevant in young adults, who often exhibit higher baseline heart rates due to stress, anxiety, or physical exertion. By slowing down the rate of breathing to around 6 breaths per minute, parasympathetic tone is enhanced, leading to immediate reductions in heart rate. This decrease in heart rate is not only a sign of relaxation but also indicates an improved ability to cope with physiological stress. A lower resting heart rate is associated with better cardiovascular health, while a persistently high heart rate is a risk factor for heart disease and other cardiovascular conditions. Therefore, the ability of slow deep breathing to modulate heart rate in a short duration can have significant implications for both short-term relaxation and long-term cardiovascular health.[4]Blood pressure, another critical marker of cardiovascular function, is similarly influenced by slow deep breathing. Blood pressure consists of two values: systolic pressure (the pressure in the arteries when the heart contracts) and diastolic pressure (the pressure when the heart is at rest between beats). Both systolic and diastolic pressures are subject to fluctuations based on physical activity, stress, and emotional state. Slow deep breathing reduces systolic and diastolic blood pressure by promoting vasodilation (widening of blood vessels) and reducing vascular resistance. This is achieved through the modulation of autonomic activity, where the sympathetic "fight or flight" response is suppressed, and the parasympathetic "rest and digest" response is activated. This shift toward parasympathetic dominance causes blood vessels to relax, allowing blood to flow more easily and reducing the force exerted on the arterial walls.[5]In healthy young adults, the immediate effects of slow deep breathing on blood pressure are particularly significant. Young adults are often at a stage in life where stress levels are high due to academic pressures, career development, and social interactions. These stressors can lead to transient increases in blood pressure, which, if unchecked, may contribute to long-term cardiovascular issues. By incorporating short sessions of slow deep breathing, young adults can experience immediate reductions in blood pressure, which may help to buffer the effects of stress and reduce the risk of developing hypertension later in life. Furthermore, the practice of slow deep breathing can be a preventive measure, helping to maintain optimal blood pressure levels even in the absence of cardiovascular disease.[6]The simplicity of slow deep breathing makes it an accessible and low-cost intervention for promoting cardiovascular health. Unlike pharmacological treatments for high blood pressure or heart rate, slow deep breathing requires no equipment or medication and can be practiced anywhere, making it a practical tool for everyday stress management. Moreover, the immediate effects of slow deep breathing on heart rate and blood pressure provide tangible benefits that can be felt after just a few minutes of practice, encouraging individuals to adopt the practice regularly.

MATERIALS AND METHODS

This prospective study was conducted in GMC Jammu in 2018.A total of 120 healthy young adults aged 20 to 25 years were recruited from the student population of a local university. The inclusion criteria were healthy participants without any history of cardiovascular, respiratory, or metabolic diseases, and not on any regular medication. Exclusion criteria included participants with recent illnesses, those who smoked or consumed alcohol regularly, or were engaged in any breathing or relaxation exercises in the past month.The study was approved by the Institutional Ethics Committee. Written informed consent was obtained from all participants prior to enrollment, ensuring confidentiality and voluntary participation.

Methodology

Participants were randomly allocated into two groups:

- **Control group (n = 60):** Participants were seated quietly and asked to breathe normally for a duration of 5 minutes.
- **Intervention group (n = 60):** Participants were instructed to perform slow deep breathing exercises for 5 minutes. Each breathing cycle consisted of 5 seconds of inhalation followed by 5 seconds of exhalation, resulting in 6 complete breathing cycles per minute.

All participants were seated comfortably in a quiet room with a stable ambient temperature to ensure uniformity. After a 5-minute rest period, baseline

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measurements were recorded. Heart rate (HR) was measured using pulse oximetry, while blood pressure (BP), both systolic and diastolic, was obtained using an automated digital sphygmomanometer. Respiratory rate (RR) was assessed by counting the number of breaths per minute. Oxygen saturation (SpO2) was monitored using pulse oximetry to ensure adequate oxygenation. In addition, heart rate variability (HRV) was assessed using a heart rate monitor, which calculated key parameters including the standard deviation of normal-to-normal intervals (SDNN), root mean square of successive differences (RMSSD), and the low-frequency to high-frequency ratio (LF/HF ratio), which are indicators of autonomic nervous system activity.Following these baseline measurements, participants were assigned to one of two groups. The intervention group performed slow deep breathing exercises for 5 minutes, with each cycle comprising 5 seconds of inhalation followed by 5 seconds of exhalation, resulting in six complete cycles per minute. In contrast, the control group continued normal breathing for the same duration. Post-intervention, the same parameters were re-measured, including heart rate, systolic and diastolic blood pressure, respiratory rate, oxygen saturation, and heart rate variability.The primary outcome measures were the changes in heart rate and blood pressure (systolic and diastolic) from the baseline values to immediately after the intervention. Secondary outcome measures included respiratory rate, where a decrease was expected in the intervention group due to the slow deep breathing exercise. Oxygen saturation was monitored to ensure that it remained stable throughout the procedure. Heart rate variability was evaluated as an indicator of autonomic nervous system function, with increases in HRV (higher SDNN and RMSSD, lower LF/HF ratio) anticipated in the intervention group following the breathing exercise.

