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ASSESSMENT OF IMMEDIATE IMPACT OF CIGARETTE SMOKING ON CARDIAC SYMPTHOVAGAL BALANCE IN YOUNG HEALTHY ADULT

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ABSTRACT

Cigarette smoking has detrimental effects on the cardiovascular system, particularly by compromising the control of the Autonomic Nervous System, which results in diminished cardiovascular function. The immediate influence of cigarette smoking on cardiac sympthovagal balance was not well studied. This study investigated the short-term effects of smoking on autonomic cardiac control in healthy young adult cigarette smokers aged 18 to 30 years. After smoking a cigarette, heart rate variability (HRV) readings were recorded 5, 10, and 30 minutes later. The values were compared with initial resting HRV recordings. Both the paired and unpaired t-tests were used to analyse the data. Our study, showed a statistically significant increase in Low Frequency (LF) in msec^2, LF nu, and LF/HF measures at 5 and 10 minutes post-smoking, remaining slightly elevated even at 30 minutes. Conversely, Total Power, High Frequency (HF) in msec^2, and HF nu showed a significant decrease during the same intervals, with lower levels persisting to a smaller extent at 30 minutes. The most pronounced changes occurred 5 to 10 minutes after smoking. Smoking cigarettes suppresses cardiac vagal modulation and causes an instantaneous rise in sympathetic activity. This results in a short-term decrease in cardiac vagal activity and an enhancement of cardiac sympathetic activity. These acute effects generally reverse approximately 30 minutes post-smoking, indicating a temporary shift in sympathovagal balance towards sympathetic dominance. Hence smoking causes autonomic dysfunction with predominantly sympathetic over activity making them more vulnerable to cardiovascular diseases.

KEYWORD:- Cigarette smoking, Heart Rate Variability, LF/HF ratio.

INTRODUCTION

There are numerous established negative effects of cigarette smoking on the cardiovascular system. Smoking affects the regulation of the Autonomic Nervous System (ANS), which is one way it affects cardiovascular health.[1] The most extensively researched biologically active ingredients found in cigarettes and tobacco products are nicotine, tar, and carbon monoxide.[2] Nicotine acts as an agonist of nicotine receptors in the central and peripheral nervous system exerting a ganglioplegic effect and causing suppression of vagal (predominantly) and sympathetic control.[3] However in the latter there seems to be a direct enhancing effect at the central nuclei level resulting in an overall increase of sympathetic tone. In addition, nicotine affects the chemoreceptors of the aorta and the carotid thus causing reflex vasospasm, blood pressure elevation and respiratory rate acceleration.[4] The establishment of Heart Rate Variability (HRV) indices created a reliable and useful tool for studying the role of the autonomic nervous system in cardiac function. The literature has established the long-term effects of smoking on heart rate variability (HRV), a noninvasive measure of autonomic impulses that show the heart's ability to respond to various physiological and environmental stimuli and compensate for diseases. It also shows that smokers have lower HRV.[5] Studying the factors that promote a decrease in HRV is crucial because such conditions are linked to an increased risk of death and a susceptibility to potentially fatal arrhythmias.[5] Acutely, it is known that cigarette smoking results in the increase in blood pressure (BP), heart rate (HR), vascular resistance, sympathetic discharge and in the decrease in baroreflex activity which can alter the HRV index.[6] However, after a review of the technical literature pertinent to this research, studies on the acute effects of cigarette smoking on the HVR index, mainly during the period immediately after the act of smoking, are scarce. The present study attempts to record the short term

changes in autonomic cardiac control that are directly caused by smoking in young healthy volunteers.

MATERIALS AND METHODS

We studied 20 nonsmoker males who have never smoked and 11 smoker males who have smoked 10 cigarettes/ day or more daily for at least one year after taking a written consent. All were apparently healthy. All were in the age group of 18 -30 years .None of them had a history of heart disease, hypertension, drug consumption or any major clinical problems. After taking a standard 12-lead baseline ECG, individuals with bundle branch block, arrhythmia (except sinus arrhythmia), atrio-ventricular block, QT prolongation, ischemia and/or previous infarction were excluded from the study.

