Sarcouncil Journal of Internal Medicine and Public Health

ISSN(Online): 2945-3674

Volume- 02| Issue- 05| 2023

Research Article **Received:** 22-07-2023| **Accepted:** 11-08-2023 | **Published:** 10-09-2023

Shape of the Right Ventricular Outflow Doppler Envelope and Severity of Pulmonary Hypertension

Dr. Mohammed khalid Abdulkareem[1], Dr. Abdulla Ismaeel Alhasan[2] and Dr. Taha Ali Sulaiman[3]

[1]M.B.Ch.B., Diploma of Child Health (DCH), Diploma of Echocardiography, Iraqi Ministry of Health, Al Alnbar Health Directorate, Al-Ramadi Teaching Hospital, Anbar, Iraq

[2]M.B.Ch.B., MASTER ECHO, Iraqi Ministry of Health, Ninawa Health Director, Al Salam Teaching Hospital, Ninawa, Iraq

[3]Internal Medicine, M.B.Ch.B., Diploma of Internal Medicine, Master Echocardiography, Iraqi Ministry of Health, Al Alnbar Health Directorate, Al-Ramadi Educational Hospital, Anbar, Iraq

Abstract: **Background:** Pulmonary arterial hypertension is defined as a group of diseases characterized by a progressive increase in pulmonary vascular load, leading to a marked increase in pulmonary artery pressure (PAP). A cross-sectional descriptive study in Baghdad Teaching Hospital and Iraqi Center of Heart Disease with Duration Beginning of September 2014 to the end of August 2015. **Aim:** We devised a study to determine whether visual assessment of the right ventricular outflow Doppler signals can be useful in identifying the severity of pulmonary hypertension. **Patients and methods:** We used a pulsed Doppler technique to examine the flow velocity pattern in the RVOT in 127 patients. **Inclusion criteria:** 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 of the lateral tricuspid annulus. In addition, all these patients included for the analysis were in normal sinus rhythm. **Exclusion criteria:** No atrial or ventricular ectopy or other cardiac arrhythmia was present at the time of the study, and none of the patients had a pacer or defibrillator wire in the right ventricle (RV). No previous cardiac surgery. **Result:** Visual inspection of RVOT spectral Doppler signals from the 127 showed four dynamic patterns; pulmonary artery systolic pressure (PASP) was significantly correlated with these different types of RVOT spectral Doppler signals. Both TR pressure gradient (PG) and PASP increased significantly from groups I to IV with p-value < 0.05; mean pulmonary PASP was 32.92 in pattern I, 56.07 in pattern II, 73.65 in pattern III and 113.45 in pattern IV and mean TR PG was 27.57 in pattern I, 47.28 in pattern II, 64.32 in pattern. III and 102.27 in pattern IV. Furthermore, Right ventricular outflow velocity time integral (VTI), acceleration time (ACT), and pulmonary vascular resistance (PVR) showed a significant correlation with the four right ventricular outflow Doppler patterns with p-value < 0.05. **Conclusion:** In this study, we show that easily appreciated differences in the shape or morphology of the right ventricular outflow tract Doppler signal provide rapid, powerful hemodynamic insight into the presence and severity of pulmonary hypertension.

Keywords: Pulmonary arterial hypertension, RVOT, spectral Doppler, pressure gradient, PASP, morphology.

INTRODUCTION

Pulmonary artery pressure (PAP) is an important marker in cardiovascular disorders, being closely associated with morbidity and mortality. Noninvasive assessment by Doppler echocardiography is recommended by current guidelines [López, A. *et al*., 2012]

Pulmonary hypertension is defined as a mean pulmonary artery pressure of more than 25 mm Hg, which typically coincides with a pulmonary artery systolic pressure of more than 40 mm Hg. [McLaughlin, V. V. *et al*., 2009; McQuillan, B. M. *et al*., 2001]

PH is considered mild if the echo estimated PASP is 35 to 45 mm Hg, moderate if it is 46 to 60 mm Hg, and severe when > 60 mm Hg .4 Recent re-evaluation of available data has shown that the normal mean PAP at rest is 14±3 mmHg, with an upper limit of normal of 20 mmHg. [Kovacs, G. *et al*., 2009; Badesch, B. D. *et al*., 2009]

The definition of PH on exercise as a mean PAP 30 mmHg as assessed by right heart catheterization (RHC) is not supported by published data, and healthy individuals can reach much higher values. [Naeije, R. *et al*., 1993]

