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5.4: HIGHER PULSE PRESSURE IN OLDER PEOPLE IS ASSOCIATED WITH SMALLER AORTIC LUMEN AREA

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a mean of 9.2 ± 3.5 years after transplantation. The first year decline was related to smoking and acute rejection but the later decline was significantly and exclusively associated with donor age and aortic stiffness. In hypertensive humans, the significant association between PP and GFR indicates a cross-talk between the two parameters with arterial stiffness, and not vascular resistance, as major mediator.

5.2

CUFF AND TONOMETER BASED DEVICE FOR ASSESSMENT OF CAROTID TO FEMORAL PULSE WAVE VELOCITY: VALIDATION ACCORDING TO ARTERY SOCIETY GUIDELINES

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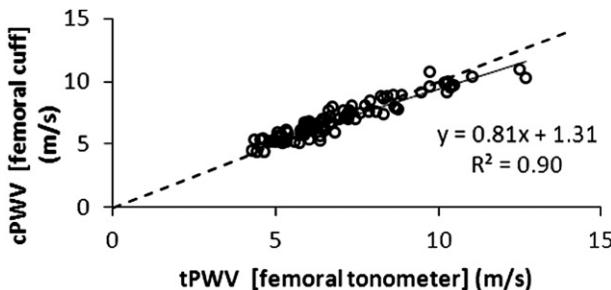
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Background: There is increasing interest in cuff-based devices for assessment of arterial pulse wave velocity (PWV). A recently developed device utilises a thigh cuff and carotid applanation tonometry for assessment of carotid to femoral PWV (cPWV; SphygmoCor XCEL, AtCor Medical; test device). Our aim was to validate the device against ECG gated tonometric measurement of PWV (tPWV) (SphygmoCor CvMS; control device) according to Artery Society Guidelines [1].

Methods: We recruited 94 subjects (48 female, 22-83 years, mean age 45.6 ± 19.4) in 3 centres (Australia, France, Italy). The thigh cuff was inflated automatically to sub-diastolic pressure and the cuff waveform was recorded simultaneously with the tonometric carotid waveform. Control and test devices were used in random order. PWV was determined from wave foot-to-foot delay and distance from suprasternal notch to femoral site or top of cuff, minus distance from suprasternal notch to carotid site. The average was computed of triplicate measurements by two operators.

Results: A high correlation was found between devices ($R^2=0.90$; Figure) with a mean difference of -0.02 ± 0.61 (SD) m/s. Mean difference and standard deviation (SD) between cPWV and tPWV was well within the "excellent" category acceptance criteria of the Artery Society guidelines (<0.5 m/sec and <0.80 m/sec, respectively).

Conclusion: The femoral cuff technique gives comparable PWV values to those acquired with the accepted standard ECG gated carotid/femoral tonometry PWV measurement technique.



[1] Wilkinson IB *et al.* Artery Society guidelines for validation of non-invasive haemodynamic measurement devices: Part 1, arterial pulse wave velocity. *Artery Research* 2010; 4:34-40

5.3

RELATIONSHIP BETWEEN SHORT-TERM BLOOD PRESSURE VARIABILITY AND LARGE-ARTERY STIFFNESS IN HUMAN HYPERTENSION

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Short-term blood pressure (BP) variability predicts cardiovascular complications in hypertension, but its association with large-artery stiffness is poorly understood and confounded by methodological issues related to the assessment of BP variations over 24h.

Carotid-femoral pulse wave velocity (cfPWV) assessment and 24-hour ambulatory BP monitoring were performed in 911 untreated, nondiabetic patients with uncomplicated hypertension (learning population) and in 2089 mostly treated hypertensive patients (test population). Short-term systolic BP (SBP) variability was calculated as the following: (1) SD of 24-hour, daytime, or nighttime SBP; (2) weighted SD of 24-hour SBP; and (3) average real variability (ARV), that is, the average of the absolute differences between consecutive SBP measurements over 24 hours. In the learning population, all of the measures of SBP variability showed a direct correlation with cfPWV (SD of 24-hour, daytime, and nighttime SBP, $r=0.17/0.19/0.13$; weighted SD of 24-hour SBP, $r=0.21$; ARV, $r=0.26$; all $P<0.001$). The relationship between cfPWV and ARV was stronger than that with 24-hour, daytime, or nighttime SBP ($P<0.05$) and similar to that with weighted SD of 24-hour SBP. In the test population, ARV and weighted SD of 24-hour SBP had stronger relationships with cfPWV than SD of 24-hour, daytime, or nighttime SBP. In both populations, SBP variability indices independently predicted cfPWV along with age, 24-hour SBP, and other factors. We conclude that short-term variability of 24-hour SBP shows an independent relation to aortic stiffness in hypertension. This relationship is stronger with measures of BP variability focusing on short-term changes, such as ARV and weighted 24-hour SD.