Young volunteers were chosen as it is a well-known fact that there is a progressive decrease of HRV after the fourth decade of life. The subjects were told to get enough sleep, get at least 8 hours of uninterrupted sleep the night before the experiment, have a regular breakfast the morning of the experiment, and refrain from smoking, caffeine, and alcohol for 36 hours before the experiment. Blood pressure and pulse rate were also recorded. Following five minutes of rest and comfortable strapping, all preparations for recording HRV parameters were made. Disposable Ag/AgCl electrodes in the standard lead II configuration were then used to record HRV while at rest. After that, the participant was permitted to smoke one cigarette. HRV recording was taken 5 minutes, 10 minutes and 30 minutes after the cigarette was finished. Data were compared between before smoking and after 5 minute, 10 minutes and then after 30 minutes successively. Using portable ECG acquisition equipment (Niviqure Meditech Systems, Bangalore, India), ECG data in the standard lead II configuration was obtained. This system is a digital data acquisition system with multiple channels. It is made up of a module that can record and show two data channels in real time. The system is made up of a package that analyses data obtained from a portable, specially designed biomedical electronic digital acquisition system. It may save digital data sampled between frequencies between 100 and 1024 Hz. The memory has time blocks that represent ECG signals. After that, the signals are transferred to the computer for "off-line" examination. Using a microprocessor chip with 12 bit resolution, the ECG was sampled at a rate of 1024 Hz, appropriately processed, enhanced, digitalized, and then saved on a computer. Following the analysis of the ECG waveform for potential artifacts or ectopic beats, a manual computation of the consecutive RR intervals was undertaken. Frequency domain analyses were performed on this set of RR intervals in accordance with the recommendations made by the European Society of Cardiology Task Force.[7] A sequence of R-R intervals was interpolated at 250 ms (4 Hz) to create an evenly spaced time series. Estimates of the power spectrum were calculated both parametrically and non-parametrically. Spectral estimates of RR intervals were calculated in both methods by integrating the power as total power from 0.04 to 0.40 Hz, LF power from 0.04 to 0.15 Hz, and HF power from 0.15 to 0.40 Hz. Furthermore, the frequencies at which the power in the LF band of the spectrum peaked were also recorded. These were referred to as "Peak LF Power" and "Peak Power Frequency." Due to its questionable physiological significance, the power contained in the VLF band was not calculated.

The absolute units of $msec^2$ are used to express spectral powers. Normalized units are also used to express high and low frequency power. While LF primarily represents sympathetic alterations, HF represents parasympathetic nerve activity to the heart. Also examined is the low frequency to high frequency ratio (LF/HF), which is a reflection of sympathovagal balance.

Statistical analysis

Results were presented as Mean ±Standard Deviation. For comparison between controls, and cigarette smokers Unpaired ‘t’ test was used. For the comparison performed between 5 minute intervals as well as between the two examinations, a paired ‘t’-test was used.

RESULTS

Table 1: Power spectral Analysis of HRV between Smokers and Non Smokers.

Parameters	Non Smokers Mean± SD	Smokers Mean± SD	*t value	p value
Total power ($msec^2$)	5143.10± 566.12	4783.10±671.54	2.42	< 0 .05 S
LF($msec^2$)	1984.42±452.69	2025.62±127.64	7.95	< 0 .001 HS
HF ($msec^2$)	1123.00±234.25	985.40±64.87	3.08	< 0.05 S
LF(nu)	42.71±5.23	49.53±5.77	5.17	< 0.05 S
HF (nu)	61.81±11.43	46.09±7.34	4.76	< 0.05 S
LF/HF	0.78±0.52	1.69±0.53	7.18	< 0.001 HS

*Unpaired’ test

$p > 0.05$: Not Significant (NS); $p < 0.05$:Significant (S); $p < 0.001$: Highly Significant (HS);

Table 2: Changes in the HRV Indices Before and after smoking at different periods.

Parameters	Before Smoking Mean± SD	5 min. after smoking Mean± SD	10 min. after smoking Mean± SD	30 min. after smoking Mean± SD
Total power	4783.10±671.54	3383.22± 654.91	3638.32± 670.56	4602.12±591.65

($msec^2$)		P < 0.05 S	P < 0 .001 HS	P < 0.05 S
LF($msec^2$)	2025.62±127.64	2922.52±120.90 P < 0 .001 HS	3034.54±131.21 P < 0 .001 HS	2343.26±24.78 < 0.05 S
HF ($msec^2$)	985.40±64.87	602.78± 45.78 P < 0 .001 HS	536.89±21.76 P < 0 .001 HS	889.76±32.45 P < 0.05 S
LF(nu)	49.53±5.77	66.34 ± 7.67 P < 0 .001 HS	69. 22± 9.21 P < 0 .001 HS	56.12± 4.89 P < 0.05 S
HF (nu)	46.09±7.34	36.12±4.55 P < 0 .001 HS	32.32±8.45 P < 0 .001 HS	42.90±7.32 P < 0.05 S
LF/HF	1.69±0.53	3.18± 0.53 P < 0 .001 HS	4.01±0.89 P < 0 .001 HS	2.64±0.46 P < 0.05 S

Statistical significance of the differences compared with the first periods' values using 'Paired' t test
p > 0.05: Not Significant (NS); p < 0.05: Significant (S); p < 0.001: Highly Significant (HS);

Table 3: Percentage differences in the HRV Indices after smoking at different periods.

