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ORIGINAL ARTICLE

Anatomy and Physiology of Right Heart Adaptation to Pulmonary Arterial Hypertension Along with Treatment

NAJMA ATTAULLAH[1], MAHVISH JAVED[1], MARIYA AZAM KHATTAK[2], MALIK SHANDAR KHAN[3], SAHAR FARHAT[4], TANVEER TARA[5], SUDHAIR ABBAS BANGASH[6], FARWA DAAR[7], UMAR AMIN MALHI[7], IRFAN ULLAH[7]

[1]*Department of Anatomy, Khyber Girls Medical College, Peshawar,*

[2]*Department of Anatomy, Peshawar Medical College, Warsak Road, Peshawar, Khyber Pakhtoonkhwa, (Affiliated with Riphah International University, Islamabad)*

[3]*Department of Physiology, Khyber Girls Medical College, Peshawar.*

[4]*Department of Physiology, Ayub Medical College, Abbotabad,*

[5]*Department of medical lab technology National Skill University Islamabad, Pakistan*

[6]*Faculty of Life Science, Department of Pharmacy, Sarhad University of Science and Information Technology, Peshawar*

[7]*Department of Life Sciences, School of Science, University of Management and Technology Lahore, Pakistan*

Corresponding authors: Mahvish Javed, Email: visho786@gmail.com & Sahar Farhat, Email: saharfarhat615@gmail.com

ABSTRACT

Background: Right ventricular (RV) performance is directly correlated with staying alive in individuals with the pulmonary arterial hypertension **(**PAH). There is still large degree of heterogeneity in the RV's response to pulmonary hypertension, despite the fact that the load of pulmonary i.e. lung, is a key factor in determining RV systolic performance in PAH.

Objective: The main reason why this study was conducted to find out the normal differential function, interrelationships, and response to pulmonary arterial hypertension apical, right ventricular (RV) input and outflow compartments (PAH).

Methods:40 individuals were examined using echocardiography which was 2-dimensional (2D) and also 3-dimensional (3D) in addition to traditional Doppler methods, including 15 controls, with the number of patients with left-sided heart failure was 25, 13 without secondary PAH which was named group-1 (G1) and 12 with secondary PAH was calledgroup-2 (G2).

Results: The apical ejection percent was lower in controls than in patients for example the ejection of control was p≤0.01 and p≤0.01, for G1 it was: p≤0.05 and p≤0.01 and the ejection fraction forG2 was: p≤0.05 and p≤0.01, as well as in patients. The Inflow was decreased i.e.: p≤0.001 for both, same in the case of the apical: p≤0.01 for both, and again also in outflow tract: p≤0.05 for both patients showed decreased ejection percent. In G2, however, the inflow compartment achieved the minimal volume simultaneously with the outflow and apex, as opposed to controls where it took 20 ms for it to do so. In patients (in G1: the apical ejection percent was p=0.02 and p=0.01 and forG2 it was: p=0.01 for both), the times for both isovolumic contraction and that of relaxation were lengthened. In controls, volumetric outflow rate decline and the peak RV ejection time were associated (r=0.6, p0.05), however G2's peak RV ejection time was connected with the apex.

Conclusion: The inflow of the right side ventricular, apical, and also the outflow tract compartments each have distinctive characteristics and share to the total combined systolic function to varying degrees. The ventricle of the right side becomes one non synchronous compartment in PAH, that can lead to a long-term impact on systemic cardiac dysfunction.

INTRODUCTION

The heart and pulmonary vasculature are both impacted by the progressive condition known as pulmonary arterial hypertension (PAH). Although the pulmonary vasculature is the site of the first PAH shock, right ventricular (RV) function is directly associated to patient survival [1]. The right ventricle adjusts to the greater afterload by thickening its walls and contracting more quickly. However, in the great majority of patients, these compensatory mechanisms fall short, leading to right ventricular dysfunction [2]. Right heart failure (RHF) is a complicated clinical phenomenon that develops in PAH patients as a result of inadequate blood flow and/or increased systemic venous pressure during rest or activity as a result of increased RV preload. The two primary clinical signs of RHF are fluid retention and restriction of physical activity. In individuals with PAH, exercise restriction is the first sign of RHF and a reliable indicator of survival [3].

