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Maximal systolic excursion of the tricuspid annulus is independent of right atrial size and function in chronic pulmonary hypertension

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Abstract

Background—Even though the tricuspid annulus (TA) is anatomically and functionally related to right atrioventricular dependence and tricuspid annular plane systolic excursion (TAPSE) is a well-known measure of right ventricular (RV) systolic function, there is paucity of data regarding the potential impact that right atrial (RA) size and function have on TAPSE. Hence, we sought to determine whether RA volumetric and longitudinal measures affect TAPSE in patients with chronic pulmonary hypertension (cPH).

Methods—In this retrospective study, echocardiographic data from 110 patients were reviewed and the studied population was divided into Group I consisting of 50 patients (50±11 years) without PH and Group II that included 60 patients (55±14 years) with cPH.

Results—Even though RA areas, RA fractional area change, and maximal long-axis length measurements were abnormal in cPH patients, TA tissue Doppler imaging systolic velocity and RV fractional area change were the only useful variables in identifying patients with abnormal TAPSE ($P<.0001$ and $P=.018$, respectively). Additionally, TAPSE was independent of all RA measures, left ventricular ejection fraction, and age ($P>.05$).

Conclusions—Based on these results, TAPSE does not appear to be influenced by either RA size or function. Additional studies using strain echocardiography to interrogate RA mechanics might now be very useful to advance our understanding of TA motion in cPH patients given recent data showing that RA function accounts for a significantly greater proportion of total right heart function in patients with cPH than in normal subjects.

Keywords

chronic pulmonary hypertension; echocardiography; right atrium; tricuspid annular plane systolic excursion

1. INTRODUCTION

Our laboratory has previously demonstrated that mechanical systolic function of the atria and the ventricle is more closely coupled on the right side of the heart than on the left.¹ However, whether this coupling is either the result of anatomic linking or chamber geometry was never clarified.

Chronic pulmonary hypertension (cPH) is known to trigger a series of right ventricular (RV) anatomical and functional remodeling changes as a result of worsening pulmonary vascular resistance.² Specifically, increasing right ventricle (RV) afterload induces its progressive dilatation and hypertrophy resulting in gradual decline of RV systolic function, the main determinant of morbidity and mortality.^{3–6} These remodeling changes also cause tricuspid annular dilation that not only worsens tricuspid regurgitation, but also contributes to the development of right atrial (RA) enlargement.^{7–9}

As most efforts in cPH have been centered in studying RV hemodynamic alterations, RA changes with cPH have been limited to increase in RA pressures (RAP) as well as changes in RA size.^{10,11} Furthermore, visualization of the RA is limited to the four-chamber apical view during echocardiographic examination.¹² To date, this transthoracic window has been invaluable to examine longitudinal displacement of the tricuspid annulus (TA) that descends during ventricular systole while it ascends during both ventricular diastole and after atrial contraction.¹ Echocardiographic identification of this longitudinal shortening during ejection has been vital to detect maximal TA descent known to correlate with RV systolic function.^{1,10,13–15}

As the apical four-chamber view not only allows most favorable angulation for longitudinal interrogation of annular motion as both displacement and velocity vectors are directed toward the echocardiographic transducer, but also allows the best echocardiographic examination of the RA and RV areas. Hence, this transthoracic window should be most useful in reflecting the full dynamic atrioventricular interaction, particularly when on the right side longitudinal fibers predominate in the RV and more muscular and prominent pectinate muscles in the RA.¹ Consequently, as the TA is both anatomically and functionally related to this right atrioventricular dependence, the main objective of this study was to determine whether changes in RA size and function alter the motion of the TA in cPH in view of recent data showing that RA function accounts for a significantly greater proportion of total right heart function in patients with cPH than in normal subjects.¹⁶

2. | METHODS

2.1. | Study group

In this retrospective study, a database from 110 echocardiograms was queried. Each echocardiography represented an independent patient referred to our echo laboratory for diagnosis, management, or follow-up of any suspected or known heart disease. A consecutive sampling technique was implemented to obtain the study sample that was later divided into two groups. Group I consisted of 50 patients (50 ± 11 years) with normal pulmonary artery systolic pressures (PASP < 40 mm Hg) while Group II included 60 patients (55 ± 14 years; $P < .04$) with cPH (PASP ≥ 40 mm Hg), as documented by echocardiography.

For inclusion in this study, all patients required a complete echocardiogram that included a discernible tricuspid regurgitation signal to estimate PASP, good endocardial border resolution of both right and left ventricular chambers as well as the RA. In addition, M-mode and tissue Doppler imaging (TDI) of the TA was also required from all patients. Finally, all patients had to be in normal sinus rhythm at the time of the study without ectopy or any other arrhythmias. In addition, all patients had normal left ventricular wall motion, systolic function, and no significant left-sided valvular diseases.

