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Acute Changes in Left Ventricular End Diastolic Pressure following the Transcatheter Closure of an Atrial Septal Defect in Adults

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ABSTRACT

Background: Acute changes in left ventricular diastolic function shortly after ASD closure in elderly patients have not been well known. We aimed to investigate acute changes in left ventricular end diastolic pressure (LVEDP) in elderly patients following transcatheter closure of atrial septal defect (ASD).

Methods: All 19 adults with ASDs who underwent transcatheter closure between June 2013 and December 2014 were enrolled. LVEDP was measured prior to device closure and compared with that immediately following device closure and 15 minutes after device closure.

Results: The median age of the patients was 48 years old. The baseline E/e' and LVEDP values were 8.3 ± 2.8 and 13 ± 3 mmHg. The LVEDP value immediately following closure was 19 ± 4 mmHg, and 15 minutes after closure was 16 ± 4 mmHg. The median increase in the LVEDP value immediately following closure was 6 mmHg, which significantly differed from that prior to closure. The LVEDP 15 minutes after closure decreased but remained significantly higher than the value observed immediately after closure. No significant changes were observed with regard to E/e' at either 1 day or 3 months following closure. The LVEDP value 15 minutes after device closure was significantly correlated with those observed before closure and immediately following closure; however, no significant correlations were observed with regard to patient age, Qp/Qs, E/e' before closure, or E/e' 3 months after device closure.

Conclusion: LVEDP in adults with ASDs significantly increases following device closure. LVEDP before closure predicts LVEDP following device closure.

INTRODUCTION

The transcatheter closure of atrial septal defect (ASD) is extremely popular, particularly among adults [Suarez De Lezo 2000; Eerola 2007]. However, the hemodynamics of elderly patients might differ from those of younger patients. The left ventricle becomes restrictive with age [Ewert 2001a; Hees 2004; Park 2007; Prasad 2007; Masutani 2012]. Several reports in the literature have discussed the consequences of pulmonary edema, which can develop immediately following the surgical closure of an atrial sepal defect (ASD) [Davies 1970; Beyer 1978]. Recently, similar phenomena have been observed among elderly patients; these findings might be because of the acute left ventricular dysfunction caused by ASD closure [Ewert 2001a; Ewert 2001b; Tomai 2002]. Although most of these patients recovered completely, their treatment courses were complicated [Ewert 2001a; Masutani 2009]. This study observed the acute changes in left ventricular end diastolic pressure (LVEDP) observed following device closure in adults.

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PATIENTS AND METHODS

All 19 adult patients with ASDs undergoing a transcatheter device closure performed by a single specialist from June 2013 to December 2014 were prospectively registered. The ASDs were hemodynamically significant, and none of the patients had high mean pulmonary arterial pressure. All ASDs were suitable for device closure, and none of the patients suffered from either coronary artery disease or atrial arrhythmia. We categorized the high-risk group of patients as those with increased LVEDP values immediately after closure (≥10 mmHg) or those with high LVEDP values 15 minutes after closure (≥20 mmHg). Patients' medical records, including their catheterization records, were reviewed. Institutional review board approved this study and waived the need for consent from patients or parents.

Cardiac Catheterization

Cardiac catheterization was performed under local anesthesia using subcutaneous lidocaine infiltration. Pulmonary arterial pressure was measured to exclude pulmonary hypertension, and the amount of shunting (Qp/Qs) was determined using Fick's method. A pigtail catheter was advanced to the left ventricle, and LVEDP was measured before device closure. Without using a balloon-sizing technique, the ASD was closed using an appropriately sized device via intra-cardiac echocardiography, and the absence of any significant residual shunting was subsequently confirmed. LVEDP was measured both immediately and 15 minutes after device closure.

Echocardiography

One day prior to the procedure, a single sonographer performed transthoracic echocardiography (TTE), and the

trans-mitral inflow pattern was measured at the tip of the mitral valve. Pulsed tissue Doppler imaging was performed on the septal mitral annulus using the apical four-chamber view. The same sonographer performed similar echocardiographic evaluations on the next day and at three months following the procedure to exclude cardiac complications and assess the resulting left ventricular diastolic changes.

Statistical Analysis

Data were expressed either as mean ± SD or as medians (ranges). Paired comparisons were analyzed using the Wilcoxon signed-rank test. Spearman's rho was obtained via a nonparametric correlation analysis. All analyses were performed using SPSS 21.0.