Statistical Analysis

Data were analyzed using SPSS version 25.0. Continuous variables (HR, BP, RR, SpO2, HRV indices) were expressed as mean ± standard deviation. Paired t-tests were used to compare pre- and post-intervention values within groups, and independent t-tests were employed to compare the intervention group with the control group. HRV indices were analyzed to determine changes in autonomic balance. A p-value < 0.05 was considered statistically significant.

RESULTS

Table 1: Demographic Characteristics of Study Participants

This table presents the demographic characteristics of the study participants. The average age of the participants in both the control group and the intervention group was very similar, with the control group having a mean age of 22.5 ± 2.8 years and the intervention group 22.8 ± 2.6 years, showing no statistically significant difference (p = 0.489). Gender distribution was balanced, with males representing 50.00% in the control group and 53.33% in the intervention group. The overall gender composition was fairly balanced across both groups (males: 51.67%, females: 48.33%), and no significant difference in gender distribution was observed (p = 0.722).The Body Mass Index (BMI) was also similar between the groups, with the control group having a mean BMI of 22.0 ± 2.5 kg/m² and the intervention group having a mean BMI of 21.8 ± 2.4 kg/m² (p = 0.638), indicating no significant variation. All participants were non-smokers (100% in both groups). In terms of physical activity, 46.67% of participants in the control group and 45.00% in the intervention group reported engaging in regular exercise, with no significant difference (p = 0.845). Overall, the demographic characteristics between the two groups were well-matched, minimizing potential confounding variables.

Table 2: Heart Rate and Pulse Pressure Comparison

This table highlights the effects of slow deep breathing on heart rate and pulse pressure. The intervention group, which performed slow deep breathing exercises, showed a significant reduction in heart rate compared to the control group. The mean heart rate in the intervention group was 65 ± 7 bpm, which was 10 beats per minute lower than the control group (75 ± 8 bpm). This difference was statistically significant (p < 0.001), indicating that slow deep breathing had an immediate effect on lowering heart rate.Similarly, pulse pressure, the difference between systolic and diastolic blood pressure, was lower in the intervention group (35 ± 4 mmHg) compared to the control group (40 ± 5 mmHg), with a mean difference of 5 mmHg (p = 0.038). This suggests that slow deep breathing not only affects heart rate but also has an impact on vascular pressure dynamics.

Table 3: Blood Pressure and Mean Arterial Pressure (MAP) Comparison

The intervention group exhibited a significant reduction in blood pressure values. The systolic blood pressure in the intervention group was 115 ± 9 mmHg, 5 mmHg lower than the control group (120 ± 10 mmHg), with a p-value of 0.045. Diastolic blood pressure also decreased significantly, with the intervention group recording 75 ± 7 mmHg compared to 80 ± 8 mmHg in the control group (p = 0.001).Mean arterial pressure (MAP), which is a calculated average of blood pressure during a single cardiac cycle, was significantly lower in the intervention group (88 ± 6 mmHg) compared to the control group (93 ± 7 mmHg), with a mean difference of 5 mmHg (p = 0.021). These results show that slow deep breathing leads to a significant reduction in both systolic and diastolic blood pressure, as well as the overall MAP, indicating its beneficial effects on blood pressure regulation.

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Table 4: Respiratory Rate, Oxygen Saturation, and Tidal Volume Comparison

This table shows that the intervention group experienced a significant reduction in respiratory rate after the breathing exercises. The respiratory rate in the intervention group dropped to 12 ± 1 breaths per minute, compared to 16 ± 2 breaths per minute in the control group, with a mean difference of 4 breaths per minute ($p < 0.001$). This reduction in respiratory rate is expected due to the slow breathing exercise.Oxygen saturation, which measures the percentage of hemoglobin that is saturated with oxygen, slightly increased in the intervention group (99 ± 1%) compared to the control group (98 ± 1%), with a mean difference of 1% ($p < 0.001$). The increase in oxygen saturation indicates that slow deep breathing helps improve oxygenation efficiency.Tidal volume, which is the amount of air displaced between inhalation and exhalation, increased significantly in the intervention group (500 ± 40 mL) compared to the control group (450 ± 50 mL), with a mean difference of 50 mL ($p = 0.029$). This demonstrates that slow deep breathing increases the depth of breaths, allowing for better air exchange.

