

Original Article



Long-Term Left Atrial Function after Device Closure and Surgical Closure in Adult Patients with Atrial Septal Defect

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OPEN ACCESS

Received: Aug 17, 2020

Revised: Oct 28, 2020

Accepted: Nov 2, 2020

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ABSTRACT

BACKGROUND: Studies comparing left atrial (LA) function after surgical closure or percutaneous closure in patients with an atrial septal defect (ASD) are lacking.

METHODS: Between 1 and 3 years after ASD treatment, we retrospectively analyzed the medical records and transthoracic echocardiographic images of patients who had been diagnosed with an ASD after 20 years of age and who had undergone surgical closure (ASD-S) or percutaneous device closure (ASD-D). We measured LA peak systolic, early diastolic, and late diastolic strain values using 2-dimensional (2D) speckle tracking echocardiography (STE) and calculated reservoir, conduit, and contraction strain.

RESULTS: The reservoir strain value of the ASD-D groups was $25.2\% \pm 7.4\%$, which was lower compared to the control group ($33.6\% \pm 5.5\%$) ($p = 0.004$). The LA conduit strain and the LA contraction values of the ASD-D group were also lower compared to the control group ($-13.8\% \pm 5.8\%$ vs. $-20.4\% \pm 4.7\%$, $p = 0.034$; $-11.3\% \pm 4.2\%$ vs. $-13.2\% \pm 2.5\%$, $p = 0.037$, respectively). The reservoir, conduit, and contraction strains of the ASD-S group were $27.8\% \pm 8.8\%$, $-15.3\% \pm 6.4\%$, and $-12.5\% \pm 5.8\%$, respectively, and were not different from those of the control group or the ASD-D group.

CONCLUSIONS: The 2D STE is a suitable method for evaluating LA function after ASD closure. Our results demonstrate that 1 year after device closure, the LA reservoir, conduit and contraction function were reduced in ASD-D group compared to healthy controls, while there was no difference between the ASD-S and ASD-D groups.

Keywords: Left atrial function; Strains; Atrial septal defect; Devices; Surgery

INTRODUCTION

An atrial septal defect (ASD) is one of common forms of congenital heart disease in adults.¹ ASD is treatable with surgery or percutaneous intervention, and patient outcomes after both surgical and interventional closure of ASD are excellent.^{2,3} Of note, percutaneous device closure of ASD is considered first line because of some advantages of the procedure over surgery, including a low rate of complications, short anesthesia time, and short admission duration.^{4,5}

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Funding

This study was supported by a grant from Korean Society of Echocardiography in 2017.

Conflict of Interest

The authors have no financial conflicts of interest.

Author Contributions

Conceptualization: Seo JS; Data curation: Seo JS, Park YA, Wi JH, Jin HY, Han IY, Jang JS, Yang TH, Kim DK, Kim DS; Formal analysis: Seo JS; Funding acquisition: Seo JS; Investigation: Seo JS; Methodology: Seo JS; Project administration: Seo JS; Resources: Seo JS; Software: Seo JS; Supervision: Seo JS; Validation: Seo JS; Visualization: Seo JS; Writing - original draft: Seo JS; Writing - review & editing: Seo JS.

Previous studies have raised concerns that electrophysiological disorders of the atrial septum may arise from the ASD itself or from surgical correction of the defect.⁶⁻⁸⁾ There are also reports that atrial stiffness may increase after percutaneous device or surgical closure of ASD, affecting functional changes in the left atrial (LA) and related outcomes. However, when clinicians choose how to close an ASD (surgical or interventional treatment), they typically don't take into account potential changes in LA function after ASD closure. Studies comparing LA function after surgical or percutaneous device closure of ASD are still lacking, and most are only short-term studies lasting from 6 months to 1 year.

Measuring either LA volume or 2-dimensional (2D) strain are used to evaluate LA function. Both methods have proven to be reliable for evaluating LA function in various cardiac diseases, and both can evaluate phasic LA function, such as LA reservoir function, conduit function, and contraction function.⁹⁻¹¹⁾

Therefore, we used volumetric measuring and 2D speckle tracking echocardiography (STE) to compare LA function more than 1 year after treatment in adult ASD patients who had undergone either surgical or percutaneous device closure.

