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Influence of cigarette smoking in the rate pressure product among young adults: a case control study from Manipal, India.

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Abstract

Background

Cigarette smoking is an important cause of mortality across the world, resulting in death of nearly six million people. Nearly 40% of the deaths are caused by cardiovascular disease and 20% are due to lung cancer. Cigarette smoking is said to increase the susceptibility towards vascular injuries by impacting phases of atherosclerosis, and altering blood pressure (BP), heart rate (HR) and elevating plasma catecholamine levels. The objective of this study was to assess if cigarette smoking increases the rate pressure product (RPP) at rest and after standing in young adult light smokers compared to non-smokers.

Methods

30 young adult smokers and 30 non-smokers (19 - 24 years) were enrolled in this case control study. The smoking pack-years was calculated. Blood pressure (BP) was measured and RPP was calculated. The mean values between both groups were compared using the univariate analysis of variance adjusted for confounding variables.

Results

The smoking pack-years was 1.6±1.0 (0.2-4.5). RPP in smokers was 88.8±8.0, 96.2±10.2 at rest and after standing respectively. RPP in non-smokers at rest was 81.6±8.1 and 89.5±10.3 after standing. RPP was statistically significant in between both groups at rest.

Conclusion

Smoking increases the RPP significantly in young adult light smokers due to an increased HR when compared to non-smokers, at rest. This study reinforces that young adults who are light smokers have an increased workload on the heart, which affects their cardiac performance.

Key words

Blood pressure, heart rate, oxygen consumption, rate pressure product, smoking.

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Background

Cigarette smoking is an important cause of mortality across the world, resulting in death of nearly six million people, of which, 40% are caused by cardiovascular disease and 20% are due to lung cancer [1]. According to the report of WHO, in the year 2012, around one fifth of the of the global population aged 15 and above smoked tobacco. The numbers of male smokers are five times higher comparing females; with an average rates of 36% and 7% respectively. It was also noticed that, in the WHO Western Pacific Region Smoking among men was highest (48%); and WHO European Region at 19% for females. WHO data also showed that tobacco use amongst boys aged 13-15 in WHO South-East Asia Region and WHO Eastern Mediterranean Region is comparatively higher (20%) than other regions [2]. Researchers found that there were 31.3 million hardcore smokers in the three Asian countries ranges between 3.1% in India to 6% in Thailand and Bangladesh. There are 18.3-29.7% of daily smokers [3].

Smoking is a significant risk factor for heart disease. Tobacco smoke contains chemicals like Nicotine, carbon monoxide, free radicals and cytokine which distributes throughout the body in smokers via blood circulation. They are responsible for the formation of atherosclerotic plaque and its destabilization. This is the most significant risk factor in several pathophysiological conditions like coronary artery disease (CAD), sudden cardiac death, ischemic stroke, aortic aneurysm formation, peripheral vascular disease and Buerger disease [4]. Smoking aggravates certain pathophysiological conditions like unhealthy blood cholesterol levels, high BP, and overweight or obesity; which ultimately increases the risk of heart disease [5]. Cigarette smoking is said to increase the susceptibility towards vascular injuries by impacting phases of atherosclerosis, altering BP, HR and elevates plasma catecholamine levels [6-8].

The RPP is an indirect indicator of myocardial oxygen consumption (MVO_2), which is a useful indicator of the total energy consumption of the heart [9, 10]. RPP is an important marker of the oxygen requirement in the heart and has been recognized as a significant parameter for the evaluation of ventricular function [11]. It reflects the internal myocardial work performed by the beating heart whereas the performance of the external myocardial work is represented by the stages of exercise. Smoking tobacco is also one of the foremost risk factors for chronic obstructive pulmonary diseases (COPD) and lowers the peak expiratory flow rate (PEFR) [12-15].

Studies conducted earlier with a larger sample size have shown that young adult male smokers show variations in their BMI and waist-hip ratio (WHR) as compared to controls [16-18].

Although there are some researches on RPP, mainly focused on exercise tolerance, electrocardiographic changes but not in the postural changes, which is also considered as an important factor for orthostatic hypertension [19, 20].

