Review Article

The tricuspid annular plane systolic excursion/PASP ratio's accuracy and validity in assessing the right ventricular function: A narrative review

Alper Sercelik[1], Lutfu Askin[2]

[1]Department of Cardiology, Sanko University, Gaziantep, Turkey, [2]Department of Cardiology, Gaziantep Islamic Science and Technology University, Gaziantep, Turkey

The tricuspid annular plane systolic excursion (TAPSE) (transthoracic apical two-chamber stretch) and pulmonary artery systolic pressure (PASP) ratio is a measure of cardiac function that is used to assess left ventricular systolic function. PASP is typically measured using a catheterization procedure, in which a small tube is inserted into a blood vessel and advanced to the pulmonary artery. A TAPSE/PASP ratio higher than 0.36 mm/mmHg has been shown in several studies to be a good sign of normal or generally well-maintained right ventricular function. It is important to note that the TAPSE/PASP ratio should be interpreted in the context of other clinical findings and should not be used as the sole indicator of cardiac function. A decrease in the TAPSEpulmonary arterial systolic pressure (PASP) ratio (i.e., (RV)-arterial uncoupling), which quantitatively depicts the function of the RV, was detected in patients with heart failure. In pulmonary arterial hypertension patients, TAPSE/PASP is linked to hemodynamics and functional class. In diseases impacting right cardiac function, the TAPSE/PASP may also be beneficial. The purpose of this review is to demonstrate how the TAPSE/PASP impacts how the (RV) functions. We believe that this is the first review on the topic written.

Key words: Arterial uncoupling, heart failure, pulmonary arterial hypertension, right ventricle arterial uncoupling, right ventricle functions, tricuspid annular plane systolic excursion/pulmonary arterial systolic pressure

How to cite this article: Sercelik A, Askin L. The tricuspid annular plane systolic excursion/PASP ratio's accuracy and validity in assessing the right ventricular function: A narrative review. J Res Med Sci 2024;29:75.

INTRODUCTION

The right ventricular (RV) is given more importance now. New data on heart failure (HF) are entering the literature on the function of the RV. Noninvasive RV performance assessments are gaining popularity. The link between longitudinal RV fiber shortening (tricuspid annular plane systolic excursion [TAPSE]) and developed pressure (pulmonary artery systolic pressure, (PASP)) is one of these that governs RV-arterial coupling.[1] The goal of this review is to show how much the TAPSE/PASP affects how the (RV) funcitons. I think that this is the first review of this subject that has been written. Table 1 shows the major theme of the references.

Access this article online	
Quick Response Code:	Website: https://journals.lww.com/jrms
	DOI: 10.4103/jrms.jrms_350_24

Conventional echocardiographic techniques present challenges in evaluating right ventricular function due to the intricacies of right ventricular morphology and physiology. Consequently, the TAPSE/PASP ratio has been suggested as a feasible alternative. The ratio in question might be better because it is easy to use, does not involve any harm, and can give a full picture of how well the (RV) is working, especially when there is pulmonary arterial hypertension (PAH) or HF. By providing this background information, you will establish a more robust justification for investigating the TAPSE/PASP ratio in your review, thereby clarifying the topic's significance and relevance.[1]

Address for correspondence: Prof. Lutfu Askin, Sanko University, Gaziantep 2700, Turkey.
E-mail: asercelik@hotmail.com

Submitted: 02-Jul-2024; **Revised:** 17-Aug-2024; **Accepted:** 20-Sep-2024; **Published:** 31-Dec-2024

Table 1: Main topic points of references

Reference number	Authors	Subjects	Main theme
[1]	Guazzi *et al*.	HFrEF patients	TAPSE versus PASP may be a step forward for a more efficient RV function assessment and is not impacted by LV dysfunction
[2]	Giardini *et al*.	HFrEF patients	Bernoulli equation measure provides an indirect estimation of the pressure in the pulmonary artery, which is crucial in assessing the haemodynamic burden on the RV
[3]	D'Alto *et al*.	HFrEF patients	Operators must be skilled in interpreting echocardiographic data, understanding potential sources of error, and recognising conditions that might affect the reliability of TAPSE or PASP
[4]	Guazzi *et al*.	HFrEF patients	They discovered a correlation between TAPSE/PASP and RV Emax/Ea (RV-arterial coupling).
[5]	Vonk-Noordegraaf *et al*.	HFrEF patients	Reduced TAPSE/PASP indicates RV dysfunction
[6]	Vonk-Noordegraaf *et al*.	HFrEF patients	RV-arterial coupling indicates afterload-adjusted RV contractility
[7]	Faragli *et al*.	HFrEF patients	End-systolic elastance (Ees) of the LV is defined as the ratio of end-systolic pressure (ESP) to end-systolic volume (ESV) (ESV)
[8]	Naeije *et al*.	HFrEF patients	RV-arterial uncoupling with hydraulic elevation is like PVR or HF. Disruption of RV-arterial copuling triggers processes that result in cardiogenic shock
[9]	Berlin *et al*.	HFrEF patients	Isovolumetric relaxation is not typical for RV. Pulmonary circulation is provided by RV longitudinal fiber contraction and tricuspid ring apex retraction
[10]	Melenovsky *et al*.	RV failure patients	TAPSE, which is equivalent to or better than conventional echo Doppler indicators of RV function, is increasingly used and can be obtained in most patients, even with atrial fibrillation or rapid heart rates
[11]	Naeije *et al*.	HFrEF patients	The RV's thin wall shows it wasn't meant for dramatic rises in pulmonary artery pressure, yet it maintains continuous gas exchange for the whole systemic venous return.
[12]	Voelkel *et al*.	RV failure patients	RV preload, afterload, and contractility are all affected by whole heartbeats, cardiac rhythm, LV interaction, and pericardial limitation
[13]	Tello *et al*.	RV failure patients	RV maintains cardiac cycle and ventriculo-arterial coupling.
[14]	Pinsky *et al*.	Pigs	RV and LV contractions interact positively. Even though twisting and rotating motions don't add much to RV contractility, LV contraction contributes 30% to both RV contraction and pulmonary flow in an intact pig heart
[15]	Al-Naamani *et al*.	RV failure patients	Reduced PAC is linked to increased pulmonary vascular stiffness, which is linked to RV dysfunction

