Comparison of Heart Rate Variability (LF/HF Ratio) and Mean Arterial Pressure in Obese Tobacco Consumers Via Different Routes for More Than a Decade: A Cross Sectional Study

Bipin Kumar¹, Tanuj Mathur², Perugu Damodar Krishna Chaitanya³

¹Senior Resident, Department of Physiology, JNMCH, AMU
²Assistant Professor, Department of Physiology, JNMCH, AMU
³Junior Resident, Department of Physiology, JNMCH, AMU

Corresponding Author: Bipin Kumar

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ABSTRACT

Introduction: Chemicals that are present in tobacco have been known to cause impaired cardiovascular health in middle aged or older population due to increase in mean arterial pressure. Long term effects of tobacco consuming via different routes is a major risk factor for autonomic neuropathy. Despite the antiquity and popularity of tobacco consuming, its adverse effect on Heart rate variability (LF/HF ratio) has not been investigated systematically in adults.

Aim: To investigate the chronic effects of tobacco on LF/HF ratio.

Materials and Methods: This case study was conducted in the Department of Physiology, Jawaharlal Nehru Medical College, Aligarh Muslim University, Aligarh, Uttar Pradesh, India. LF/HF ratio of 50 obese smokers compared with 50 obese tobacco chewers of duration more than a decade. The Heart rate variability (LF/HF ratio) was analysed with a PHYSIO-PAC SOFTWARE SYSTEM (Medical systems, Chandigarh,160002, India). Data was analysed by using unpaired t-test.

Results: In this study of comparative analysis of total 100 subjects, (50 smokers and 50 tobacco chewers were taken), statistically significant changes (p-value <0.05) were found in smokers as compared to tobacco chewers.

Conclusion: It can be concluded that changes in LF/HF ratio which cause autonomic neuropathy were more in smokers as compared to tobacco chewers.

Key words: LF/HF ratio, smokers, tobacco chewers, autonomic neuropathy

INTRODUCTION

Long term cigarette smoking is a major risk factor for cardiovascular morbidity and mortality. Previous reports suggest that smokers have a high pulse rate and high mean blood pressure, indicating sympathetic hyperactivity [1],[2],[3]. It has been shown that cigarette smoking acutely increases plasma catecholamines and cardiac norepinephrine spillover and results in an increase in blood pressure and heart rate and sympathetic outflow.
Cigarette smoking is a major cause of atherosclerotic disease and it is one of the most important risk factors for coronary heart disease along with hypertension and lipid disorders. Among the few basic instrument centering the connection between cigarette smoking and intense cardiovascular occasions, cardiac sympathetic overactivity has been noted as a prevailing element in cigarette smokers. Hayano et al [4] investigated the short and long term effects of cigarette smoking on cardiac autonomic regulation & found decreased vagal cardiac control 3 minutes after smoking and increased sympathetic activity 10-17 minutes after smoking compared to their pre smoking values. Long term effects were also examined and compared between non-smokers and smokers. They demonstrated that smoking causes an acute and transient decrease in vagal cardiac control in smokers[4].

Heart rate variability (LF/HF ratio) is the most delicate and subjective marker for both sympathetic and parasympathetic action. In clinical practice, power spectral analysis of HRV based on 5 minute ECG recording are widely used to assess autonomic regulation of cardiovascular function[5],[6],[7].

Cardiovascular disease is the leading cause of death and tobacco products estimated to directly cause 10% of all CVD worldwide [8],[9],[10]. The cardiovascular system is influenced by autonomic nervous system. Few researchers have thrown light on possible acute autonomic effects and hemodynamic changes smoking in the form of heart rate variability [HRV].

Blood pressure levels are affected by the high sodium content of smokeless tobacco, as well as by 2 of its pharmacologically active ingredients: nicotine and licorice. The sodium content differs among brands, and an increase in 24-hour urine sodium excretion by as much as 26 to 41 mEq/L has been demonstrated after the ingestion of several different types of smokeless tobacco [11]. Licorice inhibits the metabolism of mineralocorticoids and indirectly causes sodium retention, thereby increasing blood pressure levels.

