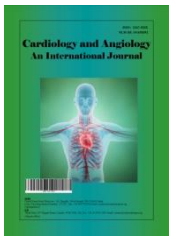

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# Two-dimensional Echocardiography in the Evaluation of Right Ventricular Systolic Function in Patients with Atrial Septal Defect before and after Closure

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***Authors' contributions***

*This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.*

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## ABSTRACT

**Background:** Atrial septal defects lead to left to right shunt, the volume of the shunt is determined by RV/LV compliance, defect size, and LA/RA pressure. RV volume overload and pulmonary over circulation are caused by a simple ASD because the RV is more compliant than the LV. The aim of our study was to assess changes in RV systolic function before and after ASD closure either by surgery or transcatheter closure.
**Methods:** This study was conducted on 70 patients diagnosed with ASD Secundum and had subdivided into two groups A (surgical closure) group, and B (percutaneous device closure) group. All patients had been assessed by transthoracic Echocardiography examination for RV systolic Function 24 h before ASD closure, and 6 months after closure.
**Results:** There was a significant decrease in the right ventricle systolic function indices (TAPSE,

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FAC, Tissue Doppler S wave velocity, and global longitudinal free wall strain) after ASD closure either by surgery or by transcatheter device closure.
**Conclusions:** The right ventricle's size and function are affected by a large shunt caused by an ASD secudium. ASD and its consequent volume overload resulted in higher RV myocardial contraction, leading to an increase in strain values and RV systolic function indices, which were reduced and returned to normal values when the left-to-right shunt was eliminated, and the defect was closed.

*Keywords: Two-dimensional echocardiography; right ventricular systolic function; atrial septal defect; TAPSE; FAC.*

## 1. INTRODUCTION

About 6–10% of all congenital heart defects are ASDs (atrial septal defects). 30–40% of clinically significant adult intracardiac shunts are caused by this condition [1,2]. It is a connection between atrial chambers that allows blood to be shunted between the systemic and pulmonary circulations [3]. The most frequent type of ASD is an ostium secundum ASD, which is caused by a true loss of septum premium tissue [4].

Hemodynamic adaptation occurs in ASD patients as a result of compensatory hyperdynamic RV contraction [5], pulmonary vasodilation [6]' and an increase in preload reserve and pulmonary vascular reserve. The baseline indices of RV contraction in patients with ASD would be substantially greater due to the compensatory hyperdynamic RV myocardium. This was explained by the fact that increased volume overload causes an increase in the initial length of cardiac fibers, which in turn causes contraction force to increase following Starling's rule, as previously stated. The increased preload imposed on the RV causes patients with open ASD to have a high baseline RV systolic function as a result of this [7,8].

Because the pulmonary vascular bed is a low-resistance system, a significant amount of blood flow can be tolerated without an appreciable increase in PA pressure [6] the PVR may be reduced even further when exercising. Because of the large RV/pulmonary preload reserve, many patients with ASD do not have any symptoms until their third or fourth xdecade [6,9].

The reduction in volume strain on the right heart occurs as a result of the ASD closure. There has been a reduction in both PAP and the size of the right heart cavity as a result of this procedure [10,11].

The aim of our study was to assess changes in RV systolic function before and after ASD closure either by surgery or transcatheter closure.

## 2. METHODS

The study was performed in Echocardiography Lab in the Cardiovascular medicine department, Tanta University Hospital. After examination by 2-D Echocardiography, we included 70 patients who were candidates for ASD closure, and we had subdivided our patients into two groups according to the suitability of ASD device closure. 35 patients were not candidates for device closure and were referred for surgical ASD closure and were included in group A. 35 patients were suitable for device ASD closure and were included in group B. All the patients were assessed for RV systolic function 12-24 H before and 6 months after ASD closure, by 2D echocardiography examination and measuring TAPSE, FAC, tissue Doppler assessment of S wave velocity, and by 2D speckle tracking assessment of RV GLS.

### 2.1 Inclusion Criteria

Inclusion criteria were patients diagnosed with isolated Secundum ASD by 2D Echocardiography and who were suitable for ASD device closure were included in group B. Other patients diagnosed with ASD secundum, and were not candidates for device closure, were referred for surgery and included in group A. Patients had Significant left to right shunt with Qp: Qs ratio of more than or equal to 1.5:1. Presence of signs of right ventricular volume overload as dilated RV, RA. Symptoms due to significant shunt.

### 2.2 Exclusion Criteria

Exclusion criteria were patients with associated other congenital cardiac anomalies. Patients with other types of ASD rather than Secundum defects. Patients with other causes of right ventricular dysfunction.

All patients were given a history that included their age, sex, body surface area, and symptoms that indicated a significant ASD. A complete general and local cardiac examination is performed. Surface electrocardiography with 12 leads (ECG). Complete blood count, international normalized ratio, clotting time, bleeding time, renal function tests, C-reactive protein, and virology are all routine laboratory investigations.

