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Original Article

Study of Heart Rate Variability in Tobacco Smokers and Smokeless Tobacco Users

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Abstract

Introduction: Tobacco use is a public health concern worldwide. It is the leading preventable agent of death in the world. It is used both in smoked or smokeless form. It is a well-known fact that tobacco smoking predisposes to atherosclerosis leading to various diseases viz. high blood pressure, myocardial infarction, and stroke. Nicotine in tobacco is found to alter the cardiovascular autonomic functions. Deleterious effects of tobacco in the smokeless form are yet to be explored. Impact of nicotine on cardiovascular autonomic functions can be best diagnosed using the Heart rate variability (HRV) assessment.

Material and Methods: The study was conducted in the Department of Physiology, Pandit Bhagwat Dayal Sharma, Post Graduate Institute of Medical Sciences, Rohtak. A total of 90 healthy adult males were included in the study. They were grouped as 30 tobacco chewers (Group I), tobacco smokers (Group II), and tobacco nonusers (Group III). Subjects with a history of hypertension, cardiopulmonary or endocrine disorder were excluded from the study. HRV was recorded and frequency domain parameters were analyzed.

Results: There is a decrease in mean values of frequency domain parameters viz. Very-low-frequency (VLF) and heart failure (HF) (nu) in both the test groups with more decrease in Group II. The decrease is highly statistically significant ($P < 0.001$) for VLF and significant ($P < 0.05$) for HF (nu) in Group II. The mean value of low-frequency LF (nu) is decreased in both test groups but decrease is more in Group I though statistically insignificant. The mean value of LF/HF ratio is increased in both test groups with more increase in Group II. This increase in LF/HF ratio is highly significant ($P < 0.001$) in Group II.

Conclusion: HRV analysis in tobacco smokers and chewers has revealed the disturbances in cardiac autonomic regulation by increasing sympathetic activity predisposing the subjects to various cardiovascular diseases. However, disturbance was more in smokers than chewers. Hence, intervention to quit tobacco even in the smokeless form is required.

Keywords: Tobacco, Smoking, Smokeless tobacco, Heart rate variability, Autonomic

INTRODUCTION

Worldwide humans have been using tobacco for about a thousand years. Smoking of tobacco dates to 5000 BC. Earlier what started as something associated with spiritual awakening eventually transformed from sacred to iniquitous, sophisticated to vulgar, an elixir to a deadly and slow poison.[1,2] It is a practice where tobacco is burnt and smoke is tasted or inhaled. This can be achieved by means of beedis, cigarettes, cigars, hookahs, etc.[3,4]

Smoke contains nicotine (a highly addictive psychoactive drug), carbon monoxide (CO), hydrogen cyanide (HCN), phenol, and several carcinogenic products such as benzopyrene, Nnitrosamine (NNK). that bind to DNA and cause many genetic mutations leading to various cancers such as bronchogenic carcinoma, cancers of the mouth, larynx, pancreas, and liver.[5,6]

The use of tobacco without burning for similar purpose is known as smokeless tobacco (SLT). It can be taken in various forms such as chewing, sniffing (naswar), khaini, zarda, plug, twist, and snus. Tobacco chewing is the most common form of SLT practiced in India.[7] SLT garnered

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immense popularity after awareness regarding hazards of smoking began to spread.

It is presumed as a safe and non-toxic alternative for smoking.[8]

Cigarette smoking is a major risk factor for the development of atherosclerosis, coronary heart disease, acute myocardial infarction (MI), and sudden cardiac death.[5] The risk of a non-fatal heart attack increases by 5.6% for every cigarette smoked.[9] However, tobacco chewing increases the risk of MI more than two folds.[10] Hypertension is significantly prevalent in both tobacco smokers and chewers. The use of tobacco either by smoking or chewing changes the lipid profile significantly.[11]

Smokeless tobacco also contains substantial amounts of nicotine, which is a cardioactive substance.[12] Impact of the nicotine on cardiovascular autonomic functions can be best diagnosed using the Heart rate variability (HRV). Various studies have either shown the effects of tobacco smoking on HRV or the effect of SLT on HRV.[11,13,14] We in our study, have compared HRV in tobacco smokers and SLT users.

MATERIALS AND METHODS

The present study was conducted in the Department of Physiology, Pt. B.D. Sharma Post Graduate Institute of Medical Sciences (PGIMS), Rohtak after getting approval from the institutional ethical committee. A total of 90 male subjects of age group 25–50 years were included in the study. The subjects were divided into three groups.

- Group I – 30 male volunteers who were chronic tobacco chewers (non-smokers) for a minimum of 10 pouch years in continuation with a duration of 7 years or more.
- Group II – 30 male volunteers who were chronic smokers (non-chewers) for minimum 10 pack years in continuation with a duration of 7 years or more.
- Group III – 30 male volunteers who had never used tobacco in any form (control group).

