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# Shape of the right ventricular outflow Doppler envelope and severity of pulmonary hypertension

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**Background** It is now well known that timing of right ventricular outflow tract (RVOT) spectral Doppler signals change with increasing pulmonary hypertension severity. We devised a study to determine whether visual assessment of these RVOT Doppler signals can be useful identifying the severity of pulmonary hypertension.

**Methods** Visual inspection of pulsed RVOT Doppler signals from 120 consecutive patients (mean age of 55 ± 12, range 29–89 years; 45 males); with a mean pulmonary artery systolic pressure (PASP) of 59 ± 29, (ranging from 18 to 150 mmHg), of whom 78 patients had PASP >40 mmHg, was performed and correlated with standard echo Doppler variables of right ventricular performance.

**Results** Visual inspection of RVOT spectral Doppler signals from the 120 patients showed four dynamic patterns and on a stepwise multiple regression analysis, PASP was the only echocardiographic variable that correlated with these different types of RVOT spectral Doppler signals. Furthermore, receiver operator curve analysis of these RVOT spectral Doppler signals correlated well with different ranges of PASP.

**Conclusions** Visual assessment of RVOT spectral signals demonstrates the presence of four dynamic patterns, independent of the aetiology of the pulmonary hypertension, that not only correlate with the severity of pulmonary hypertension, but also are useful in identifying a range of PASP with great accuracy that minimizes subjective interpretation. These simple visual assessments of RVOT Doppler signals can be done routinely when evaluating patients with pulmonary hypertension without the need of additional sophisticated equipment.

**Keywords** Echocardiography • Pulsed wave Doppler • Right ventricular ejection • Right ventricular outflow tract • Pulmonary hypertension

## Introduction

Pulmonary hypertension (PH) is a heterogeneous and pathophysiological condition comprised of a wide spectrum of clinical entities in which progressive pulmonary artery obstruction increases pulmonary vascular resistance which leads to right ventricular failure and a 15% annual mortality rate.[1–3] According to the new guidelines, PH diagnosis is an elevated mean pulmonary arterial pressure >25 mmHg at rest, a pulmonary capillary wedge pressure of <15 mmHg, and a pulmonary vascular resistance >3 Wood units.[4]

Transthoracic echocardiography remains a readily available and very useful non-invasive tool that should always be performed in cases of suspected or known PH.[2] Besides characterization of the morphological aspects of the right ventricle and exclusion of valvular, primary myocardial, or congenital anomalies that can elevate right-sided pressures; invaluable Doppler indexes to assess pulmonary haemodynamics can be obtained.[5] In addition to the traditional use of the modified Bernoulli equation to estimate pulmonary artery systolic pressures (PASP) based on the maximum velocity of the tricuspid regurgitation jet and right atrial pressure;[6,7] Doppler echocardiography can accurately estimate mean pulmonary artery pressures,[8] pulmonary artery end-diastolic pressures,[9,10] pulmonary vascular resistance,[11,12] right ventricular Tei index,[13] tricuspid annular systolic velocity,[14] pulmonary flow acceleration time,[15,16] and overall alterations in

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both time to onset and time to peak of the of the right ventricular outflow tract (RVOT) spectral signal.[17]

Since the initial work by Okamoto and associates using Doppler techniques to analyse blood flow patterns across the pulmonary artery;[18] there has been renewed interest in the use of this technique to evaluate haemodynamics.[15,16,19] However, the description of only three RVOT spectral patterns, as suggested by Kitabatake *et al.*[16] and Arkles *et al.*[19] do not appear to account for the changes in timing of the events of the RVOT signal, as a result of PH, previously described by our group.[17] Thus, we queried our echocardiographic database and visually examined RVOT Doppler signals over a wide range of pulmonary pressures to determine whether these signals not only correlate with the severity of PH, but also if these RVOT patterns can be used to identify a useful range of PASP.

# Methods

## Study group

We enrolled 120 consecutive patients (mean age of 55 ± 12, range 29–89 years; 45 males); with a mean PASP estimated by echocardiography of 59 ± 29, ranging from 18 to 150 mmHg, and a total of 78 patients in this studied population had an estimated mean PASP >40 mmHg from our echocardiographic database.

