An Exploratory Randomised Trial to Assess the Effect of Nadi Shodhan Pranayama as an Adjunct Versus Standard Non-pharmacological Management in Hypertensives

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Abstract

Background: Nadi shodhan pranayama, which is also known as Anulom-vilom pranayama or alternate nostril breathing, has been demonstrated to elicit a decrease in systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate, owing to its stress-reducing effects. One of the most promising quantitative markers of autonomic activity is heart rate variability (HRV).
Purpose: This study was done to study the immediate and long-term effects of 10 minutes of nadi shodhan pranayama, preceded by two minutes of yogic deep abdominal breathing, on blood pressure and other autonomic parameters.
Methods: Baseline parameters of BP, HRV, Pittsburgh Sleep Quality Index (PSQI) and Hamilton Anxiety Rating Scale (HAM-A) were measured at first visit. Patients were randomised into test and control groups, and the test group was given intervention. BP and HRV were measured once again immediately after intervention for test subjects. After six weeks, BP, HRV, PSQI and HAM-A were repeated.
Results: Immediately after the intervention, the test group showed a significant reduction in SBP, DBP and mean heart rate (MHR) with an increase in R-R interval, standard deviation of the NN interval (SDNN), the square root of the mean squared differences of successive NN intervals and high frequency. At follow-up, the test group had a lower SBP and low frequency/high frequency ratio and a higher SDNN and total power, compared to the control group.
Conclusion: The practice of nadi shodhan pranayama, preceded by two minutes of yogic deep abdominal breathing, reduced the systolic and diastolic BP and improved HRV-related autonomic parameters, in patients of hypertension on pharmacological therapy, immediately after 10 minutes of practice and after 10 minutes of daily practice for six weeks.

Keywords

Anulom-vilom pranayama, nadi shodhan pranayama, hypertension, heart rate variability, yoga and hypertension, autonomic nervous system

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Introduction

World over, 32% of women and 34% of men from those aged 30–79 years in 2019 were affected by hypertension, which comes to 1.28 billion adults.[1] In India, almost one in every 12 adults has hypertension, and one of the goals of India according to WHO is a reduction by 25% in the prevalence of hypertension by 2025.[2] According to the American Heart Association (AHA) guidelines, systolic and diastolic BPs fall into different categories, and the higher category is prioritised for diagnosis and treatment.[3]

Hypertension is often called a 'silent killer'. It does not show many symptoms initially, but can lead to serious health

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problems over a long period of time, if not managed.[4] Normally, our arteries are flexible and smooth inside, making it easy for blood to flow and provide organ oxygenation. But when blood pressure stays high for a long time, it can make the arterial walls stiff and narrow, leading to blockages and weak spots called aneurysms. This can cause end organ complications such as heart disease, myocardial infarction, stroke, hypertensive retinopathy, peripheral arterial disease, hypertensive retinopathy, heart failure, fibrillation and bulging blood vessels in the aorta called aneurysms.[5] Managing hypertension typically involves a combination of medications and lifestyle adjustments. Among the commonly prescribed drugs are angiotensin-converting enzyme (ACE) inhibitors, which relax blood vessels, and angiotensin receptor blockers (ARBs), which block the effects of angiotensin, a hormone that narrows blood vessels. Diuretics help the body eliminate excess sodium and water, reducing blood volume and pressure. Beta-blockers and calcium channel blockers work by slowing the heart rate (HR) and relaxing blood vessels, respectively. Alpha-blockers target the hormone adrenaline to relax blood vessels, aiding in blood flow.[6]

Traditional practices such as yoga and transcendental meditation have gained recognition for their potential benefits in hypertension management. These practices promote relaxation, stress reduction and overall well-being, which can contribute to better blood pressure control, when incorporated into a comprehensive treatment plan.[7]

Yoga incorporates three core elements: postures (Asanas), breath control (Pranayama) and meditation techniques (dharana). Yoga, thus, represents a comprehensive practice encompassing physical, mental, emotional and spiritual dimensions. The physical component of yoga enhances flexibility, strength and balance, while concurrently modulating physiological stress responses. Through mindfulness and introspection, yoga cultivates emotional regulation and psychological resilience. Additionally, yoga serves as a framework for spiritual exploration, facilitating a deeper understanding of the self.[8]

