

Temporal profile of right and left ventricular wall deformation analysis using 2D speckle tracking echocardiography following atrial septal defect closure



Shahnawaz Ali Ansari¹, DM; Aditya Kapoor^{1*}, DM; Arpita Katheria¹, DM; Harshit Khare¹, DM; Arshad Nazir¹, DM; Ankit Sahu¹, DM; Roopali Khanna¹, DM; Sudeep Kumar¹, DM; Surendra Kumar Agarwal², MCh; Shantanu Pande², MCh; Prabhat Tewari³, MD; Bipin Chandra², MCh; Naveen Garg¹, DM; Satyendra Tewari¹, DM

*Corresponding author: Sanjay Gandhi PGIMS, Raibareli Rd, Lucknow, Uttar Pradesh, 226014, India.
E-mail: akapoor65@gmail.com

ABSTRACT

BACKGROUND: Analysing temporal strain changes in right ventricular (RV) and left ventricular (LV) walls post-atrial septal defect (ASD) closure is of clinical importance.

AIMS: We aimed to evaluate acute/short-term changes in RV/LV wall deformation after ASD closure using two-dimensional speckle tracking echocardiography (2D-STE).

METHODS: A total of 43 patients with ASD and 20 controls had echocardiograms before and after ASD closure.

RESULTS: Of the 43 patients with secundum ASD (mean age 27.37 years), 48.8% were closed surgically, while 51.2% underwent device closure. At baseline, LV global longitudinal strain (GLS; 2-chamber view GLS: 16.95% vs 20.73%; $p=0.0001$, apical long-axis view GLS 16.48% vs 20.90%; $p=0.0001$, 4-chamber view GLS 16.93% vs 21.56%; $p=0.0001$, average GLS 16.75% vs 21.31%; $p=0.0001$) and RV GLS (19.22% vs 24.27%; $p=0.0001$) were significantly lower in the patients with ASD compared to controls. After closure, the average LV GLS rapidly improved at 24 hours from baseline (16.75% to 17.28%; $p=0.004$), with sustained increases at 1 and 3 months (18.16% and 19.40%; $p=0.001$). The mean RV GLS also improved at all serial timepoints (baseline, 24 hrs, 1 month, and 3 months) with values of 19.22%, 19.85%, 20.70%, and 22.23%, respectively ($p=0.0001$). As compared to surgery, LV GLS and RV GLS were much better in the device group (average LV GLS at 24 hrs, 1 month, and 3 months: 16.54% vs 17.98%, 17.34% vs 18.92%, and 18.80% vs 19.96%, respectively; mean RV GLS at 24 hrs, 1, and 3 months: 17.83% vs 21.78%, 18.73% vs 22.58%, and 20.70% vs 23.70%, respectively).

CONCLUSIONS: This GLS study demonstrates significant reverse remodelling of both the RV and LV after ASD closure. Device closure was associated with superior strain rate recovery compared to surgery at the 3-month midterm follow-up.

KEYWORDS: 2D speckle tracking echocardiography; ASD; global longitudinal strain

Secundum-type atrial septal defect (ASD) is a common congenital heart defect causing chronic right ventricular (RV) volume overload due to left-to-right shunting. If the ASD remains uncorrected, the progressive increase in RV dimensions and volume and pressure overload of the RV can also cause decreased left ventricular (LV) filling and decreased LV preload due to a leftward shift of interventricular septum and adverse biventricular remodelling^{1,2}. Transcatheter closure is now preferred over surgery for suitable candidates^{3,4}.

Reduced ventricular performance immediately after surgery for congenital heart disease has been reported, followed by a recovery over months to years, but the mechanisms behind these observations are incompletely understood⁵⁻⁷. Cardiac remodelling is an early postinterventional phenomenon and is usually expected to occur during the first few months after ASD closure⁷.

Two-dimensional speckle tracking echocardiography (2D-STE) enables precise measurement of segmental and global ventricular function, independent of angle and geometry, revealing subtle dysfunction in both ventricles⁸.

Therefore, serial assessment of LV and RV strain following ASD closure, either through transcatheter or surgical methods, is a promising approach to understanding the timeline of myocardial recovery post-ASD closure. Despite attempts to quantify changes from baseline to 24 hours, 1-6 months, and 1 year, the temporal profile of biventricular function recovery after ASD closure remains an active area of study⁹⁻¹⁴.

Some studies show LV performance improves before RV performance, while others document earlier RV strain improvements due to direct haemodynamic unloading, with LV strain improvements becoming evident later and suggesting a recovery lag¹⁵⁻¹⁷.

LV and RV strain parameters normalise faster and earlier in patients undergoing transcatheter closure due to its minimally invasive nature, reduced periprocedural myocardial stress, and absence of proinflammatory effects from cardiopulmonary bypass, hence facilitating quicker recovery^{8,10,18,19}.

Delayed diagnosis and limited access to advanced healthcare often mean that patients with ASD in India receive device or surgical closure later in life compared to their Western counterparts. Whether this long-term RV volume overload leads to chronic and persistent biventricular myocardial dysfunction, as detected by STE, represents an unmet area of clinical need. Limited Indian data show significant baseline impairments in LV and RV strain, with better and earlier strain recovery following transcatheter closure compared with surgical closure²⁰.

Despite advancements, gaps remain in understanding the long-term trajectory and clinical implications of strain recovery. Standardised protocols for serial strain assessment, especially in resource-limited settings like India, and the comparative

Impact on daily practice

By offering detailed insights into ventricular deformation, this study provides valuable knowledge for optimising closure methods and individualising patient care. This ultimately contributes to improved clinical outcomes and long-term prognosis for patients undergoing atrial septal defect closure.

efficacy of transcatheter versus surgical closure for ventricular strain recovery need further exploration. We aimed to evaluate changes in LV and RV strain parameters at 24 hours, 1 month, and 3 months after intervention in patients with secundum ASD undergoing device or surgical closure.

Methods

Patients who were diagnosed with secundum ASD on echocardiography and scheduled for device/surgical closure in the Department of Cardiology/Cardiovascular and Thoracic Surgery, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow were enrolled in this prospective analytical study.

SAMPLE SIZE

The sample size was based on the paired differences detected in measured variables of the treatment and control groups, as reported previously²¹. The effect size of the mean difference between the two groups was assumed to be 0.8. To achieve a minimum two-sided 95% confidence interval (CI) and 80% power, the estimated sample sizes for the treatment and control groups were 39 and 19, respectively. Finally, 43 patients with ASD and 20 controls were included in the study. Sample size was estimated using G*power, version 3.1.9.2 (<https://www.psychologie.hhu.de/arbeitsgruppen/allgemeine-psychologie-und-arbeitspsychologie/gpower>).

INCLUSION CRITERIA

The enrolled patients met the following eligibility requirements:

1. Patients diagnosed with isolated secundum ASD by 2D echocardiography and who were suitable for ASD device closure as per previous inclusion criteria²¹.
2. Other patients diagnosed with secundum ASD and not candidates for device closure were referred for surgery.
3. An informed consent was obtained from the patients or their legal guardians.