Inclusion criteria for this study required that all patients had a complete echocardiogram including pulsed Doppler spectral signals across the RVOT, discernible tricuspid regurgitation signal, good endocardial border resolution of both right and left ventricular chambers for tracing of the end-systole and end-diastole, as well as M-mode and tissue Doppler imaging (TDI) of the lateral mitral and tricuspid annuli. In addition, all these patients included for the analysis were in normal sinus rhythm and had normal LV systolic function. In terms of exclusion criteria, no atrial or ventricular ectopy was present at the time of the study, none of the patients had a pacer or defibrillator wire in the RV, or any significant left-sided valvular diseases.

The University of Pittsburgh Institutional Review Board (IRB # PRO10110456) approved the study (1/31/2011) and no informed consent was needed as this was a retrospective analysis.

## 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 6.0 Diagnostic Workstation, Siemens, CA, USA). Examinations were performed in accordance with the recommendations of the American Society of Echocardiography.[20] The left ventricular ejection fraction was assessed using the biplane method of disks (modified Simpson's rule) and maximal excursion of the mitral annulus (MA).[21,22] RV fractional area change and maximal excursion of the tricuspid annulus (TA) were used to determine global RV systolic function.[21,22]

To assess ejection of the right ventricle, the RVOT pulsed Doppler signal was obtained by placing a 1- to 2-mm pulsed wave Doppler sample volume just within the pulmonary valve from the parasternal short-axis view. The sample volume was placed so that the closing but not opening click of the pulmonary valve was visualized.[11,17] Pulsed wave Doppler was used rather than continuous wave Doppler to eliminate cases with increased pulmonary velocities secondary to either pulmonary valve or peripheral pulmonary artery stenosis and assess more accurately the duration of the signal.[11,17]

Velocity time integral (VTI) values were obtained by tracing the RVOT spectral pulsed Doppler signals, as previously described.[23]

Continuous wave Doppler was utilized to record the tricuspid regurgitation jet from multiple windows, and the highest velocity was then used to estimate PASP using the modified Bernoulli equation and an estimate of mean right atrial pressure using the diameter and collapse index of the inferior vena cava and the hepatic venous flow pattern.[7]

Tissue Doppler interrogation of the tricuspid annulus was performed by placing the sample volume (gate length of 0.6 cm) of the ultrasound cursor on the lateral aspect of tricuspid annulus at the junction of the right ventricular-free wall in the apical four-chamber view, parallel to the direction of annular motion.[14] A similar approach was used to interrogate the MA and determine systolic and diastolic annular velocities as previously described.[24] Peak velocities during systole, early (Ea) and late diastole (Aa), along with simultaneous ECG displayed at a sweep rate of 50 mm/s were recorded.

Finally, a visual assessment of all pulsed RVOT signals was then performed to identify signal patterns and patients were then divided into groups according to the RVOT Doppler signal. Waveform analysis was performed without knowledge of each patient's pulmonary artery pressure.

It is important to note that PH patients might have a combination of different RVOT patterns; for the purpose of this study we selected the highest estimated PASP, based on the echocardiographic examination, as well as the worst RVOT pattern found within the Doppler interrogation. Therefore, it is imperative not only to record the highest tricuspid regurgitation velocity, but also to examine RVOT pulsed Doppler signals over several cardiac cycles. In order to be able to attain this goal, capture of 4 to 6 tricuspid regurgitation as well as RVOT Doppler signals was performed during inspiration and expiration and the highest velocity and worst pattern was selected as representative for each patient as shown in *Figure 1*.

## Statistical analysis

Three measurements were obtained for each studied variable and individual mean ± standard deviation values were compared using the two-tailed Student's *t*-test for unpaired data. Univariate analysis was performed when appropriate. Analysis of variance (ANOVA) with repeated measures was used to test statistical significance of the studied variables. When ANOVA showed statistically significant differences among groups, *post hoc* analysis with the Student–Newman–Keuls test was then performed to assess differences among these groups. A stepwise multiple linear regression analysis was performed to determine the independent relationship between the different shapes of the RVOT spectral signal and all echocardiographic and Doppler variables commonly used in the assessment of RV performance. Receiver operating characteristic curves and areas under the curves were examined and compared by the method of Hanley and McNeil[25] to determine cut-off values that were most discriminative. Finally, interobserver and intraobserver variabilities were measured with both observers blinded to the other data of the subjects and without knowledge of each other's readings. Analysis of the differences between the spectral RVOT outflow patterns was calculated using an inter-rater agreement statistic (Kappa) to evaluate the agreement between two readings with 95% confidence interval technique. *P*-values of $<0.05$ were considered to be statistically significant.

