Indian J Physiol Pharmacol 2005; 49 (4) : 475–483

INFLUENCE OF ALTERNATE NOSTRIL BREATHING ON CARDIORESPIRATORY AND AUTONOMIC FUNCTIONS IN HEALTHY YOUNG ADULTS†

R. D. SRIVASTAVA*, NIDHI JAIN* AND ANIL SINGHAL**

**Departments of Physiology and **Neurosciences & Holistic Medicine,*
Himalayan Institute of Medical Sciences,
Swami Ram Nagar, Doiwala,
Dehradun – 248 140

(Received on February 2, 2005)

Abstract : Alternate nostril breathing (ANB) may modulate cardio-respiratory and autonomic functions. However, the studies are scarce and results highly conflicting. The present study was conducted in healthy young volunteers comprising of males (n=20) and females (n=20) in range of 17–22 years. In both groups respiratory rate (RR/min), heart rate (HR/min), systolic blood pressure (SBP; mm Hg), diastolic blood pressure (DBP; mm Hg), peak expiratory flow rate (PEFR; L/min) and galvanic skin resistance (GSR; μV) were recorded thrice; once as control and then after 15 min (acute exposure) and following 8 wks of training in ANB (15 min daily).

In males the control RR was 16.60 ± 2.01, HR 75.75 ± 11.07, SBP-115.9 ± 7.33, DBP 70.4 ± 6.28 and PEFR 550.00 ± 51.50. After 15 min of ANB – RR (14.75 ± 1.41, $P<0.001$), HR (68.45 ± 12.41, $P<0.01$) and SBP (113.6 ± 6.04, $P<0.05$) fell significantly. After 8 wks of ANB training RR (12.35 ± 1.35, $P<0.0001$), HR (63.20 ± 11.11, $P<0.001$), SBP (109.5 ± 5.61, $P<0.001$), declined to much greater extent and PEFR (571.50 ± 46.26, $P<0.01$) rose significantly.

In females the control RR was 17.25 ± 1.89, HR-74.90 ± 12.85, SBP-106.70 ± 6.91, DBP-68.70 ± 5.52 and PEFR-394.50 ± 44.89. After 15 min of ANB RR (15.05 ± 1.54, $P<0.001$) and HR (64.75 ± 9.80, $P<0.001$) showed significant decline with concomitant rise in PEFR (407.00 ± 2.31, $P<0.05$). Following 8 wks training the decrement in RR (12.60 ± 1.50, $P<0.0001$) and HR (63.30 ± 8.65, $P<0.001$) was maintained. SBP (103.10 ± 4.92, $P<0.001$) and DBP (65.8 ± 5.54, $P<0.001$) decreased further and PEFR (421.00 ± 38.51 $P<0.001$) rose, GSR was unaffected by ANB in both males and females.

These results suggest that in general there is a tilt towards parasympathetic dominance by alternate nostril breathing. This breathing may be a useful adjuvant to medical therapy of hypertension and COPD.

Key words : yoga alternate nostril breathing blood pressure PEFR GSR

*Corresponding Author and Present Address : Department of Physiology, SRMS Institute of Medical Sciences, 13.2 Km, Bareilly-Nainital Road, Bhojipura, Bareilly – 243 202; E-mail : rdsrivastava@rediffmail.com

†The paper was presented at Joint International Conference International Society for Heart Research (Indian Section) & International Academy of Cardiovascular Sciences, held at K.G. Medical College, Lucknow, Jan. 9–11, 2004.

INTRODUCTION

Empirical observations by ancient sages had already established that both nostrils do not work simultaneously, but alternately in a cyclic manner. According to yoga, this phenomenon is a consequence of the alternation of the flow of subtle energy in the *Ida* and *Pingala* (1). Presumably, this fact was incorporated in development of pranayama technique where one deliberately practices these breathing patterns. Kayser (2) defined nasal cycle as a phenomenon of the alternating congestion, decongestion response of erectile tissue of nasal turbinate and septum of two nostrils, which effectively altered the unilateral nasal resistance and was existent on account of prevailing sympathetic or parasympathetic tone. Sympathetic vasoconstriction will decrease air resistance allowing greater passage of air while parasympathetic vasodilatation will increase nasal resistance and will decrease the airflow. Thus, alternation may reach a transition point where air flow may be transiently equal bilaterally. The observation gains strength from the fact that in Horner's syndrome nasal cycle is absent on denervated side (3, 4). Using mask flow technique of active posterior rhinometry and other techniques, the percent incidence of nostril dominance was 72% (5) to 80% (6, 7) and cycle length has been variously described as ranging from 30 minutes (6) to 2-7 hours (3).

