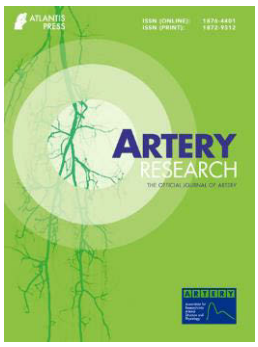

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P.022: VALIDITY OF THE TENSIOCLINIC DEVICE TO MEASURE ARTERIAL STIFFNESS IN PATIENTS ON HEMODIALYSIS

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P.022
VALIDITY OF THE TENSIOCLINIC DEVICE TO MEASURE ARTERIAL STIFFNESS IN PATIENTS ON HEMODIALYSIS

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Assessment of arterial stiffness in dialysis patients has prognostic significance. The TensioClinic device uses oscillometrically obtained wave forms to calculate pulse wave velocity (PWV) and augmentation index (AI). Our objective was assess the validity of measurements of TensioClinic (PWV_T, AI_T) compared to that of the validated PulsePen tonometer (PWV_P, AI_P).
We measured PWV and AIx in duplicate, before and after hemodialysis, in 94 hemodialysis patients. Reliability of a given device was assessed by calculating the standard deviation of the difference (SDD) between the first and second measurement. Validity of TensioClinic was evaluated by comparing its results to that obtained by the PulsePen device, using correlation analysis and Bland-Altman plots.
Predialysis SDD for PWV_P and PWV_T were –0.03±0.94 m/s and 0.51±1.35 m/s, postdialysis PWV_P and PWV_T SDD-s were 0.09±1.419 m/s and 0.07±1.81 m/s, respectively. Pre- and postdialysis SDD for AI_P and AI_T were 0.87±5.49% and 1.81±8.74%, and 0.79±4.01% and 3.54±22.69%. Mean predialysis $PWV_P$was 10.95±2.89 m/s and PWV_T 9.97±2.36 m/s. Postdialysis PWV_P was 11.59±2.92 m/s and PWV_T 10.37±3.24 m/s. Significant correlation was found between predialysis PWV_P and PWV_T ($r = 0.28$, $p < 0.05$). After dialysis the correlation was not significant ($r = 0.16$). Mean predialysis AI_P was 23.32±12.27% and AI_T 2.17±26.4%. Postdialysis AI_P and AI_T were 22.01±12.54% and –4.36±37.81%, respectively. Significant correlation was found between pre- and postdialysis AI_P and AI_T, $r = 0.40$ and 0.26, respectively.
PWV and AI measurements with PulsePen are more reproducible compared to TensioClinic. Poor correlation between the results obtained by the two devices may limit the use of TensioClinic in hemodialysis patients.

P.023
THE RELATIONSHIP BETWEEN PERIPHERAL ARTERY PULSE WAVE VELOCITY AND VASODILATOR FUNCTION IN HYPERTENSIVE PATIENTS

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Pulse wave velocity is commonly employed as a summary descriptor of physical arterial characteristics and can be measured rapidly using several commercially available devices. While brachial artery vasodilator function remains a more established index of endothelial health, its potential for widespread clinical use is limited. We examined the relationship between these two vascular measures among hypertensive patients recruited from local hospital outpatient clinics.
85 patients [mean (±SD) blood pressure 151(±15)/81(±10) mmHg] attended for vascular function assessment. Carotid-radial pulse wave velocity (CRPWV) was measured by sequential applanation tonometry (Sphygmocor, Atcor Medical). Brachial vasodilator responses to escalating doses of intra-arterial acetylcholine (endothelium-dependent agonist) and sodium nitroprusside (nitric oxide donor) were quantified by venous occlusion plethysmography.
CRPWV was inversely correlated to maximum acetylcholine-mediated vasodilation ($r = -0.287$, $p = 0.008$) but no such relationship was seen with sodium nitroprusside ($r = -0.002$, $p = 0.987$). Determinants of CRPWV were examined by setting it as the dependent variable in a multiple regression analysis which included sex, age, systolic blood pressure, diastolic blood pressure and maximum vasodilator response to acetylcholine. Significant independent predictors of CRPWV were male sex ($\beta = 0.298$, $p = 0.006$), diastolic blood pressure ($\beta = 0.248$, $p = 0.018$) and response to acetylcholine ($\beta = -0.267$, $p = 0.014$).
Among a group of hypertensive patients, there was a significant inverse relationship between CRPWV and brachial vasodilator response to acetylcholine, independent of distending blood pressure. This suggests that CRPWV may represent a rapidly obtainable estimate of arterial health.

P.024
MEASURING LOCAL PULSE WAVE VELOCITY USING NON-INVASIVE MULTIPLE M-LINE ULTRASOUND.