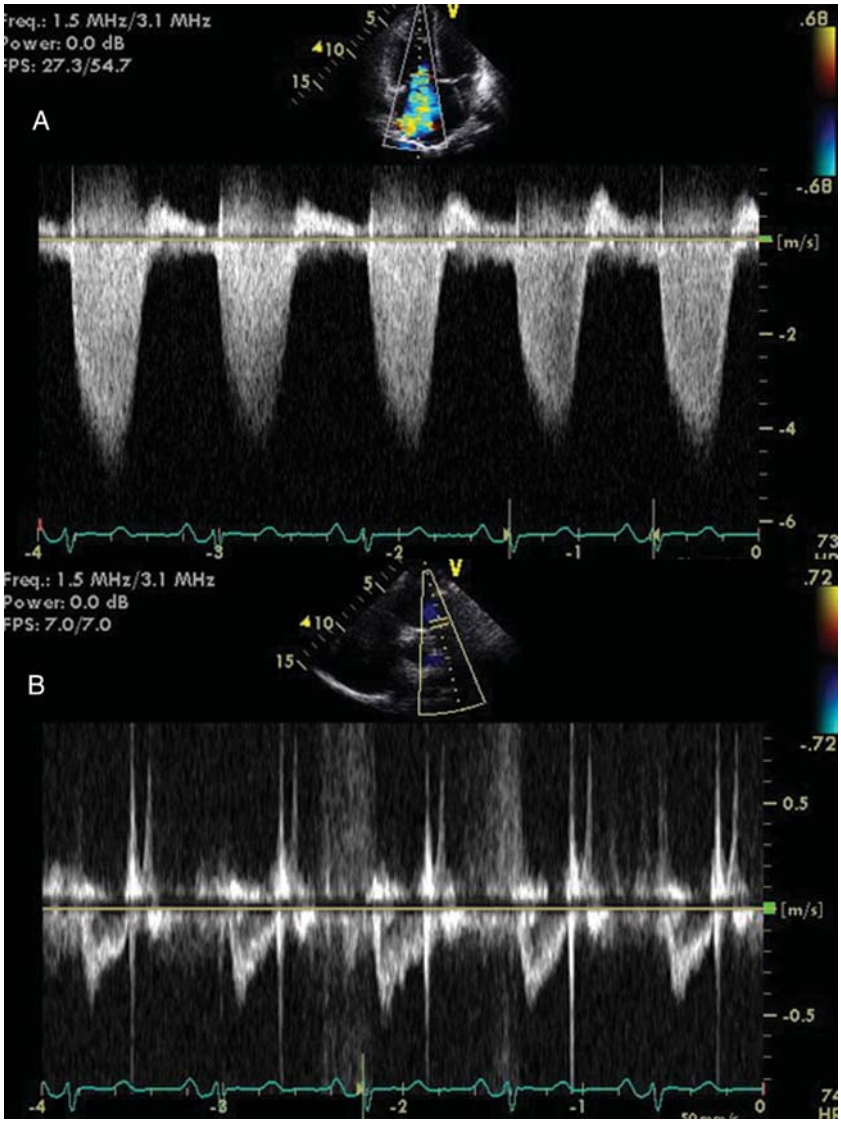


**Figure 1** (*A*) Shows capture of five tricuspid regurgitation and (*B*) right ventricular outflow tract Doppler signals performed during inspiration and expiration as mentioned under the 'Methods'.

## Results

*Table 1* lists both principal clinical diagnosis, as the reason for obtaining the echocardiographic examination, and the WHO Classification[26] for all 120 patients included in the final analysis. It is important to note that our study group is comprised of a heterogeneous group of patients with different clinical entities resulting in PH.

Visual inspection of all 120 RVOT Doppler spectral signals showed four dynamic patterns as shown in *Figure 1*. Pattern I was characterized by a parabolic contour of the ejection flow velocity envelope (*Figure 2A*); pattern II had a triangular contour as a result of an early systolic peak followed by a slow deceleration time (*Figure 2B*); pattern III and IV were similar to pattern II with the exception that pattern III had mid-systolic notching (*Figure 2C*) and pattern IV had significant reduction in signal volume resulting in a spiked appearance as seen in *Figure 2D*.