Due to RV dilatation and septal flattening (or even convexity), which negatively influence RV function, RVF not only lowers RV cardiac output but also lowers left ventricular (LV) diastolic filling. Additionally, a substantially dilated RV will lead to systemic congestion and RV ischemia, weakening essential organ functioning (such as the liver, kidneys, and gastrointestinal tract) and harming patient outcomes [4]. Acute cardiac failure may also be observed in patients with PAH. According to recent research, individuals with acute RHF and PAH who need hospital admission may experience short-term death rates of up to 40% [5].

Recently, medications that aim to treat RV dysfunction in patients with PAH have drawn a lot of attention but haven't had much luck, mainly because the mechanism(s) behind right ventricular failure, notably the change from a compensated RV to a failing RV, are poorly understood. However, ongoing research has increased our knowledge of several cellular and molecular changes that occur throughout the development of RVF, as shown by a few good studies that have recently been published on the issue [6].

To examine the inflow, apical, and outflow compartments of the ventricular of the right side (RV) normal differential function, and also their interrelationships and the reaction to pulmonary arterial hypertension, was the goal of this research (PAH)

METHODS

With good acoustic windows, 40 individuals performed3D and2D echocardiographic examinations, 15 of them were made controls (aged52±6 years 9 males) and they had none right or left cardiac anomalies conventional based echocardiography, electrocardiography, or past history of cardiovascular illness. One the bases of the history of presenting illness and clinical and medical examination there investigations, and tests like coronary vessels angiography, and 2D echocardiographic reports as the evidence for Left Ventrical dysfunction brought on by prior myocardial infarction, the 25 patients that were remained had known ischemic heart disease-related heart failure.G1ischemic patients (N=13; age: 62±15 years, 13 males); G2 ischemia patients with PAH (N=12; age: 72 ±14; 12 males) were split into two groups. Patient left lateral decubitus posture was used to get echocardiograms. As said in the Pakistan Society of Echocardiography's guidelines, the views and measurements were taken.

3D Echocardiography (ECG): 3D recordings were collected from apical window. Live 3D-mode imaging was utilized prior to data gathering to determine the ideal location for the x3 transducer. When the probe was pointed anteriorly and laterally, the RV apex and free wall along with trabecular portion were noticeably visible in image sector. After obtaining the necessary pictures, 3D images of the right ventricle were captured throughout a brief expiratory breath hold (3-4 beats).The sagittal, coronal, and four-chamber

view planes were obtained by adjusting three view planes that were generated from the entire volume and displayed on the screen following the loading of the dataset.

Software automatically made adjustments to the end-diastolic frames and end-systolic. The right ventricle's endocardial boundary was traced in sagittal plane, and in coronal view, along the endocardial boundaries two distinct contours were drawn and also along the apex border, beginning at tricuspid annulus and ending at pulmonary annular region, at end systolic and end diastolic frames, respectively (Figure 1A).

On the basis of these three images plane shapes, the program created a vibrant tripartite Beutel model whose endocardial surface adaptation to the ventricle occurs automatically. The tracing in the four-chamber plane and coronal plane, or from the proximal trabecular apical border to the tricuspid annular ring, were used to build the inflow compartment. To ensure they accurately tracked the motion of the tricuspid ring, while reconstructing the final 4D RV model, the outlines in the four-chamber plane throughout systole and diastole were individually modified frame by frame. Figure 1B illustrates a tripartite RV concept.

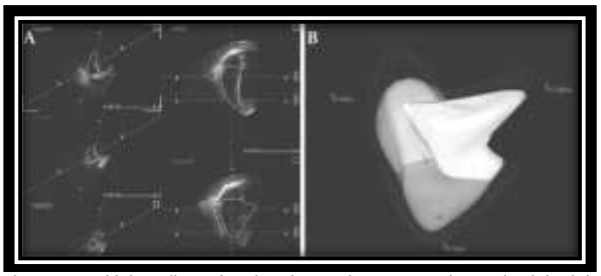

Figure 1: Initial outlines for the three planes are shown in (A); (B) four dimensions of the right ventricle.

