

## Effect of Cigarette Smoking on Heart Rate Variability in Health Adult Smokers in The Age of 20-60 Years

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### Abstract :

**Background:** Smoking has acute deleterious effects on the blood pressure and sympathetic tone, and it reduces the myocardial oxygen supply. HRV is recognized as an effective and noninvasive tool for evaluating autonomic nervous system regulations of the heart.

**AIM:** The aim of our study was to observe the effect of cigarette smoking on heart rate variability in healthy adult smokers in the age group of 20 -60 years.

**Material And Method:** A cross-sectional (observational) study was conducted on 60 healthy adult smokers and 60 healthy non- smokers in the age group of 20-60 years. Heart Rate Variability (HRV) was recorded by Physio Pac (PC-2004). The data consisted of anthropometric parameters analysis, Time Domain Analysis and Frequency Domain Analysis in smokers and controls. Data collected was analysed statistically.

**Results:** The difference in mean RR, mean Heart Rate, RMSSD (the root mean, square differences of successive NN intervals), NN50 (the number of Interval Differences of successive NN intervals greater than 50 ms), pNN50 (a measure of the number of adjacent NN intervals which differ by more than 50 ms), LF/HF ratio was found to be statistically highly significant. The difference in mean STD (SDNN)( The standard deviation of intervals between normal beats), LF(low frequency) power, HF(high frequency) power, HF power% was found to be statistically significant.

**Conclusion:** This suggests increased sympathetic activity and decrease in parasympathetic activity. Smoking severely affects the cardiac autonomic functions which is evident with the study of Heart Rate Variability, so the HRV should be included in routine investigations to assess the severity of cardiac involvement in chronic smoke.

**Keywords :** HRV, Smokers, SDNN, pNN50, LF/HF ratio.

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### I. Introduction

It is known that smoking is a serious public health problem with high incidence worldwide.<sup>[1]</sup> The Health Ministry has estimated that 40% of India's health problems stem from tobacco use.<sup>[2]</sup> Heavy smoking is the commonest cause of ischaemic heart disease and death in the 30 – 40 years of age group, who are likely to be free from other myocardial risk factors.<sup>[3]</sup>

Cigarette smoking is strongly associated with an increase in coronary artery disease. Cigarette smoking increases the relative risk of coronary artery disease by 2.8-fold and 3.1-fold in young (35–64 years) men and women, respectively.<sup>[4]</sup> Smoking has acute deleterious effects on the blood pressure and sympathetic tone, and it reduces the myocardial oxygen supply. Compared to nonsmokers, smokers have increased incidence of coronary spasm and a reduced threshold for ventricular arrhythmias.<sup>[5-7]</sup> Smoking is associated with increased ventricular premature beats, and it is a strong risk factor for sudden cardiac death.<sup>[8]</sup>

Smoking makes the heart work much harder, reduces its oxygen supply, makes clots more likely to form in blood vessels, and increases the risk of potentially fatal changes in the heart beat.<sup>[9]</sup> Smoking has both short- and long-term effects on the body. Smoking just one cigarette can have immediate health effects<sup>[10]</sup>, including: Temporary increases in blood pressure and heart rate (HR); Constriction of blood vessels, which slows down blood flow around the body; and binding of CO to hemoglobin in the bloodstream. This reduces the amount of oxygen delivered to the tissues.<sup>[11]</sup>

Currently, HRV is recognized as an effective and noninvasive tool for evaluating autonomic nervous system regulations of the heart.<sup>[12]</sup> HRV describes the variation between consecutive heart beats. The rhythm of the heart is controlled by SA node, which is modulated by both sympathetic and parasympathetic branches of autonomic nervous system. Sympathetic activity tends to increase heart rate and its response is slow (few seconds). Parasympathetic activity, on the other hand, tends to decrease heart rate and mediates faster (0.2-0.6 seconds).<sup>[13]</sup>

The parasympathetic influence on heart rate is modulated by acetylcholine released by the vagus nerve on the sinoatrial node and the sympathetic influence by the release of epinephrine and norepinephrine.<sup>[14]</sup> The HRV is evaluated by two ways: time domain analysis and frequency domain analysis.<sup>[15]</sup> VLF, LF, HF power are usually measured in absolute values of power (milliseconds squared [ms<sup>2</sup>]). LF and HF can be also measured in normalized units (nu) to emphasize the controlled and balanced behavior of the two branches of the

autonomic nervous system, as well as baroreflex responsiveness to beat-to-beat variation in arterial blood pressure. Normalization of LF and HF power tends to minimize the effect of the changes in the total power on the values of these two components.<sup>[14]</sup>

