Effect of Smoking on Sustained Handgrip Test of Young Healthy Male Tobacco Smokers

*Tasnim R,[1] Alam KI,[2] Akter S[3]

Abstract

Introduction: The tobacco epidemic is one of the biggest public health threats the world has ever faced. Cigarette smoking is major risk factor for the development of cardiovascular disease. Cessation of smoking is associated with reduced cardiovascular mortality and morbidity. Cigarette smoking alters baseline sympathetic and vagal modulation. The degree of risk of developing coronary heart disease is directly related to the number of cigarettes smoked per day.

Objectives: To assess the effects of smoking on sustained handgrip test.

Methods: This cross sectional analytical study were conducted in Department of Physiology, Rangpur Medical College, Rangpur. After obtaining permission a total 100 subjects who fulfilled the inclusion and exclusion criteria were enrolled in the study after briefing them objectives of the study. A total number of 100 subjects were selected, among them 50 were apparently healthy non-smoker subjects (group A) - control, 50 were apparently healthy tobacco smoker (group B) – experimental. For statistical analysis one way ANOVA (post-hoc) test were performed by computer based software SPSS- 23.0 version for windows. Significance for the statistical test would be predetermined at a probability value of less than 0.05 ($p<0.05$). Ethical consideration was achieving an informed consent after briefing objectives. Quality was assured through avoidance of missed data, filling of code, regular entry of data and careful data analysis.

Result: In this study smokers revealed significant rise in blood pressure as compared to nonsmokers during hand grip test.

Conclusion: Cigarette smoking affects cardiovascular functions as indicated by rise in blood pressure in smokers than nonsmokers reflect the sensitivity of sympathetic activity to nicotine due to smoking.

[M Abdur Rahim Medical College Journal, 2023 Jul; 16 (2):200-207]
[Former Dinajpur Medical College Journal]

Keywords: Sustained handgrip test, sympathetic nerve function test

Introduction

Now a days tobacco use is a well documented threat to global health. It kills more than 7 million people a year and is currently the world's single biggest cause of preventable death.[1] Smoking is one of the leading causes of premature death, disease, and disability in the world. Over 57,000 people die in Bangladesh each year from tobacco related diseases. According to Centre for Disease Control (CDC) about 3,900 teens under 18 years start smoking each day and around 1500 of these teens will become regular smokers. According to the European Society of Cardiology (ESC) - smoking increases the risk of heart disease and stroke by five-fold in people under the age of 50 and doubles risk in the over 60s. The protection of children and adolescents from taking up smoking is essential for the future health of the Europeans and stronger measures are needed.[2] Tobacco contains nicotine, an ingredient that can lead to addiction. For this reason, it is difficult to quit for so many people who use tobacco. There are also many other potentially harmful chemicals found in tobacco or created by burning it.[3]

1. **Dr. Rukhsana Tasnim, Assistant Professor, Department of Physiology, Army Medical College, Rangpur, Bangladesh. tasnimrukhsana@gmail.com*
2. *Dr. KH Imranul Alam, Medical officer, Mediscan digital Clinic, Dinajpur, Bangladesh.*
3. *Dr. Sharmin Akter, Assistant Professor, Department of Anatomy, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh.*

**For correspondence*

Over nine in 10 smokers purchased cigarettes and bidis from stores. The average cigarette expenditure per month among cigarette smokers was 378 taka/month and among bidi smokers it was 131 taka. It is estimated that in Bangladesh total expenditure on cigarettes is 1.0% of gross domestic product (GDP) and on bidis is 0.4% of GDP. The price of 100 packs of manufactured cigarettes as a percentage of per capita GDP is 5% and the price of 100 packs of bidis as a percentage of per capita GDP is 1% [3]. Cigarette smoke might favour the development of atherosclerosis by damaging the vascular endothelium and producing an unfavourable lipid profile. The adverse effects of smoking might depend, as well, on an increase in platelet aggregation and vasomotor reactivity, leading to a pro thrombotic state, favoring coronary spasm, particularly in women, and occlusion. Acute events, such as ventricular fibrillation and sudden death, are increased by smoking, particularly in the presence of pre-existing coronary artery disease. Cigarette smoking acutely increases plasma catecholamines and cardiac norepinephrine spill-over would suggest sympathetic overactivity.[4] Cigarette smoking, coupled with the recent available inhaled substances and delivery systems, including marijuana, electronic cigarettes (e-cigarettes), and waterpipes (hookahs), and the recent recognition that exposure to air pollution also increases cardiac risk. Nicotine, with a contribution from fine particulate matter (PM2.5; defined as <2.5 mm in hydrodynamic diameter), underlies the acute sympathetic excitatory effects of tobacco smoke, which are opposed by intact arterial baroreflexes. PM2.5 (fine particulate matter defined as <2.5 mm in hydrodynamic diameter) in tobacco smoke generates reactive oxygen species and inflammation, which play a crucial role in sustained sympathetic activation[5].

