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REVIEW

POCUS series: right ventricular assessment with emphasis on TAPSE, apical and subcostal variants

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Abstract

In the last decade, ultrasound has found its place in the intensive care unit (ICU). Initially, ultrasound was used primarily to increase the safety and efficacy of line insertion, but currently many intensivists use point-of-care ultrasound (POCUS) to aid in making the diagnosis, monitoring therapy, and supporting therapeutic interventions. In this series, we aim to highlight one specific POCUS technique at a time, which we believe will prove to be useful in your clinical practice. This specific article will focus on assessment of right ventricular (RV) size and function, and the application of tricuspid annular plane systolic excursion (TAPSE). RV assessment during focused cardiac ultrasound (FoCUS) depends, currently, on visual evaluation – 'eyeballing' – of the RV size and function and left and right ventricular interaction. However, 'eyeballing' is subjective, depends on experience and may be misleading if done by unexperienced sonographers. Objective measurements of RV size and function are necessary and provide an additional understanding of RV performance. There are different ways to assess the RV objectively. Many of these measurements, however, require a lot of training and are not yet available in portable devices. Evaluation of TAPSE is a validated and reproducible way of evaluating RV function and only requires the utilisation of M-mode or a 2D measurement. TAPSE, assessed in the apical four-chamber view, is sometimes difficult to measure, especially in mechanically ventilated patients. In recent years subcostal variants have been introduced: the subcostal echocardiographic assessment of tricuspid annular kick (SEATAK) and the subcostal-TAPSE (S-TAPSE). These measurements are alternatives when the 'classical' TAPSE cannot reliably be evaluated.

Introduction

This article is part of the POCUS series in the Netherlands Journal of Critical Care, in which we want to highlight POCUS techniques that will improve decision-making in daily clinical practice in the intensive care unit (ICU). We aim to provide intensivists with an overview of easy, quick, and reliable methods which may be useful in their practice.

Focused cardiac ultrasound (FoCUS) allows a quick evaluation of both the left (LV) and right ventricular (RV) size and function; it has revolutionised the evaluation of haemodynamically compromised critically ill patients. The strength of FoCUS is that it can be applied in a timely fashion, providing an 'early' or 'preliminary' assessment and management of these patients.[1] Another strength is that it does not require big complex and expensive ultrasound machines. And more importantly, FoCUS assessment of both the left and right ventricle only requires visual assessment: 'eyeballing' or simple measurements.

A subjective assessment of both ventricles has, unfortunately, its limitations.[1] In recent issues we have focused on the application of simple and reliable measurements, such as the E-point septal separation and the velocity time integral, to better characterise LV function and performance.[2,3] In this issue, we will focus on the right ventricle and dive deeper in the application of the tricuspid annular plane systolic excursion (TAPSE) and recently introduced subcostal variants such as the subcostal echocardiographic assessment of tricuspid annular kick (SEATAK) and the subcostal TAPSE (S-TAPSE).[4,5]

