Caffeine and Hypertension

Caffeine consumption is prevalent across the population. The stimulation of alertness and focus via the sympathetic nervous system (SNS) make it a routine chemical in many American diets. It is commonly ingested as part of morning rituals to wake up from a basal metabolic state in dosages of about 125 mg per cup of American coffee or to prevent the midday doldrums in the form of diet sodas, “energy” drinks, or designer coffees at 40-200 mg depending on the product and serving consumed. Caffeine is also regularly found in weight loss products serving as the most popular appetite suppressant in over-the-counter supplements and also acts as a diuretic. The widespread use and social acceptance of the stimulant leads one to assume the product is benign to the body and routine consumption would not present any negative impact to health. For many Americans consuming caffeine in moderation, this is true. But some people fail to realize that caffeine does have an impact on the body, particularly on the cardiovascular system. This fact becomes more relevant when caffeine consumption is increased in efforts to thwart the effects of mild sleep deprivation, as a support mechanism to passive (psychological) stress, or to enhance motivation for training in the gym when mental fatigue is a barrier. The addition of caffeine to these stress-present conditions increases its cardiovascular effects.

Caffeine consumption raises both heart rate and blood pressure during resting states due to an elevation in myocardial nervous stimulation and increase in vascular resistance. When exercise is applied, the combination of physical stress and caffeine stimulation of the SNS increase cardiac output and vascular resistance to a greater degree than caffeine alone, equating to significant rise in blood pressure. This suggests limited concern for the normotensive person who drinks an energy drink before a workout, but for the hypertensive exerciser this can be significantly deleterious. Studies have revealed the stress-pressure effect is larger and more prolonged in hypertensive persons than in normotensive individuals. Such combined effects on blood pressure may potentially increase the risk of hypertension and possibly reduce the effectiveness of antihypertensive therapy.

Caffeine appears to affect blood pressure through adenosine receptor inhibition and an increased release of select neurotransmitters. Caffeine levels peak 30-120 minutes after oral consumption and caffeine has a half-life range of 3-6 hours depending on different factors, including dose and regularity of exposure. Routine consumption of caffeine causes a level of tolerance which diminishes the acute effects of caffeine on blood pressure, but again hypertensive persons are more susceptible to blood pressure changes. Reviews of caffeine's acute effect on blood pressure at rest suggest dose-relevant changes ranging between 3-15 mm Hg systolic and 4-13 mm Hg diastolic. Typically, the changes in blood pressure occur within 30 minutes and will peak in 1-2 hours. Effects on blood pressure from caffeine may persist for more than 4 hours.

Aerobic exercise is used as a primary means for managing blood pressure particularly in pre-hypertensive states and in stage I hypertension. Following exercise, systolic blood pressure will drop below initial resting measures. In a recent study, hypertensive males
performing aerobic exercise experienced reductions in systolic blood pressure by 20-30 mmHg, whereas normotensive males experience a decline of 8-12 mmHg. This effect is generally sustained for two hours in normotensive males and for durations beyond 12 hours in hypertensive males. Sustained hypotension after an acute dynamic exercise bout is due to inhibitory influence in response to parasympathetic tone adjustments from the vagus nerve and adenosine-mediated vasodilation. When aerobic exercise is applied with routine frequency, post exercise hypotension is a key contributor to disease risk attenuation. In fact, exercise has demonstrated a strong effect in research trials in lowering diastolic pressure compared to beta-blocker and diuretic therapies (exercise ≥ 8mmHg/ pharmacological treatment ≥ 13 mmHg). The ingestion of caffeine poses an antagonistic threat to the post exercise hypotensive response and may actually increase the risk of exercise due to its strong neural influence.

Normotensive (BP less than 135/85 mm Hg) men between the ages of 24 and 30 performed submaximal and symptom-limited maximal cycle ergometry 1 hour apart, after ingesting either placebo or caffeine (3.3 mg/kg). Subjects were monitored for changes in heart rate, BP, cardiac output, and peripheral vascular resistance on placebo and those values were compared to outcomes for each subject using treatment of caffeine. Post treatment baseline showed that caffeine increased systolic and diastolic BP and peripheral vascular resistance and decreased heart rate. BP and vascular resistance effects of caffeine remained during submaximal exercise resulting in an additive increase in BP. When measured at maximal exercise more subjects (15 on caffeine vs. 7 on placebo) had systolic BP > 230 mmHg and/or > 100 mm Hg for diastolic measures. Additionally, cortisol was increased post-drug treatment and throughout maximal exercise on caffeine treatment days. The study data indicated that caffeine increased BP additively during submaximal exercise and caused excessive BP responses at maximal exercise for some individuals. Since cardiac output had limited deviation between measures it is presumed the pressor effects of caffeine appear to be due to increasing vascular resistance rather than cardiac output.