Alternate nostril breathing (ANB) is an integral part of pranayama. It has been known to alter cardio respiratory and autonomic parameters. However the reports on influence of ANB on heart rate (8, 9), systolic and diastolic blood pressure (8, 9) and galvanic skin resistance (8, 10) are very conflicting with investigators describing either a decline, increment or no change in individual variables.

The present study was, therefore, undertaken to investigate the effects of alternate nostril breathing on cardio-respiratory and autonomic parameters in young healthy medical students.

MATERIAL AND METHODS

Subject Population : The present study was conducted on 40 healthy medical student volunteers of first year MBBS of both sexes. These comprised of 20 males and 20 females in age range of 17–22 years. The experimental protocol was explained to them and written consent obtained. All the procedures were non-invasive and the study plan was approved by the Ethics Committee of Himalayan Institute of Medical Sciences. All the subjects were healthy and free from any cardio-respiratory ailments and were not on any medication. Smokers were excluded from the study. The subjects were of same socio-economic and nutritional status as they hailed from upper middle class and upper class of society sharing common hostel accommodation and food.

Experimental Protocol : Three students (males or females) reported daily in the laboratory. Height, weight, age, dietary habits were recorded and body surface area (BSA) calculated by 'Dubois Nomogram' (11). A baseline record (which also served as control) of respiratory rate (RR/min), heart rate (RR/min), systolic blood pressure (SBP; mm Hg), diastolic blood pressure (DBP; mm Hg), peak expiratory flow rate (PEFR; L/min) and galvanic skin resistance (GSR; μv) was obtained. Then they practiced ANB for 15 min (acute exposure) and all the variables were again recorded. By end of two wks all 20 males and 20 females had their control and acute effects (15 min) recorded. After this schedule all 20 males and 20 females started practicing ANB (15 min daily) for 8 wks. Variables were again recorded after completing their 8 wks training (following 15 min ANB). BP, ECG and GSR were recorded as quickly as possible followed by other variables. On an average all the variables were recorded within 15 min of cessation of ANB. Respiratory Rate was recorded by observing the movement of abdominal wall in lying down position. Heart Rate was calculated from R-R interval of EGG in lead II. From 20 complexes of Lead II, four R-R intervals were sampled at three places. Mean of these 12 R-R intervals was calculated. Electrocardiogram–This was recorded by ECG Machine (T-108, BPL, Banglore) in lying down position. Blood pressure was recorded by digital BP monitor AND model, No. DA-767 (Vatsalya trading Co., Dehradun) in lying down position. Peak Expiratory Flow Rate recorded by Wright's peak flow meter (INCO, Ambala) in standing position. Best of the three values was noted. Galvanic Skin Resistance was recorded by EDG machine, J & J model No. T-68 (Medicaid system, Chandigarh) in lying down position in air conditioned room by tying electrodes on the index and ring finger of right hand.

Alternate Nostril Breathing (1)

This was done in sitting posture and subject followed the instructions given below.

- Sit in a calm, quiet, airy place in an easy and steady posture with the head, neck and trunk erect and in a straight line and to keep the body still.

- Bring the right hand upto 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 (Vishnu Mudra).

- With the right nostril closed 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 the 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.

- At the end of this exhalation close the right nostril and inhale through the left nostril and repeat this for two more times. In summary, 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.

Analysis of data : Mean and standard deviation (±SD) of all observations were calculated and comparisons were done between values of control (mean and ±SD) with that of 15 min (acute exposure) and control with 8 wks training by applying paired Student's 't' test. Statistical significance was assigned at P<0.05.

RESULTS

The results are summarized in Tables I and II. In the males, the height was – 173.5 ± 6.2 cm, weight – 62.9 ± 7.4 kg and BSA 1.6 ± 0.1 m^2 and in the females the height was 157.5 ± 4.5 cm, weight 51.5 ± 6.3 kg and BSA 1.4 ± 0.1 m^2.

In males, after 15 min of alternate nostril breathing RR (P<0.01) and SBP (P<0.05) fell significantly, DBP, PEFR and GSR showed insignificant change. After 8 wks of training RR (P<0.0001), HR (P<0.001) and SBP (P<0.0001) declined to much greater extent and PEFR rose significantly (P<0.01), (Table-I).

