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Original Article

Determining the Correlation between Ventilatory Function Tests and Heart Rate Variability in Tobacco Smokers of Haryana

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Abstract

Introduction: Tobacco smoking is responsible for 90% of chronic obstructive pulmonary disease. It is also a major risk factor for the development various cardiovascular disorders. It induces autonomic imbalance typically characterized by sympathetic hyperactivity. Extent of airways involvement can be studied by ventilatory function tests. Impact of the nicotine on cardiovascular autonomic functions can be best diagnosed using the heart rate variability (HRV). We intended to seek out any association between these two types of tests. Very few studies are available on correlation between ventilatory function tests and HRV. Hence, this study is conducted.

Materials and Methods: A total of 60 male subjects in the age group of 25–50 years - 30 tobacco chewers and 30 tobacco non users were included. Subjects with history of hypertension, diabetes, oral lesion, drug intake, etc., were excluded from the study. Ventilatory function tests were carried out using RMS Med spirometer. HRV was performed by Polyrite-26D.

Results: A significant negative correlation between $FEF_{25-75\%}$ and mean HR, and $FEF_{25-75\%}$ and LF (ms2) and also between MVV and LF. Apart from this, we got ventilatory function tests' values suggesting obstructive airway changes. HRV parameters suggested increased sympathetic tone in smokers.

Discussion: Smoke and its other constituents cause structural changes in airways leading to obstruction. Nicotine is the main culprit for autonomic disturbances. As far as correlation between the two is concerned, we failed to obtain significant association between the parameters of both tests.

Key words: Correlation, HRV, Ventilatory, Smokers, Tobacco

INTRODUCTION

Tobacco smoking is responsible for 90% of chronic obstructive pulmonary disease. It is also a major risk factor for the development of atherosclerosis, coronary heart disease, acute myocardial infarction, and sudden cardiac death.[1] It also induces autonomic imbalance typically characterized by sympathetic hyperactivity. Impact of the nicotine on cardiovascular autonomic functions can be best diagnosed using the heart rate variability (HRV). Very few studies are available on correlation between ventilatory function tests and HRV. Hence, this study is conducted.

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MATERIALS AND METHODS

The present study was conducted in the Department of Physiology, Pt. B.D. Sharma PGIMS, Rohtak. A total of 90 male subjects of age group 25–50 years were included in the study. The subjects were divided into three groups. Study was carried out after ethical approval from the institutional ethical committee. Informed consent was obtained from the subjects before proceeding with the

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procedure. Information was provided in the language familiar to the subjects.

- Smokers – 30 male volunteers who were chronic tobacco smokers for minimum 10 pack years in continuation with duration of 7 years or more
- Control – 30 male volunteers who had never used tobacco in any form (control group).

Subjects with known history or symptoms of any chronic cardiopulmonary, endocrine, or metabolic disorder. Oral lesion, any drug intake was excluded from the study.

Tests Conducted

1. Ventilatory function tests
2. HRV.

Procedure for Recording Ventilatory Functions

The ventilatory functions were recorded using the RMS Med spirometer. The Med spirometer is an instrument which measures inspiratory and expiratory parameters. The test progress is shown on the computer monitor. The subjects were instructed to apply mouth piece closely to the lips and close their nose with nose clip so as to prevent any leakage of air. Following parameters were recorded:

- Forced expiratory volume in first second (FEV_1)
- Forced vital capacity (FVC)
- FEV_1/FVC%
- Forced expiratory flow rate $_{25\text{-}75\%}$ ($FEF_{25\text{-}75\%}$)
- Maximum voluntary ventilation (MVV)
- Peak expiratory flow rate (PEFR)

Procedure for Recording FEV_1, FVC, Maximal Expiratory Flow Rate $(MEFR)_{25\text{-}75\%,}$ and PEFR

For recording of FVC, FEV_1, $MEFR_{25\text{-}75\%,}$ and PEFR, the subjects were asked to breathe in and out normally into the mouth piece. Then, the subjects were asked to take deep breath to fill lungs to maximum possible and then exhale into the mouth piece as quickly as possible. All the subjects made three such attempts and the best of them was selected.

Procedure for Recording MVV

For recording of MVV, subjects were asked to inhale and exhale as deeply and quickly for 15 s. Then, MVV was calculated in liters/minute. The subjects were instructed to stop if they felt any discomfort. Spirometric indices were calculated using best out of three technically satisfactory performances as per recommendations of American Thoracic society.[2]

Procedure for Recording HRV

For recording HRV, Digitalized Power lab 26T Polyrite D was used. Sampling rate was 256 Hz. High and low filters were set at 99 and 0.1 Hzs, respectively. The screen sweep speed was kept at 30 mm/s. For R wave detector, channel 3, that is, ECG channel 3 was used. The whole channel was selected for HRV analysis. Position of event is taken as maximum after threshold. Retrigger delay is taken as 0. Ectopics are excluded from the analysis.

Method of Measurement

HRV of subjects was measured with digitalized Polyrite D as per standards laid by Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology.[3]

Procedure

The subjects were asked to lie down on the couch and made to relax in front of the Polyrite D system. The three disposable adhesive electrodes were attached to the left arm, right leg, and left leg, respectively. The basal recording of ECG (Lead II) was taken for 5 min. From the ECG, the analysis of HRV was done automatically by Fast Fourier Transformation method.

Outcome of Variables

HRV parameters generated and selected for the study.