Parameters	5 min. after smoking Percentage Difference	10 min. after smoking Percentage Difference	30 min. after smoking Percentage Difference
Total power ($msec^2$)	-34.28%	-27.14%	-3.57%
LF($msec^2$)	+36.36%	+39.97%	+14.56%
HF ($msec^2$)	-48.26%	-59.04%	-10.24%
LF(nu)	+29.56%	+33.89%	+21.81%
HF (nu)	-24.39%	-35.89%	-9.09%
LF/HF	+61.19%	+76.24%	+43.88%

Table 1 shows the power spectral analysis of HRV between smokers and Non Smokers Statistically significant increase in the LF($msec^2$), LF nu, LF/HF and significant decrease in the Total Power, HF($msec^2$), HF nu were observed in smokers.

Table 2 shows changes in the HRV Indices before and after smoking at different periods. Statistically significant increase in the LF($msec^2$), LF nu, LF/HF were seen 5 minutes after smoking and the most pronounced changes occurred 5 to 10 minutes after smoking and remains slightly elevated even after 30 minutes. Statistically significant decrease in the Total Power, HF($msec^2$), HF nu were seen 5 minutes after smoking and decrease was high at 10 minutes and continue to be in the lower side after 30 minutes. The most significant changes took place five to ten minutes after smoking. Table 3 shows a the percentage change in the power spectral analysis of HRV before and after smoking at different periods.

DISCUSSION

In our study we found a steep increase in the values of LF($msec^2$), LF nu, within 5 minutes after smoking and the highest value was recorded approximately at about ten minutes after smoking, and the values did not reach the resting value even after 30 minutes post smoking. The LF($msec^2$), LF nu of HRV reflects the sympathetic changes. Accordingly, our study found that smoking significantly increases cardiac sympathetic activity, and that this increase lasts for up to 30 minutes. The LF/HF ratio which is a mirror of sympathovagal balance also showed a higher value after smoking. Our study also found a decrease in the Total Power, HF($msec^2$), HF nu within 5 minutes after smoking and decrease was more at 10 minutes and continue to be in the lower side after 30 minutes. The above parameter reflects parasympathetic nerve activity to the heart. According to a study by Hayano and his associates, smoking significantly raises LF/HF acutely.[8] This shows that Cigarette smoking leads to an immediate increase in sympathetic activity while simultaneously suppressing cardiac vagal modulation. This results in a short-term decrease in cardiac vagal activity and an enhancement of cardiac sympathetic activity. These acute effects generally reverse approximately 30 minutes post-smoking, indicating a temporary shift in sympathovagal balance towards sympathetic dominance.

Our results are in line with earlier research that suggested smoking causes either acute sympathetic activation, parasympathetic withdrawal, or both, even though the declining tendency of HF did not reach a significant level.[9]

In smoking cessation interventions, these acute effects of smoking have also been documented, with a sharp rise in heart rate variability starting as soon as one day after quitting. It is highly likely that the parameter changes seen in our study within 5 minutes of smoking are a result of cigarette smoking because it is well known that the short-term changes in LF/HF and HF after smoking should depend on an increased release or a decreased clearance of catecholamines at the neuroeffector junctions.[10]

Acute changes in autonomic cardiac control, marked by a decrease in parasympathetic activity and an increase in sympathetic activity, as well as the recovery of these indices 30 minutes after smoking were noted by Beatriz M. M. and his colleagues when they examined the acute effects of smoking on autonomic modulation and the

post-smoking recovery of the heart rate variability (HRV) index in 25 young smokers.[11]

The acute effects of smoking on HRV indices in 15 nonsmoker volunteers were examined by Osman Karakaya et al. They found that smoking altered HRV parameters, especially in the 5–10 minutes immediately following the act of smoking. This was characterized by a decrease in HF indices and an increase in LF indices, which he attributed to a decrease in vagal modulation during smoking.[12] Additionally, Nabors-Oberg et al. demonstrated that smoking decreased the HF index. The effects of smoking on the sinus node's automaticity appear to be connected to these parasympathetic changes.[13]

The LF/HF ratio increased within 5 minutes of smoking compared to the baseline level immediately after smoking and returned to the baseline level within 15 minutes, according to Kobayashi et al.'s study on the acute effects of cigarette smoking on the heart rate variability of taxi drivers during work. They did not view LF/HF as a measure of distinct cardiac sympathetic nervous activity, but rather as an indicator of sympathetic predominance as determined by sympathetic activation or parasympathetic withdrawal.[14]

These results are also consistent with research by Narkiewikz et al. that found smoking increased sympathetic modulation.[15]

CONCLUSION

After smoking a cigarette, significant change in Autonomic Nervous System function was seen with immediate increase in sympathetic activity along with a reduction in cardiac vagal modulation. Acute cigarette smoking alters HRV parameters, particularly within the first 5 to 10 minutes after smoking, effects of smoking are negated roughly 30 minutes after smoking. This means that cigarette smoking may lead to a pronounced acute sympathovagal shift to a more powerful level of sympathetic dominance. Hence smoking causes autonomic dysfunction with predominantly sympathetic over activity making them more vulnerable to cardiovascular diseases.

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