HT=Hypertension; HF=Heart failure; HFrEF=HF with reduced ejection fraction; TAPSE=Tricuspid annular plane systolic excursion; PASP=Pulmonary artery systolic pressure; RV=Right ventricular; LV=Left ventricular; PC=Pulmonary circulation; HFpEF=HF with preserved ejection fraction; PH=Pulmonary HT; Cpc-PH=Combined pre- and post-capillary PH; SLE=Systemic lupus erythematosus; PAH=Pulmonary arterial hypertension; PA=Pulmonary artery; PASP=PA systolic pressure; 6MWD=6-min walk distance; AHF=Acute HF; PVR=Pulmonary vascular resistance; CRT=Cardiac resynchronization therapy

TRICUSPID ANNULAR PLANE SYSTOLIC EXCURSION AND PASP MEASUREMENT

To measure TAPSE, the technician will obtain an apical four-chamber view of the heart using transthoracic echocardiography. This view is obtained by positioning the transducer on the patient's chest and tilting it to visualize the heart from the apex (tip) of the heart. The technician will then measure the distance between the tips of the mitral valve leaflets in this view and divide it by the distance between the tips of the mitral valve leaflets in the parasternal long-axis view, obtained by positioning the transducer on the patient's chest and tilting it to visualize the heart from the side. The resulting ratio is the TAPSE/PASP ratio. It is important to note that the accuracy of TAPSE and PASP measurements may be affected by various factors, such as the quality of the ultrasound image and the technician's experience and skill in performing the measurements.[1]

UNDERSTANDING TRICUSPID ANNULAR PLANE SYSTOLIC EXCURSION AND PASP MEASUREMENTS

Tricuspid annular plane systolic excursion

M-mode echocardiography, which records the tricuspid annulus's motion during systole, measures TAPSE. We position the ultrasound transducer in the apical four-chamber view to measure TAPSE. We align the M-mode cursor along the lateral tricuspid annulus and record the distance the annulus moves towards the apex during systole. TAPSE reflects the longitudinal function of the RV and is a simple, reproducible measure that correlates well with RV systolic function.[1]

PASP

Doppler echocardiography estimates PASP by measuring the peak velocity of the tricuspid regurgitation (TR) jet. We use the modified Bernoulli equation (PASP = 4 (TR velocity)^2+

right atrial pressure (RAP) PASP = 4 times (TR velocity)^2 + RAPPASP = 4 (TR velocity)^2 + RAP) to calculate PASP, and we typically estimate RAP from the size and collapse of the inferior vena cava. This measure provides an indirect estimation of the pressure in the pulmonary artery, which is crucial in assessing the hemodynamic burden on the RV.[2]

ULTRASOUND PROCESS

Imaging techniques

The accuracy of TAPSE and PASP measurements depends on high-quality images, which require precise transducer placement and patient positioning. For TAPSE, clearly obtain the apical four-chamber view and place the M-mode cursor perpendicular to the tricuspid annulus. To prevent underestimating the pressure gradient in PASP, align the Doppler signal parallel to the TR jet.

