Article

Acute Effect of Cigarette Smoking On HRV in Current Cigarette Smokers

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Abstract

Background: Cigarette smoking induced increased sympathetic activity is one of the major independent risk factor for cardiac morbidity and mortality. **Objective:**To assess acute effects of smoking on neuro cardiovascular regulation by analysis of time domain measures of HRV in current regular healthy male cigarette smoker. **Methods:**This comparative analytical study was conducted in the Department of Physiology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Shahbag, Dhaka from July 2011 to June 2012. 120 apparently healthy male current regular cigarette smoker aged 20-55 years were participated in the study group. Age and BMI matched 70 apparently healthy male non smoker subjects were studied as control. To observe the acute effects data were recorded 5 and 30 min after finishing a cigarette. Time domain measures of HRV were recorded by a RMS digital polyrite D. Statistical analysis was done by independent sample t test and paired sample t test.**Results:** Resting pulse rate, SBP, DBP, mean heart rate were significantly higher ($p<0.001$) and mean R-R interval, SDNN, RMSSD and total power were significantly lower ($p<0.001$) in all smokers in comparison to those of healthy control.In addition, all time domain parameters were significantly decreased from their corresponding baseline value just 5 minutes after smoking and returned close to their baseline value after 30 minutes of smoking but it remained significantly lower than control value. **Conclusion:** The result of this study concludes that cigarette smoking had acute effect on cardiac autonomic function causing depressed vagal activity and overbalance of sympathetic function..

Keywords: Cardiac autonomic nerve function, acute effect of smoking.

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Introduction

Cigarette smoking causes increased acute cardiac events such as ventricular fibrillation, myocardial infarction particularly in the presence of pre-existing coronary artery disease[1] Studies on smoking reported impaired cardiac autonomic nerve function in long term cigarette smokers. Various studies investigated changes in cardiac autonomic nerve function caused by cigarette smoking of varying severity. [2-4]

Among the several underlying mechanism focusing the relation between cigarette smoking and acute cardiac events, cardiac sympathetic hyperactivity has been noted as a dominant feature in cigarette smokers. This observation stimulates researchers to investigate the relationship in depth between cigarette smoking and cardiac autonomic nerve function.[2,3,5]

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A study compared cardiac autonomic function between non smoker and smoker by short term HRV analysis under controlled respiration and reported reduced vagal modulation in heavy smokers[2].

Heart rate variability is the most sensitive and qualitative marker individual measurement of sympathetic and parasympathetic activity. In clinical practice, heart rate variability has been shown to be a valuable non-invasive tool for the assessment of autonomic regulation of cardiovascular function. Frequency domain & time domain parameters have been recommended for HRV analysis with 5-min (Short-term) recordings. The time domain measures used for HRV measurement are mean heart rate, mean R-R interval, SDNN, RMSSD [6].

The role of acute effect of cigarette smoking become research topic for cardiovascular health when it was found out that acute increase in plasma catecholamine and cardiac nor epinephrine spillovers follows cigarette smoking[7]. Therefore, several investigators studied the acute effect of cigarette smoking on HRV[8-10].

Some investigators studied acute effect of smoking on sympathetic and parasympathetic activity by analysis of Heart rate variability. They found significant decrease in RR, SDNN, RMSSD just 5 min after smoking in smokers. It has been suggest that cigarette smoking immediately alters cardiovascular autonomic regulation, which may causes cardiovascular complications [8] In addition, progressively decreased mean R-R interval are also found in passed smokers and habitual smokers during and after 5 min of smoking[9].

Though several studies investigated the acute effect of cigarette smoking by observing after 5 min of smoking but few studies observed it at multiple session and no study compared non smoker with smoker at acute effect of smoking on HRV in the same setting. Therefore, this study was designed to observe the acute effect of smoking on HRV parameters and its reversal in cigarette smokers.

Methods

This comparative analytical study was conducted in the Department of Physiology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Shahbag, Dhaka from July 2011 tot June 2012. Protocol of this study was approved by institutional review board of BSMMU. For this study, 190 apparently healthy male subjects with age 20-55 years were enrolled.120 apparently healthy male subjects were current regular cigarette smokers. HRV parameters of these smokers were observed at baseline (before smoking) and successively 5 min and 30 min after finishing a cigarette. For comparison, age and BMI matched 70 apparently healthy male non smoker subjects were studied as control. The smokers of this study were hospital staffs of Bangabandhu Sheikh Mujib Medical University, motor vehicle drivers and subjects living in Dhaka city with similar socio economic status.All subjects were free from history of active respiratory infection, history of coronary artery disease, congestive heart failure, Diabetes Mellitus, history of consumption of other tobacco products or alcohol, thyroid disorder, renal or hepatic dysfunction, history of taking drugs affecting autonomic nervous system and any psychiatric illness.