Materials

This cross sectional analytical study were conducted in Department of Physiology, Rangpur Medical College, Rangpur. After obtaining permission a total 100 male subjects who fulfilled the inclusion and exclusion criteria were enrolled in the study after briefing them objectives of the study. Subjects with established obstructive coronary artery disease, unstable coronary syndromes, currently alcoholics or on athletic training were excluded. The subjects were apparently healthy young adults aged 25–40 years with habit of smoking tobacco for >3 years. A thorough history regarding regular athletic training exercises, medical conditions, and the medications taken in the past 6 months was obtained followed by clinical and systemic examinations. Among them 50 were apparently healthy non-smoker subjects (group A) - control, 50 were apparently healthy tobacco smoker (group B) – experimental. For statistical analysis one way ANOVA (post-hoc) test were performed by computer based software SPSS-23.0 version for windows. Significance for the statistical test would be predetermined at a probability value of less than 0.05 ($p<0.05$). Ethical consideration was achieving an informed consent after briefing objectives. Quality was assured through avoidance of missed data, filling of code, regular entry of data and careful data analysis.

Test procedure: A rise in diastolic blood pressure is determined during isometric pressing of a handgrip dynamometer at approximately one third of the maximum contraction strength for 3–5 min. Blood pressure measurements are taken at the other arm at 1 min interval. An increase in diastolic blood pressure is a result of heart rate acceleration without an increase of peripheral vascular resistance. The test result is presented as the difference between the highest diastolic pressure during the examination and the average diastolic pressure at rest. It should normally be higher than 15 mmHg.[7]

Result

The mean ± SD of age were 32.74 ± 4.44 years in group A, 31.87 ± 6.39 years in group B (Table I & Figure I). Group A (Control) – 50 apparently healthy subjects of non tobacco chewers non- smokers. Group B (Experimental) – 50 apparently healthy subjects of tobacco smoker non- chewers. The mean ± SD of height was 1.53 ± 0.094 m in group A, 1.54 ± 0.095 m in group B (table I & figure II). The mean ± SD of weight were 67.1 ± 6.9 kg in group A, 66.066 ± 7.921 kg in group B (table I & figure III).

Table I: Showing mean ± SD of age, height and weight of the study subjects of different groups

Group	Mean ± SD Age- Year	Mean ± SD Height (m)	Mean ± SDWeight (kg)
A N=50	32.74 ± 4.45	1.53 ± 0.094	67.1 ± 6.901
B N=50	31.87 ± 6.39	1.54 ± 0.095	66.06 ± 7.921

A = Apparently healthy subjects of non-tobacco chewer non-smoker (Control).
B = Apparently healthy subjects of tobacco smoker (Experimental).
SD = Standard deviation.

Table II: Showing statistical analysis of mean ± SD of age, height and weight in different groups Analysis between the groups done by One – way ANOVA (Post Hoc Test):

Parameter (Group A/ Group B)	Mean ± SD Range (L- H)	'p'value
Age	32.74 ± 4.45/ 31.87 ± 6.39	$.924^{NS}$
Height	1.53 ± 0.094/ 1.54 ± 0.095	1.000^{NS}
Weight	67.1 ± 6.901/ 66.06 ± 7.921	$.109^{NS}$

A = Apparently healthy subjects of non-tobacco chewer non-smoker (Control).
B = Apparently healthy subjects of tobacco smoker (Experimental).
SD = Standard deviation.
NS = $p > 0.05$
** = $p < 0.01$ level.
* = $p < 0.05$ level.

