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A Comparative Study of the Acute Effect of Tobacco Smoking on Cardiovascular System in Smokers to the Non-Smoking Individuals

Mohd. Rasheeduddin Imran[1*], P. Sambasiva Rao[2]

[1,2]Assistant Professor, Department of Physiology, Alluri Sitarama Raju Academy of Medical Sciences, Eluru-534004, West Godavari Dist, Andhra Pradesh, INDIA.

[*]Corresponding Address:

dr.imran1980@gmail.com

Research Article

Abstract: **Background:** Smoking is a serious global public health hazard and has already emerged as a major cause of death due to heart diseases. It is not recognized because of inadequate programmes to deal with it. **Aims:** The study has been conducted to evaluate and explain the acute cardiovascular effects of tobacco smoking in the habitual smokers and the benefits of quitting the habit of smoking to avoid the complications. **Settings and Design:** This study was conducted in human lab of physiology department on 100 healthy male smokers and 50 healthy male non-smoker subjects in the age range of 21 to 40 years. Based on duration of exposure to cigarette smoking the smokers were again classified as short term smokers and long term smokers. The smokers were compared with non-smokers. **Methods and Material:** The smokers were given standard cigarette to smoke while the non-smokers did sham smoking using a cool drink straw. The variables were recorded at rest and immediately after smoking. Blood Pressure was measured using a mercury sphygmomanometer, heart rate was measured using ECG and Stroke volume and Cardiac Output were derived using Echocardiography. All the values were recorded and comparison tables were derived after statistical analysis using SPSS statistical software and the results were analyzed. **Results and Conclusions:** The results indicate that there is a significant hemodynamic change including an increase in heart rate, blood pressure, stroke volume and cardiac output in both long term and short term smokers immediately after cigarette smoking compared to the non-smoking group after sham smoking. The study concludes that the acute effects can lead to acute cardiovascular events which are more important risk than long-term effects of tobacco smoking and especially in heavy smokers who are always in a state of constant abnormal hemodynamic state.

Key words: Cigarette smoking, Heart rate, Blood pressure, Stroke volume, Cardiac output, Acute Cardiovascular events.

Introduction

Heart disease is not an emerging problem but has already emerged as a major cause of death in rural areas. We haven't recognized this and don't have adequate programs to deal with it. Irrespective of vast study progress in the identification of etiology and treatment of cardiovascular diseases people are still becoming prey to these diseases in the developing countries like India. Consumption of tobacco has changed from chewing tobacco and pipe smoking to smoking cigarettes. The use of tobacco was introduced to Europe more than 500 years ago by Columbus's sailors. Native Americans called the reed tabacum, hence the name tobacco [(11)]. The Egyptians are reported to have started rolling tobacco in paper around 1832. By the early 19th century tobacco was rolled in paper, smoked as cigarettes and rapidly marketed all over the world[(2)]. Cigarette smoke that is drawn through the tobacco into an active smoker's mouth is known as mainstream smoke. Mainstream cigarette smoke comprises 8% of tar and 92% of gaseous components [(20)]. Sidestream cigarette smoke is the smoke emitted from the burning ends of a cigarette. Sidestream cigarette smoke contains a relatively higher concentration of the toxic gaseous component than mainstream cigarette smoke [(13)]. Of all the known constituents, nicotine, a component of the tar phase, is the addictive substance of cigarette smoke [(19)]. The acidic pH of smoke generated by cigarettes deeply reduces adsorption of nicotine in the mouth, thus needing inhalation of the smoke into the larger surface of the lungs in order to absorb enough nicotine amounts to satisfy the smoker [(21)]. The amount of nicotine absorbed is dependent on how long the smoke remains in the mouth, whether the smoke is inhaled or not, and the frequency and depth of inhalation [(43)]. Cigarette smoking increases the risk of plaque rupture and acute thrombosis in men, while plaque erosion and consequent thrombosis is the main mechanism in female smokers [(3)] [(5)]. The thrombogenicity of atherosclerotic lesions is determined by plaque component exposure to flowing blood together with local rheological and systemic blood factors [(23)].Nicotine itself results in attenuation of platelet activation [(22)]. Current smokers have elevated circulating levels of fibrinogen which are restored after smoking cessation [(6)]. In habitual smokers, smoking one cigarette causes short term increase in arterial wall stiffness that might be harmful to the artery

