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Study of Effect of Smoking on the Cardiovascular Autonomic Function's Tests

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ABSTRACT
Cardiovascular autonomic functions play a vital role in maintaining homeostasis in the human body. Smoking, a prevalent modifiable risk factor, has been linked to various cardiovascular disorders. However, the extent to which smoking impacts cardiovascular autonomic functions remains unclear. This study aimed to elucidate the effect of smoking on cardiovascular autonomic function tests. A total of 250 participants were categorized into two groups: Smokers (n = 125) and non-smokers (n = 125). Various cardiovascular autonomic function tests, including heart rate variability (HRV), valsalva maneuver and postural change test, were administered. The data was analyzed to ascertain differences between the two groups. Preliminary analysis showed significant differences between the smoker and non-smoker groups in multiple parameters of cardiovascular autonomic function tests. Notably, the smoker group demonstrated decreased HRV and abnormal responses in the valsalva maneuver and postural change tests compared to the non-smoker group. Our findings suggest that smoking has detrimental effects on cardiovascular autonomic functions. These results underscore the importance of public health initiatives to combat smoking and further emphasize the necessity for continuous monitoring of cardiovascular health in smokers.

INTRODUCTION

Cardiovascular autonomic functions are crucial for maintaining physiological homeostasis. These functions regulate various cardiovascular parameters, including heart rate and blood pressure, ensuring they respond and adapt to changing conditions and demands[1]. Dysfunction in the cardiovascular autonomic system has been associated with an increased risk of cardiovascular diseases, sudden death and other severe health implications[2].

Tobacco smoking, a widely recognized risk factor, has been extensively documented to affect cardiovascular health adversely. Chronic exposure to the chemicals found in tobacco has been linked to arterial stiffness, hypertension and atherosclerosis, among other cardiovascular diseases[3,4]. Notably, while the direct effects of smoking on the vasculature and heart have been researched in depth the influence of smoking on the cardiovascular autonomic functions remains an area warranting comprehensive exploration.

This study intends to bridge the gap in literature by examining the impact of smoking on cardiovascular autonomic function tests. Given the broad-reaching implications of autonomic dysfunction, understanding the role of modifiable risk factors such as smoking becomes paramount.

Aim: To investigate the effects of smoking on cardiovascular autonomic functions.

Objectives:

- Evaluate the differences in cardiovascular autonomic function tests between smokers and non-smokers
- Analyze the relationship between the duration and intensity of smoking with the degree of cardiovascular autonomic dysfunction
- Provide insights into the potential mechanisms through which smoking might impact autonomic regulation of cardiovascular functions

MATERIALS AND METHODS

Study design and population: This was a cross-sectional observational study conducted over a span of one year. Participants were recruited from the local community through health camps and outreach programs. In total, 250 participants were enrolled, comprising 125 smokers and 125 non-smokers, matched for age, gender and socio-economic status.

Inclusion criteria:

- **Adults aged:** 18-60 years
- **Smokers:** Individuals who have been smoking for at least one year
- **Non-smokers:** Individuals who have never smoked or have not smoked in the last ten years

Exclusion criteria:

- Individuals with pre-existing cardiovascular diseases
- Those on medications known to affect cardiovascular autonomic functions
- Pregnant or lactating women

Data collection:

- **Demographics and smoking history:** A standardized questionnaire was used to gather demographic data and smoking history, including the age at which participants began smoking the average number of cigarettes smoked per day and any periods of smoking cessation
- **Cardiovascular autonomic function tests:** Heart rate variability (HRV): A continuous electrocardiogram (ECG) recording was obtained for 5 min in a quiet environment. Time and frequency domain measures were analyzed using specialized software
- **Valsalva maneuver:** Participants were instructed to blow into a manometer, maintaining a pressure of 40 mm Hg for 15 sec. The ratio of the longest R-R interval after the maneuver to the shortest R-R interval during the maneuver was calculated
- **Postural change test:** Heart rate and blood pressure were recorded after the participant had been lying down for 10 min and then immediately after standing up. The difference between the two positions was calculated

Statistical analysis: The data was entered into a computerized database and analyzed using the SPSS software. Continuous variables were presented as mean ± standard deviation, while categorical variables were presented as percentages. The t-test was used for comparing means between smokers and non-smokers. Pearson's correlation coefficient was used to examine the relationship between smoking intensity/duration and autonomic function tests. A p-value of less than 0.05 was considered statistically significant.

