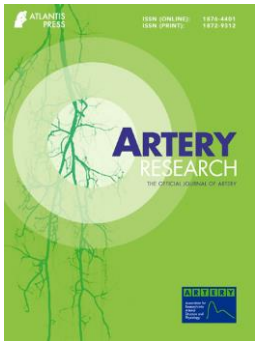

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CD2: A MULTIPHYSICS COMPUTER MODELING FRAMEWORK IN SUPPORT OF THE QUEST FOR RELIABLE AND ROBUST LOCAL ARTERIAL STIFFNESS ASSESSMENT

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ARTERY 2014 Abstracts

Invited Lecture

PULSATILE HEMODYNAMICS AND THE MICROCIRCULATION (IN ASSOCIATION WITH THE EUROPEAN SOCIETY OF HYPERTENSION WORKING GROUP ON VASCULAR STRUCTURE AND FUNCTION)

E. Agabiti-Rosei
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Macrovasculature, microvasculature, and the heart determine the structure and function of the circulatory system. Due to the viscoelastic properties of large arteries, the pulsatile pressure and flow that result from intermittent ventricular ejection is smoothed out, so that microvasculature mediates steadily the delivery of nutrients and oxygen to tissues. The disruption of this function, which occurs when microvascular structural alterations develop in response to hypertension, leads to end-organ damage. Microvascular structure is not only the site of vascular resistance but also the origin of most of the wave reflections that generate an increased central systolic blood pressure. The presence of structural alterations in the small resistance arteries may be considered an important link between hypertension and ischemic heart disease, heart failure, cerebral ischemic attacks and renal failure. An increased arterial wall thickness together with a reduced lumen may play an important role in the increase of vascular resistance, and may also be an adaptive response to the increased haemodynamic load. The increased media to lumen ratio is also a powerful predictor of subsequent cardiovascular events. In addition, essential hypertension seems to be associated with a rarefaction of arterioles and capillaries. Nowadays many data of the literature suggest that hypertension-related damage to the micro and macrovascular system may be manageable through pharmacological agents. Among them, beta-blocking agents and diuretics have never modified the microvascular structure, whereas renin-angiotensin system antagonists and calcium entry blockers had an opposite effect being able of reversing structural alterations, thereby reducing central wave reflections and, finally, causing a selective systolic blood pressure reduction.

Career Development Lecture

CD1
ARTERIAL HEMODYNAMICS IN AGING POPULATIONS

G. C. Verwoert
Erasmus MC, Rotterdam, The Netherlands

Cardiovascular disease is the number one leading cause of morbidity and mortality worldwide. A large proportion of cardiovascular diseases can be prevented by addressing risk factors and early assessment of target organ damage. The leading cardiovascular risk factor is raised blood pressure, however this relationship is more complex than only the two extremes, systolic and diastolic blood pressure. The pulsatile component of blood pressure plays a role in predicting cardiovascular disease.
The association between arterial stiffness and hypertension is of interest, because the functional relationship is likely bidirectional and can be best described as feed forward in vicious cycle. We confirmed the association of arterial stiffness with isolated systolic hypertension (ISH) and showed that subjects with ISH have a stiffer aorta compared to normotensive subjects and subjects with combined systolic and diastolic hypertension. This difference was most pronounced at older age.
Heart failure is a growing health problem in the aging population. To improve treatment of heart failure, the mechanisms regarding development of heart failure should be unraveled. We confirmed the relation between SBP and heart failure and demonstrated that both pulse pressure and aPWV are associated with the development of heart failure.
Population-wide primary prevention and individual health-care intervention strategies for cardiovascular disease have contributed to declining mortality trends. If people at risk for developing cardiovascular disease can be identified and measures taken to reduce their cardiovascular risk, a vast majority of fatal and non/fatal cardiovascular events can be prevented. We have added aortic stiffness to the Framingham risk factors and determined if the risk classification for CHD improved. However, the addition of aortic stiffness led to minor reclassification of subjects within 10-year cardiovascular disease risk categories, suggesting low additional value of aortic stiffness in the clinical management of CHD in the elderly.