The acute effects of smokeless tobacco have been documented by increases of up to 21 mm Hg in systolic blood pressure and 14 mm Hg in diastolic blood pressure and by an average increase of 19 beats per minute in heart rate [12]. These effects are likely related to activation of the sympathetic nervous system and results in autonomic neuropathy on long term usage.

Cigarette smoking has been associated with acute insulin resistance. Although the mechanism is not clearly understood, it may be due to increased levels of norepinephrine or other counter regulatory hormones, such as growth hormone or cortisol. Eliasson et al [13] demonstrated that smokeless tobacco users had higher insulin levels than nonusers, suggesting a similar link between smokeless tobacco and insulin resistance. In contrast, while growth hormone levels have been shown to rise nearly 3-fold among cigarette smokers, they do not appear to be increased in snuff users [14]. Hence, the present study was conducted with an aim to evaluate the effect of smoking and tobacco chewing on heart rate variability (LF/HF ratio).

**MATERIAL AND METHODS**

This study was conducted in the Department of Physiology, Jawaharlal Nehru Medical College, Aligarh Muslim University, Aligarh, Uttar Pradesh, India, on smokers and tobacco chewers. This study was approved from the Ethical Committee (Letter no. 249) of JN Medical College.

Total 100 sample subjects were taken- 50 as smokers and 50 as tobacco chewers. A detailed history and physical examination were carried out for every subject who entered the study as per a designed proforma and the selected cases of smoking and tobacco chewing were assessed for HRV. Both were
advised for HRV assessment and were asked to report in neurophysiology laboratory after an overnight abstinence of smoking. All the cases (100) were divided in two groups: Group 1 (n=50) smokers, Group 2 (n=50) tobacco chewers

Inclusion criteria: Only male smokers and tobacco chewers having addiction duration of more than a decade who came to the chosen study centre during the study time period were included in the study as case groups.

Exclusion criteria: Those patients who came to the study centre with any cause of neuropathy e.g., alcohol abuse, vitamin B12 deficiency, neuropathies associated with exogenous toxic agents, metal or drugs and those patients with history of trauma in the course of nerve to be examined were excluded from the study.

CLINICAL EVALUATION OF AUTONOMIC NEUROPATHY

HRV ANALYSIS

APPARATUS:
Heart rate variability was analysed with a PHYSIO-PAC SOFTWARE SYSTEM (Medical systems, Chandigarh,160002, India).

PROCEDURE

(i) Patient’s preparation:
Following instructions are given to the patient:
1. To avoid heavy meal two hours before testing.
2. No coffee, nicotine or alcohol 24 hrs prior to the testing.
3. Drugs known to affect cardiac autonomic functions like anticholinergics (including antidepressants, antihistamines, and over-the-counter cough and cold medications), 9-α - flurocorticone, diuretics, and sympathomimetic (α and β agonist) and parasympathomimetic agents may be stopped after consultation with the physician for 2 days prior to testing.
4. Clothing should be loose and comfortable.

(ii) Recording
For short term analysis of HRV, Two lead electrographic data were recorded in supine position for 5 min after 15 min of supine rest. Room temperature is maintained at 24°C. Subject is instructed to close the eyes and to avoid talking, moving hands, legs and body coughing during test –sleeping.

Quantification of HRV
The analysis of HRV is done by two methods: Frequency domain methods.

Frequency Domain Methods:
The frequency components of HRV are analyzed by using many methods. Fast Fourier Transform (FFT) is one of the commonly employed methods. The power spectrum is subsequently divided into three frequency bands: VLF- (0.001 to 0.04) Hz, LF- (0.040 to 0.15) Hz and HF- (0.15 to 0.4) Hz. Power spectral densities (PSD) are plotted in ms2/Hz against preset frequencies. Power of the spectral bands are calculated in ms2 (absolute power).