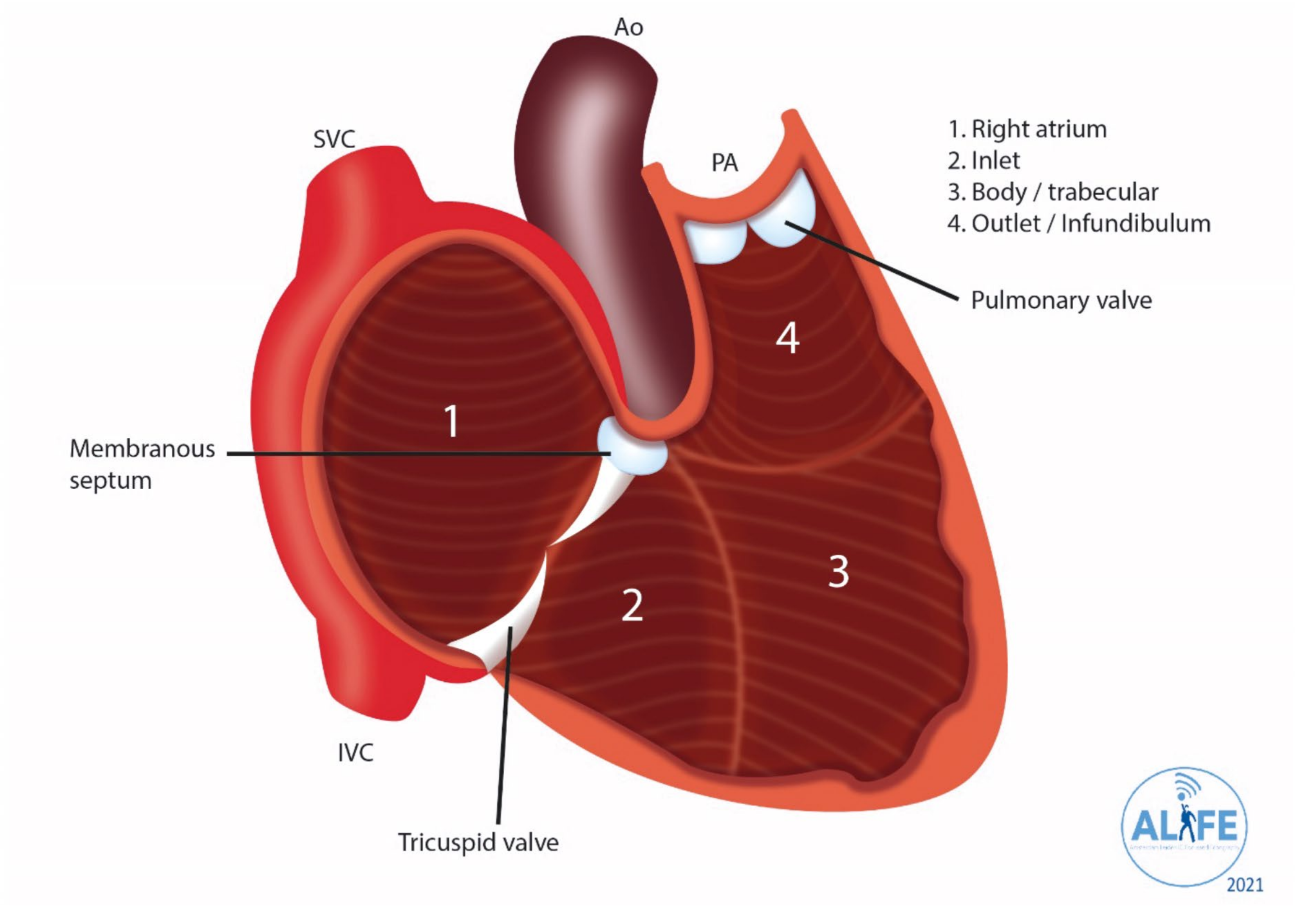

Figure 1. Diagram of the right atrium (1) and ventricle with its three major components: inflow tract (2), body (i.e., apex, 3) and outlet tract (i.e., infundibulum, 4). Adapted from: https://thoracickey.com/right-heart-anomalies/
SVC = superior vena cava; Ao = aorta; PA = pulmonary artery; IVC = inferior vena cava

RV anatomy and contraction pattern

The right ventricle, once the 'forgotten' ventricle, has regained new attention, both in cardiology and in critical care.[6,7] In the ICU, RV function can indirectly be assessed using a pulmonary artery catheter (RV haemodynamics, pulmonary vascular resistance calculation), or importantly, directly by chocardiography.[8] The echocardiographic assessment of the right ventricle is, however, difficult, because of a complex geometrical structure.

The right ventricle can be divided into three parts *(figure 1)*:[9]

A. The inflow containing the tricuspid valve and valvular apparatus
B. The trabeculated immobile apical region
C. The funnel-shaped outflow (i.e., infundibulum)

The right ventricle's myocytal organisation and its resulting contractional pattern is very different from that of the left ventricle. In comparison to the three-layered myocytal arrangement of the left ventricle only two different layers can be identified. In the superficial – epicardial – layer (20-25% of the wall thickness), the myocytes are organised in a circumferential fashion. In the subendocardial layer the organisation is longitudinal, extending from the apex to the tricuspid annulus.[10]