The significance of a mean PAP between 21 and 24 mmHg is unclear. Patients presenting with PAP in this range need further evaluation in epidemiological studies. [Galie, N. *et al*., 2009]

Furthermore, even slightly elevated PAP may have adverse prognostic implications in the general population. Therefore, the measurement of PAP has gained wide acceptance in the assessment and follow-up of patients with cardiac or pulmonary disorders. [Greiner, S. *et al*., 2014] Given the nonspecific symptoms and subtle physical signs, particularly in the early stages, a high clinical index of suspicion is necessary to detect the disease before irreversible pathophysiologic changes occur. In this regard, transthoracic echocardiography (TTE), by providing direct and/or indirect signs of elevated pulmonary pressure, is an excellent noninvasive screening test for patients with symptoms or risk factors for PH, such as connective tissue disease, anorexigen use, pulmonary embolism, heart failure, and heart

***Corresponding Author:** Dr. Mohammed khalid Abdulkareem

DOI- https://doi.org/10.5281/zenodo.8332605

murmurs. It may also provide key information on both the etiology and prognosis of PH. [Bossone, E. *et al*., 2007]

Transthoracic echocardiography remains a readily available and very useful non-invasive tool that should always be performed in cases of suspected or known PH. [Galie, N. *et al*., 2009]

PATIENTS AND METHODS

Cross-sectional descriptive study in Baghdad Teaching Hospital and Iraqi Center of Heart Diseases with a duration from the beginning of September 2014 to the end of August 2015 where. Inclusion criteria: Inclusion criteria for this study required that all patients had a PH with 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 of the lateral tricuspid annulus. In addition, all these patients included for the analysis were in normal sinus rhythm and about Exclusion criteria: no atrial or ventricular ectopy or other cardiac arrhythmia was present at the time of the study; none of the patients had a pacer or defibrillator wire in the RV.

Methods

Questioner

Including name, age, gender, address, history of medical and surgical diseases, and history of previous surgery (design by research).

Echocardiographic Examination

Two-dimensional echocardiographic (Philips CX 50) images were digitally acquired with a 1.5/3.1 MHz phased array transducer.

Parameters were Measured:

1. TR velocity and PG (mmHg)
2. PASP (mmHg)
3. Mean PAP (mmHg)
4. RVOT VTI (cm)
5. Peak velocity (cm/s)
6. AC (ms)
7. ET (ms)
8. PVR (wood)

Examinations were performed in accordance with the recommendations of the American Society of Echocardiography.

The left ventricular ejection fraction was assessed using Simpson's method. Maximal excursion of the tricuspid annulus was used to determine global RV systolic function.49

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.

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

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.

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.1 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, the capture of 4 to 6 tricuspid regurgitations as well as RVOT Doppler signals was performed during inspiration and expiration, and the highest velocity and worst pattern were selected as the representative for each patient.1

- A cut-off value of 40 mmHg and more is considered for the diagnosis of systolic pulmonary hypertension.
- Camla equation was used to calculate mean

Publisher: SARC Publisher

pulmonary artery pressure (MPAP = 0.61 SPAP + 2 mm Hg.

- Patients were categorized according to clinical diagnosis using WHO classification.43
  Group I. Pulmonary arterial hypertension: PH due to severe pulmonary vasculopathy in the absence of an identifiable underlying cause.
  Group II. Pulmonary venous hypertension (PVH): PH due to left-sided heart disease.
  Group III. PH due to chronic lung disease with hypoxia.
  Group IV. Chronic thromboembolic pulmonary hypertension.
  Group V. Miscellaneous and other causes Other definitions.
- Pulmonary vascular resistance had been calculated using the following equation: pulmonary vascular resistance = tricuspid valve regurgitation divided by right ventricular outlet tract time velocity integral multiplied by 10 plus 0.16 (TRV/ RVOT TVI×10+0.16). 29

STATISTICAL ANALYSIS

All data were coded and entered to a computer by using Statistical Package for Social Sciences (SPSS). Summarizing of data done by using no., %, and mean +/-SD. Differences between variables were measured by using analysis of variance (ANOVA) and t-test chi-square. P<0.05 is considered as a level of significance.