ASD closure: Surgical ASD closure was recommended for ASDs with inadequate or missing superior and posterior rims together, or the Inferioposterior (IVC) rim alone. The procedure was done under general or local anesthesia with echocardiographic and fluoroscopic guidance for ASDs that were eligible for transcatheter closure. Transesophageal echocardiography was employed in some cases, mainly in adults, to document the defect's complete occlusion. A 5 F or 6 F sheath was used to get vascular access to the right femoral vein, and if necessary, the right femoral artery. Amplatzer devices were used to close the defects. All patients received short-term antibiotics and antiplatelet (for 6 months) after the procedure.

## 2.3 Full two-dimension Transthoracic Echocardiography (2D TTE)

All patients underwent TTE (Vivid E9, General Electric Corporation) 12-24 h before as well as 6 months after successful closure. All data was collected offline by only one observer and averaged over 5 cardiac cycles. Measurement was done according to the recommendation of the American Society of Echocardiography [12] for the evaluation of the following: Visualization and examination of ASD and its hemodynamic consequences by obtaining subxiphoid windows (frontal long axis and sagittal short axis), the apical 4-chamber view was used for evaluation of the hemodynamic consequences of ASD and signs of right ventricle volume overload, to ensure that the patients were indicated for ASD closure, by Measurements of RVED basal diameter measurements and RVEDD\LVEDD ratio as an indicator of cardiac geometry) [12,13]. Also, an apical 4-chamber view was used for the evaluation of estimated systolic pulmonary artery pressure (ESPAP) obtained with the TR jet approach. PAH is classified According to Baseline RP: Normal PASP is less than 40 mmHg mild (40-49) mmHg, moderate (50-59 mmHg), and severe if equals or more than 60 mmHg [14].

## 2.4 Right Ventricular Systolic Function

Right ventricular systolic function was assessed using RV focused4-chamber view [14] by assessment of fractional area change (FAC), tricuspid plane annulus systolic excursion (TAPSE) Figs. 1, 2, and systolic velocity of tricuspid annulus (S/wave) Figs. 3, 4.