and increase the risk for plaque rupture with accompanying raised blood pressure and heart rate. Because acute vascular events are mainly due to plaque rupture, the short-term effects of smoking might be a more important risk than long-term effects for acute ischemic events [17]. Smoking is the leading cause of preventable morbidity and mortality in the World [25].Tobacco use claims about world-wide 5.4 million lives each year [26]. Cigarette smoking is sometimes called an independent risk factor for CHD and sometimes called a modifiable risk factor because one can reduce or stop smoking[55].Although overall cigarette consumption has declined for decades in high-income countries, smoking rates are on the rise in low-and middle-income countries[27]. Drastic changes in smoking behavior were summarized by Alfred Dunhill, a leading tobacconist from Britain. "Today the ubiquitous cigarette has robbed most of us of these former glories and gripped us by the throat. Smoking has become habit, and habit, proverbially, blunts the edge of pleasure."[11] Jean Nicot[24]extolled the medicinal values of tobacco which was supposed to cure various diseases including gout! Thus tobacco became Nicotiana in honor of Nicot. The relation between smoking and atherosclerosis was observed as early as 1908 by Burger[4], who noted severe distal ischemia among young male addicted smokers. It is said that the internist's mantra for cardiovascular health is, "Stop smoking, lose weight, and get more exercise [12]." The benefits of smoking cessation have been well demonstrated. Smoking cessation reduces health risks and improves quality of life. The cumulative risk of dying of cardiovascular and lung diseases can be drastically reduced (up to 90%) if smokers quit smoking, even late in life[1][18]. In the 20 year follow-up of the British Doctors Study, the excess cardiovascular risk was halved within 2 or 3 years of stopping smoking and by 10 years had returned to that of a never-smoker [10]. Nicotine replacement therapy — chewing gum, transdermal patches, nasal spray, oral inhalator, oral tablets — aids smoking cessation by providing a temporary source of nicotine. Therefore, this study was conducted to evaluate the acute cardiovascular hemodynamic changes in smokers and the risk of development of cardiovascular diseases, to explain the benefits of quitting the habit of smoking and to recommend regular examination and counseling to restrain from smoking.

Materials and methods

This study was conducted on 100 healthy male smokers and 50 healthy male non-smoker subjects in the age range of 21 to 40 years of age. They were all free of any systemic diseases. They were all non-alcoholics.

MATERIALS:

After obtaining informed written consent, detail history and physical examination was done in all subjects. The male subjects were chosen in age groups of 21 – 40 years of age.

The study subjects were divided into mainly two groups:

A) Non-smokers and B) Smokers

A. Non-smokers:

Subjects who had never smoked are included in the present study as control group.

Group 1: 50 controls in range of 21 – 40 years age.

B. smokers:

In the present study, Habitual smokers (nicotine dependent, daily cigarette smokers) were defined as those who reported smoking 15 – 20 cigarettes per day from the last 6months. The smokers were again classified further into two groups based on duration of exposure to cigarette smoking as:

1. Short term smokers:**Group 2**: 50 subjects in range of 21 – 40 years age, smoking fifteen to twenty cigarettes per day for past 6 months to 3 years.
2. Long term smokers:**Group 3:** 50 subjects in range of 21 – 40 years age, smoking fifteen to twenty cigarettes per day for past 10 years.

Subjects having systemic diseases, on medication, on diet restriction, alcoholics and tobacco chewers were excluded.

The subjects are instructed to stay fasting for 6 hours and refrain from smoking for 6 hours and then their Height, Weight, Heart Rate, Blood pressure, stroke volume and cardiac output were recorded.

