

Postural Instability in Response to Chronic Hyperventilation in Asthmatic Children

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Abstract: The purpose of this study was to identify the postural stability deficits in response to chronic hyperventilation in asthmatic children. Thirty children were included, their age ranged from 8 to 12 years. Fifteen asthmatic children were referred from the out patient's clinic of the Abo-Elreesh teaching hospital. Fifteen healthy children volunteers were participated to represent the control group; they were matched in age, weight and height to the asthmatic group. Biodex stability system was used to assess dynamic stability level "in the form of overall (SI), anteroposterior (AP) and mediolateral (ML) stability indices" in both groups during standing on both feet with eyes open, then with closed eyes. Results showed that independent t-tests of asthmatic children had significant stability deficits in comparison with healthy children. All children had better stability during opened-eye condition than closed-eye conditions. It had been concluded that asthmatic children have significantly lower stability control than healthy children and that visual feedback improved stability control in asthmatic and healthy children.

Key words: Hyperventilation-Asthma-Postural instability

INTRODUCTION

A simplified physiological definition of hyperventilation is breathing in excess of metabolic requirements, a pathophysiological process which can be acute or chronic [1]. Hyperventilation, or hypocapnia, has been a pervasive topic in asthma [2, 3]. Hyperventilation is caused by increased alveolar ventilation relative to metabolic carbon dioxide production. Consequently, alveolar carbon dioxide pressure tends to fall below normal levels [4].

Functionally, hyperventilation involves either fast or deeper breathing, or it may be the combination of both, resulting in an increase in minute ventilation above what is required by the organism's metabolic demand [5, 6].

The primary purpose of the respiratory system is to support metabolism, largely by facilitating the delivery of O₂ and removal of CO₂. To that end, peripheral and central neural centers provide chemical, mechanical and sensory feedback to the central nervous system in order to mount an appropriate response to metabolic demands. Sensory, chemical and mechanical feedback signals are

integrated by respiratory centers in the brainstem, which then provide efferent signals to respiratory muscles. Derangements in these processes may result in an inappropriate ventilatory response to metabolic demand, with hypo- or hyperventilation as the potential outcomes [7].

Hyperventilation differs from hyperpnea, which is increased minute ventilation without change in carbon dioxide partial pressure (PCO₂). Levels of PCO₂ falling below 35 mmHg typically indicate that breathing is in the hypocapnic range [8] and levels around 30 mmHg or lower on repeated occasions and across longer measurements periods have been proposed as being indicative of "unequivocal chronic hypocapnia" [9].

It was reported that there are many people with breathing pattern disorder (BPD) who have been labeled as asthmatics. "Thirty percent of cases of asthma are known to be induced by emotion or exercise and many symptoms are common to hyperventilation and to asthma: intermittent, labored breathing; relief from bronchodilators (transient in hyperventilation); exercise; cough; fear, anxiety and panic [10].

In the neurological context, clinicians suspect hyperventilation when patients report balance symptoms, dizziness and distal and perioral paraesthesias, in the absence of organic findings [11].

Several investigations regarding whether hyperventilation induces objective postural changes found that forced, voluntary hyperventilation increases body sway in normal subjects [12], furthermore, a group of patients with bilateral vestibular function showed a comparable enhancement of sway after hyperventilation. Since such patients do not have functioning vestibule-spinal pathways, the findings suggested that the mechanism mediating this sway increase is extra-vestibular. However, the mechanism responsible for the appearance of hyperventilation-induced symptoms is not clear. That hyperventilation influences CNS activity has been known for decades, e.g. hyperventilation is one of the most widely used methods for EEG activation [13]. Postural control is based on three distinct processes which develop through childhood: 1-a sensory organizational process, in which one or more of the orientation senses (visual, somatosensory and vestibular) are involved and integrated within the central nervous system [14]; 2- a motor adjustment process, involved in executing coordinated and properly scaled sensorimotor responses [15]; and 3-an internal representation of body scheme that slowly matures during childhood [16]. Both children and adults make use of visual, vestibular and proprioceptive information to control their body posture, but the respective contribution of these sensory inputs varies during ontogenesis [17].

Motor control is a key component in injury prevention. Loss of motor control involves failure to control joints, commonly because of incoordination of the agonist-antagonist muscle co-activation. There is evidence that the effects of breathing pattern disorders, such as hyperventilation, result in a variety of negative psychological, biochemical, neurological and biomechanical influences and interferences, capable of modifying subsystems of motor control. Breathing pattern disorders (the extreme form of which is hyperventilation), automatically increase levels of anxiety and apprehension, which may be sufficient to alter motor control and to markedly influence balance control [18].

