

Effect of coffee on blood pressure and electrocardiographic changes in nicotine users

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ABSTRACT

Objectives: The objectives of this study were to see the effect of coffee on Heart rate Blood pressure and ECG changes in nicotine users. **Methods:** The study was conducted on 120 volunteers aged 21-40 years and with body mass index (BMI) between 17.3-28.0 kg/m². The subjects were divided into two groups: Control (n=40) and Study group (n=80). **Results:** Observation suggests that increment in blood pressure, recorded in study group after coffee ingestion, was lesser than that of control group. We also observed that there were no significant changes in diastolic blood pressure in any group while the mean arterial pressure was higher in both the groups following coffee ingestion. **Conclusion:** Coffee ingestion decreases the heart rate with no changes in QTc, hence we concluded that less amount of coffee ingestion may not be harmful.

Key words: Coffee, Nicotine, Blood pressure, QTc interval

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INTRODUCTION

Coffee is one of the most widely consumed beverages in the world. It is the primary source of caffeine in many populations, but also contains several other biologically active components that may have either harmful or beneficial cardiovascular effects.¹ Caffeine is widely consumed by people of all ages in world as well as in India.² In fact, it is the world's most widely consumed stimulant, 54% of adults in America consuming on average three cups of coffee per day.³ Aside from occurring organically in tea and coffee, caffeine is now an additive in soft drinks, energy drinks, chocolates, potato chips, bottled water, chewing gum and medication which confirms its growing popularity.⁴ There has been an increase in reports of caffeine-intoxication since 1982, with 41 cases of caffeine abuse reported in the United States from 2002 to 2004.⁵ Caffeine induces various acute cardiovascular effects such as an up regulation of circulating catecholamines, leading to increases in systolic and diastolic blood pressure.⁶ An increase in the respiration rate (RR) is the prime effect dependent on the plasma caffeine levels.⁷ Recently there has been an increase in energy drink consumption leading

to caffeine abuse, with aggressive marketing and poor awareness on the consequences of high caffeine use. Prolongation or reduction of the electrocardiographic QT interval duration is associated with increased risk of ventricular arrhythmias and sudden cardiac death.⁸ Population studies have also shown associations between smaller increases in QT interval duration and total mortality, cardiovascular mortality, and sudden cardiac death.⁹ Besides genetic disorders and pharmacologic agents that can cause marked prolongation or shortening of the QT interval.¹⁰

Nicotine, which is the major component of cigarette, affects cardiovascular function and could contribute to cardiovascular diseases. Cigarette smoking accelerates atherosclerosis, producing premature atherosclerosis at epicardial coronary arteries, the aorta, the carotid and cerebral arteries and large arteries in the peripheral circulation.^{11,12} Smoking is also associated with an increased risk of acute cardiovascular events, including acute myocardial infarction, sudden death and stroke. With best of our knowledge none study has been published so far regarding effect of coffee in nicotine user Indian subjects. Therefore in present study we evaluated the blood pressure

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and electrocardiographic changes in nicotine users of north Indian population.

MATERIALS AND METHODS

Subjects

The study was approved by the ethics committee of University. The study was conducted on 120 volunteers aged 21-40 years and with body mass index (BMI) between 17.3-28.0 kg/m². The subjects were divided into two groups: Control (n=40) and Study group (n=80). Exclusion criteria for control group were any dyslipidemia, diabetes, systemic disease, any medication treatment, and smoking of any tobacco products, pregnancy or lactation in the past six months. The nicotine users (tobacco chewer/smokers) more than five years were included in study group; Exclusion criteria for study group were any dyslipidemia, diabetes, or systemic disease, any medication treatment.

Study protocol

Each subject was instructed to abstain from any form of exercise for 24 hours before each testing session. Immediately on arrival at the laboratory, the subject rested quietly in a chair.

Recording of Electrocardiogram

Basal Electrocardiogram (ECG) and blood pressure was recorded for each subject. One cup of coffee were given to subjects of both study and control group. Each cup of caffeinated coffee (CC) contained a 130 mg of caffeine. Computed standard lead-II ECG (CARDIART) was recorded at three minutes before and forty minutes after coffee ingestion. Electrocardiographic measurements like QRS, RR, HR, QT and QTc (Bazzett's formula) intervals of each ECG were automatically measured using dedicated software. Systolic and Diastolic blood pressure were recorded with sphygmomanometer (gold standard).

Statistical analysis

All data are presented as means \pm SD of the means. Basal pair wise comparisons between the two groups (study and control) were tested for statistical significance using the paired Student's t-test.

RESULTS

Our data suggests that the heart rate in nicotine users was significantly ($p < 0.05$) higher than control group (Table I). The systolic blood pressure was higher in both groups (study and control) forty minutes following the ingestion of caffeinated coffee. The change in systolic blood pressure in control and study groups was 16% and 12.5% respectively higher than its basal value. Observation suggests that increment in blood pressure, recorded in

study group after coffee ingestion, was lesser than that of control group.

