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Ventricular-arterial Coupling: Advances and Current Perspectives in Cardiovascular Research

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Abstract

The concept of ventricular-arterial coupling (VAC) was first introduced in the early 1980s to quantify the relationship between left ventricular contractility and arterial load. The mathematical formulation of VAC, expressed as the ratio of arterial elastance to ventricular elastance, has since then been refined with adjustments to allow for non-invasive assessment. By the early 2000s, advancements in echocardiography, cardiac magnetic resonance and arterial tonometry provided non-invasive alternatives to the traditional invasive method of cardiac catheterization, broadening the clinical application of VAC. Emerging technologies, such as machine learning and computational models, have further enhanced the precision and personalization of VAC, with potential applications in heart failure, hypertension and other clinical scenarios.

This review describes the physiological basis and the historical development of VAC, highlights the non-invasive assessment techniques, and discusses the potential for personalized treatment based on VAC insights. Machine learning models trained on large datasets from non-invasive imaging modalities may open new avenues in predicting individual patient responses to therapies. However, lack of standardized protocols across imaging modalities represents a challenge, making the call for standardization critical for consistent clinical application. This review underscores the need for harmonized methodologies to better utilize VAC in personalized medicine, aiming to improve cardiovascular outcomes through tailored therapies.

Keywords: Ventricular-arterial coupling; ventricular and aortic elastance; myocardial contractility; arterial compliance.

Introduction

The concept of ventricular-arterial coupling (VAC) and its mathematical formulation was first introduced by Sunagawa et al. in the early 1980s [1]. Their original work, published in 1983, laid the groundwork for understanding the interaction between the left ventricle and the arterial system in mechanical terms, using elastance-based parameters. The key formula included the arterial elastance (Ea), which represents the effective afterload of the arterial system, and the end-systolic elastance (Ees) which represents the left ventricular (LV) contractility. VAC was then defined as Ea/Ees. After the proposal of the original formula, researchers

further refined the calculation of Ea and Ees to improve its clinical application. For instance, the method was applied in humans using non-invasive techniques (such as echocardiography) and systolic blood pressure (SBP) as a surrogate for end-systolic pressure. Kelly et al. introduced the concept of using $0.9 \times$ SBP as a simplified estimate of end-systolic pressure for non-invasive VAC assessment in clinical settings [2]. This adjustment made the formula more feasible in clinical practice.

In this article we will review the physiological background, the different methods for assessment, and the directions of development of VAC.

Physiological background

The heart and the arterial system are fundamentally related, both anatomically and functionally, and VAC describes the relationship between the two systems [3]. VAC is estimated as the ratio of arterial and ventricular elastances. The ratio of the LV end-systolic pressure to the stroke volume is known as the effective arterial elastance (Ea), and it succinctly conveys the steady and pulsatile components of the arterial load.

Total peripheral resistance, as one of the properties of the steady component of the arterial load, mainly depends on microvasculature. Contrarily, the pulsatile arterial load is primarily determined by the properties of the macrovasculature, which include the impedance of the aorta (Zc), the total arterial compliance, and the wave reflections [4]. Ventricular elastance (Ees) represents the slope of the line connecting V0 to the LV end-systolic pressure-volume relation, which is unaffected by preload or afterload and is a measure of cardiac contractility. Combined with Ea, it has been used to assess heart-arterial coupling [5].

For a given beat-to-beat preload and afterload, the Ees may be obtained from the LV pressure-volume (PV) loop. The PV loop is predicted on the end-systolic pressure–volume relationship (ESPVR), which is a linear connection between the end-systolic ventricular pressure and the end-systolic LV volume. The Ees is the intracavitary pressure needed to expand its volume by one unit (mmHg ml−1; normal values 2.3 ± 1 mmHg ml−1), while Ea represents the slope of the line connecting the left ventricular end-diastolic volume to the ESPVR (normal values 2.2 ± 0.8 mmHg ml−1). Ventriculo-arterial coupling is the Ea/Ees ratio, and the normal values are 1 ± 0.36 mmHg ml−1 [4].

The Ea/Ees ratio is used to assess how well the heart and arterial system are matched. Under normal conditions, the ratio approximates 1. Increased Ea/Ees suggests higher arterial stiffness relative to ventricular function, commonly seen in heart failure and hypertension [6]. Decreased Ea/Ees indicates impaired contractility, as seen in cases of heart failure with reduced ejection fraction (HFrEF) [7]. Thus, VAC acts as a significant indicator of the mechanical function of the LV and regulation of the cardiovascular system.

