SSR Institute of International Journal of Life Sciences
ISSN (O): 2581-8740 | ISSN (P): 2581-8732
Salam *et al*., 2026
doi: 10.21276/SSR-IIJLS.2026.12.1.39

Original Article

open access

Evaluation of Sympathetic and Parasympathetic Dysfunction in Smokers Using Cardiovascular Autonomic Function Tests

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Received: 11 Sep 2025/ Revised: 27 Oct 2025/ Accepted: 26 Dec 2025

ABSTRACT

Background: Cigarette smoking is a significant risk factor that can be prevented to cause cardiovascular morbidity and mortality. Changes in the regulation of the autonomic nervous system, especially the lack of balance between the sympathetic and parasympathetic activity, are assumed to be a significant process behind the smoking-associated cardiovascular risk. This study was done to evaluate the sympathetic and parasympathetic activity in smokers.

Methods: This cross-sectional study included 80 males aged 20-30 years, randomly divided into two groups: smokers and non-smokers, with 40 in each group. The smoking history of the smokers was more than five years, and was further classified as light smokers, moderate smokers, and heavy smokers according to the smoking index. Parasympathetic activity was determined from resting heart rate, expiration-to-inspiration (E:I) ratio, Valsalva ratio, and 30:15 ratio, whereas sympathetic activity was assessed by sustained handgrip and postural hypotension.

Results: Smokers exhibited significant parasympathetic dysfunction compared with non-smokers, with lower I:E ratio, Valsalva ratio, and 30:15 ratio, alongside higher resting heart rate (all $p<0.01$). Sympathetic tests showed no significant intergroup differences ($p>0.05$). Within smokers, increasing smoking index was associated with progressively higher resting heart rate and reduced E:I and Valsalva ratios, indicating dose-related parasympathetic impairment ($p<0.05$), whereas sympathetic responses across smoking categories remained comparable ($p>0.05$).

Conclusion: The study concluded that chronic smoking had resulted in cardiovascular autonomic dysfunction, which is dominated by the parasympathetic activity and a relative deficiency of autonomic control.

Key-words: Parasympathetic dysfunction, Sympathetic function, Smoking index, Cardiovascular autonomic function, Autonomic function tests

INTRODUCTION

Smoking is the main cause of mortality and morbidity worldwide. The World Health Organization estimates that 100 million tobacco users were killed in the 20th century. By the early 2030s, tobacco-related deaths would be approximately 100 million every year.

How to cite this article
Salam MP, Haque A, Hasan N. Evaluation of Sympathetic and Parasympathetic Dysfunction in Smokers Using Cardiovascular Autonomic Function Tests. SSR Inst Int J Life Sci., 2026; 12(1): 9242-9248.

Access this article online
https://iijls.com/

Smoking increases coronary heart disease, atherosclerosis, sudden cardiac death, and acute myocardial infarction. Loss of cardiovascular mortality and morbidity is associated with smoking cessation. Cardiovascular activity is controlled by the autonomic nervous system, which requires a proper balance between its two components: the parasympathetic and sympathetic nervous systems [1,2].

The autonomic nervous system regulates smooth muscles, the heart, and the glands (endocrine and exocrine). The autonomic nervous system innervates organs, which, in itself, has activity. Autonomic innervation is solely used to regulate the activity of these

doi: 10.21276/SSR-IIJLS.2026.12.1.39

organs according to requirements. The hypothalamus, cerebellum, frontal cortex, and limbic system are brainstem centers that control the autonomic nervous system. The hypothalamus is most significant; indeed, Sherrington rightly described it as the Head Ganglion of the autonomic nervous system [3]. The sympathetic division, besides serving minimal functions such as maintaining blood pressure (BP) and body temperature, also assists the individual in adapting to emergencies. Sympathetic stimulation results in relaxation of accommodation and pupil dilation, speeding of the heartbeat, rise in BP, and augmented blood flow to muscles and reduced blood flow to skin and abdominal viscera, elevation of plasma glucose and free fatty acids. Based on these effects, Cannon referred to this emergency response as the fight-or-flight response [4,5].

