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UNDERSTANDING THE DISEASE

Understanding arterial load

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Introduction

The heart and the arterial system are anatomically and functionally connected, although they are frequently studied as separate structures with independent functions. The arterial tree does not act solely as mere conduits for blood flow distribution to the organs but it also modulates the ventricular ejection, transforming the pulsatile stroke volume (SV) into a peripheral continuous flow (essential for metabolic exchange), and maintaining blood pressure (BP) during diastole (necessary for coronary perfusion). Therefore, a complete description of the cardiovascular system should consider not only the cardiac function but also the arterial system and how both work with each other [1, 2].

Arterial load as a measure of afterload

When trying to understand how the heart interacts with the arterial system, one must know the arterial load (AL), i.e., the external opposition that must be overcome by the ventricles during ejection [2, 3]. AL, however, does not represent any specific property of the arterial system [4]. On the contrary, it gathers all extracardiac factors opposing the movement of blood out of the heart into the systemic and pulmonary systems, compromising different arterial properties, blood viscosity, and the effects of arterial wave reflections [5]. Therefore, AL can be considered as a net measure of cardiac afterload [4].

The importance of AL can be easily understood when one considers that common pathological situations, such as heart failure or systemic and pulmonary hypertension, are profoundly related to a mismatched arterial load [6, 7]. Furthermore, well-established treatments (e.g., noninvasive mechanical ventilation for acute pulmonary edema or arterial vasodilators for systemic and pulmonary hypertension) have proven beneficial through a reduction in AL [6, 8].

Arterial load as a determinant of arterial pressure

The BP is the result of the interaction between blood flow and AL [2]. AL modulates the cardiac output in order to keep constant tisular perfusion pressure. Therefore, BP depends not only on flow but also on AL (Fig. 1). This explains why caution should be applied when using BP as a surrogate for blood flow during fluid challenge [9], since variations in AL may ultimately determine the effects of fluids on BP [10].

Similarly, variations in AL are known to affect the reliability of so-called pulse-contour methods for estimation of SV from the BP [11]. Furthermore, the ability of these techniques for tracking changes in SV depends on how AL is assessed in order to establish a valid pressure–flow relationship.

Arterial load indices

Traditionally, systemic vascular resistance (SVR) has been used to characterize the arterial system and to obtain a gross simplification of AL. However, using SVR as a descriptor of the whole arterial system is clearly inadequate. SVR represents the opposition to a steady flow, but neither blood flow and BP are constant but are cyclic, because of repeated contractions of the heart. Furthermore, the distribution of SVR is not homogenous along the vascular tree [5], and the assumption that central venous pressure as a downstream pressure in classical calculation of SVR could lead to an overestimation of the actual resistance [12].

Because of the oscillatory nature of the BP and flow, the gold standard for assessing AL is the arterial impedance (Z_{in}) [5]. Unlike SVR, Z_{in} represents the complex ratio between pulsatile pressure and flow. Z_{in} provides the most comprehensive description of the AL, integrating the mechanical properties of the arterial system and the effects of arterial wave reflections [5]. Regrettably, Z_{in}