- Mean heart rate (beats/min)
- Mean RR interval (seconds)
- Very low frequency (VLF) (ms^2)
- Low frequency (LF) (nu)
- High frequency (HF) (nu)
- LF/HF ratio

Statistics

All the data obtained by above two procedures were analyzed by commercially available software package SPSS software. Statistical significance between smokers and controls was determined using student's unpaired *t*-test. Pearson's correlation coefficient (r) was used for correlation purpose. $P < 0.05$ was considered statistically significant and $P < 0.001$ was considered highly significant.

RESULTS

The anthropometric variations such as age, height, weight, and BMI were comparable in both the groups. All the ventilatory parameters were reduced in smokers and the reduction was highly significant except for PEFR [Table 1]. Among HRV parameters, there was mean reduction in all the parameters except RR interval and LF/HF ratio. The reduction was significant for HF. A highly significant increase in VLF and LF/HF ratio was also seen [Table 2].

Pearson's correlation between ventilatory function tests and HRV parameters smokers group shows a significant negative correlation between $FEF_{25\text{-}75\%}$ and mean HR, and $FEF_{25\text{-}75\%}$ and LF (ms^2). The negative correlation between MVV and LF is also significant. Correlation coefficient

Table 1: Ventilatory Function Tests in smokers and controls

Parameters	Smokers (n=30) (Mean ± SD#)	Controls (n=30) (Mean ± SD)	p value
FEV_1 (litres)	1.51±.64	2.87±.41	0001**
FVC (litres)	1.86±0.66	3.17±.52	0001**
FEV_1/FVC (%)	80.8±15.2	96.24±11.0	0001**
$FEF_{25-75\%}$ (litres/second)	1.85±1.24	3.96±1.06	0001**
MVV (litres/minute)	68.1±32.5	125.9±25.7	0001**
PEFR (litres/minute)	3.36±1.97	7.05±1.98	9784

*p <0.05 = significant
**p<0.001 = highly significant
SD= Standard deviation

Table: 2 Comparison of HRV parameters in smokers and controls

Parameters	Smokers (n=30) (Mean ± SD#)	Controls (n=30) (Mean ± SD)	*p* value
HR(beats/minute)	84.48±25.95	73.94±15.0	0.074
RR interval(seconds)	747±0.144	760±93.38	0.687
VLF (ms^2)	443.79±253.85	1977.13±1104.22	0.0001**
LF (nu)	58.10±41.77	59.91±12.87	0.861
HF (nu)	17.76±11.39	47.07±63.16	0.014*
LF/HF	3.37±0.61	2.02±0.92	0.0001**

*p <0.05 = significant
**p<0.001 = highly significant
SD= Standard deviation

among other parameters is very weak and statistically insignificant.

DISCUSSION

The findings of ventilatory function tests in our study suggested obstructive changes in the airways. This finding is similar to other studies.[4-6] Smoking is responsible for 90% of the obstructive diseases. After 20 years of smoking, pathophysiologic changes in the lungs develop and progress proportional to smoking intensity and duration. Chronic mucous hyperplasia of the larger airways results in a chronic productive cough in as many as 80% of smokers >60 years. Chronic inflammation and narrowing of the small airways and enzymatic digestion of alveolar walls resulting in pulmonary emphysema can result in reduced expiratory airflow sufficient to produce clinical symptoms of respiratory limitation in 15–25% of smokers.[1]

Most of the HRV parameters were also significantly reduced in smokers' group. However, there was highly significant increase in LF/HF ratio suggesting sympatho-vagal imbalance. Smoking acutely reduces baseline levels of vagal-cardiac nerve activity and completely resets vagally mediated arterial baroreceptor-cardiac reflex responses.[7]

Table 3: Pearsons Correlation between Ventilatory Function Tests and HRV parameters in smokers

Variables	Mean HR	Mean RR	VLF (ms^2)	LF (nu)	HF (nu)	LF/HF
FEV_1	-0.290	0.080	0.239	-0.446	-0.221	-0.247
FVC	-0.227	0.023	0.242	-0.313	0.159	-0.208
FEV_1/FVC	-0.192	0.155	0.177	-0.287	0.240	-0.188
$FEF_{25-75\%}$	-0.325*	0.216	0.278	-0.364*	0.126	-0.197
MVV	-0.263	0.126	0.162	-0.336*	0.063	-0.235
PEFR	-0.314	0.275	0.285	-0.261	-0.091	-0.048

*p <0.05 = significant

These effects are attributed mainly to the action of nicotine that binds to nicotinic cholinergic receptors present in the autonomic ganglia, neuromuscular junctions, and central nervous system, which on stimulation, increases the release of several neurotransmitters.[8] The nicotine and others substances found in cigarettes also stimulate the release of adrenalin into the sympathetic nervous system. In addition, the stimulation of the nicotinic receptors in the autonomic nervous system has been associated with the sympathetic excitatory effects of smoking.[9]

The respiratory cycle also affects autonomic control. The heart rate increases during inspiration and decreases during expiration, causing fluctuations in HRV.[10,11] This physiological phenomenon is known as respiratory sinus arrhythmia. However, we could not find correlation between all the parameters of ventilatory function tests and HRV. A significant negative correlation was seen between $FEF_{25-75\%,}$ mean HR, and Fand LF and between MVV and LF. Based on these findings, we can assume that pulmonary function is poorly associated with autonomic control. However, very few studies are available on it, so this domain needs further exploration.

CONCLUSION

It is well known fact that tobacco smoking leads to obstructive changes in the respiratory tract. Smoking also alters the sympatho-vagal imbalance by increasing sympathetic activity. However, as far as correlation between ventilatory function tests and heart rate variability in smokers is concerned, we could not find significant association except for very few parameters. Since, limited studies are available on corelation between these two tests, further research is required to explore this domain.

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