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# THE CLINICAL IMPORTANCE OF EXERCISE BLOOD PRESSURE

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## ARTERY 17 – Invited Speaker abstracts

### Career development lectures

#### THE CLINICAL IMPORTANCE OF EXERCISE BLOOD PRESSURE

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Clinical exercise stress testing is a common medical test performed in cardiology and exercise physiology clinics the world over. Measurement of blood pressure (BP) during testing is mandated. Whilst systolic BP should normally rise with incremental exercise, and diastolic BP remains relatively stable, abnormal responses can occur. Low BP or 'exercise hypotension' is a known signal of underlying cardiovascular disease and sign of poor prognosis. On the other hand, observational evidence suggests an exaggerated BP response is also associated with heightened cardiovascular disease risk. Historically, research has focused on the BP response to peak or maximum exercise intensities. However, exaggerated BP during submaximal exercise (light-to-moderate intensity) may expose the presence of high BP otherwise not detected by traditional resting measurement in the clinic. Exaggerated exercise BP is related to subclinical cardiovascular disease risk markers such as raised arterial stiffness and impaired cardiac structure and function. The mechanisms underlying such associations are complex, but physiological insight has been gained from studying changes in arterial haemodynamics in response to dynamic exercise. Similarly, there are several known modifiers of the exercise BP response, including age, disease status and aerobic capacity. An area of continued focus is to establish if modifiers, such as aerobic capacity, also modify associations between exercise BP and clinical outcomes throughout the life-course. Future work is also directed towards filling a crucial evidence gap, providing population-based thresholds of exercise BP that are associated with acute and longer-term outcomes. This should pave the way for pragmatic research aimed towards enhancing the clinical use of exercise BP.

#### THE CENTRALITY OF THE HEART IN ITS RELATIONSHIP WITH THE ARTERIAL SYSTEM: WHERE IT ALL STARTS AND ENDS

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The interaction between the heart and large arteries represent a not fully understood process, which results from the integration of pressure wave generation, flow ejection, wave travel and reflection, and geometric mismatch between structures. Left ventricular (LV) mass and geometry, two independent predictors of worse cardiac outcomes, are modelled by changes in cardiac afterload. Central pulse pressure (cPP) itself, a marker of LV afterload, is generated from heart contraction and pressure/flow propagation into the arterial system. As such, increased cPP may be viewed as both the cause and the consequence of an increased LV work. Our research group first described an inverse, age-dependent, relationship between aortic PWV, relative wall thickness, and the velocity of LV circumferential fiber shortening. We also showed that the relationship

between cPP and stroke volume is progressively lost at increasing age. Finally, we found, in a cohort of untreated hypertensives, a pressure-independent relationship between aortic characteristic impedance, LV mass and geometry. Taken together, these results suggest that changes in LV structure and function occur as part of the compensatory response of the LV to unfavourable ventricular-vascular coupling and increased aortic stiffness.

Heart rate (HR) is another relevant effect-modifier of the interconnection between cardiac and arterial function. HR is independently associated with cardiac index, PP amplification, and aortic stiffness. Pharmacological BP-lowering interventions combined with HR-lowering effect, such as beta-blockers, are ineffective in reducing LV mass. Moreover, HR changes are positively related to DBP changes. Therefore, anti-hypertensive drugs with associated HR-lowering effect only spuriously affect the ventricular-vascular coupling. More research is needed in order to find new targets of treatment able to prevent the occurrence of structural LV remodeling.

#### STIFF VESSELS APPROACHED IN A FLEXIBLE WAY: ADVANCING QUANTIFICATION AND INTERPRETATION OF ARTERIAL STIFFNESS

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##### Introduction

Although pulse wave velocity (PWV), a proxy of arterial stiffness, is a strong predictor of cardiovascular complications, it is confounded by blood pressure (BP) and heart rate at the time of examination. Furthermore, establishing whether an artery is stiffened or not does not inform a clinician on the *cause* of the stiffening.

##### Quantification of PWV's confounders

This talk will focus on BP as a confounder of PWV. We developed a method to patient-specifically determine the dependence of PWV on BP—on average 1 m/s per 10 mmHg diastolic BP—and used it to disentangle BP-dependent and -independent stiffening in hypertension and cancer patients. We furthermore showed that cardio-ankle vascular index (CAVI)—a measure that is often presented as BP-independent—shows a residual BP dependence that can be readily corrected using a modified equation (CAVI<sub>0</sub>). Both developed methods are directly applicable to clinical measurement data from individual patients.

##### Interpretation of changes in PWV

We developed a computer modelling procedure to disentangle contributions of the individual wall components—collagen, elastin, and smooth muscle—to arterial stiffening in patient data. We used this approach to show that with ageing, the biomechanical phenotype shifted from elastin-dominated to collagen-dominated load bearing.

##### Outlook

Model-based interpretation of arterial mechanics provides a promising tool to further improve understanding of arterial stiffening. In my future career, I will use such methodology to study stiffening in various mouse models. The results of these studies can potentially be used to further improve understanding and interpretation of arterial stiffening in patients.