In females after 15 min-RR (P<0.0001) and HR (P<0.001) showed significant decline with concomitant rise in PEFR; DBP and GSR did not change significantly. Following 8 wks training the decrement in RR (P<0.0001) and HR (P<0.0001) was maintained, SBP (P<0.01) and DBP (P<0.001) decreased further and PEFR rose (P<0.001) while GSR was again unaltered (Table II).

TABLE I: Effect of ANB on different variables in males (n=20).

Parameters	*HR/min*	*SBP mm Hg*	*DBP mm Hg*	*RR/min*	*PEFR L/min*	*GSR µV*
Control	75.75±11.07	115.90±7.33	70.4±6.28	16.6±2.01	550.00±51.50	12.51±5.73
After 15 min	68.45±12.41**	113.60±6.04*	70.80±6.30†	14.75±1.41***	547.50±43.99†	12.69±3.80†
After 8 wks	63.20±11.11***	109.50±5.61****	68.00±6.19†	12.35±1.35****	571.50±46.26**	13.05±5.54†

Values are expressed as mean ± SD.
*P<0.05, **P<0.01, ***P<0.001, ****P<0.0001, †P>0.05.
P value is comparison of control with after 15 min and control with after 8 wks.

TABLE II: Effect of ANB on different variables in females (n=20).

Parameters	*HR/min*	*SBP mm Hg*	*DBP mm Hg*	*RR/min*	*PEFR L/min*	*GSR µV*
Control	74.90±12.85	106.70±6.91	68.70±5.52	17.25±1.89	394.50±44.89	10.82±6.37
After 15 min	64.75±9.80***	106.20±5.39†	68.30±5.24†	15.05±1.54****	407.00±42.31*	10.86±5.82†
After 8 wks	63.20±8.65***	103.10±4.92**	65.80±5.54	12.60±1.50****	421.00±38.51***	10.80±6.33†

Values are expressed as mean ± SD.
*P<0.05, **P<0.01, ***P<0.001, ****P<0.0001, †P>0.05.
P value is comparison of control with after 15 min and control with after 8 wks.

DISCUSSION

The results of ANB could also be compared to reports available on pranayam as both are fairly similar. We found that there was a significant reduction in HR and SBP both after acute and 8 wks ANB in both sexes and significant reduction in DBP only after 8 wks in females. The earlier studies have described no change (12), decline (8, 9, 13–16) and increment (17) in HR. Our observations are thus in conformity with few (8, 9, 13–16) and contrary to others (17). The individuals differ in relation to their parasympathetic (vagal) tone and sympathetic activity levels as evident from great variation in resting heart rate from 60–90 /min (18). The HR responses will therefore depend on the algebraic summation of sympathetic and parasympathetic activation further modulated by hypocapnoea and lung stretch receptor responses (19, 20). Both tachycardia (20, 21) and bradycardia (22, 23) occur following pulmoflation which would account for bradycardia responses of ANB in our study.

The decline in SBP both in males and females and DBP in females compares well with few earlier studies (8, 10, 14, 24) but contradicted by another who observed no change in this parameter (25). SBP is determined by venous return and sympathetic and parasympathetic drive. Pulmoflation produces wide spread vasodilatation in skeletal muscle vasculature due to withdrawal of sympathetic tone (26). This would lead to less venous return and consequently SBP declined in both sexes.

The significant decline in DBP following 8 wks in females could be explained by following mechanisms – a) DBP depends on peripheral resistance and lung inflation has been known to decrease systemic vascular resistance (27). This response is initiated by pulmonary stretch receptors which bring about withdrawal of sympathetic tone in skeletal muscle blood vessels leading to wide spread vasodilatation thus decreasing peripheral resistance (26). b) Female hormone oestrogen lowers plasma cholesterol significantly and produces a rapid vasodilation by increasing the local production of Endothelium Derived Relaxing Factor (EDRF) i.e. Nitric Oxide (NO; 28). When flow to tissue is suddenly increased by arteriolar dilatation, the large arteries to the tissues also dilate, this flow induced dilation is due to local release of NO (29). Hypocholesterolemia delays onset and severity of atheroslerosis and hence relaxing effects are augmented. The absence of oestrogen in males renders them more prone to atherosclerosis which denudes the endothelium and reduces production of EDRF (30). Further, atherosclerotic vessels respond more vigorously to vasoconstrictor NE released at sympathetic efferent nervendings. These variables will eventually produce less vasodilatation in the males as compared to females, this explains absence of decline in DBP in males. The DBP did not change in females following an acute exposure of ANB (15 min) presumably because the vasodilator effect of lung inflation was balanced by sympathetic and parasympathetic interactions of ANB.