The right ventricle contracts in a peristaltic-like fashion: starting at the inlet and ending at the infundibulum *(figure 2)*. After tricuspid valve closure, RV systole begins by activation and subsequent contraction of the myocytes in the subepicardial layer of the inflow. During this isovolumetric face *(figure 2B)*, the right ventricle is circumferentially – radially – deformed, creating a 'bellow effect' by inward movement of the RV free wall. During the ejection phase *(figure 2A)*, the longitudinal myocardial fibres in the subendocardial layer (75% of total RV ejection) shorten, pulling the tricuspid annulus towards the apex (i.e., TAPSE). At the end of systole the infundibulum contracts *(figure 2C)*, contributing to 15-20% of RV ejection. LV contraction also contributes significantly to RV ejection via shared myocardial fibres, intraventricular septum and pericardium (i.e., ventricular interdependence). The interventricular septum plays a crucial role in this regard, through longitudinal shortening during the ejection phase.[11,12]

The predominately longitudinal contraction pattern changes in pathological states: there is a decline in both longitudinal and radial contraction (radial decline is more pronounced) in pressure overload and an increase in longitudinal contraction

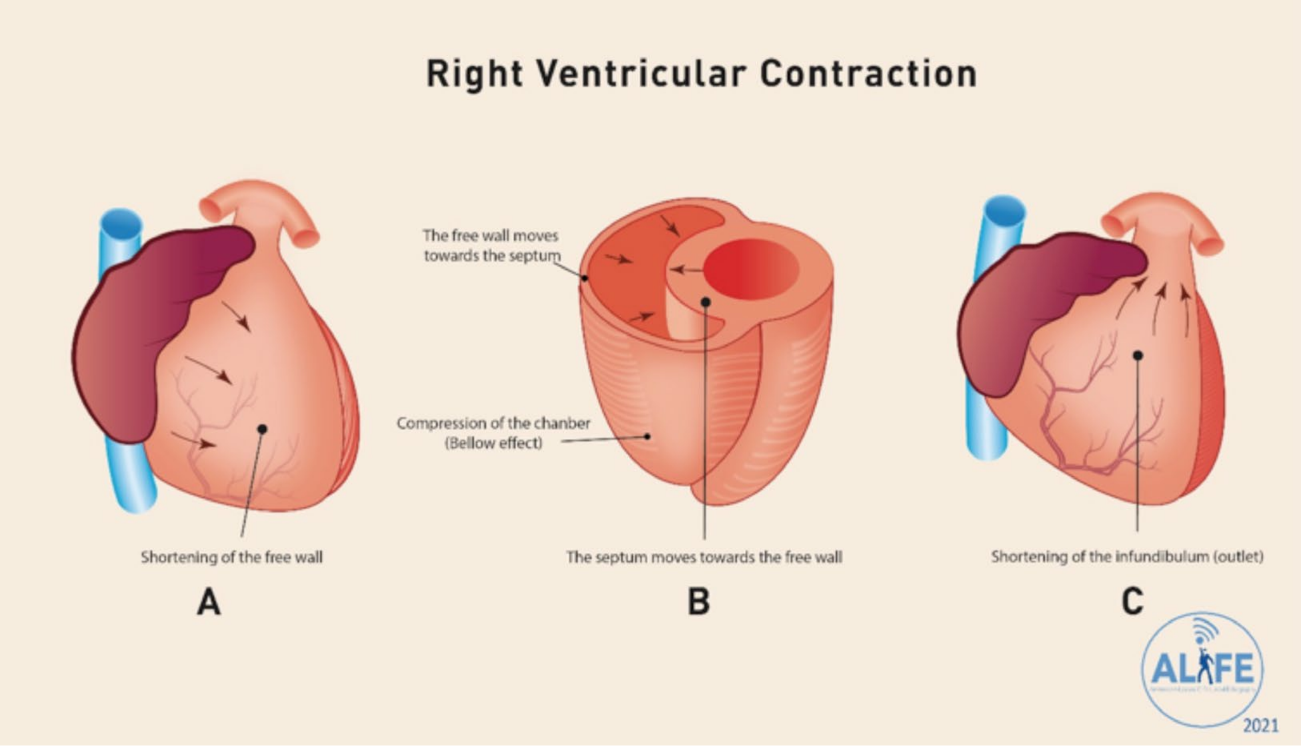

Figure 2. Diagram of right ventricular contraction. A. Ejection phase: shortening of the free wall and pulling the tricuspid annulus towards the apex (i.e., TAPSE). B. Isovolumetric face: the right ventricle is circumferentially – radially – deformed, creating a 'bellow effect' by inward movement of the RV free wall. C. End of contraction: infundibulum shortens, and blood is ejected into the common pulmonary artery. Adapted from https://aneskey.com/cardiac-anatomy-and-physiology-3/.