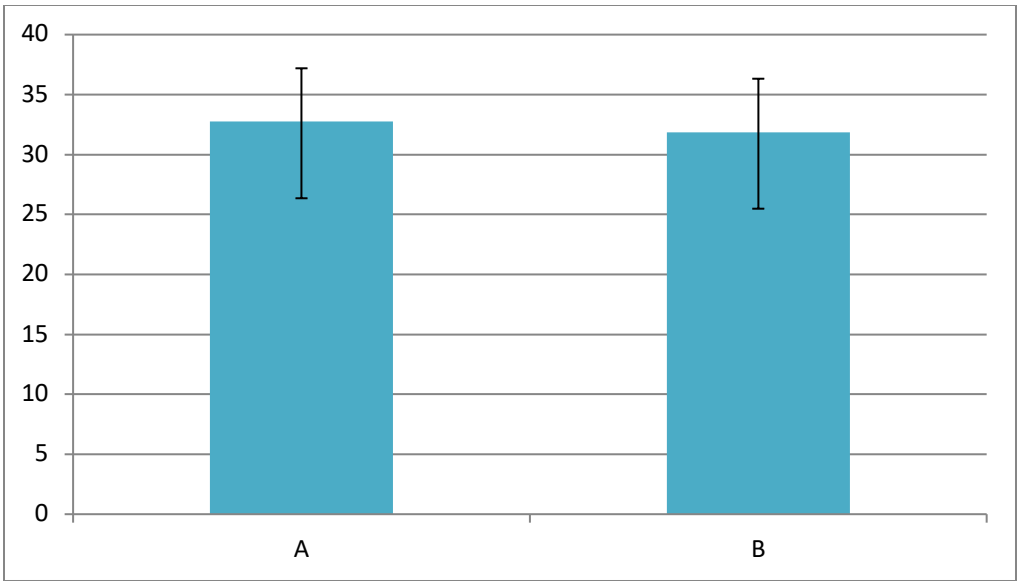

Figure 1. Bar diagram showing mean (± SD) age in group A (Control) and group B (Experimental)

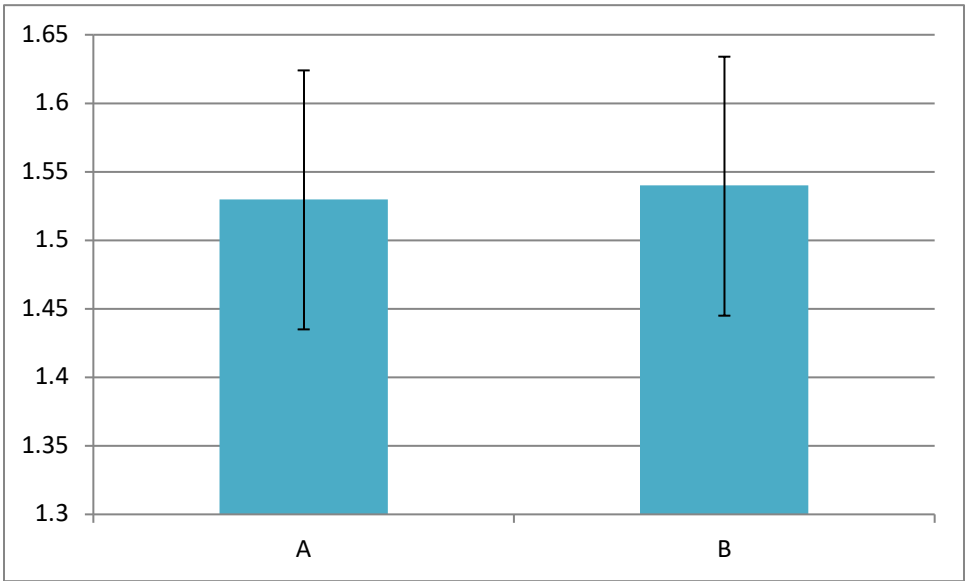

Figure 2. Bar diagram showing mean (± SD) height in group A (Control) and group B (Experimental).