The dissimilarities between our observations on HR, SBP and DBP with those of others could emanate from the fact that others have not studied the effect of ANB per-se but only as a part of many variables. Further, the time duration of exposure was also at variance, for example our 8 wks versus immediate (12). In addition our subject population was more homogeneous and from a very narrow age range.

We observed decrement in RR and rise in PEFR in both sexes. The decline in RR in the present study is substantiated by certain workers (31–34) but contradicted by other authors who found no change in RR (10, 24) The decline in RR in our study can be explained by the influence of a probable hypocapnoea on medullary respiratory center (35) and persistent voluntary effort of breathing (ANB), producing inhibition of rhythmic spontaneous breathing by a phenomenon akin to over drive suppression.

The control PEFR value of 550 L/min in males (Ht 173.3 cms) is surprisingly same as 550 L/min (mean age 22 yrs, Ht 171.6 cms) and close to 587 L/min (mean age 18.3 yrs) in healthy medical students described earlier (36, 37), similarly in females the control PEFR 394 L/min (Ht 157.5 cms) is again in the region of 320–470 L/min for young females (38). Akin to our observation of increases in PEFR in both sexes, others have also reported rise in PEFR (10, 33, 39) and FEV_1 (32, 40) while some observed no change in PEFR (10). In our study PEFR rose significantly only after 8 wks because by that time thoraco-pulmonary compliance had risen well above basal level due to training in ANB. Stimulation of pulmonary stretch receptors by inflation of the lung reflexly relaxes smooth muscles of larynx and tracheobronchial tree probably this modulates airway caliber and reduces airway resistance (41).

Previous authors have reported that yogic type exercise will increase PEFR through following changes in respiratory dynamics (39). 1) Increased respiratory muscle strength by breathing exercise 2) cleansing of airways secretions, 3) yogic breathing exercise strain practitioners to use the diaphragmatic and abdominal muscles more efficiently, thereby emptying and filling the respiratory apparatus more efficiently and completely, 4) the relaxing effect of yoga inhibiting the constrictor tone to bronchial smooth muscles.

Earlier investigators have suggested that pranayam kind of breathing will enhance muscles efficiency and compliance by reducing elastic and viscous resistance of lung present during inspiration (40) and by offering physiological stimulation for release of lung surfactant and prostaglandins into alveolar spaces which increase lung compliance (33).

Unaltered GSR of our study is in agreement with reports describing no change in GSR with ANB (8, 10). Presumably the right and left nostril alternation cancel each others effect.

Although we have analysed the data for acute exposure also due to lack of comparative studies these results will not be discussed in detail. While changes in different parameters by acute exposure of ANB could be attributed to a balance between sympathetic and parasympathetic activation and certain amount of hypocapnea which one expects once the breathing becomes voluntary and greater respiratory muscle efforts needed to inspire and expire through a single nostril, the changes following training could presumably occur from greater muscular efficiency and compliance of the thoracic wall and lung interacting with sympathetic and parasympathetic activating levels. It will be illogical to explain the results purely from viewpoint of sympathetic and parasympathetic activation per-se. The future studies involving alternate nostril breathing must take into cognizance, the tremendous variations in result reported by other workers and present study and it would necessitate monitoring of – a) the difference in eupnoeic tidal volume, and tidal volume during alternate nostril breathing, b) measurement of circulating catecholamines with this breathing pattern, c) analysis of blood gases for PO_2 and PCO_2 measurement during this maneuver, d) sympathetic and parasympathetic *in-vivo* action potential recording from thoracic sympathetic ganglia and thoracic vagus as GSR is only an indirect method of assessing sympathetic activation and in our study it was totally inconclusive.

The general decrement in RR, HR, SBP and DBP by ANB in both groups of males and females is in concert with earlier proposition that these exercises tilt the autonomic balance to parasympathetic dominance.