There are multiple types of Pranayama practiced in yogic sciences. One of them is known as nadi shodhan pranayama, also popularly called anulom-vilom pranayama or alternate nostril breathing. Nadi shodhan pranayama is an ancient Indian yogic practice to regulate the breath by alternatively closing and opening the left and right nostrils.[9] Multiple studies on this pranayama technique have already been done, and its mechanisms and effects on different parameters of the human body are being studied actively.[10] It has been demonstrated to elicit a decrease in systolic blood pressure (SBP), diastolic blood pressure (DBP) and HR, owing to its stress-reducing effects. This reduction in physiological arousal is attributed to a decrease in sympathetic nervous system activity along with the stimulation of slowly adapting stretch receptors (SARs), mediated by the Hering-Breuer inflation reflex. Consequently, this leads to the activation of the vagus nerve, resulting in parasympathetic nervous system predominance and fostering a state of relaxation and well-being.[10–12]

One of the most promising quantitative markers of autonomic activity is heart rate variability (HRV).[13] It essentially describes the oscillation in consecutive cardiac cycles or the variations in instantaneous HR and RR intervals. Following an acute myocardial infarction, HRV has emerged as a robust and independent predictor of mortality.[14] HRV analysis includes two main components: time domain methods and frequency domain methods. Time domain methods involve assessing parameters such as the mean NN interval, mean HR, standard deviation of the NN interval (SDNN) and the square root of the mean squared differences of successive NN intervals (RMSSD). On the other hand, frequency domain methods examine spectral components, which range from very low frequency (VLF) to low frequency (LF) and high frequency (HF) components.[15–17]

One of the primary aims of hypertension management is to decrease the elevated risk of cardiovascular morbidity and mortality associated with chronic high blood pressure. A decrease in sympathetic dominance and better sympathetic-parasympathetic balance leads to a decrease in the harmful effects on the heart due to multiple factors, including better control of blood pressure.[12]

The aim of this study was to elicit the short- and long-term effects of nadi shodhan pranayama preceded by two minutes of yogic deep abdominal breathing on blood pressure and other autonomic parameters.

Methods

Study Design

Our study was an outpatient department based exploratory, single-centre, parallel group and randomised control trial. Written informed consent was taken from all patients before the study procedure. The study was compliant with ethical principles aligned with the Declaration of Helsinki, Good Clinical Practice guidelines, CONSORT guidelines and local laws and regulations. The study duration was six months after due approval from an institutional ethics committee, AIIMS Rishikesh. The study protocol was registered with the Clinical Trial Registry – India (CTRI).

All patients over 30 years of age coming to the OPD were screened. Those who were known cases of hypertension on medication were recruited along with those on appropriate antihypertensive treatment with systolic BP more than 140 mmHg and/or diastolic BP more than 90 mmHg based on an average 10-day home BP measurement. Possible participants were explained about the study in their local language, and if they agreed to take part in the study and signed the consent form, they were recruited in the study.

Baseline parameters of BP, HRV, Pittsburgh Sleep Quality Index (PSQI) and Hamilton Anxiety Rating Scale (HAM-A) were measured at first visit. Patients were randomised into test and control groups, and the test group was given intervention. BP and HRV were measured once again immediately after

intervention for test subjects. Test subjects were asked daily through telephone and telephonic messenger to continue with the intervention process at home. Then, after six weeks, subjects of both test and control were called back to the OPD and their BP, HRV, PSQI and HAM-A were repeated.

Participants

The inclusion criteria were: (a) participants of age >30 years and <80 years; (b) newly diagnosed and self-reported cases of primary hypertension on medication; (c) able to follow verbal instructions; (d) having an adequate understanding of English or Hindi; (e) able to sit without physical discomfort for 10 minutes; and (f) having phones/smartphones/smart devices.

The exclusion criteria included: (a) inability to independently provide informed consent; (b) patients presenting with hypertensive emergency or urgency; and (c) already practicing some kind of pranayama, yoga or exercise.

Randomisation

Patients who passed the inclusion criteria and signed the consent form were randomised into two groups by generating a sequence through a random sequencing number generator and a sequentially numbered, opaque and sealed envelope (SNOSE) technique, which was done by a third person unrelated to the study.

Interventions

Two minutes of yogic deep abdominal breathing followed by 10 minutes of nadi shodhan pranayama, where the breath was maintained as deep and abdominal, was taught to the subjects in the intervention arm. Nadi shodhan pranayama comprises of three main components: (a) deep abdominal breathing, (b) alternate nostril breathing and (c) prevention of mind wandering by constant awareness on the breath. Thus, the preceding two minutes of deep abdominal breathing was done as a prelude to the first component. For those in the placebo arm, no intervention was given and a non-pharmacological therapy based on AHA hypertension guidelines of 2017 was given. We began with two minutes of yogic deep abdominal breathing as preparatory practice before nadi shodhan pranayama. One cycle of nadi shodhan pranayama started with closing one nostril with the ring finger and inhaling from the opposite nostril, followed by closing the other nostril and exhaling from the open nostril, and vice versa. There was no retention of breath.

