

Original Research

Assessment of Effect of Coffee on Blood Pressure and Electrocardiographic Changes in Nicotine Users at a Tertiary Centre

Sunil Kumar¹, Shazia Haque Arshad², Ajay Kumar Singh³, Sarbil Kumari⁴, Neera Kumari⁵

^{1,3}Tutor, Department of Physiology, Bhagwan Mahavir Institute of Medical Sciences, Pawapuri, Nalanda, Bihar, India.

²Tutor, Department of Physiology, Sri Krishna Medical College and Hospital, Muzaffarpur, Bihar, India.

⁴Professor and Head of Department, Department of Physiology, Bhagwan Mahavir Institute of Medical Sciences, Pawapuri, Nalanda, Bihar, India

⁵Assistant Professor and Head of Department, Department of Physiology, Sri Krishna Medical College and Hospital, Muzaffarpur Bihar, India.

Corresponding Author: Ajay Kumar Singh

Tutor, Department of Physiology, Bhagwan Mahavir Institute of Medical Sciences, Pawapuri, Nalanda, Bihar, India.

Email: aks.buxarindia@gmail.com

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ABSTRACT

Background: Coffee is rich in antioxidants, which can help neutralise harmful free radicals in the body.

Aims and Objectives: The present study was conducted to assess the effect of coffee on blood pressure and electrocardiographic changes in nicotine users.

Materials and Methods: 80 subjects aged 21–45 years with a body mass index (BMI) between 17.3 and 28.0 kg/m² of both genders were divided into 2 groups of 40 each. Group I had the habit of smoking for more than 5 years, and Group II was healthy. An assessment of the electrocardiogram (ECG) and blood pressure was done. The computed standard lead-II ECG (CARDIART) was recorded three minutes before and forty-five minutes after coffee ingestion. Electrocardiographic measurements like QRS, RR, HR, QT, and QTc intervals of each ECG were automatically measured.

Results: Group I had 25 males and 15 females, and Group II had 20 males and 20 females. Before and after coffee ingestion in groups I and II, the mean SBP (mmHg) was 121.4 and 132.2 in group I and 116.4 and 130.6 in group II. DBP (mmHg) was 74.6 and 76.2 in group I and 74.6 and 73.2 in group II. HR (bpm) was 80.4 and 74.2 in group I and 72.8 and 71.0 in group II. QRS (ms) was 86.5 and 89.2 in group I and 80.2 and 82.4 in group II. RR (ms) was 780.2 and 801.4 in group I and 854.2 and 836.4 in group II. QT (ms) was 360.6 and 372.4 in group I and 354.2 and 330.6 in group II. QTc (ms) was 416.4 and 418.2 in group I and 380.4 and 364.2 in group II. The difference was significant ($P < 0.05$).

Conclusion: Caffeinated coffee does not acutely induce any significant change in the QTc interval duration in the subjects.

Key words: coffee, ECG, SBP, DBP

Introduction

Coffee is one of the most widely consumed beverages in the world. In addition to being the main source of caffeine for many populations, it also contains several other physiologically active substances that may have advantageous or detrimental effects on the cardiovascular system. Coffee is rich in antioxidants, which can help neutralise harmful free radicals in the body. Antioxidants are generally considered beneficial for heart health. Moderate coffee drinking may be associated with a lower risk of stroke, according to some studies [1]. The exact mechanisms are not fully understood but may be related to the antioxidant and anti-inflammatory properties of coffee. The inner lining of blood vessels is called the endothelium. Some research indicates that coffee may have a positive impact on endothelial function, which is essential for maintaining healthy blood vessels [2]. For some individuals, excessive caffeine intake can lead to increased heart rate, palpitations, or even arrhythmias. People vary in their sensitivity to caffeine, so what might be a strong espresso for one person could be a mild buzz for another [3]. Caffeine can cause a temporary increase in blood pressure. While this effect is usually mild and short-lived, individuals with hypertension may be more sensitive to it. However, regular coffee drinkers may develop a tolerance for this effect over time [4]. The primary ingredient in cigarettes, nicotine, alters cardiovascular function and may be a factor in the development of cardiovascular diseases. Cigarette smoking causes premature atherosclerosis at the aorta, carotid and cerebral arteries, large arteries in the peripheral circulation, and epicardial coronary arteries. It also accelerates atherosclerosis [5].