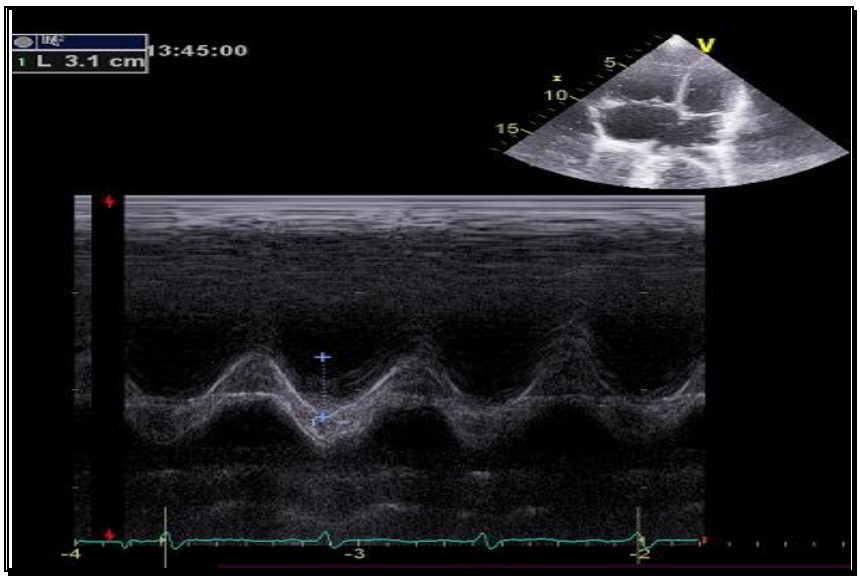


**Fig. 1. Apical 4 chamber view demonstrating assessment of TAPSE before ASD closure, showing TAPSE of 31MM**

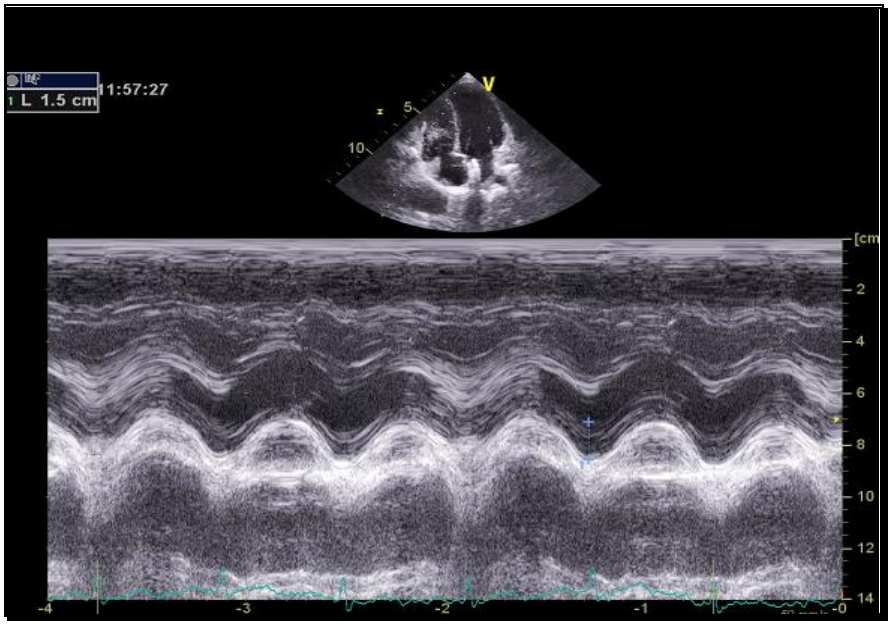


**Fig. 2. Showing a decrease in TAPSE results of 1.5 cm after device closure in the same patient**

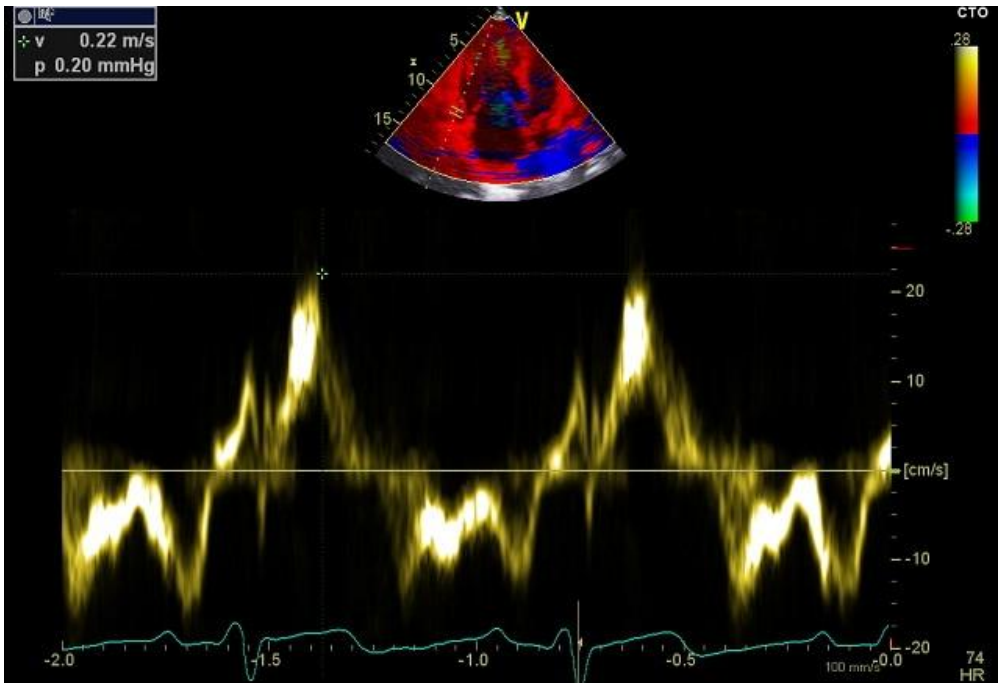


**Fig. 3. Tissue Doppler S' wave velocity measurement before ASD device closure showing S' wave velocity of 22CM \S**

### 2.5 Assessment of Global Longitudinal Strain (GLS) by 2D Speckle Tracking Imaging [12,15]

Assessment of global longitudinal strain (GLS) by 2D speckle tracking imaging [12,15] by manually tracing endocardial borders at the end of systole, and then automatically tracking the endocardial borders throughout each cardiac cycle to evaluate regional and global RV systolic function Figs. 5, 6.

The RV myocardium was subsequently separated into six segments: the basal RV lateral wall, the mid-RV lateral wall, the apical RV lateral

wall, the apical septum, the mid septum, and the basal septum).

A global longitudinal strain (GLS) value and a segment-specific peak systolic strain value PSS) were displayed after the reading analyst's approval.

The systolic strain of the six RV myocardial segments was measured. By averaging local strains (PSS) over the entire right ventricle using machine software, we were able to quantify the global longitudinal RV strain.

An average of the peak segmental strain values (PSS) of the RV free wall, supplied by the software is used to calculate RV free wall longitudinal strain.

The results were analyzed, using dedicated software (Echo PAC BT09, GE Ultrasound) [12,15].

