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Exercise induced bronchial lability: A comparison between normal men and women.

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Abstract: *The difference of bronchial lability that is bronchial hyper-reactivity between normal men and women is studied by standard exercise using Harvard steps. Preclinical medical students (M=30, F=30) with mean age of 18.64 years were assessed. Students included were nonsmokers & with no personal history of allergy or any respiratory disease. Parameters compared were age, sex, height, weight & PFTs. Parameters i.e. PFTs were recorded before, during & upto 45 minutes after exercise with computerized spirometer & for PEFR Wright's Mini Peak flow meter was used. Exercise lability index (ELI), ELI % rise & % fall calculated. Resting FVC, FEV1, FEF25-75% & PEFR in men were significantly higher ($p<0.05$) but there was no significant difference of FEV1%. ELI-PEF was not significantly different ($p>0.05$) but there was greater ELI % rise in PEFR values in women during exercise & lesser decline after it.. Women have a greater increase and lesser decrease in flow rates & showed earlier recovery as compared to men. This respiratory response pattern could account for the lower incidence, morbidity & mortality from respiratory allergic diseases seen in women.*
Keywords: *bronchial labilty, exercise, normal men and women, pulmonary function tests*

I. Introduction

Bronchial hyper-reactivity is a well recognized characteristic of asthma[1]. Hyper-reactivity is often assessed as the airway response to standard stimulus usually exercise or an inhalation challenge. Exercise bronchial lability studies done on young smokers indicate that bronchial lability is associated with less decline in pulmonary function and the best chance of improvement in asthma[2]. There are a number of factors on which pulmonary functions depend in normal individuals. Besides the balance between lung recoil and chest elasticity, that determine the mid-position at the end of spontaneous expiration and the co-ordinated neuromuscular function of maintenance of effort, the thoracic and abdominal strength play an important role in most of the pulmonary functions[3]. It has been reported that among young smokers, abnormalities in pulmonary function and risk of development of chronic bronchitis are greater among men than among women[4]. Also boys have an increased tendency to wheeze with the viral respiratory infection when compared to girls.

Smoking may affect female and male lungs differently, and these sex differences may relate to the caliber of airway or to hormonal status at different stages of life. A higher prevalence of airway hyper-responsiveness was found in women who smoked than in men who smoked, was partly explained by lower airway caliber in women (as measured by the absolute level of forced expiratory volume in one second).Exposure to cigarette smoke led to a greater increase in the number of mucus–producing tracheal goblet cells in female rats than in male rats; differences between the sexes were related to the estrous cycle[5].

Along with industrialisation, the prevalence of allergy and asthma is on the rise globally. Interestingly, it has been found that the incidence, morbidity and mortality due to allergy and asthma are less in females than in males[6]. Airway function and responsiveness may be different in men and women. Epidemiological studies also have demonstrated greater small airway dysfunction in men than in women. So to study the difference of bronchial lability between normal men and women, there are provocation tests like inhalation test (methacholine, histamine) and exercise test. But the exercise test has the advantage of not only being physiological but also being capable of reflecting both dilator and constrictor ability of bronchi.

II. Aims and Objectives

1. To study the effect of exercise on pulmonary function tests.
2. To study the difference of bronchial lability in normal men and women i.e. to compare bronchial lability in normal men and women.

III. Material and Methods

Preclinical medical students (M=30, F=30) of L.T.M.M.C., Sion, Mumbai were selected as the subjects. An informed consent was obtained. A questionnaire was filled in by the subjects before testing to confirm that each subject was a non-smoker, with no personal history of allergy with special reference to respiratory allergy

or previous history of any respiratory diseases like asthma, etc. age was obtained from date of birth. Standing height in cms was taken in erect standing position without shoes with his back close to the calibrated stand on a Standiometer. Body weight was recorded in kilograms correct to 0.05 kg on a Wegeberid weighing machine with a sensitive lever with subject in his normal clothing without shoes. The subjects were assessed during the same time of year and at the same time of day to avoid possible seasonal and diurnal variation. It was ensured that all subjects were normal at the time of testing and at least 6 weeks prior to it.

3.1 Apparatus :

Computerized Spirometer was used to record the forced expiratory spirograms. The spirometer is the computed one, the parts of spirometer i.e. pneumotach with tube having connection with proper spirometer connected with CPU of computer. Before testing each subject, the spirometer was calibrated for accuracy using 3 liter syringe. Temperature, barometric pressures were adjusted to that of room temperature and pressure. Zero flow through the pneumotach was also ensured before testing. For recording of peak expiratory flow values Wright's Mini Peak Flowmeter was used. Wright's Mini Peak Flowmeter and Pneumotach were sterilized with potassium permanganate and hydrogen peroxide respectively with every volunteer.