Operator skills

Accurate measurement of TAPSE/PASP requires proficiency in echocardiographic techniques, particularly in optimising image quality and Doppler alignment. Operators must be skilled in interpreting echocardiographic data, understanding potential sources of error, and recognising conditions that might affect the reliability of TAPSE or PASP (e.g., arrhythmias and poor image quality).[3]

CHALLENGES AND CONSIDERATIONS

Inter-observer variability

The measurements can be subject to inter-observer variability, especially in less experienced hands. Proper training and technique standardization can reduce this variability. In cases of significant TR or poor acoustic windows, both TAPSE and PASP may be challenging to assess accurately. By delving into these technical aspects, you provide a more detailed and practical understanding of the process, emphasizing the importance of operator expertise and the nuances involved in accurately measuring the TAPSE/PASP ratio.[3]

PITFALLS THAT CAN AFFECT THE ACCURACY OF TRICUSPID ANNULAR PLANE SYSTOLIC EXCURSION AND PASP MEASUREMENTS

Image quality

The accuracy of TAPSE and PASP measurements may be affected by the quality of the ultrasound image. Factors that can affect image quality include patient factors (such as obesity or emphysema), technical factors (such as transducer placement and angle), and environmental factors (such as room lighting and noise). Technician experience and skill: TAPSE and PASP measurements require careful and precise measurement of the distance between the tips of the mitral valve leaflets. The accuracy of these measurements may be affected by the technician's level of experience and skill. Patient positioning: proper patient positioning is important for obtaining accurate TAPSE and PASP measurements. The patient should be positioned in a supine position with the head slightly elevated to optimize visualization of the heart.

Cardiac motion

The movement of the heart during the cardiac cycle can affect the accuracy of TAPSE and PASP measurements. It is important to capture the image at the appropriate point in the cardiac cycle to ensure accurate measurements. Calibration of the echocardiography machine: The accuracy of TAPSE and PASP measurements may be affected by the calibration of the echocardiography machine. It is important to ensure that the machine is properly calibrated to obtain accurate measurements.[1]

CUTOFF VALUES FOR THE NORMAL TRICUSPID ANNULAR PLANE SYSTOLIC EXCURSION/RIGHT VENTRICULAR SYSTOLIC PRESSURE

A TAPSE/PASP ratio higher than 0.36 mm/mmHg has been shown in several studies to be a good sign of normal or generally well-maintained (RV) function. Researchers have linked a ratio of <0.31 mm/mmHg to poorer outcomes and may suggest (RV) dysfunction, particularly in situations such as pulmonary hypertension (HT). Individuals suffering from pulmonary HT, HF, or other cardiopulmonary ailments frequently employ the ratio, where a lower ratio indicates a greater probability of (RV) dysfunction and a more favorable prognosis. The precise threshold values may vary based on the population under investigation, with certain discrepancies observed among various age cohorts, genders, and clinical contexts. To establish a stable and globally acknowledged range for the TAPSE/PASP ratio, it would be necessary to conduct large-scale, multi-center research involving varied populations. These investigations have the potential to enhance the accuracy of the normal range and establish more exact threshold values for various clinical situations. Based on the present state of research, a TAPSE/PASP ratio >0.36 mm/mmHg is commonly considered as a general benchmark for normal (RV) function. However, it is important to consider specific patient characteristics and rely on clinical discretion when interpreting this ratio.[1]

GRADE OF RV DYSFUNCTION

The grading of RV dysfunction is typically based on the severity of the RV dysfunction and the presence of clinical signs and symptoms. The grading of RV dysfunction can be subjective and may vary depending on the specific criteria used and the expertise of the healthcare provider.

In general, mild RV dysfunction may be defined as an RV systolic pressure (RVSP) of 40–50 mmHg, with minimal or no clinical signs or symptoms. Moderate RV dysfunction may be defined as an RVSP of 50–70 mmHg, with the presence of clinical signs or symptoms such as exertional dyspnea (shortness of breath with exertion) or fatigue. Severe RV dysfunction may be defined as an RVSP >70 mmHg, with the presence of severe clinical signs or symptoms such as hypotension (low blood pressure [BP]) or syncope (fainting).

It is important to note that these definitions are general guidelines and that the grading of RV dysfunction may vary depending on the specific population being studied and the criteria used. It is always important to consult with a healthcare provider for specific information on the grading of RV dysfunction.[1]

ADVANCED RV HEART FAILURE

Guazzi *et al*.[4] validated and linked TAPSE/PASP with RV systolic/arterial elastance for everyday practice. They discovered a correlation between TAPSE/PASP and RV Emax/Ea (RV-arterial coupling). Reduced TAPSE/PASP indicates RV dysfunction. RV-arterial coupling indicates afterload-adjusted RV contractility.[5,6]

End-systolic elastance (Ees) of the left ventricular (LV) is defined as the ratio of end-systolic pressure (ESP) to end-systolic volume (ESV).[7] Mathematically, Emax (Ees)/Ea may be reduced to a volume ratio: (ESP/ESV)/(ESP/stroke volume [SV]), or “SV/ESV.” The SV/ESV ratio gives RV ejection fraction information. RV-arterial uncoupling with hydraulic elevation is such as pulmonary vascular resistance (PVR) or HF. Disruption of RV-arterial copuling triggers processes that result in cardiogenic shock.[8]

Isovolumetric relaxation is not typical for RV. Pulmonary circulation is provided by RV longitudinal fiber contraction and tricuspid ring apex retraction.[9] For this reason, TAPSE, which is equivalent to or better than conventional echo Doppler indicators of RV function, is increasingly used and can be obtained in most patients, even with atrial fibrillation or rapid heart rates.[10]

