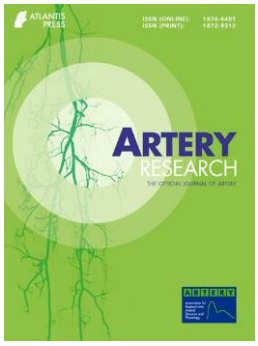

**Artery Research**

**ISSN (Online): 1876-4401** **ISSN (Print): 1872-9312**
**Journal Home Page: https://www.atlantis-press.com/journals/artres**

# P7.8: THE RELATIONSHIP BETWEEN RENAL AND CEREBRAL BLOOD FLOW PULSATILITY

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**To cite this article**: Laura Watkeys*, James Pearson, Barry McDonnell (2015) P7.8: THE RELATIONSHIP BETWEEN RENAL AND CEREBRAL BLOOD FLOW PULSATILITY, Artery Research 12:C, 31–31, DOI: https://doi.org/10.1016/j.artres.2015.10.311

**To link to this article**: https://doi.org/10.1016/j.artres.2015.10.311

Published online: 7 December 2019

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**Objective**: Morning blood pressure (BP) surge is considered to be an independent risk factor for cardiovascular diseases. On the other hand, there is increasing evidence that central systolic pressure (CSP) is stronger correlated with target organ damage and cardiovascular events than peripheral systolic pressure. Therefore, the aim of study was to evaluate the difference in morning central BP surge between men and women.
**Methods**: Fifty patients with never treated hypertension (age 40.4±11.5 years, 35 men) and 50 normotensive subjects (age 38.3±12.0 years, 35 men) were included into the study. Applanation tonometry of the radial artery and "n-point forward moving average" method have been used to derive 24-h CSP (BPro, HealtStats). The sleep-through morning surge (MS) was the difference between the morning pressure and the lowest nighttime BP. The preawakening MS was the difference between the morning BP and the preawakening BP (the average BP during the 2 hours before awakening).
**Results**: The 24-hour CSP was 129.5±10.6 mmHg in hypertensives and 110.5±12.4 mmHg in normotensives ($p<0.05$). The average daytime and nighttime CSP was 133.8±11.1 mmHg and 123.1±11.1 mmHg ($p<0.05$) in hypertensives whereas 114.3±13.7 mmHg and 104.8±11.7 ($p<0.05$) in normotensives, respectively. The values of morning BP surge are presented in the table.
**Conclusion**: Central sleep-through MS and preawakening MS are similar in men and women.

| | Men (n=35) | Women (n=15) | p |
|---|---|---|---|
| HYPERTENSIVES | | | |
| Central sleep-through MS (mmHg) | 19.38±7.59 | 16.74±5.99 | 0.24 |
| Peripheral sleep-through MS (mmHg) | 13.21±7.76 | 10.02±4.58 | 0.15 |
| NORMOTENSIVES | | | |
| Central sleep-through MS (mmHg) | 16.21±7.90 | 20.56±20.30 | 0.28 |
| Peripheral sleep-through MS (mmHg) | 9.76±7.0 | 17.37±21.4 | 0.07 |

## P7.5
## RELATIONSHIPS BETWEEN 24 HOUR URINARY CORTISOL METABOLITES AND STRUCTURAL CARDIAC AND ARTERIAL INDICES IN PEOPLE WITH OR AT RISK OF TYPE 2 DIABETES

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**Objective**: To assess if 24h urinary cortisol metabolite (UCM) profiles are related to structural cardiac and arterial parameters in those with or at risk of Type 2 diabetes mellitus (T2DM).
**Design and method**: 32 participants, 25-77 years, eGFR> 45 mL/min and no serious illness. Urine was collected over 24 hours. 2D echocardiography and arterial stiffness measures [aortic pulse wave velocity (aPWV) by Arteriograph and cardio-ankle vascular index (CAVI) by VaSera] were performed on the collection day. Steroids were extracted from urine and hydrolysed; derivatives were analysed by GC-MS.
**Results**: Seven UCMs were quantified [tetrahydrocortisol (THF), *allo*-tetrahydrocortisol (*a*-THF), tetrahydrocortisone (THE), α-cortol (α-col), β-cortol (β-col), α-cortolone (α-cone), β-cortolone (β-cone)].
Left ventricular mass index (LVMI) correlated positively with 24h cortisol:-cortisone metabolites (THF+*a*THF+αcol+βcol/THE+αcortolone+βcortolone) and negatively with α:β metabolites (αcol+αcortol/βcol+βcortol), when indexed for body surface area (BSA) and height ($r=0.37$, 0.48 and $r=-0.34$, -0.49 respectively). Further, there was a positive relationship between $LVMI_{BSA}$ and THF:THE ($r=0.35$). aPWV but not CAVI was also related to 24h cortisol:cortisone metabolites ($r=0.45$). All $p<0.05$.
Regression analysis including age, gender, systolic blood pressure (SBP), arterial stiffness (aPWV or CAVI) and body mass index (BMI; only for RWT), showed an independent association between THF:THE and $LVMI_{BSA}$ and $LVMI_{height}$ and cortisol:cortisone metabolites with $LVMI_{height}$, $p<0.02$. SBP, but not arterial stiffness, was also independently related to $LVMI_{BSA}$ and $LVMI_{height}$ in all models.
**Conclusion**: Specific 24h UCMs and UCM ratios (from glucocorticoid/mineralocorticoid, 11βHSD and 20αHSD/20βHSD actions) may be structural cardiac biomarkers in those with or at risk of T2DM.