EXCLUSION CRITERIA

Exclusion criteria included an insufficient ASD rim (except the aortic rim), other types of ASD and any associated condition

Abbreviations

2D	two-dimensional	LVEDD	left ventricular end-diastolic diameter	RV FWLS	right ventricular free wall longitudinal strain
ASD	atrial septal defect	LVESD	left ventricular end-systolic diameter	RVSP	right ventricular systolic pressure
GLS	global longitudinal strain	LVPWT	left ventricular posterior wall thickness	STE	speckle tracking echocardiography
LV	left ventricular	RV	right ventricular	TTE	transthoracic echocardiography

that may have resulted in systolic or diastolic dysfunction, such as any type of arrhythmia, especially atrial fibrillation, hypertension, diabetes, ischaemic heart disease and heart failure, or LV diastolic dysfunction.

A total of 43 patients with ASD and 20 controls were recruited for the study between September 2022 and April 2024. The study was approved by the institutional ethics committee with IEC no. 2024-171-DM-EXP-60. After satisfying the inclusion and exclusion criteria, written informed consent was obtained from eligible candidates, followed by a detailed history and physical examination, a 12-lead electrocardiogram, and an echocardiogram (including strain analysis). All patients were subjected to complete transthoracic echocardiography (TTE) using different echocardiographic modalities, such as 2D-TTE and 2D-STE, before the intervention, and at 24 hours, 1 month, and 3 months after intervention.

ECHOCARDIOGRAPHY

Echocardiography was performed using a GE Vivid E9 XDclear echo machine (GE HealthCare). Conventional echo-Doppler parameters including left ventricular end-diastolic diameter (LVEDD), left ventricular end-systolic diameter (LVESD), interventricular septal thickness (IVST), left ventricular posterior wall thickness (LVPWT), and LV ejection fraction (LVEF) were measured according to the American Society of Echocardiography guidelines²².

Speckle tracking echocardiography was used for estimating LV and RV global longitudinal strain (GLS). Standard greyscale images were obtained in three apical views (apical 2-, 3- and 4-chamber views) and were analysed offline on a dedicated workstation (EchoPAC PC, version 202 [GE HealthCare]). The average peak systolic longitudinal strain of all myocardial segments in the three views, calculated using the automated function imaging (AFI) application and demonstrated in a bull's eye plot, was defined as LV GLS and used for analysis. A 17-segment polar plot (bull's eye) was used to assess visual and quantitative representations of regional LV functions by plotting colour-coded values of peak systolic strain²³. A value of LV GLS of greater than -18% was regarded as abnormal²⁴.

In this study, strain values were expressed in absolute values, and larger absolute values indicated better cardiac ventricular function. Myocardial strain analysis was performed by two independent, blinded observers.

RV function assessment: RV free wall longitudinal strain (RV FWLS) was obtained from the standard 2D greyscale image of the RV focused apical 4-chamber view with a frame rate of ~ 50 -70 frames/s. RV FWLS was calculated as the average of the basal, mid- and apical RV free wall segments, and a value greater than -20% was defined as pathological^{25,26}.

Transcatheter ASD closure was performed under local anaesthesia utilising fluoroscopic and echocardiographic guidance. The ASD was assessed by TTE (or transoesophageal echocardiography if anatomy was not clear by TTE). Patients with at least 5 mm of rim in all planes were considered for device closure. The device chosen for closure was 0-2 mm larger than the measured diameter in cases with adequate rims. If the superior/anterior rim was deficient, a device 4 mm larger than the stretched balloon diameter was chosen.

STATISTICAL ANALYSIS

In the present study, all qualitative data were analysed using descriptive statistics followed by a Pearson's chi-square test. All quantitative data were analysed using unpaired t-tests and paired sample t-tests. Along with these, Pearson product-moment correlation analysis was also used to see correlation among different variables. All tests were performed using the computer program SPSS, version 25.0 (IBM). P-values > 0.05 were considered not significant; however, $p < 0.05$, $p < 0.01$, and $p < 0.001$ were considered statistically significant.

Results

BASELINE CHARACTERISTICS

The study included 43 patients with ASD, of whom 15 were males (34.9%), 28 were females (65.1%), and the mean age was 27.37 ± 16.37 years (**Central illustration A**). There were no differences in age or sex distribution between patients and controls. The age-wise distribution is summarised in **Table 1**. The distribution of cases according to the size of the ASD in the study group showed that out of 43 total cases, 37.2% (16 cases) had an ASD size of ≤ 20 mm, 46% (20 cases) had an ASD of 21-30 mm, 11% (5 patients) had an ASD of 31-40 mm, and 4.7% (2 cases) had an ASD larger than 40 mm. There was a statistically significant difference in the distribution of ASD sizes ($\chi^2 = 18.744$ and $p = 0.000$).

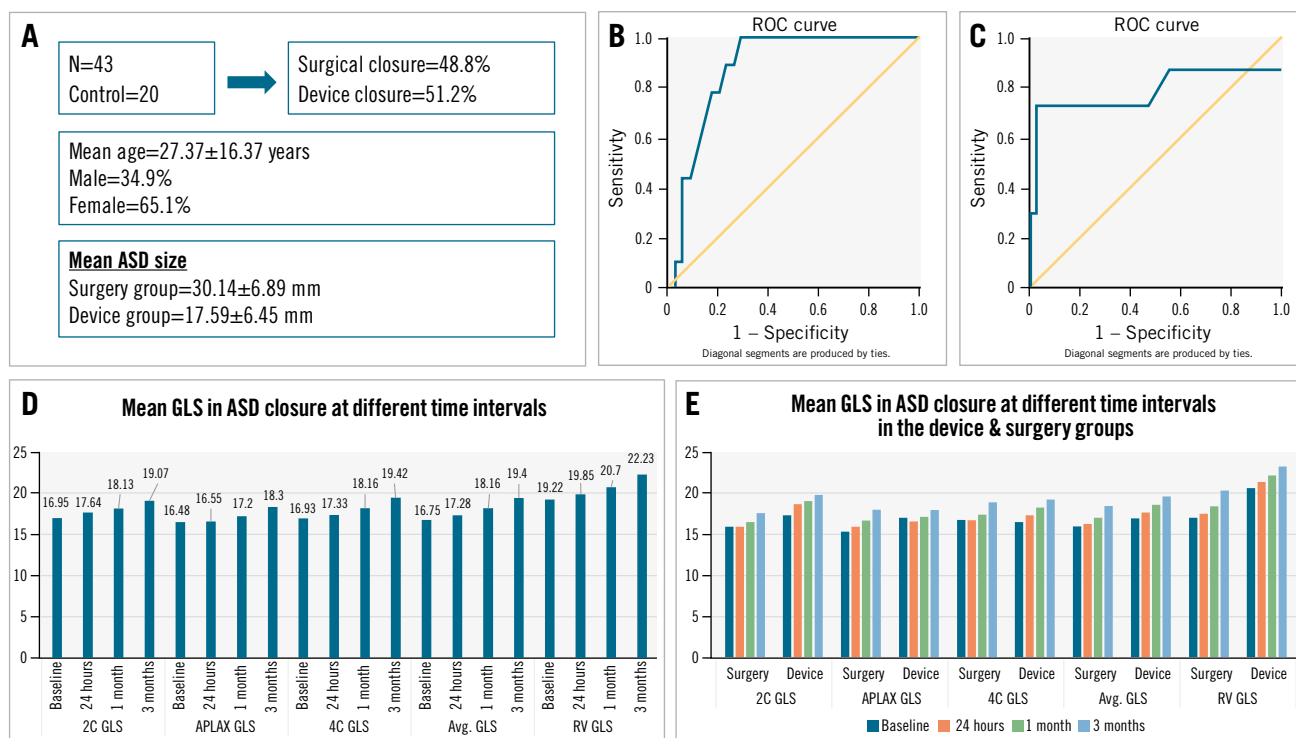
Out of 43 cases, 21 cases (48.8%) were closed using surgical techniques, while 22 cases (51.2%) utilised device-based closure methods. The surgical treatment group had a significantly larger ASD size compared to patients who underwent transcatheter closure (30.14 ± 6.89 mm vs 17.59 ± 6.45 mm; $p < 0.0001$) (**Central illustration A**). There was no statistically significant difference in the distribution of closure methods ($\chi^2 = 0.023$ and $p = 0.879$). This suggests that the two approaches to ASD closure were utilised in nearly equal proportions within the studied population (**Table 1**). Out of 22 cases, 19 cases (86.3%) were deployed with an Amplatzer septal occluder (Abbott), while 3 cases (13.6%) were deployed with a Cocoon septal occluder (Vascular Innovations).

