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INFLUENCE OF ALTERNATE NOSTRIL BREATHING ON CARDIORESPIRATORY FUNCTIONS AMONG HYPERTENSIVE SUBJECTS

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ABSTRACT

Objectives: Hypertension is the most common cardiovascular disease. Multiple regulatory mechanisms contribute to the evolution of hypertension. The change of cardio respiratory function in hypertension is associated with increase central sympathetic output to heart and skeletal muscles and peripheral resistance. Alternate nostril breathing (ANB) consists of slow deep breaths using one nostril at a time. ANB can produce different physiological responses and modulate cardio respiratory and autonomic function. Therefore the research was conducted to find out the effect of ANB on cardio respiratory functions. **Methods:** The responses of ANB on cardio respiratory function were investigated in 30 hypertensive subjects. The subjects performed ANB exercise (15min. every day in the morning) for four weeks. Cardio-respiratory parameters were recorded thrice; before ANB, after 15min. (acute exposure) and following 4 weeks of training of ANB. **Results:** Comparison of Mean arterial blood pressure (MABP), HR and RR responses were done using one –way ANOVA. Multiple comparisons were performed using post- hoc analysis- Bonferroni test. The P- value set at 0.01 levels for all the statistical tests. There is significant decrease in Mean arterial blood pressure (MABP), heart rate (HR) and respiratory rate (RR) after ANB in hypertensive subjects. When compared to baseline value ($p<0.01$). **Conclusion:** The study shows that the ANB has a significant effect on cardio respiratory function among hypertensive subjects. Therefore ANB exercise can be prescribed to hypertensive patients as an adjuvant to medical therapy.

Keywords: Hypertension, Alternate nostril breathing (ANB), Mean arterial blood pressure (MABP), Heart rate (HR), Respiratory rate (RR).

INTRODUCTION

Hypertension is the most common cardiovascular disease. It is a major risk factor for the development of coronary artery disease, cerebral vascular disease, peripheral vascular disease, and congestive heart failure.[1] Hypertension commonly begins in young adulthood and occurs in 5 to 10% of people aged 20 to 30 years. The incidence of hypertension continues with age and is found in 20 to 25% of middle aged adults and 50 to 60% of adults over 65 years of age. Males exhibit hypertension earlier than female.[1, 2]

Multiple regulatory mechanisms contribute to the evolution of primary hypertension. (1) Abnormal neuroreflex and sympathetic control of cardiac output (Q) and peripheral resistance,

(2) Abnormal renal and metabolic control of vascular volume and compliance, and (3) Abnormal local smooth muscle and endothelial control of vascular resistace.[1] The pumping effect of the heart is highly controlled by the sympathetic and parasympathetic (vagal) nerves, which abundantly supply the heart. The amount of blood pumped by the heart each minute ie the cardiac output can often be increased more than 100 per cent by sympathetic stimulation. By contrast, it can be decreased more as low as zero or almost zero by vagal (parasympathetic) stimulation.[3]

Hypertensive patients have a significantly higher sympathetic response to central and peripheral stimulation and a significantly lower parasympathetic response when compared to normotensive controls.[4] The evidence has been providing that activation of the sympathetic nervous system [1] is peculiar to the essential hypertension state. [2] Parallel the degree of blood pressure elevation.[5]

Strong sympathetic stimulation can increase the heart rate in adult humans to 180 to 200 and rarely even 250 beats per minutes in young people. Also, sympathetic stimulation increases the force with which the heart muscle contracts, therefore increasing the ejection pressure. When the activity of the sympathetic nervous system is depressed below normal, this decreases both the heart rate and the strength of ventricular contraction, thereby decreasing level of cardiac pumping to as much as 30% below normal. Strong parasympathetic stimulation can decrease the strength of heart contraction by 20 to 30%.[3]

Arterial baroreflex play an important role in the regulation of the cardiovascular system. During spontaneous variation of blood pressure, stimulation or deactivation of the arterial baroreceptors located in the carotid sinus and aortic arch causes reflex bradycardia and tachycardia, respectively at the vascular level, stimulation of the arterial baroreceptors result in sympathetic inhibition and, in consequence, reflex vasodilatation. In-contrast the deactivation of the arterial baroreceptors elicits sympathetic mediated vasoconstriction. All of these responses work in concert to maintain the BP levels in the reference range [6]

A regulatory mechanism of respiratory function is complex process. It involves multiple components of both neural and chemical control and is closely integrated with the cardiovascular system. Multiple factors can alter normal, relaxed, effortless respiration. As with temperature and pulse, any influence that increases the metabolic rate also will increase the respiratory rate.[7]

Hypertension has been well documented as a major risk factor for cardiovascular morbidity; mortality and lowering blood pressure (BP) with antihypertensive drugs which can reduce this risk.[8] The side effects and cost of antihypertensive drugs have stimulated the search for a non-pharmacological approach to control BP either as a first line or adjunctive treatment. Several studies have demonstrated that lifestyle modifications such as physical exercise, salt restriction and weight reduction can lower BP.[8, 9] Relaxation and stress-relieving techniques such as yoga, meditation and biofeedback have also been shown capable of lowering BP.[10]

