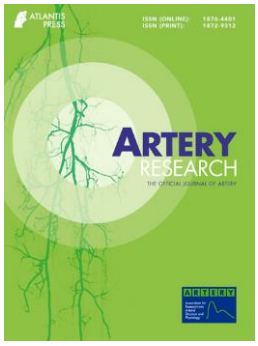

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7.4: THE INFORMATION CONTAINED IN WAVE REFLECTION

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0.28±0.04 nmol/min/mg proteins) and confirmed that GGT activity was mainly localized in the endothelium (histochemical reaction). The GSNO consumption in aorta homogenates (absorbance decay at 334 nm, 2.4±0.2 nmol/min/mg proteins) decreased by 57±3 % in the presence of serine borate complex (SBC, 20 mM), a competitive inhibitor of GGT, and increased by 24 ± 4 % when adding an acceptor of gamma-glutamyl residue, glycylglycine (glygly, 20 mM). The resulting release of •NO (Griess method) and nitrosation of proteins (Saville-Griess assay), were either reduced or improved with SBC and glygly, respectively (fig 1). Then, concentration-response curves to GSNO were performed in aortic rings (6-8 per experiment) precontracted with phenylephrine (1 μM) (fig 2). Half maximal effective concentration (EC_{50} determined by an Hill model) for GSNO (0.26±0.07 μM) increased with SBC (1.35±0.11 μM, $p<0.05$) and decreased with glygly (0.054±0.01 μM, $p<0.05$).
Such involvement of GGT in the vasorelaxant effect of GSNO should be taken into consideration for further development of new therapeutics using GSNO analogues.

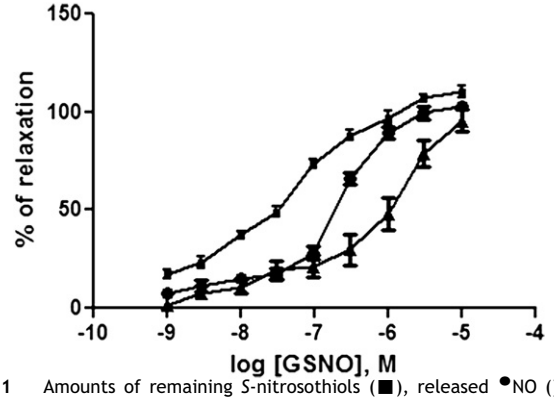

Fig. 1 Amounts of remaining S-nitrosothiols (■), released •NO () and nitrosated proteins (□) after incubation of 1 mM GSNO with aorta homogenates for 2 h at 37°C of, with and without SBC (20 mM) or glygly (20 mM). Data are means ± S.E.M. of 3 experiments. (* $p < 0.05$ *versus* GSNO alone)

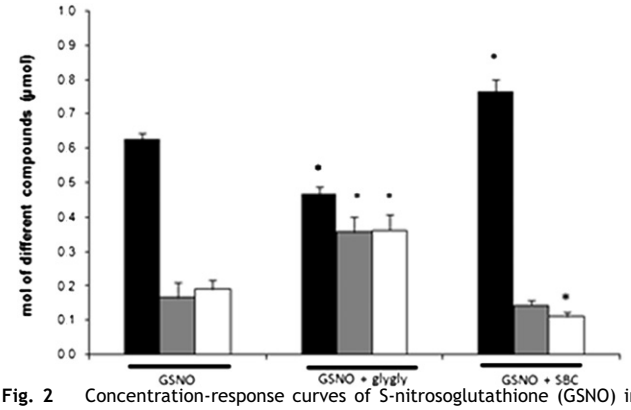

Fig. 2 Concentration-response curves of S-nitrosoglutathione (GSNO) in isolated rat aortic rings. The arteries were precontracted with 1 μM phenylephrine and concentrations (10^{-9}-10^{-5} M) of GSNO (●), GSNO + glygly (20 mM) (■) and GSNO + SBC (20 mM) () were added. Data are means ± S.E.M. of 6-8 experiments.