Exclusion Criteria

- History or symptoms of any chronic cardiopulmonary, endocrine or metabolic disorder.
- History or symptoms of any oral lesion.
- History of any drug intake.

Pack Years

Cigarette smoking was quantified in pack years. A standard package contains 20 cigarettes. This was translated into pack years as:

Pack years = number of packs per day x years smoked.

Example: 10 cigarettes per day = 1/2 pack for 10 years = 5 pack years ($1/2 \times 10 = 5$).[8]

Pouch Years

Tobacco chewing was quantified in pouch years. This was calculated as:

Pouch years = No of pouches per day x years of chewing

Example: 1 pouch per day for 10 years = 10 pouch years ($1 \times 10 = 10$).[8]

Preliminary Preparation

Consent was obtained from every subject to undergo the whole procedure. All the tests were conducted from 10 am to 1 pm to avoid diurnal variation. Overnight abstinence from tobacco use in any form was recommended. Subjects were asked to avoid tea, coffee, carbonated drinks, or heavy meals at least two hours before the test procedure. The whole procedure was explained in detail to each subject in his own language to allay any apprehension or fear. The basic parameters such as age, weight, and height of subjects were recorded. Heart rate, respiratory rate, systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial blood pressure (MAP) were taken and noted down.

After preliminary history taking and examination, 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 electrocardiograph (ECG) (Lead II) was taken for 5 min. Utmost care to minimize the movements by instructing the subjects not to move and not to speak while the recording is in progress. From the ECG, the analysis of HRV was done automatically in the machine and the printed report of HRV provided the data of required variables. The HRV is based on the duration of the time interval between two R waves, graphically represented in the form of a RR interval tachogram. The functional value of the tachogram is the duration of a RR interval (in millisecond) at a certain point of time. For analysis of HRV, there are two domains mainly "Time domain" and "Frequency domain" as described earlier. Spectral analysis of ECG was done by the Fast Fourier Transformation method. Following HRV parameters were selected for study.

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

Statistics

All the data obtained by the above two procedures were analyzed by a commercially available software package SPSS software. Statistical significance between the three groups was determined by ANOVA test and post hoc test. $P < 0.05$ was considered statistically significant and $P < 0.001$ was considered highly statistically significant.

RESULTS

The anthropometric parameters including age, height, weight, and BMI were comparable in the three groups with $P > 0.05$ Furthermore, there was no statical significance between heart rate, RR interval, SBP, DBP, and MAP in the three groups.

As evident from Table 1, the mean heart rate is increased in both Group I and Group II but the increase is more in Group II. The mean RR interval is decreased both Group I and Group II, but the reduction is more in Group I. However, changes in mean HR and mean RR are statistically insignificant. There is decrease in mean values of frequency domain parameters viz. VLF and HF (nu) in both the test groups with more decrease in Group II. The decrease is highly significant ($P < 0.001$) for VLF and significant ($P < 0.05$) for HF (nu) in Group II. The mean value of LF (nu) is decreased in both test groups but decrease is more in Group I though statistically insignificant. The mean value of LF/HF ratio is increased in both test groups with more increase in Group II, [Figure 1]. This increase in LF/HF ratio is highly significant ($P < 0.001$) in Group II.

Table 2 shows comparison in HRV parameters in Group I, Group II and Group III after post hoc analysis.

- Group I versus Group IIII- shows that there is a statistically highly significant ($P < 0.001$) difference for VLF, HF, and LF/HF ratio. There is a significant ($P < 0.05$) difference in HF- in normalized units.
- Group II versus Group III- shows that there is a statistically highly significant ($P < 0.001$) difference for VLF, HF, and LF/HF ratio. There is a significant ($P < 0.05$) difference in HF- in normalized units.
- Group I versus Group II- shows no statistically significant ($P < 0.05$) difference in the HRV parameters.

DISCUSSION

Cigarette smokers are more likely to develop both large-vessel atherosclerosis and small-vessel disease. There is a multiplicative interaction between cigarette smoking and other cardiac risk factors such that the increment in risk produced by smoking among individuals with hypertension or elevated serum lipids is substantially greater than the increment in risk produced by smoking for individuals without these risk factors. In addition to its role in promoting atherosclerosis, cigarette smoking also increases the likelihood of MI and sudden cardiac death by promoting platelet aggregation and vascular occlusion. Cessation of smoking reduces the risk of a second coronary event within 6–12 months.[8]

HRV is beat to beat variation of heart rate. It is a non-invasive method used to evaluate the autonomic regulation of heart rate. Alterations in HRV may have substantial clinical implications. Recent studies have shown decreased HRV to be associated with accelerated development of atherosclerotic coronary artery disease and increased cardiac mortality.[15] Smoking induces autonomic imbalance typically characterized by sympathetic hyperactivity. It acutely reduces baseline levels of vagal-cardiac nerve activity and completely resets vagally mediated arterial baroreceptor-cardiac reflex responses.[15]