*Table 2* depicts standard echo-Doppler parameters obtained for all patients according to their RVOT spectral Doppler pattern. Although mitral annular systolic plane excursion was normal in all four groups, the magnitude of total annular excursion for Group I patients was statistically higher than Group IV patients; however, there was no difference in the left ventricular ejection fraction using the modified Simpson's rule despite the fact that both left ventricular end-systolic and diastolic volumes were progressively smaller from Groups I to IV. With regard to the RV, end-systolic as well as end-diastolic areas increased, while fractional area change and tricuspid annular systolic plane excursion decreased significantly from Groups I to IV.

In terms of Doppler data, no difference was found with regard to RVOT pulsed velocity. However, RVOT VTI decreased

**Table 1 Indication for echocardiographic examination on the studied population**

| Diagnoses | Number of patients | WHO classification |
|---|---|---|
| Idiopathic pulmonary arterial hypertension | 18 | Group 1 |
| Human immunodeficiency virus/AIDS | 17 | Group 1 |
| Interstitial lung disease | 16 | Group 3 |
| Preoperative or other non-significant medical history | 12 | Group 5 |
| Chronic obstructive pulmonary disease/Emphysema | 10 | Group 3 |
| Hypertension/left ventricular diastolic dysfunction | 9 | Group 2 |
| Scleroderma | 8 | Group 1 |
| Systemic vasculitis | 7 | Group 1 |
| Mixed cardiac/pulmonary conditions | 6 | Group 3 |
| Valvular disease/coronary artery disease | 4 | Group 2 |
| Obstructive sleep apnoea | 3 | Group 3 |
| Congenital heart disease | 3 | Group 1 |
| Chronic thromboembolic disease | 3 | Group 4 |
| Porto pulmonary hypertension | 2 | Group 1 |
| Sickle cell disease | 2 | Group 5 |

significantly from Group I to II while it was not different between Groups II and III and then it decreased significantly between these two groups and Group IV. Both peak tricuspid regurgitation velocity and pulmonary vascular resistance increased significantly from groups I to IV.

When assessing MA TDI signals we found no difference in either systolic or early diastolic (E') wave velocities among the groups with only Group I having a lower late diastolic (A') MA velocity with a higher MA TDI E'/A' ratio than the other RVOT groups. In the case of TA TDI signals, Group IV had lower systolic and late diastolic (A') velocities than any other group. While there was no difference in terms of TA TDI E'/A' ratios; Group I had the highest early diastolic (E') TA TDI velocity.

On a stepwise multiple regression analysis, PASP was the only echocardiographic variable that correlated with the different types of RVOT spectral Doppler signals as shown in *Table 3*. Furthermore, *Figure 3* depicts the distribution of PASP that correspond to each RVOT spectral Doppler type. Most importantly the occurrence of the RVOT spectral patterns was independent of the aetiologic basis for PH and it appears it was simply dependent on the severity of the PH.

Receiver operator curve analysis of RVOT spectral Doppler signals showed that RVOT pattern I can be found in patients with PASP up to 47 mmHg with a 98% sensitivity and 100% specificity (AUC 0.999, $P = 0.0001$). In the case of RVOT spectral pattern II, this signal can be identified in patients with PASP between 48 and 68 mmHg with a 85% sensitivity and 96% specificity (AUC 0.960, $P = 0.0001$). RVOT spectral pattern III identifies patients with PASP between 69 and 94 mmHg with a 94% sensitivity and 100% specificity (AUC 0.995, $P = 0.0001$) while RVOT pattern IV is seen in those patients with PASP $>95$ mmHg with a 100% sensitivity and 100% specificity (AUC 1.0, $P = 0.0001$).

When we assessed intraobserver variability when grading the different RVOT spectral Doppler patterns, agreement between these spectral RVOT types was generally good (weighted Kappa 0.713 with a standard error (Kw' = 0) of 01.173) while the inter-observer variability agreement when grading the same RVOT outflow spectral patterns was very good for the same RVOT Doppler spectral types [weighted Kappa 0.885 with a standard error (Kw' = 0) of 01.188].

To determine whether our reported ROC analysis would be widely applicable in routine echocardiographic examinations; we prospectively applied these same cut-off analyses on a different group of 20 patients randomly selected from our echocardiography database. In this analysis, we found a good correlation between predicted and observed RVOT patterns as well as their individual PASP (weighted Kappa = 0.737 with a standard error of 0.170 when an inter-rater agreement statistic analysis) for all additional 20 patients used in this prospective analysis, confirming the utility of the above reported cut-off values, sensitivities, and specificities.