METHODS

Study design

Between 1 and 3 years after ASD treatment, we retrospectively analyzed medical records and transthoracic echocardiographic images of patients who had been diagnosed with ASD after 20 years of age and who had undergone surgical or percutaneous treatment. Patients with a residual shunt after closure, significant mitral valve disease, previous mitral valve surgery, atrial fibrillation (paroxysmal or persistent), less than 50% of left ventricular ejection fraction (LVEF), or in whom we were unable to analyze LA phasic volume or strain were excluded. In order to compare the LA function of patients who underwent surgical closure (ASD-S) and percutaneous device closure (ASD-D) with a normal control group, individuals without known underlying disease were included from among those who had undergone a routine health checkup at the Inje University Busan Paik Hospital. The control group was selected at a ratio of 3:1 to match the gender and age of ASD patients.

We reviewed the patient's electronic medical records and collected the following variables: phasic LA volume, LA longitudinal strain, demographic parameters (height, weight, blood pressure, pulse rate, underlying medical history, and social history), treatment method (surgical or percutaneous closure), surgical method (direct closure or patch closure), percutaneous device type and size, and treatment date.

The study conformed to the principles of the Declaration of Helsinki and was approved by the Institutional Review Board of Inje University Busan Paik Hospital. The institutional review board waived the need for informed consent due to the retrospective, observational nature of the present study.

Echocardiographic assessment of LA function

Echocardiography was performed using GE Vivid 7, E9 and E95 (GE Vingmed Ultrasound, Horten, Norway) ultrasound machines. Apical 4- and 2-chamber images were acquired using an M5S probe (2–4 MHz).

LA function was evaluated using volumetric measuring and 2D STE. The LA volume was measured using the biplane method of disks, according to American Society of Echocardiography guidelines.¹²⁾ The maximal LA volume was measured at end-systole, on the frame just prior to mitral valve opening, by tracing the LA inner border, and excluding the area under the mitral valve annulus, the LA appendage and the inlet of the pulmonary veins. The minimal LA volume and pre-A LA volume were measured in the same manner at end-diastole and just before the A wave, respectively. Reservoir function, conduit function, and pump function were calculated using the measured values. The formulas used are as follows.⁷⁾

$$\text{LA total emptying volume} = V_{\text{max}} - V_{\text{min}}$$

$$\text{LA total emptying fraction} = (V_{\text{max}} - V_{\text{min}})/V_{\text{max}}$$

$$\text{LA passive emptying volume} = V_{\text{max}} - V_{\text{pre-A}}$$

$$\text{LA passive emptying fraction} = (V_{\text{max}} - V_{\text{pre-A}})/V_{\text{max}}$$

$$\text{LA active emptying volume} = V_{\text{pre-A}} - V_{\text{min}}$$

$$\text{LA active emptying fraction} = (V_{\text{pre-A}} - V_{\text{min}})/V_{\text{pre-A}}$$

LA longitudinal strain was measured using a 2D STE. Images for LA strain analysis were acquired with a high-depth, narrow sector comprising only the LA to increase the frame rate, and foreshortening was avoided as much as possible. We analyzed the lateral and septal wall from the apical 4-chamber view, and the anterior and inferior wall from the apical 2-chamber view. Frame rates of 50 to 90 Hz were used. In the strain curve, the peak systolic strain, early diastolic strain, and late diastolic strain were measured, and phasic strain values were calculated using the following equations: reservoir strain=peak value of the strain curve-strain value at end-diastole; conduit strain=strain value at the onset of atrial contraction-peak value of the strain curve; pump strain=strain value at end-diastole-value at the onset of atrial contraction (**Figure 1**).¹¹⁾¹³⁾¹⁴⁾

