ISSN Print: 2394-7500
ISSN Online: 2394-5869
Impact Factor: 5.2
IJAR 2018; 4(3): 458-461
www.allresearchjournal.com
Received: 14-01-2018
Accepted: 15-02-2018

Arwa Hakim Sinnarwala
Physiotherapy Intern, P.E.S Modern College of Physiotherapy, Shivajinagar, Pune, Maharashtra, India

Dr. Gauri M Afle
Associate Professor and Head of Department of Cardio Respiratory Department PES Modern College of Physiotherapy, Shivajinagar, Pune, Maharashtra, India

Correspondence
Arwa Hakim Sinnarwala
Physiotherapy Intern, PES Modern College of Physiotherapy, Shivajinagar, Pune, Maharashtra, India

To study the effect of self guided breathing using mobile application on blood pressure in soft ware engineers with pre hypertension: A pilot study

Arwa Hakim Sinnarwala and Dr. Gauri M Afle

Abstract
Background & purpose: Software engineers face a lot of stress due to job deadlines. This leads them to have prehypertension which is a precursor of clinical hypertension. Thus the purpose of this study is to study the effect of self guided breathing using mobile application on blood pressure.
Materials and Methodology: 30 patients were selected with prehypertension. They were explained the use of the application and then asked to breathe in pace with the ques of the application for 2 minutes. SBP and DBP was measured pre and post breathing technique using digital sphygmomanometer.
Results: There was significant reduction in both systolic and diastolic blood pressure after breathing technique. Statistical analysis showed mean value of SBP pre treatment and post treatment was 129.6±5.768 and 114.8±6.552 respectively. ($p<0.0001$, $t=16.703$). While mean value of DBP pre treatment and post treatment was 81.9±5.846 and 74.13±4.732 respectively. ($p<0.0013$, $t=8.230$).
Conclusion: This study concludes that self guided breathing using mobile application is effective to reduce both systolic and diastolic blood pressure in software engineers with prehypertension.

Keywords: self guided breathing, prehypertension, software engineers

Introduction
Job stress is defined as "A set of psychosocial factors experienced by workers due to work conditions, generated as composite experiences at different levels within an organization." Among environmental factors, job stress is an important determinant of hypertension and is well-studied in developed countries [1]. It is very important to understand the role of stress at the organizational level and understand how this relates to hypertension in low and middle-income countries (LMIC) such as India. Public health researchers in these countries need to recognize the window of opportunity to address factors that can reduce CVD risk at workplaces [2, 3, 4].

Hypertension is a major contributor to the world-wide epidemic of cardiovascular disease (CVD) [5]. It is estimated that hypertension causes 7.5 million deaths world-wide amounting to 12.8% of the total of all annual deaths [6]. Many risk factors leading to hypertension are modifiable and therefore provide an opportunity for preventive efforts [7]. Hence any intervention that can successfully prevent or reduce hypertension should be viewed as promoting cardiovascular health of individuals [8].

Blood pressure is the lateral pressure exerted by the contained column of blood on the wall of the arteries. The pressure is exerted when the blood flows through the arteries. The term 'blood pressure' refers to arterial blood pressure. Arterial blood pressure is expressed in four different terms:

Systolic blood pressure, Diastolic blood pressure, Pulse pressure, Mean arterial pressure [9]
2017 High Blood Pressure Clinical Practice Guideline [10].

Categories of BP in Adults* BP

BP Category	SBP		DBP
Normal	<120 mm Hg	And	<80 mm Hg
Elevated or Prehypertensive	120-129mmhg	And	<80 mm Hg
Hypertension			
Stage 1	130-139 mm Hg	Or	80-89 mm Hg
Stage 2	≥140 mm Hg	Or	≥90 mm Hg

*Individuals with SBP and DBP in 2 categories should be designated to the higher BP category. BP indicates blood pressure (based on an average of ≥2 careful readings obtained on ≥2 occasions, as detailed in Section 4); DBP, diastolic blood pressure; and SBP systolic blood pressure.