After selection and proper counseling, the risk and benefit and detail procedure of the study were explained in details to each subject. They were encouraged for voluntary participation. Informed written consents were taken from them.

The subjects were advised to follow several instructions from the previous night before the examination day. They were advised to have finished their meal by 9:00 pm and to have a sound sleep and should be refrain of any physical or mental stress and not to take any sedatives or any drugs affecting central nervous system and

also advised to refrain from smoking cigarette for 12 hours before the nerve function tests done. Then he was asked to attend the Department of Physiology of Bangabandhu Sheikh Mujib Medical University between 9:00 a.m. to11:00 a.m. on the day of examination. They were advised to take light breakfast in the morning without any tea, coffee or smoking on the test day. All the examination was done between 9 am to 2 pm in the Autonomic Nerve Function Test laboratory under controlled laboratory condition in the Department of Physiology, BSMMU.

At first, the subject was allowed to take rest for 5 minutes. Then the interview and details of personal history, drug history, past medical history and all details of smoking history of the subject was taken. Recording of blood pressure and pulse rate was done. Then the subject was prepared to undergo Autonomic Nerve Function Test. The subject was kept in supine position in a bed for 15-20 minutes in a cool, calm and controlled environment in the lab. During this period subject was advised to refrain from talking, eating or drinking and physical or any mental activity even sleep.

Then all preparations for recording of the Heart Rate Variability parameters by a multichannel polyrite (RMS Polyrite) was finalized according to task force[6]. The HRV parameters were measured by time domain methods such as mean heart rate, mean R-R interval, SDNN, RMSSD, variance. At first a baseline 5 min recording was taken keeping the subject in supine position. Then the subject was allowed to smoke one cigarette. HRV recording was taken 5 and 30 minutes after the cigarette was finished.

Data were expressed as mean ± SE. Comparison of study variables between control and study groups and study subgroups was performed by "unpaired t" test. Paired t test compared the acute effects of smoking on different parameters in smoker. P value <0.05 was accepted as level of significance.

Results

The baseline characteristics of non smokers and smokers are presented in Table I.

In this study, the mean values of resting pulse rate, systolic and diastolicblood pressure were significantly higher (P<0.001) in all smokers than non smokers.

In this study, the mean values of mean heart rate were significantly higher in smoker than that of control. In acute effect of smoking in smokers, the mean heart rate were significantly increased after 5 min of smoking and returned towards baseline after 30 min of smoking. Again mean heart rate after 30 minutes of smoking remained significantly higher than control**.**

In the present study, significantly lower baseline values of mean R-R interval, SDNN, RMSSD and variance in smokers compared to non smokers was published in our previous publication[2,4,7,14]. In the study of acute effect in smokers, the mean R-R interval, SDNN, RMSSD and variance were significantly decreased just 5min after smoking and all values returned towards baseline after 30 min of smoking except SDNN. Again the value of mean R-R interval, SDNN, RMSSD and variance after 30 minutes of smoking remained significantly lower than non smokers.

TableI: Basline characteristics in smokers and non smokers (n=190)

Parameters	Non smoker (n=70)	Smoker (n=120)
Age (years)	32.3±0.97 (22-52)	32.56±0.66 (22-49)
BMI (Kg/m^2)	26.67±0.43 (19.88-34.24)	24.99±0.31 (17.51-33.78)
Pulse (beat/min)	72±0.58 (60-80)	78±0.5*** (62-99)
SBP(mm of Hg)	112±0.98 (100-126)	121.63±1.01** (100-140)
DBP (mm of Hg)	70±0.91 (60-85)	75.37±0.72*** (60-90)

Data expressed mean ±SE .Figure in parenthesis indicate ranges.Data are analyzed by unpaired t test ***P<0.001 SBP=Systolic blood pressure DBP= Diastolic blood pressure BMI= Body mass index

TableII: Time domain parameters in smokers and non smokers (n=190)

Parameters	Non-smoker	Smoker		
		Baseline	5 min after smoking	30 min after smoking
Mean heart rate	72.83±0.66 (65-85)	79.38±1.84*** (62-113)	81.85±1.85## (65-115)	79.3±1.52¥¥¥ (61-110)
Mean R-R interval	0.76±0.01 (0.6-0.91)	0.71±0.01*** (0.52-0.88)	0.68±0.01### (0.48-0.96)	0.70±0.01¥¥¥ (0.56-0.93)
SDNN	131.32±3.32 (89.01-189.07)	77.01±3.41*** (20.44-122.63)	70.60±3.63### (15.56-112.56)	74.35±3.32¥¥¥¶ (19.67-118.67)
RMSSD	64.73±1.23 (20.08-104.7)	49.22±4.29*** (10.31-99.5)	45.21±3.88### (8.54-92.5)	47.84± 4.07¥¥¥ (9.44-91.4)
Variance	3347.97±101.03 (897.4-5782.7)	2774.5±137.8*** (1034.5-5123.4)	2573.27±137.54### (589.2-4778.2)	2789.89±135.99¥¥¥ (1034.2-5123.3)