RESULTS

Device closure was performed successfully in all patients. No specific pre-procedural events affected the procedure. All patients had uneventful treatment courses following device closure and were discharged 2 days later. Aspirin was prescribed for all patients. Nineteen patients were enrolled in our study (14 females; 5 males). The patients' median age was 48 (32-72) years old.

The median ASD sizes were 15.5 (9.0-26.0) mm according to TTE and 17.0 (9.0-25.0) mm according to intra-cardiac echocardiography (ICE). The Qp/Qs was 2.5 ± 0.8, and the N terminal pro-brain natriuretic peptide (BNP) level was 87.5 ± 75.2 pmol/L. Neither moderate nor severe mitral regurgitation was noted, and only mild aortic regurgitation was observed using 2-dimensional echocardiography. The E/e' associated with the left ventricular diastolic function of 16 patients (8.3 ± 2.8; 4.3-16.0) was recorded before the procedure. However, an abnormally high E/e' (>12) was noted in one patient. The mean LVEDP value before device closure was 13 ± 3 mmHg, and abnormal LVEDP values (>12 mmHg) were observed in 9 patients (47.4%).

Device closure was performed using 18 Amplatzer septal occluders and 1 Figullar septal occluder. The median device size was 20 (12-30) mm.

The LVEDP immediately following closure was 19 ± 4 mmHg and 15 minutes following closure was 16 ± 4 mmHg (Figure 1, Table 1). Only three patients (15.8%) had normal LVEDP values (≤12 mmHg) 15 minutes after closure. The median increase in LVEDP immediately following closure was 6 mmHg (1-12 mmHg), which was significantly increased compared with the LVEDP value measured before closure ($P < .001$). The LVEDP value 15 minutes after closure was significantly higher than that measured before closure but lower than that measured immediately after closure ($P < .001$). The median increase in LVEDP 15 minutes after closure from baseline was 3 mmHg. No significant changes

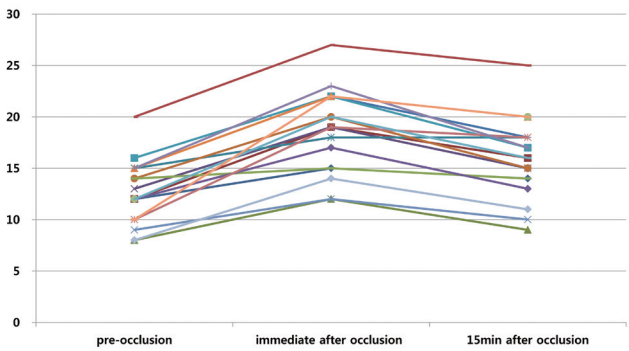

Figure 1. The changes in LVEDP following ASD closure were characterized by an acute increase in LDEVP immediately following occlusion and a gradual decrease 15 minutes after occlusion; however, the latter value remained elevated compared with that taken before occlusion.

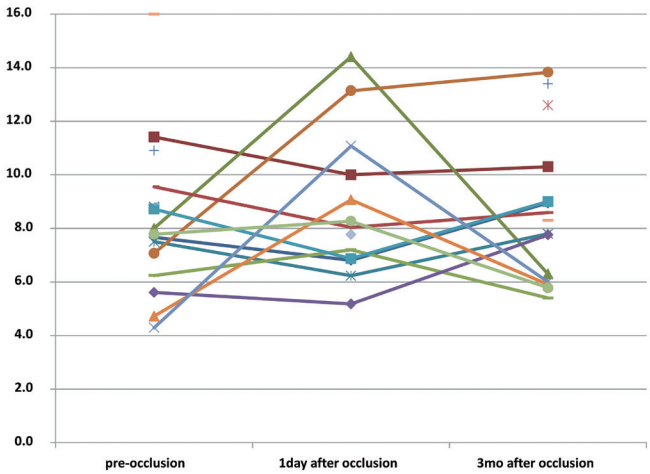

Figure 2. The changes in E/e' following ASD closure did not increase significantly for every patient.