According to research conducted by the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, in a time-domain analysis, higher values of the standard deviation of the normal to normal intervals (SDNN), root mean square successive difference (RMSSD), and percentage of adjacent normal R–R intervals 50 ms (pNN50) indicate stronger parasympathetic dominance. Among the frequency-domain measurement indicators, a high frequency (HF) denotes parasympathetic activity, and a low frequency (LF) is related to the activity of both the sympathetic nervous system and the parasympathetic nervous system.<sup>[16]</sup>

Nicotine increases the cardiac output by increasing both the heart rate and the myocardial contractility.<sup>[17]</sup> Smoking impairs the baroreflex sensitivity in humans, which may contribute to the smoking induced increase in the heart rate, as well as to the concomitant alterations in their variability.<sup>[18]</sup> Among the several underlying mechanism focusing the relation between cigarette smoking and acute cardiac events, cardiac sympathetic hyperactivity has been noted as a dominant feature in cigarette smokers. This observation stimulates researchers to investigate the relationship in depth between cigarette smoking and cardiac autonomic nerve function.<sup>[19,20,21]</sup>

Barutcu et al found SDNN and RMSSD values to be lower in smokers than the non-smokers. Baseline LF/HF ratio was also higher in smokers than the non-smokers.<sup>[20]</sup>

A study conducted by Gondim et al reported no significant difference between smoker and non-smoker in HRV parameters ( $p>0.05$ ).<sup>[22]</sup>

Our study was necessitated because of conflicting reports of effect of smoking on HRV. The present study aims to answer several questions regarding the acute autonomic consequences of smoking. Does smoking increases human muscle sympathetic nerve activity? Does smoking decreases vagal-cardiac nerve activity?

## II. Material And Methods

A cross-sectional (observational) study was conducted on 60 healthy adult smokers and 60 healthy non- smokers in the age group of 20-60 years in the Department of Physiology, Government Medical College, Patiala. The subjects for study were taken up from amongst the workers working in the Government Medical College and Rajindra hospital, Patiala. Heart rate of each subject was recorded by ECG monitoring, in RR mode (beat to beat), for 5 minutes at rest, in supine position, using ‘Physiopac hardware’ by ‘Medicaid’. The subject selection was based on exclusion-inclusion criteria.

**Inclusion criteria:** Healthy male smokers between the age of 20-60 years and non-smokers healthy males were taken as control.

**Exclusion criteria:** The males who were diagnosed Hypertensives, had any Renal and endocrine Disorders, had cardiovascular Disorder and Respiratory Disorder or had any history of anxiety or depressive disorder were excluded.

**Study Design:** In every case selected, thorough smoking history was taken including no. of cigarette smoked per day and moderate smokers (10-20 cigarettes/day) were included.

All the subjects were interviewed in detail. Correct procedure of the test was explained to all subjects.

**Prerequisites:** The subject was allowed to relax on a comfortable chair with the subject's back towards the recording machine. Physiopac Control unit was connected with computer systems through USB cable. Bio potential junctions boxes were connected with channel no. 1 available on the front panel of the Physiopac control unit. ECG disc electrodes were inserted in the sockets of Bio-potential junction boxes.

**Placement of electrodes:** ECG electrodes were placed on the subject. Electrodes were placed on RA (right arm), LA (left arm), LL (left leg), and RL (right leg).

**HRV Analysis:** After recording ECG clicked at transform button and selected HRV. Filled the required time to achieve the HRV data for that particular time. After the test was completed, clicked at stop button to stop the test.