Height and Weight: Height and weight were measured using a Detecto medical scale and weighing machine.

Blood Pressure: Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured using a mercury sphygmomanometer.

Heart Rate: Electrocardiograms (ECG) using 3 bipolar silver-silver electrodes was utilized to measure heart rate (HR).

Stroke volume and Cardiac Output: Were recorded using Echocardiography.

Methods:

The Group-A, who are non-smokers are taken as control group and Group-B who are habitual Smokers are taken as cases and comparison is done between the two groups. All the variables were first recorded in both the non-smoking group (Group 1) and the smoking groups (Groups 2 and 3) at rest. The non-smoking group were given cool-drink straw and were asked to imitate smoking for a period of 5minutes. This is known as "sham smoking". At the end of 5 minutes, all the variables were recorded again. The smoking groups were given a cigarette of standard brand most commonly smoked, Gold

Flake Kings of 84mm length, each with tar content of 15 mg and Nicotine levels of about 1.4mg. They were asked to inhale the smoke of each puff and complete the smoking of one cigarette in 5 minutes. At the end of 5 minutes, all the variables were recorded again. A p value of <0.05 was considered statistically significant and p < 0.001 is statistically highly significant.

Results

All the hemodynamic parameters included in the study was significantly higher in both smoking groups (group2&3) after cigarette smoking than at rest [Table-2 & 3] and also when compared with the non-smoking group (group1) after sham smoking [Table-4 & 5, Graphs-1, 2, 3, 4, 5 & 6]. There was no significant difference in hemodynamic parameters in non-smokers after sham smoking compared to the values at rest [Table-1].

Table 1: Comparison of Mean Hemodynamic Values in Non-Smokers after sham smoking and at rest

			P value
Variable	Mean	Mean	
Heart Rate	76.5	76.5	> 0.05
Systolic Blood Pressure	121.3	121.3	> 0.05
Diastolic Blood Pressure	73.4	73.4	> 0.05
Stroke Volume	58.59	58.55	> 0.05
Cardiac Output	4473.3	4480.26	> 0.05

Table 2: Comparison of Mean Hemodynamic Values in short term Smokers after cigarette smoking and at rest

			P value
Variable	Mean	Mean	
Heart Rate	91.8	71.7	<0.001
Systolic Blood Pressure	139.6	123.5	<0.001
Diastolic Blood Pressure	89.4	75.4	<0.001
Stroke Volume	81.4	69.5	<0.001
Cardiac Output	7488.8	4963.7	<0.001

Table 3: Comparison of Mean Hemodynamic Values in long term Smokers after cigarette smoking and at rest

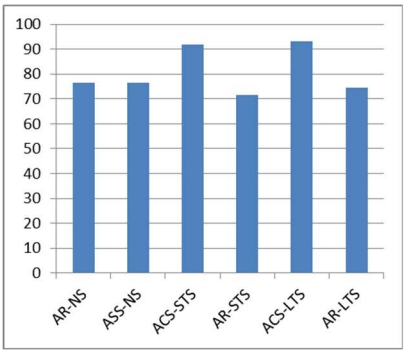
			P value
Variable	Mean	Mean	
Heart Rate	93.3	74.4	<0.001
Systolic Blood Pressure	138.7	120	<0.001
Diastolic Blood Pressure	92.8	80.6	<0.001
Stroke Volume	81.5	71.3	<0.001
Cardiac Output	7583.1	5296.4	<0.001

Table 4: Comparison of Mean Hemodynamic Values in short term Smokers after cigarette smoking and non-smokers after sham smoking