ECHOCARDIOGRAPHIC PARAMETERS

The ASD group had significantly lower LVEDD, as compared to controls (37.49 ± 4.71 mm vs 40.75 ± 3.65 mm; $p = 0.008$), while the LVESD, IVST, and LVPWT were comparable (**Table 1**). The tricuspid regurgitation velocity and right ventricular systolic pressure (RVSP) of cases were 2.78 ± 0.59 m/s and 41.37 ± 14.65 mmHg, respectively, as shown in **Table 1**.

The GLS of the LV was significantly lower in the ASD patients, as compared to controls (2-chamber view [2C] GLS was $16.95 \pm 3.68\%$ vs $20.73 \pm 1.78\%$; $p = 0.0001$, apical long-axis view [APLAX] GLS was $16.48 \pm 3.31\%$ vs $20.90 \pm 1.63\%$; $p = 0.0001$, 4-chamber view [4C] GLS was $16.93 \pm 3.87\%$ vs $21.56 \pm 1.33\%$; $p = 0.0001$, and the average GLS was $16.75 \pm 2.35\%$ vs $21.31 \pm 1.16\%$; $p = 0.0001$). The RV GLS was also significantly lower in the patient group ($19.22 \pm 3.59\%$) compared to the control group ($24.27 \pm 2.96\%$; $p = 0.0001$). The percentage of patients with LV GLS greater than -18% was 86.04% compared with 0% in the controls ($p = 0.0001$). Similarly, the percentage of patients with RV GLS greater than -20% was 69.76% compared with 10% in the controls ($p = 0.0001$) (**Table 1**, **Figure 1**).

The role of 2D strain echocardiography in assessing biventricular function in ASD closure.



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A) Patient demographics; (B) the ROC curve of RV GLS showed an AUC of 0.877, which is 87.7% ($p=0.001$); (C) the ROC curve of LV GLS showed an AUC of 0.772, which is 77.2% ($p=0.024$); (D) the improving trend of mean LV GLS and RV GLS values at different timepoints in our study; (E) the mean LV GLS and RV GLS parameters at all serial timepoints, which were significantly better in the device group than the surgery group. 2C: 2-chamber view; 4C: 4-chamber view; APLAX: apical long-axis view; ASD: atrial septal defect; AUC: area under the curve; avg.: average; GLS: global longitudinal strain; LV: left ventricular; ROC: receiver operating characteristic; RV: right ventricular

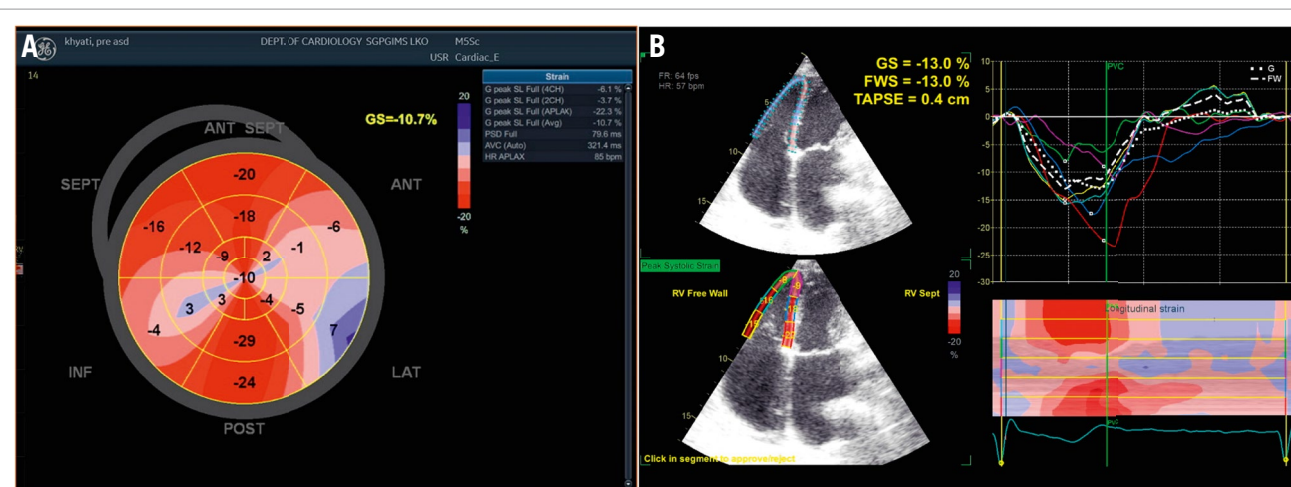


Figure 1. Impaired LV (bull's eye plot) and RV GLS values of a patient in our study. A) LV GLS values; (B) RV GLS values. 2C: 2-chamber view; 4C: 4-chamber view; ANT: anterior; APLAX: apical long-axis view; FWS: free wall strain; GLS: global longitudinal strain; INF: inferior; LAT: lateral; LV: left ventricular; POST: posterior; RV: right ventricular; SEPT: septal; TAPSE: tricuspid annular plane systolic excursion

Table 1. Baseline demographics and echocardiographic parameters of patients and control.