The inflow, outflow, and apical area values from the 4D RV model dataset were inserted in a spreadsheet, where tripartite charts demonstrating the quantification of volume fluctuations over various cardiac cycle stages were shown (Figure 2A and B). The programme automatically calculated the ejection fraction (EF) of total RV cavity. Figure 2A was used to compute the EF of outflow, inflow and apex. To do this, an end-diastolic volume was divided by the end-systolic volume of the relevant compartment, and the result was then subtracted. Quantification was done to determine how much volume changed (rose or fell) at various times during the cardiac cycle. Figure 2A illustrates the difference across two subsequent volumes measured by the interval of time which was used to compute the rate of change of volume at various places on the volume curves.

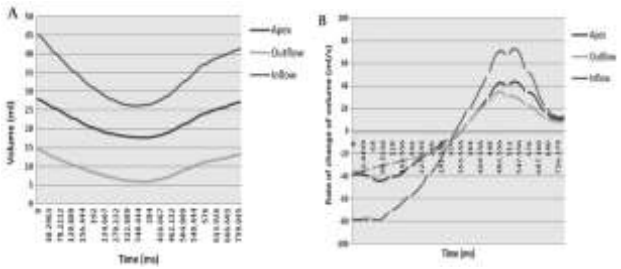

Figure 2: (A) Volume curves of a single patient are examples. (B) One patient's example of a tripartite graph showing the volume change in each of the three right ventricular chambers.

2D Echocardiography: The trans-pulmonary which is pressure difference between the pressure of alveolar and intrapleural, valve flow was measured by pulsed-wave (PW) device Doppler with a 1 cm volumic sample below the pulmonary leaflets during a standard two Dimension echocardiographic assessment of vertical view of thorax and apical views [7]. While at terminal diastole, measuring the Right Ventricle inlet diameter, and long-axis M-mode was used to gauge the TAPSE of the tricuspid annulus. At various points during the cardiac cycle, the tripartite RV function components occur: the interval, known as the isovolumic contraction time (IVCT), comparing the beginning of the ECG's q-wave and the beginning of the RV ejection, at peak and at end RV ejection and Measurements were made between the Right Ventricle final ejection and the start of filling of compartment. The beginning and peak of the pulmonary ejection were defined as the acceleration time (AccT) and the peak and end of the ejection as the deceleration time (DecT). In order to examine the volume changes of regions (inflowing, outflowing, and also that of the apex), time intervalsof these 4 i.e. IVCT, IVRT, AccT, and DecT, was overlaid on graphs volume taken for volume. The volume curve's lowest volume was determined to be the final ejection point (Figure 2A).The estimated peak systolic pressure of PA was determined using the changed Bernoulli equation and backward trans-tricuspid systolic flowing velocities.

Pulmonary artery systolic pressure =$\mathbf{4\times(v)^2+ RAP}$

The pressure of theright atrium, or RAP, is determined by the diameter of the (IV) inferior vena cava and its collapse [8]. If the systolic pressure of thepulmonary artery was more than 36 mm of Hg, PAH was taken into consideration [9].

Statistical analysis: SPSS 13.0, a statistics tool was applied in this research. For the purpose of comparing group differences, a non-parametric Mann-Whitney test was applied. Non-parametric variables within each group were compared using a Wilcoxon test. To investigate the connections between the variables, Pearson's correlation was utilized. A p value of 0.05 or below was deemed significant.

RESULTS

The knowledge demonstrates that there was a difference between the ages of the controls and Groups 1 and 2. IVCT and IVRT took longer than expected (p less than 0.01, for both), comparison with the controls (Table 1). Comparing G2 to controls and G1, the pulmonary (lung) (AccT) was somewhat lesser in group two (p less than 0.001) (table 1). The basic traits of the three groups displayed in Table 2.