The RV’s thin wall shows that it was not meant for dramatic rises in pulmonary artery pressure, yet it maintains continuous gas exchange for the whole systemic venous return. RV preload, afterload, and contractility are all affected by whole heartbeats, cardiac rhythm, LV interaction, and pericardial limitation.[11,12] RV maintains cardiac cycle and ventriculo-arterial coupling.[13]

RV and LV contractions interact positively. Even though twisting and rotating motions don’t add much to RV contractility, LV contraction contributes 30% to both RV contraction and pulmonary flow in an intact pig heart.[14] HF causes early pathological alterations in the pulmonary vasculature, resulting in increased PVR and reduced pulmonary arterial capacitance (PAC). Reduced PAC is linked to increased pulmonary vascular stiffness, which is linked to RV dysfunction.[15]

PULMONARY ARTERIAL HYPERTENSION

PAH is characterized by high mean pulmonary arterial pressure and high PVR.[16] Maladaptive RV hypertrophy and/or dilatation cause RV failure.[17,18] The TAPSE/PASP is a clinical and predictive indicator in HF patients with and without PAH.[19] TAPSE/PASP is closely related to PAC; a measure of RV afterload compliance and PVR.[20]

The TAPSE/PASP ratio, when used in conjunction with the 6-min walk distance (6MWD), can help us understand the clinical status and potential outcomes for patients with PAH associated with systemic lupus erythematosus (SLE). A low TAPSE/PASP ratio and low 6MWD score may indicate that a patient is at higher risk for a poor prognosis.[21] In individuals with PAH, using TAPSE to assess (RV) function after targeted therapy can help predict future outcomes, regardless of the therapy’s effects on blood flow.[22] The TAPSE/PASP ratio has been shown to be associated with various outcomes in patients with PAH, including functional class and hemodynamic parameters. Elevated PASP and reduced TAPSE are typically seen in patients with PAH and can be used to assess the severity of the disease and the effectiveness of treatment.[23]

Schmeisser *et al*.[24] demonstrated that TAPSE is a good surrogate measure for RV adaptation to afterload (RV–PA coupling) in secondary PAH owing to HF with reduced EF (HFREF). TAPSE/PASP did not enhance RV–PA coupling or prognosis. TAPSE/PASP might mask RV contractile dysfunction and/or RV load in secondary PH owing to HFREF since both numerator and denominator are impacted. Noninvasive PASP computation requires a TR velocity signal, which is not always available. TAPSE looks more beneficial for RV–PA coupling analysis than PASP. For clinical risk assessment of HFREF patients with secondary PAH, further TAPSE/PASP trials are required.

Tello *et al*.[25] hypothesized that TAPSE/PASP is a proxy for invasively obtained Ees/Ea to determine RV-arterial coupling. Standard bedside echocardiography may assess TAPSE and PASP. TAPSE/PASP may add value to existing risk assessment methodologies for severe PH.

OTHER CONDITIONS

Lower transferrin saturation and ferritin were linked with

TAPSE-measured RV dysfunction in individuals with acute HF. Lower transferrin saturation, not ferritin, was linked to RV–PA uncoupling. Iron status was slightly or unrelated to the left ventricular ejection fraction. Transferrin saturation negatively correlated with natriuretic peptides. Further investigations should validate these results, analyze the pathophysiology underpinning this connection, and assess the impact of iron repletion on the right systolic performance.[26] RV function may not adjust to increasing afterload in hypertensive individuals. High BP and related systemic adaptation processes damage the pulmonary system. BP affects younger men more than menopausal women. This finding's functional and prognostic importance should be researched, although it may explain arterial HT and HF with preserved EF.[27]

The noninvasive TAPSE/PASP is connected with long-term results in cardiac resynchronization therapy (CRT) participants. TAPSE/PASP contributes to risk categorization in CRT patients. A lack of meaningful change TAPSE/PASP after CRT implantation was linked to poorer survival.[28] According to Deaconu *et al*.,[29] RV-PA coupling examination using TAPSE/PASP predicts CRT response, a lower TAPSE/PASP increases cardiovascular (CV) risk. RV-PA coupling assessed by TAPSE/PASP correlated with HF prognostic indicators. It predicted CRT better than PASP and TAPSE. Bragança *et al*. demonstrated that this basic echocardiographic measure has pathophysiological and predictive significance in HF patients receiving CRT.[30]

COVID-19-induced acute respiratory distress syndrome (ARDS) is linked with early and severe RV-arterial uncoupling, and the TAPSE/PASP contributes substantially and independently to the PaO2/FIO2 ratio in these patients. These data support bedside echocardiograms in COVID-19 patients.[31] TAPSE/PASP predicts adverse outcome better in acute intermediate-risk pulmonary embolism (PE). It may enhance risk stratification and identify people who will deteriorate following intermediate-risk PE.[32] TAPSE/PASP plays a key function in detecting RV-PC coupling and gives prognostic insights in SLE-associated PAH. Low TAPSE/PASP and low 6MWD may indicate a high risk of poor prognosis and be a focus for more aggressive care.[33]

