2.1: 24 HOUR CENTRAL AMBULATORY BLOOD PRESSURE: USUAL VALUES AND RELATIONSHIP WITH MARKERS OF CARDIOVASCULAR RISK


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Background: Atherosclerotic plaque development is associated with increased oxidative stress, that promotes angiogenesis, lipid oxidation and uptake, and ensues cell death. In addition, biomechanical stress, i.e. blood-pressure variations with every heart beat, may further enhance plaque vulnerability. Ivarbivinad, a heart-rate lowering drug, is associated with reduced oxidative stress and diminished atherosclerotic plaque forma-
tion in mice, yet it’s role on plaque microvasculature and biomechanical stress is unknown.

Methods: Endothelial denudation (balloon-injury) was performed on the abdominal aorta of 18 New-Zealand-rabbits fed with a high cholesterol diet for 14 weeks. Nine rabbits received ivarbigavrinad (17 mg/kg/d) within drinking water throughout the study. Dynamic contrast-enhanced MRI was performed to quantify plaque size and microvasculature (area-under-
time-concentration curve, AUC). Blood pressure and relative distension were measured using a pressure catheter and high frame-rate ultrasound.

Results: Systolic, diastolic, and pulse pressure, and (relative) distension were similar between the ivarbigavrinad and control group (all p>0.25). But, due to 15%-reduction in heart-rate (p=0.03), the accumulated biomechan-
ical stress on the plaque is lower in the ivarbigavrinad group. MRI plaque size was similar between the groups (p=0.1). AUC was 25% lower for ivarbiga-
dine-treated animals (p=0.03). Linear regression showed a negative trend between heart-rate and AUC when adjusting for ivarbigavrinad (p=0.1).

Discussion: Ivarbigavrinad led to lowered AUC on MRI, indicating decreased plaque microvasculature, which is thought to be an important determinant of reduced plaque vulnerability. Ivarbigavrinad did not lead to reduced plaque size, despite reduced accumulated biomechanical stress. Upcoming histolog-
ical analysis might further unravel the effect of ivarbigavrinad on atherosclerosis.

1.6 AUGMENTATION PRESSURE INDEPENDENTLY ASSOCIATES WITH TIME TO PEAK SYSTOLIC MYOCARDIAL WALL STRESS

H. Gu a, M. Sinha a,b, J. Simpson a,b, P. Chowienczyck a
aKing’s College London, London, UK
bEvelina London Children’s Hospital, London, UK

Background: Central augmentation pressure (AP), an important component of central pulse pressure may be influenced by ventricular dynamics. We exam-
ined whether AP relates to time to peak systolic myocardial wall stress (MWS) independently of age, gender, body surface area (BSA), systolic blood pressure (SBP) and heart rate (HR) in subjects with a wide range of blood pressure.

Methods: We studied 133 subjects, evaluated for hypertension but other-
wise free of clinically apparent cardiovascular disease aged 47.2±16.9 (mean ± 5D) years with mean systolic blood pressure of 137±21 mmHg. Ca-
rotid pressure, obtained by tonometry calibrated from peripheral mean and diastolic BP, was used to calculate AP (difference between the second and first systolic peaks of the aortic waveform). MWS, a function of left ventricle (LV) pressure, myocardial wall volume and cavity volume was obtained using a translational study we analyzed in a cohort of 2679 patients with myocar-
dial infarction the effect of TLR4 896A/G single nucleotide polymorphism on SBP, pulse pressure and hypertension in dependency on age.

Results: C57Bl/6j and TLR4 null-mice had in adulthood similar SBP, aPWV and similar oxidative burden. During aging in C57Bl/6j mice SBP, aPWV and MDA increased (15mmHg, 2m/s, 30%, respectively). Aged TLR4 null-mice did not show these changes. In the upper age tertile of the patient cohort (age >70 years), patients with a TLR4 896A/G single nucleotide polymor-
phism had lower SBP and pulse pressure (7mmHg) and less hypertension (7% versus 60%). The TLR4 SNP remained a significant predictor for SBP in univariate and multivariate regression analysis.

Discussion: We propose that TLR4 signaling participates in SBP increase with age by inducing vascular aging.

2.1 24 HOUR CENTRAL AMBULATORY BLOOD PRESSURE: USUAL VALUES AND RELATIONSHIP WITH MARKERS OF CARDIOVASCULAR RISK

C. McEniery a,b, J. Smith a, G. Pestelli b, J. Woodcock-Smith a, K. Miles a, J. Cockcroft c, I. Wilkinson c
aUniversity of Cambridge, Cambridge, UK
bUniversity of Genova, Genova, Italy
bCardiff University, Cardiff, UK

debridement (balloon-injury) was performed on the

Brachial ambulatory blood pressure monitoring (ABPM) provides greater predictive value for cardiovascular (CV) events than clinic blood pressure (BP). However, 24hour ambulatory central BP (central ABPM) may be more closely related to surrogate markers of CV risk than brachial ABPM. The aims of this study were to determine usual values of central ABPM in apparently healthy, unmedicated individuals and to determine whether these relate to two established markers of CV risk, left ventricular (LV) mass and carotid intima-media thickness (cIMT).

24hour brachial and central ABPM was undertaken in 730 healthy individuals aged 18-88 years, using the Mobil-O-Graph device, together with clinic-

Based measurements of BP. A sub-set of individuals underwent assessment of LV mass (n=356) and cIMT (n=483), by ultrasound.

Central pulse pressure (PP) increased and PP amplification decreased significantly at night (P<0.001 for both). Daytime central, but not brachial, ABPM was significantly and independently associated with cIMT (R²=0.37, P<0.01) and, in general, correlations between central or brachial ABPM parameters and cIMT were stronger in younger (<50years) than older individu-

als. The association between 24hour central ABPM and LV mass was of borderline significance (R²=0.16, P=0.05). However, the associations be-
tween central or brachial ABPM parameters and LV mass were only signifi-
cant in older individuals.

The variation in PP amplification within individuals over 24hours, indicates that brachial and central BPs are differentially affected by the activities of daily living. Moreover, central, rather than brachial ABPM is more strongly related to surrogate markers of CV risk.

2.2 DETERMINATION OF THE RESTENOSIS DEGREE INSIDE THE IMPLANTED STENT WITH INTEGRATED WIRELESS PULSE WAVE VELOCITY (PWV) SENSOR

A. Domnich, J. Schaechtele
Fraunhofer IPA, Stuttgart, Germany

Patients with implanted stents were often (approx. 30 %) faced with the restenosis. By now no alternative to clinical examination is known to get a quick diagnosis for the actual state of possible and probable in-stent-

restenosis. At Fraunhofer IPA in Stuttgart a simple method to measure and to
determine the restenosis degree was invented and implemented. The Proof of principle was conducted on the experimental rig on an artery model. The approach is based on an inductive coupling between the external detection unit and implanted sensors. Two passive sensors were integrated in a stent and consist of a capacitive pressure sensor and an air-coil. Connected they form an oscillating circuit, the resonance frequency of which functionally depends on the local pressure. The extra-corporal detection unit generates an alternating magnetic field by 35 MHz. The spreading pulse wave changes the resonance frequency of the passive oscillating circuits inside the vessel.