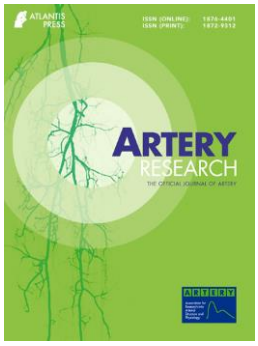

Artery Research

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

P4.14: RENAL DENERVATION IMPROVES CENTRAL HEMODYNAMICS AND PULSE PRESSURE AMPLIFICATION IN PATIENTS WITH TREATMENT RESISTANT HYPERTENSION

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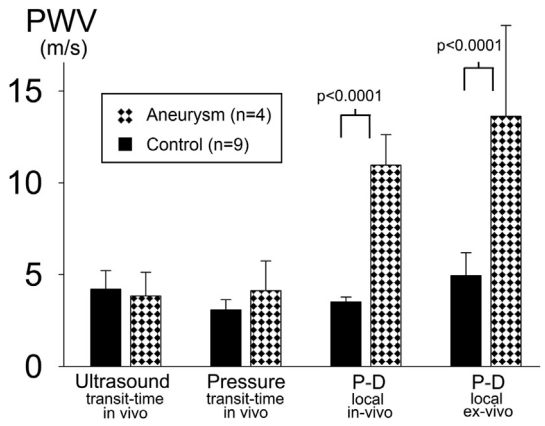
To cite this article: C. Ott, A. Schmid, T. Ditting, R. Veelken, M. Uder, R.E. Schmieder (2013) P4.14: RENAL DENERVATION IMPROVES CENTRAL HEMODYNAMICS AND PULSE PRESSURE AMPLIFICATION IN PATIENTS WITH TREATMENT RESISTANT HYPERTENSION, Artery Research 7:3_4, 139–139, DOI: https://doi.org/10.1016/j.artres.2013.10.132

To link to this article: https://doi.org/10.1016/j.artres.2013.10.132

Published online: 14 December 2019



Conclusions: PWV measurement in mice is not straightforward and results should be interpreted carefully.

P4.13
VITAMIN D SUPPLEMENTATION IMPROVES ENDOTHELIAL FUNCTION IN TYPE 2 DIABETES — A RANDOMIZED CONTROLLED TRIAL

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Background: Cardiovascular disease is prevalent in type 2 diabetes, and both endothelial dysfunction and arterial stiffness may contribute in the pathogenesis. Low levels of vitamin D are associated with both type 2 diabetes and cardiovascular disease.
Aim: To evaluate the effect of vitamin D supplementation on endothelial function and arterial stiffness in subjects with type 2 diabetes and hypovitaminosis D.
Methods: Sixty-two subjects with type 2 diabetes and serum 25-hydroxy-vitamin D [25(OH)D] <50 nmol/L were included in this randomized controlled trial (NCT 00992797). Thirty-three patients received vitamin D (400 000 IU cholecalciferol) at baseline, whereas 29 patients received placebo. Endothelial function and arterial stiffness were measured at baseline and after six months. Endothelial function was assessed as the reactive hyperaemia index (RHI) using endothelial pulse amplitude testing (Endo-PAT). Arterial stiffness was estimated as carotid-femoral pulse wave velocity (cfPWV) and augmentation index (AIx) with the SphygmoCor device. Serum 25(OH)D was measured using the DiaSorin-RIA.
Results: Mean (SD) age in the treatment and placebo group were 57.5 (9.4) and 57.8 (10.0) years, 51.5 % (n=17) and 44.8 (n=13) were females, and diabetes duration was 11.4 (6.5) and 7.5 (5.7) years. Vitamin D supplementation significantly improved RHI and increased the 25(OH)D levels, but did not change cfPWV and AIx (Table 1). In multivariable regression analysis, change in RHI was significantly associated with change in 25(OH)D levels (β [CI] = 0.009 [0.001-0.017], P=0.03).
Conclusion: Vitamin D supplementation improved endothelial function but not arterial stiffness in subjects with type 2 diabetes.