## Discussion

The results of this study appear to indicate that progressive elevation in PASP, independent of the aetiology of the PH, alter the configuration of RVOT spectral Doppler signals. Specifically, simple visual assessment revealed the presence of four RVOT Doppler spectral patterns over a wide range of PASP, determined by echocardiography, in our patient population. These RVOT spectral patterns not only correlate with the severity of PH, but also seem useful in identifying a range of PASP with great accuracy.

Non-invasive estimation of pulmonary artery pressures by Doppler echocardiography not only has been crucial during initial screening of patients referred with possible PH, but also in their routine follow-up assessments.[27–29] However, conflicting data still suggest that when right ventricular and pulmonary artery pressures are estimated via echocardiography, they more often are higher than the pressures measured directly by catheterization.[6,30–32] Consequently, additional objective echocardiographic markers of RV dysfunction have been recently described to improve interpretation of right ventricular performance in patients with PH.[14,22,33–38] Unfortunately, depending on which parameters are used, acquisition time might be prolonged, additional time might be needed for off-line calculations, or expensive equipment might be needed to perform analysis of tissue mechanics. However, Doppler remains an invaluable tool in the assessment of these PH patients based on recent data published by our group regarding changes in time to onset as well as time to peak generation of RVOT spectral Doppler signals that occur as a result of PH;[17] additionally, we were able to identify four dynamic RVOT spectral patterns.

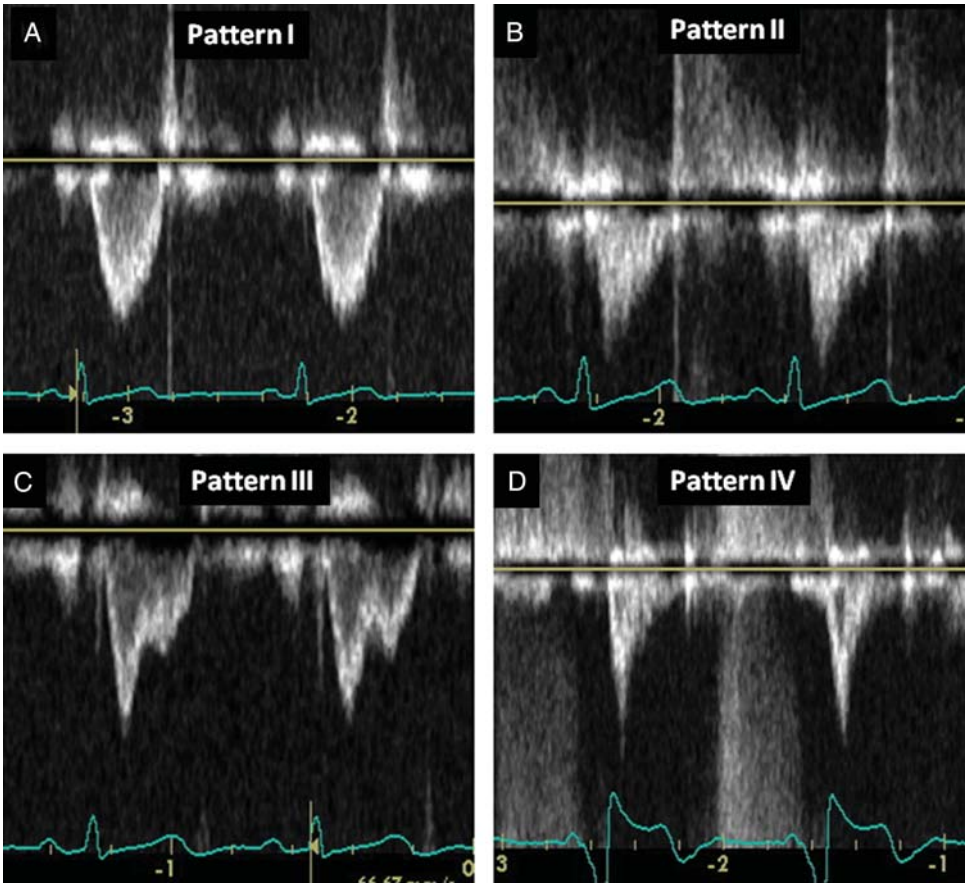


**Figure 2** Visual inspection of right ventricular outflow tract Doppler spectral signals showed four dynamic patterns. (*A*) Pattern I was characterized by a parabola-like contour of the ejection flow velocity envelope. (*B*) Pattern II had a triangular contour as a result of an early systolic peak followed by a slow deceleration time. (*C*) Pattern III has mid-systolic notching and (*D*) pattern IV has significant reduction in signal volume resulting in a spiked appearance.