7.3
MICROCIRCULATION ABNORMALITIES AS RELATED TO ARTERIAL STIFFNESS AND CENTRAL BLOOD PRESSURE IN HYPERTENSIVE PATIENTS

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Background: The possible relationships between indicators of small resistance artery structure and of large artery distensibility have not yet been evaluated. Aim: to assess the relationship between carotido-femoral pulse wave velocity (CF-PWV), central blood pressure (cBP) and vascular alterations in small resistance arteries (media to lumen ratio, M:L) in patients with primary and secondary hypertension Patients and methods: In 65 patients (mean age 53±14 years, 31 F, 21 with diabetes mellitus type 2, 14 never treated) with essential (n= 32) and secondary (n=33) hypertension, pulse wave velocity was measured (Complior)and PW analysis was performed (Sphygmocor). In all patients small-resistance arteries were dissected from subcutaneous fat biopsies and mounted on an isometric myograph, for the measurement of the M:L. Results: Mean values of PWV and of M:L ratio were 11.4±2.6 m/s and 0.09±0.019, respectively. M:L ratio was significantly related to brachial systolic blood pressure (SBP) and pulse pressure (PP) ($r=0.40$ and 0.39, $p<0.001$, respectively) and to central SBP and PP ($r=0.44$ and 0.46, $p<0.001$, respectively). A positive correlation was observed between M:L and PWV ($r=0.43$, $p< 0.001$); this correlation remained statistically significant after adjustment for age and SBP (beta= 0.29, $p=0.03$). M:L ratio was also associated to augmentation pressure ($r=0.42$, $p< 0.001$); again this correlations remained statistically significant after adjustment for age, gender, mean BP and also for CF PWV. Conclusions: In hypertensive patients the presence of structural alterations of small resistance arteries may be associated with the increase in large arteries stiffness and, possibly contribute to an increase in central pressure by earlier wave reflections.

7.4
THE INFORMATION CONTAINED IN WAVE REFLECTION

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Wave reflection associates with cardiovascular events. However, it is less clear what information on arterial function, such as arterial stiffness and vascular resistance, can be derived. We therefore set out to find the relations between parameters obtained from wave shape analysis (Augmentation Index, Inflection and Shoulder points) and from wave separation analysis (Reflection Magnitude, and arrival time of the reflected wave) and aortic stiffness and taper.
Methods. We used a distributed model of the systemic arterial tree with properties as reported in earlier studies[1,2]. We changed vascular resistance, arterial stiffness and aortic taper.
Results. Peripheral Resistance has a negligible effect on wave reflection. Backward wave amplitude results, by equal amounts, from the distal aortic reflection (at length L) and from geometric taper and (other) aortic side branches. The wave reflected at the distal aorta runs with the same Pulse Wave Velocity (PWV) as the forward wave. The return time of the reflected wave, t_{return}, varies a factor two between small and strong taper. The t_{return} decreases 20% between aortic PWV's of 6 and 12 m/s, while 50% is predicted from $2L/t_{return}$. The timing of the shoulder and inflection points change with aortic stiffness but also less than predicted from 2L/PWV. The Augmentation Index and Reflection Magnitude ($P_{backward}/P_{forward}$) depend nonlinearly on stiffness; they increase with stiffness for low stiffness but not at high stiffness.
Characteristic impedance is needed for wave separation and therefore gives no independent information.
Conclusion. The data derived from wave form analysis and wave separation give poor information on arterial stiffness, no information on resistance and cannot be used to estimate these arterial parameters.
1. Westerhof et al.J Biomech 1969;2:121. 2. O'Rourke&Avolio Circ Res 1980;46: 363.

7.5
USING FINITE ELEMENT ANALYSIS TO MODEL ACOUSTIC RADIATION FORCE IMAGING (ARFI) OF CAROTID ARTERY PLAQUES

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The sudden rupture of carotid artery plaques can trigger an ischemic stroke event and eventually lead to death. Unfortunately, the identification of unstable plaques continues to elude clinicians. The use of Acoustic Radiation Force (ARF) to non-invasively provide a relative measure of tissue stiffness has shown promise for differentiating softer, lipid filled plaque regions believed to be more vulnerable than stiffer, calcified plaque regions[1].
A Finite Element Method (FEM) model to simulate the resultant displacements and associated stresses generated by ARF *in vivo* was implemented to