The cardiovascular effects of smoking in man and experimental animals have been those brought about by nicotine itself. A puffing smoker typically receives about 0.1-0.2 mg of inhalation and 1-2mg per single cigarette. The action of nicotine on the cardiovascular system results from its multiple effects on various components of the system. The low level of nicotine concentration activates sympathetic ganglia, and the high levels paralyze them. The same occurs with the parasympathetic ganglia, but less sensitively [6]. It has nicotinic receptors at both the neuromuscular junction and the autonomic ganglia of skeletal muscle. Under some conditions, nicotine will show a nicotinic blocking effect; hence, the effect of nicotine will differ with dose and route of administration. Nicotine enhances the discharge of Epinephrine from the adrenal gland and other chromaffin tissues and the release of norepinephrine from the hypothalamus and adrenergic synapses. Physiological changes in the cardiovascular system from smoking include changes in heart rate, rhythm, BP, cardiac output, myocardial oxygen and nutrient demand, peripheral vascular resistance, ventricular fibrillation threshold, and coronary artery flow regulation [7].

The objective of the present study is to assess autonomic sympathetic and parasympathetic activity in smokers using standardized cardiovascular autonomic function tests. The present study aims to make a contribution to the improved knowledge of the subclinical cardiovascular consequences of smoking, as well as to highlight the significance of early diagnosis of autonomic dysfunction in the high-risk group of smokers.

MATERIALS AND METHODS

Study design- This study consisted of 80 male subjects of age in the range of 20 to 30 years, of which 40 smokers formed the test group and 40 non-smokers formed the control groups were selected in this study. The study sample comprised patients who visited the medicine OPD. They were given prior informed written consent after being informed by the researcher about the procedure and the objectives of the study tests. This study design was a cross-sectional survey design. The ethics committee had to provide ethical approval.

Inclusion and exclusion criteria

Test group- This study takes the smokers who have a history of over 5 years of smoking and excludes the smokers who have a history of less than 5 years of smoking. Also, the subjects who are known cases of hypertension, type 2 diabetes, peripheral neuropathy, stroke, and other cardiovascular diseases are not eligible for this study.

Control group- The control group in the current study was taken to be the subjects who never smoked in their life and who did not have any other kind of addiction related to tobacco or other forms of addiction. Subjects known to have hypertension, type 2 diabetes, peripheral neuropathy, stroke, and other cardiovascular diseases are not included in the current study.

Smoking Index- The smoking index is an easy parameter utilized to determine the smoking exposure quantitatively, as well as to quantify the risk ratio of diseases related to smoking. The smoking index is obtained by dividing the average number of cigarettes smoked per day by the number of years of smoking. The number of cigarettes is the average number of cigarettes smoked over the previous 7 days.

Based on the Smoking Index, the smokers were categorized into:

1. Light smokers (Smoking index 1-100)
2. Moderate (Smoking index 101-200) smokers.
3. Heavy smokers (Smoking index >201)

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The cardiovascular autonomic function tests were performed using a CANWIN ANALYSER instrument in the department. It was produced in Hyderabad, India. Being fully automatic, it is unnecessary to have a manual recording and reading. The arrangement of time-domain waveform analysis and blood pressure measurements is built in, which eases the process of performing all six autonomic function tests.

The cardiovascular autonomic function test included the following assessments:

Parasympathetic function tests

Resting Heart Rate: Subjects will be asked to lie in the supine position and rest for 15 minutes. Then, a 1-minute ECG recording was made. The ECG was used to determine the resting heart rate.

The Expiration: Inspiration Ratio (E:I Ratio): The subjects are required to perform deep breaths of 5 seconds, followed by deep exhales of 5 seconds. These 3 cycles were recorded in the ECG. The principle of this test is the sinus arrhythmia in each respiratory cycle, which is determined by the change in vagal tone.