Aims and objectives

The present study was conducted to assess the effect of coffee on blood pressure and electrocardiographic changes in nicotine users.

Materials & Methods

The present prospective case-control study was conducted on 80 (Eighty) volunteers aged 21-45 years and with body mass index (BMI) between 17.3-28.0 kg/m² of both genders attending the OPD of General Medicine in collaboration with the Department of Physiology at Bhagwan Mahavir Institute of Medical Sciences, Pawapuri, Nalanda, Bihar, India and Department of Physiology at Sri Krishna Medical College and Hospital, Muzaffarpur Bihar, India. The study was approved by the institutional ethical committee before its commencement. Each gave written consent to take part in the study. The duration of the study was six months, from March 2023 to August 2023.

Inclusion criteria

- The nicotine users (tobacco chewer/ smokers) more than five years
- Age between 21 - 45 years
- Those who gave informed written consent

Exclusion criteria

- Patients who refused to informed written consent
- Age <21 or above 45 years
- Any dyslipidaemia, diabetes, systemic disease or taking any medication treatment
- Smoking of any tobacco products and
- Pregnancy

Data such as name, age, gender etc. was recorded. All were divided into 2 groups of 40 each. Group I had habit of smoking for more than 5 years and group II was healthy control. An assessment of electrocardiogram (ECG) and blood pressure was done. One cup of coffee was given to all subjects containing 130 mg of caffeine. Computed standard lead-II ECG (CARDIART) was recorded at three minutes before and forty- five minutes after coffee ingestion. Electrocardiographic measurements like QRS, RR, HR, QT and QTc intervals of each ECG were automatically measured. Recording of systolic and diastolic blood pressure with a sphygmomanometer was also performed.

Statistical analysis: All statistical data were analysed using IBM SPSS (Statistical Package for the Social Sciences) software, version 22.0 and Microsoft Excel. Data are presented as means \pm SD of the means. The comparisons between the two groups (study and control) were tested for statistical significance using the paired Student's t-test. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