Vasan and colleagues [11] found that the conversion rate of prehypertension to hypertension over 4 years was 30% Slow and deep breathing ("paced breathing") has been traditionally associated with meditation and healing for hundreds of years [12]. Paced breathing plays a prominent role in behavioral methods of treating hypertension that have reported some short-term successes [13-15]. However, asking patients to perform paced breathing sessions on their own may be impractical. Apart from the prolonged training, practicing, skill, and motivation needed, effortlessly performed paced breathing involves individualized breathing patterns that typically require personal coaching. The physiological origin of the hypotensive effects of paced breathing has traditionally been attributed to the "relaxation response" [13]. While relaxation is a widely accepted, beneficial outcome of slow breathing, the antihypertensive effect of paced breathing may have a more direct physiological origin.

"Paced breathing; mobile application "Is a utility to let you improve your breathing technique. You simply set your inhale, exhale, and hold times to whatever you want, and this app will give you ques of when to start inhaling and exhaling [16].

Based on the available literature [17-22], self-guided breathing appears to be useful for prehypertensives who might benefit from reducing stress and sympathetic activity. Patients should be instructed to use the application routinely in daily 15-minute sessions, aiming to accumulate at least 45 minutes of slow breathing per week; the device can display the cumulative duration of slow breathing to assist in achieving this goal.

Slow breathing (< 10 bpm), especially with prolonged exhalation, appears to reduce sympathetic nerve traffic and thus causes arteriolar dilatation. The process is believed to be initiated by activated pulmonary mechanoreceptors, which respond to the increased tidal volume that accompanies slow breathing, and act in concert with cardiac mechanoreceptors to inhibit sympathetic outflow [23-24].

Methods

In this study 30 software engineers both male and female aged between 20 to 40 years were recruited by convenient sampling.

Subjects were included in this study if they aged between 20 to 40 years (both genders) and were diagnosed Prehypertensive software engineers (120-139mmHg/80-89mmHg) along with one year of working experience. Subjects were excluded if they were hypertensive individuals on hypertensive medications, Any recent ECG evidence of myocardial infarction, Any h/o of MI, Congenital heart disease stroke, or working for less than one year.

From the subjects demographic data was collected. Systolic and Diastolic blood pressure was taken using validated automatic oscillometric blood pressure device (ICC = 0.80) before and after intervention.

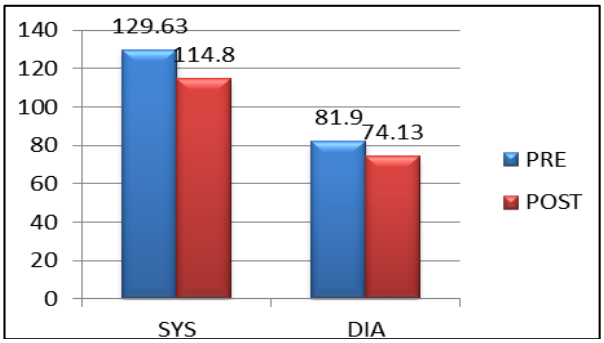
Blood pressure measurement: Subject was made comfortable and allowed 5 min of rest. SBPs and DBPs and HR was recorded by a validated automatic oscillometric device which was placed on the left arm of the patients. In the beginning of the session, three measures were made, but only the second and third measurements were averaged. During the baseline, respiratory and recovery periods, the measurements were read by the minute.

Breathing technique: Subject was explained the use of the application. Subject was asked to breathe normally for one min. Normal inhalation and exhalation time for him/her was noted. According to which Inhalation and exhalation timing was set in the application. Along with it breath per min was set with it.

Findings

Data Analysis and result: Statistical analysis was performed by using Graphpad instat 3 software. Paired t test was used to analyze systolic and diastolic blood pressure and the effectiveness of self guided breathing on it.

Outcome Measure	Training	Mean ±Sd	Paired t Value	Significant p Value	Mean Difference
Systolic BP	Pre RX	129.6±5.768	16.703	0.0001	14.833
	Post RX	114.8±6.552			
Diastolic BP	Pre RX	81.9±5.846	8.230	0.0013	7.833
	Post RX	74.13±4.732			



Results

There is extremely statistical significance (p<0.0001) between pre and post value of systolic blood pressure. The systolic blood pressure reduced post intervention which shows effectiveness of self guided breathing on blood pressure.

Also there statistical significance (p<0.0013) between pre and post value of diastolic blood pressure. The diastolic blood pressure reduced post intervention which shows effectiveness of self guided breathing on blood pressure.