Outcomes

Blood Pressure

Hypertensives, as diagnosed according to the AHA 2017 criteria or on similar medications, were recruited. Blood pressure was measured with a Diamond BPDG141 manual aneroid sphygmomanometer through palpatory and auscultatory methods.

Heart Rate Variability

HRV was measured to quantify sympathetic and parasympathetic tone.

Though, in principle, the HRV can be measured over any length of recorded ECG, as per the guidelines of the Task Force, at least five minutes of ECG must be recorded. For the patient's preparation, the following instructions were given: (a) avoid food preceding two hours of the testing; (b) no coffee, nicotine, or alcohol 24 hours prior to the testing; (c) avoid any over-the-counter cough and cold medications; and (d) wear loose and comfortable clothing.

Recording

For short-term analysis of HRV, ECG was recorded in the sitting position for five minutes after five minutes of supine rest. The room temperature was maintained at 24°C. The subject was instructed to close the eyes and avoid talking; moving hands, legs and body; coughing and sleeping.

HRV analysis was based on five minutes of ECG signal using an emWave Pro Multi-user device from HeartMath. The device sensor was placed on the ear lobe of the patient after removing any jewellery, if present, from the ear lobe. It was measured for five minutes before intervention, five minutes after intervention and for five minutes at follow-up, after a rest period of five minutes. The HR was calculated using the R-waves from artefact free graph for both stages of recordings.

The analysis of HRV is done by time and frequency domain methods. Time domain parameters include mean heart rate (MHR), R-R, SDNN and RMSSD, and frequency domain parameters include total power, LF, HF and LH/HF.

Sleep

Sleep quality was measured using the PSQI. Permission was obtained for its use. The PSQI is a self-rated validated questionnaire which assesses sleep quality and disturbances over a one-month time interval. There are 19 individual items which generate seven 'component' scores: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication and daytime dysfunction. The sum of scores for these seven components gives one total score. The higher the PSQI value, the poorer the sleep quality.

Anxiety

Anxiety was measured using the HAM-A, which is a public domain validated clinician-rated 14 questionnaire scale, with each question having four options marked from 0 to 4, with 0 being a 'fit' response denoting 'not present', 1: 'Mild', 2:

'Moderate', 3: 'Severe', 4: 'Very severe'. A high total value indicated high anxiety levels.

Sample Size Calculation

As it was an exploratory study, a minimum sample size of 32 patients was maintained.

Statistical Analysis

Ninety-one patients were recruited, out of which 23 were lost to follow-up. There were 34 subjects in each test and control group, which were analysed. Stata/BE 18.0 was the software used for statistical analysis. Skewness and Kurtosis normality test was first applied, and accordingly, a paired t-test was applied for normally distributed data within groups at baseline, immediate response and follow-up, while an unpaired t-test was applied for data between two groups of test and control at baseline and at follow-up. For skewed data, non-parametric tests were applied. Wilcoxon signed rank test was applied for data within the group at baseline, immediate response and at follow-up. And Mann–Whitney U test (Wilcoxon rank sum test) was applied for skewed data between groups of test and control at baseline and follow-up. Data are described as mean (±SD) for parametric tests and median (IQR) for non-parametric tests. P value of $< .05$ was taken as statistically significant.

Results

Among the 124 participants screened, 91 were randomised into test (47) and control (46) groups. After excluding drop-outs, 34 were analysed in each group at follow-up.

The sequence of computing result data were as follows: (a) baseline parameters were recorded for test and control groups; (b) immediately after intervention, in the test group, all the parameters were recorded and compared by baseline parameters; (c) after a six-week period of follow-up, in the test group, all the parameters were recorded and compared by baseline parameters; and (d) after a six-week period of follow-up, in the control group, all the parameters were recorded and compared by follow-up parameter values of the test group.

Parameters analysed for the test group and control group were: (a) the demographic characteristics; (b) blood pressure: systolic and DBP; (c) HRV time and frequency domain parameters; and (d) sleep quality and anxiety scores.

Sample Characteristics

The baseline characteristics are represented in Table 1. The number of patients in the test group and in the control group was 34 each. The mean age of the test patient group was 61.38 years and of the control patient group was 65.58 years. In the test group, there were 25 male and 9 female patients, and in the control group there were 16 male and 18 female patients.