Results

Table I: Gender-wise Distribution of Subjects

Gender	Group I (smokers)	Group II (Control)
Males	25	20
Females	15	20

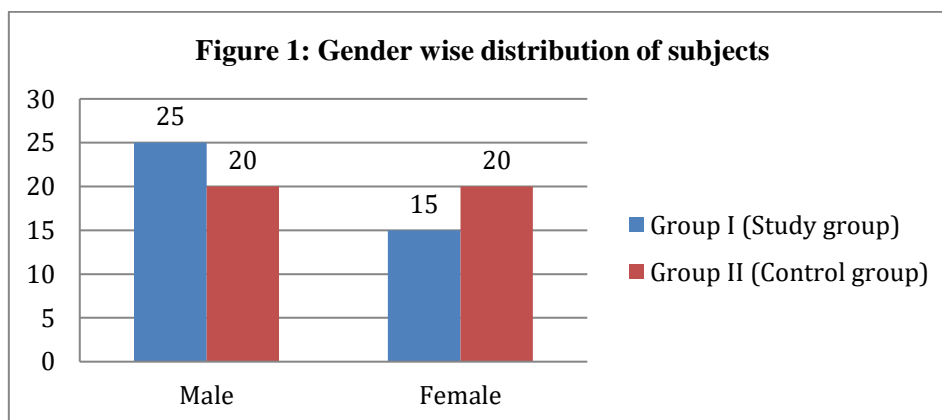
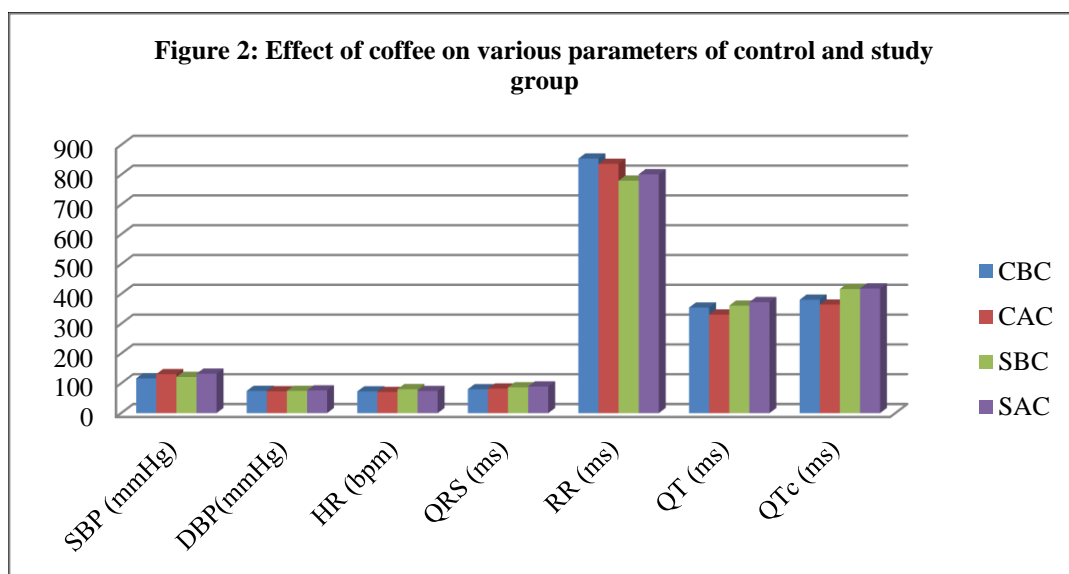


Table I, figure 1, shows that group I had 25 males and 15 females, and group II had 20 males and 20 females. The mean age of volunteers was 32 ± 6.52 years.

Table II: Assessment of parameters before and after ingestion of coffee between the study and control groups

Parameters	Group I (study group)		Group II (control group)		P value
	Before coffee ingestion	After coffee ingestion	Before coffee ingestion	After coffee ingestion	
SBP (mmHg)	121.4±15.81	132.2±5.13	116.4±8.96	130.6±10.75	0.17
DBP(mmHg)	74.6±10.68	76.2±8.61	74.6±8.90	73.2±8.01	0.35
HR (bpm)	80.4±6.81	74.2±5.69	72.8±5.61	71.0±5.21	0.81
QRS (ms)	86.5±12.25	89.25±10.05	80.2±12.03	82.4±13.65	0.62
RR (ms)	780.2±12.85	801.4±78.01	854.2±30.87	836.4±58.93	0.73
QT (ms)	360.6±24.75	372.4±30.81	354.2±12.92	330.6±24.58	0.01*
QTc (ms)	416.4±35.03	418.2±18.83	380.4±14.50	364.2±32.85	0.92

*Significant value



CBC: control group data (before coffee); SBC: study group data (before coffee); SAC: study group data (after coffee); CAC: control group data (after coffee). Table II and Figure 2, shows that before and after coffee ingestion in groups I and II, the mean SBP (mmHg) was 121.4 and 132.2 in group I and 116.4 and 130.6 in group II. DBP (mmHg) was 74.6 and 76.2 in group I and 74.6 and 73.2 in group II. HR (bpm) was 80.4 and 74.2 in group I and 72.8 and 71.0 in group II. QRS (ms) was 86.5 and 89.2 in group I and 80.2 and 82.4 in group II. RR (ms) was 780.2 and 801.4 in group I and 854.2 and 836.4 in group II. QT (ms) was 360.6 and 372.4 in group I and 354.2 and 330.6 in group II. QTc (ms) was 416.4 and 418.2 in group I and 380.4 and 364.2 in group II. The difference was significant ($P < 0.05$).

