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### **P2.02: COMPARISON OF CENTRAL BLOOD PRESSURE MEASURED BY APPLANATION TONOMOMETRY AND ECHOTRACKING**

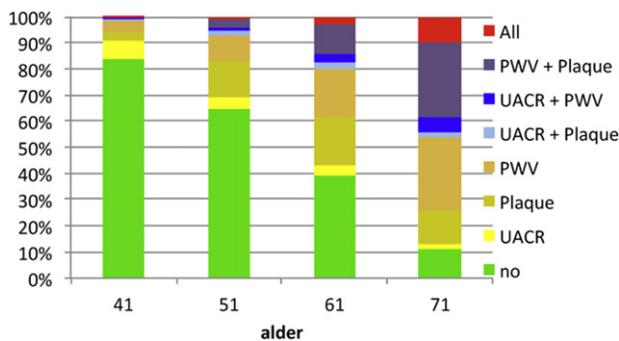
A.B. Bertaux, E.B. Bozec, M.A. Alivon, S.L. Laurent, P.B. Boutouyrie

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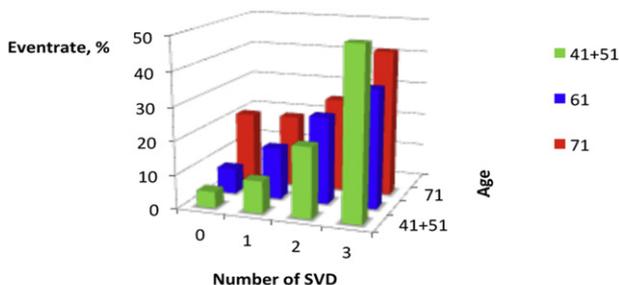
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that the advantage of combined markers for SVD is doubtful in subjects aged 41 years or SCORE<1% due to low prevalence as well as in subjects aged 71 years or SCORE≥10% due very high prevalence and low additive predictive value.



Prevalence of SVD in age groups.

Share of SVD in age groups. Illustrating number of SVD increasing with age.



Eventrate

Proportion of events, CVE, in each age group and grouped by number of SVD.

#### P1.44

##### NEURAL BARORECEPTOR SENSITIVITY IN SUBJECTS WITH METABOLIC SYNDROME

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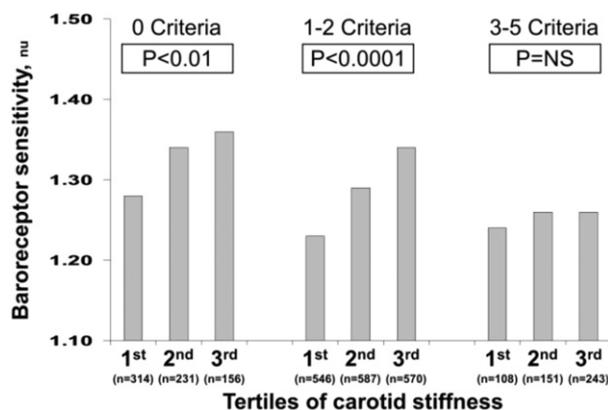
One of the most common non-invasive techniques to study the baroreflex is the spectral analysis of blood pressure (BP) and heart rate variability. The recent use of carotid distension rate instead of BP has permitted to study the neural path of the baroreflex after fully controlling for the vascular component. We previously discovered a new compensatory mechanism, reporting that the neural baroreceptor sensitivity (BRS) is higher in subjects with high carotid stiffness. We aimed to test whether this new compensatory mechanism is maintained in subjects with metabolic syndrome (MS).

**Methods:** From the PPS3 study, a large epidemiological survey of working people of age 50-75, were selected 2835 individuals non-diabetic, non-smokers, untreated by either anti-hypertensive or lipid-lowering drugs, and free from overt or familiarity for cardiovascular disease. A total of 701, 1673 and 461 subjects with respectively 0, 1-2 and 3-5 criteria for MS were studied.

**Results:** Neural BRS decreases significantly from subjects with 0 (median 1.33, IQ1.15-1.49 normalized units) to those with 1-2 (median 1.30, IQ1.10-1.47) and 3-5 criteria for MS (median 1.26, IQ1.08-1.43). Neural BRS

was not significantly increased in subjects with both high carotid stiffness and 3-5 criteria for MS (Figure 1), suggesting the presence of neuropathy in subjects with MS.

**Conclusions:** Neural BRS is reduced in subjects with MS. The compensatory, carotid stiffness-dependent, increase of neural BRS is abolished in subjects with MS.



#### P2 – Methods

##### P2.01

##### REPRODUCIBILITY OF CAROTID-TO-FEMORAL PULSE WAVE VELOCITY MEASUREMENT: QUANTITATIVE EFFECTS OF DISTANCE AND TRANSIT TIME ASSESSMENT

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**Background:** Carotid-femoral pulse wave velocity (PWV) is calculated on the basis of body surface distance (determined by either direct or indirect paths) and the corresponding transit time. The impact of distance vs time measurement on PWV reproducibility has not been quantified.