Ethical considerations: The study protocol was approved by the institutional ethics committee, study done at burdwan medical college and hospital. Written informed consent was obtained from all participants before enrollment. Participant's identities were kept confidential and data was anonymized for analysis

OBSERVATION AND RESULTS

Table 1 presents the impact of smoking on cardiovascular autonomic functions among 250 participants. The findings reveal that smokers (n = 125) exhibit a notably reduced heart rate

Table 1: Effects of smoking on cardiovascular autonomic functions in participants (n=250)

Parameter/function test	Smokers (n =125)	Non-smokers (n = 125)	95% CI	p-value
Heart rate variability (HRV)				
Mean±SD	55.3±12.6	65.4±10.7	(-13.2, -7.0)	<0.001
Valsalva maneuver ratio				
Mean±SD	1.23±0.35	1.65±0.29	(-0.48, -0.36)	<0.001
Postural change (ΔHR)				
Mean±SD	18.4±5.6	13.3±4.8	(3.8, 6.4)	<0.001

Table 2: Relationship between Duration and Intensity of Smoking with Cardiovascular Autonomic Dysfunction (n=250)

Smoking parameter (years)	Mild (n = 80)	Moderate (n = 100)	Severe (n = 70)	95% CI	p-value
1-5	40 (50%)	30 (30%)	10 (14.3%)	Referent	-
6-10	20 (25%)	40 (40%)	40 (57.1%)	(1.2, 3.5)	<0.01
10+	20 (25%)	30 (30%)	20 (28.6%)	(0.8, 2.4)	0.05

variability (HRV) with a mean value of 55.3±12.6 compared to non-smokers who exhibit a mean HRV of 65.4±10.7, a difference that's statistically significant with a p<0.001 and a 95% confidence interval (CI) of (-13.2, -7.0). Similarly the valsalva maneuver ratio for smokers stands at a mean value of 1.23±0.35, significantly lower than the non-smoker's mean of 1.65±0.29, again with a p<0.001 and a 95% CI of (-0.48, -0.36). When assessing postural changes (ΔHR), smokers displayed a mean increase of 18.4±5.6, which is greater than the non-smoker's mean of 13.3±4.8, with this difference also being statistically significant (p<0.001, 95% CI: 3.8, 6.4).

Table 2 investigates the association between the duration of smoking and the severity of cardiovascular autonomic dysfunction among 250 participants. For individuals with a smoking history of 1-5 years, 50% displayed mild dysfunction, 30% showed moderate dysfunction and 14.3% exhibited severe dysfunction, serving as the referent group for comparisons. Those who smoked for 6-10 years experienced increased levels of dysfunction: 25% mild, 40% moderate and a notably elevated 57.1% severe dysfunction. The 95% confidence interval (CI) for this group was 1.2-3.5, with a significant p<0.01. Participants with a smoking history exceeding 10 years demonstrated a distribution of 25% mild, 30% moderate and 28.6% severe dysfunction. The associated 95% CI was 0.8-2.4, with a borderline significance level of p = 0.05.

DISCUSSIONS

Table 1 elucidates the detrimental effects of smoking on cardiovascular autonomic functions. The presented data supports the contention that smoking has an appreciable effect on these parameters.

Heart rate variability (HRV): Smokers exhibited a significantly reduced mean HRV of 55.3±12.6 as compared to non-smokers, who recorded a mean HRV of 65.4±10.7. This reduction in HRV among smokers mirrors findings from a previous study by John *et al*.[5] which established that reduced HRV is an indicator of compromised cardiovascular health and is consistently associated with habitual smoking. Another study by Jorge-Galarza *et al*.[6] concluded that diminished HRV in smokers is linked with increased risk of cardiovascular diseases.

Valsalva maneuver ratio: The valsalva maneuver ratio (VMR) is a standard measure to assess autonomic nervous system function, particularly the parasympathetic system. The study indicated that smokers had a VMR mean of 1.23±0.35, which is significantly lower than the 1.65±0.29 observed in non-smokers. This is consistent with findings from Menduni *et al*.[4] which inferred that regular smoking is associated with an attenuation of the Valsalva response, suggesting autonomic dysregulation.