Primary Outcome

The results for the SBP and DBP outcomes are represented in Table 2. Immediately after intervention, in the test group, there was a significant decrease in SBP and DBP from baseline. After follow-up intervention, there was a significant decrease in SBP but a non-significant decrease in DBP of the test group, in comparison to the control group.

Secondary Outcomes

Heart Rate Variability Time and Frequency Domain Parameters

Immediately after intervention, in the test group, there was a significant decrease in MHR, a significant increase in R-R interval and a significant increase in SDNN and RMSSD from baseline. After follow-up intervention, in the test group, there was a non-significant decrease in MHR and R-R interval and a significant increase in SDNN and RMSSD from baseline. After follow-up intervention, in the test group, there was a non-significant decrease in MHR, increase in R-R interval, increase in RMSSD and a significant increase in SDNN in comparison to the control group. The results are shown in Table 3.

There was no significant difference in total power, LF, HF and LH/HF test baseline and control baseline values. Immediately after intervention, in the test group, there was a non-significant increase in total power and LF, a significant increase in HF and a non-significant decrease in LH/HF from baseline. After a follow-up intervention, in the test group, there was a non-significant increase in total power and LF, a significant increase in HF and a non-significant decrease in LH/HF from the baseline. After a follow-up intervention, in the test group, there was a significant increase in total power, a non-significant decrease in LF, a non-significant increase in HF and a significant decrease in LF/HF ratio in comparison to the control group. The results are represented in Table 4.

Sleep Quality (PSQI) and Anxiety Scores (HAM-A)

There was no statistically significant difference between test and control baseline. After follow-up intervention, in the test

Table 1. Demographic Characteristics of Test and Control Group Hypertensive Patients.

	Test		Control	
Number	34		34	
Age (years)	61.38		65.58	
Gender	Male: 25	Female: 9	Male: 16	Female: 18

Table 2. Results of SBP and DBP in Test and Control Groups at Baseline, in Test Group Immediately After Intervention, in Test Group at Follow-up and Between Test and Control at Follow-up.

Baseline BP of Test and Control Group				
	SBP Basal		**DBP Basal**	
Values	**Test**	**Control**	**Test**	**Control**
Mean	153.05	152.14	89.61	87.32
±SD	13.43	12.73	11.61	10.09
P value	>.05		>.05	
Immediate Effect of Intervention on BP in Test Group				
	SBP Test Group		**DBP Test Group**	
	Basal	**ImmediateAfter Intervention**	**Basal**	**ImmediateAfter Intervention**
Mean	153.05	142.94	89.61	87.11
±SD	13.43	14.43	11.61	10.19
P value	<.001		<.001	
Comparison of BP Between Baseline and Follow-up in Test Group				
	SBP Test Group		**DBP Test Group**	
	Basal	**Six Weeks of Follow-up with Intervention**	**Basal**	**Six Weeks of Follow-up with Intervention**
Mean	153.05	139.14	89.61	83.64
±SD	13.43	12.5	11.61	11.05
P value	<.001		<.001	
Comparison of BP Between Test and Control at Follow-up				
	SBP Follow-up		**DBP Follow-up**	
	Test	**Control**	**Test**	**Control**
Mean	139.14	147.79	83.64	87.61
±SD	12.5	12.8	11.05	8.75
P value	<.01		>.05	

Table 3. Results of MHR, R-R, SDNN and RMSSD in Test and Control Groups at Baseline, in Test Group Immediately After Intervention, in Test Group at Follow-up and Between Test and Control at Follow-up.

