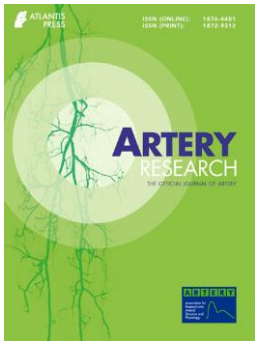

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5.6: CENTRAL BUT NOT PERIPHERAL FAT MASS IS ASSOCIATED WITH BLOOD PRESSURE COMPONENTS

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5.5
WHY DO PATIENTS WITH AORTIC STENOSIS AND UNOBSTRUCTED CORONARY ARTERIES SUFFER FROM ANGINA? INSIGHTS FROM TRANSCATHETER AORTIC VALVE INSERTION (TAVI)

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Introduction: It is unclear how aortic stenosis causes angina despite unobstructed coronary arteries. Transcatheter Aortic Valve Implantation (TAVI), permitting the instantaneous abolition of the stenosis, allows quantification of the direct physiological impact of the stenosis independent of associated pathologies such as left ventricular hypertrophy.
Methods: Intracoronary pressure and flow velocity were measured immediately before and after TAVI in six patients with unobstructed coronary arteries. We calculated the intra-coronary diastolic suction wave (the principal accelerator of coronary blood flow). To test physiological reserve to increased myocardial demand, we measured pressure and flow velocity at rest and during pacing at 90 and 120 beats per minute.
Results: Prior to TAVI the basal myocardial suction wave intensity was $2.2 \pm 1x10^{-5}$ $Wm^{-2}s^{-2}$, and increased in magnitude with increasing severity of aortic stenosis ($r = 0.82$, $p = 0.04$). This wave decreased markedly with pacing at higher heart rate (β coefficient $= -0.19$ $x10^{-4}$ $Wm^{-2}s^{-2}$, $p = 0.003$). After TAVI despite a fall in basal suction wave (2.2 ± 1 v $1.0 \pm 0.4x10^{-5}$ $Wm^{-2}s^{-2}$, $p < 0.004$), there was an immediate improvement in coronary physiological reserve as assessed with pacing (β coefficient $= 0.12$ $x10^{-4}$ $Wm^{-2}s^{-2}$, $p = 0.014$).
Conclusions: In aortic stenosis, the coronary physiological reserve is reversed: instead of increasing with increased myocardial demand, the coronary diastolic suction wave paradoxically decreases. Immediately after TAVI, this physiological reserve returns to a normal positive pattern. This may explain why patients with aortic stenosis suffer from angina despite unobstructed coronaries and the prompt relief of angina after TAVI.

5.6
CENTRAL BUT NOT PERIPHERAL FAT MASS IS ASSOCIATED WITH BLOOD PRESSURE COMPONENTS

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Aim: Both obesity and arterial stiffness are associated with cardiovascular disease. In the present study we investigated the possible associations between fat mass, measured at different locations, and measures of arterial stiffness in a geriatric outpatient's population.
Methods: Cross-sectional study. Fat mass was measured at different locations with a dual-energy X-ray absorptiometry (DEXA). Blood pressure was measured with Dynamap®, central blood pressure and augmentation index were calculated with Sphygmocor®. Associations were tested by regression analysis.
Results: 216 subjects were included. Mean age was 77.3 years. Central fat mass was associated with pulse pressure (PP) [0.579 (95CI% 0.177;0.981)], systolic blood pressure (SPB) [0.581 (95CI% 0.196;0.966)] and negatively with diastolic blood pressure (DBP) [−0.215 (95CI% −0.365;−0.064)], whereas peripheral fat mass was not (table 1). No association was found between measures of obesity and the AIx.
Conclusion: In the elderly, central but not peripheral fat mass is associated with blood pressure levels. The present study suggests the pathophysiological role of central fat mass in determining blood pressure levels.

6.1
GENETIC VARIATIONS IN FIBULIN 1 AND AGGRECAN GENES ASSOCIATED WITH ARTERIAL STIFFNESS IN YOUNG HEALTHY ADULTS

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Background: Cardiovascular disease is the commonest cause of death worldwide and premature arterial stiffening is a key contributor to this risk. Stiffness is highly heritable, but despite a clear genetic basis, the precise molecular pathways regulating stiffness are poorly understood. We aimed to identify the possible genetic risk loci that associated with arterial stiffness in young healthy adults who have little evidence of atherosclerosis or other confounding factors.
Methods: In this candidate gene based association study, the most important tagging SNPs which influenced arterial stiffness were investigated in ENIGMA study. Genotyping was performed in two-stages. First, participants were selected for top and bottom deciles of PWV ($n = 480$) and genotyping carried out using Illumina Golden Gate assays. Significant tSNPs were subsequently tested in additional ENIGMA participants ($n = 1400$) using ABI Taqman assays.
Results: In primary analyses, a number of tSNPs were identified with a significance level of <0.001. However, only those tSNPs pertaining to extracellular matrix (ECM) are presented. Two tSNPs in Fibulin 1 (rs2018279, rs2238823) and three in Aggrecan (rs3743399, rs2882676, rs2293087) genes associated significantly with PWV after adjusting for confounding factors (adjusted $R^2 = 0.38$; $p < 0.001$) when subjects were selected for deciles of PWV. Similar findings were replicated when data was analysed including the whole cohort (adjusted $R^2 = 0.28$; $p < 0.005$).
Conclusions: These data demonstrate that genetic variants of key ECM proteins were significantly associated with increased risk of large artery stiffening in young healthy adults. However, additional studies are needed to determine whether variation in these marker genes is associated with other measures of arterial stiffness.

6.2
LARGE ARTERY STIFFNESS ASSESSMENT WITH ARTERIOGRAPH DEVICE

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Introduction: Large artery stiffness is recognized as a strong, independent marker of cardiovascular risk, mainly through aortic pulse wave velocity (PWV). Carotid stiffness (CS), directly measured from echo tracking is correlated with PWV and has been associated with CV risk. Arteriograph is a new non-invasive oscillometric method, which estimates aortic PWV through brachial pressure wave analysis and provides an aortic stiffness index (ASI).
Aim: To compare CS with echotracking to ASI with arteriograph and define their determinants and discrepancies in a large unselected middle age population.
Methods: CS was assessed by echotracking system (ArtLab®) and central pulse pressure by calibrated distension waveforms, ASI by Arteriograph. R-squared Pearson's correlation coefficient (R^2) between the methods was calculated.
Results: 682 patients were included: 68 healthy control subjects (CTL), 412 patients with "non treated risk factors" (NTRF) and 202 with "treated risk factors" (TRF). Among NTRF and CTL patients, correlation between CS and ASI were weak ($R^2 = 0.019$ and 0.016 respectively, $p < 0.01$). In a robust multiple stepwise regression analysis, ASI was determined by mean blood

Table 1 Associations between central and peripheral fat mass percentages (FM) and blood pressure components.

	SBP β (95% CI)	DBP β (95% CI)	PP β (95% CI)	AI_x β (95% CI)
FM arms	0.025 (−0.052;0.101)	−0.006 (−0.036;0.024)	0.024 (−0.056;0.104)	−0.017 (−0.056;0.023)
FM legs	0.307 (−0.124;0.738)	−0.132(−0.300;0.036)	0.395 (−0.052;0.843)	0.139 (−0.083;0.360)
FM trunk	0.581 (0.196;0.966)	−0.215 (−0.365;−0.064)	0.579 (0.177;0.981)	0.148 (−0.053;0.349)

Model is adjusted for age, gender, MAP, HR and BMI.