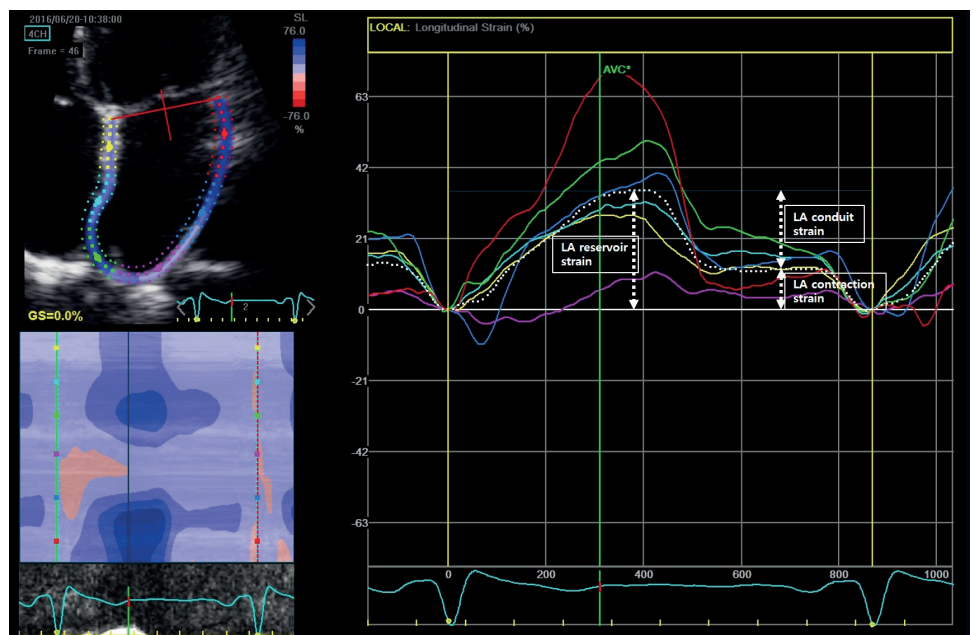


Figure 1. Strain curve of atrial myocardial longitudinal deformation.

Statistical analysis

All analyses were performed using IBM SPSS Statistics Software version 25.0 (IBM Corporation, Armonk, NY, USA). Continuous variables are expressed as mean \pm standard deviation. The Kruskal-Wallis test was used to compare the mean values of each group. To compare LA function among 3 groups, an analysis of covariance was performed after rank transformation to correct for baseline differences. The Bonferroni correction method was used for *post hoc* analysis. Categorical data are shown as frequencies and percentages, and were compared with the χ^2 test. Interobserver and intraobserver variabilities were evaluated by the intraclass correlation coefficient (ICC) with 95% limits of agreement (LOA). For all analyses, statistical significance was considered to be reached at a p-value of < 0.05 .

RESULTS

The ASD-S group included 42 patients, the ASD-D group included 20 patients, and the control group included 22 healthy controls.

The mean age at the time of ASD closure was 45.5 ± 11.6 years and 51.3 ± 11.0 years, respectively, in the ASD-S and ASD-D groups, which was not significantly different ($p = 0.066$). Thirty-three of the patients who underwent surgical closure had a patch. Of the patients who underwent percutaneous device closure, an Amplatzer device was used for 18, and a Figulla Flex II device was used for 2. The average size of the implanted device was 17.7 ± 5.7 mm. The size of the ASD was larger in the ASD-S group than in the ASD-D group (25.0 ± 12.5 mm vs. 15.4 ± 5.2 mm, respectively; $p < 0.001$).

Table 1 shows the clinical characteristics of each group at the transthoracic echocardiography follow-up time. The time from ASD closure to transthoracic echocardiography was 495.3 days in the ASD-S group and 370.5 days in the ASD-D group ($p = 0.140$). The heart rate of the ASD-S and ASD-D groups were 74.0 ± 14.0 beats per minute and 72.4 ± 14.4 beats per minute, respectively, which were higher than the control heart rate of 61.0 ± 11.9 beats per minute ($p = 0.002$). Patients with hypertension were significantly more frequent in ASD-D group than in control group ($p = 0.009$), but there was no difference between the 3 groups in prevalence of diabetes mellitus, ischemic heart disease, chronic kidney disease and valvular heart disease. There were no differences in LVEF, peak mitral E and A velocity, septal e' and a' velocity, and lateral e' and a' velocity between the 3 groups. The end-diastolic and end-systolic RV areas were larger in the ASD-S and ASD-D groups than in the control group (18.0 ± 3.4 , 18.7 ± 5.7 and 15.9 ± 3.4 , $p = 0.035$; 11.4 ± 2.8 , 11.6 ± 3.7 and 9.6 ± 3.2 , $p = 0.029$). However, there was no difference in fractional area change (FAC) between the 3 groups ($p = 0.565$). The tricuspid S' of the ASD-S group was 9.4 ± 2.5 cm/sec, which was significantly lower than that of the ASD-D and control groups (13.9 ± 2.2 cm/sec and 12.7 ± 1.8 cm/sec, respectively; $p < 0.001$). At 17.6 ± 2.8 mm, the tricuspid annular plane systolic excursion was also significantly lower than in the ASD-S group when compared to the ASD-D and control groups (23.6 ± 3.6 mm and 22.0 ± 3.3 mm, respectively; $p < 0.001$).

