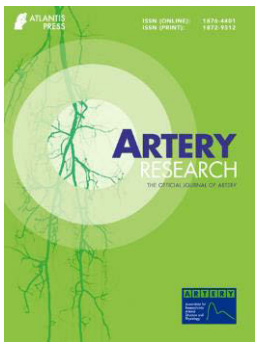

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04.04: AMBULATORY ARTERIAL STIFFNESS INDEX (AASI) PREDICTS STROKE IN A GENERAL POPULATION

T.W. Hansen*, J.A. Staessen, C. Torp-Pedersen, S. Rasmussen, Y. Li, E. Dolan, L. Thijs, J.G. Wang, E. O'Brien, H. Ibsen, J. Jeppesen

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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

T.W. Hansen[1]*, J.A. Staessen[2], C. Torp-Pedersen[3], S. Rasmussen[7], Y. Li[1], E. Dolan[6], L. Thijs[2], J.G. Wang[5], E. O'Brien[6], H. Ibsen[4], J. Jeppesen[4]. *[1]Research Center for Prevention and Health, Copenhagen, Denmark, [2]Studies Coordinating Centre, Division of Hypertension and Cardiovascular Rehabilitation, Department of Cardiovascular Diseases University of Leuven, Leuven, Belgium, [3]Department of Cardiology, Bispebjerg University Hospital, Copenhagen, Denmark, [4]Medical Department M, Glostrup University Hospital, Copenhagen, Denmark, [5]Centre for Epidemiological Studies and Clinical Trials, Ruijin Hospital, Shanghai Institute of Hypertension, Shanghai Second Medical University, Shanghai, China, [6]ADAPT Centre, Beaumont Hospital, and Department of Clinical Pharmacology, Royal College of Surgeons in Ireland, Dublin, Ireland, [7]Department of Clinical Physiology and Nuclear Medicine, Frederiksberg University Hospital, Frederiksberg, Denmark*

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 α-blockers show varying 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 polypill 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.

07.02
INFLAMMATION AND ARTERIAL FUNCTION

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During the last decade, several studies have documented the unfavorable effects of inflammation on cardiovascular function and its role in the pathophysiology of atherosclerotic disease. The interplay between inflammation and arterial system is multifaceted. On the one hand, the arterial endothelium contributes to the initiation and the perpetuation of inflammation. On the other hand, the inflammatory cascade affects adversely the endothelium-dependent processes and the mechanical properties of the arteries. These effects give rise to impaired vasomotion, arterial stiffening and increased wave reflections and thus result in an unfavorable hemodynamic loading of the heart. Chronic inflammatory diseases (such as rheumatoid arthritis, and others) as well as acute inflammatory stimuli (such as acute infections) may adversely influence the arterial performance. Moreover, systemic subclinical low-grade inflammation, as expressed by high blood levels of inflammatory markers/mediators, is a common denominator of most cardiovascular risk factors (hypertension, diabetes, etc.) and importantly, it is closely related to impaired arterial elastic properties. In addition, vasculogenic erectile dysfunction, which comprises an alternative phenotype of arterial dysfunction and an emerging cardiovascular risk predictor, is accompanied by low-grade inflammatory activation. Among the several inflammatory markers/mediators, C-reactive protein level has been consistently associated with indices of arterial function in several populations. However, data regarding a possible direct etiological role of CRP in arterial dysfunction and atherosclerosis, if any, are yet inconclusive. Current evidence suggests that anti-inflammatory strategies benefit arterial function in several clinical settings. Further research is needed to elucidate whether inflammation may comprise a worthwhile treatment target regarding the cardiovascular system.

07.03
SODIUM EXCRETION AS A MODULATOR OF GENETIC INFLUENCE ON ARTERIAL STIFFNESS AND OTHER CARDIOVASCULAR PHENOTYPES

K. Stolarz[1]*, W. Wojciechowska[1], T. Kuznetsova[2,5], K. Kawecka-Jaszcz[1], S. Babeanu[3], E. Casiglia, J. Filipovský[4], J. Peleška, Y. Nikitin[5], J.A. Staessen[2]. *On behalf of the European Project On Genes in Hypertension (EPOGH) Investigators. [1]First Cardiac Department, Medical College, Jagiellonian University, Cracow, Poland, [2]Study Coordinating Centre, Hypertension and Cardiovascular Rehabilitation Unit, Department of Molecular and Cardiovascular Research, University of Leuven, Leuven, Belgium, [3]San Luca Hospital, Bucharest, Romania, [4]Charles University, Pilsen, Czech Republic (J.F.); General Faculty Hospital, Prague, Czech Republic, [5]Institute of Internal Medicine, Novosibirsk, Russian Federation*

Hypertension is a chronic age-related disorder, affecting nearly 20% of all adult Europeans. This disease entails debilitating cardiovascular complications and is the leading cause for drug prescriptions in Europeans older than 50 years. Intensive research over the past two decades has so far failed to identify common genetic polymorphisms with a major impact on blood pressure or associated cardiovascular phenotypes, suggesting that multiple genes each with a minor impact, along with gene–gene and gene–environment interactions, play a role. The European Project on Genes in Hypertension (EPOGH) is a large-scale, family-based study in which participants from seven different populations were phenotyped and genotyped according to standardized procedures. The EPOGH demonstrated that phenotype–genotype relations strongly depend on host factors such as gender and lifestyle, in particular salt intake as reflected by the 24-h urinary excretion of sodium. Individuals with the same genetic predisposition had different vascular stiffness, left ventricular mass or heart rate variability, depending on whether they ate a high-sodium or a low-sodium diet. The EPOGH therefore highlights the concept that phenotype–genotype relations can only be studied within a defined ecogenetic context.

Free Communications (Young Investigators)

09.01
EZETIMIBE AND SIMVASTATIN BOTH REDUCE INFLAMMATION, DISEASE ACTIVITY, AORTIC STIFFNESS AND IMPROVE ENDOTHELIAL FUNCTION IN RHEUMATOID ARTHRITIS

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Background and Aims: HMG-CoA reductase inhibitors (statins) have been shown to have anti-inflammatory and disease modifying properties in patients