MEASURING ARTERIAL STIFFNESS IN VIVO

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1. Introduction - clinical interest in arterial stiffness

Over the past few years, there has been an increasing interest in the role of arterial stiffness in the development of cardiovascular disease. This clinical interest in arterial stiffness is driven by studies demonstrating an added value of measures of arterial stiffness (a.o., pulse wave velocity or PWV; see further) in the clinical decision making process (Figure 1).

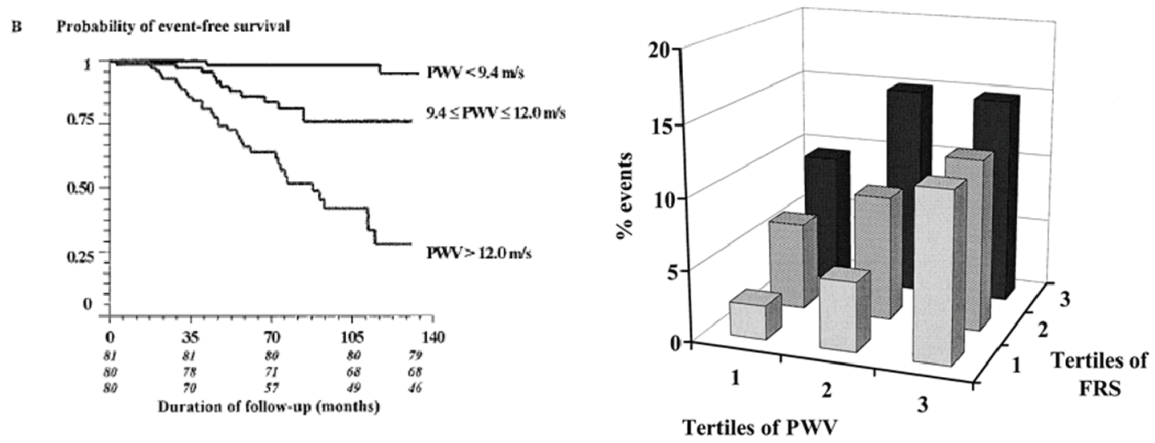

Figure 1. Left: Blacher et al. demonstrated in patients with end-stage renal disease that increased arterial stiffness is associated with lower probability of event-free survival[2]. Right: even in patients at low risk according to conventional risk scores (FRS: Framingham Risk Score), the incidence of cardiovascular events is higher in patients with high PWV according to Boutouyrie et al. [3]

The term 'arterial stiffness' can be quantified by a number of different indices, depending on the scope and area of application in which the investigator is interested[4]. Increasingly, arterial stiffness indices are included in the clinical assessment of patients, especially in large-scale clinical trials. One of these indices in particular, the measurement of carotid-femoral pulse wave velocity, has recently been recommended in the 2007 European Society of Cardiology / European Society of Hypertension guidelines for the management of hypertension in order to assess arterial damage, evaluate the level of cardiovascular risk and

adapt the therapeutic management of patients[5]. This chapter introduces the basic principles behind the most commonly reported measures of arterial stiffness.

Although the terminology is intuitively clear, quantifying “arterial stiffness" is not as straightforward as it seems, and there are many different ways to express this property. One way to classify the different methods is according to whether they quantify (i) the local elastic properties of a given arterial segment; (ii) the elastic properties of a segment, or (iii) the global, integrated buffer capacity of the complete arterial system. We will use this classification in this chapter.

Anyhow, irrespective how arterial stiffness is quantified, it is clear that the mechanical properties of the arterial wall are related to its composition and structure, which will be briefly discussed in the next subsection.

2. Composition of the arterial wall

On a macrostructure level, all arteries (or even more general, all blood vessels, except for the true capillaries) can be seen as being composed of 3 layers: the intima (inner layer), the media (middle layer) and adventitia (outer layer) (Figure 2). On a histological coupe, the media layer is bounded by the internal and external elastic lamina. On microstructure level, these layers are formed and composed of different constituents, the most important ones being:

- Endothelium: this is a continuous monolayer of cells (0,2-0,5 μm thick) which forms the inner layer of the blood vessel and hence the barrier between the blood and the arterial wall. The endothelium is involved in regulating mass-transport processes, arterial remodeling processes, … It is considered an important mechanosensor (sensing the shear stress exerted by the blood flow) and mediator of many biological processes (arterial diameter regulation, NO, …). Endothelial damage or dysfunction is considered as an important key process in the cascade of events leading to atherosclerosis.
- Collagen: this is a protein with a high stiffness (estimated Young E-modulus of about 10 MPa [6]) determining the strength of the blood vessel. Different types of collagen exist, with collagen type I and III found in arteries.