For decades it has been known that a rounded or parabolic configuration of the RVOT Doppler flow pattern was usually seen in healthy subjects while a triangular flow configuration was consistently seen in patients with PH.[16,18] Several potential mechanisms have been proposed to explain the changes that occur in flow configuration as a result of PH including rapid acceleration of the flow in the pulmonary artery due to right ventricular pressure overload; reduced capacitance and increased impedance of the pulmonary vascular tree; increased stiffness of the pulmonary artery resulting in a rapid pulse wave velocity and dilatation of the main pulmonary artery that can cause reversal of the initial forward ejection flow in the pulmonary trunk. Of course, it is important to understand that none of these factors occur in isolation; but the interaction with each other due to their interdependence is critical to alter flow dynamics as seen in this study.

It would appear that our data contradict the work already published by both Arkles[19] and Kitabatake[16] groups; since these investigators only reported the presence of three different RVOT patterns with increasing pulmonary artery pressures. However, it is important to distinguish two main differences between our study and theirs. First, in the case of Kitabatake *et al*[16] our data set was much larger. Second, the mean pulmonary pressure of the control group was $<20$ mmHg while the in the PH group the mean pulmonary pressure was equal to or $>20$ mmHg. In the case of Arkles group,[19] these investigators divided their population into three groups with corresponding mean pulmonary pressures of $33 \pm 10$, $46 \pm 12$, and $50 \pm 9$ mmHg, respectively. In comparison, the mean pulmonary artery pressures for the four groups we studied, using Chemla's formula[39] were $22 \pm 5$, $38 \pm 4$, $49 \pm 6$, and $70 \pm 11$ mmHg. This wide range of pulmonary pressures included in our study not only appears to provide a smoother transition between normal and mild PH patients; but also includes patients with higher pulmonary pressures that those reported in the other two studies.[16,19] We believe including these different ranges in PASP enabled us to better appreciate the changes in RVOT Doppler waveform patterns. Specifically, our study was able to identify the subtle transition that occurs between normal pulmonary pressures with the parabolic contour of RVOT ejection flow velocity envelope and mild PH, with its triangular contour as a

**Table 2** Standard echocardiographic and Doppler data from the studied population when divided among four groups according to their right ventricular outflow tract spectral Doppler signal