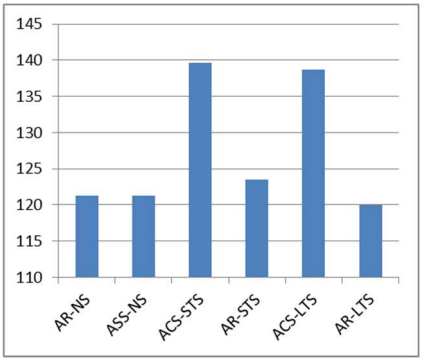
			P value
Variable	Mean	Mean	
Heart Rate	91.8	76.5	<0.001
Systolic Blood Pressure	139.6	121.3	<0.001
Diastolic Blood Pressure	89.4	73.4	<0.001
Stroke Volume	81.4	58.6	<0.001
Cardiac Output	7488.9	4473.3	<0.001

Table/Fig-1: Comparison of Mean Hemodynamic Values in long term Smokers after cigarette smoking and non-smokers after sham smoking

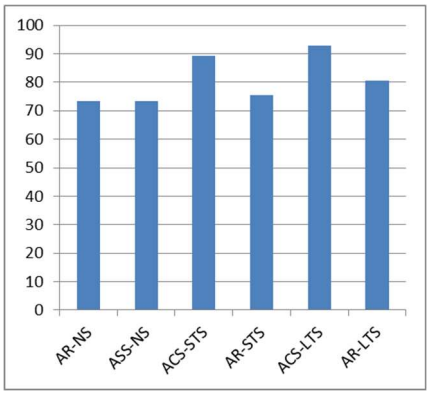
			P value
Variable	Mean	Mean	
Heart Rate	93.3	76.5	<0.001
Systolic Blood Pressure	138.7	121.3	<0.001
Diastolic Blood Pressure	92.8	73.4	<0.001
Stroke Volume	81.5	58.6	<0.001
Cardiac Output	7583.1	4473.3	<0.001



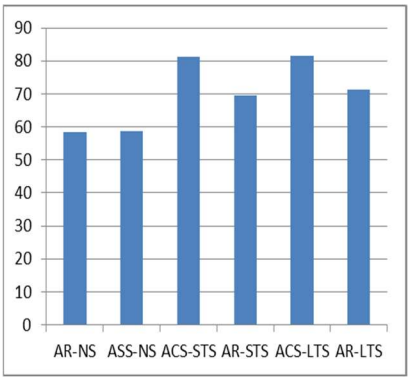
Graph 1: *Comparison of Mean Heart rate in three groups. AR-at rest, NS-nonsmokers, ASS-after sham smoking, STS-short term smokers, ACS-after cigarette smoking, LTS-long term smokers*



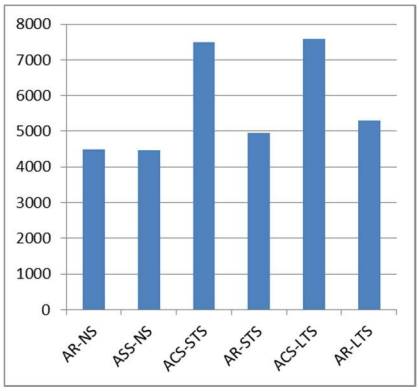
Graph 2: *Comparison of Mean Systolic BP in three groups. AR-at rest, NS-nonsmokers, ASS-after sham smoking, STS-short term smokers, ACS-after cigarette smoking, LTS-long term smokers*



Graph 3: *Comparison of Mean Diastolic BP in three groups. AR-at rest, NS-nonsmokers, ASS-after sham smoking, STS-short term smokers, ACS-after cigarette smoking, LTS-long term smokers*



Graph 4: *Comparison of Mean Stroke Volume in three groups. AR-at rest, NS-nonsmokers, ASS-after sham smoking, STS-short term smokers, ACS-after cigarette smoking, LTS-long term smokers*



Graph 5: *Comparison of Mean Cardiac output in three groups. AR-at rest, NS-nonsmokers, ASS-after sham smoking, STS-short term smokers, ACS-after cigarette smoking, LTS-long term smokers*