Baseline MHR, R-R, SDNN, RMSSD of Test and Control Groups									
	MHR Basal			**R-R Basal**		**SDNN Basal**		**RMSSD Basal**	
Values	**Test**	**Control**		**Test**	**Control**	**Test**	**Control**	**Test**	**Control**
Mean	80.2	82.6	Median	757.45	695.85	33.05	31.4	31.3	33.85
±SD	15.5	14.2	IQR*	556.3	573.4	17.8	20.3	19.1	23.7
P value	>.05			>.05		>.05		>.05	
Comparison of MHR, R-R, SDNN, RMSSD of Baseline and Immediately After Intervention in Test Group									
Values	**MHR Test Group**			**R-R Test Group**		**SDNN Test Group**		**RMSSD Test Group**	
	Basal	**Immediate After Intervention**		**Basal**	**Immediate After Intervention**	**Basal**	**Immediate After Intervention**	**Basal**	**Immediate After Intervention**
Mean	80.2	78.13	Median	757.45	765.4	33.05	45	31.3	38.7
±SD	15.5	13.8	IQR*	556.3	589.1	17.8	22.1	19.1	22
P value	<.01			<.01		<.05		<.01	
Comparison of MHR, R-R, SDNN, RMSSD of Baseline and Follow-up in Test Group									
Values	**MHR Test Group**			**R-R Test Group**		**SDNN Test Group**		**RMSSD Test Group**	
	Basal	**Six Weeks of Follow-upwith Intervention**		**Basal**	**Six Weeks of Follow-up with Intervention**	**Basal**	**Six Weeks of Follow-up with Intervention**	**Basal**	**Six Weeks of Follow-up with Intervention**
Mean	80.2	78.34	Median	757.45	754.8	33.05	43.05	31.3	38.65
±SD	15.5	14.02	IQR*	556.3	538.1	17.8	22.9	19.1	23.5
P value	>.05			>.05		<.01		<.01	
Comparison of MHR, R-R, SDNN, RMSSD at Follow-up in Test and Control Group									
Values	**MHR Follow-up**			**R-R Follow-up**		**SDNN Follow-up**		**RMSSD Follow-up**	
	Test	**Control**		**Test**	**Control**	**Test**	**Control**	**Test**	**Control**
Mean	79.85	83.18	Median	748.1	683.9	43.05	29.1	38.65	32.9
±SD	14.75	15.98	IQR*	538.1	512.1	22.9	14.3	23.5	20.3
P value	>.05			>.05		<.001		>.05	

Note: *Interquartile range.

Table 4. Results of Total Power, LF, HF and LH/HF in Test and Control Groups at Baseline, in Test Group Immediately After Intervention, in Test Group at Follow-up and Between Test and Control at Follow-up.

Baseline Total Power, LF, HF and LH/HF of Test and Control Groups								
Values	**Total Power Basal**		**LF Basal**		**HF Basal**		**LF/HF Basal**	
	Test	**Control**	**Test**	**Control**	**Test**	**Control**	**Test**	**Control**
Median	225.2	170.8	58.5	38.35	58.55	49.45	1.15	0.9
IQR*	23.8	11.4	4.8	2.8	4.8	6.6	0.2	0.2
P value	>.05		>.05		>.05		>.05	
Comparison of Total Power, LF, HF and LH/HF of Baseline and Immediately After Intervention in the Test Group								
Values	**Total Power Test Group**		**LF Test Group**		**HF Test Group**		**LF/HF Test Group**	
	Basal	**Immediate After Intervention**	**Basal**	**Immediate After Intervention**	**Basal**	**Immediate After Intervention**	**Basal**	**Immediate After Intervention**
Median	225.2	290.95	58.55	79.55	53	57.15	1.15	1.05
IQR*	23.8	36.1	4.8	4.9	8.4	13.5	0.2	0.02
P value	>.05		>.05		<.01		>.05	
Comparison of Total Power, LF, HF and LH/HF of Baseline and Follow-up in the Test Group								
Values	**Total Power Test Group**		**LF Test Group**		**HF Test Group**		**LF/HF Test Group**	
	Basal	**Six Weeks of Follow-up with Intervention**	**Basal**	**Six Weeks of Follow-up with Intervention**	**Basal**	**Six Weeks of Follow-up with Intervention**	**Basal**	**Six Weeks of Follow-up with Intervention**
Median	225.2	429	58.55	92.9	53	70.55	1.15	1.05
IQR*	23.8	48.8	4.8	9.8	8.4	14.1	0.2	0.03
P value	>.05		>.05		<.05		>.05	
Comparison of Total Power, LF, HF and LH/HF at Follow-up in the Test and Control Group								
Values	**Total Power Follow-up**		**LF Follow-up**		**HF Follow-up**		**LF/HF Follow-up**	
	Test	**Control**	**Test**	**Control**	**Test**	**Control**	**Test**	**Control**
Median	429	170.7	92.9	94	70.55	53.73	1.05	2.09
IQR*	48.8	25.9	9.8	41.4	14.1	8.2	0.03	0.7
P value	<.001		>.05		>.05		<.001	

Note: *Interquartile range.

Table 5. Results of Sleep and Anxiety in Test and Control Groups at Baseline and at Follow-up.

Comparison Between Sleep and Anxiety at Baseline				
	PSQI Baseline		**HAM-A Baseline**	
Values	**Test**	**Control**	**Test**	**Control**
Median	7	5.5	7.5	7.5
IQR	1	1	0	0
P value	>.05		>.05	
Comparison Between Sleep and Anxiety at Follow-up				
	PSQI Follow-up		**HAM-A Follow-up**	
	Test	**Control**	**Test**	**Control**
Mean	5	6	7	8
±SD	2	1	0	0
P value	>.05		>.05	

group, there was a non-significant decrease in PSQI and HAM-A from baseline. After follow-up intervention, there was a non-significant decrease in PSQI and HAM-A of the test group, in comparison to the control group. The results for sleep and anxiety parameters are shown in Table 5.