The 30:15 Ratio: (Response to standing). The subjects were asked to lie in a comfortable position on the couch, then stand. Their heart rate was monitored at the 15th and 30th beats following standing.

Ratio of the Valsalva: Subjects are requested to sit in a comfortable position. The ECG was taken of their resting heart rates. Nose clips and mouth pieces would be slipped in their noses and between their teeth and lips, respectively. The other terminal of the mouthpieces was attached to mercury manometers. Subjects were asked to blow into the mouthpieces, and a pressure of 40 mmHg was maintained for 15 seconds. The ECG was constantly recorded.

The Valsalva ratio was calculated as the following ratio: the longest RR interval after the strain/the shortest RR interval during the strain.

Sympathetic function tests

Postural Hypotension Test (Postural challenge test): Participants were asked to lie supine and relax for 15 minutes, and their blood pressure was measured. After that, they were asked to stand up, and their blood pressure was measured after 1 minute and immediately.

Sustained Handgrip Test: Subjects were instructed to place the spring dynamometers in their left hands and squeeze them as hard as possible, and the values were recorded. Afterwards, they were requested to hold the spring dynamometers with their left hand, squeeze them to 30 percent, and hold them for 4 minutes. The diastolic blood pressure increased at that point, immediately before the handgrip loosened. The test is a sign of the sympathetic inadequacy.

Statistical Analysis- The data obtained were processed using SPSS software. Statistical analysis was performed using appropriate descriptive and inferential methods. Continuous variables were expressed as mean±standard deviation. Comparisons between smokers and non-smokers were conducted using the Z test to assess differences in autonomic function parameters. A $p<0.05$ was considered statistically significant, with $p<0.01$ indicating high statistical significance. Among smokers, subgroup comparisons based on smoking index were analysed using one-way analysis of variance to evaluate trends across light, moderate, and heavy smokers. Post hoc comparisons were applied where relevant. All analyses were carried out using standard statistical software.

RESULTS

Table 1 indicates a substantial parasympathetic autonomic dysfunction in smokers as opposed to non-smokers. The inspiratory-to-expiratory (I:E) ratio, the Valsalva ratio, and the 30:15 ratio were significantly lower in smokers, indicating reduced vagal modulation and fewer reflex parasympathetic responses to stimuli. Comparatively, resting heart rate was much elevated in smokers, suggesting low parasympathetic tone and a relative increase in sympathetic tone. The Z tests for all parameters were high, and the p-values were less than 0.01, indicating that the differences between smokers and non-smokers are statistically significant at very high levels.

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Table 1: Comparison of the mean values of parasympathetic functionality test in smokers and non-smokers

Parasympathetic function tests	Smokers (N=40)	Non-smokers (N=40)	Z test value	p-value	Significance
Inspiration: Expiration ratio	1.28±0.31	1.51±0.26	8.59	<0.01	S
Resting heart rate (/min)	85.12±7.91	75.12±8.99	8.47	<0.01	S
Valsalva ratio	3.72±1.19	4.82±2.24	8.72	<0.01	S
30:15 ratio (Response to standing)	0.87±0.28	1.23±0.62	5.2	<0.01	S

S= Significance

Table 2 compares sympathetic autonomic functioning in smokers and non-smokers and presents mixed results from the tests. The rate of rise in diastolic blood pressure during the sustained handgrip test was lower in smokers than in non-smokers, suggesting a reduced sympathetic response in smokers; however, the Z value of 4.59 indicated that the difference was not significant. Contrastingly, systolic blood pressure declined during postural hypotension testing in both groups, and the difference was not statistically significant ($p>0.05$), suggesting that smokers maintain sympathetic vasomotor control during postural changes.