Subjects were asked not to have heavy meals just before this test because a full stomach may prevent lungs from full expansion. They were also asked not to exercise strenuously for 6 hours before the test, asked to wear loose clothing so that it did not restrict breathing in any way. Lung function tests were done in a room of department that has all of the lung function measuring devices. Adjustment of frequency was done with electronic metronome at one beep with every two seconds adjusted as on the metronome calibration was from 40 to 60 per minute . For getting frequency of 30 times per minute adjustment was done with 60 cycles/minute. The procedure was first demonstrated to all for basal readings of PFT. FVC was recorded by asking the subject to take a deep inspiration followed by forceful expiration which was again immediately followed by deep inspiration through pneumotach into machine with nose clip in position. The subjects were asked to make at least three satisfactory forced expiratory vital capacity manoeuvres. The computer system analysed the performance to give FVC, FEV_1, FEV_1% and $FEF_{25-75\%}$. For PEFR, the subject was being asked to take a maximum/deep inspiration and then to blow through flowmeter and basal reading was recorded.

After basal recordings, subject was allowed to do exercise i.e. Harvard's step test for 6 minutes. During exercise readings of PEFR taken 3 minutes after beginning of exercise. Then post-exercise readings of forced expiratory spirograms were recorded i.e. 1 minute and 11 minutes after exercise. PEFR recorded soon after and every 5 minutes up to maximum of 45 minutes after the end of exercise. Five readings were taken each time and the average of the highest three calculated.

Exercise bronchial lability was determined using the exercise lability index (ELI). ELI was calculated as the difference between the highest and the lowest values of the pulmonary function parameter expressed as a percent of its initial value before the test exercise. The dilator and constrictor component of ELI were also calculated. These were expressed as ELI % rise and ELI % fall, calculated using the difference between the highest and the initial, and the lowest and the initial values respectively .Statistical analysis was done using the Student t-test i.e. Unpaired t-test .

IV. Observation and Results

Age group of subjects was between 17 to 19 years. Mean height of the male was 166.5 cms and female was 156.26 cms. Mean weight of women was 53.0kgs and of men was 62.1kgs. The height and weight were significantly greater in men ($P< 0.05$)(Table I). There was no significant difference between the ages of men and women. Resting FVC, FEV_1, $FEF_{25-75\%}$ and PEFR were higher in men ($P<0.05$) but no significant difference of FEV1%. There was significant difference between ELI-PEF% Rise and ELI-PEF% Fall of men and women but no difference in ELI-PEF(Table I). Even one and 11 minutes after exercise there was significant difference in increase in FVC, FEV_1, $FEF_{25-75\%}$ but no significant difference in increase in FEV_1%(Table II & III).

On exercising, the women had a significantly greater percent increase in PEFR during exercise and lesser percent decrease throughout recovery(Table IV, V & Graph 1). By 30 minutes 53.33 % of women had recovered to baseline as compared to only 36.7 % of men ($P< 0.05$). Even at the end of 45 minutes 40% of men had not recovered as compared to only 20% of women (Table VII & Graph 3). There was greater ELI %Rise and lesser % Fall of $FEF_{25-75\%}$ & FEV_1 in women as compared to men (Table VI & Graph 2).

Table I: Showing mean age, height, weight, and the pulmonary function tests
Physical Characterstics, pre-exercise Pulmonary Function tests and ELI-PEF

Sr. No.	Parameter	Men		Women		p value	Significance
		Mean	S.D.	Mean	S.D.		
1	Age(years)	18.76	0.59	18.53	0.69	p□ 0.05	S
2	Height(cms)	166.5	6.39	156.26	6.32	p□ 0.05	S
3	Weight(kgs)	61.3	10.88	53.6	9.55	p□ 0.05	S
4	FVC(lit.)	3.43	0.65	2.59	0.49	p□ 0.05	S
5	FEV_1(lit.)	3.17	0.56	2.33	0.47	p□ 0.05	S
6	FEV_1%	89.4	4.47	91.03	5.23	p> 0.05	NS
7	$FEF_{25-75\%}$	3.97	0.97	3.47	1.51	p□ 0.05	S
8	PEF(lit/min)	484	92.01	367	48.4	p□ 0.05	S
9	PEF-ELI	9.44	1.45	9.97	2.58	p> 0.05	NS
10	ELI-PEF %Rise	3.36	1.14	6.35	1.79	p□ 0.05	S
11	ELI-PEF %Fall	6.03	1.31	3.62	1.76	p□ 0.05	S

Table II: Showing mean of FES post-exercise changes i.e. 1 minute after the end of exercise with S.D.