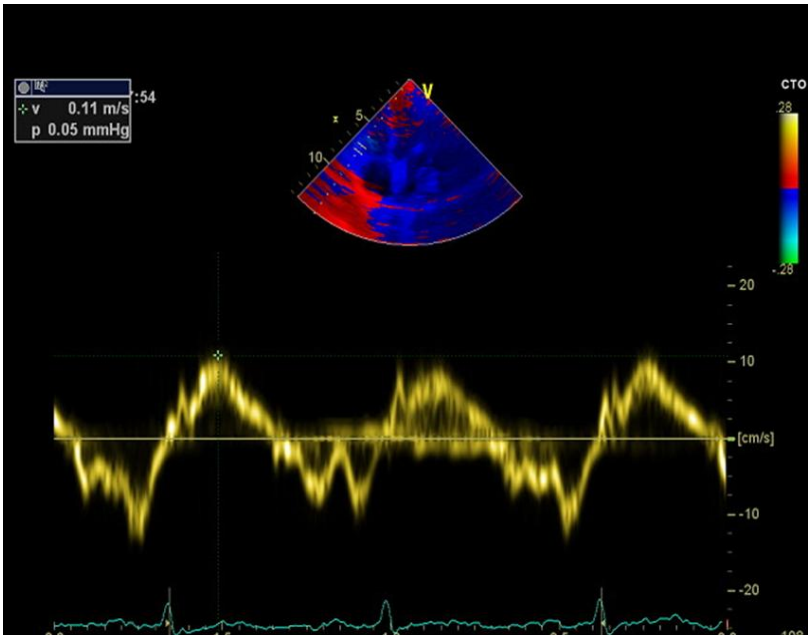


**Fig. 4. Tissue Doppler S' wave velocity decrease after ASD device closure in the same patient showing S' wave velocity of 11 CM \S**

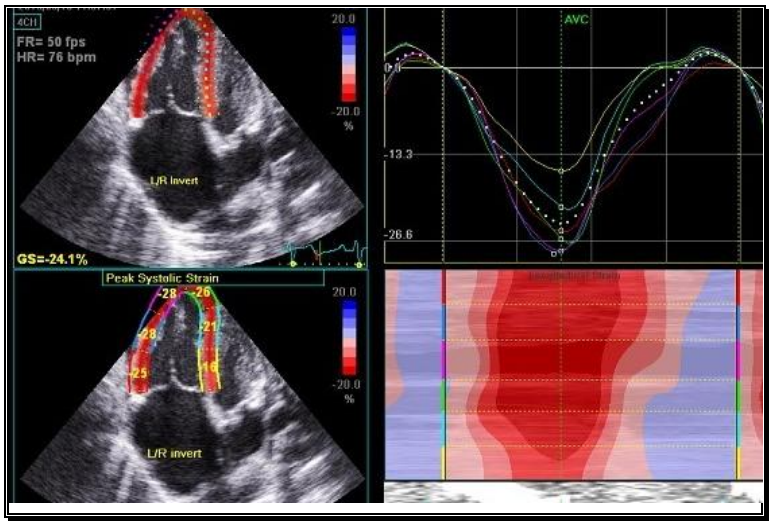


**Fig. 5. Showing GLS measurement of 24.1%, and lateral (free)wall strain of 27% before ASD device closure**

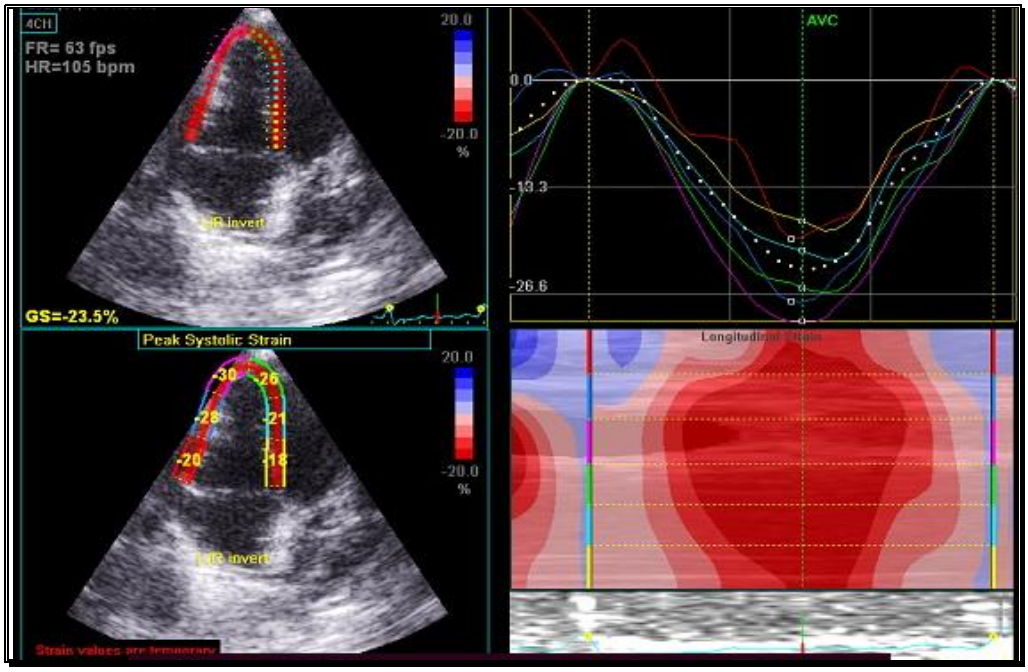


**Fig. 6. Showing decreases in GS (23.5%), and lateral (free) wall strain (26%) after ASD device closure in the same patient**

## 2.6 Statistical Analysis

IBM SPSS software package version 20.0 was used to analyze the data. (IBM Corp., Armonk, New York) Numbers and percentages were used to describe qualitative data. Kolmogorov-Smirnov tests were employed to determine whether or not the distributions were normally distributed. Minimum and maximum values, medians, and interquartile intervals were used to characterize quantitative data (IQR). The significance of the findings was evaluated at a 5% level of significance.