The aforementioned trial identified the profound effect of caffeine on exercise BP during aerobic submaximal and maximal efforts. When caffeine was combined with intense anaerobic resistance training the outcomes were similar. Twenty-two resistance trained males performed repetition maximums using 60% of 1RM on the bench press and leg press, both on placebo and when treated with caffeine (6mg/kg). Following repetition failure, heart rate and blood pressure were immediately assessed, and mean arterial pressure and rate-pressure product were calculated. Results showed significant increases in heart rate (+ 10 beats/min), systolic blood pressure (+ 8-10 mmHg), and rate-pressure product with acute caffeine ingestion compared to placebo. There were no noted changes in diastolic blood pressure across time or treatment. The relevance of these findings suggests that exercise blood pressure is elevated above normal exercise measures when caffeine is ingested prior to training both anaerobically and aerobically.

When non-medicated, hypertensive men are compared to normotensive men during exercise, both with and without caffeine treatment, the response was consistent between groups, but the magnitude of the response was significantly higher for those with
hypertension. Test subjects performed 30 minutes of extended bicycle exercise following a single dose of caffeine (3.3 mg/kg) and placebo. Hemodynamics were measured before caffeine/placebo treatment, 40-min post-drug treatment and during exercise. Pretreatment baseline measures identified higher heart rates (>10 beats/min) and BP (SBP >30 and DBP >15mmHg) as expected in the hypertensive men. At postdrug baseline, caffeine increased systolic and diastolic BP, increases peripheral vascular resistance in all cases, and consistent with the aforementioned trials, decreased heart rate without significant change in stroke volume or cardiac output for both groups. During exercise, the heart rate response was greater during caffeine treatment than the placebo treatment in the hypertensive group only. Systolic BP was consistently elevated on the caffeine treatment compared to placebo in both groups. The hypertensive group experienced an elevation in diastolic BP during exercise on the caffeine-treatment day, but this pressor effect disappeared at 15 minutes of exercise in normotensive group. As a result of elevation in heart rate and vascular resistance in the hypertensive group, the rate-pressure products were significantly higher with caffeine treatment at postdrug measures and during exercise. On caffeine, 39% of the hypertensive men and only one of the normotensive men showed an excessive BP response of > 230 mmHg systolic or > 120 mmHg diastolic during exercise. These findings suggest increased cardiovascular strain, evidenced by greater rate-pressure product and diastolic BP measures, is associated with caffeine ingestion by hypertensive males during exercise.

Additional evidence suggests that the impact of caffeine ingestion by those with hypertension is even worse than presented in this article to this point. Despite the conventional focus on peripheral blood pressure measures, the most physiologically relevant pressures for both cardiac and vascular effects are central pressures. When central measures of blood pressure were compared to peripheral measures in normotensive males and females following coffee consumption (80 mg caffeine), it was identified that the caffeine increased central systolic pressure (SBP) 5 points mmHg and diastolic pressure (DBP) 4 points mmHg compared to decaffeinated coffee. Making this even more relevant is peripheral systolic blood pressure did not change significantly after the administration of either caffeinated or decaffeinated coffee. This suggests that peripheral blood pressures measured on caffeine do not represent the significant mean arterial pressures experienced in central vessels.

One study analyzing the vascular effects of caffeine during exertion found that during exercise the increase in aortic systolic pressure was 25% greater compared with peripheral blood pressure at 30 minutes and 21% greater at the 60 minute mark. Additionally, aortic pulse pressure was 34% greater at 30 minutes and 40% greater at 60 minutes of exercise. This study shows that caffeine affects central pressures more than is apparent from the corresponding upper limb values measured at the brachial artery.

Vascular stiffness and wave reflection variance are associated with caffeine intake. This response is consistent with that seen in aging where degeneration and hyperplasia of the arterial wall contributes to increased risk of vascular disease. Chronic changes in arterial wall properties cause an increase in myocardial demand and place additional stress on the heart which promotes fatigue and development of atherosclerosis. The changes in central
pressure are grossly underestimated when blood pressure is measured in the brachial artery following exercise on caffeine. Hypertensive persons that engage in exercise while under the influence of caffeine stimulation have been found to have dramatic increases in mean arterial pressure in central measures. Therefore the mixture of exercise and caffeine for hypertensive clients may create injurious effects on the vessel wall and increase risk for diseases including stroke, coronary heart disease, myocardial infarction, heart failure, end-stage renal disease, and cardiovascular mortality. Of additional concern is caffeine-causing vascular resistance is preserved post exercise, which attenuates the parasympathetic adjustments. Caffeine’s parasympathetic inhibition blocks the favorably reduce blood pressure response for an extended period of time limiting any positive effect the exercise would have on the vascular system. Individuals with hypertension that exercise while using caffeine increase activity-adjusted blood pressure above normal and in some cases reach measures greater than 250 mmHg. The caffeine prevents post exercise hypotension in exchange for pressures greater than those measured at rest. Although more research is necessary to identify if caffeine intake with exercise has any negative effects for the normotensive, it is evident that the hypertensive client should not ingest caffeine several hours before exercise and should prudently consider avoiding caffeine intake at all.