## III. Results

**Table no. 1** Comparison of Time Domain Analysis in smokers and non-smokers

	Group		Significance
	Non-smokers	smokers	
	Mean	Mean	
Mean RR (s)	0.71	0.83	HS
STD (SDNN)(s)	0.03	0.00	S
Mean HR(beats/min)	87.11	72.38	HS
RMSSD(ms)	19.77	31.10	HS

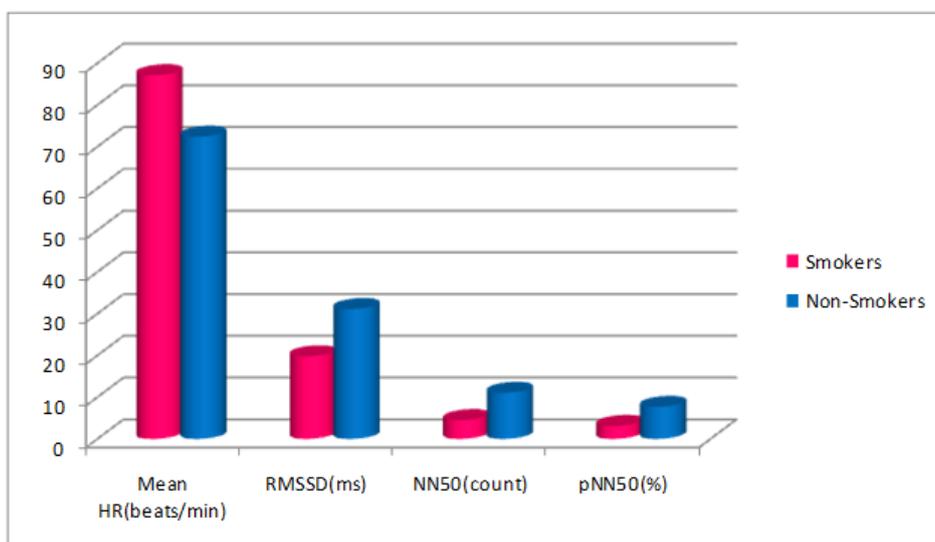
NN50(count)	4.48	11.03	HS
pNN50(%)	3.09	7.65	HS

(NS : p>0.05, S : p<0.05, HS : p<0.01)

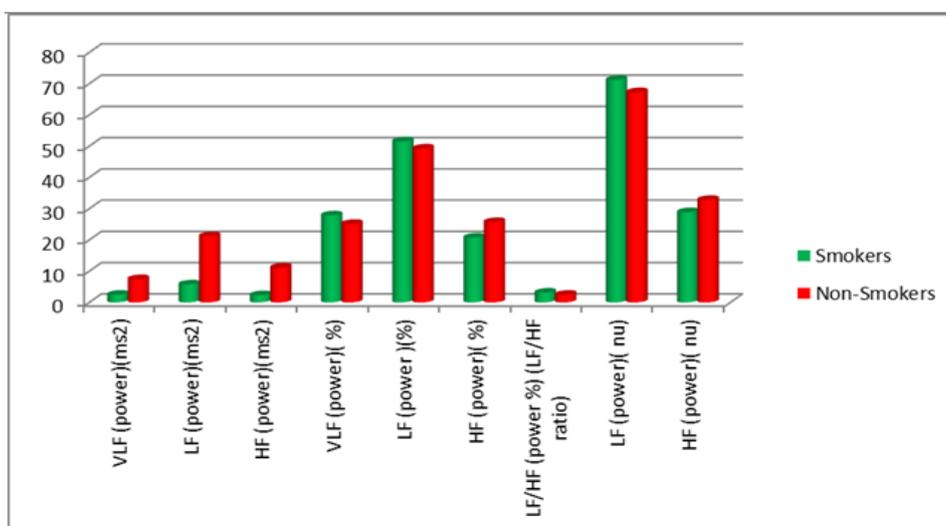
**Table no.2** Comparison of Frequency Domain Analysis in smokers and non- smokers

Parameters	Group		Significance
	Smokers	non-Smokers	
	Mean	Mean	
VLF (power)(ms <sup>2</sup> )	2.45	7.42	NS
LF (power)(ms <sup>2</sup> )	5.72	21.13	S
HF (power)(ms <sup>2</sup> )	2.28	11.12	S
VLF (power)(%)	27.81	25.11	NS
LF (power)(%)	51.48	49.21	NS
HF (power)(%)	20.72	25.69	S
LF/HF (power %)(LF/HF ratio)	3.10	2.44	HS
LF (power)( nu)	71.16	67.22	NS
HF (power)( nu)	28.84	32.78	NS

(NS : p>0.05, S : p<0.05, HS : p<0.01)