Parameter	Patients (n=43)	Control (n=20)	p-value
Baseline demographics			
Age, years			
Mean age	27.37±16.37	28.65±17.63	0.779
≤20 years	16	7	0.58
21-30 years	5	4	
31-40 years	13	3	
41-50 years	6	3	
51-60 years	3	3	
Sex			
Male	15 (34.9)	9 (45.0)	0.441
Female	28 (65.1)	11 (55.0)	
ASD size, mm			
Mean ASD size	23.72±9.15	NA	0
ASD size ≤20 mm	16	NA	
ASD size 21-30 mm	20	NA	
ASD size 31-40 mm	5	NA	
ASD size >40 mm	2	NA	
Closed surgically	21 (48.8)	NA	0.879
Closed by device	22 (51.2)	NA	
Mean device size used, mm	20.45±6.84	NA	
Echocardiographic parameters			
LVEDD, mm	37.49±4.71	40.75±3.65	0.008
LVESD, mm	22.40±3.52	21.45±2.82	0.297
IVST, mm	9.19±0.88	9.25±0.85	0.787
LVPWT, mm	9.21±0.86	9.35±0.81	0.541
TR velocity, m/s	2.78±0.59	NA	0.0001
RVSP, mmHg	41.37±14.65	NA	0.0001
2C GLS, %	16.95±3.68	20.73±1.78	0.0001
APLAX GLS, %	16.48±3.31	20.90±1.63	0.0001
4C GLS, %	16.93±3.87	21.56±1.33	0.0001
Avg. GLS, %	16.75±2.39	21.31±1.16	0.0001
LV GLS greater than -18%	37 (86.04)	0 (0)	0.0001
RV GLS, %	19.22±3.59	24.27±2.96	0.0001
RV GLS greater than -20%	30 (69.76)	2 (10)	0.0001

All values are mean±SD, n, or n (%). 2C: 2-chamber view; 4C: 4-chamber view; APLAX: apical long-axis view; ASD: atrial septal defect; avg.: average; GLS: global longitudinal strain; LV: left ventricular; LVEDD: LV end-diastolic diameter; LVESD: LV end-systolic diameter; LVPWT: LV posterior wall thickness; NA: not applicable; RV: right ventricular; RVSP: RV systolic pressure; SD: standard deviation; TR: tricuspid regurgitation

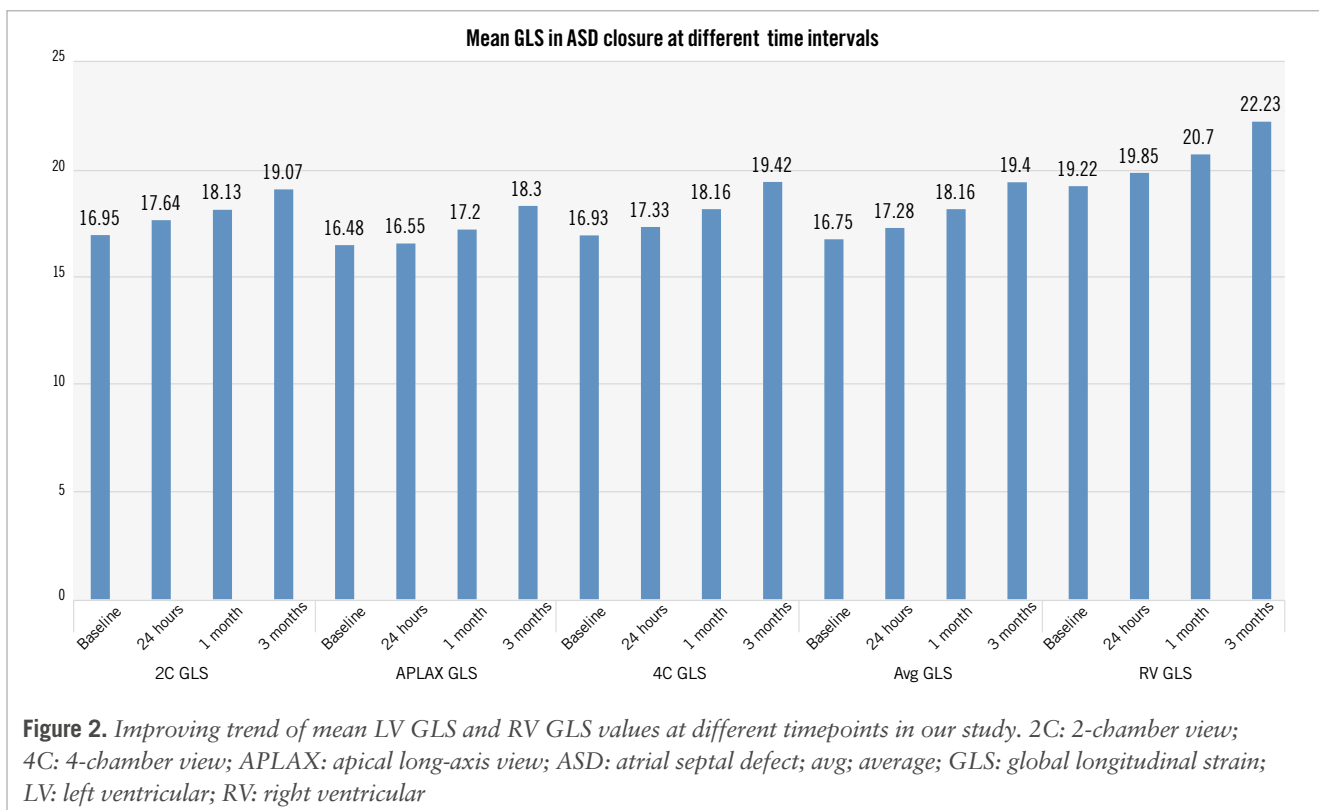
TEMPORAL CHANGES IN LV AND RV STRAIN FOLLOWING ASD CLOSURE

The mean 2C GLS improved from a baseline of 16.95±3.68% to 17.64±3.11% at 24 hours ($p=0.045$) (**Table 2, Figure 2, Central illustration D**). At 1 month after the procedure, the mean 2C GLS further increased to 18.13±2.75% ($p=0.001$), and at 3 months, the mean 2C GLS was 19.07±2.54% ($p=0.000$), reflecting substantial improvements over time. The change in the mean APLAX GLS from baseline to 24 hours (16.48±3.31% to 16.55±2.42%; $p=0.856$) was not significant. However, at 1 month and 3 months, the mean APLAX GLS showed sustained improvements (17.20±2.29% and 18.30±2.15%,

respectively; $p=0.000$). Similarly, the mean 4C GLS did not change significantly from baseline to 24 hours (16.93±3.87% to 17.33±3.03%; $p=0.154$). Significant and continued improvements were observed at 1 month (18.16±2.67%; $p=0.001$) and at 3 months (19.42±2.66%; $p=0.000$).

The mean average GLS showed a rapid improvement at 24 hours compared with baseline (from 16.75±2.39% to 17.28±2.46%; $p=0.004$), with continued increases at 1 month and 3 months (18.16±2.30% and 19.40±2.05%, respectively; $p=0.000$).

The mean RV GLS demonstrated significant improvements at all serial timepoints measured (baseline: 19.22±3.59, 24 hrs:



19.85±3.66, 1 month: 20.70±3.63, and 3 months: 22.23±3.36, p=0.0001) (Table 2, Figure 2, Central illustration D).

The mean percentage changes in 2C GLS, APLAX GLS, 4C GLS, and average GLS at 3 months were 12.50%, 11.04%, 14.70% and 15.82%, respectively, and the corresponding value for the mean percentage change in RV GLS was 15.66%. The percentage of patients with LV GLS greater than -18% at 3 months was 16.27% and those with RV GLS greater than -20% was 20.93%.

COMPARISON OF SURGERY VERSUS DEVICE GROUP

The mean LV GLS and RV GLS parameters at all serial timepoints was significantly better in the device group compared with those who underwent surgery (Table 3).