Sr. No.	**Parameter**	Men		Women		p value	Significance
		Mean	S. D.	Mean	S.D.		
1	FVC(lit.)	3.47	0.65	2.38	0.51	p< 0.05	S
2	FEV_1(lit.)	2.94	0.49	2.08	0.45	p< 0.05	S
3	FEV_1%	85.1	4.66	88.4	5.22	p> 0.05	NS
4	$FEF_{25-75\%}$	3.72	1.0	3.14	1.2	p< 0.05	S

Table III: Showing means of FES post-exercise changes i.e. 11 minutes after the end of exercise with S.D.

Sr. No.	**Parameter**	**Men**		**Women**		**p value**	**Significance**
		Mean	S. D.	Mean	S.D.		
1	FVC(lit.)	3.5	0.61	2.37	0.47	p< 0.05	S
2	FEV_1(lit.)	2.99	0.68	2.15	0.44	p< 0.05	S
3	FEV_1%	88.16	4.49	89.5	5.36	p> 0.05	NS
4	$FEF_{25-75\%}$	3.84	0.98	3.16	1.2	p< 0.05	S

Table IV: Showing means of ELI-PEF , ELI-PEF %Rise and ELI-PEF %Fall with S.D.

Parameter	Men		Women		Significance
	Mean	S.D.	Mean	S.D.	
ELI	9.44	1.45	9.77	2.58	NS
%Rise	3.36	1.14	6.35	1.79	S
%Fall	6.03	1.31	3.62	1.76	S

Table V: Showing comparison of ELI %Rise (+) and %Fall (-) in men & women at different time duration of exercise (Graph 1).

Sr. No.		ELI	
		Men	Women
1	During exercise	+0.32	+0.36
2	Immediately after exercise	+0.28	+0.30
3	5 minutes	-0.40	-0.32

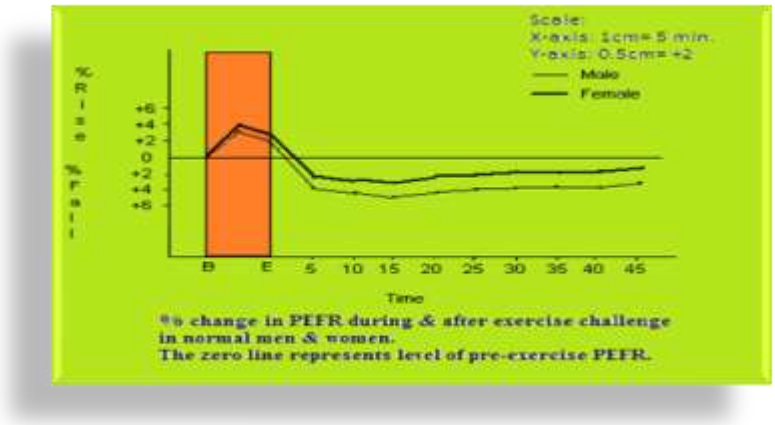
4	10 minutes	-0.43	-0.33
5	15 minutes	-0.47	-0.32
6	20 minutes	-0.42	-0.31
7	25 minutes	-0.41	-0.30
8	30 minutes	-0.40	-0.28
9	35 minutes	-0.40	-0.30
10	40 minutes	-0.40	-0.28
11	45 minutes	-0.38	-0.26

Table VI: Showing ELI, %Rise & %Fall of PEF, $FEF_{25-75\%}$ & FEV_1 in men & women (Graph 2).

