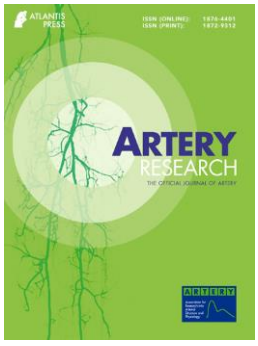

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2.5: DOES WAVE REFLECTION PROTECT THE MICROVASCULATURE FROM HIGH PULSE PRESSURE?

Avinash Kondiboyina, Joe Smolich, Michael Cheung, Berend Westerhof, Nico Westerhof, Jonathan Mynard

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SD greater dBPV was associated with lower IPS (beta [SD difference]; 95% CI: -0.10;-0.20 to -0.00) and EF (-0.12; -0.22 to -0.01), and borderline associated with lower MF (-0.09; -0.20 to 0.01). A 1-SD greater sBPV, however, was not associated with IPS (-0.040; -0.14 to 0.06), or EF (-0.09; -0.20 to 0.022), but was borderline associated with lower MF (-0.11; -0.21 to 0.00). This effect of greater dBPV on cognitive performance is equivalent to ±3 additional years of ageing. The stronger association of dBPV than sBPV with cognitive performance may be explained by the fact that DBP is the main determinant of MAP. Excessive dBPV may then lead to inadequate cerebral perfusion. In conclusion, greater very short-to mid-term dBPV and, to a lesser extent, sBPV could be a modifiable risk factor for cognitive impairment.

2.3

OCCUPATIONAL, SPORT AND LEISURE RELATED PHYSICAL ACTIVITY HAVE CONTRASTING EFFECTS ON NEURAL BAROREFLEX SENSITIVITY. THE PARIS PROSPECTIVE STUDY III

Rachel Climie [1], Pierre Boutouyrie [1], Marie-Cecile Perier [1], Edouard Chaussade [2], Matthieu Plichart [2], Lucile Offredo [1], Catherine Guilbout [1], Thomas van Sloten [1], Frederique Thomas [3], Bruno Pannier [3], James Sharman [4], Stephane Laurent [5], Xavier Jouven [6], Jean-Philippe Empana [7]
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Background: Physical activity (PA) is beneficial for baroreflex sensitivity (BRS), but it is unclear whether the type of PA has similar effects on the neural (nBRS) or vascular (carotid stiffness) components of BRS. We sought to determine this in healthy adults from a community- based study via assessment of occupational (OPA), sport (SPA), leisure (LPA) and total PA (TPA).
Methods: In 8649 adults aged 50 to 75 years, resting nBRS (estimated by low frequency gain, from carotid distension rate and heart rate) and carotid stiffness were measured by high-precision carotid echotracking. PA was self-reported using the Baecke questionnaire, which distinguishes OPA, SPA, LPA and TPA. The associations between PA and nBRS and carotid stiffness were quantified using multivariate linear regression analysis. Analyses were conducted separately in the working and non- working population.
Results: In working adults (n = 5039), OPA was associated with lower nBRS function (p = 0.026) and borderline higher carotid stiffness (p = 0.08). When stratified by education, this association remained only in those with less than tertiary education. SPA was associated with higher nBRS (p = 0.0005) and borderline lower carotid stiffness (p = 0.052). Neither LPA nor TPA was associated with nBRS or carotid stiffness. In non-working adults (n = 3610), SPA and TPA were both associated with lower carotid stiffness (p = 0.012 and p = 0.020), but not nBRS. LPA was not associated with either parameter.
Conclusion: Occupation-related PA is associated with lower nBRS function and higher carotid stiffness, especially in those with lower education. Higher amounts of sport-related PA are associated with higher nBRS and lower carotid stiffness.