Table 2 presents the LA structure and function of the 3 groups, as assessed by volumetric measuring. There was no difference in LA maximal volume, minimal volume, or pre-A volume between the 3 groups. Moreover, there was no difference in the calculated total emptying volume, passive emptying volume and active emptying volume between the 3 groups.

Table 1. Clinical and conventional echocardiographic characteristics

Characteristics	Control (n = 22)	ASD-S (n = 42)	ASD-D (n = 20)	p-value
Age at TTE follow-up	47.6 ± 12.7	46.9 ± 11.5	52.4 ± 10.7	0.217
Male	8 (36.4)	13 (31.0)	8 (40.0)	0.794
Time after ASD closure to TTE (days)	NA	495.3 ± 310.2	370.5 ± 300.8	0.134
Body mass index (kg/m ²)	22.7 ± 2.3	24.0 ± 6.6	26.6 ± 5.7	0.051
Body surface area (m ²)	1.7 ± 0.1	1.7 ± 0.2	1.8 ± 0.3	0.200
Systolic blood pressure (mmHg)	122.5 ± 12.8	120.9 ± 17.4	125.6 ± 19.4	0.612
Diastolic blood pressure (mmHg)	74.5 ± 8.6	71.0 ± 11.0	73.2 ± 13.1	0.467
Heart rate, beats per minute	61.0 ± 11.9 ^{††}	74.0 ± 14.0 [†]	72.4 ± 14.4 [†]	0.002
Hypertension	0 (0) [†]	4 (9.5)	6 (30.0) [†]	0.009
Diabetes mellitus	0 (0)	4 (9.5)	2 (10.0)	0.355
Ischemic heart disease	0 (0)	0 (0)	0 (0)	NA
Chronic kidney disease	0 (0)	1 (2.4)	0 (0)	1.000
Valvular heart disease	0	1 (2.4)	0 (0)	1.000
LVEF	63.9 ± 4.5	63.9 ± 5.0	65.2 ± 3.6	0.578
Peak E velocity (cm/sec)	66.0 ± 15.0	68.7 ± 26.0	70.7 ± 19.7	0.761
Peak A velocity (cm/sec)	56.2 ± 12.5	60.9 ± 16.0	66.7 ± 15.6	0.084
Septal e' velocity (cm/sec)	8.9 ± 2.5	8.1 ± 2.3	7.6 ± 2.7	0.235
Septal a' velocity (cm/sec)	8.5 ± 1.5	8.6 ± 2.7	8.9 ± 2.1	0.840
Lateral e' velocity (cm/sec)	12.4 ± 3.0	12.2 ± 2.3	10.7 ± 3.4	0.103
Lateral a' velocity (cm/sec)	8.8 ± 1.8	9.2 ± 2.8	9.3 ± 2.4	0.790
RV area, end-diastole (cm ²)	15.9 ± 3.4 [*]	18.0 ± 3.4 [†]	18.7 ± 5.7	0.035
RV area, end-systole (cm ²)	9.6 ± 3.2 [*]	11.4 ± 2.8 [†]	11.6 ± 3.7	0.029
RV FAC (%)	40.2 ± 9.8	35.5 ± 16.2	38.3 ± 7.7	0.565
Tricuspid s' velocity (cm/sec)	12.7 ± 1.8 [*]	9.4 ± 2.5 ^{††}	13.9 ± 2.2 [*]	< 0.001
TAPSE (cm)	22.0 ± 3.3 [*]	17.6 ± 2.8 ^{††}	23.6 ± 3.6 [*]	< 0.001

Values are presented as mean ± standard deviation or number (%).

ASD: atrial septal defect, ASD-S: atrial septal defect after 20 years of age and who had undergone surgical closure, ASD-D: atrial septal defect after 20 years of age and who had percutaneous device closure, TTE: transthoracic echocardiography, LVEF: left ventricle, TAPSE: tricuspid annular plane systolic excursion, RV: right ventricle, FAC: fractional area change, NA: not available.