## 3. RESULTS

Regarding group A patients' age ranged from (6.60 – to 45.0) years, and the mean age was 15.21 ± 12.53 years. Group B patients' ages ranged from (6.0 – to 38.0) with a mean age of 53 ± 7.51. So, there was no statistically significant difference between both groups regarding age. Regarding sex distribution There were 21 females (60%),14 males (40%) were enrolled in group A, and 28 females (80%), 7 males (20%) were enrolled in group B. The mean size of ASD was 24.73 ± 3.77 mm in group A, and 15.53 ± 1.42 mm in group B. There was a significant difference of p <0.001 between both groups regarding ASD size Table 1.

There was a significant decrease in RVEDD at 6 months after both surgical and transcatheter device closure, when comparing pre and post-closure results with a p-value of <0.001* in both groups. as well as the RVEDD\LVEDD ratio showed a significant decrease at 6 months after both surgical and device closure when comparing pre and post-closure results with a p-value of <0.001* in both groups. PASP showed a significant reduction after both surgical and transcatheter device closure when comparing pre and post-closure results with a p-value of <0.001*. in both groups Table 2.

**Table 1. Comparison between both groups regarding ASD size**

| ASD size (mm) | Group A | Group B | Test of sig | p |
|---|---|---|---|---|
| Min-max | 15.36-30.25 | 12.90-17.96 | | |
| Mean ± SD. | 24.73 ± 3.77 | 15.53 ± 1.42 | t= | <0.001* |
| Median (IQR) | 25.68 (22.18 – 27.05) | 15.69 (14.61 – 16.69) | 13.508* | |

*IQR: Interquartile range, SD: Standard deviation, t: Student t-test, ASD: Atrial septal defect, p: p-value for comparing the studied groups, *: Statistically significant at p ≤ 0.05*

**Table 2. Comparison between pre and post-closure results in both groups regarding right ventricle geometry and pulmonary artery systolic pressure**

| | Group A | | | | Group B | | | |
|---|---|---|---|---|---|---|---|---|
| **RVEDD in (MM)** | **Pre** | **Post** | **Test of sig** | **P value** | **Pre** | **Post** | **Test of sig** | **P value** |
| Min. – Max. | 40.0 – 45.90 | 29.90 – 35.20 | t=59.506* | <0.001* | 33.60 – 45.50 | 22.90 – 32.0 | t=35.306* | <0.001* |
| Mean ± SD. | 43.13 ± 1.63 | 32.39 ± 1.60 | | | 39.03 ± 2.34 | 28.15 ± 2.31 | | |
| Median (IQR) | 43.60 (42.0 – 44.25) | 32.0 (30.90 – 34.0) | | | 39.60 (37.60 – 40.40) | 28.60 (26.70 – 29.45) | | |
| **RVED\LVED ratio** | | | | | | | | |
| Min. – Max. | 1.07 – 1.25 | 0.70 – 0.99 | t=20.387* | <0.001* | 0.80 – 1.20 | 0.60 – 1.03 | t=15.262* | <0.001* |
| Mean ± SD. | 1.17 ± 0.05 | 0.87 ± 0.08 | | | 1.03 ± 0.10 | 0.73 ± 0.10 | | |
| Median (IQR) | 1.19 (1.13 – 1.20) | 0.90 (0.80 – 0.92) | | | 1.08 (0.95 – 1.09) | 0.70 (0.70 – 0.80) | | |
| **PASP mmhg** | | | | | | | | |
| Min. – Max. | 25.0 – 57.0 | 17.0 – 39.0 | t=29.276* | <0.001* | 22.0 – 41.0 | 15.0 – 32.0 | t=47.469* | <0.001* |
| Mean ± SD. | 37.11 ± 8.69 | 26.11 ± 7.15 | | | 28.31±5.12 | 18.63±4.66 | | |
| Median (IQR) | 36.0 (30.0 – 40.50) | 26.0 (19.0 – 30.0) | | | 28.0 (24.0 – 30.0) | 18.0 (15.0 – 20.0) | | |

*IQR: Interquartile range, SD: Standard deviation, t: Paired t-test, p: p-value for comparing the studied groups: Statistically significant at p ≤ 0.05*