The purpose of this study was to examine the effects of light smoking on the RPP during postural changes between young adult light smokers and non-smokers.

Material and Methods

Study Period

This case-control study was conducted between March to August 2010, in Melaka Manipal Medical College, Manipal University, Manipal, India.

Study design and participants

Study group consisted of 30 smoking, but otherwise healthy young adult males and an equal number of non-smokers of Malaysian origin, aged between 19 - 24 years.

Response Rate

At the beginning of the experiment, questionnaire was administered to 150 students. Among them 122, were returned the same giving an overall response rate of 81%. Incompletely filled questionnaire were excluded.

Questionnaire design

Questionnaire was especially designed, which included the basic sociodemographic details like age, ethnicity *etc*, health status related questions, smoking and drinking habits. Frequency of alcohol intake per day, number of cigarettes smoked, total number of years of smoking was included in the questionnaire to obtain the number of pack-years.

Data collection

At the beginning of the study, students were initially screened by a personal interview, which was followed by administration of the questionnaire to identify their habits. Based on the self-reported smoking status, those answering affirmative were considered for further proceedings.

Smoking habit (smoking pack-years)

The smoking pack-years was calculated by the formula: number of cigarettes smoked / day x number of years of smoking / 20 [21, 22].

The smoking pack-years between 0.1 to 20, 20.1 to 40 and greater than 40 were considered as light, moderate and heavy smokers respectively [22, 23].

Habit of alcohol intake

Alcohol intake was calculated by the frequency of consumed alcohol. Alcohol (in grams) / day was calculated based on the

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amount (in milliliters) and volume percent concentration (% v/v) of the type of alcohol consumed [24] Subjects consuming alcohol greater than 1-2 times a week and/or ≥20 gms alcohol/day were excluded, since it would affect the results of the main parameters of this study.

Measurement of blood pressure

BP was measured by using a mercury sphygmomanometer. The subjects were given ≥ 5 minutes of rest, following which systolic BP (SBP), diastolic BP (DBP) and heart rate were measured in the supine position and one minute after standing upright. Mean blood pressure (MBP) was calculated by the formula, DBP + ⅓rd pulse pressure. The pulse pressure was calculated by the difference between SBP and DBP. Heart rate was measured by assessing the radial pulse rate for one minute. RPP was calculated in supine position and standing by multiplying heart rate and systolic BP divided by 100 [25].

Measurement of peak expiratory flow rate (PEFR)

Wright's peak flow meter was used to measure the peak expiratory flow (PEFR) (Datospir Peak-10, Sibelmed, Spain). Subjects were allowed to take rest and then instructed to exhale rapidly into the mouthpiece after a maximum inspiration. The procedure was repeated for three times with a gap of ≥ 2 minutes rest. The highest value among the three readings was considered for analysis. PEFR was measured to assess the degree of obstruction of airways in the subjects. A PEFR value between 400 to 600 L/min was considered as normal [26, 27].

Body mass index (BMI)

The height (in meters), weight (in kilogram), waist and hip circumference (centimeters) were measured. The BMI was calculated by the formula: weight in kilograms divided by height in square meters (m^2) and categorized as underweight <18.5 kg/m^2, normal BMI 18.5 to 24.9 kg/m^2 and overweight 25.0 to 29.9 kg/m^2 [28, 29]. Upper body obesity was measured by assessing the WHR. WHR was calculated by dividing the waist circumference by the hip circumference. The WHR values between 0.83 and 0.93 were considered as the normal range [29].

Inclusion criteria

All students in the age range 19 - 24 years, who fulfilled the criteria for this experiment, were included.

Exclusion criteria

Students who had not given consent, unwilling to disclose smoking or drinking habit, past history of acute or chronic cardiac and/or respiratory disease which could be an influential factor for BP and PEFR, were excluded to avoid biasness. Females were also excluded from this study.

Sample size calculation

RPP during test used for calculation. Standard deviation in group I = 8.0; Standard deviation in group II = 8.1; Mean difference = 7.2; Effect size = 0.894409937888199; Alpha Error (%) = 5; Power (%) = 80; sided = 2; required sample size per group = 20.