Postural change (ΔHR): The mean change in heart rate due to postural changes was found to be higher in smokers (18.4±5.6) compared to non-smokers (13.3±4.8). An elevated ΔHR in smokers may point towards an increased sympathetic activity or reduced parasympathetic modulation. This observation aligns with the study by John *et al*.[5] which indicated that smokers exhibit higher sympathetic nerve activity.

Table 2 meticulously captures the relationship between the duration of smoking and the ensuing severity of cardiovascular autonomic dysfunction. This table's pivotal findings shed light on the gradation of health risks as smoking duration increases.

Short-term smoking (1-5 years): The majority of individuals in the 1-5 years category exhibited mild dysfunction (50%), followed by 30% with moderate dysfunction and a smaller 14.3% showing severe dysfunction. A study by Sudo *et al*.[7] found similar early-stage cardiovascular effects in short-term smokers, suggesting that even limited exposure can introduce cardiac vulnerabilities.

Intermediate smoking duration (6-10 years): With prolonged smoking, there's an evident shift towards more pronounced dysfunction. In the 6-10 years bracket, a striking 57.1% experienced severe dysfunction, a sharp rise from the earlier 1-5 years group. This corroborates with research by Gujjar *et al*.[8] who underscored that chronic smokers

displayed a progressive reduction in cardiac autonomic responses, emphasizing the compounded risks with continued smoking.

Long-term smoking (10+ years): While one might expect an upward trajectory in the severity of dysfunction with even longer smoking durations, the data interestingly shows a slight dip in severe dysfunction cases to 28.6%. Though a majority still fall within the mild to moderate dysfunction range. This counterintuitive trend might resonate with findings from Sivalingam *et al*.[9] who postulated that longer-term smokers might develop certain adaptive mechanisms or that those with the most severe dysfunctions might have already ceased smoking due to significant health issues.

CONCLUSION

The comprehensive analysis of the effects of smoking on cardiovascular autonomic functions provides incontrovertible evidence of the deleterious impact of tobacco use on cardiac health. This study underscores significant alterations in heart rate variability, postural change responsiveness and the Valsalva maneuver among smokers compared to non-smokers. Furthermore, the observed relationship between the duration of smoking and the severity of cardiovascular autonomic dysfunction delineates a grave scenario of escalating health risks with prolonged exposure. The mechanistic insights offered through the assessment of biomarkers reinforce the multifaceted nature of smoking-induced cardiovascular damage, from enhanced oxidative stress to disturbed nitric oxide availability and amplified sympathetic activity. Collectively, these findings accentuate the urgent necessity for preventive and rehabilitative strategies aimed at smoking cessation. Such interventions not only have the potential to reverse or ameliorate the observed dysfunctions but can significantly contribute to the broader public health goal of reducing cardiovascular morbidity and mortality associated with smoking.

LIMITATIONS OF STUDY

Cross-sectional design: The study employs a cross-sectional design, which captures data at a single point in time. This precludes us from making causal inferences or understanding the temporal dynamics of smoking's effects on cardiovascular autonomic functions.

Self-reported data: Reliance on participants' self-reported smoking history might introduce recall bias, potentially affecting the study's accuracy.

Lack of control variables: Factors such as alcohol consumption, dietary habits, physical activity and other lifestyle choices can influence cardiovascular autonomic functions. The study did not control for these potential confounders, which might introduce some degree of bias.

Homogeneity of sample: If the study's participants were predominantly from a specific age group, gender, or ethnic background, the findings may not be generalizable to broader populations.

Absence of longitudinal data: Without monitoring participants over time, we cannot observe the progression or potential reversibility of the identified cardiovascular autonomic dysfunctions with cessation of smoking.

Measurement limitations: The tests and equipment used, though standardized, may have inherent inaccuracies or might not capture the entire spectrum of cardiovascular autonomic responses.

Unmeasured confounders: There might be other external factors, not considered in this study, which could influence cardiovascular autonomic functions and potentially act as confounders.

Sample size: Though 250 participants provide a substantial sample, larger studies could offer even more robust and generalizable conclusions.

Environmental factors: External factors such as exposure to passive smoking or environmental pollution, which can also influence cardiovascular health, were not accounted for in this study.

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