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Original Research Article

A Case Control Study Assessing the Heart Rate Variation between Smokers and Non-Smokers

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Abstract

Aim: To compare the heart rate variability between male heavy smokers and non-smokers

Materials and Methods: The SKMCH Department of Physiology at Muzaffarpur, Bihar, India, performed this case-control research. The Smoking Index: The index was developed by multiplying the average daily cigarette consumption by the years smoked. The amount of cigarettes is the average daily consumption during the last week. Light (Smoking index1-100), moderate (101-200), and heavy (>201) smokers were considered. This research comprised 50 healthy male heavy smokers and 50 non-smokers aged 20-50.

Results: Resting pulse rate and blood pressure were significantly greater in smokers compared to non-smokers ($p<0.05$). Smokers had significantly lower levels of high frequency power in normalised unit values compared to non-smokers ($p<0.05$) in terms of total power (ms2) and high frequency power (ms2). Smokers had greater LH/HF ratios compared to non-smokers, with a significant difference ($p<0.05$). Smokers had significantly lower SDNN and RMSSD values compared to non-smokers ($p<0.05$).

Conclusion: Compared to non-smokers, those who smoke have less parasympathetic activity and more sympathetic activity. Therefore, they are more prone to cardiovascular disorders caused by autonomic dysfunction, namely sympathetic overactivity, which is caused by smoking. Measurement of HRV may therefore serve as a screening tool for the early detection of ANS changes prior to the onset of clinical symptoms.

Keywords: Heart rate variability, Parasympathetic activity, Sympathetic activity, Smokers.

Introduction

The HRV is a non-invasive way to monitor the function of the autonomic nervous system (ANS), which is responsible for maintaining a balance between the parasympathetic and sympathetic nervous systems. Heart rate variability (HRV) analysis has become more popular as a method for evaluating cardiovascular health. Pathological disorders are generally linked to decreased parasympathetic activity and greater sympathetic dominance, which are both indicated by lower HRV. One of the leading causes of death worldwide, smoking has a negative impact on the heart's ability to pump blood and regulate its own heart rate. [1]

The risk of cardiovascular disease and death is significantly elevated among heavy smokers. Smoking impairs autonomic regulation of heart function due to endothelial dysfunction, inflammation, oxidative stress, and carbon monoxide, two of the hazardous components of tobacco smoke. Cigarette smoke contains the active ingredient nicotine, which raises blood pressure and heart rate by activating the release of catecholamines. Tobacco poisoning, including nicotine, alters the autonomic balance so that the sympathetic nervous system is more dominant, interfering with the regular fluctuation of heart rate. [2,3]

When comparing HRV in smokers and non-smokers, researchers have shown that smokers often have lower HRV. If HRV drops below this level, it means autonomic function is weakened and the risk of cardiovascular disease is higher. For example, lower time-domain and frequency-domain HRV measurements are indicative of greater sympathetic activity and decreased parasympathetic activity in chronic smokers, according to the research. [4]

In addition, heart rate variability (HRV) analysis can pinpoint the first signs of cardiovascular failure in smokers. Reduced HRV may be used as a predictor for cardiovascular events in smokers

since it occurs before clinical symptoms do. Contrarily, non-smokers often have elevated HRV, which is indicative of a more balanced autonomic system and reduced risk of cardiovascular disease. [5]

In order to better understand how long-term smoking affects autonomic function, this research will compare HRV in men who smoke heavily with those who do not. These distinctions help us comprehend the dangers of smoking to our cardiovascular systems and the need of quitting the habit for greater autonomic health and lower rates of cardiovascular disease.

Materials and Methods

For this case-control study, researchers spent a year at SKMCH's Department of Physiology in Muzaffarpur, Bihar, India. Multiplying the average daily cigarette smoking rate by the total number of years of smoking yielded the smoking index. Cigarette use during the last seven days is represented by the average daily cigarette consumption. A light smoker was defined as having a smoking index between 1 and 100, a moderate smoker as having an index between 101 and 200, and a heavy smoker as having an index more than 201.

Inclusion Criteria

- Healthy 50 male heavy smokers and 50 non-smokersof the age group 20-50 years.

Exclusion Criteria

- Subjects with respiratory, cardiovascular diseases andcerebrovascular diseases.
- Subjects taking any drug modulating the autonomic nervoussystem.
- Obese and underweight subjects were excluded.