1. The average LV GLS at 24 hrs, 1 month, and 3 months in the device versus surgery group were 16.54±1.86% versus 17.98±2.78% (p=0.054), 17.34±1.79% versus 18.92±2.49% (p=0.023), and 18.80±1.75% versus 19.96±2.19% (p=0.051), respectively (Central illustration E).
2. Similarly, RV GLS at 24 hrs, 1 month, and 3 months in the device versus surgery group were 17.83±2.37% versus 21.78±3.66% (p=0.0001), 18.73±2.29% versus 22.58±3.70% (p=0.0001), and 20.70±2.16% versus 23.70±3.67% (p=0.002), respectively (Central illustration E).
3. The mean percentage changes in 2C GLS, APLAX GLS, 4C GLS, and average GLS at 3 months were 10.41% versus 14.34% (p=0.429), 17.37% versus 5.60% (p=0.014), 12.71% versus 16.67% (p=0.465), and 15.83% versus 15.77% (p=0.991), respectively.
4. The mean percentage change in RV GLS at 3 months was 19.37% versus 12.80% (p=0.246), for the surgery and device groups, respectively.

5. The percentage of patients with LV GLS greater than -18% at 3 months were 23.80% versus 9.09% (p=0.010), while the percentage with RV GLS greater than -20% at 3 months were 38.09% versus 4.54% (p=0.001), for the surgery and device groups, respectively.

Correlation analysis showed that the mean LV GLS and RV GLS significantly and negatively correlated with the ASD size at all timepoints. The r-values for the mean LV GLS at baseline, 24 hrs, 1 month, and 3 months were -0.309 (p<0.05), -0.418 (p<0.01), -0.409 (p<0.01), and -0.328 (<0.05), respectively, while the r-values for RV GLS were -0.499, -0.536, -0.516, -0.455 (all p<0.01), suggesting that a larger defect size correlated with impaired biventricular strain values.

Correlation analysis also showed that the mean LV GLS negatively correlated with age with non-significant results (r=0.323; p=0.035), whereas RV GLS positively correlated with age, meaning that the residual dysfunction (RV GLS greater than -20%) patients were of higher age, and these were significant results (r=-0.191; p=0.221).

SIGNIFICANCE OF ASD SIZE AND CHANCES OF GLS IMPROVEMENT AT 3 MONTHS POST-ASD CLOSURE

The seven patients with persistent impairment of LV GLS at 3 months had a significantly larger ASD size (32.00±13.15 mm) as compared to the 36 patients whose GLS had normalised (22.11±7.38 mm; p<0.0001). Similar trends were seen in the 9 patients with residual impairment of RV GLS at 3 months (32.56±5.61 mm vs 21.38±8.48 mm; p<0.001). Hence, those with an ASD larger than 29 mm demonstrated impaired LV and RV GLS at 3 months despite successful closure either by device or surgery.

Table 2. Changes in 2C GLS, APLAX GLS, 4C GLS, avg. GLS, RV GLS at different timepoints post-ASD closure.

Parameter	Patients (n=43)	p-value
2C GLS		
2C GLS at baseline, %	16.95±3.68	
2C GLS at 24 hours, %	17.64±3.11	0.045
2C GLS at 1 month, %	18.13±2.75	0.001
2C GLS 3 months, %	19.07±2.54	0
Mean % change at 3 months	12.5	
APLAX GLS		
APLAX GLS at baseline, %	16.48±3.31	
APLAX GLS at 24 hours, %	16.55±2.42	0.856
APLAX GLS at 1 month, %	17.20±2.29	0.095
APLAX GLS at 3 months, %	18.30±2.15	0
Mean % change at 3 months	11.04	
4C GLS		
4C GLS at baseline, %	16.93±3.87	
4C GLS at 24 hours, %	17.33±3.03	0.154
4C GLS at 1 month, %	18.16±2.67	0.001
4C GLS at 3 months, %	19.42±2.66	0
Mean % change at 3 months	14.7	
Avg. GLS		
Avg. GLS at baseline, %	16.75±2.39	
Avg. GLS at 24 hours, %	17.28±2.46	0.004
Avg. GLS at 1 month, %	18.16±2.30	0
Avg. GLS at 3 months, %	19.40±2.05	0
Mean % change at 3 months	15.82	
Avg. LV GLS		
Avg. LV GLS greater than -18% at 3 months	7 (16.27)	
RV GLS		
RV GLS at baseline, %	19.22±3.59	
RV GLS at 24 hours, %	19.85±3.66	0.001
RV GLS at 1 month, %	20.70±3.63	0.0001
RV GLS at 3 months, %	22.23±3.36	0.0001
Mean % change at 3 months	15.66	
RV GLS greater than -20% at 3 months	9 (20.93)	

All values are mean±SD, %, or n (%). 2C: 2-chamber view; 4C: 4-chamber view; APLAX: apical long-axis view; ASD: atrial septal defect; avg.: average; GLS: global longitudinal strain; LV: left ventricular; RV: right ventricular; SD: standard deviation

An ASD size >29 mm had a sensitivity of 84.85% and 80.00%, specificity of 20% and 25%, positive predictive value of 77.78% and 82.35% and a negative predictive value of 28.57% and 22.22% in predicting LV GLS greater than -18% and RV GLS greater than -20%, respectively, at 3 months (**Table 4**).

ROC CURVE ANALYSIS

The receiver operating characteristic (ROC) curve for LV GLS showed an area under the curve (AUC) of 0.772, indicating there is a 77.2% chance that a randomly selected

Table 3. Changes in different LV and RV strain parameters in surgery versus device groups.

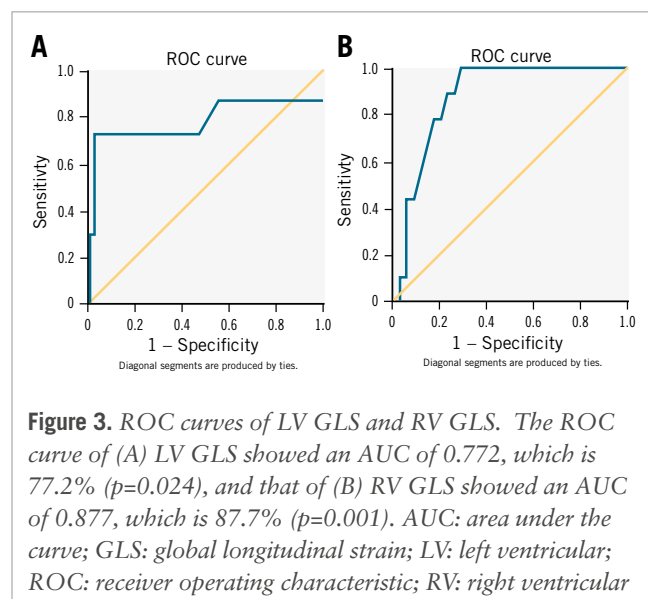
Parameter	Surgery (n=21)	Device (n=22)	p-value
2C GLS			
2C GLS at baseline	16.22±2.49	17.64±4.48	0.211
2C GLS at 24 hours	16.20±1.89	19.01±3.44	0.002
2C GLS at 1 month	16.79±1.78	19.39±2.93	0.001
2C GLS at 3 months	17.91±1.55	20.17±2.83	0.003
Mean % change at 3 months	10.41	14.34	0.429
APLAX GLS			
APLAX GLS at baseline	15.60±2.65	17.31±3.69	0.089
APLAX GLS at 24 hours	16.22±2.24	16.86±2.58	0.052
APLAX GLS at 1 month	16.97±2.08	17.42±2.49	0.021
APLAX GLS at 3 months	18.31±1.79	18.28±2.48	0.961
Mean % change at 3 months	17.37	5.6	0.014
4C GLS			
4C GLS at baseline	17.06±2.46	16.79±4.91	0.819
4C GLS at 24 hours	17.03±2.36	17.62±3.58	0.051
4C GLS at 1 month	17.71±2.34	18.59±2.92	0.021
4C GLS at 3 months	19.23±2.52	19.59±2.82	0.041
Mean % change at 3 months	12.71	16.67	0.465
Avg. GLS			
Avg. GLS at baseline	16.23±1.74	17.24±2.82	0.169
Avg. GLS at 24 hours	16.54±1.86	17.98±2.78	0.054
Avg. GLS at 1 month	17.34±1.79	18.92±2.49	0.023
Avg. GLS at 3 months	18.80±1.75	19.96±2.19	0.051
Mean % change at 3 months	15.83	15.77	0.991
Avg. LV GLS			
Avg. LV GLS greater than -18% at 3 months	5 (23.80)	2 (9.09)	0.01
RV GLS			
RV GLS at baseline	17.34±2.04	21.01±3.86	0.0001
RV GLS at 24 hours	17.83±2.37	21.78±3.66	0.0001
RV GLS at 1 month	18.73±2.29	22.58±3.70	0.0001
RV GLS at 3 months	20.70±2.16	23.70±3.67	0.002
Mean % change at 3 months	19.37	12.8	0.246
RV GLS greater than -20% at 3 months	8 (38.09)	1 (4.54)	0.001