2.4

CENTRAL SYSTOLIC BLOOD PRESSURE PROVIDES ADDITIONAL INFORMATION IN RISK PREDICTION IN HEMODIALYSIS PATIENTS

Christopher C. Mayer [1], Julia Matschkal [2], Pantelis A. Sarafidis [3], Stefan Hagmair [1], Georg Lorenz [2], Susanne Angermann [2], Matthias C. Braunisch [2], Marcus Baumann [2], Uwe Heemann [2], Christoph Schmaderer [2], Siegfried Wassertheurer [1]
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Background: Association of Ambulatory Blood Pressure Monitoring (ABPM) with mortality depends on cardiac function in hemodialysis patients. Evidence for the predictive power of central Systolic Pressure (cSBP) is inconclusive. Thus, this study aimed to investigate the additional information of ambulatory cSBP in risk prediction in a cohort of hemodialysis patients.
Methods: Within the ISAR-study cohort, 344 hemodialysis patients underwent 24 h ABPM on the dialysis day. All-cause and cardiovascular mortality served as endpoints. Risk prediction was performed using Cox regression in patients with or without atrial fibrillation (AF) or heart failure (HF) for peripheral (pSBP) and central systolic pressure calibrated with peripheral systolic and diastolic pressure (cSBP1) or peripheral mean and diastolic pressure (cSBP2).
Results: During a mean follow-up of 37.6 (17.5 SD) months, 115 patients died, of whom 47 due to cardiovascular reasons. In patients with AF or HF, a negative association to mortality could be observed, independent of pressure location and calibration (see Table). In patients without AF or HF, these associations were to the opposite directions and cSBP2 was superior to pSBP and cSBP1 for all-cause (pSBP: HR = 1.01, p = 0.30; cSBP1: HR = 1.00, p = 0.77; cSBP2: HR = 1.01, p = 0.06) and cardiovascular (pSBP: HR =1.03, p = 0.02; cSBP1: HR = 1.02, p = 0.06; cSBP2: HR = 1.03, p = 0.003) mortality. This circumstance was confirmed in multivariable analysis combining pSBP and differences between pSBP and cSBP (see Table).
Conclusions: This study provides evidence for the additional information of central systolic blood pressure and its dependency on calibration in risk prediction in hemodialysis patients. Further studies are needed to confirm these findings.

		AForHF (n = 105)		noAForHF (n = 239)	
		HR	p	HR	p
All-cause Mortality		*59 events*		*56 events*	
Univariate	pSBP	0.97 (0.96, 0.98)	<0.001	1.01 (0.99, 1.03)	0.30
	cSBP1	0.97 (0.95, 0.98)	<0.001	1.00 (0.98, 1.02)	0.77
	cSBP2	0.97 (0.96, 0.99)	<0.001	1.01 (1.00, 1.03)	0.06
	pSBP-cSBP1	0.93 (0.85, 1.01)	0.09	1.10 (1.04, 1.17)	<0.001
	pSBP-cSBP2	1.01 (0.97, 1.05)	0.59	0.95 (0.91, 0.98)	0.005
Multivariable	pSBP	0.97 (0.95, 0.99)	<0.001	1.00 (0.98, 1.01)	0.60
	pSBP-cSBP1	1.01 (0.92, 1.12)	0.80	1.11 (1.04, 1.19)	0.002
	pSBP	0.97 (0.96, 0.98)	<0.001	1.00 (0.99, 1.02)	0.79
	pSBP-cSBP2	1.00 (0.96, 1.04)	0.83	0.95 (0.91, 0.99)	0.009
Cardiovascular Mortality		*20 events*		*27 events*	
Univariate	pSBP	0.95 (0.93, 0.98)	<0.001	1.03 (1.00, 1.05)	0.02
	cSBP1	0.95 (0.93, 0.97)	<0.001	1.02 (1.00, 1.05)	0.06
	cSBP2	0.96 (0.94, 0.98)	<0.001	1.03 (1.01, 1.05)	0.003
	pSBP-cSBP1	0.86 (0.74, 1.02)	0.08	1.12 (1.03, 1.20)	0.006
	pSBP-cSBP2	1.01 (0.94, 1.07)	0.87	0.93 (0.88, 0.98)	0.006
Multivariable	pSBP	0.95 (0.93, 0.98)	<0.001	1.02 (0.99, 1.04)	0.22
	pSBP-cSBP1	0.98 (0.81, 1.19)	0.87	1.08 (0.98, 1.18)	0.12
	pSBP	0.95 (0.93, 0.98)	<0.001	1.02 (1.00, 1.04)	0.12
	pSBP-cSBP2	0.98 (0.91, 1.05)	0.59	0.94 (0.89, 1.00)	0.04

Table: Univariate and multivariable hazard ratios (95% confidence intervals) per mmHg increase and significance levels (p) for all-cause and cardiovascular mortality. Abbreviations: pSBP, peripheral systolic pressure; cSBP, central systolic pressure (1=brachial systolic and diastolic pressure calibration; 2= brachial mean and diastolic pressure calibration); HR, hazard ratio; AF, atrial fibrillation; HF, heart failure.