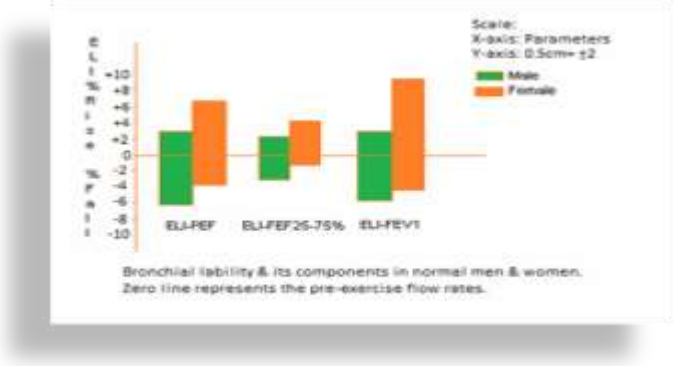
Parameter	Men			Women		
	ELI	%Rise	% Fall	ELI	%Rise	% Fall
PEF	9.44	3.36	6.03	9.97	6.35	3.62
$FEF_{25-75\%}$	6.6	2.25	3.6	5.46	4.1	1.1
FEV_1	8.2	2.98	5.9	14.36	8.46	5.59

Table VII: Showing recovery of subjects after exercise i.e. by 30 minutes and at the end of 45 minutes (Graph 3)

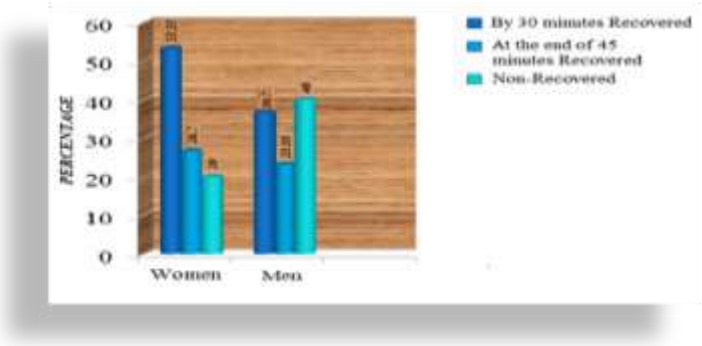
	By 30 minutes Recovered (%)	At the end of 45 minutes Recovered (%)	Non-Recovered (%)
Women	53.33	26.7	20
Men	36.7	23.33	40



Graph 1



Graph 2



Graph 3

V. Discussion

The present study was conducted to find out the difference of bronchial lability induced by acute exercise, in students of first year M.B.B.S., L.T.M.M.C., Sion, Mumbai,.

This study shows that the lung volumes and flow rates in men are significantly higher than in women. FVC, FEV_1, and PEFR have been shown to be higher in men even after adjusting for height and weight but FEV_1 % is greater in women. The published reports in the literature support this finding. As lung growth keeps pace with body growth, age, height and weight are the three important variables which influence the pulmonary functions. Hence there may be variation in resting values between men and women[7].

Mc Ilroy et al found a decreased non-elastic resistance with exercise. There is biphasic response with an initial bronchodilation followed by a bronchoconstriction with exercise[8]. So the exercise while it lasts has a protective effect on airways against bronchoconstriction. The bronchodilation could be due to a combination of factors. A decrease in vagal tone and increased beta-adrenergic stimulation are known to occur during exercise[9]. Inhibitory mediators such as some prostaglandin (PGE_2) may also play a role in causing the bronchodilation. Prostaglandin E_2 is not a directly acting relaxant, it may inhibit histamine-induced muscle contraction. This response is transient and followed by bronchoconstriction, which is believed to be caused by mediator release resulting from airway cooling or osmolality changes. The dry, cold air inspired during exercise can stimulate thermally active receptors with resultant bronchoconstriction. Bronchoconstriction may also be caused by mediators like prostaglandins (PGI_2) and leukotrienes released from locally resident cells (such as mast cells) or cells brought to the airways by the circulation (such as basophils, eosinophils and neutrophils)[10,11].

Airway smooth muscle function is regulated by the autonomic nervous system. Both diminished beta-adrenergic function and enhanced cholinergic reflex broncho-spasm have been found in asthma. Beta adrenergic agonists relax smooth muscle, decrease the leukocyte inflammatory response by inhibiting mediator release during infections. Over 20 years ago, it was postulated that diminished beta-adrenergic function was a component of asthma that contributed to the bronchial hyper-responsiveness and bronchoconstriction found in asthma. Moreover, it was proposed that respiratory infections might diminish beta-adrenergic function to further intensify airway obstruction. Acetylcholine via cholinergic receptors modulate vagally-mediated bronchoconstrction[12].