All values are mean±SD, %, or n (%). 2C: 2-chamber view; 4C: 4-chamber view; APLAX: apical long-axis view; avg.: average; GLS: global longitudinal strain; LV: left ventricular; RV: right ventricular; SD: standard deviation

Table 4. Value of an atrial septal defect >29 mm in predicting impaired LV GLS and RV GLS at 3 months.

	LV GLS at 3 months	RV GLS at 3 months
Sensitivity	84.85%	80.00%
Specificity	20.00%	25.00%
Positive predictive value	77.78%	82.35%
Negative predictive value	28.57%	22.22%
Accuracy	69.77%	69.77%

patient with LV GLS greater than -18% will have a larger ASD size than a patient with LV GLS less than or equal to -18% (p -value=0.024). It confirms that ASD size is a reliable predictor of abnormal LV GLS values in this population. The ROC curve for RV GLS showed an AUC 0.877 (p =0.001). This means there is an 87.7% probability that a patient with RV GLS greater than -20% will have a larger ASD size than a patient with RV GLS less than or equal to -20% . The p -value of 0.001 signifies strong statistical significance, confirming that the relationship between ASD size and impaired RV GLS is not due to chance. Compared to the LV GLS analysis (AUC=0.772), the predictive value of ASD size is even stronger for RV GLS (**Figure 3, Central illustration B-C**).



Discussion

In this study of 43 patients (mean age 27.37 ± 16.37 years, range 3-56 years), 34.9% were males with secundum ASD (mean defect size 23.72 ± 9.15 mm). In these patients, who underwent device or surgical closure, impaired baseline LV and RV GLS was observed when compared with age- and sex-matched controls.

The 2C GLS was $16.95 \pm 3.68\%$ versus $20.73 \pm 1.78\%$ (p =0.0001), APLAX GLS was $16.48 \pm 3.31\%$ versus $20.90 \pm 1.63\%$ (p =0.0001), 4C GLS was $16.93 \pm 3.87\%$ versus $21.56 \pm 1.33\%$ (p =0.0001), and the average GLS was $16.75 \pm 2.39\%$ vs $21.31 \pm 1.16\%$ (p =0.0001). The RV GLS was also significantly lower in the patient group ($19.22 \pm 3.59\%$) compared to the control group ($24.27 \pm 2.96\%$; p =0.0001).

Impaired LV GLS (greater than -18%) was seen in 86.04%, while abnormal RV GLS greater than -20% was observed in 69.76% of patients with ASD at baseline (p =0.0001 vs controls). Such baseline abnormalities of LV and RV strain parameters are possibly secondary to the volume overload and interventricular dependence, which emphasises the systemic impact of left-to-right shunting and often correlates with shunt size and pulmonary pressures. Similar to our observations, previous studies have also reported impaired myocardial performance (using the Tei index) and GLS of not

only the RV but also of the LV, presumably due to septal flattening, interventricular dependence, and altered diastolic filling dynamics that compromise LV strain^{15,17,22}.

Following closure of an ASD, we observed a significant and progressive reduction in RVSP from a baseline of 41.37 mmHg to 35.63 mmHg at 24 hours (p =0.0001), with further sustained decreases at 1 month (mean 30.30 mmHg) and 3 months (mean 24.51 mmHg; p =0.0001).

IMPROVEMENTS IN LV AND RV STRAIN FOLLOWING ASD CLOSURE

We demonstrated significant reverse remodelling of both the RV and LV after transcatheter and surgical closure of ASD. All parameters of LV strain (2C, APLAX, 4C and average LV strain) and RV strain improved immediately (at 24 hours) and showed continued and sustained improvement at 1-3 months of follow-up. The mean 2C GLS improved from a baseline of $16.95 \pm 3.68\%$ to $17.64 \pm 3.11\%$ at 24 hours (p =0.045), $18.13 \pm 2.75\%$ at 1 month (p =0.001), and $19.07 \pm 2.54\%$ at 3 months (p =0.000). Although the APLAX GLS did improve, the values at baseline, 24 hours, and 1 month were not significantly different. However, at 3 months, the mean APLAX GLS was significantly higher ($18.30 \pm 2.15\%$; p =0.000) compared to the value at 1 month. The change in the mean 4C GLS from baseline to 24 hours was not significantly different while at 1 month ($18.16 \pm 2.67\%$; p =0.001) and at 3 months ($19.42 \pm 2.66\%$; p =0.000), the changes were significant.

The mean average GLS improved significantly at all measured time intervals ($17.28 \pm 2.46\%$ at 24 hours, $18.16 \pm 2.30\%$ at 1 month, and $19.40 \pm 2.05\%$ at 3 months; p =0.000). The mean RV GLS mirrored the changes in LV GLS with significant improvement at 24 hours, 1 month and 3 months ($19.85 \pm 3.66\%$, $20.70 \pm 3.63\%$, $22.23 \pm 3.36\%$; p =0.0001).

The mean percentage change in the average GLS at 3 months was 15.82%, while the percentage change in RV GLS was 15.66%, following ASD closure.

Biventricular volumetric and dimensional changes post-ASD closure have been studied with various technologies including conventional 2D echocardiography, tissue Doppler imaging and 3D echocardiography, STE and magnetic resonance imaging^{10-12,27-29}. Some studies have assessed ventricular function only at 24 hours, while others have done serial assessments at 24 hours, 1-6 months and 1 year following closure. These data highlight the role of strain analysis in detecting early myocardial recovery, even before changes in conventional echocardiographic parameters become evident. Usually, the changes in RV strain have been noted to be significant only in the RV lateral wall, as we also noted, while the strain parameters of the RV septum may not show as much change²⁰. Although some studies have documented a differential rate of recovery of strain in the ventricles, with either the LV improvement preceding that of the RV or vice versa, we observed a similar temporal profile of recovery in both the LV and RV^{12,15,16}.