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Introduction: Local pulse wave velocity (PWV) provides direct information about the mechanical properties of an artery. Although PWV is related to arterial stiffness and both are predictors of cardiovascular events, no methods are currently available to measure local PWV non-invasively.
Method: The common carotid artery (CCA) of 4 young subjects were measured with multiple M-line ultrasound, resulting in 14 distension waveforms spaced over 17 mm. PWV was determined by applying linear regression to the foot of the distension waveforms and the corresponding echo line position. The PWV was accepted for further analysis if the correlation coefficient of both parameters was above 0.95.
Results: The local PWV measurement method had a good intra-subject coefficient of variation (CV) of <5%. The average PWV was 3.5±0.7 m/s with an inter-subject CV of 20%. One subject, with high blood pressure (140/90 mmHg), had a PWV of 4.6±0.2 m/s.
Discussion: These results show a low PWV in the CCA compared to conventional carotid-femoral (CF) PWV. The muscular arteries that are part of the CF-trajectory increase the CF-PWV. Moreover since pulse waves travel in opposite direction, using the distance between carotid and femoral artery causes CF-PWV to overestimate true PWV.
Conclusion: Local PWV can be measured with good precision using multiple M-line ultrasound. The inter-subject variation (CV = 20%) exceeds the intra-subject variation (CV = 5%), enabling distinction between individual differences. More measurements are required to evaluate the accuracy of the local PWV method in vivo.

P.025
ABSENCE OF SYNDECAN-1 RESULTS IN INCREASED INFARCT HEALING AND DEPRESSED CARDIAC FUNCTION AFTER ACUTE MYOCARDIAL INFARCTION

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Syndecan-1 (Syn1) has been implicated in angiogenesis during tumor formation and wound healing. To investigate the unknown role of Syn1 in cardiac healing, remodeling and function, acute myocardial infarction (AMI) was performed in Syn1-deficient (Syn1KO) and wild type (WT) mice.
Cardiac function and structure did not differ between sham-operated WT and Syn1KO mice at 14 days. However, significantly less necrotic cardiomyocytes remained in the infarct of Syn1KO *versus* WT mice (% necrotic cardiomyocytes; 16±4.1 in WT vs 1.3±0.1 in KO, $n = 5$, $p < 0.05$) suggesting accelerated infarct healing in absence of Syn1 at 14 days. Increased number of CD45-staining leukocytes and CD31 staining capillaries in Syn1KO *versus* WT mice (CD45; 1068±283 in WT vs 2076±193 in KO; CD31; 138±5.9 in WT vs 233±19 in KO, $n = 5$, $p < 0.05$) confirmed accelerated infarct healing in absence of Syn1. Cardiac contractility was significantly depressed in Syn1KO as compared to WT mice in response to dobutamine (dP/dtmax, mmHg/s; 9080±830 in WT vs 5550±1140 in KO, $n = 5$, $p < 0.05$). Decreased fractional shortening and increased end-diastolic dimensions in Syn1KO *versus* WT mice at echocardiography (%FS: 19±2.5 in WT vs 11±1.3 in KO; EDD, mm: 5.6±1.2 in WT vs 6.6±0.3 in KO, $n = 5$, $p < 0.05$) confirmed depressed cardiac function in Syn1KO mice, resulting in a 70% higer lung to body weight index in Syn1KO as compared to WT mice.
In conclusion, absence of Syn1 results in increased inflammation, angiogenesis and accelerated healing after AMI, leading to increased dilatation and decreased cardiac function.

P.026
A PIVOTAL ROLE OF THE ANTI-ANGIOGENETIC FACTOR THROMBOSPONDIN-2 IN THE PROGRESSION OF CARDIAC HYPERTROPHY TO FAILURE.

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Introduction: Thrombospondin-2 has been implicated in angiogenesis during wound healing and tumour formation, but its role in cardiac hypertrophy and function during cardiac overload is unknown.
Methodology: First, micro-array analysis of cardiac biopsies taken in hypertensive renin-overexpressing rats at the compensated phase (10 weeks) revealed that increased cardiac expression of the matricellular protein TSP2 identified the failure-prone hearts. Subsequently, thrombospondin-2 (TSP2) knockout and their wild type littermates were submitted to Angiontensin II infusion (0.5 μg/g/day). After detailed hemodynamic analysis, hearts were taken out and prepared for further histological and molecular analysis.
Results: TSP2 immunostaining in WT mice revealed increased expression in areas of active remodeling after AngII infusion, with a maximum at 4 and 7 days and declining at 14 days. When TSP2 knockout (KO) mice were submitted to Ang II infusion, 70% of TSP2 KO mice succumbed due to fatal cardiac rupture. The surviving TSP2 KO mice showed severe cardiac failure, as indicated by decreased fractional shortening and increased diastolic dimensions, whereas cardiac rupture, dilatation or dysfunction were absent in TSP2 WT mice. Ultrastructural analysis of AngII treated TSP2-KO hearts revealed oedema and disruption of the extracellular matrix, associated with increased activity of the collagen degrading enzymes MMP-2 and MMP-9.
In conclusion, increased expression of the matricellular proteins TSP2 and SPARC during cardiac overload or ischemia protects against adverse cardiac remodeling, thereby preventing cardiac dilatation, failure or fatal cardiac rupture.