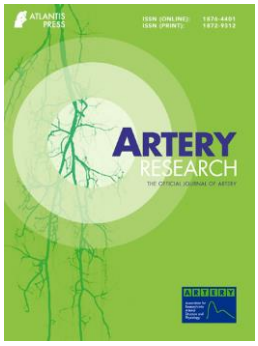

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P6.09: ULTRASONOGRAPHIC MEASUREMENT OF ARTERIAL WALL AND INTRAPLAQUE STRAIN IN THE CAROTID BULB AND COMMON CAROTID ARTERY

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4. Lumped Models

a. Two-element, Frank's, Windkessel. Required: *P(t)* and CO. Many ways to derive *C* (e.g. RC-time). Interpretation: R_p, and *C*, see 2. Aspects: No Z_c.

b. Three-element Windkessel. Required: *P(t)* and *F(t)*. Computer fits. Interpretation: Model description of 2, R_p, *C*, and Z_c. Aspects: Mean $P = CO(R_p + R_c)$ not only R_p.

5. *Wave Intensity Analysis*. Not a characterization of the arterial system alone. Reflections as 3b above.

CONCLUSION: only 2 and 4b characterize the whole arterial system.

P6.09

ULTRASONOGRAPHIC MEASUREMENT OF ARTERIAL WALL AND INTRAPLAQUE STRAIN IN THE CAROTID BULB AND COMMON CAROTID ARTERY

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Background: Transient ischemic attacks and stroke are commonly caused by vulnerable plaques in the carotid bulb. Noninvasive ultrasound imaging techniques, revealing spatial inhomogeneities in strain distribution, could possibly improve the timely diagnosis of plaque at risk of rupturing.
Methods: In 20 subjects with cerebrovascular ischemia, radiofrequency ultrasound data, covering 20 by 24 mm, of the carotid bulb and distal common carotid artery (CCA) were twice and bilaterally recorded during 6 cardiac cycles to assess 2D wall strain rate distribution at radial and lateral increments of 0.45 mm and 1.8 mm, respectively at peak distension rate.
Results: Reproducible strain rates were obtained for distal CCA and carotid bulbs free of plaques (unilateral intrasubject variation of 1.1 ± 0.6%/s and 1.2 ± 0.7 %/s, respectively). Strain rate was generally higher in the distal CCA (7.5 ± 3.9%/s) than in carotid bulb (3.4 ± 2.5%/s). Strain inhomogeneity was significantly associated to plaque area ($p < 0.001$) and was lower in distal CCA (1.2 ± 0.6) than in carotid bulb (1.6 ± 1.0).
Discussion: High resolution strain distribution in carotid segments reveals inhomogeneities in mechanical characteristics of the artery wall, and of the carotid artery plaques. Strain in the distal CCA is different in magnitude and inhomogeneity from the strain in the carotid bulb. Therefore, the proposed method for noninvasive identification of wall composition using ultrasonography might be a powerful tool to assess the plaque burden in an arterial segment particularly prone to plaque formation.

P6.10

A COMPARISON BETWEEN THE VICORDER AND SPHYGMOCOR DEVICES FOR THE NON-INVASIVE ASSESSMENT OF CENTRAL BLOOD PRESSURE

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Background: There is increasing evidence that central (aortic) blood pressure (BP) may be a more important determinant of cardiovascular risk than brachial BP. Most of actual devices to assess central BP non-invasively are partially operator-dependent, or rely on the detection of the late systolic shoulder as an estimation of central systolic BP (cSBP). The Vicorder is a new cuff-based, operator-independent device which obtains brachial BP waveforms using a volume displacement method and derives aortic waveforms using a previously published brachial-to-aortic mathematical transfer function[1].
Aim: to compare central BP measurements using the Vicorder and SphygmoCor devices.
Methods: 27 subjects (38 ± 11 years, 44% males) without cardiovascular diseases were studied after 10 minutes of supine rest. Brachial BP was assessed using the Vicorder. Brachial pressure waveforms calibrated to brachial systolic and diastolic pressure were recorded using the Vicorder and radial pressure waveforms calibrated to brachial mean and diastolic pressure were recorded using the SphygmoCor. The corresponding cSBP measurements were compared between devices (vcSBP versus sphygcSBP).
Results: The average (±SD) brachial BP was $113 \pm 12/63 \pm 8$ mmHg. There was good agreement between vcSBP (106 ± 12mmHg) and sphygcSBP (105 ± 15mmHg; $P = 0.56$). Similar results were observed for central pulse pressure (cPP), with a mean difference of 1.6 mmHg ($P = 0.18$). Conclusions: the values of cSBP and cPP provided by the Vicorder and SphygmoCor devices show good agreement when radial artery waveforms are calibrated to brachial mean and diastolic pressure. Further comparative data are required in a larger sample size, and with invasive BP measurements.