*p < 0.05 vs. ASD-S; †p < 0.05 vs. ASD-D; ††p < 0.05 vs. control.

Table 2. Effect of ASD closure on LA structure and function assessed by volumetric method

Variables	Control (n = 22)	ASD-S (n = 42)	ASD-D (n = 20)	p-value (ANCOVA)
LA maximal volume (mL)	42.8 ± 12.6	51.0 ± 22.3	59.1 ± 28.9	0.148
LA minimal volume (mL)	20.4 ± 7.7	27.6 ± 20.9	32.0 ± 25.0	0.107
LA pre-A volume (mL)	28.7 ± 10.1	35.5 ± 18.9	42.5 ± 24.3	0.061
Total emptying volume (mL)	22.4 ± 7.6	23.3 ± 9.3	27.2 ± 14.0	0.862
Total emptying fraction	0.5 ± 0.1	0.5 ± 0.1	0.5 ± 0.2	0.285
Passive emptying volume (mL)	14.1 ± 7.3	15.4 ± 6.9	18.2 ± 11.5	0.912
Passive emptying fraction	0.3 ± 0.1	0.3 ± 0.1	0.3 ± 0.1	0.393
Active emptying volume (mL)	8.4 ± 3.4	8.0 ± 5.8	9.5 ± 5.3	0.761
Active emptying fraction	0.3 ± 0.1	0.3 ± 0.2	0.3 ± 0.1	0.425

Values are presented as mean ± standard deviation.

LA: left atrial, ASD, atrial septal defect, ASD-S: atrial septal defect after 20 years of age and who had undergone surgical closure, ASD-D: atrial septal defect after 20 years of age and who had percutaneous device closure, ANCOVA: analysis of covariance.

Table 3 shows the LA longitudinal strain values as measured by 2D STE and the LA phasic function calculated using strain values. The LA reservoir strain value of the ASD-D group was 25.2% ± 7.4% which was significantly lower compared to the control group (33.6% ± 5.5%) (p = 0.004). The LA conduit strain and the LA contraction strain values of the ASD-D group were also lower compared to the control group (-13.8% ± 5.8% vs. -20.4% ± 4.7%, p = 0.034; -11.3% ± 4.2% vs. -13.2% ± 2.5%, p = 0.037, respectively). These differences were maintained in the apical 4-chamber, but there was no difference between the 3 groups in the apical 2-chamber.

Table 3. Effect of ASD closure on LA function assessed by 2-dimensional speckle tracking method

Variables	Control (n = 22)	ASD-S (n = 42)	ASD-D (n = 20)	p-value (ANCOVA)
LA reservoir strain (%)	33.6 ± 5.5*	27.8 ± 8.8	25.2 ± 7.4†	0.004
LA reservoir strain, 4-chamber	34.1 ± 7.1*	26.0 ± 7.6	22.9 ± 6.3†	0.001
LA reservoir strain, 2-chamber	33.1 ± 4.9*	29.7 ± 11.3	27.4 ± 9.5†	0.052
LA conduit strain (%)	-20.4 ± 4.7*	-15.3 ± 6.4	-13.8 ± 5.8†	0.034
LA conduit strain, 4-chamber	-21.7 ± 5.9*	-15.0 ± 6.0	-13.8 ± 5.4†	0.026
LA conduit strain, 2-chamber	-19.1 ± 4.5	-15.6 ± 7.6	-13.9 ± 7.1	0.066
LA contraction strain (%)	-13.2 ± 2.5*	-12.5 ± 5.8	-11.3 ± 4.2†	0.037
LA contraction strain, 4-chamber	-12.4 ± 3.7*	-11.0 ± 5.5	-9.1 ± 3.8†	0.006
LA contraction strain, 2-chamber	-14.0 ± 2.2	-14.1 ± 6.8	-13.6 ± 5.4	0.366

LA: left atrial; ASD-S: atrial septal defect after 20 years of age and who had undergone surgical closure, ASD-D: atrial septal defect after 20 years of age and who had percutaneous device closure, ANCOVA: analysis of covariance. *p < 0.05 vs. ASD-D; †p < 0.05 vs. control.