| Variables | RVOT type I | RVOT type II | RVOT type III | RVOT type IV | ANOVA |
|---|---|---|---|---|---|
| MAPSE | 1.4 ± 0.2 | 1.3 ± 0.2 | 1.3 ± 0.2 | 1.2 ± 0.3 | <0.05 |
| LVESV | 26 ± 10 | 18 ± 13 | 19 ± 14 | 11 ± 8 | <0.001 |
| LVEDV | 99 ± 28 | 70 ± 35 | 74 ± 41 | 38 ± 18 | <0.001 |
| LVEF | 73 ± 8 | 75 ± 9 | 75 ± 9 | 73 ± 9 | NS |
| TAPSE | 2.5 ± 0.3 | 2.0 ± 0.5 | 1.9 ± 0.5 | 1.5 ± 0.4 | <0.001 |
| RVESA | 6 ± 2 | 14 ± 6 | 17 ± 7 | 25 ± 7 | <0.001 |
| RVEDA | 18 ± 4 | 23 ± 7 | 27 ± 9 | 33 ± 9 | <0.001 |
| RVFAC | 64 ± 9 | 41 ± 16 | 38 ± 15 | 24 ± 10 | <0.001 |
| RVOT pulsed velocity | 0.7 ± 0.1 | 0.8 ± 0.2 | 0.7 ± 0.2 | 0.7 ± 0.2 | NS |
| RVOT VTI | 18 ± 6 | 15 ± 4 | 15 ± 4 | 10 ± 2 | <0.001 |
| Peak TR velocity | 2.4 ± 0.4 | 3.4 ± 0.3 | 4.0 ± 0.3 | 4.8 ± 0.5 | <0.001 |
| PVR | 0.14 ± 0.04 | 0.26 ± 0.09 | 0.29 ± 0.10 | 0.48 ± 0.11 | <0.001 |
| MA TDI systolic velocity | 0.09 ± 0.02 | 0.08 ± 0.02 | 0.08 ± 0.02 | 0.10 ± 0.02 | NS |
| MA TDI E' velocity | 0.11 ± 0.03 | 0.09 ± 0.03 | 0.10 ± 0.03 | 0.11 ± 0.04 | NS |
| MA TDI A' velocity | 0.10 ± 0.03 | 0.12 ± 0.04 | 0.12 ± 0.03 | 0.13 ± 0.03 | <0.05 |
| MA TDI E'/A' ratio | 1.25 ± 0.52 | 0.88 ± 0.46 | 0.91 ± 0.42 | 0.88 ± 0.36 | <0.05 |
| TA TDI systolic velocity | 0.13 ± 0.02 | 0.12 ± 0.03 | 0.11 ± 0.03 | 0.10 ± 0.03 | <0.05 |
| TA TDI E' velocity | 0.12 ± 0.03 | 0.10 ± 0.04 | 0.09 ± 0.03 | 0.09 ± 0.06 | <0.001 |
| TA TDI A' velocity | 0.15 ± 0.04 | 0.16 ± 0.05 | 0.14 ± 0.05 | 0.12 ± 0.05 | <0.05 |
| TA TDI E'/A' ratio | 0.89 ± 0.28 | 0.65 ± 0.23 | 0.74 ± 0.52 | 1.02 ± 1.06 | NS |

MAPSE, mitral annular plane systolic excursion; LVESV, left ventricular end systolic volume; LVEDV, left ventricular end diastolic volume; LVEF, left ventricular ejection fraction; TAPSE, tricuspid annular plane systolic excursion; RVESA, right ventricular end systolic area; RVEDA, right ventricular end diastolic area; RVFAC, right ventricular fractional area change; RVOT, right ventricular outflow tract; VTI, velocity time integral; TR, tricuspid regurgitation; PVR, pulmonary vascular resistance; MA, mitral annulus; TDI, tissue Doppler imaging' TA, tricuspid annulus.

**Table 3** Results of the multiple linear regression analysis of two-dimensional and spectral Doppler signals on the different right ventricular outflow tract spectral Doppler signals

| Independent variables | Coefficient | Std. Error | *P*-value |
|---|---|---|---|
| Peak tricuspid regurgitation velocity | −0.008088 | 0.2093 | 0.9692 |
| RVOT pulsed velocity | −0.1834 | 0.2668 | 0.4935 |
| RVOT VTI | −1.1823 | 1.1084 | 0.2886 |
| PVR | −0.8081 | 0.8117 | 0.3217 |
| RV end-systolic area | −0.02652 | 0.03096 | 0.3937 |
| RV end-diastolic area | 0.03368 | 0.01913 | 0.0812 |
| RV fractional area change | −0.0048 | 0.008412 | 0.5694 |
| TAPSE | −0.06729 | 0.1602 | 0.6753 |
| TA TDI systolic velocity | 0.02461 | 0.0356 | 0.4908 |
| TA TDI E' velocity | −1.8311 | 0.9608 | 0.0594 |
| TA TDI A' velocity | 1.1905 | 0.9139 | 0.1955 |
| Pulmonary artery systolic pressure | 0.03378 | 0.007097 | <0.0001 |

TAPSE, tricuspid annular plane systolic excursion; RVOT, right ventricular outflow tract; VTI, velocity time integral; PVR, pulmonary vascular resistance; TDI, tissue Doppler imaging TA, tricuspid annulus.