Table 1. Changes of LVEDP and E/e' after ASD Occlusion

	Pre-occlusion	0 min after occlusion	15 min after occlusion	1 day after occlusion	3 mo after occlusion	*P*
LVEDP	13 ± 3	19 ± 4	16 ± 4			<.001
E/e'	8.3 ± 2.8			8.8 ± 2.7	8.7 ± 2.8	>.05
NT Pro-BNP (pg/mL)	87.5 ± 75.2					

LVEDP indicates left ventricular end diastolic pressure; NT Pro-BNP, N terminal pro-brain natriuretic peptide.

Table 2. Comparison between High-Risk Group and Control

	High-risk group	Control	*P*
Patients	8	10	
Age, y	52.6 ± 11.6	45.8 ± 9.5	.213
Device size, mm	20.3 ± 3.1	20.0 ± 6.4	.315
Qp/Qs	2.6 ± 0.5	2.3 ± 1.0	.122
Pro-BNP pre	92.9 ± 75.5	84.4 ± 82.6	.762
LVEDP pre, mmHg	14.6 ± 2.9	11.3 ± 2.5	.021
LVEDP immediate, mmHg	22.3 ± 2.2	16.0 ± 2.8	<.001
Increase of LVEDP immediate, mmHg	7.6 ± 1.9	4.7 ± 2.4	.012
LVEDP 15 minutes, mmHg	18.5 ± 3.2	13.8 ± 3.1	.009
E/e' pre	9.4 ± 3.5	7.4 ± 2.2	.232
E/e' 1 day	9.3 ± 2.7	8.6 ± 3.0	.570
E/e' 3 month	9.8 ± 3.1	8.1 ± 2.4	.282

Pro-BNP indicates pro-brain natriuretic peptide; LVEDP, left ventricular end diastolic pressure.

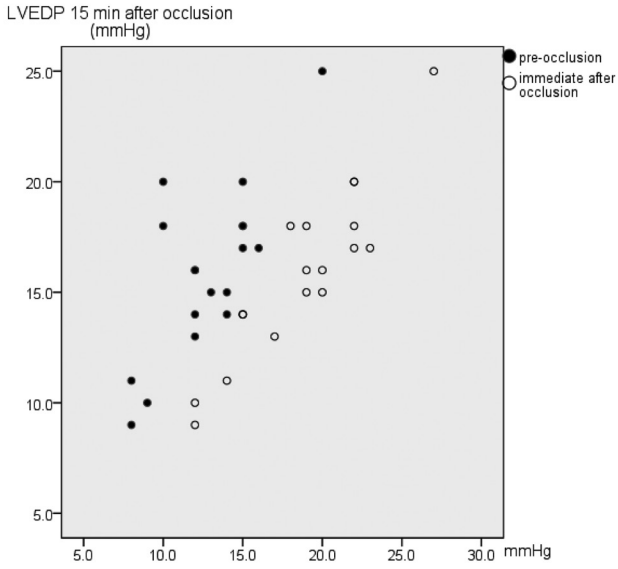

Figure 3. LVEDP at 15 minutes following ASD closure was significantly correlated with LVEDP before occlusion and immediately following occlusion.

in E/e' were observed at either 1 day or 3 months following closure (8.8 ± 2.7 and 8.7 ± 2.8; Figure 2, Table 1).

The LVEDP measured 15 minutes after device closure was significantly correlated with the LVEDP values measured before closure and immediately after closure (Spearman's rhos = 0.545 and 0.843; ps = 0.016 and <0.001, respectively; Figure 3); however, no significant correlations were observed with regard to age, Qp/Qs, E/e' before closure, or E/e' 3 months after device closure ($P > .05$).

The high-risk group consisted of 8 patients, including 1 patient with significantly increased LVEDP immediately following closure and 8 patients with elevated LVEDP 15 minutes after closure. These high-risk patients exhibited significantly higher LVEDP both before closure and immediately following closure. However, age, Qp/Qs, defect size, and E/e' did not significantly differ between groups ($P > .05$; Table 2).

DISCUSSION

This observational study described acute changes in both LVEDP and acute left ventricular remodeling following ASD device closure. These findings were similar to those of previous studies; to the best of our knowledge, however, LVEDP immediately following device closure has not been assessed previously [Ermis 2015].