Sympathetic vasoconstriction will decrease air resistance allowing greater passage of air while parasympathetic vasodilatation will increase nasal resistance and will decrease the airflow. Thus alteration may reach a transition point where air flow may be transiently equal bilaterally.[11] ANB affects brain hemisphere by alternately stimulating the right-brain and then the left-brain. This process is brought about by the action of the air flowing though the nostrils that stimulate the contra-lateral (opposite) side of the brain. It alters cardio respiratory and autonomic parameter.[12]

Slow and regular breathing elicits acutely a number of beneficial effects via the cardiovascular reflex control system, [13, 14] including increased heart rate variability and baroreflex sensitivity, BP reduction [15, 16] and increase of oxygen saturation in chronic heart failure.[17]

The previous study was the influence of ANB on cardio respiratory and autonomic function in healthy young adults. These results suggest that in general there is a tilt towards parasympathetic dominance by ANB. This breathing may be a useful adjunct to medical therapy of hypertension and COPD.[18]

METHODOLOGY

In this experimental study, a total number of 30 hypertensive subjects of both sexes in the age group of 40 to 60 years were taken on the basis of inclusion criteria as follows:

- Hypertensive subjects in age groups of 40 to 60 years (both Male and Female)
- Blood pressure: systolic blood pressure ≥ 140 and diastolic blood pressure ≥ 100

The exclusion criterion is as follows: Renal, heart or liver failure**,** Recent cardiovascular event, Pulmonary disease, Diabetes Mellitus, Neuropathies**,** Alcohol consumption**,** Smokers**,** Use of anti-depressants and anti-arrhythmic

Procedure: Each subject was allowed to participate in the study after he or she satisfied the inclusion criteria and given the written consent. The aim and objective of the study were explained to each of them. Instructions about the procedure were given to each enrolled subject as explained below. The procedure of ANB was explained and each subject was instructed to follow the same as follows.

Alternate Nostril breathing: ANB was done in sitting posture and subject followed the instructions given below. Bring the right hand up to the nose, fold the index and middle fingers so that the right thumb can close the right nostril and the ring finger can close the left nostril, than close right nostril by the right thumb, exhale completely through the left nostril. The exhalation should be controlled and free from exertion and jerkiness.

At the end of the exhalation close the left nostril with ring finger, open the right nostril and inhale slowly and completely. Inhalation should be smooth, controlled and of the same duration as exhalation.

Repeat this cycle of exhalation through the left nostril and inhalation through right nostril, exhale completely through the same nostril keeping the left nostril closed with ring finger.

One exercise consisted of 3 cycles of exhalation through the left nostril and inhalation through the right nostril followed by 3 cycles of exhalation through the right nostril and inhalation through the left nostril and this was repeated for about 15 min.

Experimental protocol: A baseline record of HR, MABP and RR were recorded on first day before starting ANB session. Then the subject was instructed to do ANB for 15 min (acute exposure) and all the variables were again recorded. After this schedule instruction was given to subjects to start practicing ANB (15 min daily) for 4 weeks. Variables were again recorded after completing their 4 weeks training. RR was recorded by palpating the movement of abdominal wall. Systolic and diastolic blood pressure were recorded by using standardize sphygmomanometer and stethoscope from brachial artery at elbow in supine position. Heart rate recorded beat to beat, continuously by non – invasive monitor, pulseoximeter, (L & T, STELLAR).The MABP was obtaining by using this formula:

MABP = DBP + (1/3 SBP-DBP)

RESULTS

Findings show the MABP at baseline was 119.87 ± 5.11, after 15min.118.4 ± 4.9, after 4 wks 115.70 ±5.107. There is significant decrease from baseline to 4 wks on ANOVA, p<0.01.

Findings shows the further multiple comparisons by Bonferroni method, there is significant decrease from baseline to 15min., baseline to after 4 wks and 15min to 4 wks by 1.46, 4.16 and 2.70.

Findings shows the HR at baseline was 80.93 ±5.47, after 15min.77.60 ±4.77, after 4 wks 75.47 ±4.28. There is significant decrease from baseline to 4 wks on ANOVA, p<0.01.

Findings shows the further multiple comparisons by Bonferroni method, there is significant decrease from baseline to 15min., baseline to after 4 wks and 15min to 4 wks by 3.33, 5.46 and 2.13.

Findings shows the RR at baseline was 19.30 ±2.10, after 15min.18.20 ±1.76, after 4 wks 17.23 ±1.54. There is significant decrease from baseline to 4 wks on ANOVA, p<0.01.

Findings shows the further multiple comparisons by Bonferroni method, there is significant decrease from baseline to 15min., baseline to after 4 wks and 15min to 4 wks by 1.10, 2.06 and 0.96.