The University of Cincinnati Institutional Board Review (IRB #12012604) gave approval for this study and no informed consent was therefore needed, as this was a retrospective analysis.

2.2 | Echocardiographic examination

Two-dimensional echocardiographic (Vivid 7; GE Medical Systems, Milwaukee, WI, USA) images were digitally acquired with a 1.5/3.1 MHz phased array transducer using standard harmonic imaging, in gently held end-expiration, for subsequent offline analysis using the Acuson Syngo system (V 7.0 Diagnostic Workstation, Siemens, Berkeley, CA, USA). Examinations were performed in accordance with the recommendations of the American Society of Echocardiography.^{10,17} Left ventricular ejection fraction (LVEF) was assessed using the biplane method of disks (modified Simpson's rule) and tricuspid annular plane systolic excursion (TAPSE).¹⁷ RV fractional area change (RVFAC), TAPSE, as well as systolic velocity attained by the lateral TA were all used to determine global RV systolic function.^{10,18–20}

Continuous-wave Doppler was utilized to record the tricuspid regurgitation jet from multiple windows, and the highest velocity was then used to estimate pulmonary artery systolic pressure using the modified Bernoulli equation and an estimate of mean RAP using the diameter and collapse index of the inferior vena cava and the hepatic venous flow pattern.²¹

Right atrial measurements were performed in accordance with published guidelines recommended by the American Society of Echocardiography.¹⁰ Specifically, RA maximal long-axis distance was taken from the center of the TA to the center of the superior RA wall, parallel to the interatrial septum. In addition, RA area was traced at the end of ventricular systole (largest volume) from the lateral aspect of the TA to the septal aspect, excluding the area between the leaflets and annulus, following the RA endocardium, excluding the IVC

and superior vena cava and RA appendage.^{10,11} In a similar fashion, RA measurements were also obtained at the end of ventricular diastole (smallest RA dimension) as previously described.¹¹ Finally, RA fractional area change was calculated in the standard method.¹⁰

2.3 | Statistical analysis

The mean±standard deviation obtained from these measurements was compared using the two-tailed Student's *t* test for unpaired data to determine differences between the studied groups. Shapiro–Wilk test was applied to test for normality. Correspondingly, Spearman's correlation test was used to determine the association between all measurements. A stepwise multiple linear regression analysis was then performed to determine which of the echocardiographic measurements used in the study was most predictor of maximal TA systolic excursion. Reliability of RA measurements for this patient population has already been published.¹¹ All statistical analyses were performed using STATA version 14.2 StataCorp, College Station, TX, USA. *P*-values of less than .05 were considered to be statistically significant.

3 | RESULTS

Standard echo Doppler parameters used in this study are depicted in Table 1. As expected, patients in Group I had normal pulmonary artery systolic pressure (30 ± 6 mm Hg) when compared to Group II (76 ± 23 mm Hg; $P<.0001$) patients. Similarly, Group I patients had normal main RV chamber end-systolic (7 ± 2 cm² vs 17 ± 8 cm²; $P<.0001$) and end-diastolic (17 ± 5 cm² vs 26 ± 8 cm²; $P<.0001$) areas when compared to Group II. Consequently, Group I patients had significantly better echocardiographic measures of RVFAC ($63\pm9\%$ vs $38\pm16\%$; $P<.0001$), TAPSE (2.5 ± 0.3 cm vs 2.0 ± 0.5 cm; $P<.0001$), and TA TDI systolic velocity (14 ± 2 cm/s vs 12 ± 3 cm/s; $P<.001$) than Group II patients. Moreover, Group I patients not only had significantly smaller end-systolic as well as end-diastolic RA areas, but also corresponding RA maximal long-axis length dimensions were also significantly smaller than in patients. In a similar fashion, fractional area change of the RA was also higher in Group I patients.

We then performed a Spearman correlation analysis to identify which associations were significant between TAPSE and the studied echocardiographic variables and identified a strong association with RVFAC ($r=.8205$, $P<.0001$), TA TDI systolic velocity ($r=.7326$, $P<.0001$), and PASP ($r=.6803$, $P<.0001$) as seen in Figures 1 and 2. In contrast, no such correlation was observed with regard to age ($r=.0094$, $P=.9226$) or LVEF ($r=-.1566$, $P=.0738$). Interestingly, TAPSE was associated with all RA size measurements (Figures 2 and 3).

To determine which of the measured right chambers echocardiographic variables was most useful in predicting TAPSE, we performed a stepwise multiple regression analysis using the studied measurements that showed significant association with TAPSE (Table 2). RVFAC and TA TDI systolic velocity were the only echocardiographic variables useful in identifying patients with abnormal TAPSE. Age and gender were not included in the analysis as these variables did not show a significant association with TAPSE when Spearman's correlation analysis was performed.

