The Arterial Load and Its Role on the Heart

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The article by Zamani et al.1 shows a clear association between the major systemic arterial parameters vascular resistance (SVR) and total arterial compliance (TAC, inverse of arterial stiffness) and left ventricular (LV) mass and LV wall/LV cavity volume ratio (here called remodeling, for short). The forward and backward components of the central pressure (Pf and Pb) also show associations with cardiac remodeling.

Otto Frank suggested the Windkessel model as a description of the entire arterial system. The 2 Windkessel parameters are SVR (mainly in the microvasculature) and TAC (in the conduit arteries, mainly the aorta). In the mid-1960s input impedance could be determined as a comprehensive description of the arterial system. However, input impedance is difficult to derive and grasp and the contributions of arterial properties such as SVR and TAC are not easily distinguished. Nevertheless, we learned from impedance information that Frank’s Windkessel closely approximates impedance when the so-called characteristic impedance (Zc, equal to ascending aortic pulse wave velocity divided by aortic area) is added as a third element.2 The pressure–flow relationship of Frank’s Windkessel falls short in systole and the addition of Zc resolves this. Thus, the arterial system, although being a complicated tube system with traveling waves of pressures and flows, can be described in terms of SVR and TAC, with preferably Zc added.

Recent technical developments have made it possible to obtain quantitative information on SVR and TAC noninvasively.3 For their determination the mean, systolic and diastolic pressures were derived. Cardiac MRI was used to derive LV wall, lumen volumes, and stroke volume (the difference between diastolic and systolic lumen volumes), and aortic flow (cardiac output) was calculated from stroke volume and heart rate as cardiac output = stroke volume × heart rate. These data are sufficient to derive SVR and TAC. Mean pressure/cardiac output gives SVR, and stroke volume/pulse pressure is an accepted measure of TAC. Both were then associated with LV mass and LV wall/LV cavity volume ratio, here together called remodeling.

The importance of Zamani’s article1 lies in the finding that the (purely) arterial parameters associate with ventricular remodeling, which suggests that treatment of small and large artery properties may reduce or prevent cardiac remodeling.

A limitation of the article is that an association of characteristic impedance with ventricular remodeling is not reported, although it has been shown that Zc contributes to pressure in systole and thus to ventricular loading.4

Of course, important questions remain. First, what mechanisms cause the changes in the resistance arteries, that is, an increase in SVR and what changes the conduit arteries, that is, a decrease in TAC? Knowledge about these mechanisms is required to develop targeted therapies to decrease SVR and increase TAC. Although reduction of SVR by peripheral vasodilators is generally applied, treatments to increase large artery compliance are still limited.5 Second, how do changes in TAC and SVR affect the heart? The authors, rightfully, assume this to be through (systolic) pressure and the resulting (myocardial) wall stress. Whether it is mean or maximum systolic wall stress remains a point in discussion.6

This is where (pressure) waves come in. The measured pressure wave was separated in its forward and backward components. In the article by Zamani et al.,1 this was done by assuming a standard flow wave shape and a generally accepted wave separation calculation.7 The pressure waves can give information on the timing of maximal pressure load, and, via wall and lumen volumes (Laplace’s law) myocardial stress can be derived. The maximal loading in terms of wall stress may differ from maximal pressure loading8 because the ejecting ventricle decreases in volume and increases in wall thickness. As a result, maximal stress is highest at the moment of maximal ejection, whereas maximal pressure is not.

The backward pressure wave was shown to have the strongest relationship with LV remodeling, but it affects mid-systolic loading in terms of pressure, not necessarily early maximal wall stress. This may suggest that the maximal loading stress is not the major determinant of remodeling.

However, it should be realized that pressure (and flow) waves, as well as their forward and backward components, are the result of heart-load interaction. Thus, Pf and Pb cannot give information on the contribution of the arterial system to cardiac morphology and function per se. The contribution of the cardiac pump to pressure waves has been reported before and depends on cardiac function.8 Thus, changes in LV pump function, even if the arterial system would be unaltered, affect

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the measured pressure (and flow) including their forward and backward components. Therefore, the finding that the Pf and Pb associate with ventricular remodeling results, at least in part, from the ventricular remodeling itself.

Moreover, the changes in the forward and backward pressure waves are difficult to relate to changes in the arterial system. It has been shown that the return time of the reflected wave is hardly related to PWV, and thus also hardly related to TAC.10 The so-called major reflection site was shown to be elusive.11

This, in turn, implies that targeted interventions based on forward and backward waves are out of reach.

We conclude (Figure) that the article by Zamani et al1 is important in that it shows that 2 main parameters characterizing the arterial system, systemic vascular resistance and total arterial compliance, associate and possibly contribute to ventricular remodeling. The information on left heart remodeling obtained from wave separation is limited because forward and backward pressures change by the cardiac remodeling process.

**Disclosures**

B.E. Westerhof works for Edwards Lifesciences BMEYE. The other author reports no conflicts.

**References**


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