Table 1: Demographic data of the studied group

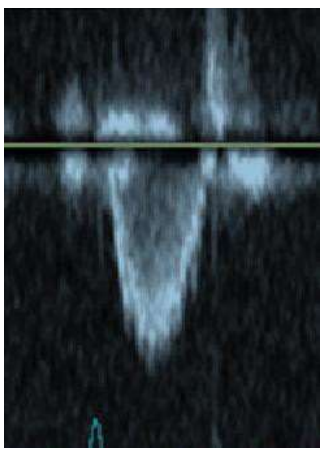
Variables	No	Minimum	Maximum	Mean	Std. Deviation
Age years	127	2	95	47.87	16.78
TR PG mmHg	127	33	130	53.38	17.94
PASP mmHg	127	40	140	55.09	26.72
mPAP mmHg	127	25.37	87.40	40.99	16.05
VTI cm	127	4.3	38.0	16.25	5.55
Peak RVOT velocity cm /s	127	38.00	207.00	85.81	25.59
ACT ms	127	33.00	169.00	90.03	32.40
ET ms	127	122.00	459.00	271.35	52.33
ACT/ ET	127	0.13	0.62	0.33	0.10
PVR Wood	127	0.99	10.56	2.60	1.49

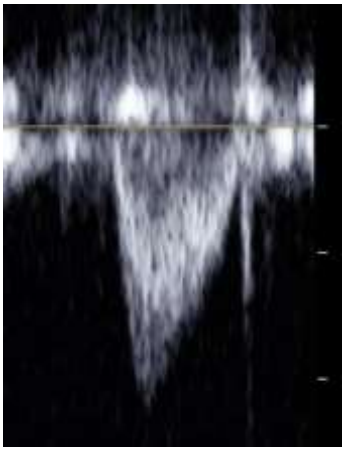
Table 2: Clinical diagnosis and the WHO Classification for patients included in the final analysis

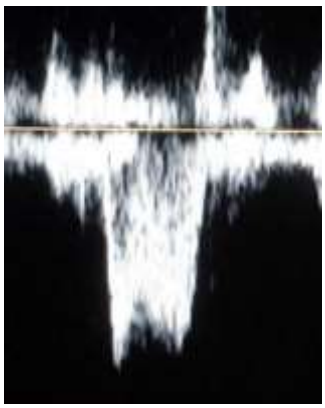
Probable diagnosis	NO. (%)	WHO classification
Increased LV filling pressure	30 (24)	Group 2
Valvular heart disease	23 (18)	Group 2
non-significant medical history	12 (9)	Group 5
Lung diseases	11 (9)	Group 3
Congenital heart diseases	7 (6)	Group 1
Unclassified	35 (28)	Group 5
Idiopathic pulmonary arterial hypertension	3 (2)	Group 1
Eisenmenger's syndrome	3 (2)	Group 1
Pulmonary embolism	3 (2)	Group 1

We found that a visual inspection of all 127 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 1A); pattern II had a triangular contour as a result of an early systolic peak followed by a slow deceleration time (Figure 1B); pattern III and IV were similar to pattern II with the exception that pattern III had mid-systolic notching (Figure 1C) and pattern IV had significant reduction in signal volume resulting in a spiked appearance as seen in Figure 1D

Publisher: SARC Publisher


1 A


1 B



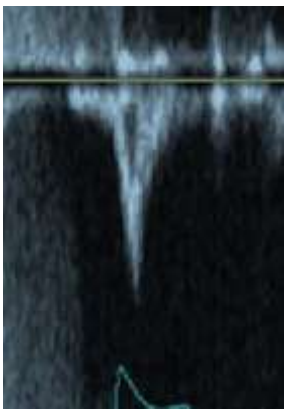


Table 3: Comparison between different RVOT pulsed envelope patterns according to echocardiographic data

Variables	Mean ±SD				P-value
	Pattern I	Pattern II	Pattern III	Pattern VI	
TR PG mmHg	27.57±10.63	47.28±6.75	64.32±11.53	102.27±18.11	0.0001
PASP mmHg	41.92±11.11	56.07±6.23	73.65±11.79	113.45±16.64	0.0001
Mean PAP mmHg	29±2	36.20±3.80	46.92±7.19	68.65±13.90	0.0001
RVOT VTI cm	18.16±3.84	15.65±6.30	15.30±6.23	11.59±5.02	0.0001
Peak velocity cm/s	85.49±19.41	89.35±34.47	84.61±24.81	85.55±23.36	NS
ACT ms	120.75±25.31	74.41±9.80	68.35±11.55	50.18±11.56	0.0001
ET ms	281.89±42.31	262.52±53.40	285.82±50.51	199.18±39.24	0.003
PVR wood	1.63±.42	2.70±.98	3.12±1.03	5.44±2.51	0.0001
AT/ET	0.42±.06	0.29±.08	0.24±.05	0.26±.08	0.0001