In addition to classic adrenergic and cholinergic airway innervation, non-adrenergic and non-cholinergic nervous system has been proposed. The adrenergic inhibitory nerves relax airway smooth muscle and are likely to be activated by chemical stimulation. Bronchodilating neurotransmitters in the airway, such as vasoactive intestinal peptide (VIP), co-exist with acetylcholine and serve to antagonize the effect of airway

cholinergic nerves. During airway inflammation, VIP is inactivated to have unopposed cholinergic bronchoconstriction. Conversely, non-cholinergic excitatory nerves induce bronchoconstriction through neuropeptides, such as substance P; this response can counter the bronchodilation stimulated by the non-adrenergic inhibitory reflex. In addition, substance P can cause mast cell mediator release to produce airway inflammation and induce bronchoconstriction.

The exercise effect is due to increased patency and possibly increased number of conducting airways. The maximum inflation and deflation, which occurs during exercise, is an important physiological stimulus for the release of lung surfactant and prostaglandin (PGE_2) into the alveolar spaces thereby increasing the lung compliance and decreasing bronchial smooth muscle tone respectively[13].

Airway resistance greatly increases after cigarette smoking. This increases the cost of breathing, which could be detrimental to performance during prolonged exercise but this bronchoconstrictior effect, caused by inhalation of cigarette smoke, was rapidly reversed by brief exercise. Administration of propranolol does not block the exercise effect observed, because the predominant mechanism is more likely due to lessened parasympathetic activity than to beta adrenergic stimulation. Smooth muscle tone, mediated through cholinergic vagal pathways, is probably increased through the bronchoconstricing effect of cigarette smoke.

It has been reported that boys have an increased tendency to wheeze with the respiratory infection when compared to girls. Male-female differences also exist in the age at which the volume and flow rates begin to decline. In the studies of Anderson and associates, the difference in prevalence of chronic obstructive airway disease (COAD) between men and women was much less striking than that observed for chronic bronchitis, although a small excess male prevalence existed[14].

Lability is the sum of airway constriction and dilation, therefore a high exercise lability does not necessarily indicate a high bronchial reactivity. Bronchial reactivity is probably reflected by constrictor component of ELI[15]. An increase in exercise induced bronchial lability can be produced by an increase in bronchodilation during exercise, increase in bronchoconstriction after exercise or a combination of both. The observation also shows that this bronchial response to exercise differs qualitatively in men and women. Women have a greater increase and lesser decrease in flow rates as compared to men, indicating a greater degree of bronchodilation and less of bronchoconstriction. This difference may be due to the modulation exerted by the female sex hormones on the various interactive mechanisms that cause the bronchomotor changes.

Increase in alveolar ventilation during pregnancy and luteal phase of menstrual cycle is well known and has been attributed to increased levels of progesterone because progesterone has been shown to have a smooth muscle relaxant effect and therefore may have a bronchodilator action. The reports indicate the influence of progesterone on central (medullary) and peripheral receptors causing hyperventilation and hypocapnia in the luteal phase of normal menstrual cycle and pregnancy by increasing the sensitivity of the receptors, and this increased sensitivity of respiratory system to progesterone in luteal phase has been attributed to cause a beneficial compensatory mechanisms to meet the increased demands during pregnancy. Foster et al showed progesterone mediated relaxation of the bronchial smooth muscle in animal models through a beta-adrenoreceptor mediated mechanism. Exogenous administration of progesterone is often prescribed for hypoventilation syndrome. It is also known to cause relaxation of smooth muscle especially of the reproductive and gastrointestinal system. Keane et al demonstrated reduced spontaneous contraction and decreased magnitude of maximal contraction in human gall bladder exposed to progesterone in vitro. Everson et al have reported relaxation of gall bladder muscle in luteal phase of menstrual cycle. Further due to beneficial role in pre-menstrual asthma, wherein the asthmatic attacks are increased when the serum levels of progesterone fall has also been suggested[16,17].

Rao et al reported significant lower values of PEFR and FEF25-75% in oestrogenic phase and Das et al found non-significant higher values in the progesteronic phase respectively in their adult female subjects of mean age 18 years. This greater variability could be due to the changing level of female sex hormones with different phases of the menstrual cycle. Thus, interplay of these complex mechanisms regulate airway caliber andairway response to stimuli may be operative in inducing the biphasic response with exercise[18].

Summary and conclusion

This study shows that

1. Respiratory response patterns of men and women during exercise and after it are significantly different.
2. Women have greater dilator component as compared to men.
3. Women recovered faster after a short bout of exercise.
4. Women have greater bronchial lability than men.

This is advantageous as it is associated with better chance of improvement and faster recovery in those with respiratory diseases as compared with men. This finding indicates that the sex differences in bronchial response and lability may have to be considered during prescription and maintenance of therapeutic regimens for obstructive airway disease.

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