**Table 3. RV function indices results in pre and post-closure in both groups**

| | Group A | | | | Group B | | | |
|---|---|---|---|---|---|---|---|---|
| **Systolic function indices** | **Pre** | **Post** | **Test of sig** | **P-value** | **Pre** | **Post** | **Test of sig** | **P-value** |
| **TAPSE (mm)** | | | | | | | | |
| Min. – Max. | 21.10 – 35.50 | 16.30 – 29.20 | t=28.539* | <0.001* | 21.10 – 30.50 | 16.30 – 24.20 | t=23.859* | <0.001 |
| Mean ± SD. | 29.75 ± 3.98 | 23.64 ± 4.03 | | | 26.09 ± 2.88 | 20.40 ± 2.20 | | |
| Median (IQR) | 30.0 (27.27 – 33.30) | 24.60 (20.10 – 27.55) | | | 26.80 (24.35-28.17) | 20.10 (18.60-22.15) | | |
| Fractional area change (%) | | | | | | | | |
| Min. – Max. | 51.0 – 69.0 | 41.0 – 59.0 | t=23.931* | <0.001* | 50.0 -68.0 | 40.0-57.0 | t=72.955* | <0.001 |
| Mean ± SD. | 57.80 ± 5.06 | 47.63 ± 5.20 | | | 56.40±4.24 | 46.31±4.28 | | |
| Median (IQR) | 56.0 (54.0 – 61.0) | 45.0 (44.0 –51.50) | | | 55.0 (53.0-59.50) | 45.0 (42.50-49.50) | | |
| SWAVE (cm\s) | | | | | | | | |
| Min. – Max. | 13.20 – 18.90 | 9.70 – 15.20 | t=12.352* | <0.001* | 12.0-18.50 | 9.50-15.20 | t=14.659* | <0.001 |
| Mean ± SD. | 17.28 ± 1.42 | 13.03 ± 1.32 | | | 15.56±1.71 | 11.90±1.87 | | |
| Median (IQR) | 17.70 (16.40 – 18.35) | 13.20 (12.40 – 14.10) | | | 15.80 (14.15-16.75) | 12.00 (10.20-13.35) | | |

*IQR: Interquartile range, SD: Standard deviation, t: Paired t-test, p: p-value for comparing the studied groups' pre and post results, *: Statistically significant at p ≤ 0.05*

**Table 4. Strain indices results in pre and post-closure in both groups**

| | Group A | | | | | Group B | | | |
|---|---|---|---|---|---|---|---|---|---|
| Global longitudinal free wall strain % | | | | | | | | | |
| Min. – Max. | -32.66 – 31.66 | -26.33 – -20.66 | Z=4.590* | <0.001* | Min. – Max. | -32.0 – -27.0 | -26.0 – -20.33 | Z=5.163* | <0.001* |
| Mean ± SD. | -28.48 ± 10.52 | -23.24 ± 1.43 | | | Mean ± SD. | -29.39 ± 1.25 | -22.47 ± 1.42 | | |
| Median (IQR) | -30.33 (-30.66 – -29.49) | -23.0 (-24.17 – -22.33) | | | Median (IQR) | -29.33 (-30.33 – -28.47) | -22.33 (-23.0 – -21.33) | | |
| Global longitudinal systolic strain% | pre | post | | p | Global longitudinal systolic strain % | pre | post | | |
| Min. – Max. | -29.16 – -25.16 | -25.50 – 23.50 | Z=5.161* | <0.001* | Min. – Max. | -28.60 – -24.16 | -25.16 – -21.66 | Z=5.162* | <0.001* |
| Mean ± SD. | -27.13 ± 0.84 | -22.31 ± 8.02 | | | Mean ± SD. | -26.73 ± 0.78 | -23.23 ± 0.95 | | |
| Median (IQR) | -27.0 (-27.58 – -26.66) | -23.50 (-24.40 – -22.80) | | | Median (IQR) | -26.66 (-27.23 – -26.33) | -23.16 (-23.90 – -22.50) | | |

*IQR: Interquartile range, SD: Standard deviation, Z: Wilcoxon signed ranks test, p: p-value for comparing between pre and post subgroups, *: Statistically significant at p ≤ 0.05*

Both surgical and transcatheter closure groups had shown a significant decrease (P value<0.001*) in TAPSE, FAC, and Tissue Doppler S wave velocity after 6 months of closure Table 3.

The RV free wall longitudinal strain % showed a significant reduction (P<0.001*) after 6 months in both surgical and transcatheter ASD closure groups. As well as the global RV strain % which showed a significant decrease in both groups, surgical and transcatheter closure P<0.001*. Regarding peak systolic strain (PSS) of 6 segments of lateral (free) and septal walls, there was a significant decrease in peak systolic strain of apical, mid, and basal RV free walls after surgical and transcatheter ASD closure (p-value was P<0.001*for all three segments). While the peak systolic strain (PSS) of all three segments of the septum (apical, mid, and basal) showed insignificant differences after surgical ASD closure with p values of (0.751, 0.711, 0.377) respectively. As well as in group B (PSS) of septal segments could show insignificant differences after 6 months of ASD device closure with p-values of (0.879, 0.304, and 0.282) respectively Table 4.

## 4. DISCUSSION

In patients with a left to right shunt or other RV disorders, the systolic function is an important prognostic sign. Before ASD repair, prior studies found that RV systolic function parameters were normal or exaggerated in open ASD and that these parameters were reduced to normal or subnormal values following the procedure [16,17]. We measured TAPSE, S' wave velocity, FAC, and global longitudinal strain by 2D speckle tracking analysis as the main parameters for RV systolic function. hence, cardiac remodeling occurs in the first few months after ASD closure, and a 3- to 6-month follow-up period is sufficient to demonstrate the RV remodeling effect [18,19].

