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Original Research Article

Study of pulmonary function tests in response to localized cold stimuli in age group between 19-30 years of Guwahati city

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ABSTRACT

Background: The aim of the study was to observe any variation in the pulmonary functions in response to localized cold stimuli in an externally controlled environment.
Methods: The baseline pulmonary function parameters were obtained before the introduction of localized cold stimuli. Now the stimuli were introduced by immersing both feet up to ankle in a bucket full of cold water maintained at temperature between 8-10 degree Celsius in 30 male healthy subjects (age group 19-30 years) and the parameters were noted after 2 minutes and 5 minutes respectively using a Medspiror (HELIOS) Electronic spirometer and keeping room temperature at 24^{o}celsius in the Department of Physiology, Gauhati Medical College, Guwahati, Assam, India. For statistical analysis, the value of the pulmonary function parameters were presented as Mean±standard deviation. Analysis of variance (ANOVA) using Statistical Package for Social Sciences (SPSS) version 20 was employed for comparing the parameters and $p<0.05$ was considered as significant.
Results: It was observed that the tidal volume and Inspiratory capacity showed a significant increase ($p<0.05$) whereas the Inspiratory reserve volume, expiratory reserve volume and forced vital capacity showed a significant decrease ($p<0.05$) in response to the cold stimuli.
Conclusions: A significant effect was obtained in the pulmonary function tests exposed to cold stimuli showing the multidimensional response of the respiratory mechanics to cold, making a base for further information into the cold climatic effect in an individual.

Keywords: Age group 19-30 years, Localized cold stimuli, Male, Medspiror (HELIOS) electronic spirometer Pulmonary function tests

INTRODUCTION

Cold elicits several effects on the respiratory system. Pulmonary mechanics are worsened due to bronchoconstriction, airway congestion; secretions and decreased mucociliary clearance.[1] These responses are active during cold environment and are possibly responsible for decreased immune function and protection against airborne pollutants. Cold causes local vasoconstriction and local cessation of sweating. These reactions are caused by local effects of temperature directly on the blood vessels and also by the local cord reflexes conducted from skin receptors to the spinal cord and back to the same skin area and the sweat glands.[2] The primary ventilatory effect of cold is to decrease baseline ventilation and respiratory chemo sensitivity, but the acute exposure to cold may cause sympathoadrenal discharge which in turn may cause increase in the respiratory rate.[1,3]

Although these responses provide significant protection against heat losses in many animals, the study towards

these effects are also limited.[4,5] In studies using cold pressor test it has been seen that there is a significant increase in the lung function parameters induced by pain causing sympathetic and parasympathetic overactivity.[5,6]

Another study stated that the pathways by which acute pain increases ventilatory drive was based on three possibilities: spinal reflex pathways, activation of supra-pontine structures provoking the modulation of respiratory centers in the central nervous system, and a direct effect of nociceptive afferents on the respiratory centers in the brain stem.[7] These findings indicate to a large extent that stimulation of respiration should form an integral part of cold induced pain.

However, literature towards the change of respiratory parameters induced by the rapidly adapting receptors which responds initially to the cold is very limited. Exposure to cold elicits a generalized cutaneous vasoconstriction that is especially pronounced in hands and feet. If a peripheral limb is chilled, reflex generalized vasoconstriction is caused in part by the cooled blood that returns to the general circulation. This returned blood then stimulates the temperature regulating centre in the anterior hypothalamus which responds to direct application of cold to evoke cutaneous vasoconstriction and causes change in ventilation.[8]

Thus, the primary objective of this study is to determine the pulmonary function parameters in response to localized cold stimuli in individuals of this region and to find any significant variation in the parameters.

METHODS

The study was conducted in the Department of Physiology, Gauhati Medical College, Guwahati, Assam, India for a duration of 2 months. After obtaining permission from the Institutional Ethics Committee, individuals were approached to collect data. Study was performed after obtaining full consent from them. Thirty (30) healthy male individuals between 19-30 years were selected by simplified random sampling from Guwahati city. Individuals suffering from Obstructive lung diseases like bronchial asthma, emphysema; Restrictive lung diseases like pneumoconiosis, tuberculosis; hypertension, diabetes were excluded from the study. Materials used were Medspiror (HELIOS) electronic spirometer, mercury thermometer, cold water (8-10^{0}C), and bucket.