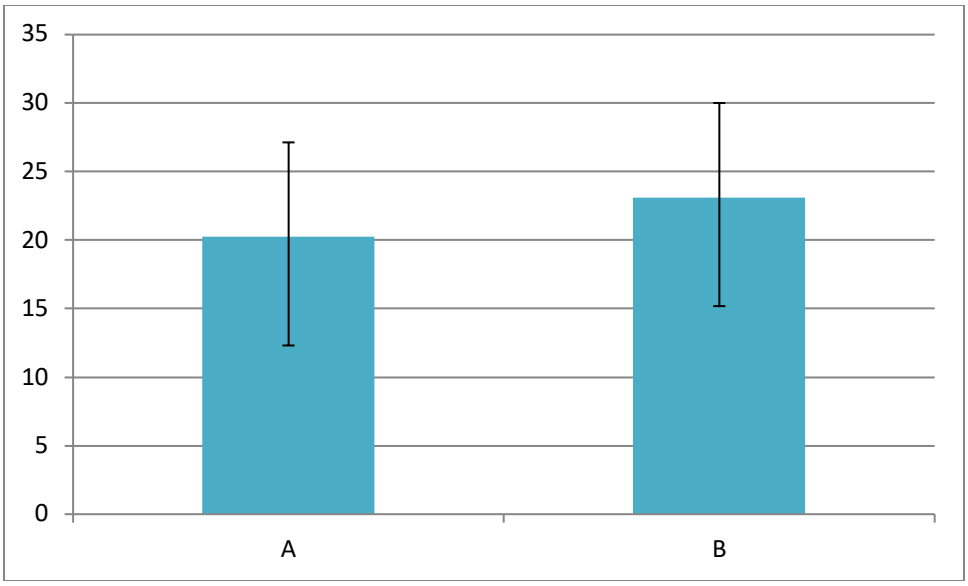

Figure 3. Bar diagram showing mean (± SD) weight in group A (Control) and group B (Experimental)

Sympathetic nerve function parameter

The sympathetic nerve function statuses of all subjects were evaluated by blood pressure response to sustained handgrip test.

Blood pressure response to sustained handgrip test

The mean ± SD of sustained handgrip tests were 20.2333 ± 2.3846 and 23.1063 ± 3.1084 in group A and B. (Table – III and Figure - 4).

In this study mean values of sustained handgrip tests were compared between group A and group B.

The mean values were significantly ($p < 0.001$) lower in group A than group B (Table IV).

Table III: Showing mean ± SD of sympathetic nerve function parameters in study subjects of different groups

Group	Sustained Handgrip test (mm of Hg)
A (n=50)	20.2333 ± 2.3846
B (n=50)	23.1063 ± 3.1084

A = Apparently healthy subjects of non-tobacco chewer non-smoker (Control).
B = Apparently healthy subjects of tobacco smoker (Experimental).
SD = Standard deviation.

Table IV: Showing statistical analysis of mean ± SD of blood pressure response to sustained handgrip test in study subjects of different groups

Groups	Mean ± SDm Range	'p'value
A / B (n=50) / (n=50)	20.2333 ± 2.3846/ 23.1063 ± 3.1084	.003**

Analysis between the groups done by One – way ANOVA (Post Hoc Test)

A = Apparently healthy subjects of non-tobacco chewer non-smoker (Control).
B = Apparently healthy subjects of tobacco smoker (Experimental).
SD = Standard deviation.
NS = $p > 0.05$
** = $p < 0.01$ level.
* = $p < 0.05$ level.