Discussion

The aim of the present study was to determine whether slow breathing training results in a modification of central neural pathways controlling blood pressure.

Hence this study found out that self guided breathing using mobile application helped software engineers with pre hypertension to reduce their systolic and diastolic blood pressure.

The baroreceptor reflex (baroreflex) is a negative feedback mechanism involving stretch receptors, present primarily in the aortic arch and carotid sinuses, that monitor arterial blood pressure and respond to acute changes *via* central–neural–autonomic pathways, The baroreflex is therefore tightly coupled to, perhaps even predominantly accountable for, LF HRV oscillations [27, 28-32].

It has been indicated that slow breathing causes the pulse harmonics of blood flow (*i.e*. blood pressure oscillations) to synchronize with the rhythm of the heart [29]. Various studies have found that slow breathing increases amplitudes of blood pressure oscillations and HRV, and that this is particularly significant at a respiration rate of 6 breaths per min (0.1 Hz) [33-36]. At 6 breaths per min, the LF HRV oscillations are said to be augmented by respiration [37-38].

The mechanism underlying the BP-lowering effect is [39, 13, 41].

- One hypothesis holds that autonomic imbalance plays a major role in the origin of hypertension [42-44]. Relative overactivity of the sympathetic nervous system eventually desensitizes cardiopulmonary and arterial baroreflex/chemoreflex receptors, leading to a resetting of threshold BP values at which regulatory signals are triggered [46]. Paced breathing with prolonged breath cycles may favorably alter (ie, reduce) chemoreceptor sensitivity, thereby decreasing arterial baroreceptor inertia and sympathetic outflow [47].
- Another possible mechanism involves the fact that augmentation of tidal volume activates the Hering-Breuer reflex mediated by pulmonary stretch receptors [48]. This reduces chemoreflex sensitivity, in turn up regulating baroreflex receptor sensitivity and thereby decreasing arterial BP.

It has also been suggested that paced slow breathing entrains central nervous system nuclei in which respiratory and cardiovascular centers crosstalk, thus favorably altering rhythmic sympathetic outflow to the vasculature. Small mechanistic studies suggest that the reduction in BP occurs mainly via a reduction in systemic vascular resistance and total arterial compliance [39, 40, 49, 24].

It is known that regular practice of breathing exercises increases parasympathetic tone, decreases sympathetic activity, improves cardiovascular and respiratory functions, decreases the effect of stress on the body and improves physical and mental health (Adhana *et al*., 2013, Kulur *et al*, 2009 and Mourya *et al*, 2009). Hence, breathing is considered to be a regulator of the autonomic nervous system and consequently of mental processes. Controlling the breath and thus calming the nerves is a prerequisite to controlling the mind and the body. By voluntarily changing the rate, depth, and pattern of breathing, the messages being sent from the body's respiratory system to the brain can be changed. In this way, self guided breathing techniques provide a gateway to the autonomic communication network through which the individual can, by changing the breathing patterns, specific messages send to the brain using the body language, and the body responds to it.

Conclusion

This demonstrated that slow breathing is indeed capable of inducing a modification in respiratory and cardiovascular control, and that appropriate training could induce a long-term effect.

Conflict of interest

There is no conflict of interest in the conduct of the study.