We also observed that there were no significant changes in diastolic blood pressure in any group while the mean arterial pressure was also higher in both the groups following coffee ingestion.

We also observe that there was decrease in heart rate in both groups 40 minutes after caffeinated coffee ingestion but percentage in heart rate decrease was lesser in study group. The Figure 1 reports the changes in QT and QTc intervals after caffeinated coffee ingestion. Significant changes in the QT duration (352.00 ± 15.49 vs 329.00 ± 25.14 ms $P < 0.05$) was observed forty minutes after coffee ingestion in control group but not in study group (359.00 ± 25.49 vs 374.00 ± 32.72). In fact the QRS and QTc durations did not significantly changes after caffeinated coffee ingestion in either group.

DISCUSSION

Present study has been planned to elucidate the effect of coffee in control and nicotine user subjects. A cup of coffee can contain 100-150 mg of caffeine, and this may elevate plasma caffeine levels to its peak level.¹³ Caffeine enters all tissue compartment¹⁴ and through its actions at the adenosine receptor,¹⁵ it has widespread effects on the central nervous system and all peripheral tissues. Caffeine has been shown to inhibit both A1 and A2 adenosine receptors.¹⁶ Our observations showed that there was significant difference in basal heart rate in control and study group. The Heart rate in subjects of study group was higher; it may be due to nicotine present in blood of study group. Nicotine binds to nicotinic cholinergic receptors, which are located in the brain, autonomic ganglia, the adrenals and neuromuscular junction¹⁷ Nicotine works primarily by enhancing the release of various neurotransmitters, including epinephrine, norepinephrine, dopamine, acetylcholine, serotonin, vasopressin and glutamate.¹⁸ The main cardiovascular effect of nicotine is sympathetic neural stimulation.

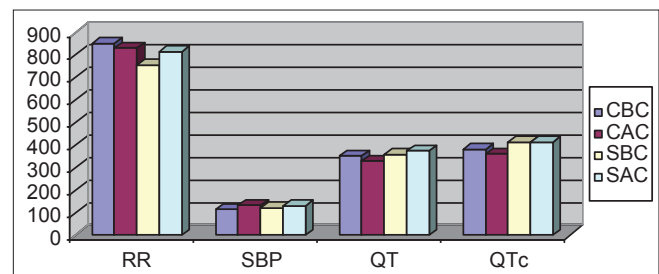


Figure 1: Effect of coffee on various parameters of control and study groups. CBC: Data of control group (before coffee). CAC: Data of Control group (after coffee), SBC: Data of study group (before coffee), SAC: Data of study group (after coffee)

Table 1: Various parameters in control and study group

Parameter	Control group		Study group	
	Before coffee ingestion	After coffee ingestion	Before coffee ingestion	After coffee ingestion
HR (bpm)	71.25±4.96	70.60±5.06	80.8±08.34	73.7±6.46
SBP (mmHg)	114.37±10.68	132.61±12.56	120.87±16.28	130.43±6.25
DBP (mmHg)	74.50±9.06	73.50±8.86	73.50±8.86	76.89±7.36
QRS (ms)	80.00±10.54	83.00±11.59	86.00±11.73	89.00±9.94
RR (ms)	852.00±32.93	832.00±63.38	756.00±36.50	815.00±72.76
QT (ms)	352.00±15.49	329.00±25.14	359.00±25.49	374.00±32.72
QTc (ms)	381.40±15.67	361.50±34.48	413.00±37.94	414.10±22.1

Our data suggests that the heart rate was decreased in control as well as in study groups while the systolic blood pressure was raised in both the groups. It reveals that the ingested caffeine may not work through the sympathetic system because sympathetic stimulation causes secretion of norepinephrine which has positive chronotropic and as well as positive inotropic effects. Although this preliminary study did not able to identify any mechanism responsible for the increase in systolic blood pressure; it may speculate that caffeine may act via increase in cAMP.

The methylxanthine caffeine is a nonselective adenosine receptor antagonist and phosphodiesterase inhibitor it may leads to increase in the cytosolic calcium. Basal QTc interval was found to be raised in study group showed that nicotine alters the repolarization state.

In fact, it has been recently reported¹ that caffeine is responsible for a reduced myocardial blood flow response to physical exercise. The possibility also exists that caffeine may influence both left ventricular repolarization and diastolic function modifying the sympathetic/parasympathetic balance; however no hormonal or instrumental measures were considered in this study in order to investigate this hypothesis. We conclude that despite coffee acutely increases systolic and diastolic blood pressure but not the heart rate. Caffeinated coffee does not acutely induce any significant change in the QTc interval duration in the subjects. QTc interval represented the repolarization of myocardium and K⁺ channels set the membrane potential as well as the excitability of most living cells. The K⁺ ions are predominantly responsible for the long QTc.

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Authors Contribution:

PK: Development of hypothesis, **DKV & JN:** Collection of data, **PK & AG:** Analysis of data.

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