Discussion

Caffeine induces various acute cardiovascular effects, such as an up-regulation of circulating catecholamine, leading to increases in systolic and diastolic blood pressure [6]. An increase in the respiration rate (RR) is the prime effect dependent on plasma caffeine levels [7]. There is a correlation between prolongation or shortening of the QT interval on electrocardiography and an increased risk of ventricular arrhythmias and sudden cardiac death [8, 9]. The caffeine content of a cup of coffee can range from 100 to 150 mg, which can raise plasma caffeine levels to their peak.¹⁰ Research has also shown associations between smaller increases in QT interval duration and total mortality, cardiovascular mortality, and sudden cardiac death. Besides genetic disorders and pharmacologic agents that can cause marked prolongation or shortening of the QT interval [10,11]. The effects of caffeine on the central nervous system and all peripheral tissues are widespread due to its absorption in all tissue compartments and its actions at the adenosine receptor [11]. Research indicates that caffeine inhibits adenosine receptors A1 and A2 [12]. The current study was carried out to assess the effect of coffee on blood pressure and electrocardiographic changes in individuals who use nicotine. We found that group I had 25 males and 15 females, and group II had 20 males and 20 females. Kumar et al. [13], determined the effect of

coffee on heart rate, blood pressure, and ECG changes in nicotine users. The study was conducted on 120 volunteers aged 21–40 years with a body mass index (BMI) between 17.3 and 28.0 kg/m². The subjects were divided into two groups: the control group (n = 40) and the study group (n = 80). Observation suggests that the increment in blood pressure recorded in the study group after coffee ingestion was less than that of the control group. They observed that there were no significant changes in diastolic blood pressure in any group, while the mean arterial pressure was higher in both groups following coffee ingestion. We found that before and after coffee ingestion in groups I and II, the mean SBP (mmHg) was 121.4 and 132.2 in group I and 116.4 and 130.6 in group II. DBP (mmHg) was 74.6 and 76.2 in group I and 74.6 and 73.2 in group II. HR (bpm) was 80.4 and 74.2 in group I and 72.8 and 71.0 in group II. QRS (ms) was 86.5 and 89.2 in group I and 80.2 and 82.4 in group II. RR (ms) was 780.2 and 801.4 in group I and 854.2 and 836.4 in group II. QT (ms) was 360.6 and 372.4 in group I and 354.2 and 330.6 in group II. QTc (ms) was 416.4 and 418.2 in group I and 380.4 and 364.2 in group II. Noha et al. [14], studied 165 healthy men and women in 6 groups: men and premenopausal women (35–49 years) vs. men and postmenopausal women (50–64 years), with postmenopausal women divided into those taking no hormone replacements (HR), oestrogen alone, or oestrogen and progesterone. Testing during one week of the study involved six days of caffeine maintenance at home (80 mg, 3x/day), followed by testing of responses to a challenge dose of caffeine (250 mg) in the laboratory. The other week, it was necessary to ingest placebos on maintenance and lab days. Using automated monitors, resting blood pressure responses to caffeine were assessed at baseline and 45–60 minutes after ingesting caffeine compared to a placebo. All six groups had a significant increase in systolic blood pressure after ingesting coffee (4 ± 0.6 , $p < 0.01$). Diastolic BP significantly increased in response to caffeine in all (3 ± 0.4 , $p < 0.04$) but in the group of older men (2 ± 1.0 , $p = 0.1$). The observed pressure responses to caffeine did not vary by age. Namdaret al. [15], assessed the acute effect of caffeine in a dose corresponding to two cups of coffee on myocardial blood flow (MBF) in coronary artery disease (CAD). MBF was measured with (15) O-labelled H₂O and Positron Emission Tomography (PET) at rest and after supine bicycle exercise in controls (n = 15, mean age 58 ± 13 years) and in CAD patients (n = 15, mean age 61 ± 9 years). In the latter, segments that were subtended by remote and stenotic coronary arteries were used for assessing regional MBF. Fifty minutes following the 200 mg of oral caffeine ingestion, all measurements were performed again. The ratio of myocardial blood flow (MBF) at rest to MBF during bicycle stress was used for calculating myocardial perfusion reserve (MPR). Caffeine had no effect on resting MBF in either group. In comparison to controls (2.26 ± 0.56 vs. 2.02 ± 0.56 , $P < 0.005$), remote (2.40 ± 0.70 versus 1.78 ± 0.46 , $P < 0.001$), and stenotic segments (1.90 ± 0.41 vs. 1.38 ± 0.30 , $P < 0.001$), the exercise-induced MBF response significantly decreased following caffeine. Caffeine decreased MPR significantly by 14% in controls ($P < 0.05$ vs. baseline). In CAD patients, MPR decreased by 18% ($P < 0.05$ vs. baseline) in remote and by 25% in stenotic segments ($P < 0.01$ vs. baseline).