in volume overload.[12] Interestingly, after pericardiotomy, there is a loss of longitudinal contraction (i.e., TAPSE) with a compensatory increase in the RV free wall and concentric contraction, thereby maintaining the global RV systolic function. This demonstrates the pivotal role an intact pericardium plays in allowing normal longitudinal contraction.[12,13]

Echocardiographic assessment

In echocardiography, the right ventricle appears triangular when viewed from the front and has a crescent-like shape in a transverse view. To have a comprehensive FoCUS view of the right ventricle, different views have to be evaluated:[9]

1. Parasternal long-axis view: proximal right ventricular outflow tract (RVOT) diameter, free wall contraction and LV/RV ratio
2. Parasternal short-axis view at papillary muscle level: RV dimension, septal position and kinetics
3. Parasternal short-axis view at aortic valve level: proximal RVOT diameter
4. Apical four-chamber (A4C) view: qualitative assessment of LV and RV function and dimensions, and LV/RV ratio
5. Subcostal four-chamber view (S4C): qualitative assessment of LV and RV function and dimensions, and LV/RV ratio
6. Subcostal inferior vena cava (IVC) view: IVC diameter and respiratory variations.

There is an additional view, the 'focused' RV view, which can give a better evaluation of the RV free wall. From the A4C view, the transducer is moved more laterally on the patient, and rotated until the maximal diameter of the base can be seen.

Visual versus quantitative assessment

The above-explained 'comprehensive' assessment is done mainly by visual inspection — 'eyeballing'. But how good are we at 'eyeballing'?

Several studies have investigated the accuracy of 'eyeballing' as a way of evaluating the RV size and function.[5,14-17] These studies, conducted in different settings, demonstrated that 'eyeballing' has limited accuracy and high interrater variability for detecting RV dysfunction as compared with cardiac magnetic resonance imaging (cMRI) – the 'gold standard'– or by 3D or speckle-tracking echocardiography. Accuracy improves and variability decreases when only differentiating normal from abnormal RV function, with the level of experience (experts 'outperform' beginners) and more importantly, by adding a quantitative assessment to the evaluation.[5,15-17]

Not surprisingly, current guidelines recommend that RV function should not only be assessed visually but also be evaluated by at least one additional quantitative parameter.[18] There are many objective echocardiographic parameters: TAPSE, tissue Doppler imaging of the RV free wall (RV S'), RV index of myocardial

Table 1. TAPSE and subcostal variants SEATAK and S-TAPSE

Echo parameter	View	Modus	Abnormal value	Reference
TAPSE	A4C	MM	< 17 mm	[28]
TAPSE	A4C	2D	< 17 mm	[44]
SEATAK	S4C	MM	< 16 mm	[4, 42, 43]
S-TAPSE	S4C	2D	ND	[24]

TAPSE = tricuspid annular plane systolic excursion, s-TAPSE = subcostal TAPSE, SEATAK = subcostal echocardiographic assessment of tricuspid annular kick, 4AC = apical four chamber view, S4C = subcostal four-chamber view, 2D= two dimensions, MM = M-mode echocardiography, ND = not (yet) defined

performance (RIMP) or Tei index, and fractional area change (FAC).[19,20] More recent additions encompass RV strain analysis and 3D RV-ejection fraction measurement.[9] Excluding TAPSE and probably FAC, these parameters require additional training and, more importantly, more computational power (i.e., Doppler tissue imaging, speckle tracking, 3D analysis) which are not yet available in portable devices.

TAPSE was one of the first quantitative measurements introduced to objectively assess RV function and is, besides eyeballing, still the most widely used method.[21,22] It has been incorporated in many international FoCUS training programs and was recently selected as a core critical care ultrasound competency to assess RV function by the European Society of Intensive Care Medicine.[23,24]

But is it necessary to include measurements in a FoCUS assessment as ICU physicians seem able to accurately quantify RV size and function (including visual evaluation of tricuspid valve displacement, i.e., TAPSE) visually?[25] A study conducted in the emergency department (ED) shed some light on this discussion: after training and performing at least 20 TAPSE measurements, ED fellows and residents could visually estimate TAPSE with good accuracy.[26] They concluded: '… once a practitioner has gained some experience measuring TAPSE, it is reasonable to visually estimate TAPSE and only measure it using M-mode in cases of uncertainty'.