This study was carried out to find out the influence of chronic hyperventilation on postural stability in children with bronchial asthma.

MATERIALS AND METHODS

Subjects: Thirty children (13boys, 17girls) were included in the study. Their age ranged from 8 to 12 years old with a mean value of 10 ± 2.7 years. Fifteen children with mild to moderate degree of bronchial asthma since at least 5 years have been selected to represent the study group. They were referred from the out patient's clinic in Abo-Alreesh pediatric teaching hospital. The other fifteen children were matched healthy volunteers and were participated as control group. Written informed consent was obtained from the participants' parents of the children participating in this study.

Exclusion Criteria: Patients with the following clinical disorders were excluded from participating in the study

- Middle ear infections, neural diseases or any other disease contribute to balance and stability.
- Musculoskeletal problems as deformities of the spine or extremities.

Biodex Balance System: (Biodex Medical Systems, Shirley, NY, USA) is a commercially available dynamic postural stability assessment and training system. It was used to assess double and single leg static balance. It consists of movable balance platform, which provides 20° of surface tilting in 360° range and is interfaced with a microprocessor based actuator. The actuator controls the manually present degree of surface instability, which ranges from a completely firm surface with stability level 8, to a very unstable surface, with stability level 2. Biodex stability system is used to evaluate stability indices (SI) in degrees, anteroposterior stability (AP) in degrees and mediolateral stability (ML) in degrees.

Assessment Procedures: Each testing session took a total period of 15-20 minutes, including participant orientation and assessment procedures. The participant should center him-self on the platform before starting the test. Then the participant is instructed to keep this position while investigator identifies the Participant foot or feet position on platform grid through recording the heel coordinates and foot angle. All these values of position are recorded on the balance system computer software (centered position of patients). The participants underwent the tests at stability level 8 for a period of 20 seconds for each test (the test was performed for 3 trials

and the end result as a mean of 3 trials was calculated). The subject was instructed to maintain a level platform for a period 20 seconds for each test and rest by sitting for one minute after each test. The test was performed in opened-eye condition then with closed eye condition [18].

Statistical Analysis:

- The data were analyzed by calculating: Mean \pm Standard deviation.
- Student t: test was used to compare the mean stability level between control group and study group.

Paired t-test was used to compare the mean stability level between opened-eye condition and closed-eye conditions within each group.

RESULTS

The results of the study showed that there was no statistical significant differences concerning age, weight and height in both groups (Table 1).

The results showed a significant difference in overall stability indices (SI), anteroposterior stability indices (A/P) and mediolateral (M/L) between Open eye condition and closed eye condition in asthmatic children with more stability control during open eye condition than closed eye condition (Table 2).

In addition there was a significant difference in overall stability indices (SI), anteroposterior stability indices (A/P) and mediolateral (M/L) between open eye condition and closed eye condition in normal subjects with more stability control during open eye condition than closed eye condition (Table 3).

Table 1: Demographic Data of children in both groups:

Characteristics	Group A (n=15) Group B (n=15)		P-VALUE	Sig
	mean \pm SD	mean \pm SD		
Age (years)	10 \pm 1.4	10.2 \pm 1.5	0.712	NS
Weight (kg)	32.46 \pm 6.2	30.66 \pm 5.8	0.981	NS
Height (cm)	137.6 \pm 4.5	141.2 \pm 6.1	0.12	NS

Results in Mean \pm Standard Deviation

Table 2: The mean difference of SI, A/P and M/L stability indices in degrees during open eye and closed eye conditions in asthmatic group

variables	Open eye	Closed eye	P-value	Sig
SI	6.7° \pm 0.95°	7.19° \pm 0.38°	.000*	Sig
A/P	5.9° \pm 1.17°	7.11° \pm 1.12°	.000*	Sig
M/L	5.32° \pm 1.19°	7.21° \pm 1.32°	.000*	Sig

Results in Mean \pm Standard Deviation

*Sig. P < 0.05 "°": degrees Sig: significant

A/P: Anteroposterior. M/L: Mediolateral. SI: Overall stability indices

Table 3: The mean difference of SI, A/P and M/L stability indices in degrees during open eye and closed eye conditions in control group

Variables	Open eye	Closed eye	P-value	Sig
SI	5.24° \pm 1.26°	6.49° \pm 1.25°	0.000 *	Sig
A/P	4.15° \pm 1.09°	5.55° \pm 1.33°	0.000 *	Sig
M/L	5.13° \pm 0.98°	6.24° \pm 1.09°	0.000 *	Sig

Results in Mean \pm Standard Deviation

"°": degrees *Sig. P < 0.05

With comparison of the stability indices between patients in study group and control group in open eye condition the results showed that there were significant reduction in overall, SI, A/P and M/L stability indices in control group than study group (Fig. 1).

