07.02: INFLAMMATION AND ARTERIAL FUNCTION

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stiffness remained significant [1.22 (1.02-1.47)] whereas estimates of pulse pressure were slightly decreased [1.13 (0.93-1.37)].

Conclusions: Aortic stiffness is an independent predictor of coronary heart disease in apparently healthy subjects.

04.04 AMBULATORY ARTERIAL STIFFNESS INDEX (AASI) PREDICTS STROKE IN A GENERAL POPULATION


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Background: The ambulatory arterial stiffness index (AASI), defined as one minus the regression slope of diastolic on systolic blood pressure in individual subjects, can be computed from 24-h ambulatory blood pressure recordings and predicted stroke in a large cohort of referred patients.

Methods: We investigated the prognostic value of AASI and 24-h pulse pressure (PP) in a sex- and age-stratified random sample of 1829 Danes, aged 40-70 years. We used Cox regression to adjust for sex, age, body mass index, mean arterial pressure, smoking, diabetes mellitus, and a history of cardiovascular disease. We also adjusted AASI for PP and vice versa.

Results: Over a median follow-up of 9.4 years, the incidence of fatal and nonfatal endpoints amounted to 40 for stroke, 150 for coronary heart disease, and 212 for cardiovascular events. In fully adjusted models, the relative hazard ratios associated with a 1 SD increase (0.14 units) in AASI were 1.61 (95% confidence interval, 1.14 to 2.27; P = 0.007) for stroke, 0.94 (0.78 to 1.12; P = 0.46) for coronary heart disease, and 1.04 (0.89 to 1.20; P = 0.64) for cardiovascular events. For PP, none of the fully adjusted ratios reached significance (P >0.45). AASI still predicted stroke after excluding subjects with previous cardiovascular disease or after adjustment for systolic blood pressure instead of mean arterial pressure.

Conclusions: In middle-aged and older individuals randomly recruited from a European population, AASI was a strong predictor of stroke over and beyond traditional cardiovascular risk factors, including mean arterial pressure and PP.

07.01 REDUCING ARTERIAL STIFFNESS AND WAVE REFLECTION – QUEST FOR THE HOLY GRAIL?

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Arterial stiffness and wave reflection are fast emerging as therapeutic targets in their own right. While thiazide diuretics have little or no effect on either arterial stiffness or wave reflection, vasodilators including nitrates and phosphodiesterase type-5 inhibitors e.g., sildenafil, reduce wave reflections and aortic pressures but not aortic stiffness. ß-blockers have the opposite effect; they reduce aortic stiffness but increase aortic pulse pressure and wave reflections while calcium antagonists and ß-blockers vary with effects on the vascular wall. Drugs targeting the renin-angiotensin-aldosterone system, namely angiotensin-converting enzyme inhibitors (ACEI), angiotensin receptor blockers (ARBs) and aldosterone antagonists have been shown as the most effective in reducing both arterial stiffness and wave reflection, and in some cases, to a greater extent than predicted from the extent of blood pressure (BP) reduction. Also, there is evidence of an additive effect on arterial stiffness with combined ACEI and ARBs. Exploring further the synergistic effects of anti-hypertensive drugs on arterial stiffness, a polymorph containing a low-dose combination of a thiazide diuretic, calcium antagonist, ß-blocker and an ACEI, decreased arterial stiffness more than the individual drugs in standard doses. However, beyond the dynamic effects of anti-hypertensive drugs, future therapies may directly target vascular structural alterations including collagen degradation, advanced glycation end-products, the matrix metalloproteinases and vascular inflammation. Finally, one can speculate about the role of pharmacogenomics which may help tailor “de-stiffening therapy” in individuals with stiff arteries.