Original Research Article

A comparative study of heart rate variability in male heavy smokers and non-smokers

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Abstract

Background: Smoking has acute deleterious effects on the blood pressure and sympathetic tone. Smoking is one of the important lifestyle risk factor associated with increase morbidity and mortality from cardiovascular disease. Spectral analysis of heart rate variability is a sensitive tool and non- invasive method for assessment of autonomic dysfunctions in clinical diseases. **Aims and Objectives:** The aim of our study was to observe and compare 1) Total power, 2) Low frequency, 3) High frequency, 4) LF/HF Ratio, 5) SDNN (Standard deviation of all NN intervals), 6) RMSSD (The square root of the mean of the sum of the squares of differences between adjacent NN intervals) in healthy adult male smokers and non-smokers in the age group of 20 -50 years. **Materials and Methods:** The present case-control study was conducted in apparently healthy 40 male smokers and 40 non-smokers in the age group 20-50 years. Subjects with heavy smoker's category (>201) were included for the study. Written informed consent of all the subjects were taken. Spectral analysis of heart rate variability was done by taking continuous standard Lead II ECG recording by RMS polyrite- D Machine. The data consisted of Time domain and Frequency domain analysis. Data collected was analyzed statistically. **Results:** Resting pulse rate, blood pressure, Low frequency LF/HF Ratio was significantly increased in smokers. Total power, High frequency, SDNN and RMSSD were significantly decreased in smokers ($p<0.05$). **Conclusion:** Sympathetic activity is increased and parasympathetic activity is decreased in smokers as compared to non-smokers. Hence smoking causes autonomic dysfunction with predominantly sympathetic over activity making them more vulnerable to cardiovascular diseases.

Key Words: Heart rate variability, Parasympathetic activity, Sympathetic activity, Smokers.

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INTRODUCTION

Smoking causes serious public health problem with high incidence worldwide.[1] In India, smoking is a common habit particularly in young adults for psychosocial reasons and they get addicted to smoking due to nicotine[2]. It has been reported that cigarette smoking is a strong risk factor for atherosclerosis, stroke, myocardial infarction and sudden death. It is observed in many studies that smoking is one of the independent risk factor for coronary artery disease.[3]. Study conducted to investigate these clinical findings shows that changes in cardiac autonomic nerve function were due to variable intensity of cigarette smoking.[4]. In clinical practice, heart rate variability (HRV) is recognized as an effective and noninvasive tool for evaluating autonomic dysfunction of the heart.[5]. In HRV, there are frequency domain and time domain parameters. In HRV analysis, frequency domain parameters that is total power (TP), High frequency component, HF power in normalized unit indicates parasympathetic activity. Low frequency component, LF power in normalized unit indicates sympathetic activity and ratio of LF to HF power indicate sympathovagal balance. Time domain parameters that are SDNN and RMSSD indicate parasympathetic dominance. So HRV analysis was used to assess

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autonomic dysfunction in smokers as compared to non-smokers.[6]. In support to these findings it was observed that heart rate variability was lower in smokers who smoke more than 10 cigarettes per day as compared to smokers who smoke less than 10 cigarettes per day. [3] In some studies in comparison between heavy smokers and non-smokers the frequency domain parameters, LF power, LF in normalized units and LF/HF ratio were significantly increased in heavy smokers as compared to non-smokers.[7,8] Therefore this study was designed to find out the effect of cigarette smoking on cardiac autonomic functions by heart rate variability in apparently healthy heavy smokers as compared to non-smokers.

OBJECTIVES: To observe and compare heart rate variability in between 40 male heavy smokers and 40 male non-smokers of the age group 20-50 years.

MATERIALS AND METHODS

The present case –control study was conducted under the auspices of Department of Physiology.

The Smoking Index: Smoking index was calculated by multiplying the average number of cigarettes which was smoked per day and the duration of smoking in years. The number of cigarettes means, the average number of cigarettes which was smoked per day in the last seven days.[9]

1. Light smokers (Smoking index1-100)
2. Moderate smokers (Smoking index101-200)
3. Heavy smokers (Smoking index>201)

Inclusion Criteria

Healthy 40 male heavy smokers and 40 non-smokers of the age group 20-50 years.