Also in eye-closed condition the stability indices in control group were significantly lower (better stability) than that in study group (Fig. 2).

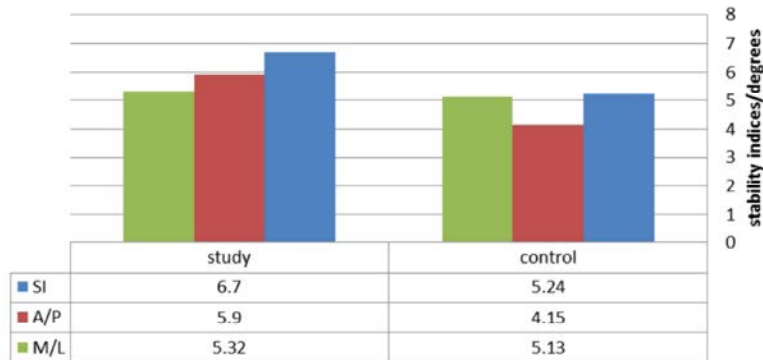


Fig. 1: The mean values of overall stability (SI), A/P and M/L stability indices in degrees in study group versus control group during open eye condition.

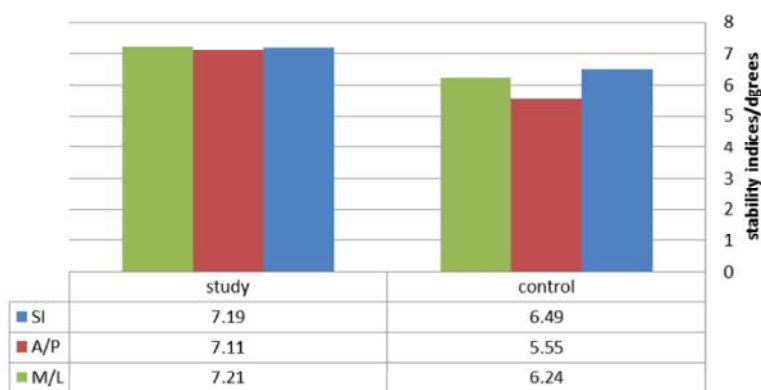


Fig. 2: The mean values of overall stability (SI), A/P and M/L stability indices in degrees in study group versus control group during eye-closed condition

DISCUSSION

This study was conducted to investigate the effect of chronic hyperventilation on postural stability in children with bronchial asthma.

Assessment of balance was done using Biodex system for both groups.

The results of this study showed that the overall SI, A/P and M/L stability indices were significantly lower in degrees (better postural control) in eye-open condition than eye-closed condition in both the study and control groups. These results indicated that visual feedback provides better balance control and minimize postural sway either in normal or pathological condition. The importance of visual feedback and its contribution to balance control even in healthy subjects had been declared several times in literature and the results of the present study is in agreement with several previous studies which emphasized that vision is a major contributor to balance control [19,20].

The results of the present study showed also that all stability indices (overall, A/P and M/L) were significantly lower in the control group in both eye-open and eye-closed conditions. These results indicated that children with bronchial asthma had higher degree of postural sway, and compromised balance control in comparison with the matched healthy subjects.

The results of this study were supported by previous studies which found that there is a link between control of respiration and control of posture, although their mechanisms are differently organized [21].

It was confirmed that hyperventilation causes an objective disturbance of postural control. The causes of this unsteadiness are likely to be multiple in the light of

the widespread metabolic changes induced by hyperventilation [22].

It was found that chronic breathing pattern disorders (BPD) as chronic hyperventilation negatively influences motor control, neurological sensitization, muscle behavior, pain threshold and balance [23].

It was concluded that hyperventilation with its associated hypocapnia can alter the resting muscle tone and ultimately motor pattern changes via the increased excitability in the nervous system and muscular system [24].

Kinnula and Sovijarvi [25] using cycle ergometry, noted consistent hyperventilation in all the asthmatic females, despite no evidence of bronchospasm at one minute after exercise or differences in exercise capacity. The findings are similar to a study by Hammo and Wienburger [26] which assessed 32 patients diagnosed with exercise-induced asthma, for hyperventilation. Of the 21 patients who experienced asthma symptoms, 11 had no significant decrease in FEV1, but demonstrated the lowest PCO2, suggesting hyperventilation.