Introduction of assessment methods

Since the early 2000s, there has been a growing interest in non-invasive methods for measuring VAC. Techniques such as arterial tonometry, echocardiography, and cardiac MRI, allowed for estimates of both Ea and Ees without the need for invasive PV loop recordings. This shift was critical for applying VAC in broader clinical settings, especially in patients with cardiovascular diseases such as heart failure and hypertension. More recent research has integrated advanced imaging techniques like MRI and echocardiography to assess VAC in greater detail. These developments allow for dynamic VAC assessment, considering how coupling changes under different physiological conditions (e.g., exercise or pharmacological stress) [8]. Additionally, some studies have refined how ventricular elastance is estimated noninvasively through models that incorporate ventricular strain and tissue Doppler imaging data, further enhancing the practical utility of the VAC formula [9]. The relative merits of invasive and non-invasive methods for VAC assessment are summarized in Table 1.

Invasive method

● High fidelity conductance microcatheters

During cardiac catheterization, a PV loop can be generated, that is a graphical representation of the relationship between LV pressure and volume throughout the heartbeat. It provides valuable insights into both systolic and diastolic function, as well as the interaction between the heart and the arterial system. A catheter equipped with sensors is introduced into the left ventricle through a major artery (usually the femoral artery). The catheter records LV pressure and volume continuously. Volume changes can be measured using conductance technology or by integrating echocardiographic imaging with the catheter's data [3]. To determine the Ees and Ea, a brief occlusion of the inferior vena cava is performed to reduce venous return. This allows clinicians to generate multiple PV loops under different loading conditions and calculate the ESPVR (Figure 1) [10].

Figure 1 – SThis series of loops, that represent multiple cardiac cycles, allow us to observe how the heart's pressurevolume relationship changes over time under different loading conditions, contractility, or heart rates. By shifting the preload (end-diastolic volume), afterload (arterial pressure), or contractility, a family of loops can help assess cardiac function. The line connecting the end-systolic volumes represents the ventricular elastance. Ees: Ventricular elastance; ESPVR: Endsystolic pressure-volume relationship.

An invasive simplified method is based on mathematical extrapolation of Ees from single-beat measures [11, 12]. Single PV loop measurement represents a single cardiac cycle with one contraction and relaxation phase. It can be used to examine detailed changes in pressure and volume during systole and diastole. From the PV loop, Ees is the slope of the line connecting V0 to the end-systolic pressure-volume relation, while Ea is the slope of the line connecting the LV end-diastolic volume to the end-systolic pressure-volume relation (Figure 2).

● Advantages and disadvantages of invasive approach

Since catheterization involves direct measurements of pressure and volume, it is by far more precise than non-invasive methods like echocardiography or MRI. For assessing complex cardiovascular dynamics like VAC, cardiac catheterization is considered the gold standard because it provides quantitative, real-time data that can be used to calculate indices of ventricular contractility and arterial load with unmatched accuracy. Furthermore, during catheterization, clinicians can also perform therapeutic procedures (e.g., coronary angioplasty or valvuloplasty) while assessing cardiac function, making it a diagnostic and therapeutic tool.

One of the disadvantages of the method is its invasiveness, requiring catheter insertion into the heart or arteries, which carries risks of complications like bleeding, infection, or vessel damage. Assessing VAC through catheterization requires expertise in both the procedure and the interpretation of data, including the calculation of Ees and Ea. Due to its invasiveness, cardiac catheterization is often reserved for patients with suspected or known significant cardiovascular disease, rather than being used for routine VAC assessment.

Non-invasive methods

● Echocardiographic estimation of Ees and Ea

Echocardiography is a non-invasive method used to estimate VAC through complex mathematical calculations, which allow to extrapolate Ees from the information obtained in a single cardiac cycle. The method developed by Chen et al. includes the calculation of stroke volume (from the velocitytime integral in the LV outflow tract and the LV outflow tract area), the LV ejection fraction, and the BP at two different moments of the heart cycle [13]. Doppler imaging are the key techniques for estimating these parameters. Arterial elastance is

Figure 2 – Schematic drawing of the pressure-volume loop with lines identifying the arterial and ventricular elastance (panel A). Pressure-volume loop obtained in a normal subjects from feature-tracking cardiac magnetic resonance imaging. From the pressure-volume loop, arterial and ventricular elastance are derived (panel B). Ea: Arterial elastance; EDV: End-diastolic volume; Ees: Ventricular elastance; ESPVR: End-systolic pressure-volume relationship; ESV: End-systolic volume; PE: Potential energy; SW: Stroke work

calculated as the ratio of end-systolic pressure (often estimated as 0.9 x SBP) to stroke volume [2]. Echocardiography may be used to estimate end-systolic elastance, which is a measure of ventricular contractility, by monitoring end-systolic pressure and end-systolic volume [9].