Unfortunately, TAPSE cannot always be obtained reliably (e.g., because of mechanical ventilation, wound dressing); then, what often remains is the subcostal view, 'the rescue window for the intensivist'.[27] In this situation two recently introduced subcostal variants - SEATAK and S-TAPSE - could be useful.[4,5] Taken together, the general intensivist can improve his FoCUS skills by measuring TAPSE and variants. It not only helps to improve his visual assessment but also contributes to the characterisation and prognosis of critically ill patients (see Applications of TAPSE).

TAPSE, SEATAK and subcostal TAPSE

TAPSE

TAPSE, first reported by Kaul and colleagues in 1984, is the distance (in millimetres) the tricuspid annulus travels towards the RV apex, between end-diastole and end-systole.[21] Therefore, it quantifies the longitudinal contraction aspect of the right ventricle.

TAPSE is measured in the A4C view by M-mode *(figure 3A)* or 2D *(figure 3B)* echocardiography. M-mode is more commonly used, but both methods yield similar results.[28] We prefer M-mode because it allows a higher temporal resolution. After obtaining the 4AC view, the M-mode is activated, and the M-mode line is aligned parallel to the motion of the tricuspid annulus *(figure 3A)*. According to cardioserv.net[29] there are different ways to optimise the measurement: decreasing the gain to avoid 'noise artefacts', using a medium-fast M-mode speed (~50 mm/s), looking for a consistent signal throughout systole and diastole, and measuring vertically via a leading-edge to leading-edge method.

An average of three measurements should be used, with a minimum of five when the patient is in atrial fibrillation.[30] The normal TAPSE value in healthy subjects is 24.0±3.5 mm, more or less independent of sex or age.[31] A TAPSE <17 mm is considered abnormal *(table 1)*.[18] Normal and abnormal values have not yet been clearly defined in critically ill patients.

Whether TAPSE is preload dependent is debatable. Some studies have found a good correlation between end-diastolic volume (preload) and TAPSE.[31,32] However, acute fluid administration in healthy volunteers and in critically ill patients did not affect TAPSE, and fluid withdrawal in haemodialysis patients revealed contradictory results.[33-36]

TAPSE correlates well (r~0.90) with the global RV function, measured by radionuclide angiography, 3D echocardiography or cMRI.[21,37,38] In critically ill patients it correlates with thermodilution-derived RV ejection fraction (r=0.78).[39] TAPSE can therefore serve as a surrogate marker for the RV systolic function. This relationship is unsurprising since longitudinal contraction determines 75-80% of RV ejection.[11] In some pathological situations, this coupling is, however, lost: in patients with LV dysfunction (especially with reduced longitudinal septal motion), regional wall motion abnormalities (TAPSE measures only a small RV segment), severe tricuspid regurgitation (overestimation of global function due to malalignment between transducer and tricuspid annulus),[40-42] and after pericardiotomy (loss op pericardial integrity).[13]