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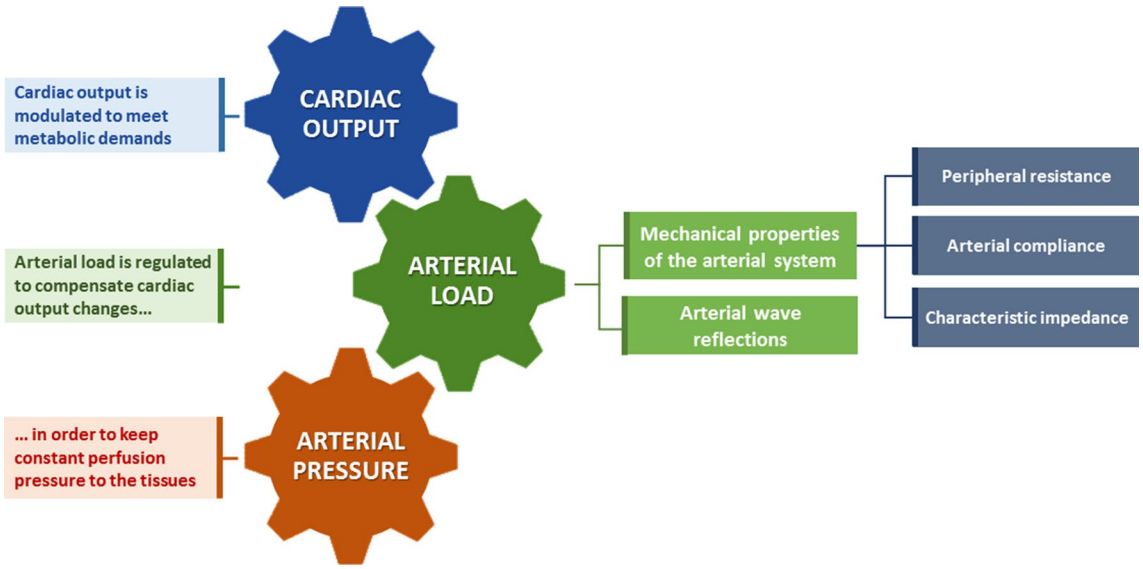

Fig. 1 Arterial load: main components and its interaction with blood flow. Arterial load represents all extracardiac factors that oppose ventricular ejection. It compromises not only mechanical properties of the arterial system, such as compliance or arterial resistance, but also the effects of arterial wave reflections. The interaction between arterial load and blood flow determines the arterial pressure

can be challenging to measure and to interpret in clinical practice, thus Z_{in} has been appropriately simplified in familiar interpretable surrogates, such as the 3-element Windkessel model of arterial system [13].

Effective arterial elastance as an index of arterial load

Back in the early 1980s, Sunagawa et al. published a landmark paper in which they proposed that all elements integrating AL can be characterized into a single variable called effective arterial elastance (Ea) [2], which is the change in pressure for a given change in volume. However, Ea is not a measure of arterial stiffness, but it incorporates the principal features of AL, including steady and pulsatile components [2, 3]. Ea is therefore an integrative measure of afterload [3].

An advantage of Ea is that it is computed as the ratio between ventricular end-systolic pressure and SV. End-systolic pressure can be estimated using 90 % of systolic BP [3, 6]. SV can be measured using current hemodynamic monitors or noninvasively by echocardiography. So, Ea can be calculated at the bedside without requiring any complex technology. The reliability of Ea for estimating Z_{in} has been repeatedly demonstrated, and its usefulness for characterizing AL has been recognized over the last three decades [3, 7, 14]. Moreover, since Ea shares the same units of ventricular elastance (E_{max}), the gold standard for estimating the ventricular contractility, both parameters can thus be related, allowing the assessment of right and left ventriculo-arterial coupling, an index of cardiovascular efficiency [2, 6, 7, 15].

Functional assessment of arterial load: the dynamic arterial elastance

The dynamic arterial elastance (Ea_{dyn}), or the ratio between pulse pressure variation and SV variation, has been suggested as a functional assessment of AL [16]. Ea_{dyn} represents the dynamic interaction between BP and SV during a respiratory cycle, and it may be useful for the prediction of BP response to fluid challenge in preload-dependent patients [16].

Conclusions

Over recent years, a growing interest in cardiovascular physiology has been aroused. This interest has been mostly focused on venous return and cardiac function. However, our knowledge of the arterial system is still mostly simplified to the estimation of vascular resistance. A better understanding of AL will lead to enhanced knowledge of the underlying processes involved in cardiovascular diseases and will hopefully aid the development of new tools and interventions to monitor and treat these diseases.

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Compliance with ethical standards

Conflicts of interest
The authors declare no conflict of interest regarding this commentary.

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