Preface

We welcome the reader to this book on different aspects of Early Vascular Aging (EVA), a concept that has attracted considerable attention since it was first described in 2008. A number of skilled authors have contributed to provide a multifaceted description of the pathophysiological and clinical aspects that are associated with EVA. Previous research has for decades described and investigated atherosclerosis, a process that starts in the intima layer of the arterial wall and becomes proximal to many cardiovascular events caused by athero-thrombotic disease. As the core component of EVA is arterial stiffness, arteriosclerosis, which is mainly influenced by morphological changes in the arterial *media* layer, but also in other layers, we have focused on different characteristics and mechanisms associated with stiffness of the large elastic arteries. We also consider EVA based on an integrated view linking the macro- with the microcirculation. This is because hemodynamic forces influenced by stiffness may also cause harm to the peripheral smaller vessels due to the increased pulsatile energy that is transmitted, for example, in the brain. Another aspect of a more integrated approach identifies important contributing factors for EVA also from the intima (endothelial dysfunction) and the adventitia (impaired function of vasa vasorum and innervation, accompanied by increased secretion of cytokines from the perivascular adipose tissue causing local inflammation) when impaired glucose metabolism could further contribute to stiffening by glycosylation. Therefore, we consider EVA to be a fruitful scientific concept to promote research on early changes of the arterial wall, programmed already *in utero* and early life and influenced by genetic and environmental factors. As metaanalyses have documented that arterial stiffness (increased aortic pulse wave velocity, aPWV) is an independent risk marker for future cardiovascular risk and total mortality, adjusted for conventional risk factors, we consider it of importance to find new ways to find, diagnose, and treat subjects with signs of EVA. Still however, neither an exact definition nor a targeted treatment exists for EVA, but several attempts are being made to find such alternatives. We therefore

invite the reader to contribute to the lively discussion on EVA with data from different populations and ethnic groups, as well as with data from basic and clinical science. This could contribute to early detection of atrisk individuals, for example, from at-risk families with early onset cardiometabolic disease, for prevention based on improved life style as well as drug therapy when needed. This is not to deny the importance of atherosclerosis and the evidence-based methods that exist to prevent cardiovascular events by control of hypertension and hyperlipidemia as well as smoking cessation, but we consider that EVA is a feature starting early in life and that later in life components of arteriosclerosis and atherosclerosis will be intertwined in further promoting cardiovascular disease risk.

In an historical perspective, the interest in arterial function and stiffness contributing to hemodynamic changes predates the clinical measurement of blood pressure and diagnosis of hypertension as we know it. In London, the physician Fredrik Akbar Mahomed carried out studies on pulse wave properties in *arteria radialis* with his own sphygmograph and published in 1877:

It is very common to meet with people apparently in good health who have no albumen in the urine, who constantly present a condition of high arterial tension when examined by the aid of the sphygmograph. [1]

We therefore date the interest in large arteries and stiffness to an era before clinical hypertension was recognized [2], and thus the EVA concept [3-5] attempts to bridge more than a century to revive the importance of large arteries and their properties in cardiovascular medicine.

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