Scholars Journal of Applied Medical Sciences (SJAMS)
Abbreviated Key Title: Sch. J. App. Med. Sci.
ISSN 2320-6691 (Online)
ISSN 2347-954X (Print)

A Unit of Scholars Academic and Scientific Society, India
www.saspublisher.com

Physiology

Comparative Study of Autonomic Nervous System Status between Normal Individuals and Asthmatic Patients

Dr. Nishanta[1], Bharti Maan[2*], Pooja Sharma[3], Purnima Rathore[4]
[1]3rd year MD student Department of Physiology, Sardar Patel Medical College Bikaner, India
[2]Senior Demonstrator, Department of Physiology, Pandit Jawaharlal Nehru Government Medical College Chamba, India
[3]Senior Demonstrator, Department of Physiology, Sardar Patel Medical College Bikaner, India
[4]M. Sc Student, Department of Physiology, Sardar Patel Medical College Bikaner, India

Original Research Article

***Corresponding author**
Bharti Maan

Article History
Received: 03.03.2018
Accepted: 13.03.2018
Published: 30.03.2018

DOI:
10.21276/sjams.2018.6.3.69

Abstract: Asthma is a common clinical disorder and is characterized by reversible obstruction of the bronchial airways leading to symptoms such as shortness of breath, wheezing, and coughing. Aim of present study was to compare the effect of autonomic nervous system status in normal individual and asthmatic Patients. Selected patients were divided randomly in two groups each comprised of 25 patients. Group A served as a control group. Group B served as a asthmatic group. In response to autonomic nervous system both sympathetic and parasympathetic test can be done. The result of our study showed that cold pressure test shown significant result while S/L ratio and LST (30:15) ratio shown insignificant result. Thus this study indicates that the autonomic defects seen in asthmatics could be secondary to asthma and not because of autonomic aberrations inheritance in asthmatics.
Keywords: Autonomic nervous system (ANS), Systolic blood pressure (SBP), Diastolic blood pressure (DBP), Cold pressure test (CPT), Standing to lying ratio (S/L), and Lying to standing ratio (LST).

INTRODUCTION

Asthma is a common clinical disorder and is characterized by reversible obstruction of the bronchial airways leading to symptoms such as shortness of breath, wheezing, and coughing. The obstruction of the bronchial airways can be produced by one or more of the following Contraction of bronchial smooth muscle, hypertrophy of the bronchial wall, dilatation of bronchial mucosa, and secretions in the bronchial lumina.

Airway hyper responsiveness and accompanying bronchospasm are the principal hallmarks of asthma [1].

The human airways are innervated by autonomic nerves, which regulate many aspects of airway function via its effects on smooth muscle tone, mucus secret ion, microvascular permeability, blood flow, migration of inflammatory cells and release inflammatory mediators from them. Thus, autonomic nerves can influence airway caliber [2-4].

The autonomic nervous system (ANS), formerly the vegetative nervous system, is a division of the peripheral nervous system that supplies smooth muscle and glands, and thus influences the function of internal organs [5]. The autonomic nervous system is a control system that acts largely unconsciously and regulates bodily functions such as the heart rate, digestion, respiratory rate, pupillary response, urination, and sexual arousal [6]. This system is the primary mechanism in control of the fight-or-flight response.

Within the brain, the autonomic nervous system is regulated by the hypothalamus. Autonomic functions include control of respiration, cardiac regulation (the cardiac control center), vasomotor activity (the vasomotor center), and certain reflex actions such as coughing, sneezing, swallowing and vomiting. Those are then subdivided into other areas and are also linked to ANS subsystems and nervous systems external to the brain. The hypothalamus, just above the brain stem, acts as an integrator for autonomic functions, receiving ANS regulatory input from the limbic system to do so [7].

The autonomic nervous system has three branches: the sympathetic nervous system, the parasympathetic nervous system and the enteric nervous system [8-11]. Some textbooks do not include the enteric nervous system as part of this system [12].

The sympathetic nervous system is often considered the "fight or flight" system, while the parasympathetic nervous system is often considered the "rest and digest" or "feed and breed" system. In many cases, both of these systems have "opposite" actions where one system activates a physiological response and the other inhibits it.