● Advanced imaging to assess ventricular and arterial function

MRI offers precise measurements of both ventricular elastance and arterial elastance. Ea is calculated as the ratio of end-systolic pressure (ESP) to stroke volume (SV), which can be accurately measured through MRI's ability to quantify LV volumes and aortic blood flow during the cardiac cycle [14]. MRI provides high-resolution images to calculate ESV and assess myocardial strain, which is essential for evaluating ventricular function and contractility [8]. Recently, advanced software based on a mathematical model allows noninvasive analysis of PV loops from feature-tracking MRI or echocardiography [15]. This new method derives PV loops by combining CMRderived volumes with brachial blood pressure measurements, providing insights into cardiac function without the need for invasive catheterization. This method has been validated against traditional invasive techniques, showing strong correlation for important hemodynamic parameters such as stroke work, ventricular efficiency, and potential energy [16].

● Arterial tonometry: pulse wave velocity and arterial compliance measurement.

Arterial tonometry is a non-invasive technique used to assess VAC by measuring pulse wave velocity (PWV) and arterial compliance. PWV reflects arterial stiffness, which is a key determinant of arterial elastance. PWV is calculated by measuring the speed of the pressure wave traveling through the arteries, with higher values indicating increased arterial stiffness and thus higher Ea [17].

PWV is measured by recording the pressure waveforms at two arterial sites (e.g., carotid and femoral arteries). The time it takes for the pressure wave to travel between these two points gives PWV, which correlates with arterial stiffness. Increased PWV indicates stiffer arteries, meaning higher arterial elastance. Arterial compliance is derived from the shape of the aortic pressure waveform, which is recorded through tonometry. Reduced compliance (stiffer arteries) increases Ea, thus increasing the afterload on the heart. Since tonometry focuses on arterial parameters, Ees is estimated using complementary data from imaging (e.g., echocardiography) or blood pressurevolume relationships.

Emerging technologies and computational models

The integration of computational models and machine learning algorithms is transforming how VAC is assessed. Computational models use mathematical representations of the heart and vasculature to simulate the interaction between ventricular and arterial function under various conditions. These models can incorporate real-time data from non-invasive imaging (e.g., echocardiography or MRI) and adjust to dynamic physiological changes. More recently, machine learning algorithms have been used to analyze large datasets from noninvasive imaging to predict VAC parameters, such as Ea and Ees, without the need for direct invasive measurements. Artificial neural networks and other machine learning models have been trained to recognize patterns in echocardiographic data that correlate with VAC, improving the speed and accuracy of VAC assessment [18]. These models have the potential to automate

the analysis of large imaging datasets, providing personalized assessments of VAC for individual patients.

Comparative analysis of assessment methods

In the table provided below we compared invasive and noninvasive methods of the VAC assessment (Table 1).

Clinical application of VAC

VAC in heart failure with reduced ejection fraction (HFrEF)

Ees and Ea are mismatched in HFrEF due to a reduction in myocardial contractility. The mismatch between the heart's pumping ability and the arterial load exacerbates heart failure symptoms, resulting in decreased stroke volume and higher arterial pressures. In individuals with HFrEF, VAC evaluations can predict unfavorable outcomes including hospitalization and death [19].

VAC in heart failure with preserved ejection fraction (HFpEF)

Even though VAC responds dynamically in HFrEF (raised values with the lowered Ees and increased Ea), its relevance as a dimensionless number is less clear in HFpEF. In fact, in this case both Ees and Ea are elevated resulting in a "normal" VAC [6].

VAC in hypertension

Vascular artery stiffness and elastance are increased in hypertension, a common disorder that profoundly affects VAC. The long-term mismatch between artery and ventricular elastance deteriorates cardiovascular outcomes. Earlystage hypertension is compensated for by the LV enhanced contractility, which keeps VAC constant despite the increased afterload. But gradually, when arterial stiffness increases and VAC is affected, it leads to a decrease in cardiac output and the onset of HF symptoms [22]. Through VAC examination, this shift can be identified early and treated promptly. A greater risk of cardiovascular events, including myocardial infarction and stroke, is linked to significant VAC impairment in resistant or advanced hypertension. More potent antihypertensive methods or gadget-based treatments, like baroreceptor stimulation, may fall under this category [30].