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P6.11

RELATIONSHIPS BETWEEN ENDOTHELIAL FUNCTION, ARTERIAL ELASTICITY AND BAROREFLEX SENSITIVITY

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Endothelium derived nitric oxide (NO) increases conduit artery distensibility, through relaxation of vascular smooth muscle. Barosensory wall distensibility influences the mechanical component of baroreflex sensitivity (BRS) by modifying the baroreceptor stimulus. NO bioavailability may also affect the neural component of BRS, by influencing afferent sensitivity, central neural processing or neuroeffector mechanisms.

We aimed to study the relationships between the above variables in young healthy volunteers ($n = 28$, male:female 10:18; age 16.5 ± 1.4 years).

To this end we measured brachial artery endothelial function by brachial flow mediated dilatation normalized with peak mean shear rates (nFMD) and carotid artery biomechanical parameters and blood pressure by ultrasonographic wall-tracking and tonometry. From these variables elastic parameters were calculated. BRS was measured by the spontaneous sequence method (Seq+).

The mean values (mean ± SD) were 5.41 ± 1.49 [10^{-3}/mmHg] and 4.79 ± 1.14 for carotid artery distensibility coefficient and stiffness index β, respectively, 2.59 ± 1.00 for nFMD and 25 ± 16 [ms/mmHg] for Seq+. Using correlation and linear regression analysis, we found that elastic parameters were not related, but BRS was significantly related to nFMD ($r = 0.486$, $p < 0.05$). However, the correlation was limited only to males ($r = 0.681$ $p < 0.05$ vs. $r = 0.368$ $p = 0.133$, females).

Our results suggest that the positive correlation between endothelial function and BRS can not be explained by the mechanical vessel wall properties. This association may be due to neural mechanisms transmitted by NO. The gender differences and the relationship between endothelial function and the neural component of the BRS need further studies.

P6.12

BRACHIAL ARTERY REACTIVITY IS DIRECTLY PROPORTIONAL TO BASAL BRACHIAL ARTERY TONE: POSSIBLE CONFOUNDER IN MEASURING ENDOTHELIAL FUNCTION

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Brachial artery reactivity following compression is used as a surrogate marker of endothelial function. Brachial diameter increases exceeding 10% indicate normal endothelial function. We investigated if basal brachial artery tone influenced brachial artery reactivity.

One hundred never smoking, healthy, normotensive, normolipidaemic subjects (41 + 9 yrs, 71F, 29 M) underwent brachial artery assessments. Basal, Reactive (endothelial dependant diameter change following 4 minutes of cuff compression) and Post GTN (endothelial independent dilatation) diameters were recorded.

Tone was calculated as the percent difference in Post GTN and Basal brachial diameters.

Basal diameter 3.6 + 0.05 mm, Reactive diameter 4.0 + 0.06 mm, Post GTN Diameter 4.4 + 0.1 mm

There was a significant correlation between Brachial artery reactivity and basal brachial artery tone (All $r = 0.60$, $p < 0.0001$, Women $r = 0.57$, $p < 0.0001$, Men $r = 0.68$, $p < 0.0001$).

These data indicate that basal brachial tone has a strong association with the degree of brachial artery reactivity in men and women and consideration should be given to the influence of tone on percent reactivity when measuring endothelial function by this methodology.

A Reactivity/Tone ratio should be considered once new cut-off points are determined.