Representative TAPSE M-mode images from a patient with normal pulmonary pressures and RV systolic function as well as from a cHP patient with RV systolic dysfunction are shown in Figure 4.

4 | DISCUSSION

Three separate mechanisms have been used to describe RV contractility including (1) inward movement of the RV free wall, (2) longitudinal basal to apical motion of the TA, and (3) traction effect on the RV free wall as a result of left ventricular contractility,²² being longitudinal RV shortening the mechanism that accounts for the majority of RV contraction.^{23,24} The latter mainly accounts for the predominant abundance of longitudinal RV myocardial fibers along the RV main chamber long axis²⁵ that is also responsible for the strong correlation seen with TAPSE. Therefore, maximal excursion of the TA in systole has been identified as an excellent surrogate measure of RV systolic function in several clinical situations.^{1,10,11,20,26}

In sharp contrast, the potential role that RA size or function might have on TAPSE has not received much attention. In a recent study, Sivak and associates showed that RA function contributes approximately 32% to total TA displacement.¹⁶ This is in agreement with findings previously documented by our laboratory.^{1,19} In our study, although most of RA measures were significantly associated with TAPSE, after adjusting for possible confounders, none of them were found to have any significant influence over maximal TA excursion.

It is well established that cPH causes proximal stiffening of the pulmonary artery,^{27–29} which then triggers a series of RV remodeling changes that in some patients has the potential to result in RV failure,^{30–32} currently considered the leading cause of death in PH.³³ The consequent increase in RV afterload raises RAP, which is not only a well-characterized hemodynamic abnormality in cPH, but also a recognized adverse outcome variable in these patients.^{34,35} An increased RA size has also been associated with adverse outcomes in cPH.³⁶ Neither the contribution of RA function nor size to TAPSE has been given enough attention, particularly when the TA is anatomically flanked by the RA on one side and the RV on the other. Although with regard to the mitral annulus, Kovasc and his group best exemplified the concept of atrioventricular geometry using an idealized cylinder to emphasize the longitudinal motion of the annulus in both systole and diastole.³⁷ Although the same myocardial fiber arrangement between right and left heart is not applicable, the mechanistic construct of an oscillating annulus between atria and a ventricle does hold true for both annuli. Hence, it makes perfect sense to consider the potential effect of RA size and function on TA motion given that abnormalities of both RA size and function do occur as cPH worsens.

We would like to acknowledge some study limitations. First, the retrospective nature of the study and the small sample size analyzed represent an important weakness of this investigation. Second, patients with cardiac rhythms besides normal sinus rhythm were not included, and therefore, our results might not be applicable to other patients. However, on a recent study by Torii et al.,³⁸ patients with atrial fibrillation were shown to have lower

TAPSE values than patients in sinus rhythm regardless of age. Simply stated, in the presence of atrial fibrillation the distance that TA has to traverse from base to apex at the end of ventricular diastole when compared to normal sinus rhythm is significantly less^{1,17,19} and consequently the overall TAPSE magnitude will be smaller (Figure 5). These differences are likely due to loss of the atrial ascent component of TA motion in the absence atrial contractility. Third, our patient population had no invasive hemodynamic data performed at the time of the echocardiogram; hence, some potentially valuable information that could have improved our correlations was not included. However, our purpose was to assess impact between echocardiographic variables. Finally, we lack speckle tracking imaging data to determine whether peak longitudinal strain or strain rate is important. Sakata and associates recently published interesting data using speckle tracking echocardiography and these investigators demonstrated that peak RA longitudinal global strain as well as peak strain rates were lower in cPH patients when compared to normal controls.³⁹ Unfortunately, these investigators made no comparisons between RA and RV measures to determine which of these measurements was most important in determining TAPSE.

Based on these results, our data seem to suggest that overall TA motion is mostly dependent on RV rather than RA anatomical and functional standard echocardiographic variables. From a mechanistic point of view, even when both RA and RV appear to be anatomically and functionally related to an intervening atrioventricular TA in between both chambers, the longitudinal motion of this right-sided annulus appears to be mainly driven by the predominant RV longitudinal myocardial fibers composing the main architecture of the RV chamber, rather than by the thick and prominent RA pectinate muscles.^{1,18} Whether RA contribution is important in maintaining RV systolic and diastolic function requires further analysis. Similarly, the full spectrum of TA motion in relation to right atrioventricular dependence is not completely elucidated, particularly as the RV remodels and the TA dilates causing significant tricuspid regurgitation also known to affect TAPSE interpretation.⁴⁰

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FIGURE 1.

Line graphs showing correlation analysis between TAPSE and (A) pulmonary artery systolic pressure (PASP) and (B) TA TDI systolic velocity. TA=tricuspid annulus; TDI=tissue Doppler imaging; TAPSE=tricuspid annular plane systolic excursion

FIGURE 2.