Our results met with Kumar P et al. [20] observations on 14 patients who had surgical correction of ASD, they had reported a significant reduction of TAPSE (23.71 ± 1.6 mm vs –19.50 ± 1.41 mm after 3 months of surgical closure, P < 0.0001). Contrarily, in disagreement with our results Balci et al. [21] included 19 patients with mean age (40.0 ± 13.51) and couldn't observe changes in TAPSE after ASD percutaneous closure after 3 months of closure (2.49 ± 0.46 cm vs 2.51 ± 0.32 cm, p=0.078). This discrepancy between our results might be due to the short-term follow-up in their study and limited patient sample size as well as the difference in the mean age between our patients.

In concordance with our results, Vijayvergiya et al. [22] Also had revealed a significant reduction in RV systolic function following 6 months of surgical closure of ASD, by comparing results for S' wave, (17.75±3.5) cm\s in open ASD vs (11.55±4.9) cm\s at one-month post-surgery vs 11.60±3.4 cm/s at 6-month post-surgery (p<0.001).

In line with our results, Akula et al. [7] found a significant reduction in tricuspid annular systolic velocity from 16.0±2.8 cm/sec to 13.1 ±3.0 cm\s after one-month vs 13.7-2.8 cm\s after 6 months of ASD closure after including Seventy-three patients with a range of age 26 - 17 years in their study.

Our results were in agreement with Vitarelli et al. [23] who evaluated 39 patients before and 6 months after closure and demonstrate a significant reduction in RVFAC (%) 58 ± 12 to 52 ±9 (p<0.05).

Also, Eroglu et al. [24] results regarding RVFAC met our results as FAC (%) decreased significantly from 53.2 ± 4.3 to 40.6 ± 4 at 24 H post-closure vs 45.7 ± 5 after 1 month of closure p<0.0001, of note, that in Eroglu's study the RV systolic function indices (as RVEF, TAPSE, RVFAC, RVMPI) had shown decrease immediately after 24 hours of closure, with re increase to near baseline values at first month which could be similar to acute, transient deterioration of RV function immediately after unloading of RV.

On the contrary, Islami et al. [25] results regarding RVFAC in the post-closure group were significantly increased compared to pre-closure results, which was not in agreement with our results. Their results regarding RV FAC were 37.6±8.8 in open ASD vs 48.6±7.3 in the post-closure group, this discrepancy with our results may be related to significant lower basal FAC in their open ASD patients compared to their control group, so their ASD patients might have a mild basal degree of RV systolic dysfunction which improved after ASD closure, on contrary to our open ASD patients' results, which couldn't show any abnormal lower basal FAC, and all the post-closure results decreased to normal ranges.

Keeping with our results Di Salvo et al. [26] showed a significant decrease in the global longitudinal strain of the RV free wall, although their values dropped to subnormal levels after 6 months of ASD closure in the post-surgical group and this was consistent with prior research, (194-195) [27,28] they had attributed their results to the direct effect of cardiopulmonary bypass on RV function, as well as the effect of greater myocardial temperature during surgery owing to its exposed position in the mediastinum [27].

Our results were also augmented by Suzuki et al. [28] findings after investigating the effect of ASD closure either surgically or by percutaneous device closure on 120 adult patients with ASD at 6 months after closure as in our study. In their group of patients without residual symptoms RV-GLS % had shown significant reduction (from − 28 ± 11 to − 24 ± 7% $P < 0.01$) after closure which met our results. However, RV-GLS % showed a non-significant increase in ASD patients with residual symptoms after ASD closure in their results (−20± 7 in pre-closure vs −21±8 at 6 months follow up after closure), of note that we didn't report any patients with residual symptoms after closure in our cohort, and this may be the cause of the discrepancy between the results of this symptomatic group and our asymptomatic patients.

Additionally, our results were augmented by Bussadori et al. [29] results after including 21 adult patients with a Mean age of 28 ± 9.5 ranging from 18–49 years, they could report a significant reduction in the global longitudinal RV strain%, ( GLS of −23.95±5.24 before closure vs −17.04 ±5.94) after 24 hours of ASD Closure with a p-value of 0.00016. Also, Bussadori et al. [29] results of the 3 segments free wall PSS could demonstrate significant reduction, with no significant reduction in the three segments of the septal PSS, which met with our finding after 6 months of ASD closure.

Also, our results were agreed by Jategaonkar et al. [16] reported a significant reduction in the GLS of the right ventricle after ASD closure at 3 months of follow up 23.4±4.5 vs 21.4±4.3 p<0.05. Additionally, their results revealed a significant reduction of the PSS of the lateral mid-wall segment 29.4±7.7 in pre-closure vs 26.6±6.3 in the post-closure group p<0.05, and in the lateral wall apical segment PSS 27.0±6.7 in pre-closure vs 24.3±6.4 in post-closure group, p< 0.05 which met with our results.

However, in contrast to our results of the septal apical segments PSS, Jategaonkar et al. [16] showed a significant reduction in their results 21.2±7.4 in the preclosure group vs 17.9±7.1 in the post-closure group p<0.05, and this didn't match our results as we demonstrated non-significant changes in PSS of the septal apical segment in post-closure group.