*nonsmoker vs baseline, (*** = p<0.001), # 5min after smoking vs baseline, (### = p<0.001, ## = p<0.01)
¥ 30 min after smoking vs nonsmoker, (¥¥¥= p<0.001), ¶30 min after smoking vs baseline,(¶ = p<0.05)

Discussion

In the present study, the influence of cigarette smoking on cardiovascular health was investigated by assessing cardiac autonomic nervous activity in healthy male smokers. Furthermore, acute effect of smoking on cardiac autonomic nerve activity was studied by comparing the data before and after smoking of cigarette in smokers. In all the cases cardiac autonomic nerve activity was evaluated by time domain of HRV analysis.Simple time domain measures including mean R-R interval and mean heart rate reflects vagal activity. Statistical time domain measures, SDNN and RMSSD for general autonomic balance. Variance reflects the overall variability autonomic activity[10].

The present results showed that smoking of cigarette caused immediate effect on CANA indicated by decreased values of all the time domain parameters and increased HR after 5 minute of smoking. It is suggestive of vagal suppression and sympathetic overbalance due to immediate effect of nicotine[3].

A group of researchers observed that nicotine dependent stimulation of sympathetic control and inactivation of vagal cardiovascular control is responsible for hemodynamic changes in smokers [3,11].

Nicotine is the principle mediator of acute effects of smoking on neurocardiovascular regulation. In addition to its chronic effect, nicotine also exerts harmful effects during the acute period of smoking. These effects are mediated through its action on central nervous system as well as afferent and efferent division of autonomic nervous system[3].The effect of nicotine from cigarette smoke on cardiovascular control can be further added by its effect on catecholamine release from adrenal gland. Nicotine binds with ganglionic type nicotinic receptor in adrenal medulla and this stimulates adrenal medullary secretion of catecholamine resulting in increased flow of adrenaline and noradrenalin into the bloodstream. The rise of circulatory epinephrine causes an increase in heart rate, blood pressure and blood glucose level[12].

The attenuated vagal activity and concomittant rise of sympathetic activity in smoker can be explained by the fact that discharge in cardiac sympathetic fibers inhibits cardiac vagal effect by releasing neuropeptide Y as a co-transmitter from its ending [13].

In the context of the present study, the higher values of some simple autonomic indexes such as resting pulse rate, resting blood pressure were found in all smokers than those of non-smoker subjects. Though these values in both smoker and non smoker subjects were within physiological limit and all the subjects were apparently healthy but the trend of these values in the smokers were at the upper limit of their normal value.

This study also investigated the immediate effect of smoking on cardiac autonomic nerve function in smokers by assessing HRV measures after 5 minutes following smoking of a cigarette. Again, to assess the recovery of acute effect of smoking, this study also observed cardiac autonomic nerve function 30 minutes after smoking a cigarette. The present result suggest significantly increased sympathetic and decreased parasympathetic activity during the acute period of smoking and the reversal of this changes towards its pre smoking value 30 minutes after cessation of smoking. These findings are comparable to the observation of Hayanoet al[5].

Again, Hayano et al. noted these autonomic changes 3 minutes after smoking. They reported that change of parasympathetic activity 3 minutes after smoking and sympathetic changes 5 minutes after smoking and both return to baseline value 25 minutes after smoking cessation[7,9].Moreover, Andrikopoulos et al. observed transient depression of vagal control and moderate elevation of sympathetic activity was caused by cigarette smoking.They also found these changes.All these literatures viewed that all these changes are consequence of suppression of cardiac vagal activity after smoking which supports our results[7,13].

All these features suggest that smoking on short term basis causes an acute and transient decrease in cardiac vagal activity and also exaggerate cardiac sympathetic activity. Furthermore, heavy smoking also induces long term reduction in cardiac vagal control and sustained overall increased sympathetic activity; even it can be further increased though transitory following an acute episode of cigarette smoking[13].

These observations from the present study suggest that cigarette smoking induced autonomic changes in the acute period of smoking was remarkably sustained for a notable period of time after cessation of smoking. Persistence of this altered state of cardiac autonomic nerve function compared to non smoker is suggestive of development of impaired cardiac autonomic nerve function in cigarette smokers.

Conclusion

From the results of the present study, it has been observed that even in the resting state, increased sympathetic activity with concomitant suppression of cardiac vagal modulation occurs in regular cigarette smokers which are further exaggerated immediately after smoking a cigarette. Throughout the study, there is clear indication that cardiac autonomic functions are deranged due to cigarette smoking.

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