DISCUSSION

Result of this study shows there is significant reduction in MABP score from baseline to 15min and after 4 wks. The MABP values depend upon SBP and DBP. In patients with hypertension, these changes in BP are correlated with changes in breathing patterns and autonomic function.

It is well known that both the nostril does not take part equally in breathing at a time. One nostril predominate the other and follows a definite cycle. In the breathing right nostril dominance corresponds to activation of sympathetic activity and left nostril correspond to activation of parasympathetic activity.ANB effects the proper balance between sympathetic and parasympathetic activity.

The change of autonomic balance in hypertensive subjects results from an absolute or relative reduction in sympathetic activity or an increase in parasympathetic tone. This cause improved baroreflex sensitivity. This promotes vasodilatation in skeletal musculature and decrease in peripheral resistance due to withdrawal of sympathetic tone. This would lead to less venous return and consequently decrease in MABP

It found that there is a significant reduction in HR score from baseline to 15min and after 4 wks. The individuals differ in relation to their parasympathetic tone and sympathetic activity levels as evident from great variation in resting heart rate from 60 to 80 /min. In patient with hypertension, decrease in HR during ANB is associated with stimulation of the parasympathetic nerves to the heart which causes the hormone acetylcholine to be released at the vagal endings. It decreases the rate and rhythm of the sinus node and decrease the excitability of the AV junctional fibres.

Result of this study shows that mean RR values were found to be lowest after 15min and after 4 wks compared to RR at baseline. The cardiovascular and respiratory system has a close relationship regarding their mechanisms of neural control.

CONCLUSION

The study shows that there is significant effect of ANB on cardio respiratory function among hypertensive subjects. The effects of ANB were improving autonomic balance and respiratory control and decrease MABP, HR and RR in patients with hypertension. Therefore, this simple exercise can be prescribed to hypertensive patients (with proper monitoring) as an adjunct to medical therapy.

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REFERENCES

1. Michael L. Pollock, Donald H. Schmidt. Heart disease and rehabilitation; ed-3rd: 343-353.
2. Gifford, R.W, Kirkendall, W., O' connor, D.T, Weidman, W. AHA scientific council special report: Office evaluation of hypertension. Circulation. 1989; 79: 721-731.
3. Arthur C. Guyton, John E. Hall. Text book of medical physiology; 9th edition.
4. Benjelloun H, Aboudrar S, Jroundi I, Benjelloun- Benani H, Coghlan L, Benomar M. Sympathetic response in primary hypertension. Ann cardiol angeiol (Paris). 2008 Jul 1
5. Grassi, Guido 1, 2. Role of the sympathetic nervous system in human hypertension. Journal of Hypertension. Dec. 1998; 16 (12) supplement: 1979-1987.
6. Chapleau MW, Li Z, Meyrelles SS, Xiuying MA, Abboud FM. Mechanism determining sensitivity of baroreceptors afferent in health and disease. Ann N Acad Sci. 2001; 940: 1-19.
7. Susan B. O'Sllivan, Thomas J. Schmitz. Physical Rehabilitation: Assessment and Treatment; ed- 4th: 71-97.
8. The sixth report of the Joint National Committee on prevention, detection, and treatment of high blood pressure. Arch Intern Med. 1997; 157: 2413-2446.
9. Nonpharmacological approaches to the control of high blood pressure. Final report of the subcommittee on Nonpharmacological Therapy of the 1984 Joint National Committee on Detection, Evaluation, and Treatment of high blood pressure. Hypertension. 1986; 8: 444-467.
10. Fahrion SL. Hypertension and biofeedback. Prim Care 1991; 18: 663-682.
11. Keuning J. On the nasal cycle. Int Rhinol. 1968; 99-139.
12. Stancak A. Jr, Kuna M. EEG changes during forced alternate nostril breathing. Int J Psychophysiol. 1994 Oct; 18(1): 75-9.
13. Daly M de B. Interactins between respiration and circulation. In: Fishman AP (ed). Handbook of Physiology. American Phsiology Society: Bethesda, 1986; pp 529-594.
14. Daly M de B. Aspects of the integration of the respiratory and cardiovascular system. In: Jordan D, Marshall J (Eds). Cardiovascular Regulation. Portland Press: Londan, 1995; pp 15-35.
15. Pitzalis MV et al. Effect of respiratory rate on the relationships between RR interval and systolic blood pressure fluctuations: a frequency-dependent phenomenon. Cardiovascular Res. 1998; 38: 332-339.
16. Novak V, Novak P, de Champlain J, Nadeau R. Altered cardio respiratory transfer in hypertension. Hypertension. 1994; 23: 104-113.
17. Bernardi L et al. Effect of breathing rate on oxygen saturation and exercise performance in chronic heart failure. Lancet 1998; 351: 1308-1311.
18. Srivastav RD, Jain N, Singhal A. Influence of alternate nostril breathing on cardio respiratory and autonomic function in healthy young adults. Indian J Physiol Pharmacology. 2005; 49: 475-83.