Table 2: Comparison of the mean values of the sympathetic functionality test in smokers and non-smokers

Sympathetic function tests	Smokers (N=40)	Non-smokers (N=40)	Z-test value	p-value	Significance
Sustained test for handgrip (increase in diastolic pressure) (mmHg)	5.34±4.8	10.34±5.34	4.59	<0.01	NS
Test for postural hypotension (decrease in systolic pressure) (mmHg)	8.92±5.39	8.23±6.54	0.25	>0.05	S

NS= Non-Significance; S= Significance

Table 3 demonstrates a graded association between smoking index and parasympathetic function parameters among smokers. Resting heart rate increased progressively from light smokers (77.32±6.42/min) to moderate smokers (80.09±2.89/min) and heavy smokers (81.78±4.35/min), indicating a rising basal sympathetic tone with increasing smoking exposure. However, the intergroup difference did not reach statistical significance ($p>0.05$). The E:I ratio declined across smoking categories, with the highest values observed in light smokers (1.29±0.11) and the lowest in heavy smokers (1.09±0.21), reflecting impaired cardiovagal function with a higher smoking index ($p<0.05$). Similarly, the 30:15 ratio was reduced from light (1.13±0.24) to moderate (0.95±0.09) and heavy smokers (0.98±0.08), suggesting attenuation of parasympathetic reactivity, though the difference between moderate and heavy smokers was marginal ($p>0.05$). The Valsalva ratio declined with increasing smoking index, from 3.78±2.83 in light smokers to 1.96±2.56 in heavy smokers, indicating substantial impairment of autonomic reflexes in heavy smokers ($p<0.05$).

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Table 3: Association between smoking index and parasympathetic function tests in smokers

Smoking Index	Resting heart rate (/min) mean±SD	E: I ratio mean±SD	30: 15 Ratio mean±SD	Valsalva ratio mean±SD
01-100 (light smokers) (N=16)	77.32±6.42	1.29±0.11	1.13±0.24	3.78±2.83
101-200 (moderate smokers) (N=13)	80.09±2.89	1.12±0.19	0.95±0.09	3.24±2.89
Above 200 (heavy smokers) (N=11)	81.78±4.35	1.09±0.21	0.98±0.08	1.96±2.56

Table 4 compares sympathetic function tests across smoking index categories and reveals relatively modest variations. The test for postural hypotension showed slightly higher mean values in heavy smokers (9.82±2.87) than in light (7.95±3.54) and moderate smokers (7.45±3.78), suggesting a tendency toward an exaggerated blood pressure response with increasing smoking burden; however, this difference was not statistically significant ($p>0.05$). The sustained handgrip test demonstrated comparable mean responses across light (6.26±3.2), moderate (6.29±2.72), and heavy smokers (5.92±2.59), indicating no meaningful association between smoking index and this parameter of sympathetic function ($p>0.05$). Overall, while parasympathetic dysfunction showed a clearer dose-related decline with increasing smoking index, sympathetic function tests exhibited only minimal and statistically non-significant differences between groups.

Table 4: Comparison of sympathetic function tests and smoking index in smokers

Smoking Index	Test for postural hypotension mean±SD	Test for sustained handgrip mean±SD
01-100 (light smokers) (N=16)	7.95±3.54	6.26±3.2
101-200 (moderate smokers) (N=13)	7.45±3.78	6.29±2.72
Above 200 (heavy smokers) (N=11)	9.82±2.87	5.92±2.59

DISCUSSION

A study aimed to investigate the impact of smoking on the cardiovascular autonomic activity in smokers. It was observed that there was no significant difference between the mean values of the Postural hypotension test, and that there was a highly significant difference between the mean values of the Sustained handgrip test in the smokers and the non-smokers [8].

Smoking cigarettes has been found to cause more activity in the sympathetic nervous system. The variation in cardiac autonomic functioning of heavy smokers and non-smoker controls was studied using heart rate variability (HRV). Heavy smokers have vagal modulation of the heart that is suppressed, especially when performing a parasympathetic maneuver.