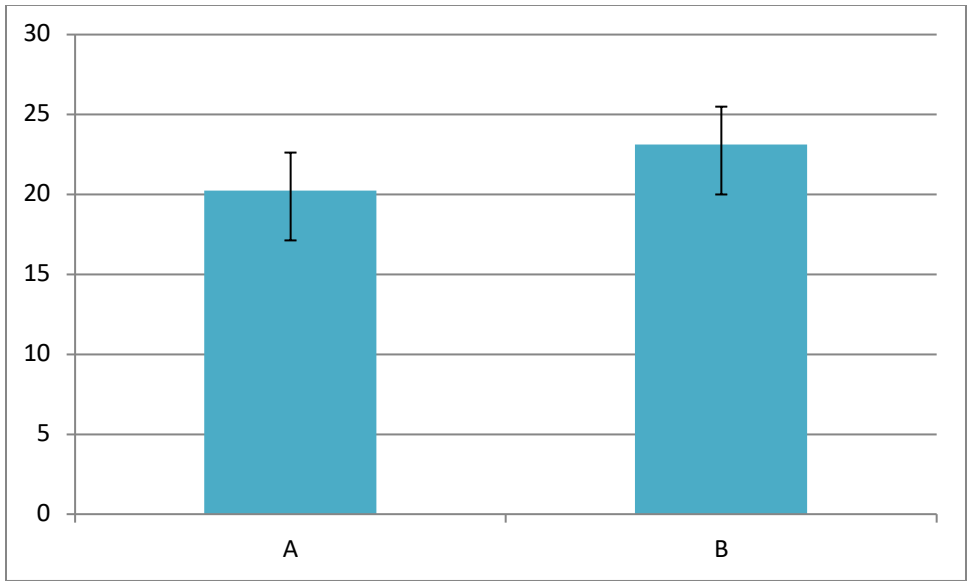

Figure 4. Bar diagram showing mean (± SD) BP response to sustained handgrip in group A (Control) and group B (Experimental)

Discussion

In Bangladesh smoking is a common habit prevalent in both urban and rural area irrespective of the item smoked i.e. cigarettes, bidis, pipes, cigar, hookah etc. About 17% smokers in the world live in India. Smoking has been identified as a major coronary heart disease risk factor with several possible mechanisms like carbon monoxide induced atherogenesis ,nicotine stimulation of adrenergic drive raising both blood pressure and myocardial oxygen demand.[6]

The present study was carried out to assess the blood pressure response to sustained hand grip test in tobacco smoker non-tobacco chewer subjects. The blood pressure response to sustained hand grip test is significantly higher ($p < 0.05$) level in group B than group A.

In the present study, the findings of the parameters in apparently healthy control group were within normal ranges and also similar to those reported by the various investigators from different countries.

In tobacco chewer smoker subjects, the mean blood pressure response to sustained handgrip were significantly higher ($p<0.001$) than those of control subjects. These findings are in agreement with those reported by Gupta BK et al[23] and Srivastava RK, Jha RK and Kumer R.[27]

In tobacco chewers nonsmokers' subjects, the mean blood pressure response to sustained handgrip were significantly higher ($p<0.01$) than those of control subjects. These findings are in agreement with those reported by Begum N et al[13] and Shivaji J, Joshi A and Vasantrao A[28] and Haragopal R, Aruna BMK[35]

In smokers non tobacco chewers subjects, the mean blood pressure response to sustained handgrip were significantly higher ($p<0.001$) than those of control subjects. These findings are in agreement with those reported by Malge M and Ranjith Bk,[8] Motilal C and Modala S, Ahmed QR and Sau SK.[9]

They reported that the pressor and tachycardiac effects of cigarette smoking are associated with plasma catecholamines suggesting an adrenergic stimulation. They also suggested that habitual smoking affects the sympathetic activity earlier than parasympathetic activity. Their observations indicate that cigarette smoking might induce α adrenergic tone with considerable impact on cardiovascular regulations. Further these results implied even if α receptor function unaltered in cigarette smokers their down regulation of β receptor may result in relative increase in α adrenergic tone at any given catecholamine concentration. In their study cold pressor and handgrip test the rise in BP was higher in smokers over nonsmokers could be explained on the basis of increased α adrenergic tone leading to vasoconstrictive tendency of blood vessels, due to increased sensitivity of sympathetic nervous system.

These findings are in agreement with those reported by Gupta BK et al[10] and Srivastava RK, Jha RK and Kumer R.[11]

They reported that tobacco chewing and smoking can result in significantly lethal cardiovascular effects might be due to a larger overall exposure owing to prolonged absorption. Smoking and chewing tobacco is a possible etiological agent for hypertension because it contains three ingredients known to elevate blood pressure i.e. nicotine, sodium, and liquorice.