Table 5: Heart Rate Variability (HRV) Comparison – SDNN, RMSSD, and Total Power

Heart rate variability (HRV) was significantly enhanced in the intervention group. The standard deviation of normal-to-normal intervals (SDNN), an indicator of overall HRV, was higher in the intervention group (70 ± 12 ms) compared to the control group (50 ± 10 ms), with a mean difference of 20 ms ($p < 0.001$). This indicates an improvement in autonomic regulation of the heart.Similarly, the root mean square of successive differences (RMSSD), which reflects parasympathetic activity, was also higher in the intervention group (50 ± 9 ms) compared to the control group (30 ± 8 ms), with a mean difference of 20 ms ($p < 0.001$). This suggests that slow deep breathing significantly enhances parasympathetic (relaxation) tone.The total power of HRV, which reflects overall autonomic function, was significantly greater in the intervention group (1800 ± 250 ms²) compared to the control group (1500 ± 200 ms²), with a mean difference of 300 ms² ($p = 0.013$). These results demonstrate that slow deep breathing has a positive effect on autonomic regulation, as reflected in the increased HRV parameters.

Table 6: Heart Rate Variability (HRV) Comparison – LF/HF Ratio and High-Frequency Power

In this table, the LF/HF ratio, which indicates the balance between sympathetic and parasympathetic activity, was significantly lower in the intervention group (1.2 ± 0.4) compared to the control group (1.8 ± 0.5), with a mean difference of -0.6 ($p < 0.001$). A lower LF/HF ratio suggests a shift towards parasympathetic dominance and a reduction in sympathetic activity, which is associated with relaxation and reduced stress.High-frequency power (HF), which is associated with parasympathetic (vagal) activity, was significantly higher in the intervention group (1000 ± 180 ms²) compared to the control group (800 ± 150 ms²), with a mean difference of 200 ms² ($p = 0.027$). This increase in high-frequency power further supports the notion that slow deep breathing enhances parasympathetic activity, promoting a relaxed physiological state.

Table 1: Demographic Characteristics of Study Participants (n = 120)

Characteristic	Control Group (n = 60)	Intervention Group (n = 60)	Total (n = 120)	p-value (ANOVA)
Age (years)	22.5 ± 2.8	22.8 ± 2.6	22.6 ± 2.7	0.489
Gender				
Male	30 (50.00%)	32 (53.33%)	62 (51.67%)	0.722
Female	30 (50.00%)	28 (46.67%)	58 (48.33%)	-
BMI (kg/m²)	22.0 ± 2.5	21.8 ± 2.4	21.9 ± 2.4	0.638
Smoking Status				
Non-smoker	60 (100.00%)	60 (100.00%)	120 (100.00%)	-
Physical Activity				
Regular Exercise	28 (46.67%)	27 (45.00%)	55 (45.83%)	0.845
No Regular Exercise	32 (53.33%)	33 (55.00%)	65 (54.17%)	-

Table 2: Heart Rate and Pulse Pressure Comparison (Control vs. Intervention Group)

Parameter	Control Group (Mean ± SD)	Intervention Group (Mean ± SD)	Mean Difference	p-value (ANOVA)
Heart Rate (bpm)	75 ± 8	65 ± 7	-10	< 0.001
Pulse Pressure (mmHg)	40 ± 5	35 ± 4	-5	0.038

Table 3: Blood Pressure and Mean Arterial Pressure (MAP) Comparison

Parameter	Control Group (Mean ± SD)	Intervention Group (Mean ± SD)	Mean Difference	p-value (ANOVA)
Systolic BP (mmHg)	120 ± 10	115 ± 9	-5	0.045

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Diastolic BP (mmHg)	80 ± 8	75 ± 7	-5	0.001
Mean Arterial Pressure (MAP)	93 ± 7	88 ± 6	-5	0.021

Table 4: Respiratory Rate, Oxygen Saturation, and Tidal Volume Comparison

Parameter	Control Group (Mean ± SD)	Intervention Group (Mean ± SD)	Mean Difference	p-value (ANOVA)
Respiratory Rate (breaths/min)	16 ± 2	12 ± 1	-4	< 0.001
Oxygen Saturation (%)	98 ± 1	99 ± 1	+1	< 0.001
Tidal Volume (mL)	450 ± 50	500 ± 40	+50	0.029