In addition, the contradiction between our results and Jategaonkar et al. [16] was regarding basal lateral segment PSS, as we could observe a significant reduction in PSS of the basal lateral segment after ASD closure, which was not in line with Jategaonkar et al. [16] hence they couldn't find significant difference regarding this segment after ASD closure. This discrepancy between results might be because they examine their patients after 3 months of closure so Basal segment remodeling hadn't occurred yet as proved by Ko HK et al. [30] when they demonstrated remodeling in the basal segment at 6 months follow up post closure.

Our results regarding RV lateral basal segment were in agreement with Di Salvo et al. [26] when they enrolled two groups the first one included 15 patients with successful ASD device closure with a mean age of 9-3 years), While the second group included 15 age- and gender-matched patients after successful ASD surgical closure with the mean age (of 9-3 years). They had documented reduced RV lateral basal segment deformation in post-closure patients and also noticed that reverse remodeling of the basal segment might not have happened until 6-months post-closure because all of their patients had ASD closure more than 6 months before their research. And this was in line with our results regarding the RV lateral basal segment in post device closure group.

However, our results of insignificant reduction in mid and basal septal segments in the post catheter closure group were not in line with Di Salvo et al. [26] results in their ASD-Device post-closure group as On the basal and mid-segments of the interventricular septum, they found a considerable decrease in longitudinal regional deformation values. This was due to the deposition of a non-contractile component within the interatrial septum, which impacts the shortening of the septal wall's basal and mid segments. This supported a previous study showing that device size, site, and distance between the device and the mitral annulus all

affect the severity of septal longitudinal function impairment [31].

On contrary with our results, Agha et al. [32] results after assessment of strain by 2D speckle tracking study of RV after 6 months of device closure in pediatric age group. There was a significant increase in the RV global longitudinal strain (GLS) 6 months post-ASD closure. (-20.17 ± 3.14% in the pre-closure group vs -25.86 ± 5.02% in the post-closure group, $p < 0.0001$), which was not in agreement with our results, however in Agha et al. [32] the range of their patients 'age was (3-9) years, and a mean value of (6.01±3.19) so they included only the pediatric age group and didn't include adult patients as in our study.

On the other hand, Suzuki M et al. [28] results had some discrepancy with ours regarding strain indices, as after including 120 adult patients with ASD in their study, they could observe blunted response and non-significant difference in RV GLS % − 20 ±7 Vs − 21 ± 8 after 6 months of ASD closure in their group of patients with residual symptoms. Furthermore, Suzuki M could also observe a significant reduction of RV GLS in the subgroup of patients without persistent symptoms after ASD closure (from − 28 ± 11 to − 24 ± 7%, $P < 0.01$), and this was in agreement with our study.

RV-GLS was considered by Suzuki M et al. [28] to be an important marker of RV myocardial irreversible impairment, which may lead to persistent symptoms even after ASD closure has been completed. As a result of RV volume overload and compensatory mechanisms like RV dilation and myocardial hyper contraction, the previously stated disparity in their study's outcomes may be explained [28]. Myocardial damage will ensue as a result of prolonged RV hyper contraction and myocardial overstretch [6]. Because of the lower PVR, the pulmonary vasculature can initially receive a large volume of cardiac output. Chronic pulmonary overflow generates shear stress on the pulmonary artery endothelium, leading to pulmonary vasculopathy [33,34]. Due to the persistent rise in PA pressure, even after ASD closure, RV "unloading" could not be completed in those patients with persisting symptoms, suggesting that these patients had already exhausted their hemodynamic reserve capacity. It should be noticed that there was no increase in pulmonary artery pressure nor residual symptoms in our patients after ASD closure and this may explain the discrepancy between our results.

Due to current technology's limitations, which don't allow for measurements of RV radial or circumferential deformation, we were only able to measure longitudinal strain, yet RV contraction is thought to be mainly longitudinal. It was not possible to anticipate the long-term effects of ASD closure on RV function from our study, as the sample size was small and the follow-up was limited to a mid-term period. As a result, in order to corroborate our findings, studies with a bigger sample size and long-term follow-up should be conducted, as well as additional research. Absence of control group to compare pre and post results. Adults and children are included in our study. In future studies, pediatric and adult patients should be assessed separately, particularly when examining changes in the size of the heart chambers.

## 5. CONCLUSION

The right ventricle's size and function are affected by a large shunt caused by an ASD secudium. ASD and its consequent volume overload resulted in higher RV myocardial contraction, leading to an increase in strain values and RV systolic function indices, which were reduced and returned to normal values when the left-to-right shunt was eliminated, and the defect was closed. Two-dimensional strain measurements provide a distinct tool for the assessment of global and regional cardiac function. Myocardial fibers on the right side of the interventricular septum appear to be less affected by right ventricular volume overload than those on the free wall of the right ventricular. A consistent indicator of right ventricular function, particularly RV free wall strain, appears to be based on loading conditions.

## CONSENT AND ETHICAL APPROVAL

The study was done after being approved from the institutional ethical committee, Tanta University. Informed written consent was obtained from all patients included.

## COMPETING INTERESTS

Authors have declared that no competing interests exist.

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