RV Regional EF and its relationships to time: The infilling and out filling tract of heart compartments of the EF in controls were identical, but the both Group one and group two had higher total Right side ventricle (emptying) end-diastolic and (filling) end-systolic volumes than controls. apical EF was considerably lower than both (inflow and outflow p≤0.01). The total RV end-diastolic and end-systolic volumes in Groups 1 and also 2 were higher than in controls (p≤0.001 for both), while the overall ejection fraction was lower. Regional EF was also lowered in both patient groups similarly when contrasted to controls at the inflow, apical, and outgoing flow tract p less than 0.00.1 for both respectively. Although the infilling and out filling tract EFs of the already mentioned groups of two patient were similar,, apical EF of G1was smaller in (p less than 0.05 and p less than 0.01) than in group two (p≤0.05 and p≤0.01).In the control group, the filling in compartment attained that of minimal volume that was twenty ms faster than the filling out tract (p less than 0.01) and also faster than the apex (p less than 0.001). It was not different from that outflow tract in G1, but rather simply before the apical heart compartment (p less than0.05). Between the three compartments in G2, there was no duration difference (p=NS) (table 1, Figure 3 A to D).

Table 1: Echocardiography tripartite right ventricular characteristics of both groups

Characteristics	Controls (n=15)	G1 (n=13)	G2 (n=12)	p (1)	p (2)	p (3)
Age, years	52 ± 6	62 ± 15 years	72 ± 14	0.01	≤0.01	0.08

End-diastolic volume, ml	77.5 ± 18.5	110.6 ± 36	123.6 ± 30	≤0.01	≤0.001	0.13
End-systolic volume, m	42.2 ± 11.2	73.9 ± 29.2	82.9 ± 25.4	≤0.001	≤0.001	0.24
Ejection fraction, %	47.8 ± 5.7	32.3 ± 8.2	34.3 ± 9.2	≤0.001	≤0.001	0.74
Ejection fraction (I), %	51.8 ± 7.2	32.9 ± 9.1	35.8 ± 10.28	≤0.001	≤0.001	0.68
Ejection fraction (O), %	51.7 ± 9.8	41.5 ± 16.2	41.3 ± 14.1	0.04	0.06	0.93
Ejection fraction (A), %	39.5 ± 8.7	27 ± 8.6	27.9 ± 9.8	0.001	≤0.01	0.66
Isovolumic Contraction Time (IVCT)	71.8 ± 16.8	91.2 ± 25.9	92.2 ± 27.7	0.16	0.001	0.01
Acceleration Time (AccT)	137.4 ± 15.2	119.1 ± 25.7	92.4 ± 27.7	0.16	0.001	0.01
Deceleration Time (DecT)	163 ± 22.1	155.4 ± 40.2	177.3 ± 34.3	0.01	0.01	0.75
Isovolumic Relaxation Time (IVRT)	49.8 ± 8.2	61 ± 20.6	73.5 ± 13.3	0.01	0.01	0.78
Rate of volume fall (ml/ms)						
Outflow	-19.2 ± 5.3	-18.8 ± 4.1	-27.5 ± 11.1	0.82	0.05	0.01
Inflow	-67.4 ± 21.5	-51.3 ± 22.2	-79.3 ± 31.2	0.03	0.68	0.02
Apex	-28.1 ± 13.8	-31.4 ± 9.8	34.8 ± 10.1	0.32	0.11	0.41

Table 2: The three groups' basic characteristics

Characteristics	Controls (n=15)	G1 (n=13)	G2 (n=12)	p (1)	p (2)	p (3)
Left ventricular end-diastolic diameter, mm	44 ± 8	61 ± 4	61 ±5	≤0.001	≤0.001	0.06
Heart rate, bpm	66 ± 2	67 ± 3	71 ± 2	0.6	0.1	0.1
LVEF, %	57 ± 2	36.1 ±5.5	32.2 ± 4.5	≤0.001	≤0.001	0.12
Mitral regurgitation (patients)		9	9			
Mild		6	3			
Mild moderate		4	9			
Right ventricular inlet diameter, mm	32 ± 2	41 ± 4	42 ± 4	≤0.001	≤0.001	0.051
TAPSE, cm	2.6 ± 0.3	1.6 ± 0.4	1.3 ± 0.2	≤0.001	≤0.001	0.1
RVSP, mm Hg	30 ± 3	31 ± 3.3	51.5 ± 6.1	0.3	≤0.001	≤0.001