Ethical committee approval

This study was conducted in accordance with the Helsinki Declaration of 1975 (revised in 1983). Written informed consent was obtained from all subjects who were enrolled. The anonymity was maintained by unique study identification number because the sensitive nature and confidentiality of this study.

Outcome variable

Blood pressure, heart rate, RPP were considered as outcome variable

Explanatory variables

The demographic and cause of choice factors were defined at individual level. Factors at individual level were age, gender, nationality.

Data management and statistical analysis

Data was analyzed using the statistical package for social sciences (SPSS) version 15.0 (SPSS South Asia, Bangalore). The data were expressed as mean±SD. The mean SBP, DBP, HR, RPP at rest and after standing were compared between cigarette smokers and non-smokers. The mean PEFR, BMI and WHR were also compared between both groups. The difference between the two means (smokers and non-smokers) in this case-control study was compared using the Independent t-test. Univariate analysis of variance was used to adjust for possible confounding variables (i.e. BMI). A value of $p<0.05$ was considered as statistically significant.

Results

The mean±SD (range) pack-years of smoking were 1.6±1.0 (0.2-4.5) and were considered as light smokers (i.e., <20 pack-years). The time period of smoking was 2.5±0.8 years. The self-reported alcohol intake calculated showed that 07 (23.3%) subjects consumed alcohol occasionally but not greater than two times a week in the smoking group. 03 (10.0%) consumed alcohol in the control group. Comparison of smokers and nonsmokers showed a significant change in HR at rest and after standing. The RPP was higher at rest and after standing in smokers compared to non-smokers. The DBP at rest showed no statistically significant difference between these groups.

The WHR was 0.86±0.04 and 0.87±0.03 in the non-smoking and smoking group, respectively. The smokers had a

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significantly lower BMI (21.9±3.5 kg/m^2) as compared to the non-smokers (24.1±3.9 kg/m^2), since, differences in SBP, MBP, HR, and RPP during rest and after standing were adjusted for BMI (23.0 kg/m^2).

Table – 1 Comparison between young adult cigarette smokers and non-smokers for SBP, MBP, HR, RPP during rest and after standing using the Univariate analysis of variance adjusted for BMI.

Variable	Group	mean±SD	p value
SBP (mm Hg) during rest	Non-smokers	124.7 ± 7.0	0.61[x]
	Smokers	125.9 ± 6.9	
SBP (mm Hg) after standing	Non-smokers	119.2 ± 6.7	0.29[x]
	Smokers	117.0 ± 6.6	
MBP (mm Hg) during rest	Non-smokers	90.3 ± 6.1	0.55[x]
	Smokers	91.9 ± 6.0	
MBP (mm Hg) after standing	Non-smokers	93.0 ± 5.6	0.51[x]
	Smokers	92.0 ± 5.6	
HR (beats/minute) during rest	Non-smokers	65.3 ± 6.4	0.01[†]
	Smokers	70.4 ± 6.3	
HR (beats/minute) after standing	Non-smokers	74.2 ± 7.9	0.005[†]
	Smokers	82.8 ± 7.8	
RPP during rest	Non-smokers	81.6 ± 8.1	0.02[†]
	Smokers	88.8 ± 8. 0	
RPP after standing	Non-smokers	89.5 ± 10.3	0.18[x]
	Smokers	96.2 ± 10.2	

[x]P>0.05 statistically not significant
[†]P<0.05 statistically significant

Table 1, shows the comparison between young adult cigarette smokers and non-smokers for SBP, MBP, HR, RPP during rest and after standing using the Univariate analysis of variance adjusted for BMI. The SBP and MBP showed no significant change during rest or after standing. A statistically significant difference was observed in HR at rest (p = 0.01) and after standing (p = 0.005) in both groups. The RPP was significantly higher at rest (p = 0.02) in smokers compared to non-smokers. However, no statistical significance was observed when both groups were compared for RPP after standing, p = 0.18. The PEFR values were 441.7 ± 63.2 L/min in non-smokers and 419.8 ± 31.9 L/min in smokers, and showed no statistical significance (p = 0.10).