In terms of the RV-PA unit's integrated evaluation, the preoperative TAPSE/PASP and PVR index were strong predictors of remaining symptoms following atrial septal defect closure.[34] Normalizing RV echocardiographic parameters did not enhance prognosis, according to Ishiwata *et al*.[35] In patients with HF due to dilated cardiomyopathy, combining fractional area change (FAC), and RV longitudinal strain enhanced risk stratification. TAPSE/PASP may enhance risk classification and candidate selection in MitraClip patients.[36] TAPSE/PASP is connected with RV fibrosis which boosts its prognostic importance in ischemic and nonischemic cardiomyopathy patients.[37] In mechanically ventilated (MV) septic shock patients, the TAPSE/PASP predicted 1-year all-cause death, intensive care unit mortality, and MV duration.[38]

Middle-aged people had lower resting and exercise RV-PA coupling.[39] Mild-to-moderate asthma is linked to RV dysfunction and higher pulmonary arterial stiffness (PAS). TAPSE/PASP was similarly lowered, showing RV-PA uncoupling even without PAH. RV afterload predicts TAPSE/PASP independently.[40] Because the TAPSE/PASP assesses RV systolic function, it may predict mortality in transcatheter aortic valve implantation (TAVI) patients. It may help with prognosis in TAVI patient selection.[41]

LIMITATIONS

TAPSE denotes RV shortening. TAPSE reaches a minimum as RV dysfunction worsens. In later phases of RV breakdown, TAPSE's dependability as a function parameter (and the TAPSE/PASP usefulness) may be diminished. The TAPSE/PASP value in failing hearts or acute decompensated states was outside the scope of our research. In addition, intra- and interobserver variability may restrict TAPSE/PASP. Pulmonary veno-occlusive disease may have affected the prognostic power of the TAPSE/PASP, since they had a greater mortality than PAH patients.

The right and LVs share the interventricular septum and pericardium, which leads to the interdependence of ventricular function. LV failure can result in elevated left atrial pressure, pulmonary venous congestion, and ultimately, heightened pulmonary artery pressures. When left heart pressures increase, the TAPSE/PASP ratio may change. This change not only indicates the function of the RV, but also reflects a combination of both RV and LV pathologies. In cases of LV failure, an increase in pulmonary artery systolic pressure (PASP) may result in a decreased ratio of TAPSE/PASP. People may mistakenly perceive this as RV dysfunction. Nevertheless, the main problem may originate from the left side of the heart, causing subsequent impacts on the right side of the heart. Accurately diagnosing and managing primary RV dysfunction versus secondary RV dysfunction caused by LV illness is essential.[42]

THE EFFECTS OF VALVE DISEASES

TR can lead to an incorrect reading of (PASP) because there is less pressure difference across the tricuspid valve than expected. A misleading increase in the TAPSE/PASP ratio may indicate a higher level of RV function than the actual observation. On the other hand, if the TR jet is not correctly orientated during Doppler evaluation, there is a possibility of overestimating PASP, which can result in a falsely low TAPSE/PASP ratio. Mitral Valve Disease: If you have mitral stenosis or regurgitation, the pressure in the left atrium can rise. This can lead to PAH and, in turn, a high PASP. This can complicate the TAPSE/PASP ratio, particularly if RV function is largely intact. In cases of mitral valve illness, the TAPSE/PASP ratio may not provide an adequate assessment of the right RV function because left-sided valvular pathology is the main cause of increased PASP.[43]

ANALYSING TRICUSPID ANNULAR PLANE SYSTOLIC EXCURSION/PASP IN CASES OF DUAL VENTRICULAR DYSFUNCTION

Interpreting the TAPSE/PASP ratio becomes more difficult when patients have malfunctions in both the left and RVs. Significant RV failure, increased PASP due to LV dysfunction, or a combination of both factors could account for a low ratio. It may be necessary to use clinical judgement and additional imaging techniques such as MRI or three-dimensional echocardiography to differentiate the individual contributions of each ventricle to the overall hemodynamic situation. When doing a holistic assessment, it is crucial to take into account the TAPSE/PASP ratio alongside other clinical and echocardiographic findings. These include the size of the RV, FAC, size of the left atrium, and the existence of substantial valvular disease. An all-encompassing evaluation that incorporates these characteristics can provide a more precise understanding of the patient's total heart function and direct appropriate treatment.[44]

ADDITIONAL VARIABLES COULD POTENTIALLY INFLUENCE THE RESULTS

Preload and afterload conditions affect heart blood flow before and after contraction. Both TAPSE and PASP can be affected by changes in preload (like low or high blood volume) or afterload (like high BP or a blockage in the lungs). This can change the ratio between them. The TAPSE/PASP ratio can vary depending on whether the patient is experiencing an acute or chronic disease. Temporary variations in the ratio due to sudden changes in the application of a load may not accurately reflect the stable function of the RV.[44]

