Effect of Caffeine on Heart Rate and Blood Pressure

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Abstract- Caffeine increases systolic blood pressure to extent of 17% and mean arterial blood pressure by about 11%. Heart rate was also increased. The increase in systolic blood pressure was attributed more to the increased stiffness of the aorta and larger blood vessels rather than increased stroke volume. The increase of aortic stiffness is attributed to the increased production of angiotensin II and catecholamines potentially nor adrenaline. These changes are attributable to the inhibition of adenosine A2a receptors activity in the smooth muscles of blood vessels. The concomitant increase in the release of rennin from the kidneys because of the direct stimulation by caffeine and similar activity on sympathetic ganglions releasing noradrenaline contributes to the increased activity of vascular smooth muscles. In the present study, attempt has been made to know the changes occurring in heart rate and blood pressure after the intake of caffeine.

Index Terms- Caffeine, Angiotensin, Adenosine Receptors

I. INTRODUCTION

Caffeine is the world’s most widely consumed psychoactive substance but like many other psychoactive substances it is legal and unregulated in all countries of the world. The chemical name is 1,3,7-trimethyl-1H purine-2,6(3H,7H)-Dionne, also named as 1,3,7-trimethyl xanthenes or 7 methyl theophylline. Its molecular formula is C8H10N4O2 and molecular mass 194.19g/mole. It is an odourless white crystalline substance or powder. Because of its liquefactive nature and worldwide usage, a lot of work has been done and obtained quite contradictory results by different groups. It was presumed that caffeine is a major source of antioxidants contributing up to 65% of dietary supplement. Epidemiologic studies have found that ingestion of coffee is associated with reduced biomarkers of oxidative stress and helpful in reduction of morbidity in rheumatoid arthritis, chronic obstructive pulmonary disease, asthma, ischemic perfusion, ulcerative colitis, diabetes, sexual type of neurodegenerative diseases and atherosclerosis.

Adenosine is a potent vasodilator but caffeine, an adenosine analogue competitively inhibits the adenosine receptors and brings out vasoconstriction. The psychoactive effects of caffeine are due to the blockade of adenosine receptors of brain. It can increase the systolic blood pressure by enhancing angiotensin II and epinephrine. It can also increase the heart rate by increasing the levels of catecholamines.

II. MATERIALS AND METHODS

Forty healthy nonobese, non-smoking male healthy volunteers of age group between 19-22 years and weight 45-60 kgs not suffering from any cardiac or pulmonary diseases were recruited for this study. The participants were randomly divided into two groups, control and test groups. Twenty students were allocated in control group and twenty in test group. All the participants gave the informed written consent for the study. The study was approved by Institutional Human Ethical Committee on 26/03/2010. The study was conducted according to the guidelines of SVS Medical College, Mahabubnagar.

All the participants were instructed not to take coffee or tea atleast four days prior to the test. Once they were in the lab they were permitted to take rest for 30 minutes. Blood pressure and heart rate were recorded for all the individuals using sphygmomanometer and pulse rate. Later the test group were given caffeine 5mg/kg body weight in gelatine capsules and placebo for control group. They were allowed to take rest for 60 minutes. Later blood pressure and heart rate were recorded in both the test group and control group. The data was tabulated and analysed by using SPSS 16 statistical soft ware.

III. RESULTS

Caffeine caused significant increase in the mean systolic blood pressure (60 minutes post ingestion) from 116.6 to 128.3 mmHg as compared to placebo conditions. The mean rate increased from 72.9 to 77.3 Bpm in test group. No change was observed in control group.

<table>
<thead>
<tr>
<th>TEST GROUP</th>
<th>RESTING</th>
<th>60 MIN AFTER CAFFEINE</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>BP</td>
<td>HR</td>
</tr>
<tr>
<td>MEAN</td>
<td>116.6/72.2</td>
<td>72.9</td>
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<tr>
<td>SD</td>
<td>6.65/4.44</td>
<td>4.52</td>
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### IV. DISCUSSION

Under resting conditions caffeine has been shown to cause increase in blood pressure and systemic vascular resistance\(^5\). Azra Mohammed et al\(^4\) have shown that caffeine can change stiffness of blood vessels may be independent of blood pressure changes. They here also shown that arterial stiffness increases with caffeine and pressor effect of caffeine is predominantly on the vessel resistance rather than an increase in the cardiac output. They also claim that the increase in the sympathetic nervous system activity, serum adrenaline and rennin have been linked, the acute pressor effect is also seen in adrenelectamised patients. Jonson w. daniels et al\(^3\) showed that angiotensin II levels were increased with caffeine. As we know that angiotensin II is a powerful vasoconstringtor, it causes increase in blood pressure. In the present study we noticed increase in values of blood pressure after caffeine intake which clearly substantiates the above statement.

Angiotensin II (ANG) potently enhances catecholamine release from the peripheral sympathetic system\(^5\). Catecholamines released by this mechanism contribute to the vasoconstricting and sodium-retaining properties of ANG\(^6\). In particular, the chronic effects of ANG at moderately elevated levels are promoted by adrenergic pathways that are significantly involved in the development of hypertension\(^7\) \^8\(^. \) Catecholamines can also cause increase in heart rate.

Caffeine with its multiple effects is (1) an adenosine receptor blocker. (2) increasing levels of angiotensin II (3) increasing the levels of catecholamines.

In the present study, an increase in the values of heart rate and blood pressure were noticed with the intake of caffeine when compared to placebo. It has been explained due to the above said multiple effects of caffeine.

### REFERENCES


### AUTHORS

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