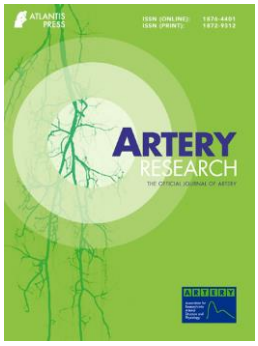

**Artery Research**

**ISSN (Online): 1876-4401** **ISSN (Print): 1872-9312**
**Journal Home Page: https://www.atlantis-press.com/journals/artres**

# CEREBRAL CIRCULATION & COGNITION IN THE ELDERLY

G.J. Biessels

**To cite this article**: G.J. Biessels (2014) CEREBRAL CIRCULATION & COGNITION IN THE ELDERLY, Artery Research 8:4, 120–120, DOI: https://doi.org/10.1016/j.artres.2014.09.042

**To link to this article**: https://doi.org/10.1016/j.artres.2014.09.042

Published online: 7 December 2019

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