SHORT COMMUNICATION

EFFECT OF SLOW BREATHING TRAINING FOR A MONTH ON BLOOD PRESSURE AND HEART RATE VARIABILITY IN HEALTHY SUBJECTS

Background: Slow and deep breathing leads to an immediate decrease in blood pressure and increase in heart rate variability (HRV). Maximum HRV is reported during breathing at 0.1 Hz frequency (6 breaths/minute). In hypertensive patients, slow breathing at 0.1 Hz frequency has been shown to improve the arterial baroreflex sensitivity (BRS) and decrease the blood pressure.
Aims & Objective: This study was designed to see whether regular practice of slow breathing exercises will bring about changes in the HRV and blood pressure even during natural breathing in healthy participants.
Materials and Methods: Eight healthy participants performed slow breathing exercises at 6 breaths per minute, for 30 minutes daily for 4 weeks. Their respiratory rates, mean heart rate, standard deviation of the normal-to-normal RR intervals (SDNN) and mean arterial pressure (MAP) were compared before and after the 4 weeks of breathing exercise.
Results: The resting MAP decreased significantly from 82.33 ± 3.40 to 79.17 ± 3.64 mm Hg ($P < 0.05$), after the 4 weeks of respiratory training. The respiratory rates of the participants also showed a significant decrease ($P < 0.01$). Although there was an increase in the SDNN during supine rest, it was not statistically significant. Nevertheless, the SDNN during quiet standing increased significantly from 36.63 ± 4.44 to 46.25 ± 4.20 msec ($P < 0.05$). Training did not significantly change the mean heart rate.
Conclusion: This study shows that deep slow breathing training reduces the spontaneous respiratory rate and MAP while increasing the HRV during quiet standing in healthy participants.
Key Words: Slow Breathing Training; Blood Pressure (BP); Respiratory Rate; Heart Rate Variability (HRV)

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INTRODUCTION

Heart rate variability (HRV) is the beat to beat variations occurring in the intervals between consecutive heart beats, due to instantaneous changes in the cardiac autonomic inputs. Respiration alters the autonomic flow to the heart, resulting in RSA mainly by modulating the vagal cardiac outflow. RSA is a major contributor to HRV. HRV is an index of cardiovascular health and declines with age and also in certain pathological conditions that affect the autonomic control of the heart like diabetes, hypertension, congestive cardiac failure and myocardial infarction.[1] A diminished HRV is associated with increased morbidity and mortality.[1] Studies have also shown that RSA and hence the HRV vary with the frequency and depth of breathing, increasing when the breathing rate is reduced voluntarily and reaching a maximum at 6 breaths/minute.[2,3] Studies have also reported that the spontaneous respiratory rate can be decreased by the practice of slow breathing exercises.[4] So, it was hypothesized that if the spontaneous respiratory rate is reduced by slow breathing training, then it would be accompanied by an increase in HRV, which is beneficial.

Sympathetic hyperactivity is one of the main causes of essential hypertension.[5,6] BRS has also been found to be diminished in essential hypertension.[7] Slow breathing at 6 breaths/minute has been shown to improve BRS, and reduce the blood pressure in essential hypertension[8], improve the BRS and oxygen saturation in chronic heart failure patients[4], and reduce the sympathoexcitation found in COPD patients[9]. Routine practice of certain device guided slow breathing exercises have also been shown to decrease the blood pressure in hypertensive patients.[10] Hence this study also explores the effect of slow breathing training at 6 breaths/minute on the MAP of healthy participants.

MATERIALS AND METHODS

Participants

Eight healthy participants, 5 males and 3 females, of age 26.25 ± 3.88 years (mean ± S.D) and body mass index of 24 ± 2.85 kg/m^2 volunteered for the study. They were matched with 6 controls, 4 males and 2 females of age 26.66 ± 4.45 years and body mass index 24.26 ± 4.32 Kg/m^2. All the participants were healthy and non-smokers. None of them were on any medication or had performed any kind of breathing exercises prior to the study. All of them gave written informed consent for the

study. The study was approved by the institutional research committee.

Equipment

ECG and respiration signals were digitized and acquired on to a PC using a data acquisition card (National Instruments, USA) and custom software. Blood pressure was recorded manually using a sphygmomanometer.

Baseline Recording of Parameters

For the baseline recording of parameters, the participants were instructed to refrain from severe physical activity for 24 hours, refrain from smoking and drinking beverages containing caffeine or alcohol for 12 hours prior to the test and come to the laboratory 2-3 hours after a light breakfast. After 20 minutes of quiet supine rest, ECG and respiration were recorded continuously for 5 minutes, from which the mean heart rate, SDNN and respiratory rate (average rate over 5 minutes) during supine rest were calculated. Subsequently, supine blood pressure was recorded twice at an interval of 15 minutes, using a manual sphygmomanometer and the lower value was taken as the baseline value. The participants were then instructed to stand up from the supine position quickly with minimal effort and continue to stand quietly for 5 minutes. After the first 2 minutes, when hemodynamic adjustments to the change in posture would have stabilized, ECG and respiration were recorded for the next 3 minutes of standing from which mean heart rate, SDNN and respiratory rate during quiet standing were calculated.

Slow Breathing Training

The participants were then taught to breathe slowly and deeply at the rate of 6 breaths/minute. Each breathing cycle involved inhalation for 4 seconds and exhalation for 6 seconds, to match the normal breathing cycle. All the participants were able to do the breathing exercises comfortably. They were then asked to perform it daily for half an hour, guided by audio CDs with recorded verbal prompts, at any convenient time of the day, for 4 continuous weeks. The participants were also instructed to maintain a record of their daily breathing practices. Except for occasional lapses, all the participants reported performing the breathing exercises daily. The controls did not do any breathing exercises and followed their usual daily routine. During the study period, all the participants were instructed to continue with their existing level of physical activity and not to start or stop any other exercises.