VAC in aortic stenosis

Arterial elastance is increased with aortic stenosis because the left ventricle is challenged with a fixed outflow barrier. Ventricular geometry and contractility are eventually affected by this increased afterload. Thus, VAC might be a useful tool for monitoring patients with aortic stenosis to identify the best time to replace the valve [31]. Quantifying ventricular-arterial mismatch is often done using non-invasive techniques such as MRI and echocardiography [24].

VAC in coronary artery disease (CAD)

Ischemia in CAD impairs arterial function (increased Ea) and ventricular contractility (decreased Ees), which results in ineffective coupling. Clinicians can estimate the degree of ischemia and choose the best revascularization techniques with the use of VAC evaluation [20]. For long-term therapy and prognosis, routine VAC evaluation can be useful in patients with CAD.

VAC as a prognostic indicator

In patients with heart failure, the composite events of hospitalization for heart failure and cardiovascular mortality were substantially correlated with Ea/Ees [32]. VAC has a crucial role in forecasting long-term cardiovascular mortality in individuals with prior myocardial infarctions and is an independent echocardiographic correlate of B-type natriuretic peptide levels in these patients [33]. In patients admitted to the intensive care unit, VAC was a powerful and independent predictor of in-hospital clinical outcomes (acute heart failure, hypoperfusion, requirement for invasive ventilation, intra-aortic balloon pump, renal replacement therapy, and mortality) [34]. Furthermore, the researchers found that both very high and very low VAC values were associated with worse outcomes, including higher mortality and fewer ventilator-free days in patients with sepsis or septic shock [35]. This highlights VAC as a potential marker for patient prognosis in critical care settings.

Implications for therapeutic interventions

Clinicians can evaluate responses to therapy and modify treatment plans by assessing non-invasive evaluation of VAC [20]. Assessing VAC is critical for directing therapy methods, such as diuretics and vasodilators [29]. Reducing Ea by therapeutic procedures that target the arterial system (ACE inhibitors, angiotensin II receptor blockers, etc.) can improve cardiac output and restore a normal balance [21].

Future implications

Need for standardization of non-invasive techniques

Different non-invasive techniques provide different ways to estimate ventricular elastance and arterial elastance, leading to inconsistent results across studies and clinical settings. For example, echocardiography can measure Ees using either global longitudinal strain or TDI-derived myocardial velocities, which can yield different estimates of contractility [9]. The lack of standardized non-invasive techniques makes it difficult to establish clear clinical cut-offs for impaired VAC. Studies using different imaging modalities often report varying thresholds for pathological coupling [36]. The lack of standardized methods limits the use of non-invasive VAC assessment in routine care, where the ability to accurately monitor VAC could help optimize treatment for heart failure patients. Without standardized protocols, the variability in these measurements makes it difficult to compare findings between studies and ensure consistent clinical interpretation.

Potential for personalized treatment based on VAC

In order to provide individualized care, VAC evaluation provides a window into the degree of ventricular dysfunction and arterial stiffness. Based on VAC findings, tailored treatment is being further enhanced through the integration of big data and machine learning. AI algorithms are able to forecast individual patient responses to specific medicines by examining vast datasets from imaging, genetics, and electronic health records. For example, VAC parameters may be processed by machine learning algorithms to forecast outcomes in heart failure or hypertension, enabling more accurate drug and intervention modifications.

Conclusion

VAC provides a crucial framework for understanding the dynamic interplay between the heart and arterial system. VAC assessment has evolved significantly over time, integrating both invasive and non-invasive methods for assessing its arterial and ventricular components. Clinical applications of VAC span various cardiovascular conditions such as heart failure, hypertension, and coronary artery disease, offering valuable insights into disease prognosis and treatment optimization.

The transition from invasive methods like cardiac catheterization to non-invasive techniques such as echocardiography, MRI, and arterial tonometry has widened the clinical application of VAC assessment, making it more accessible in routine clinical practice. Each method comes with its own strengths and limitations, with MRI offering high precision and echocardiography providing widespread applicability.

Further advancements in computational models and machine learning algorithms have the potential to revolutionize VAC assessment by predicting patient-specific outcomes and tailoring therapeutic interventions. However, the variability in non-invasive measurement techniques underscores the need for standardized protocols to ensure consistency across clinical and research settings. Ultimately, VAC may serve as an essential diagnostic and prognostic tool in cardiovascular medicine, offering new avenues for personalized treatment strategies and improved patient outcomes. Ongoing research and technological developments are expected to refine VAC assessment and enhance its clinical utility.

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