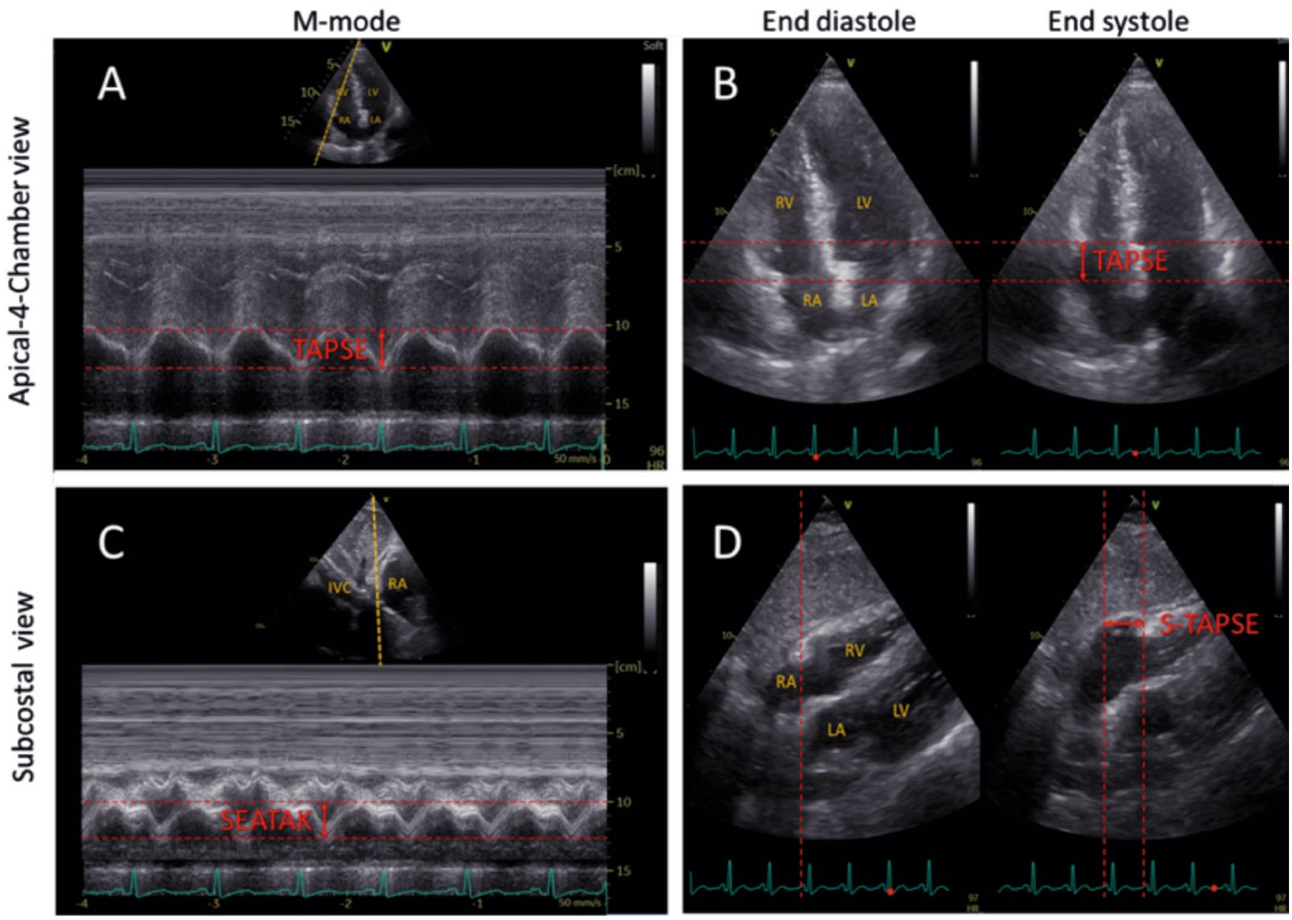

Figure 3. Measurement of tricuspid annular plane systolic excursion (TAPSE), subcostal echocardiographic assessment of tricuspid annular kick (SEATAK) and the subcostal-TAPSE (S-TAPSE)

A: Apical-4-chamber view (A4C): M-mode TAPSE measurement. In the A4C view, a M-mode line is aligned parallel to the motion of the tricuspid annulus: TAPSE is the distance in millimetres (mm) between minimum tot maximal excursion

B: A4C view: 2D TAPSE measurement. In the A4C view a cursor is positioned over the TA in end-diastole, the image is then progressed to end-systole, where a second cursor is placed over the TA. TAPSE is the distance (in mm) between these two points

C: Subcostal short-axis view: SEATAK (M-mode) measurement. In the subcostal-4-chamber (S4C) view the probe is turned counter-clockwise and a M-mode line is aligned with the TA: SEATAK is distance in mm between the minimum and maximal excursion

D: S4C view: 2D S-TAPSE measurement. In the S4C view, a cursor is positioned at the TA in end-diastole, the image is then progressed to end-systole, and a second cursor is placed at the TA. S-TAPSE is the distance between these two points (in mm)

RA = right atrium view; RV = right ventricle; LA = left atrium; LV = left ventricle. IVC = inferior vena cava

Subcostal echocardiographic assessment of tricuspid annular kick

SEATAK is measured by first obtaining the S4C view. The probe is subsequently rotated counter-clockwise to obtain a subcostal short-axis view. This allows visualisation of the right atrium and ventricle, tricuspid annulus, and inferior vena cava *(figure 3C)*. After activating the M-mode, the M-mode line is aligned with the tricuspid annulus to obtain a linear measurement in millimetres from end-diastole to end-systole (tricuspid annular klick).