**Methods:** In 34 volunteers (age 47±19 years), carotid-femoral distance and transit time were measured twice by each of 2 trained observers, 2 hours apart, using a tape and a caliper. Two commonly used estimates of the travelled distance were calculated, namely "subtracted" (suprasternal notch to femoral artery minus suprasternal notch to carotid artery) and "direct" distance (carotid to femoral artery, multiplied by 0.8). Transit time was measured by high-fidelity tonometry (SphygmoCor, average of 3 readings for each of the 2 sessions). Variability was expressed as interobserver coefficient of variation (CV) and intra-class correlation (ICC).

**Results:** The CV was lowest for transit time (3.0%; interobserver difference ± SD, 0.0±1.8 ms), highest for subtracted distance (6.8%; 3.1±29 mm), and intermediate for direct distance (4.2%; 12.5±20 mm). The resulting interobserver differences in PWV were +0.0±0.2 m/s, +0.0±0.5 m/s, and +0.2±0.3 m/s, respectively. ICC was 0.98 for transit time (95% confidence interval [CI], 0.97-0.99), 0.73 for subtracted distance (95% CI, 0.53-0.86), and 0.81 for direct distance (95% CI, 0.66-0.90).

**Conclusion.** Interobserver variability of aortic PWV depends more on the measurement of body surface distance than on transit time. Estimates of the distance based on direct paths may generate a lower interobserver variability than those resulting from the combination of 2 paths ("subtracted" distance).

##### P2.02

##### COMPARISON OF CENTRAL BLOOD PRESSURE MEASURED BY APPLANATION TONOMOMETRY AND ECHOTRACKING

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**Background:** Some studies have shown the interest of measuring central blood pressure (CBP), which can be lowered differently by drugs at same systemic BP response. CBP is usually measured noninvasively by the Sphygmocor® device using applanation tonometry on radial artery.

Nevertheless, CBP can be calculated with echotracking at the common carotid artery (CCA) assuming the fact that the distension waveform is similar to a pressure curve.

**Objectives:** To analyse the accordance between CBP measured by the Sphygmocor® and by echotracking.

**Materials and Methods:** CBP measurements were performed on 115 patients of the unit. Applanation tonometry was performed on radial artery and CBP was estimated by the onboard transfer function of the Sphygmocor®. Echotracking measurements were performed on right CCA (ART.LAB) and treated to obtain Systolic Blood Pressure (SBP) and Pulse Pressure (PP). Pearson's correlation and Bland and Altman graph were performed.

**Results:** We showed a 14 mmHg overestimation of central SBP and PP by echotracking compared to applanation tonometry.

The correlations between central BP estimated by transfer function and systolic blood pressure calculated by echotracking were good (SBP:  $R^2=0,81$ ; PP:  $R^2=0,66$ ,  $p<0.001$ ).

**Conclusion:** This study has shown a good agreement between the two techniques to calculate central SBP in spite of a 14 mmHg overestimation by echotracking. As applanation tonometry has been shown to underestimate central pressure, echotracking could be the best technique to measure it. But more patients should be performed.

### P2.03

#### COMPARISON OF COMMON CAROTID ARTERY DISTENSION WAVE WITH TONOMETRY FOR CENTRAL PULSE PRESSURE ASSESSMENT

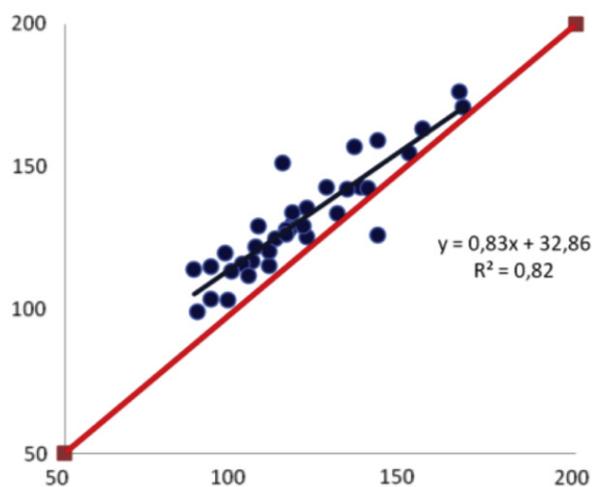
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**Aims.** Measurement of central blood pressure remains difficult. Also a good proxy for aortic pressure, carotid pressure is difficult to get through tonometry. Moreover, tonometry being a contact method, motion of the wall, especially if large, might alter the pressure waveform. We thus compared echotracking carotid distension waveforms with radial derived aortic waveforms for assessment of central pulse pressure.