2.5

DOES WAVE REFLECTION PROTECT THE MICROVASCULATURE FROM HIGH PULSE PRESSURE?

Avinash Kondiboyina [1,2], Joe Smolich [3,2], Michael Cheung [3,2,4], Berend Westerhof [5], Nico Westerhof [5], Jonathan Mynard [1,2,6]
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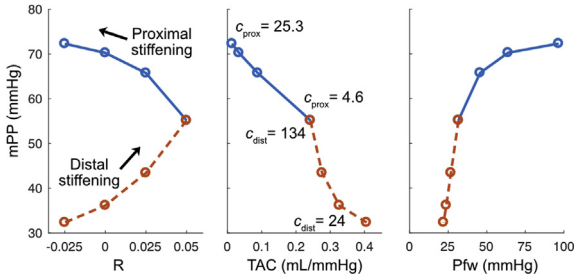
Background: Wave reflection (caused by a stiffness increase from large to small arteries) has been considered to protect against high microvasculature Pulse Pressures (mPP) (1). However, according to transmission line theory, Transmission (T) and Reflection (R) coefficients are proportional (T = 1+R), implying that reflection would not be protective. Proximal arterial stiffening with aging is associated with reduced Total Arterial Compliance (TAC) and increased forward Pressure (Pfw). We hypothesized that a high TAC and low Pfw, rather than high R, are responsible for protection from mPP.
Methods: We constructed a fractal arterial tree containing 5008 vessels across 14 generations (fractal exponent 2.76, asymmetry ratio 0.8). Wave speed in each vessel was prescribed to achieve a uniform reflection coefficient (R = -0.025, 0, 0.025 or 0.05) at every junction, achieved by progressively stiffening distal vessels while keeping aortic wave speed constant ("distal-stiffening") or by progressively stiffening proximal vessels while

keeping average wave speed in all terminal vessels constant ("proximal-stiffening", see Figure). An elastance heart model was applied at the inlet and simulations were performed with a one-dimensional flow solver (2).
Results: Proximal-stiffening and distal-stiffening had opposing effects on R but the same effects on mPP, whereas mPP increased monotonically with decreasing TAC and increasing Pfw in both settings (Figure).
Conclusion: Wave reflection per se does not provide protection from high mPP since greater reflection also entails greater transmitted pressure. Although a decreased R may accompany proximal arterial stiffening, the likely mechanism of increased mPP with aging is decreased TAC and greater Pfw.

Figure. Reflection coefficient, R (at every junction), TAC and Pfw vs mPP with proximal and distal stiffening. Arrows indicate effect of stiffening. c_{prox} and c_{dist} are wave speeds (m/s) in the proximal inlet segment and distal terminals (mean).



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2.6

FEASIBILITY OF AORTIC WAVE INTENSITY ANALYSIS FROM SEQUENTIALLY ACQUIRED CARDIAC MRI AND NON-INVASIVE CENTRAL BLOOD PRESSURE

Anish Bhuva [1,2], Niro Nadarajan [3], Andrew D'Silva [4], Camilla Torlasco [5], Redha Boubertakh [2], Siana Jones [3], Paul Scully [3,2], Rachel Bastiaenen [4], Guy Lloyd [2], Sanjay Sharma [4], James Moon [3,2], Kim Parker [6], Charlotte Manisty [3,2], Alun Hughes [3,7]
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Background: Wave intensity analysis (WIA) in the aorta offers important clinical and mechanistic insights but is difficult non-invasively. We performed WIA by combining high temporal resolution cardiovascular magnetic resonance (CMR) flow velocity and non-invasive central blood pressure (BP) waveform data.
Method: 206 healthy volunteers (36 ± 11 years, 47% male) underwent sequential phase contrast CMR (Siemens Aera 1.5T, 1.97 x 1.77 mm^2, ~9 ms temporal resolution) and supra-systolic oscillometric central BP (Uscom Ltd BP+) measurement. Velocity (U) and central pressure (P) waveforms (200 Hz) were aligned using the wave foot, and local wave speed was calculated both from the P-U slope during early systole (c) and the sum of squares method (cSS) (Figure 1), and compared with CMR aortic arch pulse wave velocity (PWV) by transit time.
Results: The peak intensity of the initial compression wave (dI+1), backward compression wave (dI-) and protodiastolic decompression wave (dI + 2) were 69.5 ± 28, -6.6 ± 4.2 and 6.2 ± 2.5 W/m^2 respectively. PWV correlated with c or cSS (r = 0.60, and 0.68 respectively; bias -1.3 [limits of agreement: -3.8 to 1.2 m/s], and bias -0.64 [limits of agreement: -3.0 to 1.7 m/s] respectively), Figure 1.
Conclusion: Wave intensity patterns and values are similar to those measured using invasive methods. Local wave speed showed good agreement with PWV. CMR and central blood pressure provides a novel non-invasive technique for performing wave intensity analysis and is feasible for large scale studies.