When the size of ASD was added as a covariate and corrected, there was no difference in reservoir, conduit and contraction function between ASD-S and ASD-D groups (p = 0.129, p = 0.071 and p = 0.869, respectively).

The ICC with 95% LOA for the interobserver and intraobserver variabilities are shown in **Table 4**. The intraobserver and interobserver ICC for LA reservoir strain were 0.965 (95% LOA, 0.918–0.985) and 0.977 (95% LOA, 0.945–0.990), respectively. The intraobserver and interobserver ICC for LA conduit strain were 0.963 (95% LOA, 0.914–0.984) and 0.975 (95% LOA, 0.941–0.989), respectively. For LA contraction strain, the intraobserver and interobserver ICC were 0.950 (95% LOA, 0.881–0.979) and 0.934 (95% LOA, 0.843–0.972), respectively.

DISCUSSION

In this study, we found that the reservoir function, conduit function and contraction function, as evaluated using 2D STE, were reduced in the ASD-D groups compared to the control group. These differences were derived from the functional differences of the apical 4-chamber. However, there was no difference in the *post hoc* analysis between the ASD-S and ASD-D groups. LA function, as evaluated using volumetric measuring, was not significantly different between the 3 groups.

It is well known that, in patients with an ASD, the defect itself can change the function of the LA. However, there have also been concerns about the mechanical and electrophysiological effects of the patches or devices used for treatment of ASD on LA function. Reduced LA function is known to be a predictor for the development of atrial arrhythmias and adverse

Table 4. Interobserver and intraobserver variability for 2-dimensional speckle tracking echocardiography-derived strain of left atrial

Variables	Interobserver			Intraobserver		
	ICC	95% LOA	p-value	ICC	95% LOA	p-value
Reservoir strain	0.977	0.945–0.990	< 0.001	0.965	0.918–0.985	< 0.001
Reservoir strain, 4-chamber	0.950	0.881–0.979	< 0.001	0.897	0.759–0.956	< 0.001
Reservoir strain, 2-chamber	0.968	0.925–0.986	< 0.001	0.948	0.877–0.978	< 0.001
Conduit strain	0.975	0.941–0.989	< 0.001	0.963	0.914–0.984	< 0.001
Conduit strain, 4-chamber	0.970	0.928–0.987	< 0.001	0.922	0.816–0.967	< 0.001
Conduit strain, 2-chamber	0.959	0.903–0.983	< 0.001	0.960	0.905–0.983	< 0.001
Contraction strain	0.934	0.843–0.972	< 0.001	0.950	0.881–0.979	< 0.001
Contraction strain, 4-chamber	0.912	0.792–0.963	< 0.001	0.867	0.686–0.943	< 0.001
Contraction strain, 2-chamber	0.856	0.661–0.939	< 0.001	0.889	0.739–0.953	< 0.001

ICC: intraclass correlation coefficient, LOA: limits of agreement.

outcomes associated with various morbid conditions.^{9,11,15,16)} Interest in changes of LA function after ASD treatment has increased recently, and the results of the few studies that have been reported have been inconsistent. Some possible reasons for these discrepancies include differences in the timing of evaluation of LA function after ASD treatment and differences in methods used for evaluation.

The remodeling of the LA after ASD treatment is most pronounced immediately after treatment, and it has been reported that remodeling terminates within 6 months following treatment.^{17,18)} Based on these data, more recent studies investigating changes in LA function after ASD treatment have been conducted immediately after or within 6 months of treatment. Boyd et al.¹⁹⁾ reported that the strain value of the LA septum measured using color Doppler tissue imaging 6 months after treatment in patients who underwent device closure was reduced compared to the control group. Aslan et al.²⁰⁾ evaluated LA phasic function before, 1, and 6 months after ASD percutaneous device closure in 41 patients using volumetric measuring. They reported no difference in reservoir function, improved conduit function, and decreased contraction function 1 and 6 months after treatment when compared to LA functioning before the procedure. Suzuki et al.²¹⁾ compared LA function before and after surgical or percutaneous device closure in a study of 43 children. In the surgical closure group, the strain value of the LA lateral wall did not change; however, in the device group, the strain value of the LA lateral wall immediately after the procedure was decreased, and the strain value was inversely correlated with the size of the device used.