PERSISTENT STRAIN ABNORMALITIES OF THE LV AND RV AT 3 MONTHS

The percentage of patients with abnormal LV GLS greater than -18% at 3 months was 16.27%, and 20.93% of patients

had impaired RV GLS greater than -20% . The mean average LV GLS and RV GLS significantly and negatively correlated with the ASD size at all timepoints. This trend suggested that a larger ASD size at baseline correlated with impaired biventricular strain values at 3 months, irrespective of the closure method.

All 7 patients with persistent impairment of LV GLS at 3 months and the 9 patients with residual impairment of RV GLS at 3 months had a significantly larger ASD size as compared with those whose strain had normalised.

The sensitivity, specificity, positive predictive value, and negative predictive value of an ASD size >29 mm predicting abnormal LV GLS (greater than -18% at 3 months) were 84.85%, 20.00%, 77.78%, and 28.57%, respectively. The corresponding values of ASD size >29 mm predicting persistently impaired RV GLS greater than -20% at 3 months were 80.00%, 25.00%, 82.35%, and 22.22%, respectively.

The long-term impairment of biventricular longitudinal strain following ASD closure may predispose patients to adverse outcomes such as atrial fibrillation, heart failure, or pulmonary arterial hypertension. Although the current literature lacks direct evidence linking reduced strain to these complications, the observed mechanical dysfunction raises concern. This potential association highlights a key area of interest for future studies aimed at understanding long-term ventricular mechanics and their role in predicting post-ASD closure morbidity.

DEVICE VERSUS SURGICAL CLOSURE: IMPACT ON STRAIN IMPROVEMENT

The choice of closure method – device versus surgery – has implications for the degree and timeline of myocardial recovery. We noted a more superior strain recovery of both the LV and RV in the patients undergoing device closure as compared to surgery. The average LV GLS at 24 hrs, 1 month, and 3 months in the device group was much higher than those undergoing surgery ($16.54 \pm 1.86\%$ vs $17.98 \pm 2.78\%$; $p=0.054$, $17.34 \pm 1.79\%$ vs $18.92 \pm 2.49\%$; $p=0.023$, and $18.80 \pm 1.75\%$ vs $19.96 \pm 2.19\%$; $p=0.051$).

Similarly, RV GLS at 24 hrs, 1 month, and 3 months was also higher in the device group ($17.83 \pm 2.37\%$ vs $21.78 \pm 3.66\%$; $p=0.0001$, $18.73 \pm 2.29\%$ vs $22.58 \pm 3.70\%$; $p=0.0001$, and $20.70 \pm 2.16\%$ vs $23.70 \pm 3.67\%$; $p=0.002$).

The percentage of patients with LV GLS greater than -18% at 3 months were 23.80% vs 9.09% ($p=0.010$), while the percentage with RV GLS greater than -20% at 3 months were 38.09% vs 4.54% ($p=0.001$), for the surgery and device groups, respectively.

These findings align with previous reports that have also shown that device closure is associated with faster and superior improvement in LV and RV strain parameters compared to surgical closure^{8,10,18}. This is postulated to be due to the minimally invasive nature of device closure and the vulnerability of the myocardium to the adverse effects of surgery, particularly perioperative conditioning and cardiopulmonary bypass^{6,30}.

Surgical closure of ASD can lead to RV free wall tethering due to pericardial or postsurgical adhesions. This mechanical restriction alters RV geometry and hampers its contractile function, especially in the longitudinal direction. As a result,

a reduction in RV longitudinal strain is commonly observed postoperatively. This phenomenon is attributed to impaired RV free wall motion caused by tethering, rather than intrinsic myocardial dysfunction. Hence, the assessment of RV strain after ASD closure must consider this mechanical impact to avoid misinterpretation of right ventricular performance.

Limitations

The study being a single-centre study with limited patient numbers is an obvious limitation. Another limitation is that we only evaluated RV free wall myocardial function and not the RV septal area (which may be perhaps less affected in such cases). A longer-term follow-up would also elucidate better if the residual strain abnormalities recover over time. Moreover, the hypothesis that cardiopulmonary bypass adversely affects myocardial function leading to inferior strain recovery in those undergoing surgery as compared to device closure needs validation.

Conclusions

The assessment of LV and RV strain parameters using echocardiography provides valuable insights into myocardial recovery following ASD closure. Early assessment of biventricular function with highly sensitive modalities like strain and strain rate imaging before closure can predict acute RV and LV remodelling and functional changes after closure. We observed baseline strain impairments in ASD patients, underscoring the impact of chronic volume overload on myocardial function. The reduction in tricuspid regurgitation velocity and RVSP immediately after closure that we noted further highlights the haemodynamic benefits of intervention, while sustained strain improvements over time reflect ongoing myocardial remodelling and recovery. We further found that device closure offers advantages over surgical closure in terms of superior strain improvement at midterm follow-up of 3 months, emphasising the importance of minimally invasive techniques in contemporary clinical practice. Future research should focus on long-term outcomes and the integration of strain analysis into routine care to optimise patient management and improve clinical outcomes.

Authors' affiliations

1. Department of Cardiology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, India; 2. Department of Cardiovascular and Thoracic Surgery, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, India; 3. Department of Cardiac Anesthesia, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, India

Conflict of interest statement

The authors have no conflicts of interest to declare.

References

1. Geva T, Martins JD, Wald RM. Atrial septal defects. *Lancet*. 2014;383:1921-32.
2. Van De Bruaene A, Buys R, Vanhees L, Delcroix M, Voigt JU, Budts W. Regional right ventricular deformation in patients with open and closed atrial septal defect. *Eur J Echocardiogr*. 2011;12:206-13.
3. Cuyper JA, Opić P, Menting ME, Utens EM, Witsenburg M, Helbing WA, van den Bosch AE, Ouhlous M, van Domburg RT, Meijboom FJ, Bogers AJ,

Roos-Hesselink JW. The unnatural history of an atrial septal defect: longitudinal 35 year follow up after surgical closure at young age. *Heart*. 2013;99:1346-52.