Discussion

The results of the present study conducted with an aim to study the effect of smoking tobacco cigarette on cardiovascular system in both short and long term smokers shows that smoking causes significant acute hemodynamic changes in both the groups. After removing the potential confounding factors we found that smoking tobacco alone causes these changes irrespective of age and duration of exposure. The significant acute hemodynamic effects includes immediate rise in heart rate, blood pressure, stroke volume and cardiac output. Therefore, in men, our findings support the other studies, which observed that cigarette consumption has acute hemodynamic effects. Not only heavy, but also moderate smokers showed significant acute hemodynamic changes. "Sham" smoking does not cause a change, while smoking and inhaling does cause a considerable increase in the cardiac output and stroke volume [(7)]. The presence of significant effects of cigarette smoking on heart rate and blood pressure observed in this study confirms early reports that found significant elevation in both the cardiovascular parameters and strengthens the findings by showing that the functional changes after cigarette smoking can be detected immediately. In the current study, the evidence of changes in stroke volume and cardiac output after cigarette smoking is also in accordance with current available data [16, 27]. This study has shown that smoking of first cigarette increases the heart rate and both systolic and diastolic blood pressure and the functional changes persisted even after thirty minutes. Groppelli et.al.[(45)] has observed that the changes in heart rate and blood pressure were persistently higher during the smoking hour than during the nonsmoking hour. Miraian.J.F.Kool et.al.[(68)]also found a significant acute increase in heart rate and blood pressure after smoking one cigarette and concluded that this might be a more important risk than long term effects for the acute ischemic events. The results of this study agree with this fact, as there was a much greater increase in the heart rate, blood pressure, stroke volume and cardiac output when smoke was inhaled than when it was not [(22)]. The current study has shown that both the short term and long term smokers has shown significant hemodynamic changes immediately after smoking first cigarette and the rise in the parameters continued throughout the duration of the study. The study done by David C. Moses et al[(23)] agrees with the results of the present study. The mechanism of an increased cardiac output occurring as a result of the inhalation of cigarette smoke is due to the combined effects of an increase in the heart rate and the stroke volume and both these variables increased in all the smokers studied. Cigarette smokers have increased risks of various manifestations of cardiovascular disease at all ages, and with increasing age the absolute excess risks become substantial. In counseling patients who smoke, health professionals can provide abundant information on the large and almost immediate decrease in cardiovascular risks following smoking cessation. Since the risk of cardiovascular disease is reduced significantly in even elderly smokers after cessation, the clear public health message is that it is never too late to quit. In summary, the present study shows significant hemodynamic changes in smokers after smoking one cigarette compared to the values at rest and also to the nonsmokers who were restricted to sham smoking. The acute hemodynamic differences associated with smoking that were observed in this study did not differed with age and duration of exposure and the changes persisted for longer duration even after end of smoking.These acute changes can lead to acute cardiovascular events which is more important risk than long-term effects of tobacco smoking and in heavy smokers who are always in a state of constant abnormal hemodynamic state.Furthermore, because BP levels in smokers are

rarely recorded during or immediately after smoking when acute rises in BP occur, usual BP levels of smokers tend to be systematically underestimated. Because smoking and BP have been shown to exert a synergistic adverse effect on the risk of coronary heart disease,[3] it is critical that persons with raised BP are advised to stop smoking. The alteration in hemodynamics should raise the concern with respect to the acute cardiovascular risks and recommendation for counseling the smokers to quit smoking and routine.

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Authors:

1. Dr.MohdRasheeduddin Imran.
Assiatant Professor, Department of Physiology,
AlluriSitaramaRaju Academy of Medical Sciences, Eluru-534004,
West Godavari Dist, A.P. India.

2. Dr.P.SambasivaRao.
Assiatant Professor, Department of Physiology,
AlluriSitaramaRaju Academy of Medical Sciences, Eluru-534004,
West Godavari Dist, A.P. India.

Contact details:
Dr.MohdRasheeduddin Imran.
Assiatant Professor, Department of Physiology,
AlluriSitaramaRaju Academy of Medical Sciences, Eluru-534004,
West Godavari Dist, A.P. India.
Email: dr.imran1980@gmail.com