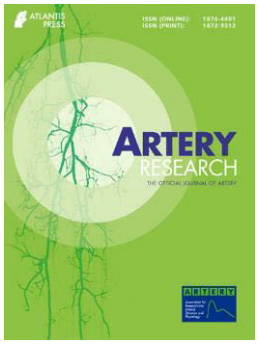

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P87: CEREBROVASCULAR REACTIVITY DURING COGNITIVE ACTIVATION IN ADULTS WITH CONTROLLED HYPERTENSION

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(Aix) with ABI in individuals with and without peripheral arterial disease (PAD). The study group included 670 subjects mean age 57 ± 16 years (248 PAD (ABI ≤ 0.9) and 422 No-PAD (ABI ≥ 0,91 −1.3). The aPWV, and Aix was estimated using applanation tonometry. 2. The ankle systolic pressure measurements for the calculation of the ABI was obtained using an 8-mHz Doppler probe. After stepwise selection process, in PAD patients aPWV and Aix were not related to ABI.

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Poster Session II — Brain

P85
CEREBRAL SMALL VESSEL DISEASE AND RISK OF INCIDENT STROKE, DEMENTIA AND DEPRESSION, AND ALL-CAUSE MORTALITY: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Background: MRI features of cerebral small vessel disease, i.e. white matter hyperintensities, lacunes, microbleeds, perivascular spaces, and cerebral atrophy, may be associated with clinical events, but the strength of these associations remains unclear.
Methods: We conducted a systematic review and meta-analysis on the association between these features and incident ischaemic and haemorrhagic stroke, all-cause dementia and depression, and all-cause mortality.
Results: For the association with stroke, 36 studies were identified (number of individuals/events [n] = 38,432/4,136), for dementia 28 (n = 16,458/1,709), for depression nine (n = 9,538/1,746), and for mortality 28 (n = 23,031/2,558). Only two studies evaluated perivascular spaces; these results were not pooled. Pooled analyses showed that all other features were associated with all outcomes (hazard ratios ranged 1.22—2.72). Combinations of two features were more strongly associated with stroke than any individual feature.
Conclusions: Individual features and combinations of CSVD features are strongly associated with incident ischaemic and haemorrhagic stroke, all-cause dementia and depression, and all-cause mortality. If these associations are causal, the strength of these associations suggests that a substantial burden of disease is attributable to CSVD.

P86
BLUNTED CEREBRAL MICROCIRCULATION OXYGENATION DURING EXERCISE IN NEWLY DIAGNOSED HYPERTENSIVE PATIENTS: LINKS WITH INDICES OF MACROCIRCULATION AND ARTERIAL STIFFNESS

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Purpose/Background/Objective: Hypertension has been considered as one of the most common modifiable risk factors for stroke and cognitive impairment. Decreased cerebral perfusion and oxygenation, as a result of capillary rarefaction and microvascular impairment of brain vessels, have been suggested as potentials underlying mechanisms. However, there is no study investigating whether these parameters are present in newly-diagnosed hypertensive patients (HP), without any co-morbidities. Thus, we examined (i) whether functional activation of the human cerebral cortex during handgrip exercise is altered in newly diagnosed HP and (ii) whether cerebral oxygenation correlates with different markers of arterial stiffness.
Methods: Forty-five newly diagnosed HP and 36 normotensives underwent an exercise protocol, consisting of a 3-min-rest, a 3-min-handgrip exercise (30% MVC), and a 3-min-recovery. Continuous-near-infrared-spectroscopy (NIRS) was used to monitor changes in cerebral-[O2Hb]. IMT, Augmentation Index, Central-BP and PWV (Sphygmocor) were assessed.
Results: No significant differences were detected between groups in age, BMI, sex, MVC and force maintained during handgrip. During handgrip, cerebral[O2Hb] increased in both groups; however, hypertensive patientsexhibited a significantly lower average response than normotensives [1.6(1.1—2.7) vs. 2.4(1.4—3.2)μM], respectively, $p < 0.05$ and a lower peak [O2Hb] [4.2(3.3—6.2) vs. 5.9(4.3—9.2), $p < 0.01$]. Significant negative correlations were found between cerebral-[O2Hb] and aortic BP, AI, and PWV.
Conclusions: Hypertensive patients exhibited a blunted cerebral [O2Hb] response during handgrip exercise compared to their normotensive counterparts. This blunted increase in cerebral oxygenation during exercise was present in patients with recent diagnosis of hypertension and without evident TOD and correlated with macrovascular stiffening, indicating a cross-talk between micro- and microcirculation.

P87
CEREBROVASCULAR REACTIVITY DURING COGNITIVE ACTIVATION IN ADULTS WITH CONTROLLED HYPERTENSION

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Hypertension, even when pharmaceutically controlled, may accelerate arterial stiffening and impair changes in blood flow necessary to support neural activity (neurovascular coupling [NVC]). Optimal NVC requires continuous, non-pulsatile flow, which is partially determined by extra- and intra-cranial vessel function.
Purpose: Examine extra- and intra-cranial hemodynamics during cognitive activity in adults with well-controlled hypertension and without hypertension.
Methods: 30 middle-aged, medicated hypertensive and 30 age-, sex-, and Body Mass Index (BMI)-matched non-hypertensive adults (56 ± 6 yrs, BMI 28.2 ± 2.9 kg/m^2; 32 men) underwent cerebrovascular measures at rest and during a Stroop task. Applanation tonometry and ultrasound were used to assess aortic and carotid (single-point) Pulse Wave Velocity (PWV), respectively. Ultrasound and Doppler were used to measure carotid and Middle Cerebral Artery (MCA) blood velocity pulsatility. Near-infrared spectroscopy was used to measure prefrontal oxygenation (tissue saturation index; TSI). Accuracy and reaction times were computed to assess cognitive performance.
Results: Stroop performance was similar between groups ($p > 0.01$). Aortic and carotid PWV increased, carotid pulsatility decreased (p0.01; Table 1). Reductions in CCA pulsatility during the Stroop were associated with increases in cortical TSI in the combined sample ($r = 0.27$), suggesting extracranial hemodynamics may play a role in optimizing intracranial NVC.
Conclusions: Our findings indicate that middle-age adults with medically-controlled hypertension display similar intra- and extra-cranial cerebrovascular reactivity to adults without hypertension. Additionally, adults with and without hypertension may utilize reductions in extracranial pulsatility during NVC to minimize intracranial pulsatility and improve downstream cerebral oxygenation.