



An Overview of Pulmonary Artery Pulsatility Index and Aortic Pulsatility Index

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Abstract

Pulsatility indices are hemodynamic parameters used to evaluate vascular compliance, cardiac function, and circulatory performance in both the pulmonary and systemic circulations. Two such indices — the Pulmonary Artery Pulsatility Index (PAPi) and the Aortic Pulsatility Index (AoPI) — have emerged as useful tools in assessing right and left heart function respectively, especially in critically ill patients. The PAPi is calculated using the formula: $\text{PAPi} = (\text{Pulmonary Artery Systolic Pressure} - \text{Pulmonary Artery Diastolic Pressure}) / \text{Right Atrial Pressure}$. This index provides insight into right ventricular (RV) function and pulmonary artery compliance. A low PAPi (typically <1.0) is associated with right ventricular failure, particularly in patients with acute myocardial infarction or those being evaluated for mechanical circulatory support. PAPi is also used in heart transplantation and advanced heart failure management to guide therapy and predict outcomes. The AoPI is a less standardized but increasingly explored marker, often defined as: $\text{AoPI} = (\text{Systolic Blood Pressure} - \text{Diastolic Blood Pressure}) / \text{Central Venous Pressure (CVP)}$ or sometimes relative to mean arterial pressure or pulse pressure. AoPI is considered reflective of systemic arterial compliance and left ventricular performance. It has potential applications in identifying left-sided heart dysfunction, fluid responsiveness, and vascular tone assessment in the setting of shock, particularly septic or cardiogenic shock. Both indices can be derived from invasive hemodynamic monitoring in intensive care or catheterization settings, and they offer dynamic insights beyond static pressure measurements.

Keywords: Pulmonary Artery Pulsatility Index and Aortic Pulsatility Index, hemodynamic parameters.

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1. Introduction

Invasive hemodynamic monitoring is recommended in patients with advanced heart failure and cardiogenic shock specially that are unresponsive to initial therapy. These hemodynamic parameters are sufficiently to detect early, subtle deterioration in these patients and can be safely applied and repeated continually can also predict the value of intervention and clinical outcomes in these patients. The PAPi has found acceptance into clinical practice, particularly in the management of advanced heart failure, cardiogenic shock and left ventricular assist device therapy. This index has multiple determinants and will reflect changes in any of the components of the right heart system – systemic venous system, RV function and the pulmonary circulation. By extension, PAPi will vary significantly in different patient populations based on the underlying pathophysiology, which would render the application of a single PAPi threshold across different patient Groups invalid [1]. PAPi is a non-invasive measure that calculates the ratio of pulmonary artery

pulse pressure (PAPP) to right atrial pressure (RAP) calculated by echocardiography.

The PAPi has been adopted into clinical practice and was a central parameter in the algorithm described by Tehrani et al. in the management of cardiogenic shock. However, although the calculation for PAPi is simple, the physiological interpretation is more nuanced [2]. It is one of the most valuable hemodynamic measures which has role to predict clinical outcome in patients with decompensated heart failure. Our study used a non-invasive PAPi hemodynamic parameter which calculated through one echocardiographic equation to predict the prognosis and clinical outcome in patient with decompensated heart failure PAPi equals (PA systolic pressure – PA diastolic pressure) / RAP. PAPi is defined as the ratio of pulmonary artery pulse pressure (sPAP – dPAP) to right atrial pressure (RAP). Systolic pulmonary artery pressure (sPAP) is determined from tricuspid regurgitation (TR) velocity using the simplified Bernoulli equation: $\text{sPAP} = 4x (\text{peak TR jet velocity})^2 + \text{RA pressure}$.

Diastolic pulmonary artery pressure (dPAP) is calculated as: $dPAP = 4 \times (\text{end-diastolic pulmonary regurgitant velocity})^2 + \text{RA pressure}$ [3].

- Measuring of right atrial pressure by Echocardiographic assessment of right atrial pressure (RAP). Normal IVC (IVC < 2.1 cm) and > 50% collapsible with inspiration, RAP = 5 mmHg. IVC dilated (> 2.1 cm), with > 50% collapsibility with inspiration, RAP = 10 mmHg. Dilated IVC (> 2.1 cm), with no inspiratory collapse, RAP = 15 mmHg.