## P7.7
## TESTOSTERONE LEVELS IN HYPERTENSIVE PATIENTS WITH VASCULAR ORGAN DAMAGE

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**Purpose**: Testosterone levels are decreased in hypertensive patients compared to normotensive subjects with similar age. Measurements of carotid IMT or aortic stiffness are reasonable for detecting vascular organ damage (OD) in patients with arterial hypertension. We investigated whether low testosterone concentration is associated with vascular OD in hypertensive patients.
**Methods**: 178 consecutive asymptomatic hypertensive males (40-60 y/o) were evaluated using exercise treadmill test and stress echocardiography. Men with positive one or both of the two tests were referred for coronary angiography in order to document coronary artery disease (CAD). All patients underwent carotid-femoral pulse wave velocity (PWV) and carotid IMT evaluation. Vascular OD was detected when IMT>0.9 mm (or plaque) and/or PWV>10 m/s. Total testosterone (TT) levels were measured in all participants. Testosterone deficiency (TD) was defined when TT levels were below 3.4 ng/ml.
**Results**: Coronary angiography revealed significant stenosis in 31 (17%) patients. The prevalence of Grade II/III hypertension was not different between CAD patients and subjects without CAD. Subjects without CAD were further divided according to presence/absence of vascular OD. Patients with vascular OD had lower TT level ($p<0.001$) and a greater prevalence of TD ($p<0.01$) compared to hypertensive subjects without OD after adjustment for age and blood pressure. Interestingly, CAD patients and non CAD subjects with vascular OD had comparable TT concentration and prevalence of TD.
**Conclusion**: TT concentration is decreased in hypertensive patients with vascular OD compared to subjects without OD. These findings underscore the predictive value of TD in hypertensive males with OD.

## P7.8
## THE RELATIONSHIP BETWEEN RENAL AND CEREBRAL BLOOD FLOW PULSATILITY

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**Introduction**: Increased pulsatile flow has been implicated in the progression of microvascular damage. The pulsatile blood flow transmitted into both the brain and kidney are often assumed to be similar, however specific studies confirming these associations are lacking. Therefore, the aim of this study was to investigate the relationship of pulsatility index (PI) and resistive index (RI) [both measures of pulsatile flow] between the renal artery (RA) and middle cerebral artery (MCA).
**Methods**: Thirty-one participants (aged 36±11 years) were recruited from Cardiff Metropolitan University's Staff Health and Wellbeing study. Peak systolic velocity, end diastolic velocity and mean velocity were all measured and used to calculate PI (Gosling's) and RI (Pourcelot's) of the RA (renal ultrasound, Vivid Q, GE) and MCA (Transcranial Doppler, DWL). Correlation and level of agreement (LOA) between RA PI and MCA PI were analysed using Pearson correlation coefficient and Bland-Altman plots.
**Results**: Although, a linear relationship was observed between RA PI and MCA PI ($r=0.477$, adjusted $R^2=0.201$, $P=0.007$) and between RA RI and MCA RI ($r=0.513$, adjusted $R^2=0.238$, $P=0.003$), further Bland-Altman analysis demonstrated a PI bias of 0.37±0.26 and LOA of -0.15 to 0.89.
**Conclusion**: These data suggest that the agreement between flow pulsatility within the renal and middle cerebral arteries are not similar. Our data indicate that the measurement of PI in the renal artery only explains 20% of the variance in PI observed in the MCA. Therefore, caution should be taken when assuming similarity in PI and RI between different organs.