Lum [27] has discussed the reasons for people becoming hyperventilators: "Neurological considerations can leave little doubt that the habitually unstable breathing is the prime cause of symptoms. Why they breathe in this way must be a matter for speculation, but manifestly the salient characteristics are pure habit."

Respiratory alkalosis leads to an accumulation of incompletely oxidized products of metabolism, due to the activation of anaerobic energy pathways. The products of the anaerobic pathway are acids such as lactic acid and pyruvic acid. This acidification is more extreme in deconditioned individuals. When ATP production is supplemented by anaerobic glycolysis, lactate

accumulates in muscle cells and the bloodstream reducing pH. Relative acidosis then encourages bicarbonate retention, resulting in increased CO₂ production, stimulating a more rapid breathing rate, leading to the respiratory threshold being breached. In a deconditioned individual this threshold is lower, resulting in dyspnea and fatigue early in aerobic activity. The deconditioned individual relies more on anaerobic metabolism for energy supply [28].

The significant increase reported in postural sway can be attributed to the outcomes of deconditioning that include; loss of muscle mass, decreased ability to use energy substrates efficiently, decreased neuromuscular transmission, decreased efficiency in muscle fiber recruitment with indications of disruption of normal motor control being apparent [29].

It was proved that hyperventilation can cause asymmetric phenomena, i.e. unilateral paraesthesia such mechanisms include anatomical differences in the peripheral nerves and their nutrient vessels and asymmetrically decreased cerebral perfusion, alone or in combination with asymmetrical hemispheric processes [11].

This study confirmed that hyperventilation causes an objective disturbance of postural control, The causes of this unsteadiness are likely to be multiple which are attributed the widespread metabolic changes induced by hyperventilation, however, in the main, the effects are not mediated by vestibular mechanisms but rather by interference with somatosensory and vestibular-compensation processes.

Literature supports that the increase occurs mainly in the sagittal plane, with a smaller increase in the frontal plane. It is to be noted that the sway increase is not due to respiratory movements because sway recordings were always due to cardiovascular effects of hyperventilation, it is difficult to ascertain but experiments with variable workloads indicate that heart rate and body sway do not correlate [30].

Proprioceptive information from the lower limbs is probably the single most important sensory input for the control of postural balance in man. Hyperventilation was proved to modify the somatosensory potentials from the lower limb which in turn could underlie the unsteadiness observed after hyperventilation. Study revealed involvement of the sural nerve in postural control mechanism, it is representative of the changes brought about by hyperventilation in the whole of the distal somatosensory system, which conveys proprioceptive as well as tactile information from the ankle and foot [22, 31].

CONCLUSIONS

The findings of the present study support that hyperventilation affects postural control and significantly increase postural sways in all recorded parameters. This could have significance for the treatment of bronchial asthma patients with balance disorders, since the rehabilitation specialists should be aware about nature of hyperventilation and recognize that it may decompensate an underlying vestibular disorder.

REFERENCES

1. Gardener, W., 1990. Hyperventilation disorders. J.R. Soc. Med., 83: 755-757. Make references like this style.
2. Clarke, P.S. and J.R. Gibson, 1980. Asthma hyperventilation and emotion. Australian Family Physician, 9: 715-719. [PubMed: 7425962].
3. Bruton, A. and S.T. Holgate, 2005. Hypocapnia and asthma: A mechanism for breathing retraining. Chest, 127: 1808-1811.
4. McDonough, J.T., 1994. Stedman's Concise Medical Dictionary. Williams and Wilkins; Baltimore.
5. Gardner, W.N., 1996. The pathophysiology of hyperventilation disorders. Chest, 109: 516-534. [PubMed: 8620731].
6. Wientjes, C.J., 1992. Respiration in psychophysiology: Methods and applications. Biological Psychology, 34: 179-203. [PubMed: 1467393].
7. Bates, *et al.*, 2013. Ventilatory Control in Infants, Children and Adults with Bronchopulmonary Dysplasia. Respir Physiol Neurobiol., 1; 189(2): 329-37.
8. Oakes, D.F., 2013. Clinical practitioner's pocket guide to respiratory care. 8th ed. Health Educator Publications Inc., Old Town, MN., pp: 65-100.
9. Bass, C. and W.N. Gardner, 1985. Respiratory and psychiatric abnormalities in chronic symptomatic hyperventilation. BMJ, 290: 1387-1390. [PubMed: 3922504].
10. Lum, C., 1996. Hyperventilation and asthma: the grey area. Biological Psychology, 43(3): 262.
11. Evans, R.W., 1995. Neurologic aspects of hyperventilation syndrome. [Review]. Semin Neurol., 15: 115-25.
12. Sakellari, V. and A.M. Bronstein, 1997. Hyperventilation effect on postural sway. Arch Phys Med Rehabil, 78: 730-6.