Conclusion

In the present study, both in males and females, the two outstanding effects of alternate nostril breathing were — a) there was fall in blood pressure and b) rise in PEFR. Therefore, this simple exercise can be prescribed to hypertensive patients as an adjuvant to medical therapy. This practice attended by rise in PEFR obviously offers an increment in cardio-respiratory efficiency, and may be useful in COPD patients also.

ACKNOWLEDGEMENTS

Authors are grateful to Himalyan Institute of Medical Sciences for providing facilities to conduct this study. Thanks are also due to SRMS-IMS Bareilly, and Medical Director Prof. S.D. Pandey for the help in preparation of manuscript. Secretarial assistance by Shashi Kant Srivastava and Ram Mohan Prajapati is also appreciated.

REFERENCES

1. Swami Rama, Ballentine R, Hymes A. The Sciences of Prana: Science of breath : A practical guide. Himalayan International Institute of Yoga Science and Philosophy of the USA. *Honesdale, Pennsylvania* 1979; 91–115.

2. Kayser R. 1895. Cited by : Hasegawa M, Kern EB. The Human Nasal Cycle. *Mayo Clin Proc* 1977; 52: 28–34.

3. Keuning J. On the nasal cycle. *Int Rhinol* 1968; 99–136.

4. Eccles R, Maynard RL. Studies on the nasal cycle in the immobilized pig. Proceeding of the Physiological Society Middlesex Hospital Meeting 1975; 1–2, 17–18.

5. Hasegawa M, Kern EB. The Human Nasal Cycle. *Mayo Clin Proc* 1977; 52: 28–34.

6. Heetderks DR. Observations on the reaction of normal nasal mucous membrane. *Am J Med Sci* 1927; 174: 231–244.

7. Williams HL. A reconsideration of the relation of

the mechanics of nasal airflow to the function of the nose in respiration. *Rhinology* 1972; 10: 145–161.

8. Bhargava R, Gogate MG, Mascarenhas JF. Automatic responses to breath holding and its variations following Pranayam. *Indian J Physiol Pharmacol* 1988; 32: 257–264.

9. Khanam AA, Sachdeva U, Guleria R, Deepak KK. Study of pulmonary and autonomic functions of asthma patients after yoga training. *Indian J Physiol Pharmacol* 1996; 40: 318–324.

10. Telles S, Nagarathna R, Nagendra HR. Breathing through a particular nostril can alter metabolism and autonomic activities. *Indian J Physiol Pharmacol* 1994; 38: 133–137.

11. Dubois D, Dubois DF. A formula to estimate the approximate surface area if height and weight be known. *Arch Int Med* 1916; 17: 863–871.

12. Raghuraj P, Ramakrishan AG, Nagendra HR, Telles S. Effect of two selected yogic breathing techniques on heart rate variability. *Indian J Physiol Pharmacol* 1998; 42: 467–472.

13. Udupa KN, Singh RH, Settiwar RM. Studies on the effect of some yogic breathing exercises (Pranayams) in normal persons. *Indian J Med Res* 1975; 63: 1062–1065.

14. Joseph S, Sridharan K, Patil SKB, Kumaria ML, Selvamurthy W, Joseph NT et al. Study of some physiological and biochemical parameters in subjects undergoing yogic training. *Indian J Med Res* 1981; 74: 120–124.

15. Selvamurthy W, Nayar HS, Joseph NT, Joseph S. Physiological effects of yogic practice. *NIMHANS Journal* 1983; 1: 71–80.

16. Bera TK, Gore MM, Oak JP. Recovery from stress in two different postures and in Shavasana – A yogic relaxation posture. *Indian J Physiol Pharmacol* 1998; 42: 473–478.

17. Madan Mohan, Saravanane C, Surange SG, Thombre DP, Chakrabarty AS. Effect of yoga type breathing on heart rate and cardiac axis of normal subject. *Indian J Physiol Pharmacol* 1986; 30: 334–340.

18. Ganong WF. Cardiovascular regulatory mechanism. In : Review of Medical Physiology. Edition 20, McGraw-Hill, New York 2001; 579–580.

19. Kontos HA, Mauck HP Jr., Richardson DW, Patterson JL Jr. Circulatory responses to hypocapnoea in anaesthetised dog. *Am J Physiol* 1965; 208: 139.

20. Hayashi KD. Responses of systemic arterial pressure and heart rate to increased intra pulmonary pressure in anaesthetized dog. *Proc Soc Exptl Biol Med* 1969; 131: 426.