Adverse events attributed to cigarette smoking may be partly related to blunted autonomic control of the heart [9].

Preventable cardiovascular disease (CVD) is a major cause of cigarette smoking. The ANS is an important part of cardiovascular regulation, which is why its dysfunction is associated with its pathogenesis. The physiological change in the interval between successive heartbeats, known as HRV, is an effective, non-invasive indicator of cardiac autonomic activity. Reduced HRV indicates inadequate sympathovagal balance and is a predictive variable on its own of adverse cardiovascular events. Although smoking is known not to influence the ANS, a more detailed characterization of the change of HRV in chronic smokers is still relevant. Long-term cigarette

doi: 10.21276/SSR-IIJLS.2026.12.1.39

smoking has a close connection with serious changes in HRV, including the decrease in general variability, sympathetic withdrawal, and sympathetic dominance. These results show subclinical cardiac autonomic dysfunction among smokers, a potentially pivotal process in explaining why they are at high risk of cardiovascular morbidity and mortality [10].

One of the best risk factors for predisposing to cardiovascular morbidity and mortality is cigarette smoking. Autonomic dysfunction caused by chronic smoking puts smokers at risk of developing cardiovascular complications. A study compared cardiovascular autonomic function in smokers. Cardiovascular autonomic function tests are effective, non-invasive, and easy to implement. These simple tests will help identify early autonomic nervous system dysfunction before clinical symptoms manifest. According to the study, smoking negatively impacts the cardiovascular autonomic functions [1].

A cross-sectional study was planned to evaluate the impact of active smoking on ANS functioning in the form of HRV or DBT abnormalities. An electrocardiogram was recorded at rest for 5 min and during forced breathing using metronomic pacing. The HRV and DBT were determined in accordance with accepted standards. Finally, the relatively short-term smoking of healthy adults does not appear to cause any major impairment of ANS functioning. However, the effects of smoking appear to be increased with the cumulative exposure burden [11].

HRV was measured using time-domain techniques in healthy cigarette smokers. Reduced HRV generalization and impaired cardiac vagal regulation are experienced in seemingly healthy cigarette smokers [12].

The role of tobacco in having detrimental effects on health is well documented. Nonetheless, the impact of low doses of smoking has not been studied properly. Although heavy smoking has reduced because of public awareness, light smoking is being indulged in because it is believed not to be as unhealthy. As light smoking is not easy to define, a study was done to examine the impact of 1- 10 cigarettes on cardiovascular autonomic parasympathetic activities. The research established that the parasympathetic activity was significantly reduced, and it also had a negative correlation with the smoking period [13].

A study was conducted to identify and compare the autonomic function tests of male cigarette smokers and healthy non-smoking control subjects. The study concluded that it was related to cigarette smoking, leading to an increase in sympathetic and decreased parasympathetic activity compared to control healthy subjects [14].

A study was conducted to examine the interrelationships among Autonomic Function parameters in current smokers versus non-smokers. Smoking is so disabling that the first step of the program to enhance one's health is always to quit the habit first. Autonomic failure can manifest clinically in the late stages of the disease. Autonomic dysfunction can be evaluated early to avoid dangerous complications associated with smoking [15].

CONCLUSIONS

The study concludes that chronic smoking results in severe cardiovascular autonomic dysfunction, which is largely dominated by the loss of parasympathetic activity and a relative autonomic control deficiency. Resting heart rate was greater, and parasympathetic reflex reactions were significantly less in smokers than in non-smokers, and dose-dependent deterioration of the parasympathetic response with an increase in the smoking index. These results show that parasympathetic dysfunction is an early cumulative smoking occurrence, even in young adults, and cardiovascular autonomic functional testing is important to detect the need for smoking cessation to avoid the development of long-term cardiovascular complications.

CONTRIBUTION OF AUTHORS

Research concept- Md Perwez Salam, Ansarul Haque, Noor Hasan
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doi: 10.21276/SSR-IIJLS.2026.12.1.39

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