Genetic factors are also known to influence not only the occurrence but also the severity of asthma. It has been found that a child's asthma or wheezing is highly associated with mother's or father's asthma, other atopic condition in mother, father or with other siblings. Many of the siblings of asthmatic children who were apparently normal with no overt clinical symptoms of asthma have positive exercise result and are prone develop asthma later in life[13,14].

MATERIALS AND METHODS

This study was conducted in the Department of Physiology, S.P. Medical College, Bikaner subjects were selected from the, Medicine Department P.B.M. Hospital Bikaner.

The study has been undertaken to observe the effect of Autonomic nervous system in normal individuals and asthmatic patients of adult group. The selected subject has a history of asthma from last 6 months.

Patients suffering from liver disease, arthritis, pulmonary tuberculosis, malabsorption, alcoholism, myocardial infarction, heart block disease and non-co-operative patients will be excluded from the study.

METHODS

A total 50 subjects will be enrolled in this study and divided in two groups of 25 each:-
Group A – Control group
Group B – Asthmatic group
PROCEDURE-

The autonomic nervous system was tested by performing the following tests:- The following standard autonomic function tests were conducted with the help of an ECG machine. A standard limb lead II was recorded and R-R intervals were calculated manually.

- Blood Pressure response to standing to supine posture (S/L) Ratio- Longest R-R interval during 5 beats before lying down/Shortest R-R interval during 10 beats after lying down.
- Blood pressure response to 30:15 Ratio- R-R intervals at beat 30/ R-R interval at beat 15 .
- Blood pressure response to Cold pressure test- Subjects were asked to immerse both feet in ice water (1–4C) for 1 min (20). The blood pressure was recorded just before the test and at 1 min, after the immersion of feet in the water. The systolic and diastolic pressure difference at 1 min from the baseline was calculated.

RESULTS

Table-1: Comparison of Mean value of S/L Ratio in study and control group

S/L Ratio	Control Group	Study Group
Mean	13.00	14.64
SD	4.06	4.44
P-value	> 0.05 (Not Significant)	

Table 1 shows the standing to Lying ratio (S/L) in study and control group. It is observed that there is no significant rise in blood pressure in (S/L) ratio in study as compared to control group.

Table-2: Comparison of Mean value of LST 30:15 Ratio in study and control group

LST 30/15	Control Group	Study Group
Mean	1.02	1.02
SD	0.01	0.02
P-value	> 0.05 (Not Significant)	

Table 2 shows the Lying to standing test (LST) 30/15 Ratio in study and control group. It is observed that there is no significant rise in blood pressure in (LST) in study as compared to control group.

Table-3: Comparison of Mean value of SBP in Cold pressure test in study and control group

Systolic BP	Control Group	Study Group
Mean	124.64	124.32
SD	5.56	6.37
P-value	> 0.05 (Not Significant)	

Table 3 shows the Cold pressure test results in study and controls. It is observed that there is no significant rise in Systolic blood pressure (SBP) in study group as compared to control group.

Table-4: Comparison of Mean value of DBP in Cold pressure test in study and control group

Diastolic BP	Control Group	Study Group
Mean	80.68	83.68
SD	3.50	4.23
P-value	< 0.05 (Significant)	

Table 4 shows the cold pressure test results in study and controls. It is observed that there is significant rise in Diastolic blood pressure (DBP) in study group as compared to control group.

DISCUSSION

Autonomic function tests for sympathetic division and parasympathetic division which were carried out in this study have been extensively used in the past. They are all standard, noninvasive, safe and easily reproducible.

Asthma represents an imbalance of the autonomic nervous system. Several types of autonomic defects have been proposed in asthma; enhanced cholinergic, α-adrenergic, excitatory non-adrenergic non-cholinergic mechanisms promoting bronchoconstriction and glandular secretion [15,16]. Hyposensitivity at β-adrenergic receptors promote bronchodilation, reduction in bronchial secretions and impairment of IgE-mediated mast cell degranulation [17,18].

In our study we have taken asthmatic subjects and assessed their autonomic nervous status by comparing it with controls. The autonomic tests shows that the mean value of S/L ratio and LST ratio did not shown any significant rise in study group when compare with control group. In cold pressure test SBP did not shown any significant result but DBP shows significant result.

The rise in diastolic blood pressure in these tests is a measure of sympathetic efferent vasoconstrictor function mediated via -adrenergic receptor [19]. In asthma, where adrenergic hyper responsiveness has been proposed [20], there should be an exaggerated blood pressure response with these tests.