21. Tandon HC, Varma P, Vyas S, Rizvi SAH, Pandey RC, Srivastava RD, Mishra RK, Mishra MLB. Evidence for arrhythmolytic effects of lung inflation in anaesthetized coronary occluded dogs. *Gems Int J Cl Res* 1988; 1(3): 91–95.

22. Salisbury PF, Galletti PM, Lewin RJ, Reiben PA. Stretch reflexes from the dog's lung to the systemic circulation. *Circulation Res* 1959; 5: 62.

23. Glick G, Wechsler AS, Epstein SE. Reflex cardiovascular depression produced by stimulation of pulmonary stretch receptors in the dog. *J Clin Invest* 1969; 48: 467.

24. Udupa KN, Singh RH, Settiwar RM. Physiological and Biochemical studies on the effect of yogic and certain other exercises. *Indian J Med Res* 1975; 63: 620–624.

25. Udupa KN, Singh RH, Settiwar RM. A comparative study on the effect of some individual yogic practices in normal persons. *Indian J Med Res* 1975; 63: 1066–1071.

26. Daly M, De B, Robinson BH. An analysis of the reflex systemic vasodilator response elicited by lung inflation in dog. *J Physiol London* 1968; 195: 387.

27. Hainsworth R. Circulatory responses from lung inflation in anaesthetised dogs. *Am J Physiol* 1974; 226: 247.

28. Ganong WF. The gonads : Development & function of the reproductive system. Ed Ganong WF In : Review of Medical Physiology edition 21, *McGraw Hill New- Delhi* 2003 P 446.

29. Ganong WF. Cardiovascular regulatory mechanism, chapter 31 Ed Ganong WF. In Review of Medical Physiology edt. 21, *McGraw Hill, New-Delhi* PP 600–601.

30. Thomas G, Ram well PW. Nitric Oxide donors and inhibitors. Ed Katzung BG In : Basic and Clinical Pharmacology, edt 9th, *McGraw-Hill, New-Delhi* 2004, P 317.

31. Madan Mohan, Rai DC, Balavittal V, Thombre DP, Swami Gitananda. Cardio-respiratory changes during Savitri Pranayam and Shavasan. *The Yoga Review* 1983; 111: 25–34.

32. Makwana K, Khirwadkar N, Gupta HC. Effect of short term yoga practice on ventilatory function tests. *Indian J Physiol Pharmacol* 1988; 32: 202–208.

33. Joshi LN, Joshi VD, Gokhale LV. Effect of short term pranayam practice on breathing rate and, ventilatory functions of lung. *Indian J Physiol Pharmacol* 1992; 36: 105–108.

34. Bharshankar JR, Bharshankar RN, Deshpandey VN, Kaore SB, Gosavi GB. Effect of yoga on cardio-vascular system in subject above 40 years. *Indian J Physiol Pharmacol* 2003; 47: 202–206.

35. Keele CA, Neil Erik, Joel Noman. Part III. The chemical regulation of respiration. In : Keele CA, Neil Erik, Joel Noman eds. Samson Wright's applied physiology. edt 13th, *Oxford University Press, New Delhi* 2003; p. 209.

36. Agarwal MJ, Jain DS, Dikshit MB, Raje S. PEFR data on healthy young male medical students. Unpublished observations. *IJ PP* 2005; 49:(1) 8–18.

37. Dikshit MB, Prasad BAK, Jog NV. Peak expiratory flow rates in elderly Indians. *Indian J Physiol Pharmacol* 1991; 35: 39–43.

38. Singh HD, Peri S. Peak expiratory flow rate in South Indian adults. *Indian J Physiol Pharmacol* 1979; 23: 315–320.

39. Yadav RK, Das S. Effect of yogic practice on pulmonary functions in young females. *Indian J Physiol Pharmacol* 2001; 45: 493–496.

40. Gopal KS, Anantharaman V, Balachander S, Nishith SD. The cardio-respiratory adjustments in 'Pranayam' with and without 'Bandhas' in 'Vajrasana'. *The Indian Journal of Medical Sciences* 1973; 27: 686–692.

41. Keele CA, Neil Erik, Joel Norman. Role of Pulmonary stretch receptors. In : Keele CA, Neil Erik, Joel Norman eds Samson Wright's applied physiology, edt 13th. *Oxford University Press New Delhi* 2003; pp. 170–171.