Acute and long-term exposure to cigarette smoke leads to changes in the balance of autonomic nervous system, resulting in sympathetic predominance. The acute effects of smoking are mainly the action of nicotine that binds to cholinergic receptors present in the autonomic ganglia, neuromuscular junctions and central nervous system, which, when stimulated, increase the release of several neurotransmitters. The nicotine and others substances found in cigarettes also stimulate the release of adrenalin into the sympathetic nervous system. Additionally, the stimulation of the nicotinic receptors in the autonomic nervous system has been associated with the sympathetic excitatory effects of smoking. There are three possible mechanisms to explain this sympathetic activation. The first is a direct effect on the central nervous system; and the second is a stimulatory effect on the ganglionic sympathetic transmission that leads to a subsequent increase in the postganglionic efferent sympathetic activity; and the third, is an effect on the sympathetic peripheral nervous terminations.

Conclusion

Cardiovascular autonomic function tests are reliable, noninvasive and easy to carry out. By using these simple tests, we can detect early involvement of autonomic nervous system before clinically related symptoms appear and thus are useful in taking steps to prevent further progress of the disease. The present study has been carried out in 50 smokers and 50 nonsmokers (control) to study the effect of habitual smoking on autonomic function, which include hand grip test for checking sympathetic activity. The haemodynamic effect of tobacco appears to be mediated by nicotine. Nicotine

increases the sympathetic activity by increasing both heart rate and blood pressure.

Acknowledgement
Authors of this study are thankful to the Physiology Department of Rangpur Medical College and the students whose initiative, active support and guidance makes this study successful.

References

1. World Health Organization. Tobacco and its environmental impact: an overview. [Internet]. [cited 2017]. Available from http://apps.who.int/iris/bitstream/handle/10665/255574/9789241512497-eng.pdf.
2. Khan FI, Afrin S, Huq ME, Zaman UKS, Rahman MR. Socio demographic factors related to smoking among rural adolescent. Delta medical college journal, 2014; 2(2): 58-63.
3. National Institute on Drug Abuse. Drug Facts: Cigarettes and other tobacco products. [Internet]. [cited 2017]. Available from http://www. drugabuse. gov/publications/drugfacts/cigarettes-other-tobacco-products.
4. Lucini D, Bertocchi F, Malliani A, Pagani M. A controlled study of the autonomic changes produced by habitual cigarette smoking in healthy subjects. Cardiovuscular Research, 1996; 31: 633-639.
5. Holly R, Middlekauff, Park J, Roya S, Moheimani BS. Adverse effects of cigarette and noncigarette smoke exposure on the autonomic nervous system. Journal of the American college of cardiology, 2014; 64(16): 1740-1750.
6. Motilal C, Tayadeand Nandkumar BK. Effect of smoking on sustained handgrip test and valsalva ratio among smokers: A cross sectional study. International Journal of Healthcare and Biomedical Research. 2015; 3(4): 84-89.
7. Zygmun A and Stanczyk J. Methods of evaluation of autonomic nervous system function. Archieves of Medical Science, 2010; 6(1): 11-18.
8. Magle M and Babu KR. Study of autonomic functions in cigarette smokers. International Journal of Advances in Scientific Research, 2015; 1(7): 293-299.
9. Modala S, Rais QA and Sau KS. Effect on Autonomic Nervous System in Smokers and Non Smokers- A Comparison Study. National Journal of Medical and Allied Sciences. 2012; 1(1): 25- 28.
10. Gupta BK, Kaushik A, Panwar RB, Chaddha VS, Nayak KC, Singh VB et al. Cardiovascular Risk Factors in Tobacco-chewers: A Controlled Study. Journal of the Association of Physicians of India. 2014; 55: 27- 31.
11. Shrivastava KR, Kumar RJ and Kumar R. Comparative Study of Tobacco Chewer & Smokers as a Risk Factor For Cardiovascular Disease. Indian Journal of Applied Research. 2014; 4(1): 26-27.