POTENTIAL FUTURE RESEARCH DIRECTIONS

Examine the significance of the TAPSE/PASP ratio in individuals with cardiometabolic illnesses such as obesity, diabetes, and metabolic syndrome, where there is a growing awareness of RV dysfunction. Gaining insight into its usefulness in these populations could result in a better assessment of risk and treatment. Look into how the TAPSE/PASP ratio can be used in critical care settings, especially for people who have been diagnosed with ARDS, sepsis, or an acute PE. During these critical circumstances, researchers were able to ascertain whether the ratio can function as a prognosis indicator or assist in making therapeutic choices.[45]

Develop AI-based software to automate TAPSE and PASP measurement from echocardiographic images. Research should prioritise examining the precision, uniformity, and practical value of AI-supported computations in comparison to manual techniques. Explore novel echocardiographic methodologies or protocols that have the potential to improve the precision of Doppler measurements employed in the TAPSE/PASP ratio. This may entail optimizing the transducer's angles or utilizing contrast agents to improve the visualization of the TR jet. Conduct a study to determine the efficacy of the TAPSE/PASP ratio in predicting the prognosis of elderly patients with complex heart diseases, such as HF with preserved ejection fraction and age-related PAH. Look into whether the TAPSE/PASP ratio works well for kids and people who were born with heart problems that cannot be fixed. Pay special attention to how well it can predict outcomes and help guide treatment in these groups.[45]

Do longitudinal studies to look at how the TAPSE/PASP ratio changes over time in people who have long-term illnesses such as HF, PAH, or chronic obstructive pulmonary disease. Research could ascertain whether successive alterations in the ratio are associated with the advancement of a disease or the effectiveness of a treatment. Look into the possibility of adding more clinical and imaging factors to the TAPSE/PASP ratio to make risk scores that are more accurate at predicting bad outcomes in people with CV conditions. This has the potential to improve individualized care and treatment planning. Research should prioritise the establishment of normative values for the TAPSE/PASP ratio in various populations, encompassing diverse ethnicities, age groups, and genders. This could help refine the interpretation of the ratio and enhance its therapeutic significance. When quantifying the TAPSE/PASP ratio, researchers should investigate ways to minimize disparities among and within observers. For instance, they could employ established measurement techniques or utilize more sophisticated imaging technologies. They could conduct clinical trials to evaluate the effectiveness of the TAPSE/PASP ratio in monitoring the response to therapeutic interventions such as pulmonary vasodilators, diuretics, or RV-specific medicines.[45]

This study has the potential to ascertain if the ratio can serve as a guiding factor for making therapy adjustments and enhancing patient outcomes. Examine the use of the TAPSE/PASP ratio in surgical decision‑making, specifically in patients undergoing procedures such as mitral valve replacement, LV assist device installation, or heart transplantation. Research could ascertain whether the ratio is indicative of postoperative RV function and outcomes. Create and evaluate training programs for echocardiographers with the goal of enhancing the precision and uniformity of TAPSE/PASP ratio readings. Research should assess the effects of these programs on clinical outcomes and the wider acceptance of the TAPSE/PASP ratio in clinical guidelines. Establishing a standardised method for reporting TAPSE/PASP ratios in echocardiography guidelines should be a priority. Research could facilitate the establishment of consensus guidelines about the appropriate use of the ratio in various therapeutic contexts.

CONCLUSION

In patients with advanced HF, a drop in the TAPSE/PASP (i.e. RV‑arterial uncoupling), which quantitatively represents the function of the RV, was observed to be linked. TAPSE/PASP is closely linked with hemodynamics and functional class in PAH patients. The TAPSE/PASP may also be helpful in diseases affecting right cardiac function.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES

1. Guazzi M, Bandera F, Pelissero G, Castelvecchio S, Menicanti L, Ghio S, *et al.* Tricuspid annular plane systolic excursion and pulmonary arterial systolic pressure relationship in heart failure: An index of right ventricular contractile function and prognosis. Am J Physiol Heart Circ Physiol 2013;305:H1373‑81.
2. Giardini A. Limitations inherent to the simplified bernoulli equation explain the inaccuracy of Doppler echocardiographic estimates of pulmonary artery pressures in patients with pulmonary hypertension. Chest 2011;140:270.
3. D'Alto M, Pavelescu A, Argiento P, Romeo E, Correra A, Di Marco GM, *et al*. Echocardiographic assessment of right ventricular contractile reserve in healthy subjects. Echocardiography 2017;34:61‑8.
4. Guazzi M, Dixon D, Labate V, Beussink‑Nelson L, Bandera F, Cuttica MJ, *et al*. RV contractile function and its coupling to pulmonarycirculation in heart failure with preserved ejection fraction: stratification of clinical phenotypes and outcomes. JACC Cardiovasc Imaging 2013;10:1211‑21.
5. Vonk‑Noordegraaf A, Haddad F, Chin K. Right heart adaptation to pulmonary arterial hypertension: physiology and pathobiology. J Am Coll Cardiol 2013;62:22‑33.
6. Vonk‑Noordegraaf A, Westerhof N. Describing right ventricular function. Eur Respir J 2013;41:1419‑23.
7. Faragli A, Tanacli R, Kolp C, Abawi D, Lapinskas T, Stehning C, *et al*. Cardiovascular magnetic resonance‑derived left ventricular mechanics‑strain, cardiac power and end‑systolic elastance under various inotropic states in swine. J Cardiovasc Magn Reson 2020;22:79.
8. Naeije R, Manes A. The right ventricle in pulmonary arterial hypertension. Eur Respir Rev 2014;23:476‑87.
9. Berlin DA, Bakker J. Understanding venous return. Intensive Care Med 2014;40:1564‑6.
10. Melenovsky V, Kotrc M, Borlaug BA, Marek T, Kovar J, Malek I, *et al*. Relationships between right ventricular function, body composition, and prognosis in advanced heart failure. J Am Coll Cardiol 2013;62:1660‑70.
11. Naeije R, Brimioulle S, Dewachter L. Biomechanics of the right ventricle in health and disease. Pulm Circ 2014;4:395‑406.
12. Voelkel NF, Gomez‑Arroyo J, Abbate A, Bogaard HJ. Mechanisms of right heart failure—a work in progress and a plea for failure prevention. Pulm Circ 2013;3:137‑43.
13. Tello K, Seeger W, Naeije R, Vanderpool R, Ghofrani HA, Richter M, *et al*. Right heart failure in pulmonary hypertension: Diagnosis and new perspectives on vascular and direct right ventricular treatment. Br J Pharmacol 2021;178:90‑107.
14. Pinsky MR. Dynamic right and left ventricular interactions in the pig. Exp Physiol 2020;105:1293‑15.
15. Al‑Naamani N, Preston IR, Hill NS, Roberts KE. The prognostic significance of pulmonary arterial capacitance in pulmonary arterial hypertension: single‑center experience. Pulm Circ 2016;6:608‑10.
16. Galiè N, Humbert M, Vachiery JL, Gibbs S, Lang I, Torbicki A, *et al.* 2015 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension: The Joint Task Force for the Diagnosis and Treatment of Pulmonary Hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS): Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC), International Society for Heart and Lung Transplantation (ISHLT). Eur Heart J 2016;37:67‑119.
17. Harrison A, Hatton N, Ryan JJ. The right ventricle under pressure: evaluating the adaptive and maladaptive changes in the right ventricle in pulmonary arterial hypertension using echocardiography (2013 Grover Conference series). Pulm Circ 2015;5:29‑47.
18. Vonk Noordegraaf A, Westerhof BE, Westerhof N. The Relationship Between the Right Ventricle and its Load in Pulmonary Hypertension. J Am Coll Cardiol 2017;69:236‑43.
19. Gorter TM, van Veldhuisen DJ, Voors AA, Hummel YM, Lam CSP, Berger RMF, *et al*. Right ventricular‑vascular coupling in heart failure with preserved ejection fraction and pre‑ vs. post‑capillary pulmonary hypertension. Eur Heart J Cardiovasc Imaging 2018;19:425‑32.
20. Gerges M, Gerges C, Pistritto AM, Lang MB, Trip P, Jakowitsch J, *et al*. Pulmonary Hypertension in Heart Failure. Epidemiology, Right Ventricular Function, and Survival. Am J Respir Crit Care Med 2015;192:1234‑46.
21. Guo X, Lai J, Wang H, Tian Z, Wang Q, Zhao J, *et al*. Predictivevalue of non‑invasive right ventricle to pulmonary circulationcoupling in systemic lupus erythematosus patients withpulmonary arterial hypertension. Eur Heart J Cardiovasc Imaging 2021;22:111‑8.
22. Ghio S, Pica S, Klersy C, Guzzafame E, Scelsi L, Raineri C, *et al*. Prognostic value of TAPSE after therapy optimisation in patients with pulmonary arterial hypertension is independent of the haemodynamic effects of therapy. Open Heart 2016;3:e000408.
23. Tello K, Axmann J, Ghofrani HA, Naeije R, Narcin N, Rieth A, *et al*. Relevance of the TAPSE/PASP ratio in pulmonary arterial hypertension. Int J Cardiol 2018;266:229‑35.