Exclusion criteria

1. Subjects with respiratory, cardiovascular diseases and cerebrovascular diseases.
2. Taking any drug modulating the autonomic nervous system.
3. Obese and underweight subjects were excluded.

The study was approved by Institutional Ethics Committee. Subjects were briefed about the nature of the study and written informed consent was obtained. Personal details as name, age, sex, occupation, contact number were obtained. The anthropometrical data measured were height in cm, weight in kg and body mass index was calculated.[10] General precautions followed before conducting Heart rate variability Tests,

1) Before heart rate variability testing, participants were advised not be on fasting.
2) Not to take tea/coffee or other beverages which are likely to affect heart rate variability.
3) Participants were advised to remain relaxed throughout the test.
4) Not to undergo any strenuous physical exercise in 24 hours before the study.
5) To avoid smoking tobacco, chewing tobacco, alcoholic beverages for at least 24 hours prior to study.
6) To take sound sleep at previous night.

All the tests were done between 10 am and1pm.Subject was rested in supine position for 15 minute before commencement of test. Right radial artery pulse was counted for full one minute. We manually measured the systolic and diastolic blood pressure by palpatory and auscultatory method on right arm with aneroid sphygmomanometer and stethoscope. POLYRITE -D system (RMS India private limited, Chandigarh) was used for HRV analysis. HRV recording: A short term 5 minute continuous tracing of lead II electrocardiography (ECG) was taken at a speed of 30mm/sec, with POLYRITE-D. LF (ms2 and nu) was measured in 0.04-0.15 Hz range and HF (ms2and nu) was measured in 0.15-0.40 Hz range.[5] Statistical analysis was performed with SPSS software version 16.0. Values were expressed in Mean (S.D.). Difference between mean of the two groups was tested using unpaired t-test, where p value < 0.05 was considered to be statistically significant.

OBSERVATIONS AND RESULTS

Table 1: Age , anthropometric and basal cardiovascular parameters of smokers and non-smokers

Parameters	(Non-smokers) Control	(Smokers) Cases	P Value	Statistical significance
	Mean (S.D.)	Mean (S.D.)		
Age (in years)	30.23(6.59)	30.9(8.40)	0.69	NS
BMI (in kg/m2)	24.04(3.20)	23.58(3.36)	0.53	NS
Resting pulse rate (in beats/min)	71.95(1.24)	72.62(1.46)	0.02*	S
Resting- Systolic blood pressure (in mmHg)	117.4(3.82)	119.05(3.23)	0.04*	S
Resting- Diastolic blood pressure (in mmHg)	79.85(0.70)	80.9(2.51)	0.01*	S

*p value <0.05 is statistically significant.

Table1 shows that Resting pulse rate and blood pressure values were higher in smokers as compared to non-smokers, and the difference was statistically significant (p<0.05).

Table 2: Comparison of Frequency domain HRV parameters between smokers and non-smokers

HRV Parameters	(Non-smokers) Control	(Smokers) Cases	P Value	Statistical significance
	Mean(S.D.)	Mean(S.D.)		
TP(ms2)	2947.47(2796.25	1972.83(1003.84)	0.04*	S
LF(ms2)	569.85(1276.12)	1001(185.61)	0.03*	S
LF(n.u.)	78.60(5.39)	102.575(44.55)	0.001**	HS
HF(ms2)	122.95(92.09)	84.92(9.95)	0.01*	S
HF(n.u.)	21.45(5.38)	17.4(5.43)	0.001**	HS
LF:HF	3.65(3.10)	5.45(1.51)	0.001**	HS

*p value <0.05 is statistically significant. **p value<0.001 is stastically highly significant.

Table2 shows that Total power (ms2), High frequency power (ms2), High frequency power in normalized unit values were lower in smokers as compared to non-smokers, and the difference was statistically significant($p<0.05$) Low frequency power (ms2) Low frequency power in normalized unit, LH/HF ratio were higher in smokers as compared to non-smokers, and the difference was statistically significant ($p<0.05$)

Table 3: Comparison of Time domain HRV parameters between smokers andnon-smokers

HRV Parameters	(Non-smokers) Control	Cases (smokers)	P Value	Statistical significance
	Mean (S.D.)	Mean (S.D.)		
SDNN (ms)	38.61 (13.96)	30.60(3.81)	0.000***	HS
RMSSD (ms)	58.55 (182.68)	18.10(4.72)	0.000***	HS

*p value<0.05 is statistically significant **p value<0.001 is stastitically highly significant

Table 3 shows that SDNN and RMSSD values were lower in smokers as compared to non-smokers, and the difference was significant statistically ($p<0.05$).