Room temperature was maintained optimally at 24 degrees Celsius. All the subjects were tested upon between 9 am to 12 noon in order to rule out any alterations imposed by diurnal variations in respiratory parameters. The subject was allowed to sit comfortably in a chair and breathe normally. The mouthpiece placed snugly in his mouth and nose clip applied to his nose so that he breathes only through the mouth piece.

The subject was asked to take normal inspiration and expiration through the mouthpiece and the baseline slow vital capacity was noted. Now the subject was asked to take deep maximal inspiration and then expire forcefully up to its maximum point and the baseline forced vital capacity noted. After that the subject was allowed to maximally inspire and expire as quickly as they can for 15 seconds and the baseline maximal voluntary ventilation obtained. Now the subject's both feet up to the ankle joint was immersed in cold water placed in a bucket with temperature maintained between 8-10 degree Celsius and the readings of the parameters were obtained respectively at 2 minutes and 5 minutes.

Stastical analysis

Analysis of variance (ANOVA) using Statistical Package for Social Sciences (SPSS) version 20 was employed for comparing the parameters. A p value of <0.05 was considered significant.

RESULTS

Among the 30 male individuals the tidal volume showed a significant increase ($p<0.05$) from the baseline to 2 minutes and 5 minutes of the stimuli. The Inspiratory capacity showed fairly equal response from baseline to 2 minutes but increased significantly after 5 minutes of application of stimuli (Table 1). Similar results were obtained for FEV_1/FVC (Figure 2) and peak expiratory flow rate from baseline to 2 minutes to 5 minutes of the cold stimuli showing a significant decrease in the value (Table 3).

Table 1: Effect of localized cold stimuli on pulmonary function parameters.

Pulmonary function parameters	Baseline (mean±std)	2 minutes (mean±std)	5 minutes (mean±std)	P value
Tidal volume (TV)* (Litres)	(0.525±0.20)	(0.629±0.24)	(0.647±0.20)	0.000
Inspiratory capacity (IC) (Litres)	(1.93±0.39)	(1.93±0.36)	(2.04±0.31)	0.001

ANOVA; p<0.05=significant; *Tidal volume during the cold stimuli at 2 minutes and 5 minutes is significantly increased from that of the baseline.

The inspiratory reserve volume from baseline showed initial significant decrease ($p<0.05$) in first two minutes of stimuli but almost returned to its normal value after 5 minutes. Similar results were obtained for expiratory reserve volume from baseline to the period of stimuli at 2 minutes and 5 minutes. The peak expiratory flow rate

also showed a steady significant decrease ($p<0.05$) from baseline to 2 minutes of the stimuli but almost rose to its normal baseline value after 5 minutes. The forced vital capacity and forced expiratory volume in 1st sec showed a significant decrease ($p<0.05$) from baseline to 2 minutes and 5 minutes after the stimuli was applied (Table 2) (Figure 1).

Table 2: Effect of localized cold stimuli on pulmonary function parameters.

Pulmonary function parameters	Baseline (mean±std)	2 minutes (mean±std)	5 minutes (mean±std)	P value
Inspiratory reserve volume (IRV)* (Litres)	(1.377±0.49)	(1.30±0.31)	(1.39±0.29)	0.002
Expiratory reserve volume (ERV)* (Litres)	(1.20±0.79)	(0.98±0.47)	(1.05±0.59)	0.000
Forced vital capacity (FVC)* (Litres)	(3.25±0.39)	(3.03±0.36)	(2.97±0.41)	0.000
Forced expiratory volume in 1st sec (FEV1)* (Litres)	(3.03±0.41)	(2.79±0.39)	(2.76±0.45)	0.000

ANOVA; $p<0.05$=significant; *Value before the cold stimuli is significantly different from that during the duration of cold stimuli at 2 minutes and 5 minutes.

Table 3: Effect of localized cold stimuli on FEV_1/FVC and peak expiratory flow rate.

Pulmonary function parameters	Baseline (mean±std)	2 minutes (mean±std)	5 minutes (mean±std)	P value
FEV_1/FVC*	(93.1±.5.60)	(92.06±5.32)	(92.46±4.65)	0.000
Peak expiratory flow rate (PEFR) (Litres/min) *	(6.17±1.03)	(5.36±0.92)	(5.97±1.17)	0.000

ANOVA; $p<0.05$=significant; *Value during the cold stimuli of 2 minutes and 5 minutes showed a significant decrease from the baseline.