However, the LA remodeling observed by previous researchers was only a structural change based on LA volume, and it is not clear whether functional remodeling continues after 6 months. In addition, the effect of ASD closure on LA function over longer periods time remains unclear, so 6 months may not be a sufficient amount of time for monitoring the LA function of patients who underwent ASD treatment. This can also be inferred from the fact that, although atrial arrhythmia most frequently occurs within 1 month after ASD closure, it can continue to occur more than 6 months after closure, and occurs more frequently with increasing age in this patient population than in healthy controls.²²⁾

In this study, the parameters evaluated to assess structural changes were not significantly different between the 3 groups. However, LA strain as assessed using 2D STE, demonstrated reduced LA reservoir, conduit and contraction function in patients who underwent ASD device closure when compared to healthy controls.

Few studies have directly compared the effects of surgical closure and percutaneous device closure on LA function. In 2005, Di Salvo et al.²³⁾ first studied LA function after surgical and percutaneous device closure. In this study, the LA function was evaluated using the tissue Doppler image-derived strain at least 6 months after treatment, and the authors found that the peak systolic strain was not preserved after successful surgical closure. They hypothesized that this was a result of interstitial fibrosis caused by surgical closure and changes in LA structure following treatment. On the other hand, after percutaneous device closure, LA reservoir function was not different from that of the normal control group. Conversely, Suzuki et al.²¹⁾ and Boyd et al.¹⁹⁾ reported that immediately after treatment, the reservoir function was preserved in the surgical group and decreased in the percutaneous device group compared to controls. This discrepancy may originate from differences in the length of the follow-up period after treatment between the 2 studies.

Compared to previous studies showing results either immediately after treatment or after approximately 6 months, we analyzed data from 1 to 3 years after treatment. The reason for the 1- to 3-year limit was to minimize the differences over the follow-up period. In this long-term follow-up, the LA reservoir function, conduit and contraction function of the ASD-D were impaired compared to the control group, but there was no difference in these functions between the ASD-D and ASD-S groups. Our results are very similar to those of Hajizeinali et al.,²⁴⁾ who analyzed data from 1 to 5 years after treatment. They reported reduced reservoir function in the device group, and reduced conduit function in both the device and surgery groups, compared to controls. Therefore, these data from long-term follow-up after ASD treatment suggest that percutaneous device closure have a negative effect on the reservoir function and conduit function of the LA.

Traditionally, LA function has been evaluated by measuring the size or volume of the LA using 2D echocardiography. Recently, strain values using tissue Doppler imaging or 2D STE have been used to detect early functional changes, before structural changes occur.¹¹⁾¹³⁾²⁵⁻²⁷⁾ Here, we evaluated LA function using volumetric measuring and STE. Both of these methods allow phasic function analysis according to the cardiac cycle. The 2D STE, but not volumetric measuring, was able to demonstrate the functional impairment of the LA in the ASD-D group compared to controls.

European Association of Cardiovascular Imaging and American Society of Echocardiography achieved consensus to standardize the LA deformation imaging using 2D STE.²⁸⁾ When analyzing 2D strain of LA, they suggest averaging the measurements of the anterior, inferior, lateral, and septal walls, but several previous studies have excluded the septum from the analysis because of its displacement due to the device or patch.²¹⁾²⁴⁾ We analyzed global strain without excluding the LA septum, because 2D STE has the ability to distinguish normal deformation from passive device motion without being affected by the global motion of the heart or tethering of the adjacent segments.²⁹⁾³⁰⁾

The present study has several limitations. First, due to the retrospective observational nature of the study, there is the potential for the existence of selection bias and differences in the baseline characteristics of the ASDs. Second, because we did not have the baseline data before treatments in patients with ASD we could not show the changes between pre-treatment and post-treatment. Therefore, it is not clear whether the device itself has degraded the LA function. Third, because our data do not include clinical outcomes, the clinical implications of impaired LA strain after ASD closure is unclear. Finally, the software we used to analyze LA strain was originally developed to analyze the strain of the left ventricle.

In conclusion, the 2D STE is a suitable method for evaluating LA function after ASD closure. One year after treatment, the LA reservoir function, conduit function and contraction function, which were evaluated using global longitudinal strain values, were reduced in patients who underwent device closure when compared to the control group, and there was no difference in these functions between the 2 treatment groups. These findings could provide a reasonable method for long-term atrial evaluation after ASD treatment, which could eventually help determine optimal treatment strategies.

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