Table 5: Heart Rate Variability (HRV) Comparison – SDNN, RMSSD, and Total Power

Parameter	Control Group (Mean ± SD)	Intervention Group (Mean ± SD)	Mean Difference	p-value (ANOVA)
SDNN (ms)	50 ± 10	70 ± 12	+20	< 0.001
RMSSD (ms)	30 ± 8	50 ± 9	+20	< 0.001
Total Power (ms²)	1500 ± 200	1800 ± 250	+300	0.013

Table 6: Heart Rate Variability (HRV) Comparison – LF/HF Ratio, High-Frequency Power

Parameter	Control Group (Mean ± SD)	Intervention Group (Mean ± SD)	Mean Difference	p-value (ANOVA)
LF/HF Ratio	1.8 ± 0.5	1.2 ± 0.4	-0.6	< 0.001
High-Frequency Power (ms²)	800 ± 150	1000 ± 180	+200	0.027

DISCUSSION

In the current study, both the control and intervention groups were well-matched in terms of age, gender, BMI, smoking status, and physical activity, minimizing confounding factors. This demographic alignment is similar to the study by Pal et al. (2019), where no significant differences were noted between intervention and control groups regarding baseline characteristics like age, gender, and BMI. This consistency in demographics across studies strengthens the reliability of observed outcomes and ensures that variations in results are due to the intervention rather than underlying participant differences.[7]The significant reduction in heart rate (by 10 bpm) in the intervention group aligns with the findings of Soni et al. (2019), where slow deep breathing resulted in a decrease of 8-12 bpm, reflecting enhanced parasympathetic activity.[8] Additionally, the observed decrease in pulse pressure (by 5 mmHg) in this study supports the conclusions from Patil et al. (2018), which showed that controlled breathing exercises improve arterial elasticity, resulting in lower pulse pressure. Both studies suggest that slow breathing can positively affect cardiovascular dynamics, enhancing heart rate control and reducing vascular tension.[9]

This study showed significant reductions in both systolic and diastolic blood pressure (by 5 mmHg each), similar to the findings of Bhavsar et al. (2020), where slow breathing led to reductions of 4-6 mmHg in systolic BP and 3-5 mmHg in diastolic BP. These changes are attributed to a combination of increased parasympathetic tone and reduced sympathetic outflow, leading to vasodilation.[10] Additionally, Fonkoue et al. (2018) also demonstrated that slow breathing significantly lowers MAP, similar to the 5 mmHg decrease observed in this study. These consistent reductions across studies suggest that slow breathing is an effective non-pharmacological intervention for blood pressure regulation.[11]The decrease in respiratory rate (by 4 breaths/min) observed in this study aligns with results from Jayaraman et al. (2020), where controlled breathing led to a similar reduction in respiratory rate.[12] The increase in oxygen saturation (by 1%) is consistent with the findings of Sharma et al. (2019), who showed that slow deep breathing improves oxygenation by optimizing lung ventilation and gas exchange.[13] Moreover, the increase in tidal volume (by 50 mL) parallels the results of Mishra et al. (2021), where breathing exercises were found to increase lung capacity, enhancing tidal volume and improving respiratory efficiency.[14]

This study demonstrated significant improvements in HRV parameters, including a 20 ms increase in both SDNN and RMSSD, reflecting enhanced parasympathetic activity. These findings align with those of Khodakarami et al. (2018), who reported similar improvements in HRV indices following slow breathing interventions.[15] The increase in total HRV power (by 300 ms²) supports the results of Prinsloo et al. (2020), who found that deep breathing significantly enhances overall autonomic regulation. These results consistently show that slow breathing enhances HRV, indicating improved autonomic function and stress resilience.[16]The reduction in the LF/HF ratio (by 0.6) observed in this study is consistent with the work of Laborde et al. (2018), who also noted reductions in the LF/HF ratio following slow deep breathing exercises, indicating a shift toward parasympathetic dominance.[17] Additionally, the increase in high-frequency power (by 200 ms²) is comparable to the

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findings of Lehrer et al. (2021), who demonstrated that slow breathing significantly increases vagal activity, as reflected in higher HF power. These studies collectively suggest that slow deep breathing promotes relaxation by enhancing parasympathetic activity and reducing sympathetic arousal.[18]

CONCLUSION

In conclusion, this study demonstrates that short-duration slow deep breathing has immediate beneficial effects on heart rate and blood pressure in healthy young adults. The practice significantly reduces heart rate and systolic and diastolic blood pressure, promoting parasympathetic activation while suppressing sympathetic activity. These findings suggest that slow deep breathing is an effective non-pharmacological intervention for enhancing cardiovascular function and managing stress-related physiological responses. Its simplicity and accessibility make it a practical tool for improving autonomic regulation and overall cardiovascular health, especially in younger populations.

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