In our study, we found in Group II, an increased heart rate even though it was statistically insignificant, indicating decreased vagal tone of heart. There was reduction in LF though statistically insignificant, suggesting impaired sympathetic activity. Furthermore, there was significant reduction in HF (nu) and highly significant increase in the LF/HF ratio suggesting that during smoking, associated with the decrease in the parasympathetic activity, there was increase in sympathetic activity.[13] Since HRV assesses the state of sympathovagal balance, it can be used to determine susceptibility to developing autonomic dysfunctions in

Table 1: Comparison of HRV in Group I, Group II, and Group III

Parameters	Group I (Chewers) (n=30) (Mean±SD#)	Group II (Smokers) (n=30) (Mean±SD)	Group III (Control) (n=30) (Mean±SD)	*P* value
HR (beats/min)	80.21±9.56	84.48±25.95	73.94±15.0	0.094
RR interval (s)	0.744±0.15	0.747±0.144	0.76±0.09	0.636
VLF (ms2)	501.58±416.83	443.79±253.85	1977.13±1104.22	<0.001**
LF (nu)	53.72±10.95	58.10±41.77	59.91±12.87	0.696
HF (nu)	22.94±6.39	17.76±11.39	47.07±63.16	0.009*
LF/HF	2.52±0.91	3.37±0.61	2.02±0.92	<0.001**

*P<0.05: Significant, **P<0.001: Highly significant, # SD: Standard deviation

Table 2: Post hoc test in HRV in Group I, Group II, and Group III

Groups	*P* value					
	Mean HR	Mean RR	VLF (ms^2)	LF (nu)	HF (nu)	LF/HF
Group I versus Group III	0.100	0.689	<0.001**	0.676	<0.001**	<0.001**
Group II versus Group III	0.061	0.344	<0.001**	0.2680	<0.001**	<0.001**
Group I versus Group II	0.673	0.584	0.749	0.489	0.372	0.246

*P<0.05=Significant, **P<0.001=Highly significant

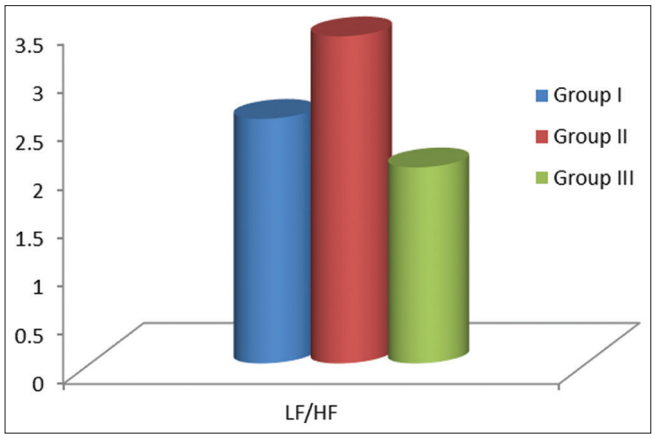

Figure 1: LF/HF in Group I, Group II, and Group III

conditions such as prehypertension and hypertension.[16] These results point towards autonomic imbalance with an increase in sympathetic tone in smokers.

In chewers, we observed, decrease in the mean RR interval and increase in the mean HR and though insignificant. Nicotine causes vasoconstriction possibly through alteration of a cyclic-GMP-dependent vasoactive mechanism. Significant rise in the diastolic blood pressure is of great concern as any increase in the diastolic BP is an indicator of hypertension.[14] Significant decline in VLF and LF (nu) suggests an increased sympathetic activity in chewers. And a highly significant decline in HF (nu) reflected decreased parasympathetic activity in. The significant increase in LF/HF ratio in Group I shows disturbances in the sympathovagal balance, with decreased vagal tone and increased sympathetic activity. Increased LF/HF ratio reflects decreased HRV.[16]

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.[14] 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.[17] There are three possible mechanisms to explain this sympathetic activation. (i) Direct effect on the central nervous system; (ii) stimulatory effect on the ganglionic sympathetic transmission that leads to a subsequent increase in the postganglionic efferent sympathetic activity; and (iii) effect on the sympathetic peripheral nervous terminations.[14,18,19]

CONCLUSION

Tobacco in any form affects the cardiovascular system adversely by altering the sympathovagal balance. Although deleterious effects of smoking were more than tobacco chewing, tobacco chewing should not be considered as a safe alternative. Awareness regarding cardiac effects of SLT is necessary. The use of tobacco in any form must be discouraged. Furthermore, HRV can emerge as a screening test for assessing cardiac autonomic disturbances in preclinical stages.

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