24. Schmeisser A, Rauwolf T, Groscheck T, Kropf S, Luani B, Tanev I, *et al*. Pressure-volume loop validation of TAPSE/PASP for right ventricular arterial coupling in heart failure with pulmonary hypertension. Eur Heart J Cardiovasc Imaging 2021;22:168-76.
25. Tello K, Wan J, Dalmer A, Vanderpool R, Ghofrani HA, Naeije R, *et al*. Validation of the Tricuspid Annular Plane Systolic Excursion/Systolic Pulmonary Artery Pressure Ratio for the Assessment of Right Ventricular-Arterial Coupling in Severe Pulmonary Hypertension. Circ Cardiovasc Imaging 2019;12:e009047.
26. Miñana G, Santas E, de la Espriella R, Núñez E, Lorenzo M, Núñez G, *et al*. Right ventricular function and iron deficiency in acute heart failure. Eur Heart J Acute Cardiovasc Care 2021;10:406-14.
27. Vriz O, Pirisi M, Bossone E, Fadl ElMula FE, Palatini P, Naeije R. Right ventricular-pulmonary arterial uncoupling in mild-to-moderate systemic hypertension. J Hypertens 2020;38:274-81.
28. Stassen J, Galloo X, Hirasawa K, Chimed S, Marsan NA, Delgado V, *et al.* Right ventricular-pulmonary artery coupling in cardiac resynchronization therapy: Evolution and prognosis. ESC Heart Fail 2022;9:1597-607.
29. Deaconu S, Deaconu A, Scarlatescu A, Petre I, Onciul S, Vijiac A, *et al*. Right ventricular-arterial coupling – A new perspective for right ventricle evaluation in heart failure patients undergoing cardiac resynchronization therapy. Echocardiography 2021;38:1157-64.
30. Bragança B, Trêpa M, Santos R, Silveira I, Fontes-Oliveira M, Sousa MJ, *et al.* Echocardiographic assessment of right ventriculo-arterial coupling: Clinical correlates and prognostic ımpact in heart failure patients undergoing cardiac resynchronization therapy. J Cardiovasc Imaging 2020;28:109-20.
31. D'Alto M, Marra AM, Severino S, Salzano A, Romeo E, De Rosa R, *et al.* Right ventricular-arterial uncoupling independently predicts survival in COVID-19 ARDS. Crit Care 2020;24:670.
32. Lyhne MD, Kabrhel C, Giordano N, Andersen A, Nielsen-Kudsk JE, Zheng H, *et al.* The echocardiographic ratio tricuspid annular plane systolic excursion/pulmonary arterial systolic pressure predicts short-term adverse outcomes in acute pulmonary embolism. Eur Heart J Cardiovasc Imaging 2021;22:285-94.
33. Guo X, Lai J, Wang H, Tian Z, Wang Q, Zhao J, *et al*. Predictive value of non-invasive right ventricle to pulmonary circulation coupling in systemic lupus erythematosus patients with pulmonary arterial hypertension. Eur Heart J Cardiovasc Imaging 2021;22:111-8.
34. Suzuki M, Matsumoto K, Tanaka Y, Yamashita K, Shono A, Sumimoto K, *et al*. Preoperative coupling between right ventricle and pulmonary vasculature is an important determinant of residual symptoms after the closure of atrial septal defect. Int J Cardiovasc Imaging 2021;37:2931-41.
35. Ishiwata J, Daimon M, Nakanishi K, Sugimoto T, Kawata T, Shinozaki T, *et al*. Combined evaluation of right ventricular function using echocardiography in non-ischaemic dilated cardiomyopathy. ESC Heart Fail 2021;8:3947-56.
36. Trejo-Velasco B, Estevez-Loureiro R, Carrasco-Chinchilla F, Fernández-Vázquez F, Arzamendi D, Pan M, *et al*. Prognostic Role of TAPSE to PASP Ratio in Patients Undergoing MitraClip Procedure. J Clin Med 2021;10:1006.
37. Keranov S, Haen S, Vietheer J, Rutsatz W, Wolter JS, Kriechbaum SD, *et al*. Application and Validation of the Tricuspid Annular Plane Systolic Excursion/Systolic Pulmonary Artery Pressure Ratio in Patients with Ischemic and Non-Ischemic Cardiomyopathy. Diagnostics (Basel) 2021;11:2188.
38. Zhang H, Lian H, Zhang Q, Chen X, Wang X, Liu D. Prognostic implications of tricuspid annular plane systolic excursion/pulmonary arterial systolic pressure ratio in septic shock patients. Cardiovasc Ultrasound 2020;18:20.
39. Forton K, Motoji Y, Caravita S, Faoro V, Naeije R. Exercise stress echocardiography of the pulmonary circulation and right ventricular-arterial coupling in healthy adolescents. Eur Heart J Cardiovasc Imaging 2021;22:688-94.
40. Karasu BB, Aydıncak HT. Right ventricular-pulmonary arterial uncoupling in mild-to-moderate asthma. J Asthma 2022:1-10.
41. Sultan I, Cardounel A, Abdelkarim I, Kilic A, Althouse AD, Sharbaugh MS, *et al*. Right ventricle to pulmonary artery coupling in patients undergoing transcatheter aortic valve implantation. Heart 2019;105:117-21.
42. Dandel M, Hetzer R. Evaluation of the right ventricle by echocardiography: particularities and major challenges. Expert Rev Cardiovasc Ther 2018;16:259-75.
43. Bahira S, Hahn RT. Right ventricular-pulmonary arterial coupling and outcomes in heart failure and valvular heart disease. Structural Heart 2021;5:128-39.
44. Ghio S, Guazzi M, Scardovi AB, Klersy C, Clemenza F, Carluccio E, *et al*. Different correlates but similar prognostic implications for right ventricular dysfunction in heart failure patients with reduced or preserved ejection fraction. Eur J Heart Fail 2017;19:873-9.
45. Guazzi M. Use of TAPSE/PASP ratio in pulmonary artery hipertension. An easy shortcut in a cogested road. Int J Cardiol 2018;266:242:4.