2. Clinical significance of PAPI

The prognostic value of PAPI is assessed by its capacity to predict future events or clinical endpoints, aiding clinicians in risk stratification and treatment decision-making in patients with heart failure, pulmonary embolism, right ventricular infarction and pulmonary hypertension [4].

2.1. Clinical Outcome Prediction and prognosis

The prognostic value of PAPI involves its capability to predict relevant clinical outcomes, such as mortality, hospitalizations, or the need for advanced therapeutic interventions, in patients with pulmonary hypertension and heart failure [5]. The physiological basis for PAPI as an indicator of right heart function is predicated on PASP as an indirect indicator of RV contractile function against a given afterload, and high RAP as a sign of failing right ventricle. However, the interpretation of PAPI is more nuanced, as pulmonary artery pulse (and systolic) pressure is dependent on both RV stroke volume and pulmonary arterial capacitance (PAC) [6].

2.2. Risk Stratification

PAPI serves as a tool for risk stratification, allowing clinicians to categorize patients into different risk groups based on their PAPI values. Lower PAPI values may be indicative of increased right ventricular dysfunction, translating into a higher risk of adverse outcomes. This information guides the intensity of monitoring and therapeutic interventions [7].

2.3. Clinical Decision-Making

Clinicians use the prognostic information derived from PAPI to inform clinical decision-making. For example, patients with a high PAPI indicative of greater right ventricular afterload may be considered for more aggressive therapeutic interventions, advanced therapies, or closer follow-up to prevent adverse events [8].

2.4. Post-Cardiac Surgery Monitoring

Monitoring PAPI post-cardiac surgery, especially in cases involving pulmonary artery catheterization, may offer insights into right ventricular function and help guide postoperative management [9].

2.5. Congenital Heart Diseases

PAPI might be useful in assessing right ventricular function in patients with congenital heart diseases, especially those with pulmonary circulation involvement [10].

2.6. Response to Pulmonary Hypertension Therapies

PAPI could be used to monitor the response to specific pulmonary hypertension therapies, providing

clinicians with an additional parameter to evaluate treatment efficacy [11].

2.7. Cardiogenic Shock

In cases of cardiogenic shock, where right ventricular dysfunction can be a significant factor, PAPI may be considered as part of the hemodynamic assessment to guide therapeutic interventions [12].

3. Drawbacks of PAPI

3.1. Sensitivity to Measurement Techniques

PAPI is sensitive to the measurement techniques employed during right heart catheterization. Variability in measurements, such as differences in catheter placement or calibration, can introduce inconsistencies and affect the accuracy of PAPI values [13].

3.2. Influence of Right Atrial Pressure

Invasive PAPI hemodynamic parameter is calculated by dividing the pulmonary artery pulse pressure by the right atrial pressure. Changes in right atrial pressure can significantly impact PAPI values. Variations in right atrial pressure due to factors like tricuspid regurgitation or intravascular volume status may introduce confounding effects [14].

3.3. Dynamic Nature of Right Ventricular Function

Right ventricular function is dynamic and can change rapidly in response to physiological and pathological conditions. PAPI, being a static parameter, may not capture the dynamic fluctuations in right ventricular function over time or during acute changes in clinical status [15].

3.4. Inter patient Variability

The response of PAPI to right ventricular dysfunction can vary among individuals. Some patients may exhibit low PAPI values with preserved right ventricular function, while others may have high values despite significant dysfunction. This inter patient variability may limit the specificity of PAPI [16].

3.5. Lack of Standardization

Standardization of PAPI measurements across different centers and studies is often lacking. The absence of standardized protocols for measurement and interpretation may contribute to inconsistencies in reported PAPI values and hinder comparisons between studies [17].

3.6. Research Gaps

Despite the potential applications of PAPI, there are still research gaps in understanding its utility in various clinical scenarios. Additional studies are needed to validate its use and explore its limitations in different patient populations and disease states [18].

3.7. Dependency on invasive PAPI in Right Heart Catheterization

PAPI measurements depend on the invasive procedure of right heart catheterization. This limits its application in routine clinical practice and may pose challenges, especially in patients who are not candidates for or are reluctant to undergo invasive hemodynamic assessments [19].