**Figure 1:** Comparison of Mean HR, RMSSD, NN50 and pNN50 in Smokers and Non-Smokers



**Figure 2:** Comparison of VLF power(ms<sup>2</sup>), LF power (ms<sup>2</sup>), HF power (ms<sup>2</sup>), VLF power%, LF power%, HF power%, LF/HF ratio, LF power (nu) and HF power (nu) in Smokers and Non-Smokers

The present study tested the HRV of healthy adult male smokers and non-smokers. Comparison of HRV parameters were done in both groups (in smokers and control). It was evident from the result as shown in Table no.1 and Table no. 2 that Mean RR, Mean STD(SDNN), Mean HR, RMSSD, , NN50, pNN50 ,Mean LF power, Mean HF power, Mean VLF power%, Mean HF power% Mean LF/HF ratio are higher in smokers as compared to the controls.

#### IV. Discussion

Our study suggest increased sympathetic activity and decrease in parasympathetic activity.<sup>[11]</sup> Sympathetic activation induced by smoking, depends on an increased release and/or a reduced clearance of catecholamines. The impairment of baro-reflex sensitivity caused by smoking further worsens the condition, due to the inability to counteract the sympathetic activation.<sup>[23]</sup> Values of Mean RR and Mean HR in the present study are in agreement with the values of Swathi et al<sup>[24]</sup> (2015), Mallikarajuna et al<sup>[25]</sup> (2015), Ohta et al<sup>[26]</sup> (2016). Values of STD (SDNN) in present study are in agreement with values of Cagirci et al<sup>[27]</sup> (2009), Saperova et al<sup>[28]</sup> (2014), Erdem et al<sup>[29]</sup> (2015). Values of Mean RMSSD in the present study are in agreement with with Alyan et al<sup>[19]</sup> (2008), Cagirci et al<sup>[27]</sup> (2009), Saperova et al<sup>[28]</sup> (2014). Values of mean NN50 in present study are in agreement with Doss et al<sup>[11]</sup> (2016). Values of mean pNN50 in present study are in agreement with Saperova et al<sup>[28]</sup> (2014), Doss et al<sup>[11]</sup> (2016). This may be due to impaired sympathovagal balance and decreased heart rate variability in healthy subjects due to smoking.<sup>[19]</sup>

The values of LF power are in agreement with Min et al<sup>[30]</sup> (2009). The values of mean HF power are in agreement with Min et al<sup>[30]</sup> (2009), Taralov et al<sup>[31]</sup> (2015), Doss et al<sup>[11]</sup> (2016). This may be due to impaired sympathovagal balance and decreased heart rate variability in healthy subjects due to smoking.<sup>[19]</sup>

Values of LF power % of our study are in agreement with the values of Behera et al<sup>[32]</sup> (2010). Values of mean HF power% of our study are in agreement with the values of Cagirci et al<sup>[27]</sup> (2009), Behera et al<sup>[32]</sup> (2010), Saperova et al<sup>[28]</sup> (2014). This may be due to impaired sympathovagal balance and decreased heart rate variability in healthy subjects due to smoking.<sup>[19]</sup>

Values of mean LF/HF ratio in the present study are in agreement with the values of Taralov et al<sup>[31]</sup> (2015), Doss et al<sup>[11]</sup> (2016), Saini et al<sup>[23]</sup> (2016). This suggest increased sympathetic activity and decrease in parasympathetic activity.<sup>[11]</sup> Values of mean LF nu and mean HF nu in the present study are in agreement with the values of Barutcu et al<sup>[20]</sup> (2005).

Our study suggest the effect of cigarette smoking on cardiac autonomic parameters by increased activity of sympathetic nervous system and decrease in vagal cardiac control which brings about changes in Heart Rate Variability. Smoking severely affects the cardiac autonomic functions which is evident with the study of Heart Rate Variability.

#### V. Conclusion

Our study concluded increased sympathetic activity and decrease in parasympathetic activity. Sympathetic activation induced by smoking, depends on an increased release and/or a reduced clearance of catecholamines. The impairment of baro-reflex sensitivity caused by smoking further worsens the condition, due to the inability to counteract the sympathetic activation. HRV should be included in routine investigations to assess the severity of cardiac involvement in chronic smokers.

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