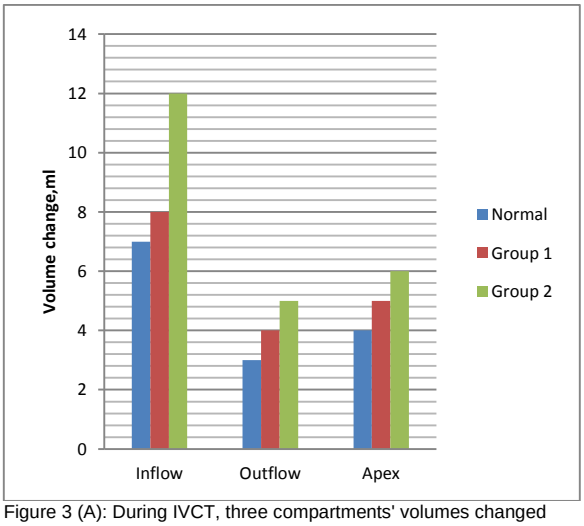

Figure 3 (A): During IVCT, three compartments' volumes changed

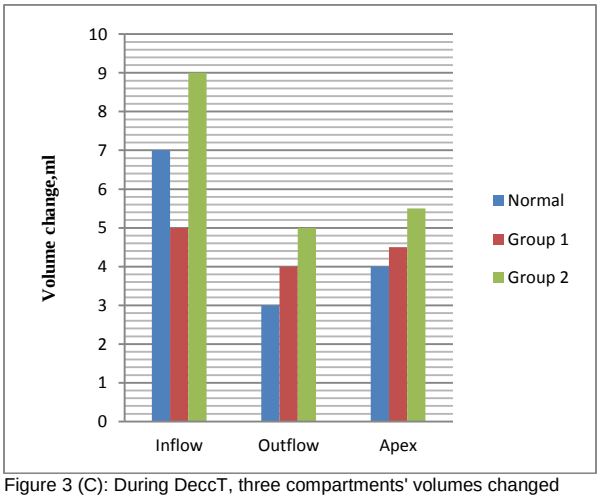

Figure 3 (C): During DeccT, three compartments' volumes changed

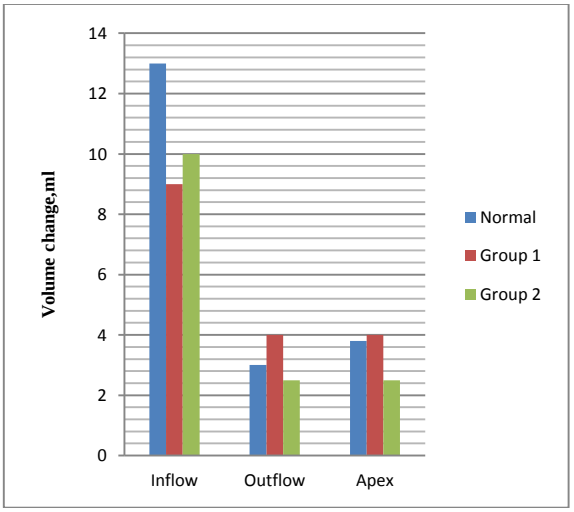

Figure 3 (B): During AccT, three compartments' volumes changed

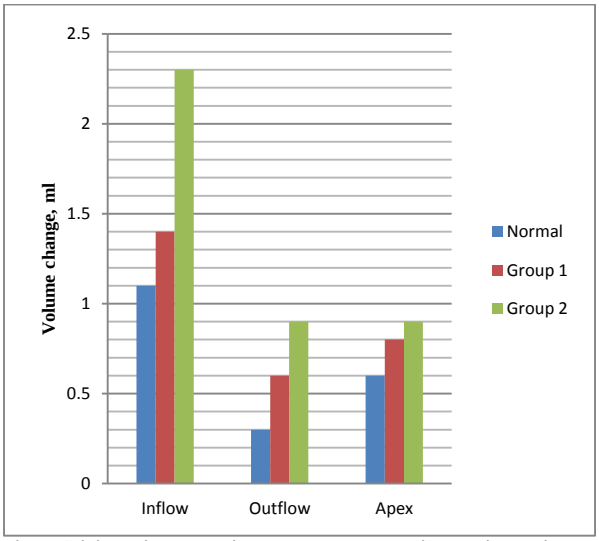

Figure 3 (D): During IVRT, three compartments' volumes changed

The cardiac cycles various stages events

Isovolumic contraction time: In comparison to the outflow tract andapex, the RV inflow volume fell more rapidly in controls i.e. p≤0.001 for the both controls. The relative decrease in the inflow of the volume in group one was similar to the controls, but it was nearly twice in the other 2nd group for that P was less than 0.001 (Figure 4A and 4B).

Acceleration time: The two patient groups' inflow volume falls were substantially less severe than in the control group which was p less than 0.01 and p less than 0.05, respectively, despite being much smaller than the controls. Only G2 had a decrease in apical volume (p≤0.05).