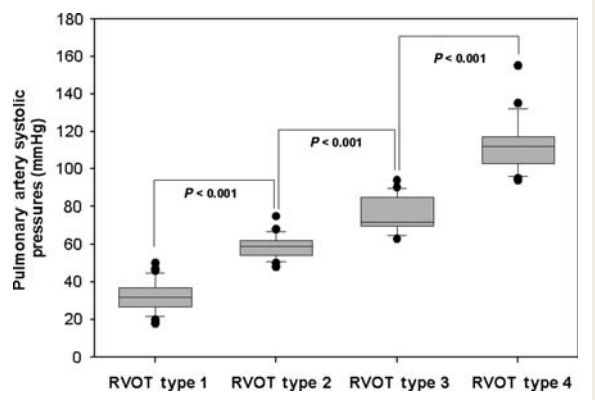


**Figure 3** Box plot representing the distribution of pulmonary artery systolic pressures that correspond to each right ventricular outflow tract spectral Doppler type. Grey boxes represent the main bulk of the patients per group and the line within each grey box represents the mean for that group. The individual black dots either above or below each respective grey boxes are individuals in each group that deviate from the standard deviation. The black lines connecting each grey box are the statistical analysis between groups to determine whether any statistical difference is found.

result of an early systolic peak followed by a slow deceleration time.

Even though we did not include in this study specific RVOT measurements, progression of the triangular RVOT spectral signal shape as seen in this study can be explained by data previously published by our group where increasing degrees of PASP result in shortening of both time to peak RVOT signal and overall total RVOT signal duration.[17] Sequential alterations in these time intervals seem to be in agreement with the transition noted in RVOT spectral signals in PH and specifically with the four RVOT patterns noted by our results.

We speculate that these different RVOT spectral Doppler patterns are shaped not only by changes in elasticity as well as impedance of the pulmonary circulation; but also by conformational changes of the right atrial–ventricular system, as a result of increasing degrees of PH.[40–43] Since pulmonary circulation is pulsatile, alteration of the after mentioned components of this closed circuit will certainly alter wave reflection as demonstrated in this study as well as by others;[16,19] Doppler echocardiography can be useful in documenting these changes in PH.

We acknowledge the following study limitations. First, there is only one study reported on each patient and no sequential imaging was included in this analysis to truly validate the utility of these findings over time. However, the intent of this study was to examine RVOT Doppler signals over a wide range of pulmonary pressures to determine whether these signals not only correlate with the severity of PH, but also if these RVOT patterns can be used to identify a useful range of PASP. Second, we certainly lack concomitant right-sided haemodynamic pressures to determine whether the severity of PH based on echo-derived estimates were accurate. However, data from these cPH patients have been previously used to validate both tissue Doppler and speckle tracking imaging modalities.[12,14,17,22,33,34,36–38] Third, it could be argued that our methodology can only be applied to a certain profile of patients with cPH as listed in *Table 1* and that we cannot extrapolate our results to another significant group of patients with PH such as those with either LV systolic dysfunction or significant LV-sided valvular abnormalities. However, our intent was to isolate changes in the spectral patterns of RVOT Doppler signals as a result of pure RV afterload abnormalities rather than confound the analysis of these signals due to left ventricular abnormalities. Therefore, we cannot make any assumptions on how abnormal LV systolic function or the presence of arrhythmia or frequent ectopy could influence the shape of these spectral signals. Finally, a discrepancy in the final interpretation of which RVOT pattern or for that matter the maximal tricuspid velocity signal might occur if a long spectral Doppler capture is not routinely obtained. It is important to realize that respiratory variation could affect these signals, particularly in patients with significant degrees of PH.

In summary, simple visual assessment of RVOT spectral patterns revealed the presence of four dynamic patterns over a wide range of PASP, determined by echocardiography. Based on our results, identification of these RVOT spectral patterns appears to be dependant only on the severity of PH. Therefore, routine assessment of the RVOT spectral Doppler signal, in conjunction with other well described echo parameters, not only appears to be useful in helping to determine whether PH is present, but also changes in the contour of the RVOT spectral signal appear to correlate with the severity of PASP with great accuracy. This particular Doppler marker can certainly aid in the assessment of patients in whom measurement of the maximal tricuspid regurgitation velocity is limited by either a poor transthoracic window or interrogation of eccentric jets. PH is a dynamic phenomenon that is clearly displayed by the change in the contour in the RVOT spectral signal in the same individual during the course of an echocardiographic examination, likely reflecting the continuous change in PASP that occur with changing loading conditions on a remodelled RV under conformational changes of the interventricular septum and opposing pericardial constrictive forces. Additional studies are now required to further examine the role of echocardiography to non-invasively assess cardiac haemodynamics in PH.

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Each author contributed to the evaluation of data and writing of the manuscript. Dr Lopez-Candales is the guarantor of the paper, taking responsibility for the integrity of the work as a whole, from inception to publication.

**Conflict of interest:** none declared.

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