P4.14
RENAL DENERVATION IMPROVES CENTRAL HEMODYNAMICS AND PULSE PRESSURE AMPLIFICATION IN PATIENTS WITH TREATMENT RESISTANT HYPERTENSION

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Background: Renal denervation (RDN) was shown to be effective in reducing peripheral BP in treatment resistant hypertension. Accumulating data suggest that central pressures may be a better predictor of cardiovascular events and outcomes than the corresponding peripheral pressure. PP amplification is among others inversely related to stiffer arteries and peripheral arterial resistance.
Methods: 57 patients with treatment resistant hypertension (office BP ≥140/90 mmHg, while on at least 3 antihypertensive agents, and diagnosis confirmed by 24-h ABPM ≥130/80 mmHg) underwent catheter-based RDN using the Symplicity Flex™ catheter (Medtronic Inc., Palo Alto, CA). In addition, in our lab pulse wave analysis was assessed with the SphygmoCor™ device (AtCor Medical, Australia) before and after 6 months of RDN. PP amplification is determined as ratio of peripheral PP to central PP.
Results: Patients (59±12 years) were treated with 6.0±1.3 antihypertensive drugs on average. Peripheral as well as central systolic and diastolic BP were reduced (all p<0.01) 6 months after RDN. In accordance, peripheral PP (77.5±22 versus 71.5±23 mmHg, p=0.008) and central PP (63.2±21 versus 56.7±22 mmHg, p=0.001) were reduced 6 months after RDN. Consistently, there was a significant improvement in PP amplification (1.25±0.2 versus 1.30±0.2, p=0.012). Also central augmentation pressure (20±12 versus 16±13 mmHg, p<0.001) and cAIx@75 (24±10 versus 21±11 %, p=0.005) decreased 6 months after RDN. There was no change on heart rate (63±11 versus 64±10 bpm, p=0.499).
Conclusion: Our data suggest that RDN might exert beneficial effects indicated by an improvement of central PP beyond peripheral PP, and hence PP amplification.

P4.15
EFFECT OF RENIN ANGIOTENSIN SYSTEM BLOCKADE ON SOLUBLE KLOTHO, ARTERIAL STIFFNESS AND ALBUMINURIA IN PATIENTS WITH TYPE 2 DIABETES AND SYSTOLIC HYPERTENSION

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Background: Soluble Klotho is an anti-ageing phosphaturic protein associated with cardiovascular and renal protection. In-vitro and in-vivo studies have demonstrated that rennin-angiotensin-system (RAS) blockade increases soluble Klotho levels. The effect of RAS blockers on soluble Klotho in patients with diabetic-kidney-disease (DKD) is unknown.
Methods and measurements: Plasma soluble Klotho was measured in a secondary analysis of a randomised controlled clinical trial performed at a single university centre. Seventy-six patients with Type-2 diabetes, and DKD (all with albuminuria and serum creatinine <1.7mg/dl) were studied at baseline and at 24-weeks (end of study), following randomisation to valsartan/hydrochlorothiazide (n=37) or amlodipine (n=39) treatment. Aortic-pulse wave velocity (Ao-PWV) by applanation tonometry and albuminuria (from 3-timed urine collections) were also measured at baseline and 24-weeks.
Results: Valsartan/hydrochlorothiazide treatment significantly increased soluble Klotho mean±standard deviation, from 432.7±179 to 506.4±226.8

Table 1 Baseline values and change in endothelial function, arterial stiffness and vitamin D from baseline to 6 months. Values are given as mean (SD). *P* represents the significance of between-group-comparisons for baseline values and changes after 6 months respectively.

	Baseline			Change		
	Treatment (*n=33*)	Control (*n=29*)	*P*	Treatment (*n=33*)	Control (*n=29*)	*P*
RHI	1.7 (0.4)	1.7 (0.5)	0.74	0.21 (0.49)	-0.03 (0.37)	0.04
cfPWV, m/s	10.18 (1.85)	9.84 (2.33)	0.54	-0.21 (0.92)	-0.10 (0.64)	0.56
AIx, %	20.0 (8.7)	20.0 (10.1)	0.98	0.8 (5.0)	3.5 (8.4)	0.54
25(OH)D, nmol/L	38.5 (9.1)	38.1 (8.5)	0.74	15.0 (11.0)	0.7 (16.0)	<0.001