4. Kutty S, Hazeem AA, Brown K, Danford CJ, Worley SE, Delaney JW, Danford DA, Latson LA. Long-term (5- to 20-year) outcomes after transcatheter or surgical treatment of hemodynamically significant isolated secundum atrial septal defect. *Am J Cardiol*. 2012;109:1348-52.
5. de Boer JM, Kuipers IM, Klitsie LM, Blom NA, Ten Harkel AD. Decreased biventricular longitudinal strain shortly after congenital heart defect surgery. *Echocardiography*. 2017;34:446-52.
6. Klitsie LM, Roest AA, Blom NA, ten Harkel AD. Ventricular performance after surgery for a congenital heart defect as assessed using advanced echocardiography: from doppler flow to 3D echocardiography and speckle-tracking strain imaging. *Pediatr Cardiol*. 2014;35:3-15.
7. Thilén U, Persson S. Closure of atrial septal defect in the adult. Cardiac remodeling is an early event. *Int J Cardiol*. 2006;108:370-5.
8. Menting ME, van den Bosch AE, McGhie JS, Cuypers JA, Witsenburg M, Geleijnse ML, Helbing WA, Roos-Hesselink JW. Ventricular myocardial deformation in adults after early surgical repair of atrial septal defect. *Eur Heart J Cardiovasc Imaging*. 2015;16:549-57.
9. Monfredi O, Luckie M, Mirjafari H, Willard T, Buckley H, Griffiths L, Clarke B, Mahadevan VS. Percutaneous device closure of atrial septal defect results in very early and sustained changes of right and left heart function. *Int J Cardiol*. 2013;167:1578-84.
10. Di Salvo G, Drago M, Pacileo G, Carrozza M, Santoro G, Bigazzi MC, Caso P, Russo MG, Carminati M, Calabró R. Comparison of strain rate imaging for quantitative evaluation of regional left and right ventricular function after surgical versus percutaneous closure of atrial septal defect. *Am J Cardiol*. 2005;96:299-302.
11. Walker RE, Moran AM, Gauvreau K, Colan SD. Evidence of adverse ventricular interdependence in patients with atrial septal defects. *Am J Cardiol*. 2004;93:1374-7, A6.
12. Agha HM, Mohammed IS, Hassan HA, Abu Seif HS, Abu Farag IM. Left and right ventricular speckle tracking study before and after percutaneous atrial septal defect closure in children. *J Saudi Heart Assoc*. 2020;32:71-8.
13. Suzuki M, Matsumoto K, Tanaka Y, Yamashita K, Shono A, Sumimoto K, Shibata N, Yokota S, Suto M, Dokuni K, Tanaka H, Otake H, Hirata KI. Preoperative coupling between right ventricle and pulmonary vasculature is an important determinant of residual symptoms after the closure of atrial septal defect. *Int J Cardiovasc Imaging*. 2021;37:2931-41.
14. Shaban GS, Kassem HK, El Setiha MES, Elsheikh RG, Shaban A, Saed IS. Two-dimensional Echocardiography in the Evaluation of Right Ventricular Systolic Function in Patients with Atrial Septal Defect before and after Closure. *Cardiology and Angiology: An International Journal*. 2022;11:308-22.
15. Wu ET, Akagi T, Taniguchi M, Maruo T, Sakuragi S, Otsuki S, Okamoto Y, Sano S. Differences in right and left ventricular remodeling after transcatheter closure of atrial septal defect among adults. *Catheter Cardiovasc Interv*. 2007;69:866-71.
16. van der Ven JPG, van den Bosch E, Kamphuis VP, Terol C, Gnanam D, Bogers AJJC, Breur JMPJ, Berger RME, Blom NA, Koopman L, Ten Harkel ADJ, Helbing WA. Functional Echocardiographic and Serum Biomarker Changes Following Surgical and Percutaneous Atrial Septal Defect Closure in Children. *J Am Heart Assoc*. 2022;11:e024072.
17. Dragulescu A, Grosse-Wortmann L, Redington A, Friedberg MK, Mertens L. Differential effect of right ventricular dilatation on myocardial deformation in patients with atrial septal defects and patients after tetralogy of Fallot repair. *Int J Cardiol*. 2013;168:803-10.
18. Castaldi B, Vida VL, Argiolas A, Maschietto N, Cerutti A, Gregori D, Stellin G, Milanesi O. Late Electrical and Mechanical Remodeling After Atrial Septal Defect Closure in Children: Surgical Versus Percutaneous Approach. *Ann Thorac Surg*. 2015;100:181-6.
19. Samiei N, Bayat F, Moradi M, Parsaei M, Haghighi SZ, Mohebbi A, Hamzepour N, Noohi F. Comparison of the response of the right ventricle with endovascular occlusion and surgical closure in adults with atrial septal defect one year after intervention. *Clin Med Insights Cardiol*. 2010;4:143-7.
20. Kumar P, Sarkar A, Kar SK. Assessment of ventricular function in patients of atrial septal defect by strain imaging before and after correction. *Ann Card Anaesth*. 2019;22:41-6.
21. Alkhateeb A, Roushdy A, Hasan-Ali H, Kishk YT, Hassan AKM. The changes in biventricular remodelling and function after atrial septal defect device closure and its relation to age of closure. *Egypt Heart J*. 2020;72:85.
22. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, Flachskampf FA, Foster E, Goldstein SA, Kuznetsova T, Lancellotti P, Muraru D, Picard MH, Rietzschel ER, Rudski L, Spencer KT, Tsang W, Voigt JU. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *Eur Heart J Cardiovasc Imaging*. 2015;16:233-70.
23. Voigt JU, Pedrizzetti G, Lysyansky P, Marwick TH, Houle H, Baumann R, Pedri S, Ito Y, Abe Y, Metz S, Song JH, Hamilton J, Sengupta PP, Kolias TJ, d'Hooge J, Aurigemma GP, Thomas JD, Badano LP. Definitions for a common standard for 2D speckle tracking echocardiography: consensus document of the EACVI/ASE/Industry Task Force to standardize deformation imaging. *Eur Heart J Cardiovasc Imaging*. 2015;16:1-11.
24. Yang H, Wright L, Negishi T, Negishi K, Liu J, Marwick TH. Research to Practice: Assessment of Left Ventricular Global Longitudinal Strain for Surveillance of Cancer Chemotherapeutic-Related Cardiac Dysfunction. *JACC Cardiovasc Imaging*. 2018;11:196-201.
25. Muraru D, Onciul S, Peluso D, Soriani N, Cucchini U, Aruta P, Romeo G, Cavalli G, Iliceto S, Badano LP. Sex- and Method-Specific Reference Values for Right Ventricular Strain by 2-Dimensional Speckle-Tracking Echocardiography. *Circ Cardiovasc Imaging*. 2016;9:e003866.
26. Wang TKM, Grimm RA, Rodriguez LL, Collier P, Griffin BP, Popović ZB. Defining the reference range for right ventricular systolic strain by echocardiography in healthy subjects: A meta-analysis. *PLoS One*. 2021;16:e0256547.
27. Weber M, Dill T, Deetjen A, Neumann T, Ekinic O, Hansel J, Elsaesser A, Mitrovic V, Hamm C. Left ventricular adaptation after atrial septal defect closure assessed by increased concentrations of N-terminal pro-brain natriuretic peptide and cardiac magnetic resonance imaging in adult patients. *Heart*. 2006;92:671-5.
28. Burgstahler C, Wöhrle J, Kochs M, Nusser T, Löffler C, Kunze M, Höher M, Gawaz MP, Hombach V, Merkle N. Magnetic resonance imaging to assess acute changes in atrial and ventricular parameters after transcatheter closure of atrial septal defects. *J Magn Reson Imaging*. 2007;25:1136-40.
29. Gao CH, Zhang H, Chen XJ. The impacts of transcatheter occlusion for congenital atrial septal defect on left ventricular systolic synchronicity: a three-dimensional echocardiography study. *Echocardiography*. 2010;27:324-8.
30. Reddy S, Bernstein D. The vulnerable right ventricle. *Curr Opin Pediatr*. 2015;27:563-8.