DISCUSSION

In our study, resting pulse rate and basal blood pressure were significantly increased in healthy male heavy smokers as compared to non-smokers. George *et al.* also observed significant increase in pulse rate in smokers as compared to non-smokers which may be due to impaired baroreflex function.[11]. This indicates increased sympathetic activity in heavy smokers. In frequency domain analysis, Total power along with SDNN indicates overall autonomic regulation. In our study, total power was significantly decreased in heavy smokers as compared to non-smokers. Ferdousi *et al.* also observed that total power decreases in heavy smokers as compared to non-smokers which may be due to decreased vagal tone.[3]. This indicates decreased parasympathetic activity. High frequency (ms^2 and normalized units) indicates parasympathetic activity. In our study, high frequency significantly decreased in heavy smokers as compared to non-smokers. Similar findings were observed by Lee and Chang *et al.*.[8]. Contrary to this finding Karakaya observed significantly increased HF in smokers as compared to non-smokers on acute smoking.[12]. This indicates decreased parasympathetic activity in heavy smokers as compared to non-smokers Low frequency (ms^2 and normalized units) indicates both sympathetic and parasympathetic activity. In our study, low frequency was significantly increased in heavy smokers as compared to non-smokers. Similar findings were observed in Taralov *et al.*[13]. Contrary to this finding Lee and Chang *et al.* found that LF was significantly reduced in smokers as compared to non-smokers.[8]. This indicates that sympathetic activity is increased in heavy smokers as compared to non-smokers. LF/HF ratio indicates balance between sympathetic and parasympathetic tone. In our study we observed that LF/HF ratio was significantly increased in heavy smokers as compared to non-smokers. Similar finding was observed by Doss *et al.* and Saini *et al.*[14,15]. This indicates increased sympathetic activity in heavy smokers as compared to non-smokers. Similar findings for LF increased, HF decreased and LF/HF Ratio was increased in heavy smokers were observed in Ferdous *et al.*[16] In time domain analysis, SDNN indicates parasympathetic dominance. In our study, SDNN was significantly decreased in heavy smokers as compared to non-smokers. Similar finding was observed by Barutcu *et al.* they concluded that vagal modulation of the heart was blunted in heavy smokers during a parasympathetic maneuver.[4]. Values of SDNN in present study are in agreement with values of Cagirci *et al.*, Erdem *et al.*[6,17]. This indicates decreased parasympathetic activity in heavy smokers as compared to non-smokers. RMSSD indicates parasympathetic dominance. In our study, RMSSD was significantly decreased in heavy smokers as compared to non-smokers. Similar finding was observed in Alyan *et al.*[7].This indicates decreased parasympathetic activity in heavy smokers as compared to non-smokers. Similar findings for SDNN and RMSSD were observed by

Urooj *et al.*[18] In the present study, the significant changes in autonomic functions in heavy smokers have been suggested to be resulting from consumption of nicotine and other substances contained in cigarette smoke. In smokers there is autonomic imbalance which can be due to effect of nicotine mediated stimulation of autonomic ganglia and adrenal medulla which results in increased discharge of sympathetic fibers, increased release of catecholamine's, muscle sympathetic nerve excitation and increased peripheral chemoreceptor sensitivity. Increased sympathetic activity increases hear rate and blood pressure, myocardial contraction by action on beta1 adrenergic receptors. It also increases tone of the coronary blood vessels by acting on alpha2 adrenoceptor. In chronic nicotine users there is decreased baroreceptor sensitivity and increased sympathetic activity due to baroreflex centers are directly affected in brainstem [3]. Thus the results of the present study suggest autonomic dysfunction in heavy smokers.

CONCLUSION

Sympathetic activity is increased and parasympathetic activity is decreased in smokers as compared to non-smokers. Hence smoking causes autonomic dysfunction with predominantly sympathetic over activity making them more vulnerable to cardiovascular diseases. Therefore measuring HRV could be a screening test for detecting ANS alterations before the clinical signs appear.

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