Deceleration time: Comparing G1 to controls, only the decline in inflow volume was reduced (p≤0.05). In comparison to G1, the relevant changes in G2 were larger (p≤0.01). In compared to controls, G2 showed similar results in the outflow filling tract which was P less than 0.01.

Isovolumic relaxation time: In comparison to those controls, the outflow tract's volume fell by four times (p≤0.001) and the inflow volume by twice (p≤0.01) in the apical region. In G1, these relative variations did not alter considerably. G2was exceeded controls by 2.4 and 3 times by the respective values, respectively (p ≤0.001).

The only compartment in the control group where the speed per time of volume decline linked with the peak ejection time was the outflow tract, the value of r was equal to 0.62, and that of p was equal to 0.03. This link vanished in group one and changed to being with the inflow blood filling compartment r is equal to 0.61, p is equal to 0.01. The strongest association in G2 was not with the outflow tract, but rather with the apex (r=0.60, p0.05).

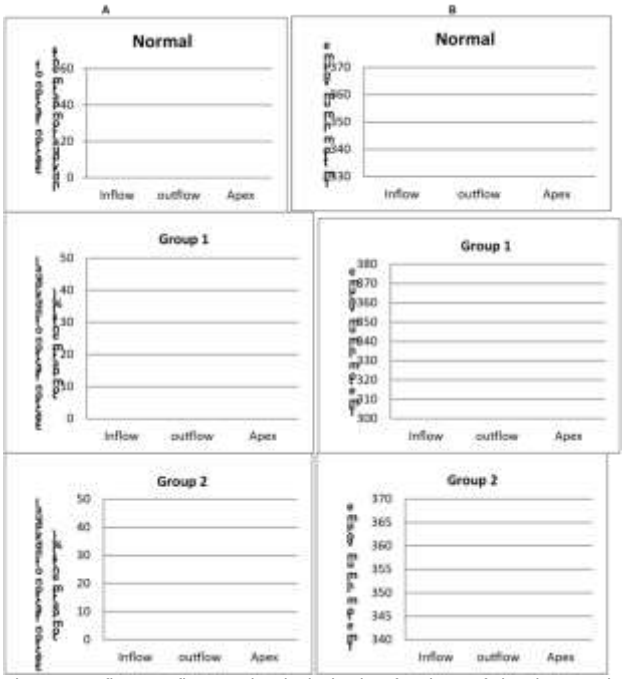

Figure 4: Inflow, outflow, and apical ejection fractions of the three patient groups are compared in (A). (B) a comparison of the lowest volume at 0 ms from the reference line in the three patient groups' 3D volume curves.