Table 4: Distribution of the studied group according to increased LV filling pressure and Left sided Valvular heart diseases

variables	Total No (%)	Pattern 1 No (%)	Pattern 2 No (%)	Pattern 3 No (%)	Pattern 4 No (%)
No (%)	127	53 (42)	29 (23)	34 (26)	11 (9)
Increased LV filling pressure No (%)	38 (30)	4 (14)	17 (58)	15 (44)	2 (5)
Left-sided Valvular heart diseases No (%)	23 (18)	5 (22)	5 (22)	9 (39)	4 (17)

Publisher: SARC Publisher

DISCUSSION

Echocardiography remains one of the most useful and widely available non-invasive imaging modalities that have been used in the screening and follow-up of patients with PH. Even though the idea of measuring indices of RVOT and relating them to PH is not new, the concept of measuring time to onset, time to peak, total duration, and shape of the RVOT systolic spectral signal in relation to PH are not well addressed.

In our study, we found that the mean TR PG was 53.38, which is more than the upper limit of normal physiological TR PG (<25mmHg). Also, the mean PASP was 55.09 (normal cut-off value < 40mmHg that is because all patients were complaining of pulmonary hypertension. Mean ACT was 90 ms, that was lower than the normal limit; Granstam1 S found that an ACT of less than 100 ms indicates a high probability of pulmonary hypertension**.** Mean PVR 2.60 wood showed moderate elevation, (WU) normal PVR

<1.5 WU, mildly elevated 1.5–2.49 WU, moderately elevated 2.5–3.49 WU, and severely elevated >3.5 WU.

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 [Kitabatake, A. *et al*., 1983; Forfia, P. *et al*., 2012].

It would appear that our data contradict the work already published by both Arkles [Arkles, J. S. *et al*., 2011] 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*., our data set was much larger. Second, much higher levels of pulmonary pressure were observed in in our study. In the case of the Arkles group [Arkles, J. S. *et al*., 2011], these investigators divided their population into three groups with corresponding mean pulmonary pressures of 33+10, 46+12, and 50+ nine mmHg, respectively; in comparison, the mean pulmonary artery pressures for the four groups we studied, using Chemla's formula [Chemla, D. *et al*., 2004] were 29±2, 36.20±3.80, 46.92±7.19, and 68.65±13.90 mmHg respectively. Our study agrees with Lo´ pez A and Edelman K1 22+5, 38+4, 49+6, and 70+11 mmHg, in which four patterns were noticed with similar data seen in each pattern.

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 a reversal of the initial forward ejection flow in the pulmonary trunk.1 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.

The factors responsible for the triangular type were principally the reduced capacitance and increased impedance of the pulmonary vascular tree. Those responsible for the reverse flow were the curved path of the blood flow and the dilatation of the pulmonary artery46, or the actual cause of Doppler notching is the early arrival of reflected arterial waves from the pulmonary vasculature that leads to "real-time" impedance to RV ejection. [Furuno, Y. *et al*., 1991]

The lack of Doppler notching was strongly associated with a relative lack of PVD; when PH was present in these patients, it was the result of an increased LA pressure and a relatively normal PVR (PH-PVD). In contrast, in the presence of increased PA stiffness and high PVR, reflected waves return to the RV during systole, impede RV ejection, and cause "notching" of the Doppler profile. [Forfia, P. *et al*., 2012]

By far, the most common cause of PH relates to passive elevation of the PAP from an increase in LA pressure. [Tribouilloy, C. M. *et al*., 1997] That is agree with our study; about half of all cases and 70% of pattern 3 (notched) and 46% of pattern 4 group were associated with left heart disease, assuming there is no notching or flow reverse physiology related to the increased impedance of the pulmonary vascular tree. A variable degree of reactive pulmonary vascular remodeling may also occur, typically related to the severity and chronicity of the LA hypertension. [Vachiéry, J. *et al*., 2013]

In a retrospective study on 3,107 patients, the population was separated according to the current

***Corresponding Author:** Dr. Mohammed khalid Abdulkareem

DOI- https://doi.org/10.5281/zenodo.8332605

definition of "passive" versus "reactive" PH, the latter being referred to as "out of proportion" PH. Pulmonary hypertension due to LHD accounted for 35% of all cases of PH. More than one-half (55%) presented with passive PH-LHD, and 45% had "reactive" PH,57 this agree with our result, 61 patients (47%) have LHD, 95% of them have PVR ≥ 1.5 Wood, 27 patients (27%) have PVR ≥ 3 Wood.

