Caffeine ingestion alters blood pressure (BP), however, the interactive effect of caffeine and exercise on central BP is unknown. PURPOSE: Examine the acute influence of caffeine and moderate-intensity aerobic exercise on post-exercise central BP and arterial stiffness. METHODS: Ten males (ages 55±5; range 31-71 years) completed two exercise trials after ingestion of caffeine (400 mg) or placebo. Peripheral (brachial) and central (aortic) BP were assessed via pulse wave analysis (PWA) and arterial stiffness via pulse wave velocity (PWV) obtained before and 30 min post-ingestion. Participants performed 40-min of cycling at 70% of HRmax using identical workloads between trials. PWA and PWV were collected again 10 and 30 min post-exercise. Data were analyzed via two-way ANOVA with repeated measures. RESULTS: Prior to exercise, compared to placebo, caffeine increased (P<0.05) brachial systolic blood pressure (bSBP) (+12mmHg), brachial diastolic blood pressure (bDBP) (+8mmHg), central systolic blood pressure (cSBP) (+11mmHg) and central diastolic blood pressure (cDBP) (+7mmHg). PWV was higher (0.75 vs. 0.22m/s) 30 minutes post caffeine ingestion, independent of trial (P<0.05) while there was a trend for an interaction (P=0.074), suggesting an increase in PWV with caffeine. Post-exercise, bSBP (-4.8 vs. -6.1mmHg) and PWV (-0.40 vs. -0.74m/s) were higher in caffeine (P<0.05), likely due to the influence of caffeine prior to exercise. cSBP (-5 vs. -6mmHg) and cDBP (-3.5 vs. -1.8mmHg) were lower after exercise, independent of trial (P<0.05) while bSBP (-4.8 vs. -6.1mmHg) and cDBP (-3.1 vs. -1.5mmHg) trended (P=0.07) to be lower after exercise, independent of trial. PWV (-0.11 vs. -0.06m/s) remained higher (P<0.05) after exercise in caffeine compared to placebo but was not influenced by exercise. Accordingly, AP (2.7 vs. -1.1mmHg) and AIX (-5.5 vs. -1.2%) were lower (P<0.05) after exercise in placebo only. CONCLUSION: These findings suggest that the stimulatory effects of caffeine ingestion elevates central hemodynamics and arterial stiffness, which persists even after exercise, exerting a greater afterload on the heart.

Chronic kidney disease (CKD) is an independent risk factor for the development of cardiovascular disease, with both diseases characterized by reduced nitric oxide (NO) bioavailability and vascular dysfunction. Passive leg movement (PLM) has previously been shown to produce NO-mediated hyperemia in the lower extremity, however this technique has not yet been utilized to assess vascular function in patients with CKD. PURPOSE: To assess vascular function in patients with CKD using PLM, in addition to the traditional flow-mediated dilatation (FMD) technique. METHODS: Assessment of vascular function via PLM and FMD was performed on 12 patients (CKD 67±3 yrs) and 12 healthy controls (CON, 59±2 yrs). Hemodynamics and artery diameters during PLM and FMD were measured utilizing ultrasound Doppler of the femoral and brachial arteries, respectively. RESULTS: Patients with CKD had reduced peak leg blood flow (LBF) (CKD, 384±39 vs. CON, 626±93 mL/min, p<0.05) and a reduced change in LBF from baseline to peak (A, P<0.05) during PLM compared to CON. Additionally, rA in LBF was significantly correlated with kidney function as assessed by estimated glomerular filtration rate for all participants (r=0.53, p<0.05). As anticipated, FMD was also significantly attenuated in CKD patients compared to CON. CONCLUSION: Vascular function as assessed by PLM and FMD is attenuated in patients with CKD compared to controls, supporting a reduction in NO bioavailability in this chronic disease state. Additionally, PLM appears to be a novel and feasible approach to assess NO-mediated vascular function in CKD and is associated with kidney function.