References

1. Landsbergis PA, Schnall PL, Warren K, Pickering TG, Schwartz JE. Association between ambulatory blood pressure and alternative formulations of job strain. Scand J Work Environ Health. 1994; 20:349-63.
2. Raymond S. Foreign assistance in an aging world. Foreign Affairs. The Council on Foreign Relations, Inc, Tampa, FL 33662-0001. 2003: 91-105.
3. Reddy KS. India wakes up to the threat of cardiovascular diseases. J Am Coll Cardiol. 2007; 50:1370-2.
4. Bloom DE, Canning D, Sevilla J. The Demographic Dividend: A New Perspective on the Economic Consequences of Population Change. Santa Monica, CA 90407-2138: Rand Corp. 2003.
5. Kearney PM, Whelton M, Reynolds K, Muntner P, Whelton PK, He J. Global burden of hypertension: Analysis of worldwide data. Lancet. 2005; 365:217-23.
6. World Health Organization. Global Health Risks: mortality and Burden of Disease Attributable to Selected Major Risks. Geneva: World Health Organization, 2010, 2009.
7. Srinath RK. Protecting the heart of global development. Heart Beat, The World Heart Federation Newsletter. 2005. Last accessed on 2014. web http://www.worldheartfederation.org/fileadmin/user_upload/documents/press-releases/HB/publications-heartbeat-2005-june.pdf.
8. Babu G, Detels R. Prioritizing social actions and involving community for prevention of the non-communicable diseases. Internet J Epidemiol. 2011. Last accessed. 2014, 9. http://ispub.com/IJE/9/2/11556.
9. K Sembulingam, Prerna Sembulingam. Essentials of medical physiology 5th edition.2010, 580-581.
10. Carey RM, Whelton PK. Prevention, detection, evaluation, and management of high blood pressure in adults: synopsis of the 2017 American College of Cardiology/American Heart Association hypertension guideline. Annals of internal medicine. 2018.
11. Vasan RS, Larson MG, Leip EP, Kannel WB, Levy D. Assessment of frequency of progression to hypertension in non-hypertensive participants in the Framingham Heart Study: a cohort study. Lancet, 2001; 358(9294):1682-6.
12. Bernardi L, Sleight P, Bandinelli G, *et al*. Effect of rosary prayer and yoga mantras on autonomic cardiovascular rhythms: Comparative study. BMJ. 2001; 323:1446-1449.
13. Benson H, Rosner BA, Marzetta BR, Klemchuk HM. Decreased blood pressure in pharmacologically treated hypertensive patients who regularly elicited the relaxation response. Lancet. 1974; (1):289-291.
14. Patel C. 12-month follow-up of yoga and biofeedback in the management of hypertension. Lancet. 1975; (1):62-64.

