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VENTRICULAR-ARTERIAL COUPLING: INVASIVE AND NON-INVASIVE ASSESSMENT

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Invited Speaker Abstracts

Opening lecture

ARTERIAL WALL MODELING: STATE OF THE ART AND POTENTIAL CLINICAL IMPACT

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Mechanics regulates biological processes at the molecular, cellular, tissue, organ and organism levels. Biomechanics has the goal to better explain phenomena in bioengineering, biology, chemistry and medicine, and hence to improve, for example, diagnostic methods, therapeutic interventions, medical devices and tissue engineering.

By means of examples we will show and discuss the importance of biomechanics in quantifying the mechanical environment within arterial walls in health, disease or injury. We also emphasize the interdisciplinary nature of such analysis embracing bioengineering, biology, chemistry and medicine, and the importance of connecting arterial mechanics with biological processes such as growth, remodeling, adaptation and repair. Without undue detail, we discuss important mechanobiological aspects for modeling arterial walls, allude to challenges in modeling pathologies such as atherosclerosis and aneurysms, and highlight the potential clinical impact of using patient-specific modeling.

In particular, computational (multi-scale) models may lead to better understanding of the function of arteries by synthesizing medical images, powerful computers, experimental data and mechanics; they may eventually allow doctors to use computers, together with a patient's medical data, to generate and analyze "virtual arteries" and help identify the best treatment strategy, for example, to use an optimal stent for a particular patient's lesion.

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FOCUS: Ventricular – vascular coupling

VENTRICULAR-ARTERIAL COUPLING: INVASIVE AND NON-INVASIVE ASSESSMENT

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The interaction between the left ventricle (LV) and the arterial system, (ventricular–arterial coupling, V-AC) is a key determinant of cardiovascular function. V-AC is most frequently assessed in the pressure–volume plane using the ratio of effective arterial elastance (Ea) to left ventricular (LV) end-systolic elastance (Ees). Ea (a composite index of arterial load) is computed as end-systolic pressure/stroke volume, whereas Ees (a load-independent

measure of LV chamber performance) is ideally assessed invasively using data from a family of pressure–volume loops obtained during an acute preload alteration. Single-beat methods have also been proposed, allowing for non-invasive estimations of Ees using simple echocardiographic measurements. The Ea/Ees ratio is very useful because it provides information regarding operating energetic and mechanical efficiency of the ventricular–arterial system. However, it should be recognized that V-AC encompasses many different physiologic aspects, many of which are not captured in the pressure–volume plane. Therefore, additional assessments can provide important incremental physiologic information about the cardiovascular system and should be more widely used. In particular, it should be recognized that: (1) Comprehensive analyses of arterial load are important because Ea incompletely characterizes (and does not depend exclusively on) arterial properties; (2) The myocardial (systolic) loading sequence is important for V-AC and can profoundly impact LV function, remodeling and progression to heart failure; (3) LV time-varying LV elastance does not equal time-varying myocardial wall stress, which better addresses the myocardial loading sequence; (4) When a stenotic aortic valve is present, special considerations are required to assess V-AC and the combined valvular–arterial load.

ARTERIAL STIFFNESS AND DIASTOLIC HEART FAILURE

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Diastolic heart failure (or heart failure with preserved ejection fraction – HFPEF, which is the contemporary denomination of the condition) and increased arterial stiffness/wave reflections are closely linked from an epidemiological perspective. Both are usually found in elderly patients, with long-standing hypertension, with a predominance of female gender. Consecutive cross-sectional studies indeed established the presence of increased arterial stiffness/wave reflections in individuals with diastolic dysfunction and in HFPEF-patients. More mechanistic trials showed that key characteristics of diastolic dysfunction (invasively or non-invasively derived left ventricular filling pressures, natriuretic peptides, tissue-Doppler measurements of relaxation) are closely related to estimates of arterial stiffness (Pulse Wave Velocity) and wave reflections (Pressure Augmentation, Augmentation Index, backward wave amplitude). These findings are supported by previous experimental studies, proving that an increase in late-systolic load can impair diastolic function. As HFPEF often manifests as exertional dyspnea, measures of arterial stiffness may be relatively normal at rest, but rise inadequately during exercise, and exactly this phenomenon has been shown in HFPEF patients. With respect to treatment of HFPEF, we are currently facing a serious lack of evidence-based recommendations. Studies using angiotensin-receptor antagonists have largely failed, but the presence of the condition (HFPEF) in many of the patients included in the largest trial performed so far has been questioned. Interestingly, the extent of improvement of diastolic function has been directly linked to the extent of blood pressure fall with treatment. Recently, a study investigating the effects of spironolactone, a drug with proven beneficial effects on pulsatile hemodynamics, has shown promising results in HFPEF. Furthermore, interventional treatment (renal sympathetic denervation) of patients with resistant hypertension has resulted in improvements in diastolic function as well as arterial stiffness and wave reflections.