<table>
<thead>
<tr>
<th>Frequency bands</th>
<th>Frequency</th>
<th>Mediated by</th>
</tr>
</thead>
<tbody>
<tr>
<td>Very low frequency (VLF)</td>
<td>0.001-0.04 Hz</td>
<td>Possibly renin-angiotensin system</td>
</tr>
<tr>
<td>Low frequency (LF)</td>
<td>0.04-0.15 Hz</td>
<td>Parasympathetic and sympathetic influences</td>
</tr>
<tr>
<td>High frequency (HF)</td>
<td>0.15-0.4 Hz</td>
<td>Parasympathetic influence</td>
</tr>
<tr>
<td>LF/HF ratio</td>
<td></td>
<td>Sympatho-vagal influence</td>
</tr>
</tbody>
</table>
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**OBSERVATION AND RESULTS**

Table 1: Comparison of Mean Arterial Pressure in smokers and tobacco chewers

<table>
<thead>
<tr>
<th></th>
<th>Smokers</th>
<th>Tobacco chewers</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean arterial pressure</td>
<td>104.4 ± 4.23</td>
<td>94.69 ± 2.72</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>

(Independent ‘t’ test for unpaired samples was applied)

*P value <0.05: Statistically Significant

Table 2: Comparison of body mass index (BMI) in smokers and tobacco chewers

<table>
<thead>
<tr>
<th></th>
<th>Smokers</th>
<th>Tobacco chewers</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass index</td>
<td>25.90 ± 1.42</td>
<td>25.47 ± 1.96</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(Independent ‘t’ test for unpaired samples was applied)

**HRV PARAMETERS**

<table>
<thead>
<tr>
<th>PARAMETERS</th>
<th>Smokers (n=50) M±SD</th>
<th>Tobacco chewers (n=50) M±SD</th>
<th>P VALUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>LF/HF (ms²)</td>
<td>2.91±1.70</td>
<td>1.33±0.2</td>
<td>&lt;0.05*</td>
</tr>
</tbody>
</table>

(Independent ‘t’ test for unpaired samples was applied)

*P value <0.05: Statistically Significant

A significant change is seen in LF/HF ratio.

**RESULTS**
The mean arterial pressure of smokers was 104.4 ± 4.23 and that of tobacco chewers was 94.69 ± 2.72. It shows that mean arterial pressure of smokers was more as compared to tobacco chewers (Table 1).
The LF/HF ratio of smokers was 2.91 ± 1.70 and that of tobacco chewers was 1.33 ± 0.2. It shows that LF/HF ratio was more in smokers as compared to tobacco chewers (Table 2).

**DISCUSSION**
On assessment of autonomic neuropathy through heart rate variability, it was seen that heart rate variability (LF/HF ratio) was increased in smokers as compared to tobacco chewers. HRV analysis shows that smokers have an autonomic imbalance suggestive of an increased sympathetic tone or decreased parasympathetic tone which shows that smoking has more adverse than tobacco chewing which results in development of autonomic neuropathy more in smokers. Sympathetic over activity may lead to development of cardiovascular diseases like myocardial infarction, ischemic heart diseases, cardiac arrhythmias, hypertension and others. Our study shows that smokers are more prone to autonomic dysfunction as compared to tobacco chewers. This study was carried out to predict the autonomic imbalance in smokers and tobacco chewers in relation with mean arterial pressure and obesity, which will be helpful for planning the novel therapeutic and preventive approaches. Further, this study emphasis on the importance of harmful effect of smoking, tobacco chewing, increase blood pressure and obesity on cardiac autonomic neuropathy which can be prevented by appropriate lifestyle, early screening and drug interventions.

**CONCLUSION**
On assessment of autonomic neuropathy through heart rate variability, it was seen that heart rate variability (LF/HF ratio) was increased in smokers as compared to tobacco chewers, showing the involvement of cardiovascular system more in obese smokers due to increased mean arterial pressure. These results make a strong foundation for future neuropathic changes in obese smokers and tobacco chewers as observed in different
studies. Further studies are needed to confirm the findings with larger sample size.

**Declaration by Authors**

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**Conflict of Interest:** The authors declare no conflict of interest.

**REFERENCES**


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