15. Irvine J, Johnson DW, Jenner D, Marie GV. Relaxation and stress management in the treatment of essential hypertension. J Psychosom Res. 1986; 30:437-450.
16. Paced breathing. paced breathing [INTERNET] Friday, 2015. Available from:file:///D:/project%20%20final%20year%20/Paced%20Breathing.html
17. Viskoper R, Shapira Priluck I. *et al*. Non-pharmacological treatment of resistant hypertensives by device-guided slow breathing exercises. Am J Hypertens. 2003; 16:484-487. [PubMed]
18. Schein M, Gavish B, Herz M *et al*. Treating hypertension with a device that slows and regularizes breathing: A randomized double-blind controlled study. J Human Hypertens. 2001; 15:271-278.
19. Elliott WJ, Izzo JL, Jr White WB, *et al*. Graded blood pressure reduction in hypertensive outpatients associated with use of a device to assist with slow breathing. J ClinHypertens. 2004; 6:553-559.[PubMed]
20. Grossman E, Grossman A, Schein MH, Zimlichman R, Gavish B. Breathing control lowers blood pressure. J Human Hypertens. 2001; 15:263-269.
21. Rosenthal T, Alter A, Peleg E, Gavish B. Device-guided breathing exercises reduce blood pressure – ambulatory and home measurements. Am J Hypertens. 2001; 14:74-76.
22. Meles E, Giannattasio C, Failla M, *et al*. Nonpharmacologic treatment of hypertension by respiratory exercise in the home setting. Am J Hypertens. 2004; 17:370-374.
23. Parati G, Izzo JL, Jr, Gavish B. Respiration and blood pressure. In: Izzo JL Jr, Black HR, editors. Hypertension Primer. 3rd ed. Baltimore, Md: Lippincott, Williams, and Wilkins. 2003, 117-120.
24. Joseph CN, Porta C, Casucci G, *et al*. Slow breathing improves arterial baroreflex sensitivity and decreases blood pressure in essential hypertension. Hypertension. 2005; 46:714-718.
25. Sleight P, La Rovere MT, Mortara A, *et al.* Physiology and pathophysiology of heart rate and blood pressure variability in humans: is power spectral analysis largely an index of an baroreflex gain? Clin Sci. 1995; 88:103-109.
26. Akselrod S, Gordon D, Madwed JB, *et al.* Hemodynamic regulation: investigation by spectral analysis. Am J Physiol. 1985; 249:867-875.
27. Baselli G, Porta A, Cerutti S, *et al.* RR-arterial pressure variability relationships. Auton Neurosci 2001; 90:57-65.
28. Cevese A, Gulli G, Polati E, *et al.* Baroreflex and oscillation of heart period at 0.1 Hz studied by alpha-blockade and cross-spectral analysis in healthy humans. J Physiol (Lond). 2001; 531:235-244.
29. Lanfranchi PA, Somers VK. Arterial baroreflex function and cardiovascular variability: interactions and implications. Am J Physiol Regul Integr Comp Physiol 2002; 283:815-826.
30. Guzik P, Piskorski J, Krauze T, *et al.* Correlations between the Poincare plot and conventional heart rate variability parameters assessed during paced breathing. J Physiol Sci. 2007; 57:63-71.
31. Sin PY, Galletly DC, Tzeng YC. Influence of breathing frequency on the pattern of respiratory sinus arrhythmia and blood pressure: old questions revisited. Am J Physiol Heart Circ Physiol 2010; 298:1588-1599.
32. Tharion E, Samuel P, Rajalakshmi R. *et al.* Influence of deep breathing exercise on spontaneous respiratory rate and heart rate variability: a randomised controlled trial in healthysubjects. Indian J Physiol Pharmacol 2012; 56:80-87.
33. Paprika D, Gingl Z, Rudas L. *et al.* Hemodynamic effects of slow breathing: does the pattern matter beyond the rate? Acta Physiol Hung 2014; 101:273-281.
34. Bernardi L, Gabutti A, Porta C. *et al.* Slow breathing reduces chemoreflex response to hypoxia and hypercapnia, and increases baroreflex sensitivity. *J Hypertens* 2001; 19:2221-2229.
35. Brown TE, Beightol LA, Koh J, *et al.* Important influence of respiration on human R-R interval power spectra is largely ignored. J Appl Physiol 1993;75: 2310-2317.
36. Beda A, Simpson DM, Carvalho NC, *et al.* Low-frequency heart rate variability is related to the breath-to-breath variability in the respiratory pattern. Psychophysiology. 2014; 51:197-205.
37. Resperate for hypertension. Med Lett Drugs Ther. 2007; 49:55-56.
38. Gavish B. Device-guided breathing in the home setting: technology, performance and clinical outcomes. Biol Psychol. 2010; 84:150-156.
39. Pagani M, Somers V, Furlan R, Dell Orto S, Conway J, Baselli G. *et al*, Changes in autonomic regulation induced by physical training in mild hypertension. Hypertension. 1988; 12:600-610.
40. Mahtani KR, Nunan D, Heneghan CJ. Device-guided breathing exercises in the control of human blood pressure: systematic review and meta-analysis. J Hypertens. 2012; 30:852-860.
41. Brook RD, Julius S. Autonomic imbalance, hypertension, and cardiovascular risk. Am J Hypertens. 2000; 13(2):112-122.
42. Mancia G. Björn Folkow Award Lecture: the sympathetic nervous system in hypertension. J Hypertens. 1997; 15(2):1553-1565.
43. Radaelli A, Bernardi L, Valle F, Leuzzi S, Salvucci F, Pedrotti L, *et al*, Cardiovascular autonomic modulation in essential hypertension: effect of tilting. Hypertension. 1994; 24:556-563.
44. Oneda B, Ortega KC, Gusmão JL, Araújo TG, Mion D Jr. Sympathetic nerve activity is decreased during device-guided slow breathing. Hypertens Res. 2010; 33:708-712.
45. Schelegle ES, Green JF. An overview of the anatomy and physiology of slowly adapting pulmonary stretch receptors. Respir Physiol. 2001; 125:17-31.
46. Sharma M, Frishman WH, Gandhi K. RESPeRATE: nonpharmacological treatment of hypertension. Cardiol Rev. 2011; 19:47-51.
47. Sica DA. Device-guided breathing and hypertension: a yet to be determined positioning. Cardiol Rev. 2011; 19:45-46.
48. Pramanik T, Sharma HO, Mishra S. *et al*. Immediate Effect of Slow Pace Bhastrika Pranayama on BP and HR. J Altern Complement Med 2009; 15(3):293-295.
49. Somers VK, Mark AC, Abbound FM. Potentiation of Sympathetic Nerve Responses to Hypoxia in Borderline Hypertensive Subjects. Hypertension. 1988; 11:608-612.