**Methods:** We included 115 subjects aged 18-75 with valid carotid echotracking (ET) (Artlab, Esaote, NL) and radial derived aortic waveforms (TO) (Sphygmocor, Atcor, Aus). Central SBP and PP were compared with correlations and Bland-Altman method. ET was calibrated using radial mean and diastolic blood pressure

**Results:** Correlation equation for SBP was  $ET-SBP=0.83*TO-SBP+32.9$ ,  $R^2=0.82$ . Correlation equation for PP was  $ET-PP=0.80*TO-PP+21.5$ ,  $R^2=0.71$ . Mean bias for SBP was  $-12\pm 8$  mmHg and  $-12\pm 7$  mmHg for PP.

### Echotraking SBP (mmHg)



### Tonometry SBP (mmHg)

**Conclusion:** Although waveforms appeared very similar and correlation were very narrow, there was a systematic overestimation of SBP and PP by echotracking, compared to tonometry. Whether this is overestimation by ET or underestimation by TO remains to be demonstrated

### P2.04

#### PULSE WAVE VELOCITY AND WAVE INTENSITY IN THE CAROTID ARTERY OF HEALTHY HUMAN: WINDKESSEL AND WINDKESSEL-LESS ANALYSIS

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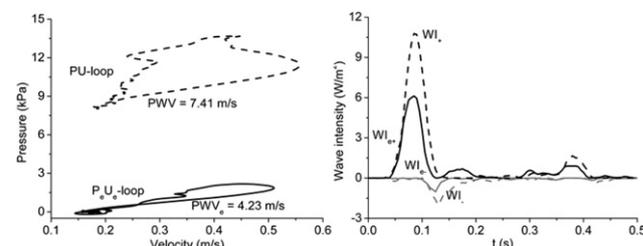
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Pulse wave velocity (PWV) is an acknowledged marker to arterial compliance. A time-domain approach to study wave propagation taking into account the Windkessel effects in arteries was introduced (1). The model assumes that the measured pressure (P) and velocity (U) are the sum of two components; a reservoir ( $P_r$ ,  $U_r$ ) due to the buffering capacity of arteries and an excess ( $P_e$ ,  $U_e$ ) due to the waves. A direct comparison of PWV and wave intensity (WI) with and without considering  $P_r$  is lacking and quantifying the differences constitutes the objective of this work.

We measured P and U in the carotid artery of 328 healthy human subjects ( $45\pm 6$  years old); a subset of the Asklepios study to examine pulse velocity and intensity with (PWV and WI) and without (PWV<sub>e</sub> and WI<sub>e</sub>) the Windkessel effects. PWV is 45% higher than PWV<sub>e</sub> ( $8.4\pm 2.4$  vs.  $5.8\pm 2.0$  m/s,  $p<0.001$ ). The intensities of the forward and backward compression waves of WI are 35% and 166% higher than those of WI<sub>e</sub> ( $21.8\pm 8.0$  vs.  $16.2\pm 7.1$  W/m<sup>2</sup> and  $3.2\pm 2.5$  vs.  $1.2\pm 0.9$  W/m<sup>2</sup>,  $p<0.001$ ).

Values of pulse wave velocity and intensities vary massively depending on whether  $P_r$  is taken into account. A  $P_r$ -independent methods for calculating these parameters are needed to determine the relative accuracy and importance of  $P_r$ .



**Figure 1** Wave speed (left) and intensity (right) using the Windkessel and Windkessel-less analysis.

(1) Wang J.J. et al. Time-domain representation of ventricular-arterial coupling as a windkessel and wave system. American Journal of Physiology - Heart and Circulatory Physiology 2003;284(4 53-4):H1358-H1368.

### P2.05

#### INVESTIGATION OF FLOW AND WALL SHEAR STRESS DURING PULSATILE FLOW IN A HUMAN AORTA WITH A COARCTATION AND POST-STENOTIC DILATATION USING LARGE EDDY SIMULATIONS

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Flow field and wall shear stress (WSS) in an idealized model of a human aorta with a coarctation and a post-stenotic dilatation, Figure 1, were investigated with large eddy simulation. A physiologic pulse was prescribed at the inlet, Figure 1. Phase averages of axial WSS, from 30 consecutive pulses, were determined between the throat ( $x=0$ ) and the exit ( $x=0.2$  m) along lines where the x-y plane and x-z plane cross the vessel wall. Figure 2 shows these values at late systole; the pattern is representative for the entire systolic phase. WSS peaks in the stenosis, as expected, but also at the end of the dilatation ( $x=0.06$  m). In the dilatation backflow causes a negative peak. Diastolic WSS is characterised by low amplitude oscillations. Also noticeable is the asymmetric pattern between the inner ( $y>0$ ) and outer ( $y<0$ ) sides caused by the flow through the arch. Thus, large spatial, temporal, and probably asymmetric WSS gradients in the already diseased region suggest increased risk for further endothelial dysfunction [1]. This reflects a complex, partly turbulent, flow pattern that may disturb the blood flow in the abdominal aorta.