This increased adrenergic drive to combat parasympathetic hyperactivity causing bronchoconstriction is also supported by the fact that in asthmatics β-receptor blockade causes severe bronchoconstriction's, whereas in normal subjects no significant effect on airway caliber occurs[21,22]. This bronchoconstriction can be inhibited by the anticholinergic pretreatment [23]. From this observation it is concluded that there is increased cholinergic input in asthmatics causing up regulation of the adrenergic system. When this is blocked therapeutically it results in bronchoconstriction.

REFERENCES

1. Insel PA, Wasserman SI. Asthma: a disorder of adrenergic receptors. The FASEB Journal. 1990 Jul;4(10):2732-6.
2. Verhein KC, Prayer AD, Jacoby DB. Neural control of airway inflammation. Curr Allergy Asthma Resp 2009; 9(6): 484–490.
3. Van der Velden VH, Hulsmann AR. Autonomicm innervations of human airways. Neuromodulation 1999; 6(3): 145–159.
4. Nadel J, Barnes PJ. Autonomic regulation of airways. Annu Rev Med 1984; 35: 451–467.
5. Gabella G. Autonomic nervous system. John Wiley & Sons, Ltd; 2001.
6. Jänig W. Autonomic nervous system. InHuman physiology 1989 (pp. 333-370). Springer, Berlin, Heidelberg.
7. Allostatic load notebook. Parasympathetic Function-1999, MacArthur research network, UCSF.
8. Langley JN. The autonomic nervous system W. Heffer; 1921.
9. Jänig W. Integrative action of the autonomic nervous system: Neurobiology of homeostasis. Cambridge University Press; 2008 Jun 26.
10. Furness, John (9 October 2007). "Enteric nervous system". Scholarpedia.doi:10.4249/scholarpedia.4064. Retrieved 14 january 2018.
11. Jartti TT, Kuusela TA, Kaila TJ, Tahvanainen KU, Välimäki IA. The dose–response effects of terbutaline on the variability, approximate entropy and fractal dimension of heart rate and blood pressure. British journal of clinical pharmacology. 1998 Mar 1; 45(3):277-85.
12. Pocock, Gillian. Human Physiology (3rded). Oxford University Press. 2006 pp. 63–64. ISBN 978-0-19-856878-0.
13. Ownby Dr. Environmental Factors vs. Genetic Determination of Childhood Inhalant Allergies. J Allergy Clin immunology 1990; 86: 279–287.
14. Rona RJ, Duran. Family Size, Atopic Disorders in parents, asthma in children and ethnicity, J Allergy-clin-immuno 1997; 99(4): 454–460.
15. Kumar S, Babbar R, Varshney VP, Daga MK, Dalvi VS. A study of autonomic nervous system status in children of asthmatic parents. Indian J Physiol Pharmacol 2012; 56:74-9.
16. Seaton A, Crompton G. Asthma: Clinical features. In: Seaton A, Seaton D, Leitch AG. Crofton and Douglas's Respiratory Diseases. 5th Ed. New Delhi: Wiley India; 2008. p. 922-72.

17. Szentivanyi A. The beta adrenergic theory of the atopic abnormality in bronchial asthma. Journal of Allergy. 1968 Oct 1;42(4):203-32.
18. Reed CE. Abnormal autonomic mechanisms in asthma. J Allergy Clin Immunol 1974; 53:34-41.
19. Bannister R. Diseases of the autonomic nervous system. In: Recent advances in clinical neurology, 4th ed. (Mathews WB, Gilbert GH, Eds.) Edinburgh: Churchill-Livingstone 1984; 67–85.
20. Barnes PJ. Third nervous system in the lung: Physiology and clinical perspectives. Thorax 1984; 39: 561–167.
21. Zaid G, Beall GN. Bronchial response to beta-adrenergic blockade. N Eng J Med 1966; 275: 580–584.
22. Richardson PS, Sterling GM. Effects of beta-adrenergic receptor blockade on airway conductance and lung volume in normal and asthmatic subjects. Br Med J 1969; 3: 143–145.
23. In PW, Dixon CMS, Fuller RW, Barnes PJ. Anticholinergic blockade of beta-blocker induced bronchoconstriction. Is Rev Respire Dis 1989; 139: 1390–1394.