Limitations of the study

The small sample size and duration of the study are some of its limitations.

Conclusion

In the present study, we found that 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 volunteers. The 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 QT.

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References

1. Mednick SC, Cai DJ, Kanady J and Drummond SPA. Comparing the benefits of caffeine, naps and placebo on verbal, motor and perceptual memory. *Behav Brain Res* 2008; 193:79-86.
2. Reissig CJ, Strain EC and Griffiths RR. Caffeinated energy drinks a growing problem. *Drug Alcohol Depend* 2009; 99: 1-10.
3. Riksen NP, Rongen GA and Smits P. Acute and long-term cardiovascular effects of coffee implications for coronary heart disease. *Pharmacol Ther* 2009; 121: 185-191.
4. Chou T. Wake up and smell the coffee. Caffeine, coffee and the medical consequences. *West J Med* 1992;157: 544-553.

5. Priori SG, Schwartz PJ, Napolitano C, Bloise R, Ronchetti E, Grillo M, et al. Risk stratification in the long-QT syndrome. *N Engl J Med* 2003; 348: 1866-1874.
6. Algra A, Tijssen JG, Roelandt JR, Pool J and Lubsen J. QT interval variables from 24-hour electrocardiography and the two year risk of sudden death. *Br Heart J* 1993; 70: 43-48.
7. Montanez A, Ruskin JN, Hebert PR, Lamas GA and Hennekens CH. Prolonged QTc interval and risks of total and cardiovascular mortality and sudden death in the general population: a review and qualitative overview of the prospective cohort studies. *Arch Intern Med* 2004; 164: 943-948.
8. McBride PE. The health consequences of smoking: Cardiovascular diseases. *Med Clin North Am* 1992; 76:333-353.
9. Wilhelmsen L. Coronary heart disease: epidemiology of smoking and intervention studies of smoking. *Am Heart J* 1988; 115:242- 249.
10. O'Connell SE and Zurzola FJ. Rapid quantitative liquid chromatographic determination of caffeine levels in plasma after oral dosing. *J Pharm Sci* 1984; 73:1009-1011.
11. Spindel E. Action of the methylxanthines on the pituitary and pituitary dependent hormones. *Prog Clin Biol Res* 1984; 158: 355- 363.
12. Smits P, Bockema P, De Abreu R, Thien T and van't Laar A. Evidence for an antagonism between caffeine and adenosine in the human cardiovascular system. *J Cardiovasc Pharmacol* 1987; 10: 136-143.
13. Kumar P, Verma DK, Narayan J, Kanawjia P, Ghildiyal A. Effect of coffee on blood pressure and electrocardiographic changes in nicotine users. *Asian Journal of Medical Sciences*. 2015;6(3):46-8.
14. Noha HF and Thomas LW. Caffeine and Blood Pressure Response: Sex, Age, and Hormonal Status. *Journal of women's health* 2010; 19, 6: 1171-1177.
15. Namdar M, Schepis T, Koepfli P and Gaemperli O. Caffeine impairs myocardial blood flow response to physical exercise in patients with coronary artery disease as well as in age matched controls. *PLoS One* 2009; 22; 4(5):5665.