13. Patel, V.M. and R.L. Mulsby, 1987. How hyperventilation alters the electroencephalogram: a review of controversial viewpoints emphasizing neurophysiological mechanisms. [Review]. *J Clin. Neurophysiol.*, 4: 101-20.
14. Steindl, R., K. Kunz, A. Schrott-Fischer and A.W. Scholtz, 2006. Effect of age and sex on maturation of sensory systems and balance control. *Dev Med Child Neurol.*, 48: 477-82.
15. Assaiante, C., S. Mallau, S. Viel, M. Jover and C. Schmitz, 2005. Development of postural control in healthy children: a functional approach. *Neural Plast.*, 12: 263-272.
16. Schmitz, C. and C. Assaiante, 2002. Acquisition of a New Coordination in a Bimanual Load-lifting Task: Developmental Aspects. *NeurosciLett*, 330: 215-218.
17. Assaiante, C. and B. Amblard, 1995. An ontogenetic model for the sensorimotor organization of balance control in humans. *Hum Mov Sci*, 14: 13-43.
18. Aly, F., E. Fawzy, M. Ibrahim and A. Mohamed, 2007. Assessment of Stability Deficits in Patients with Diabetic Peripheral Neuropathy. *Bull. Fac. PhTh. Cairo Univ.*, 12(1): 31-41.
19. Lord, S.R. and D.L. Molyneaux, 2005. The physiology of falling: assessment and prevention strategies for older people. *J Sci MedSport*, 8(1): 35-42.
20. Raouf Hi, G. David, Mokhtarch and Aymen Ben Othman, 2014. Comparison of Static Balance and the Role of Vision in Elite Athletes. *J Hum Kinet.* Jun 28; 41: 33-41.
21. Pascal David, David Laval, Jérémy Terrien, Isabelle Mora and Michel Petitjean, 2009. Effects of two types of hyperventilation on standing balance. Christian Collet, Emma Guillet, Jean Saint-Martin, Isabelle Rogowski. XIIIème Congrès International de l'ACAPS, Oct 2009, Lyon, France. *Approche Pluridisciplinaire de la Motricité Humaine*.
22. Sakellari, V., A.M. Bronstein, S. Corna, C.A. Hammon, S. Jones and C.J. Wolsley, 1997. The effects of hyperventilation on postural control *Brain*, 120: 1659-1673.
23. Chaitow, L., 2004. Breathing pattern disorders, motor control and low back pain *Journal of Osteopathic Medicine*, 7(1): 34-41.
24. McLaughlin, L., 2009. Breathing evaluation and retraining in manual therapy. *J. Body Mov Ther.*, 13: 276-82.
25. Kinnula, V. and A. Sovijarvi, 1996. Hyperventilation during exercise: independence on exercise-induced bronchoconstriction in mild asthma. *Respir Med.*, 90: 145-51.
26. Hammo, A. and M. Weinberger, 1999. Exercise-induced hyperventilation: a pseudoasthma syndrome. *Ann Allergy Asthma Immunol.*, 82: 574-8.
27. Lum, L., 1987. Hyperventilation syndromes in medicine and psychiatry. *Journal of the Royal Society of Medicine*, pp: 229-231.
28. Fried, R., 1987. *Hyperventilation Syndrome*. Baltimore: Johns Hopkins University Press.
29. Wittink, H. and T. Michel, 2002. *Chronic Pain Management for Physical Therapists*. 2nd ed. Boston: Butterworth Heinemann.
30. Alexopoulos, D., J. Christodoulou, T. Toulgaridis, G. Sitafidis, A. Klinaki and A.G. Vagenakis, 1995. Hemodynamic response to hyperventilation test in healthy volunteers. *ClinCardiol.*, 18: 636-41.
31. Bergin, P.S., A.M. Bronstein, N.M.F. Murray, S. Sancovic and D.K. Zeppenfeld, 1995. Body sway and vibration perception thresholds in normal aging and in patients with polyneuropathy. *J. Neurol Neurosurg Psychiatry*, 58: 335-40.