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

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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)

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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

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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