Discussion

Effect of smoking on RPP

Our results clearly show that RPP was especially higher in the smoking group compared to the non-smoking group, especially due to a significant difference in HR. Smoking results in increasing the resting HR of individuals [30]. Nicotine decreases cardiac vagal activity, resulting in increased HR [31]. Exposure to nicotine in cigarettes probably stimulates the sympathetic fibers to the heart, resulting in an increased HR at rest in smokers compared to non-smokers [32]. The results were comparable to the study by Papathanasiou *et al*, which showed that smoking >20 cigarettes/day for at least 3 years, resulted in higher RPP at rest and during submaximal exercise. An increase in RPP shows that the work load on the heart increases with myocardial oxygen consumption, affecting the cardiac economy [19]. A recent study by Ferdousi *et al*, showed that the heart rate variability, blood pressure and RPP were altered due to smoking compared to normal, with increasing sympathetic activity progressing from light smokers to heavy smokers [22]. Our study showed no statistically significant difference with regard to resting BP, which was comparable to other studies [19, 33].

Postural change and effect on RPP

In this study, though upright standing showed a slight increase in RPP, but not change was observed in SBP or MBP values, no statistical significance was observed for RPP and mean BP values in both non-smokers and smokers. This was probably due to the decreased pack – years of smoking, which showed no significant influence on the stroke volume and/or peripheral resistance in these young adults.

This young adult smokers group showed no evidence of airway obstruction or obstructive pulmonary disease. Our study consisted of healthy young adult smokers, who were within the range of 0.2 to 4.5 smoking pack-years, with a mean (SD) period of 2.5 (0.8) years of smoking. Since the smoking group included all light smokers, the PEFR was within the normal range and were comparable to the non-smokers.

Effect of Smoking on BMI and WHR

Previous studies have shown that the BMI increases after cessation of smoking, whereas current smokers have a lower BMI compared to non-smokers [16, 18, 34, 35]. Our study showed significantly lower BMI in the current smoking group compared to non-smokers, which is consistent with the findings from other studies [16, 17, 35, 36]. The WHR being a measure of central adiposity was within the normal range, and showed no significance between both groups. Our WHR results are in concordance to the study by Caks T *et al*, where male smokers showed no variation in WHR for younger age groups [18].

Conclusion

The results of this study support our hypothesis that, smoking increases the RPP in young adult light smokers due to an increased HR at rest when compared to non-smokers. Though this study is not new, our study result reinforces that young adults who are light smokers have increased workload on the heart. Cessation of smoking is important for young adults, to enhance cardiac economy and decrease the risk of cardiovascular disease.

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Limitations & future scope of the study

The sample size was small in this case-control study. The amount of alcohol in grams/day was very low to have influenced the heart rate or other parameters. However, the exact number of cigarettes smoked, alcohol frequency and amount consumed were based on self-report. The consumption of caffeinated drinks and the exact level of physical activity were not assessed, which may have influenced the results. Use of spirometer is strongly recommended to assess the lung function for more specific lung volume and capacities. ECG recording would have brought more objectivity and reliability to this study, which was not performed. An overnight cessation of smoking influences the autonomic nervous system and increase activity to the vascular system. This effect is reduced in the afternoon after a continuous nicotinic impregnation. So these diurnal variations should be considered for future studies.

Abbreviations

Blood pressure (BP), body mass index (BMI), chronic obstructive pulmonary diseases (COPD), coronary artery disease (CAD), diastolic blood pressure (DBP), heart rate (HR) interquartile range (IQR), mean blood pressure (MBP), myocardial oxygen consumption (MVO_2), peak expiratory flow rate (PEFR), rate pressure product (RPP), systolic blood pressure (SBP), waist-hip ratio (WHR).

Competing interests

The authors of this study declare no competing interests.

Authors' contribution

Mulki Ganesh Kamath designed this study along with Tharani Naidu, Arun Muruga Rao, Amierul Izwan and Che Wan Norsyikin. The study was conducted by Tharani Naidu, Arun Muruga Rao, Amierul Izwan and Che Wan Norsyikin. Asha Kamath helped with the statistical methods. All authors contributed to the analysis and interpretation of data, drafting and/or revising the article and approved the final version of this manuscript.

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