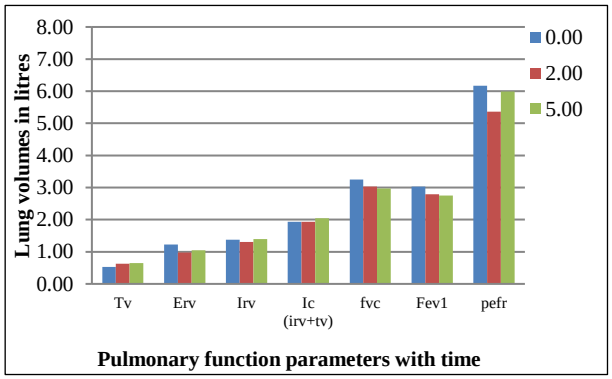

Figure 1: Change in lung volumes of pulmonary function parameters in relation to time.

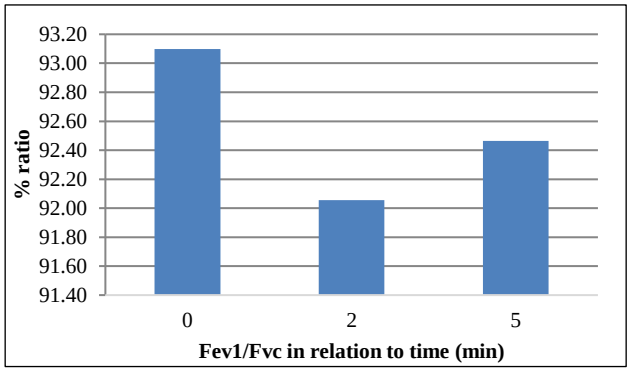

Figure 2: Change in % ratio of Fev_1/Fvc in relation to time.

DISCUSSION

After introducing cold stimuli an acute gasp response is initiated, this causes a steady state rise in the baseline ventilation which is responded by the significant increase in tidal volume and Inspiratory capacity in this study. It is a known fact that in intact humans, breathing rate usually increases when tidal volume increases. This response occurs for about few minutes and returns to its base after a brief period. During a maintained hyperinflation of the lungs induced by the cold stimuli the rapidly adapting receptors or irritant receptors, due to their superficial location in the lungs, exhibit a high frequency discharge which declines by 80% or more within the first second of inflation.[9] They are connected to myelinated vagal afferents which lie mostly in the epithelial and sub epithelial layers of the airways and are responsible for bronchoconstriction and hyperpnea as well as mucus secretion.[10] Increasing lung volume increases the caliber of the airways and as a result, resistance to airflow decreases and vice versa. Cold stimulates the efferent vagal fibers either directly or reflexively which increases airway resistance and decreases anatomic dead space secondary to airway constriction which is responded by the decrease in the Inspiratory reserve volume, expiratory reserve volume, forced vital capacity, forced expiratory volume in 1st sec. Various studies state that certain lower-airway sensory receptors can be sensitive to cold and capable of inducing bronchoconstriction in animals.[11,12] Cooling of the lower airways may induce vasoconstriction in the bronchial mucosa, followed by

reactive hyperemia and edema, which would narrow the airways after hyperpnea.[13,14] Interestingly in this study the forced vital capacity showed a significant decrease from the baseline after the introduction of the cold stimuli. This result is contradictory to the studies that showed a significant increase in forced vital capacity as a general arousal response to pain during cold stress.[5,6] Another possibility could be that a change in the bronchomotor tone i.e., relaxation and dilatation of large airways thus showing a different pathway mediated by pain in the older studies and the pathway of acute gasp response and increased ventilation induced by the localized stimuli in this study. The Fev1/Fvc ratio also significantly decreased from the baseline value indicating a more obstructive pattern developed by the exposure to cold stimuli.

CONCLUSION

Pulmonary response to cold stimuli occupies an integral portion of the body defense mechanism. The deleterious effects of pulmonary mechanics towards acute exposure to cold should be further elaborated to prevent them from causing any permanent damage to the body. Further studies and similar experiments with larger study groups and longer duration of study along with gender and ethnic comparison should be conducted to form a basis of the acute response to cold generated by the body to counteract it in all age, sex and religion.

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