Reassessment of Parameters

At the end of 4 weeks, the parameters that were measured during the baseline recording were measured again using the same procedure in both the participants and controls.

HRV Analysis

Guidelines of the Task force of the European society of Cardiology and the North American Society of Pacing and Electrophysiology were followed.[1] ECG signals were analyzed using Custom software to detect the R-peaks and compute the RR intervals. HRV analysis software version 1.1 from Biomedical Signal Analysis group, University of Kuopio, Finland was used for further analysis. Mean of all the RR intervals (mean RR) and standard deviation of the normal-to-normal RR intervals (SDNN) were calculated in the time domain. Frequency domain analysis could not be done for the following reason. The spontaneous respiratory rates fell below 9/minute (0.15 Hz) in some of the participants after the training and hence the power due to respiration which is usually in the HF range (0.15 – 0.4 Hz) got transferred to the LF range (0.04 – 0.15 Hz). Therefore, the power spectral analyses before and after the training were not comparable.

Statistical Analysis

Statistical analysis was done using GraphPad Prism 6 software. After confirming the normality of the data using Shapiro Wilk test, Paired 't'- test was used to compare the values obtained before and after the study period. P value of less than 0.05 was considered significant.

RESULTS

The results are presented in Table 1.

Table-1: Comparison of the parameters of the test subjects before and after deep breathing training for 4 weeks

Parameter	Before Training	After Training	P Value
RR – supine rest	16.63 ± 1.22	11.13 ± 1.88*	**0.002**
RR - quiet standing	16.25 ± 1.32	10.75 ± 1.53*	**0.001**
Mean HR- supine rest	64.04 ± 3.07	63.52 ± 2.77	0.779
Mean HR – quiet standing	78.61 ± 3.06	77.46 ± 2.16	0.657
SDNN (ms) – supine rest	59 ± 6.02	62.63 ± 4.95	0.401
SDNN (ms) – quiet standing	36.63 ± 4.44	46.25 ± 4.20*	**0.031**
MAP mm Hg – supine rest	82.33 ± 3.48	79.17 ± 3.64*	**0.049**

All values are mean ± standard error of mean. * Significantly different from the value before training. RR – Respiratory rate; Mean HR – mean heart rate; SDNN – Standard deviation of the normal-to-normal RR intervals; ms – milliseconds; MAP – mean arterial pressure

After the breathing training, the spontaneous respiratory

rates of the subjects decreased significantly during supine rest as well as quiet standing. The MAP of the subjects during supine rest also reduced significantly after the training. While the SDNN during quiet standing increased significantly, the SDNN during supine rest showed an increase, which was not statistically significant. None of the parameters significantly changed in the control group.

DISCUSSION

The most remarkable outcome of the breathing training is the significant decrease in MAP. Such an effect observed in young healthy adults just after 4 weeks of deep breathing training is quite impressive compared to the multiple life-style modifications needed by the hypertensive patients to produce a small reduction in blood pressure.

The significant reduction in the spontaneous respiratory rates of the subjects after the training would have to be accompanied by increase in the tidal volumes to maintain minute ventilation. This would increase the vagal discharge due to activation of the Hering-Breur's inflation reflex. Narkiewicz et al.[11] have reported that in healthy men, muscle sympathetic nerve activity (MSNA) is very much related to spontaneous respiratory rate and lower the respiratory rate of a person, lesser is the MSNA, and hence the central sympathetic outflow. So, the combination of decreased sympathetic outflow and increased vagal discharge accompanying the slower spontaneous respiratory rate is a plausible explanation for the reduction in blood pressure. The study did not reveal a significant change in the mean heart rate. There could be two possibilities for the above. Firstly, the small sample size being inadequate to bring out a difference which existed. Secondly, the decrease in blood pressure could be due to a decrease in sympathetic vasomotor tone alone, a scenario similar to essential hypertension where, in spite of the sympathoexcitation, there is no increase in heart rate, and the elevated blood pressure being attributed to increased peripheral vascular resistance.

After the training, there was a significant increase in the SDNN during standing, though the increase was not significant in the supine position. The reduction in breathing frequency caused by the breathing training allows the respiration to modulate the sympathetic cardiac outflow along with the vagal outflow resulting in increased SDNN. During standing, the increased sympathetic outflow provides more scope for modulation, and therefore, the increase in HRV is more prominent during standing. Further studies in larger populations are required to corroborate these findings and to explore the usefulness of deep breathing exercises in treating pathological conditions with diminished HRV.

Limitations of this Study

The major limitation of this study is the small sample size, which increases the probability of false negative results (beta error). So, we cannot conclude that there was no significant change in the mean heart rate or supine SDNN. However, to identify a significant difference with a small sample size, the difference must be very prominent. Hence, we can safely conclude that the respiratory rate, MAP and the standing SDNN were significantly changed by the breathing training. Another limitation of the study is that, BRS could not be assessed.

CONCLUSION

In conclusion, this study shows that in healthy individuals, the practice of slow breathing exercises at the rate of 6 breaths/min for half an hour daily for 4 weeks, causes a significant reduction in the spontaneous breathing rate and MAP, while increasing the HRV during quiet standing. This opens up a vast array of possibilities of using deep breathing exercise as a tool to correct the states of autonomic dysregulation. Further studies in this domain are undoubtedly warranted.

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