Line graphs showing correlation analysis between TAPSE and (A) right chambers fractional area changes and (B) RA maximal long-axis distance at the end of systole/diastole.

RAFAC=right atrial fractional area change; RVFAC=right ventricular fractional area change;

RAESL=right atrial end-systolic length; RAEDL=right atrial end-diastolic length;

TAPSE=tricuspid annular plane systolic excursion

FIGURE 3.

Line graphs showing correlation analysis between TAPSE and right-sided chambers. (A).

Individual representation of RA areas and (B) RV areas at the end of systole/diastole.

RAESA=right atrial end-systolic area; RAEDA=right atrial end-diastolic area;

RVESA=right ventricular end-systolic area; RVEDA=right ventricular end-diastolic area;

TAPSE=tricuspid annular plane systolic excursion

FIGURE 4.

Representative M-mode tricuspid annular plane systolic excursion images in (A) patient with normal pulmonary artery systolic pressure and right ventricular (RV) ejection fraction and in (B) patient with chronic pulmonary hypertension and RV dysfunction

FIGURE 5.

Representative apical four-chamber view illustration showing the effect of atrial fibrillation on TAPSE. Please note that when compared to normal atrial contraction, TAPSE is smaller in magnitude. Dashed lines represent tricuspid valve position in systole. RA=right atrium; RV=right ventricle, LA=left atrium; LV=left ventricle; TAPSE=tricuspid annular plane systolic excursion

TABLE 1

Demographic and echocardiographic data of the study population

Variables	Group I Normal PASP ($\pm$ SD)	Group II Elevated PASP ($\pm$ SD)	P-value
Number of patients (gender)	50 patients (28 males)	60 patients (17 males)	
PASP (mm Hg)	30 $\pm$ 6	76 $\pm$ 23	<.0001
Age (y)	50 $\pm$ 11	55 $\pm$ 14	.04
MAPSE (cm)	1.4 $\pm$ 0.2	1.4 $\pm$ 0.3	.05
LV end-systolic volume (cm ³)	29 $\pm$ 12	17 $\pm$ 14	<.0001
LV end-diastolic volume (cm ³)	98 $\pm$ 26	65 $\pm$ 37	<.0001
LVEF (%)	71 $\pm$ 8	76 $\pm$ 10	.004
TAPSE (cm)	2.5 $\pm$ 0.3	2.0 $\pm$ 0.5	<.0001
RV end-systolic area (cm ²)	7 $\pm$ 2	17 $\pm$ 8	<.0001
RV end-diastolic area (cm ²)	17 $\pm$ 5	26 $\pm$ 8	<.0001
RVFAC (%)	63 $\pm$ 9	38 $\pm$ 16	<.0001
TA TDI s' (cm/s)	14 $\pm$ 2	12 $\pm$ 3	<.0001
RAFAC (%)	54 $\pm$ 13	41 $\pm$ 16	<.0001
RA end-systolic area (cm ²)	13.0 $\pm$ 3.7	18.3 $\pm$ 6.8	<.0001
RA end-systolic length (cm)	4.2 $\pm$ 0.7	4.8 $\pm$ 0.8	<.0001
RA end-diastolic area (cm ²)	6.0 $\pm$ 2.4	11.2 $\pm$ 6.5	<.0001
RA end-diastolic length (cm)	2.6 $\pm$ 0.6	3.6 $\pm$ 1.0	<.0001

SD=standard deviation; RA=right atrium; LV=left ventricle; RV=right ventricle; TA=tricuspid annulus; PASP=pulmonary artery systolic pressure; MAPSE=mitral annular plane systolic excursion; LVEF=LV ejection fraction; TAPSE=tricuspid annular plane systolic excursion; RVFAC=RV fractional area change; TDI=tissue Doppler imaging; RAFAC=RA fractional area change.

TABLE 2

Stepwise multiple regression analysis to determine the best right chamber echocardiographic predictors of TAPSE

Independent variables	Coefficient	SE	<i>t</i>	<i>P</i> -value
PASP	-.0024853	.001737	-1.43	.156
RV end-systolic area	-.0106043	.0220792	-0.48	.632
RV end-diastolic area	.010943	.0134981	0.81	.419
RVFAC	.0135496	.0056345	2.40	.018
TA TDI s'	.0610842	.0125523	4.87	<.0001
RA end-systolic area	.0163171	.0199158	0.82	.415
RA end-diastolic area	-.0236049	.0255551	-0.92	.358
RA end-diastolic length	.0055264	.0785637	0.07	.944
RAFAC	-.0040644	.005115	-0.79	.429

RA=right atrium; RV=right ventricle; TA=tricuspid annulus; PASP=pulmonary artery systolic pressure; RVFAC=RV fractional area change; TDI=tissue Doppler imaging; RAFAC=RA fractional area change.