Díaz-Gómez et al. were the first to study SEATAK in critically ill patients. SEATAK and TAPSE were highly correlated (r=0.86) with a mean difference of -2.6 mm (95% CI -1.9 to -3.5 mm).[4] Similar results were recently found in cardiology patients (mean difference of 0.15 mm, r=0.82; p<0.0001). A SEATAK of ≥16 mm predicted normal RV function (FAC ≥35%) with a sensitivity of 86% and specificity of 67% *(table 1)*.[43] In children, SEATAK is also highly correlated to TAPSE, and the value increases with increasing age, body weight, body length, and body surface area in a nonlinear fashion.[44] Older adolescents have similar values as reported in adults by Díaz-Gómez et al.[4]

Subcostal TAPSE (S-TAPSE)

In the S4C view, the tricuspid annulus is first identified in diastole, and a cursor is positioned over it *(figure 3D)*. The image is then progressed to systole, where a second cursor is placed over the tricuspid annulus. The distance between these two points is the S-TAPSE.

In a retrospective study of 56 critical care patients, S-TAPSE and TAPSE were well correlated, with a mean difference of 1.2 mm (95% CI 0.04-2.36).[45] However, the Bland–Altman plot analysis demonstrated that the majority of measurements fell outside the confidence interval, limiting its applicability. S-TAPSE performed well when discriminating 'normal' from 'abnormal' TAPSE (sensitivity 97.8%, specificity 87.5%), so this may be used to quickly screen for abnormal RV function.

Applications of TAPSE

TAPSE has been evaluated in different critical care scenarios, such as mechanical ventilation, acute reparatory distress syndrome (ARDS), pulmonary embolism (PE), sepsis and recently in Covid-19 pneumonia.

TAPSE in ICU patients

The complex effect of mechanical ventilation on RV and LV function has been studied extensively. These studies demonstrated that mechanical ventilation, PEEP, and recruitment manoeuvres can have a detrimental effect, particularly in the already compromised right ventricle.[46]

TAPSE can decrease significantly after the start of positive mechanical ventilation, as was recently described in elective cardiac surgery patients (-1.6 mm, 95% CI -2.6 to -0.7 mm; p=0.0013).[47] However, 3D echocardiography derived RV-ejection fraction and stroke volume were not reduced, so the clinical implication of this finding is not fully understood. There is an inverse relationship between central venous pressure (CVD) and TAPSE nonetheless, especially in patients with a low LV ejection fraction (<55%). A high CVD and low TAPSE are both associated with the development of acute kidney injury after cardiac surgery and in a general ICU population.[48-50] A low TAPSE in combination with high LV filling pressures is a strong predictor of weaning failure.[51] Moreover, a low TAPSE is associated with an increased ICU length of stay, and even increased mortality.[52]

TAPSE in acute reparatory distress syndrome

Many studies have looked at the RV function, including measurement of TAPSE, in ARDS patients.[53] ARDS and mechanical ventilation affect RV function (i.e., TAPSE) through various mutually influencing mechanisms. Increased pulmonary vascular resistance (microcirculatory changes, low PO_2/FiO_2 <150 mmHg, and high pCO_2 ≥48 mmHg) and intrathoracic pressures (high driving pressures >18 cmH_2O and high PEEP >10 cmH_2O) negatively affect RV function and TAPSE.[53-56] On the other hand, prone position, lung protective mechanical ventilation and CO2 removal and, interestingly, a patent foramen ovale (unloading right ventricle), have a positive influence on TAPSE.[57,58]

In a meta-analysis of 16 studies (1661 patients) on lung-protective ventilation, the pooled incidence of acute cor pulmonale was 23% (95% CI 18-28%). About 27-48% of the patients (studied in five of the 16 studies) had a TAPSE <16 mm.[59] A reduced TAPSE <17 mm is correlated with the severity of ARDS, reflected by a low PO_2/FiO_2 ratio and high pCO_2, and is independently associated with increased mortality.[60,61] A sequential FoCUS RV evaluation (including TAPSE) could enable a timely selection of a more 'RV-protective ventilation' strategy, thereby reducing the incidence of cor pulmonale and positively impacting mortality.[53]