Career Development Lecture

CD2
A MULTIPHYSICS COMPUTER MODELING FRAMEWORK IN SUPPORT OF THE QUEST FOR RELIABLE AND ROBUST LOCAL ARTERIAL STIFFNESS ASSESSMENT

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Arterial stiffness has proven to be a powerful, early marker of cardiovascular diseases, with most clinical data relying on carotid-femoral pulse wave velocity (PWV) measurements, a rather global assessment of arterial stiffness. Direct, local evaluation of carotid stiffness is clinically useful, but remains technically more challenging. Hence, we have been investigating the performance of such local strategies, both from a biomechanical and image acquisition perspective. In particular, the PU-loop method (and its derived techniques) as well as ultrasonic tissue characterization techniques have been under consideration. In the former approach, PWV is derived from the slope of the blood pressure (P) versus velocity (U) signal in early systole. The latter refers to our investigation of shear wave elastography, assessing tissue stiffness by tracking shear waves artificially evoked in the tissue via the acoustic energy of an ultrasound probe.
However, previously mentioned measurement strategies are hampered in the presence of intricate vascular anatomy or tissue mechanics, inducing complex pulse/shear wave phenomena, erroneously affecting stiffness assessment. Hence, we developed a computer modeling platform for in-depth investigation and validation of these measurement strategies, allowing comparison of the simulated measurement outcome with the true tissue properties, fully defined in the simulation but typically lacking during in-vitro/in-vivo evaluation. Hence, this is a multi-physics model, integrating both the biomechanics and imaging, which has allowed us to

1872-9312/$ — see front matter

analyze arterial stiffness assessment techniques in varying biomechanical conditions as well as to investigate new imaging approaches and signal processing.

Career Development Lecture

CD3
DIABETES CONFERS A GREATER EXCESS RISK CARDIOVASCULAR DISEASE IN WOMEN THAN IN MEN: CURRENT EVIDENCE, POTENTIAL MECHANISMS, CLINICAL IMPLICATIONS, AND FUTURE DIRECTIONS

S. Peters
University of Oxford, UK

A lack of sex-specific data from early epidemiological studies has typically led to the assumption that the associations between risk factors and disease outcomes are equivalent in women as in men. But increasingly, evidence to support the existence of clinically meaningful sex differences in the relationships between certain risk factors, such as smoking and diabetes with chronic disease is becoming apparent — often to the detriment of women. Determining reliably whether there are clinically meaningful sex-differences in risk factor-disease associations is important, not solely to better understand the aetiology of CVD, but also from a population and public health vantage. Current estimates of the burden of CVD, that are used to inform public health policy, assume that these risk factors effect cardiovascular risk similarly in women as in men. However, if this assumption is proven to be invalid then it would necessitate the revision of the estimates to more accurately reflect the true nature of the relationships in women and men. Moreover, just as possible racial differences in the relationships between risk factors and diseases are considered when tailoring specific interventions for different communities, so could information on important sex differences be used to provide an added impetus for targeted interventions aimed at the treatment and management of these risk factors in both sexes.
Recently we have conducted two systematic reviews and meta-analyses of the sex-specific effects of diabetes on risk of stroke and coronary heart disease. Both studies suggested that women with diabetes had a substantially greater excess risk compared with their male equivalents, even after consideration of differences in baseline levels of other major risk factors. In this talk, the results from these studies will be presented, potential biological, behavioral, or social mechanisms involved will be discussed, clinical implications will be considered, and directions for future research will be provided.