Discussion

Blood Pressure

Cardiac output and peripheral vascular resistance are the main determinants of arterial blood pressure. Thus, both parameters, if on the higher side, lead to hypertension.[19]

The regulation of cardiac output and total peripheral resistance involves the relay of sensory inputs from peripheral receptors, including baroreceptors sensitive to stretch and cardio-pulmonary sensors responsive to volume. This is an almost continuous process with sensory signals being received every minute by the baroreceptors and cardio-pulmonary sensors. These signals converge at the medullary cardiovascular control centre (MCCC), where they are processed to modulate sympathetic and parasympathetic activity. The primary aim is to uphold a consistent mean arterial pressure (MAP) for sufficient organ perfusion. This detailed process ensures cardiovascular equilibrium and optimal functioning of vital organs.[20]

In our study, nadi shodhan pranayama was preceded by two minutes of yogic deep abdominal breathing, and this breathing pattern was maintained during the 10 minutes of nadi shodhan practice. During deep breaths, pulmonary SARs are stimulated, which cause a decrease in the sympathetic tone of the skeletal musculature, leading to a fall in vascular resistance, which might explain the fall in DBP in our study. The nasal airflow is regulated by sympathetic innervation, which in turn is regulated by the hypothalamus and the brainstem vasomotor areas.[21]

Slow pranayama breathing causes parasympathetic dominance to increase through two pathways. The first is the activation of slow adapting SARs above tidal volume inspiration, which generate inhibitory impulses in neuronal tissue and decrease its action potential.[22] These inhibitory impulses have been observed to play a role in controlling autonomic cardiorespiratory mechanisms such as the tone of the smooth muscles in the airways, HR, etc. Second, the stretch of fibroblasts during yogic deep abdominal breathing generates a hyperpolarisation current, which, apart from synchronising the neural tissues such as the hypothalamus and brainstem, also decreases the metabolic activity of the surrounding tissues by increasing the resting membrane potential polarity. Both these mechanisms lead to increasing the parasympathetic dominance and decreasing BP, HR and O2 consumption. -vagal responses decrease with [23] Immediately after intervention, there was a statistically significant decrease in SBP and DBP in the test group from baseline. During follow-up visit, again, there was a statistically significant decrease in SBP and DBP from baseline, and the difference of test vs control group at follow-up was statistically significant for SBP but not statistically significant for DBP.

These results were consistent with another study done by Saisupriya et al.,[24] where subjects who were given nadi shodhan pranayama in the ratio of 1:3:2 breaths with a six seconds inhalation, breath hold for 18 seconds, then exhalation for 12 seconds through each nostril alternatively, for 12 rounds, showed significant improvement in their SBP immediately after practicing pranayama. Although, in contrast to our study, in this study, there was no statistically significant improvement in DBP immediately after the intervention. This might be attributed to the difference in nadi shodhan breathing techniques between the two studies. In another study by Upadhyay et al.,[12] 20 minutes of nadi shodhan pranayama showed an immediate significant decrease in SBP and DBP, which was similar to our study. A study by Uikey and Sandel[25] on hypertensives showed significant improvement in SBP and DBP after a daily nadi shodhan practice of 15 minutes for four weeks.

HRV

The 'time domain' parameters include MHR, R-R interval, standard deviation of the NN interval (SDNN) and root-mean square of successive differences between R-R intervals (RMSSD), and the 'frequency domain' parameters include total power in the entire frequency range <0.4 Hz (total power), power in the LF range: 0.04 to 0.15 Hz (LF), power in the HF range: 0.15 to 0.4 Hz, as well as the LF/HF ratio.

HRV-time Domain Parameters: Mean Heart Rate and R-R Interval

Since slow breathing inhibits excitatory pathways which regulate the cardio-respiratory system, autonomic effects of parasympathetic dominance can be seen on HR. One of two reasons why our study showed a decrease, although non-significant, in HR at six weeks follow-up might be the mean age of our study subjects, which was above 60 years. It has been documented that cardio-vagal responses decrease with age.[26,27] Also, in contrast to the other studies, the daily intervention duration for our test group was shorter compared to the other studies. In a study by Saisupriya et al.,[24] there was a significant decrease in MHR. immediately after nadi shodhan pranayama. In another study by Kumaresan et al.,[28] there was a non-significant decrease in MHR. In a study by Goel et al.,[29] there was a significant decrease in MHR after 10 minutes of nadi shodhan pranayama. In yet another study by Srivastava et al.,[11] immediately after 15 minutes of nadi shodhan pranayama and follow-up of eight weeks, there was a significant decrease in MHR in both gender subjects from baseline. In concurrence with our study, a study by Vaksh and

Pandey,[30] normotensive individuals showed a significant decrease in MHR immediately after 15 minutes of nadi shodhan pranayama. Upadhyay et al.'s[12] study showed that there was a significant increase in RR interval immediately after nadi shodhan.