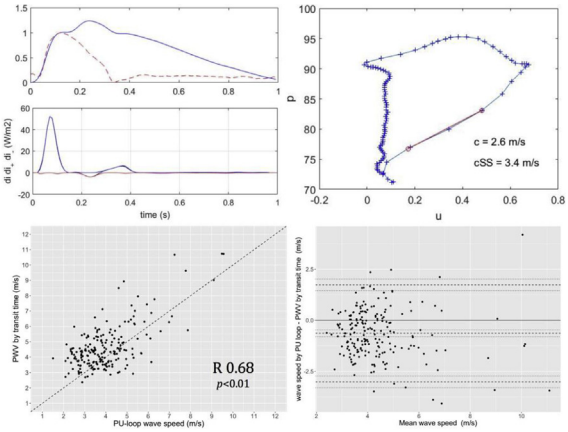

Figure 1. Calculation of wave speed from a pressure – velocity (P-U) loop and comparison with pulse wave velocity by transit time. Top left: alignment of scaled pressure (blue) and velocity (red) waveforms and example of wave intensity analysis showing initial compression (dI+1), backward compression (dI-) and protodiastolic decompression (dI+2) waves. Top right: P-U loop showing wave speed measurement in early systole (*c*); and using sum of squares (*c*SS). Bottom left and Bottom right: Correlation and Bland- Altman analysis of *c*SS and PWV from phase-contrast MRI showing good correlation and slight underestimation.

2.7

FITNESS MODIFIES THE ASSOCIATION BETWEEN EXERCISE BLOOD PRESSURE AND LEFT-VENTRICULAR MASS IN ADOLESCENCE

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Objective: Exaggerated exercise blood pressure (BP) is associated with higher left-ventricular mass index (LVMI). Paradoxically, exercise BP and LVMI may be higher with greater fitness, but underlying factors are poorly understood. This study aimed to determine the influence of fitness on exercise BP and its relationship with LVMI in adolescents.
Methods: 4835 adolescents from the Avon Longitudinal Study of Parents and Children, aged 15.4(0.3) years, 49% male completed a submaximal cycle test. Exercise BP was measured immediately on test cessation and fitness calculated as physical work capacity 170 adjusted for lean body-mass. LVMI (n = 1589), cardiac output (CO, n = 1628) and total peripheral resistance (TPR, n = 1628) were measured by echocardiography 2.4 (0.4) years later.
Results: Each unit of fitness was associated with a 6.46 mmHg increase (95% CI: 5.83, 7.09) in exercise systolic BP. Exercise systolic BP increased step-wise by third of fitness (difference 6.06 mmHg, 95% CI:4.99, 7.13 first vs. middle; 11.13 mmHg, 10.05, 12.20 middle vs. highest). Each 5 mmHg increase in exercise systolic BP was associated with 0.25 g/m^2. 7 (0.16—0.35) greater LVMI, attenuated with adjustment for fitness. There was evidence of an interaction between fitness and exercise BP on LVMI, more-marked in the middle fitness third (difference -0.27g/m2.7, -0.51,0.04 vs. first third), but similar in lowest and highest fitness thirds. CO increased (difference 0.06 L/min, -0.05,0.17; 0.23 L/min, 0.12,0.34), TPR decreased (difference -0.13AU, -0.84,0.59; -1.08AU, -0.1.80,-0.35 with fitness.
Conclusion: Fitness may modify associations between exercise BP and LVMI in adolescence. Higher CO, but lower TPR suggests a physiological exercise BP-LVMI relationship with higher fitness, rather than pathological elevations in exercise BP and LVMI.

2.8

RELATIONSHIPS BETWEEN ADIPOSITY AND LEFT VENTRICULAR FUNCTION IN ADOLESCENTS: MEDIATION BY BLOOD PRESSURE AND OTHER CARDIOVASCULAR MEASURES

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