4. Aortic Pulsatility Index (API)

4.1. Definition

The Aortic Pulsatility Index (API) is a hemodynamic parameter that quantifies the pulsatility of blood flow in the aorta, which is the major artery that carries oxygenated blood from the heart to the rest of the body. Pulsatility index (PI) was defined as the difference between the peak systolic flow and minimum diastolic flow velocity, divided by the mean velocity recorded throughout the cardiac cycle. It is a non-invasive method of assessing vascular resistance with the use of Doppler ultrasonography. It was first introduced in 1974 by Gosling and King and is also known as the Gosling Index [20]. Recently, the aortic pulsatility index (API) is a haemodynamic variable derived from systemic pulse pressure (systolic-diastolic) divided by pulmonary capillary wedge pressure (PCWP) – has been proposed as a new marker of left heart performance with evidence of prognostic predictive abilities superior to the established haemodynamic measurements in patients with acute decompensated HF [21]. The API is analogous to the pulmonary artery pulsatility index (PAPI), which is the best invasive hemodynamic predictor of right ventricular failure and clinical outcomes in patients with advanced heart failure. Furthermore, both pulse pressure, defined as the difference between systolic blood pressure and diastolic blood pressure, and left ventricular end-diastolic pressure, for which PCWP serves as a surrogate, have been associated with short-term mortality in patients with acute coronary syndromes [1].

- PCWP can be calculated through echocardiographic equation: $PCWP = 1.9 + (E/e' \times 1.24)$ as prescribed by Nagueh *et al.*, [22] and approved in clinical practice by Sugimoto *et al.*, [23].

4.2. Clinical significance and importance of non-invasive API

- Significance in hypertensive patients

Many available reports discuss the association between the loss of arterial elasticity and hypertension. A study explains that increased blood pressure values are associated with an increase in intimal-media thickness, lumen enlargement, and increased stiffness of proximal elastic arteries. Conversely, several publications have shown that arterial stiffness precedes and strongly contributes to the development of hypertension [24]. In hypertensive patients, the pulsatility index can be used as a predictor of complications and help assess the chronicity of the disease. In a recently published study from 2019, Sasaki *et al* have shown that Aortic Pulsatility Index, as well as resistance index measurements, are associated with the log NT-pro-BNP levels in hypertensive patients, suggesting that PI could be a useful marker of hemodynamic stress on left ventricle, while mean intima-media thickness, number of plaques, and plaque score had no association with log NT-proBNP [25].

- Significance of API in decompensated heart failure patients

Belkin *et al.*, [20] conducted a study titled Aortic Pulsatility Index: a novel hemodynamic variable for evaluation of decompensated heart failure. Right heart catheterization for invasive hemodynamics has shown only a modest correlation with clinical outcomes. They designed a novel hemodynamic variable that incorporates ventricular output and filling pressure. They anticipated that the aortic

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pulsatility index (API) would correlate with clinical outcomes in patients with heart failure the API is considered as a novel invasive hemodynamic measurement that can predict need to use of vasoactive medications or temporary mechanical circulatory support devices, implantation of a left ventricular assist device, heart transplantation, or death within 30 days.

4.3. Challenges and drawbacks of API

While the potential of API in heart failure management is evident, challenges exist. Standardization of measurement variables and consideration of confounding variables, and addressing the dynamic nature of heart failure pose ongoing challenges. Addressing these issues is crucial to establishing API as a reliable and reproducible parameter in clinical practice [26]. As individuals age, the elasticity of the arterial walls typically decreases, leading to increased stiffness and a higher pulsatility index. This reduced compliance means that the arteries are less able to expand and contract with each heartbeat, resulting in greater pressure fluctuations. Similarly, atherosclerosis, characterized by the buildup of plaques in the arterial walls, further contributes to arterial stiffness. This condition narrows the arteries and disrupts normal blood flow, often causing an increase in the API [27]. API influenced by aspects such as body size, blood pressure, and the presence of comorbidities like diabetes or hypertension. For instance, individuals with a higher body mass index may experience different arterial compliance compared to those with a lower BMI, affecting the API. Additionally, lifestyle factors such as diet, physical activity, and smoking can alter vascular health and elasticity, leading to variability in the pulsatility index.

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