Invited Lecture

CEREBRAL CIRCULATION & COGNITION IN THE ELDERLY

G. J. Biessels
University Medical Center, Utrecht, The Netherlands

Vascular disease is an important cause of cognitive decline and dementia. Approximately 20% of patients with a stroke develop dementia within a year after the event. Even more often, cerebrovascular disease is involved in cognitive decline or dementia in people without an obvious history of stroke. Autopsy studies identify vascular pathology in the majority of patients with dementia, also in those with a clinical diagnosis of Alzheimer's disease. The concept "Vascular cognitive impairment" (VCI) has been introduced as an umbrella term to capture all forms of cognitive impairment — regardless of severity or cognitive profile - that are associated with and presumably caused by cerebrovascular disease. Hence, VCI is heterogeneous, both with regard to aetiology and course of development, ranging, for example, from an acute strategic brain infarct to insidious diffuse white matter pathology.
Importantly, causes of VCI may not be restricted to vascular brain lesions. Abnormalities in vascular function, resulting in altered cerebral haemodynamics, may also be involved, and might represent a potentially modifiable cause of cognitive dysfunction. Currently, the role of haemodynamics in VCI is addressed in a Dutch multidisciplinary research program, called "The Heart-Brain connection". This program addresses the following questions. 1) To what extent do hemodynamic changes contribute to VCI? 2) What are the mechanisms involved? 3) Does improvement of the hemodynamic status lead to improvement of cognitive dysfunction?
In my presentation I will provide background on VCI, review evidence for links between haemodynamics and cognition and introduce the research program of the Heart-Brain connection consortium.

Invited Lecture

MOLECULAR MECHANISMS OF ARTERIAL STIFFENING: ROLE OF VASCULAR SMOOTH MUSCLE CELLS

P. Lacolley
Vandoeuvre-les-nancy, France

Vascular Smooth Muscle Cells (VSMCs) are the stromal cells of the vascular wall, continually exposed to mechanical signals and biochemical components generated in the blood compartment. They are involved in all the physiological functions and the pathological changes responsible for arterial stiffening. Due to their contractile tonus, VSMCs of resistance vessels participate in the regulation of blood pressure and also in arterial stiffness. VSMCs of conduit arteries respond to hypertension-induced increases in wall stress by an increase in cell protein synthesis and extracellular matrix secretion. These responses are mediated by complex signaling pathways, mainly involving RhoA and extracellular signal-regulated kinase1/2. Serum response factor and miRNA expression represent main mechanisms controlling the pattern of gene expression. A progressive decrease in plasticity and reprogramming potential of VSMCs plays a complementary role contributing to the increase in arterial stiffness and associated cardiovascular risk factors in old humans. These key signaling pathways have become the focus of modern aging research and will undoubtedly provide a rich resource for the development of selective drugs interfering with either of these processes and prevention of the number one cause of death in the modern world.

Invited Lecture

MODELLING THE ARTERIAL SYSTEM: BIOMECHANICS OF CENTRAL ARTERY STIFFENING

J. D. Humphrey
Yale University, New Haven, CT, USA

Central arterial stiffening is both an indicator and an initiator of cardiovascular disease, and aging is a ubiquitous cause of stiffening. In this talk, we will discuss the utility of biomechanical models in understanding particular effects of arterial stiffening on systemic hemodynamics and we will discuss advantages of using mouse models to obtain detailed longitudinal information on regional variations in arterial wall properties. In particular, we will focus on delineating intrinsic material and structural stiffness as a function of location along the aorta and we will show results from 3-D computational simulations of the hemodynamics that account for interactions between the blood and regional wall properties. Amongst the different findings, one emerging concept is that adaptive responses appear to favor the maintenance of material stiffness near normal values while offsetting increased hemodynamic loads or genetic defects with changes in structural stiffness. If true, such a response would have important mechanobiological implications.
Related Articles
Bersi MR, Ferruzzi J, Eberth JF, Gleason RL, Humphrey JD (2014) Consistent biomechanical phenotyping of common carotid arteries from seven different genetic, pharmacological, and surgical mouse models. Annl Biomed Engr 42: 1207-1223.
Roccabianca S, Figueroa CA, Tellides G, Humphrey JD (2014) Quantification of regional differences in aortic stiffness in the aging human aorta. J Biomech Behavior Biomed Matl 29: 618-634.

Invited Lecture

THE DESIGN OF AN OPTIMAL ARTERIAL NETWORK

A. D. Hughes
University College London

The arterial circulation is a network that delivers oxygen and nutrients to cells. Blood flow is achieved by means of a muscular pump but diffusion plays a key role at a cellular level necessitating a branching structure where no cell is more than ~25microm from a capillary.[1]
The design of the cardiovascular system is subject to a variety of 'constraints' and 'costs'. It has been postulated that the design of the arterial network might be understood in terms of the need to minimize competing 'costs' within the context of physical or material limits to the system.[2] These designs can also be envisaged as being subservient to space filling or fractal considerations.[3] The signalling mechanisms underlying these