DISCUSSION

Four significant findings are presented in this research. First, it demonstrates that the right ventricle's three segments typically each contribute independently to ejection fraction of heart in which apex makes the smallest contribution to the entire ejection fraction [10]. The RV systolic and diastolic volumes were raised in the 2 groups with ischemic cardiac failure, and the EF of each compartment, as well as the entire EF, was lowered, once more in a sequence identical to the physiological ones. The apical compartment showed least contribution to the EF across the groups of the patients, despite the fact that there was little change between the three compartments. Secondly, there was a 20 ms gap between the inflow and outflow tracts, followed by the apex, before the compartments reached their minimal volume. As all three compartments concurrently approached minimal capacity in G2 patients, this pattern was entirely erased [11].

Thirdly, IVCT and IVRT considerably took longer than expected; this was slightly more prominent in G2. When compared to normal people, however, AccT was lower in, particularly in group twopatients. There were also notable changes between and among groups throughout the two isovolumic times [12]. Regarding the apex and out filling tract heart compartments, the decrease in RV volume largely occurred at the infilling level in all those mentioned three groups [13]. Only in the group two did the out filling tract volume decrease increase by three times the amount it did in controls.Fourthly,the factor which is connected with the peak RV ejection time, regulates the pace at which the volume of the outfilling tract falls. Peak heart volume emptying was synchronized with the timing of the infilling tract in group one and that of the apex in group two of the other two RV compartments [14].

Analysis of data: Its total systolic function is not evenly distributed over the three RV compartments, in comparison to the inflow and outflow tract the apex contributing the least. This pattern of activity appears to exist regardless of the presence of illness, suggesting that the RV apex may have another essential function in addition to contraction. Given that it is situated where the axes of the inflow and outflow tracts intersect at a rather broad angle, it is likely that little energy must be generated and there should be less inward motion to allow for the well organised non turbulentand fluent lammenar flow of the blood in direction of the emptying tract [15]. In order to sustain the RV intracavitary circulation, the apex is crucial. Additionally, RV compartments often does not have a synchronous drop in peak volume. The peristaltic RV pump function depends on this differential time delay because of the way it is shaped. For patients with ischemic LV disease, this time delay was still there; however, for those who had concomitant PAH, it was almost completely gone. This study is quite interesting since it shows that right ventricles in patients with PAH lose their separate segmental functions and behave as an unified chamber [16].

Only a major alteration in the right ventricle's shape to a cylindrical one as opposed to a triangular one can accomplish this. In order to show such alterations in PAH, 3D echocardiography is unique. In addition, compared to controls, change in the shape was linked to notable working disruptionslike synchronization problems between the two isovolumic periods [17]. Due to a notable depletion in the degree of volume during speeding up and slowing down as a result of insided myocardial failure, the right ventricle must effectively utilize same volumic phases in order to create enough tension to survive or retain the stroke volume. In patients with PAH, the RVOT function that determines peak ejection has been transferred to the apical area, which lends more credence to this conclusion. As the outflow tract serves as a component of the monocompartmental chamber, this indicates once more the total loss of outflow tract integrity. We discovered that the Right ventrical infilling tract, which can be a step between the normal and PAH phases, had an effect on G1's peak ejection [18].

Therapeutic implications: The usual idea of the RV inflow tract moving along a long axis, also known as TAPSE, using M-mode or tissue Doppler velocities as the only measure of total Right Ventrical blood empting pressure performance is an understatement and an oversimplification despite its strong relationship to Right ventrical three dimentional volumes [19].A straightforward fractional shortening calculation and M-mode recording of RVOT diameter should nevertheless offer significant information even in the lack of 3D equipment, especially in patients with heart and lung flow circulation insufficiency. Atlast, the shift toward the apex of RVOT heart empting pressure time relations in PAH patients raises the possibility of a advantage from that known

electrical resynchronization of the heartchamber in an effort to maximise Right Ventrical ejection volume [20].

CONCLUSION

Asymmetrical in design, the normal RV has three distinct compartments with varying systolic capacities and timing relationships. Individuals with PAH present a clear sign of substantial dyssynchrony throughout the two isovolumic phases, which practically eliminates this distinctive pattern in these patients. Electrically re-timing the RVOT may increase pump efficiency and reduce symptoms in patients whose RV decompensation is progressing despite receiving the best medical care.

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