In a series of 320 HFrEF patients, Butler *et al*. 22 found that PVR was >82%. More recent data suggest that the prevalence of reactive PH is similar in all ejection fraction groups. Indeed, in a recent study, 80% to 90% of patients with HFrEF (Heart Failure with Reduced Ejection Fraction) and HFpEF displayed PVR _1.7 WU, and over half had PVR _3 WU or TPG _15 mm Hg59. This agrees with our study; the mean PVR pf those with HFrEF is 3.67± 1.68, and 100% of patients with HFpEF have PH with a mean PVR of 3.21±1.57.

In the PH cohort, any FVERVOT notching (MSN or LSN) was highly associated with PVR 3 WU (odds ratio, 22.3; 95% confidence interval, 5.2–96.4), whereas the No notch pattern predicted a PVR less than or equal to 3WU. (odds ratio, 30.2; 95% confidence interval, 6.3–144.9).

That is Agree with our study; in the pattern 3 (notched) group, the mean PVR was 3.28±1.1, and in the pattern 2 (No notch) group, the PVR was 2.67±1.

Thus, our study confirms that the flow velocity envelope of the right ventricular outflow tract (FVERVOT) notching is not specific for thromboembolic processes and was strongly associated with pulmonary vascular disease regardless of whether the clinical nidus for the pulmonary vascular disease was an isolated pulmonary vasculopathy (PAH), chronic lung disease, or left heart.

Regarding the RVOT VTI, it was significantly correlated with different RVOT envelope patterns and inversely related with p value <0.05; its value decrease from 18.16±3.84 in pattern I, to become 15.65±6.30 in pattern II, 15.30±6.23 in pattern three and 11.59±5.02 in pattern IV, that agree with Lo´ pez A and Edelman K1 study in which RVOT VTI 18+6, 15+4 in pattern I and II, 15+4, 10+2 in pattern III and IV respectively.

In normal individuals, ACT exceeds 140 milliseconds and progressively shortens with increasing degrees of pulmonary hypertension. The shorter the acceleration time, the higher the pulmonary artery pressure. Most studies have suggested that at an ACT of less than 70 to 90 milliseconds, PASP will exceed 70 mm Hg. [Ginghină, C. *et al*., 2009]

Early work from Kitabatake and associates41 that the time to peak velocity, or ACT, of the RV outflow velocity curve provides an estimate of mPAP. An average ACT of 137±24 ms was seen in subjects with a mean PA pressure of 19 mm Hg or less, whereas ACT of 97±20 ms and 65±14 ms were seen in patients with a mPAP of 20 to 39 mm Hg and 40 mm Hg or more, respectively.

In our study, we found that ACT decreases progressively from pattern I to pattern IV with a significant correlation. Arkles, S. *et al*., found that mean ACT.

Was 113±29 in no notch pattern and 67± 21. Similar results were recorded in our study; ACT was 114.9±22.9 in round type and 72.8±9.9 in notch type.44 However, the Doppler index, either ACT or ACT/RVET, measured from the velocity pattern, is thought to be independent of beam direction since the vector of ejection flows hardly changes during systole.

CONCLUSION

1. Visual assessment of RVOT spectral signals demonstrates the presence of four dynamic patterns, independent of the etiology of pulmonary hypertension, that correlate with the severity of pulmonary hypertension. These simple visual assessments of RVOT Doppler signals can be done routinely when evaluating patients with pulmonary hypertension.
2. Our study demonstrated that TR PG, PASP, and mPAP were significantly correlated with the different types of RVOT spectral Doppler signals.
3. ACT, ET, and ACT/ET showed significant association with the different patterns of RVOT flow envelope.
4. Both PVR and RVOT VTI increased significantly from pattern I to IV.
5. No significant correlation was observed between the RVOT peak velocity and the shape of the RVOT envelope.

REFERENCES

1. López, A. & Edelman, K. "Shape of the right ventricular outflow Doppler envelope and severity of pulmonary hypertension." *European Heart Journal – Cardiovascular Imaging* 13(2012): 309–316.