HRV-time Domain Parameters: SDNN and RMSSD

In a study by Malhotra et al.,[31] SDNN increased significantly during and just after nadi shodhan pranayama. RMSSD decreased slightly without any significant change immediately or after intervention. In accordance with our study and the study by Upadhyay et al.,[12] RMSSD increased after 20 minutes of nadi shodhan practice. In another study by Telles et al.,[32] RMSSD increased significantly during the 20 minutes intervention of nadi shodhan, but it showed a non-significant increase post intervention. In a study by Saisupriya et al.,[24] after 12 rounds of nadi shodhan pranayama for 36 seconds each, there was a significant increase in RMSSD.

HRV-frequency Domain Parameters: Total Power, LF, HF, LF-HF Ratio

A study by Ghiya and Lee[33] showed an increase in LF immediately after 30 minutes of nadi shodhan pranayama, but contrary to our study, the change in their study was significant. In concurrence with our study, the increase in HF in their study was statistically significant. Similar to our study, their LF-HF ratio also decreased without being statistically significant. In a study by Upadhyay et al.,[12] there was a non-significant decrease in LF in contrast to our study and a non-significant increase in HF, which was in concurrence with our study results. The LF-HF ratio was significantly decreased immediately after 20 minutes of nadi shodhan pranayama.

Time domain parameters of MHR and RR interval reflect sympathetic as well as parasympathetic control. MHR increases with adrenergic stimuli and decreases with cholinergic stimulus, while RR interval has an inverse relationship with MHR. SDNN is used as a quantitative measure of HRV. Higher SDNN is related to a lesser mortality due to cardiac issues. RMSSD reflects HF parasympathetic activity. Among the frequency domain metrics, total power, LF, HF and LF/HF ratio, power reflects both sympathetic and parasympathetic activity, where higher power denotes a parasympathetic dominance. LF is the power in the LF range and denotes the sympathetic activation, while HF is the power in the HF range and reflects the vagal dominance. LF-HF ratio denotes the balance between sympathetic-parasympathetic activity. Right nostril breathing has a sympathetic dominant effect on the body, while left nostril breathing has a parasympathetic effect. Alternate nostril breathing causes alternating sympathetic and parasympathetic effects, which in continuation may be leading to autonomic homeostasis.

Autonomic and central controls regulate nasal airflow.[35] Nasal airflow is also influenced by the nasal vasculature. Sympathetic nerves, which regulate the nasal vasculature, are regulated by the hypothalamus and vasomotor centres in the brainstem.[36] Sympathetic activation through hypothalamic nuclei is believed to be closely related to airflow through the right nostril. Hence breathing through alternate nostrils is believed to bring about a balance between sympathetic and parasympathetic nervous systems, and, hence, showed improved parameters related to autonomic functioning.

Studies have highlighted that HRV reflects the intricate communication between the brain and heart, allowing the autonomic nervous system (ANS) to regulate cardiac function in response to internal and external stimuli. This communication pathway, known as the intrinsic cardiac nervous system, encompasses various components such as ganglia, neurotransmitters, supportive cells and proteins. Through the activation of sympathetic and parasympathetic pathways, the ANS adjusts cardiac functions based on stimuli affecting heart physiology, with sensory neurons rapidly transmitting signals to the cardiac nervous system for further regulation.[34] The intrinsic nervous system of the heart communicates with the brain through ascending nerve fibres of the vagus nerve and the spinal column, targeting specific brain regions such as the medulla, hypothalamus, thalamus, amygdala and cerebral cortex for interpretation. Conversely, the brain influences cardiac activities via complex pathways involving peripheral ganglia, premotor neurons and myocardiocytes. Additionally, cardiac reflexes, such as baroreceptors and chemoreceptors, along with central autonomic responses to stress, exercise and sleep cycles, contribute to the regulation of cardiac function. It is noteworthy that chronic neurodegenerative diseases may impact ANS function, while vascular pathologies or neurological issues can lead to ANS hyperactivity.[37,38]