5.4

HIGHER PULSE PRESSURE IN OLDER PEOPLE IS ASSOCIATED WITH SMALLER AORTIC LUMEN AREA

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High pulse pressure (PP) contributes to the pathogenesis of hypertension and is associated with adverse cardiovascular disease outcomes. There is consensus that aortic wall stiffening contributes to higher PP. However, the role of lumen size in the pathogenesis of elevated PP remains controversial. Prior studies showing an unexpected inverse association between PP and lumen area have been criticized for using echocardiography, which affords limited views of the thoracic aorta. Therefore, we performed cine magnetic resonance imaging (MRI) of the ascending aorta 5 mm above the aortic valve and the proximal and distal descending thoracic aorta in 423 older participants (age 72 to 94, mean 79 years; 57% women) in the Age, Gene/Environment Susceptibility-Reykjavik Study (AGES-Reykjavik). Immediately prior to MRI, supine auscultatory blood pressure ($141\pm19/64\pm9$, $PP=77\pm18$ mmHg) and tonometry of brachial, radial, femoral and carotid arteries were performed. Mean aortic lumen area during the cardiac cycle was computed at each level and averaged across the 3 levels to give average lumen area (LA_M). Wall area (WA) and elastance ($E = PP \times A_D / (A_S - A_D)$) were similarly averaged. In linear regression models, LA_M was negatively related to PP when considered alone (Model 1) and in a model that adjusted for age, sex, height, weight, heart rate, total and HDL cholesterol, triglycerides, estimated GFR, diabetes, glucose, HbA1c and history of smoking (Model 2). The relation persisted after further adjustment for E and WA (Model 3). In our sample of older people, higher pulse pressure is associated with a smaller lumen area of the thoracic aorta.

Table Relations between pulse pressure and average aortic lumen area.

	Beta	SE	P
Model 1	4.5	0.9	<0.0001
Model 2	5.7	1.1	<0.0001
Model 3	5.9	1.2	<0.0001

Effects are expressed as pulse pressure increase per SD decrease in mean lumen area (mm Hg/SD).

5.5

ANDROGEN DEFICIENCY: A CRITICAL DETERMINANT OF AORTIC STIFFNESS IN MEN

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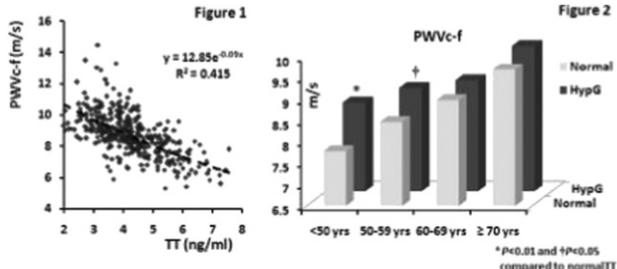
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Background: Low testosterone levels and increased aortic stiffness are predictive markers for those at high risk of cardiovascular disease. The influence of androgen level on the age/aortic stiffness relationship is unknown.

Methods: Total testosterone (TT) levels were measured in 382 men with no evidence of clinical atherosclerosis. Carotid-femoral Pulse Wave Velocity (PWVc-f) was measured as an index of aortic stiffness.

Results: Figure 1 illustrates the exponential increase in PWVc-f values with linearly decreasing total testosterone concentration. The inverse correlation between PWVc-f values and TT remained significant in multivariate analysis after adjustment for confounders ($\beta = -0.170$, $P < 0.001$). Subjects were then categorized by age decade and further subdivided according to presence/absence of hypogonadism ($TT < 3.4$ ng/ml). PWVc-f values of each age/testosterone category after adjustment for confounders are shown in figure 2. In the first two age categories, patients with hypogonadism (HypG) had higher adjusted PWVc-f (by 0.80 m/s, $P < 0.01$ and 0.45 m/s, $P < 0.05$, respectively) compared to subjects with TT concentration above the cut off level for biochemical definition of HypG. On the contrary, in older age categories, PWVc-f between patients with HypG and men with normal levels did not differ. It can be noted also that young men (<50 yrs and 50-59 yrs) with HypG had already elevated PWVc-f as compared to older men (50-59 and 60-69 yrs, respectively) with normal TT levels.

Conclusion: TT levels are independently associated with aortic stiffening. The effect of low testosterone concentration on aortic stiffness is emphasized in young men. These findings underline the important role of testosterone as a marker of arterial damage.



5.6

PULSE PRESSURE AND AGE

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We investigated individual and combined effects of increased aortic stiffness and resistance on (pulse, PP) pressure with aging. Wave travel and reflection determine the contribution of the arterial system to pressure wave shape and magnitude. Reflections occur at all branch points, and these local reflections cause amplification (distal pressure equal forward plus backward pressure). We used an anatomically accurate model of the human systemic arterial tree in this analysis. We found that ascending aorta pressure wave shape and Reflection Magnitude ($PP_{\text{forw}}/(PP_{\text{backw}}+PP_{\text{forw}})$) are not changed with changes in peripheral resistance. This suggests that pressure wave shape depends on multiple local reflections, i.e., large artery (aortic) geometry and stiffness. Mean pressure depends on resistance only. Over the physiologic range changes in geometry have little effect, but stiffness does. Using aortic PWV and area as function of age (20–80 years, PWV 4–10 m/s) we calculated aortic stiffness. For constant resistance systolic pressure increased and diastolic pressure decreased with age (stiffness). An increase in peripheral resistance of 20% and decrease in CO of 5% between 20 and 80 yrs, increased both systolic and diastolic pressures but left PP unaltered. Both effects together let systolic and mean pressure increase (106–161 and 93–108 mmHg, resp.) and diastolic pressure decrease (81–71 mmHg). We conclude that increased pulse pressure results from increased aortic stiffening not from increased vascular resistance. Increased mean pressure results from increased resistance. Both effects together causes increased systolic pressure and may cause a decrease in diastolic pressure, depending on the resistance change.