Publisher: SARC Publisher

2. McLaughlin, V. V, *et al*. "ACCF/AHA 2009 expert consensus document on pulmonary hypertension: a report of the American College of Cardiology Foundation Task Force on Expert Consensus Documents and the American Heart Association developed in collaboration with the American College of Chest Physicians; American Thoracic Society, NC.; and the Pulmonary Hypertension Association." *Am Coll Cardio*l 53.17(2009):1573-1619.
3. McQuillan, B. M, *et al*. "Clinical correlates and reference intervals for pulmonary artery systolic pressure among echocardiographically normal subjects." *Circulation* 104.23(2001):2797-2802.
4. Kovacs, G, *et al*. "Pulmonary arterial pressure during rest and exercise in healthy subjects: A systematic review." *Eur Respir J* (2009).
5. Badesch, B. D, *et al*. "Diagnosis and assessment of pulmonary arterial hypertension*." J Am Coll Cardiol* 54 (2009): 55–56.
6. Naeije, R, *et al*. "Mechanisms of improved arterial oxygenation after peripheral chemoreceptor stimulation during hypoxic exercise." *J Appl Physiol* 74:1666 (1993): 1671.
7. Galie, N, *et al*. "ESC Committee for Practice Guidelines (CPG). Guidelines for the diagnosis and treatment of pulmonary hypertension: the 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 the International Society of Heart and Lung Transplantation (ISHLT)." *Eur Heart J*. 30.20 (2009):2493– 537.
8. Greiner, S, *et al*. "Reliability of noninvasive assessment of systolic pulmonary artery pressure by Doppler echocardiography compared to right heart catheterization: analysis in a large patient population." *Journal of the American Heart Association* 3.4 (2014): e001103.
9. Bossone, E, *et al*. "Echocardiography in pulmonary arterial hypertension: an essential tool." *Chest* 131 (2007):339-41.
10. Galie, N, *et al*. "ESC Committee for Practice Guidelines (CPG). Guidelines for the diagnosis and treatment of pulmonary hypertension: the 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 the International Society of Heart and Lung Transplantation (ISHLT)." *Eur Heart J*. 30.20(2009): 2493– 537.
11. Kitabatake, A, *et al*. "Noninvasive evaluation of pulmonary hypertension by a pulsed Doppler technique." *Circulation* 68 (1983):302–9.
12. Forfia, P. & Susan, E. "Echocardiographic Evaluation of the Patient with Pulmonary Hypertension." In Catherine M. Otto (Ed.), The Practice of Clinical Echocardiography (4th ed.). *Saunders, an imprint of Elsevier Inc, Philadelphia,* (2012): 629-645.
13. Arkles, J. S, *et al*. "Shape of the right ventricular Doppler envelope predicts hemodynamics and right heart function in pulmonary hypertension." *Am J Respir Crit Care Med*. 183.2(2011):268–76.
14. Chemla, D, *et al*. "New Formula for Predicting Mean Pulmonary Artery Pressure Using Systolic Pulmonary Artery Pressure." *CHEST* 126 (2004):1313–1317.
15. Furuno, Y, *et al*. "Reflection as a cause of mid-systolic deceleration of pulmonary flow wave in dogs with acute pulmonary hypertension: comparison of pulmonary artery constriction with pulmonary embolisation." *Cardiovasc Res*. 25.2(1991):118-24.
16. Tribouilloy, C. M, *et al*. "Determinants of the pulmonary artery pressure rise in left ventricular dysfunction." Cardiologia 42:1051-1058, 1997) (Forfia PR. Approach to patients with heart failure and pulmonary hypertension." *Curr Treat Options Cardiovasc Med* 9 (2007):302-309.
17. Vachiéry, J., Adir, Y. & Barberà, J. "Pulmonary Hypertension Due to Left Heart Diseases." *Journal of the American College of Cardiology* 62 (2013): D100–8.
18. Ginghină, C, *et al*. "Doppler Flow Patterns in the Evaluation of Pulmonary Hypertension." *Rom J Intern Med.* 47.2(2009):109-21.

Source of support: Nil; **Conflict of interest:** Nil.

Cite this article as:
Abdulkareem, M.K., Alhasan, A.I. and Sulaiman, T.A. "Shape of the Right Ventricular Outflow Doppler Envelope and Severity of Pulmonary Hypertension." *Sarcouncil Journal of Internal Medicine and Public Health* 2.5 (2023): pp 07-14.

Publisher: SARC Publisher