The body possesses various systems and features allowing it to adapt to different environments and activities. HRV serves as a measure of this adaptability. High HRV indicates the body's ability to adjust to changes, often associated with lower stress levels and increased happiness. Conversely, low HRV suggests potential health issues, signalling reduced resilience and difficulty in handling alterations. It is more prevalent in individuals with higher resting HRs, as faster heartbeats leave less room for variability between beats. Conditions such as diabetes, high blood pressure, heart arrhythmia, asthma, anxiety and depression are commonly linked to low HRV.[39]

Sleep and Anxiety

In contrast to our results, a study by Weheda et al.[40] showed significant improvement in sleep scores from baseline to one week and then again at four weeks after 15 minutes of nadi shodhan pranayama three times per day for four weeks with emphasis on maintaining quality time duration of inhalation and exhalation from each nostril. The higher frequency of their intervention might have a role to play in the enhanced improvement in sleep quality as compared to our study, which, although showed improved results, were not statistically significant.

In a study by Telles et al.,[32] after five minutes of nadi shodhan pranayama, there was a non-significant decrease in anxiety score done by State trait anxiety inventory from 38.66 ± 10.94 to 38 ± 11.44, which was in concurrence with our study. In another study done by Kamath et al.,[41] 15 minutes of Nadi shodhan practice was given to subjects before a public speech performance and their anxiety scores were measured five times till the speech performance was completed. In concurrence with our study results, there was improvement in the anxiety scores, but the improvement was not statistically significant. Although statistically non-significant, the improving values in anxiety and sleep scores point towards the possible effects of lowering of sympathetic tone, leading to a more anxiety-free and relaxed state in the practicing subjects.

The feeling of energy depletion or diminished 'prana' in the body is often attributed to improper breathing habits. Many individuals only utilise a fraction of their lung capacity, leading to inadequate oxygen supply and inefficient waste removal from the body. This oxygen deficit slows down bodily functions and impedes cellular and tissue regeneration due to insufficient energy. Through regular pranayama practice, one can significantly enhance oxygen intake, up to five times the usual amount, and expel five times as much carbon dioxide. Consequently, this would lead to improved health and a reduction in anxiety levels.[42]

The study illustrates an enhancement in the overall balance of the body's autonomic state through pranayama. This improvement is attributed to various processes, including the activation of mechanoreceptors, SARs and chemoreceptors located in nasal passages, respiratory tract, lungs, heart and blood vessels. These receptors transmit signals to the respiratory centre in the brainstem. These stretched tissues generate inhibitory signals and hyperpolarisation currents, which propagate through neural and non-neural tissues, synchronising neural elements in the lungs, heart, limbic system and cortex. This inhibitory influence and hyperpolarisation result in decreased metabolic activity, indicative of parasympathetic activation. The brainstem and hypothalamus probably play a crucial role in eliciting the parasympathetic response, promoting a more relaxed state that improves sleep.[43]

Physiological mechanisms underlying nadi shodhan pranayama are intricate and involve several bodily systems. It starts with the activation of mechano and chemoreceptors in the respiratory and circulatory system, which send signals to the brainstem's respiratory control centre. From there, the activation spreads to areas, such as the cerebellum and limbic system, finally reaching the cerebral cortex. This process creates a smooth physiological mechanism that readjusts breathing patterns according to the specific techniques of pranayama. These proposed mechanisms suggest that pranayama can have applications in rehabilitation across not only clinical populations but also for enhancing general health and well-being.[44]

Conclusion

The practice of nadi shodhan pranayama, preceded by two minutes of yogic deep abdominal breathing, reduced the systolic and diastolic BP, in patients of hypertension on pharmacological therapy, immediately after 10 minutes of practice and a daily 10 minutes practice for six weeks. It also improved parameters of HRV related to parasympathetic dominance and sympatho-vagal balance. The results of this study point towards the benefits nadi shodhan pranayama can provide to patients with hypertension, in controlling blood pressure and improving autonomic balance.

Authors' Contribution

Gauri Mittal, Monika Pathania formulated and conceptualized the idea, aims and goals. Gauri Mittal was the principal investigator. Praag Bhardwaj was part of the project administration and investigation. Monika Pathania and Minakshi Dhar were resource and supervising faculty. Meenakshi Khapre took part in the formal analysis. Sunita Mittal was part of the visualization process.

Statement of Ethics

Due approval was obtained from the Institutional Ethics Committee, AIIMS Rishikesh, via letter number 153/IEC/IM/NF/2023.

Declaration of Conflicting Interest

The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

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Patient Consent

Possible participants were explained about the study in their local language, and if they agreed to take part and signed the informed consent form, they were recruited into the study.

Trial Registration

CTRI/2023/11/059625.

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