Before any subjects gave their written informed permission, they were told on the study's purpose. Name, age, sex, profession, and phone number were among the personally identifiable information gathered. Body mass index, weight in kilogrammes, and height in centimetres were the anthropometric variables assessed.[10] Measures taken generally prior to doing heart rate variability tests. It was recommended that individuals refrain from fasting and avoid consuming tea, coffee, and other liquids that might impact heart rate variability before the testing. Keep calm and collected throughout the exam; don't exercise too hard in the hour leading up to the study; don't smoke or chew tobacco; don't drink alcohol for at least that long; and have a good night's sleep the night before. Between 10 a.m. and 1 p.m., all analysis was conducted. The subject was allowed to lie down for fifteen minutes prior to the start of the test. We counted the pulse in the right radial artery for a whole minute. We used an aneroid sphygmomanometer and a stethoscope to take the patient's systolic and diastolic blood pressure readings on their right arm. In order to analyse HRV, the POLY-RITE -D system was used for recording of heart rate variability: The patient had a brief 5-minute continuous tracing of lead II electrocardiography (ECG) at a speed of 30 mm/sec using POLY-RITE-D. Low-frequency (LF) measurements were collected in the range of 0.04-0.15 Hz, while high-frequency (HF) measurements were taken in the range of 0.15-0.40 Hz.5

Software version 16.0 of SPSS was used for statistical analysis. This data was presented as the Mean (S.D.). Time domain HRV measurements included things like mean heart rate, standard deviation of normal-to-normal intervals (SDNN), and root mean square of successive differences (RMSSD).

Results

The research comprised a total of 100 participants, 50 of whom did not smoke (controls) and 50 of whom smoked (cases). In terms of age and basal mass index (BMI), the non-smokers had a mean of 30.23 (6.59) and the smokers of 30.9 (8.40), while the former had a value of 24.04 (3.20) and the latter of 23.58 (3.36). The values of resting pulse rate and blood pressure were significantly higher in smokers compared to non-smokers ($p<0.05$). Table 1.

Smokers had significantly lower total power (ms2), high frequency power (ms2), and high frequency power in normalised unit values compared to non-smokers ($p<0.05$). Megs of low-frequency power In comparison to non-smokers, smokers had a larger low frequency power in normalised unit, LF/HF ratio, and this difference was statistically significant ($p<0.05$). Table 2

In comparison to non-smokers, smokers had lower SDNN and RMSSD values, and this difference was statistically significant ($p<0.05$). Table 3

Table 1: Age, anthropometric and basal cardiovascular parameters of smokers and non-smokers

Parameters	Control (Non-smokers) (n=50)	Cases (smokers) (n=50)	P Value	Statistical significance
	Mean (S.D.)	Mean (S.D.)		
Age (in years)	30.23(6.59)	30.9(8.40)	0.69	NS
BMI	24.04(3.20)	23.58(3.36)	0.53	NS

(in kg/m2) Resting pulse rate	71.95(1.24)	72.62(1.46)	0.02*	S
(in beats/min) Resting- Systolic blood pressure (in mmHg)	117.4(3.82)	119.05(3.23)	0.04*	S
Resting- Diastolic blood pressure (in mmHg)	79.85(0.70)	80.9(2.51)	0.01*	S

*p value <0.05 is statistically significant

Table 2: Comparison of Frequency domain HRV parameters between smokers and non-smokers

HRV Parameters	Control (Non-smokers)(n=50)	Cases (smokers) (n=50)	P Value	Statistical significance
	Mean (S.D.)	Mean (S.D.)		
TP (ms2)	2947.47(2796.25	1972.83(1003.84)	0.04*	S
LF (ms2)	569.85(1276.12)	1001(185.61)	0.03*	S
LF (n.u.)	78.60(5.39)	102.575(44.55)	0.001**	HS
HF (ms2)	122.95(92.09)	84.92(9.95)	0.01*	S
HF (n.u.)	21.45(5.38)	17.4(5.43)	0.001**	HS
LF:HF	3.65(3.10)	5.45(1.51)	0.001**	HS

*p value <0.05 is statistically significant. **p value<0.001 is statistically highly significant;

Table 3: Comparison of Time domain HRV parameters between smokers and non-smokers

