5.6: ASSOCIATION BETWEEN ENDOGENOUS SERUM TESTOSTERONE CONCENTRATIONS AND AORTIC PRESSURES AND PULSE WAVE AMPLIFICATION INDICES IN ERECTILE DYSFUNCTION PATIENTS

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In a cross-sectional design, we analyzed 1077 subjects from the Czech population-based study ("Post-MONICA"). Aortic pulse wave velocity (aPWV) was measured using a SphygmoCor device. sRAGE concentrations were assessed in frozen samples by ELISA methods (R&D Systems).

Aortic PWV significantly (p < 0.0001) increased across the sRAGE quartiles. After adjustment for all potential confounders, non-diabetic subjects in the bottom quartile of sRAGE (-9.18 m/sec) had odds ratio of raised aortic PWV (9.3 m/sec) was higher for older adults pre/post. (p < 0.05). However, FMD significantly decreased only in young males.

Conclusion: The results suggest that differential responses occur between young and older adults and between males and females in response to acute inflammation. Although inflammation increased similarly, inflammation had limited effects on vascular function in both young and old females, suggesting there are significant sex effects for arterial function in response to acute inflammation.

5.4 MATRIX GLA PROTEIN IN RELATION TO LEFT VENTRICULAR DIASTOLIC FUNCTION
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Background: Aging is associated with increased arterial stiffness and chronic low-grade inflammation. Acute inflammatory stimulus in the presence of low-grade chronic inflammation briefly increases the risk of cardiovascular events. The risk of cardiovascular events also increases substantially in older compared to younger women, therefore the relationship between sex and aging is important to understand.

Purpose: To describe the differential effects of age following induced systemic inflammation on arterial function, endothelial function, and wave reflection.

Method: Healthy volunteers aged 18–35 years (n = 18, 26yr, male = 6) or 55–75 years (n = 21, 64yr, male = 9) participated. Ultrasound of the common carotid and brachial artery was performed. Beta-stiffness (β) and flow mediated dilation (FMD) were calculated. Aortic mean arterial (sMAP), pulse wave analysis with wave separation, pulse wave velocity (PWV) measurements were obtained in the supine position at rest using an-applanation tonometry. Participants received an influenza vaccine to induce acute inflammation following baseline measurements and returned for 24-hour follow-up.

Results: C-reactive protein and interlukin-6 increased pre-to-post similar in all groups (p < 0.05). β was higher for older adults pre/post. (p < 0.05). Older males exhibited decreased MAP and reflected wave pressure (RP), but increased PWV following vaccination (p < 0.05). However, FMD significantly decreased only in young males.

Conclusion: The results suggest that differential responses occur between young and older adults and between males and females in response to acute inflammation. Although inflammation increased similarly, inflammation had limited effects on vascular function in both young and old females, suggesting there are significant sex effects for arterial function in response to acute inflammation.

5.5 MEASUREMENT OF ARTERIAL STIFFNESS BY ULTRAFAST ECHO: COMPARISON WITH ECHOTRACKING IN NORMOTENSIVE SUBJECTS AND HYPERTENSIVE PATIENTS
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Because measurement of arterial stiffness is highly dependent on blood pressure (BP), methods independent of BP are required. Ultrafast echography (UFE, Supersonix Imagine, Aix en Provence, France) makes use of very fast sampling rate (up to 10 kHz), so transient events such as pressure wave arrival can be tracked. This method has never been tested against classical echotracking (Artlab, Esatoire, Maastricht, NL) and carotid-femoral wave velocity (cf-PWV, SphygmoCor, AtCor, Sydney, Australia).

We included 56 subjects, 27 normotensives (NT) and 9 essential hypertensives (HT), matched for age and sex. We optimized UFE algorithms for pressure wave detection and tracking, for both foot of the wave (FW) and dicrotic notch (DN) PWV.

Feasibility appears good (FW: 78%, DN: 96%). The relations of arterial stiffness with age and blood pressure were stronger for echotracking and cf-PWV than for UFE. DN wave fronts appeared better associated with cf-PWV (r = 0.32, p < 0.001) and carotid PWV (r = 0.47, p < 0.001). FN was not associated with cf-PWV nor with carotid PWV. The residuals between DN and carotid PWV were not associated with BP or age. Similar associations between DN and cf-PWV/carotid PWV were found in NT and HT.

Conclusion: After optimizing algorithms for wave front identification and tracking, UFE appears as a promising technique for assessing arterial stiffness. DN showed the best associations with echotracking, whereas FW did not provide meaningful data. As previously shown by Hermelin et al (J Hypertens 2008 and 2009), FW is not appropriate for local stiffness measurement likely because of very early wave reflections.
between testosterone, central pressures and wave reflection indices in ED patients is unknown.

Methods: Total testosterone (TT) levels were measured in 407 consecutive ED patients (55 ± 8 y/o) without CVD. Central (aortic) systolic and pulse pressure, augmentation index (AIx) and augmented pressure (AP) as indices of pulse wave amplification across the arterial tree were measured with SphygmoCor device (AtCor Medical).

Results: TT levels were inversely correlated with systolic and pulse central pressures ($r = -0.195$ and $r = -0.249$, respectively) and wave reflection indices (AIx: $r = -0.208$ and AP: $r = -0.168$) (all P < 0.001). In multivariate regression models adjusting for age and risk factors, TT was an independent predictor of central pressures and wave reflection indices (all P < 0.001).

The combination of low TT level (<4.0 ng/ml) with higher central pulse pressure (>40 mmHg) and AIx (>27%) values showed greater effect on 10-year risk of a CV event (figure).

Conclusions: Our study is the first, to the best of our knowledge, to demonstrate in ED patients the independent association of low testosterone with central pressures and indices of pulse wave amplification across the arterial tree. This observation highlights the role of testosterone as a marker of arterial disease and predictor of CV events and imply a pathophysiological contribution of testosterone deficiency to age and blood pressure-related processes associated with generalized arterial disease.