TAPSE and pulmonary embolism

Pulmonary embolism is a common diagnosis in the ICU and is associated with high ICU and in-hospital mortality.[62] In the setting of acute PE, pulmonary vascular resistance and RV afterload increase abruptly due to mechanical obstruction and release of vasoconstrictive mediators. An obstruction of >25-30% of the pulmonary vasculature is associated with an increase in pulmonary pressures and a 30% reduction in RV stroke volume; whilst an obstruction of 50-75% leads to RV failure.[63] This increased strain on the right ventricle results in RV dilatation and high wall stress, and later systolic dysfunction and septal deviation. Echocardiography plays a pivotal role in the diagnosis, risk stratification, and prognosis estimation.[64] Low TAPSE is strongly and independently associated with risk of death and decompensation in multiple studies, though the cut-off value varies between 15 and 18 mm.[63] It also predicts acute decompensation in patients undergoing acute pulmonary artery embolectomy.[65] According to Paczynska et al., patients with a TAPSE ≤15 mm should be admitted to the ICU and closely monitored; on the other hand, a TAPSE ≥18 mm identifies a very low-risk population.[63,66] TAPSE was recently successfully incorporated in a modified pulmonary embolism severity index (PESI) score, the PESI echo (PESI echo = PESI + pulmonary artery systolic pressure (PASP) -TAPSE). PESI echo was superior to PESI for predicting in-hospital mortality.[67]

TAPSE in sepsis

Sepsis-induced 'reversible' LV dysfunction, first described by Parker et al. in 1984, is well documented.[68] RV dysfunction, in up to 50% of cases, can also be found in sepsis and septic shock. However, its negative effect on mortality has been controversial.[69] Newer studies, with a total of 1569 patients, found a strong correlation between RV dysfunction (including TAPSE <16 mm) and 28-day mortality.[70-72] In three recent papers, a low TAPSE (optimal cut-off between 18-21 mm) or a high TAPSE/PASP ratio were independent factors of increased duration of mechanical ventilation, ICU mortality and one-year all-cause mortality.[73-75]

TAPSE in Covid-19 pneumonia

Recent studies have evaluated the impact of RV dysfunction in Covid-19 patients.[76-78] In a multicentre retrospective study of 164 Covid-19 patients, RV dilatation (38% of patients) and impaired RV systolic function (reduced FAC or TAPSE) were the main echocardiographic findings. LV function was usually preserved or hyperdynamic. Interestingly, a reduced TAPSE <17 mm was associated with an increased D-dimer and highly sensitive cardiac troponin. Reduced RV function was an independent predictor of all-cause mortality.[76] In a recent meta-analysis, every 1 mm decrease in TAPSE was associated with an increase in mortality of approximately 20%.[78]

Limitations

An important point to emphasise is that TAPSE only measures longitudinal RV function. However, since longitudinal contraction accounts for 75% of RV ejection, TAPSE has proven to be a good alternative for overall RV function in many situations. There are important exceptions to this rule: regional wall motion abnormalities (TAPSE measures only a small RV segment), severe tricuspid regurgitation (overestimation of RV function), and post-cardiac surgery (loss of pericardial integrity). Furthermore, in case of severe pressure overload, TAPSE does not reflect global RV function properly, since in these circumstances the longitudinal shortening is less important than the radial shortening.

Another limitation is that TAPSE, like any other M-mode measurement, is angle-dependent. Thus, a lot of effort has to be made to find the right alignment between the tricuspid annulus movement and the M-mode cursor.

Sometimes, proper alignment is not possible, then SEATAK or S-TAPSE appear to be adequate replacements. However, because both are measured in the subcostal view, this angle issue is even more pronounced. Furthermore, the influence of loading conditions, use of inotropic agents, and arrhythmia on SEATAK or S-TAPSE are currently unknown.

Conclusion

The right ventricle can visually be inspected or objectively evaluated using FoCUS. However, 'eyeballing' is, at best, a semi-accurate and highly experience-based way of assessing RV function. Many objective echocardiographic measurements are available to better characterise the right ventricle. TAPSE and, to a lesser extent, its subcostal counterparts - SEATAK and S-TAPSE - are easy and reliable ways to objectively assess RV function. Nevertheless, further research is needed prior to wide implementation of these TAPSE variants. In many critical care scenarios, TAPSE (and variants) are independent predictors of an increased duration of mechanical ventilation, ICU length of stay, and mortality. In addition, sequential TAPSE measurements can be used to monitor the effect of protective ventilation settings on the right ventricle. In our opinion, TAPSE should be a mandatory FoCUS assessment.

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Disclosures

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