HRV Parameters	Control (Non-smokers) (n=50)	Cases (smokers) (n=50)	P Value	Statistical significance
	Mean (S.D.)	Mean (S.D.)		
SDNN (ms)	38.61 (13.96)	30.60 (3.81)	0.0001**	HS
RMSSD (ms)	58.55 (182.68)	18.10 (4.72)	0.0001**	HS

*p value<0.05 is statistically significant **p value<0.001 is statistically highly significant;

Discussion

In our research, we found that healthy male heavy smokers had considerably higher resting pulse rate and baseline blood pressure compared to non-smokers. George et al. also found that smokers' pulse rates were much higher than non-smokers', which might be because of reduced baroreflex function. [6] People who smoke heavily show more sympathetic activity. Total power and SDNN in frequency domain analysis show general autonomic control. Heavy smokers had much lower overall power compared to non-smokers in our research. Another finding by Ferdousi et al. is that overall power is lower in heavy smokers compared to non-smokers. This might be because to a reduction in vagal tone. [7] Less parasympathetic activity is indicated by this. Parasympathetic activity is indicated by a high frequency, measured in ms2 and normalised units. Heavy smokers had a much lower high frequency compared to non-smokers in our research. Lee as well as Chang et al. also found similar results. [8] On the other hand, Karakaya found that, in response to acute smoking, HF was much higher in smokers than in non-smokers. [9] This suggests that heavy smokers have less parasympathetic activity compared to those who don't smoke. Both parasympathetic and sympathetic activity are indicated by low frequency (ms2 and normalised units). In our investigation, we found that heavy smokers had a much higher low frequency than non-smokers. The research conducted by Taralov et al. also found similar results. [10] This result is at odds with that of Lee and Chang et al., who discovered that LF was much lower in smokers than in non-smokers. The results show that heavy smokers' sympathetic activity is higher than that of non-smokers. The ratio of sympathetic to parasympathetic tone, or LF/HF, shows how well the two systems are balanced. Heavy smokers had a much higher LF/HF ratio than non-smokers, according to our research. Both Saini et al. and Doss et al. found something similar. [9,10] This suggests that heavy smokers have more sympathetic activity compared to non-smokers, as shown in references. [11,12] Ferdous et al. likewise found that heavy smokers had elevated LF, reduced HF, and an increased LF/HF ratio, same as the previous study. [13] Parasympathetic dominance is shown by SDNN in time domain analysis. Heavy smokers had a much lower SDNN than non-smokers in our research. In a similar vein, Barutcu et al. found that heavy smokers' vagal regulation of the heart was decreased during a parasympathetic manoeuvre. [14] The current study's SDNN values align with those of Cagirci et al. and Erdem et al.Results

show that heavy smokers have lower parasympathetic activity compared to non-smokers. [15,16] A parasympathetic dominance indicator is RMSSD. When comparing heavy smokers to non-smokers, we found that RMSSD was much lower in the former group. Alyan et al. also found something similar. [17] This suggests that heavy smokers have less parasympathetic activity compared to those who don't smoke. Urooj et al. found the same thing for RMSSD and SDNN. [18] The current research suggests that nicotine and other chemicals in cigarette smoke are responsible for the substantial alterations in autonomic systems seen in heavy smokers. The autonomic nervous system becomes unbalanced in smokers as a result of the effects of nicotine on the adrenal gland and autonomic ganglia, which in turn cause an increase in catecholamine release, stimulation of the muscle sympathetic nerve, and sensitivity of peripheral chemoreceptors. By acting on beta1 adrenergic receptors, an increase in sympathetic activity causes myocardial contraction, which in turn raises blood pressure and the heart rate. Additionally, it acts on alpha2 adrenoceptors, which raise the tone of the coronary blood arteries. Due to direct effects on the baroreflex centres in the brainstem, long-term nicotine users have diminished baroreceptor sensitivity and elevated sympathetic activity. Therefore, autonomic dysfunction in heavy smokers is suggested by the current study's findings.

Conclusion

Compared to non-smokers, those who smoke have less parasympathetic activity and more sympathetic activity. Cigarette smokers are at increased risk for cardiovascular disease due to autonomic dysfunction, namely sympathetic overactivity. So, taking HRV readings might be a way to screen for ANS changes before any symptoms show up in the body.

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