Restrictive left ventricles are more common among elderly patients and can cause left ventricular failure following ASD closure in this population [Park 2007; Prasad 2007]. Although it is difficult to identify restrictive left ventricles, Chen et al described a significant increase in left ventricular stiffness associated with aging [Chen 1998]. Left ventricle volume loading is decreased under ASD because of the left-to-right shunt across the ASD; closure of the ASD causes acute left ventricle volume loading. If left ventricular diastolic dysfunction exists, then the left ventricle cannot cope with the resulting volume overload, causing marked increases in LVEDP and pulmonary congestion. However, ASDs might mask restrictive left ventricles in elderly patients, and surgical closure might cause serious complications during the post-operative period [Davies 1970; Ewert 2001b]. Similar findings have been observed following transcatheter ASD closure in elderly patients; however, surgical closure can cause more serious adverse effects because of the use of a bypass pump. However, these effects do not occur in every elderly patient. The recovery of conventional left ventricular anatomy following ASD closure enables the left ventricle to adjust to increases in LVEDP despite the underlying left ventricular diastolic dysfunction [Booth 1988; Walker 2004; Pascotto 2005; Pascotto 2006]. Conditioning therapy administered 14-72 hours prior to ASD closure is reportedly helpful for high-risk patients [Ewert 2001a; Schubert 2005]. There have been several reports of ASD closure using fenestrated devices among patients with restrictive LVs [Holzer 2005; Kenny 2011]. However, the risk factors for left ventricular failure following ASD closure, regardless of the method used, have not been identified. Prophylactic left ventricular conditioning prior to ASD closure is recommended for patients with high left ventricular filling pressure and those exhibiting increases in LVEDP or left atrial pressure of more than 10 mmHg during the balloon occlusion of the ASD [Ewert 2000; Ewert 2001a; Schubert 2005]. Swan et al proposed that a patient age of older than 60 years is an additional indication [Swan 2006].

Previous authors have emphasized the effect of age on ventricular diastolic dysfunction following transcatheter ASD closure and have argued that ASD closure should occur at a younger age [Masutani 2009; Masutani 2012]. Left ventricular diastolic dysfunction is often associated with age [Prasad

2007]. However, Ermis et al described significant LVEDP changes following an ASD balloon occlusion that did not correlate with age, body mass index, or Qp/Qs [Ermis 2015]. Based on the results of the echocardiographic evaluations performed before and after ASD closure, E/e' increased, and e' decreased significantly, whereas the myocardial performance index did not change [Masutani 2009; Masutani 2012; Ermis 2015]. Satoshi et al observed left ventricular failure following device closure in two patients with low early diastolic mitral annular velocities and noted higher BNPs following closure, which were correlated with both e' and age [Masutani 2009]. Therefore, tissue Doppler imaging might help predict left ventricular dysfunction after device closure.

This study observed a hyper-acute increase in LVEDP immediately following ASD closure, followed by a gradual decrease. However, the LVEDP measured 15 minutes after device closure remained elevated compared with that before closure. Ermis et al observed similar findings but did not mention when LVEDP was measured following the occlusion of the defect [Ermis 2015]. LVEDP was estimated immediately following occlusion; therefore, rapid LV remodeling was not recorded following device closure.

None of our patients exhibited symptoms of left ventricular diastolic failure following ASD closure, including one patient with LVEDP values of 27 mmHg and 25 mmHg immediately following device closure, and 15 minutes after device closure, respectively. In contrast, two patients in Ermis's study with LVEDPs of 22 mmHg and 26 mmHg exhibited mild symptoms after device closure following balloon occlusion [Ermis 2015]. The reasons for this difference are unclear; however, our patient had an elevated LVEDP (20 mmHg) at baseline. Only one patient exhibited an increase in LVEDP greater than 10 mmHg immediately following device closure in our study. Therefore, our patients' left ventricular diastolic functions were superior to those of the patients in other studies despite our patients' older average age.

Tissue Doppler imaging parameters change following ASD closure. In the current study, however, no changes in E/e' were observed either at 1 day or 3 months following closure. This finding might have resulted from a baseline E/e' that was lower than the values reported by other studies.

Only baseline LVEDP predicts elevations in LVEDP following ASD closure. Ermis et al found that ASD device size was significantly correlated with changes in LVEDP [Ermis 2015]. Despite the beneficial effects of LV conditioning before ASD closure among patients with restrictive left ventricles, left ventricular restrictive physiology should be more clearly defined among patients with ASDs because LVEDP following occlusion might depend on the time of measurement following occlusion, as was the case in our study. However, an elevated LVEDP at baseline and a significant increase in LVEDP immediately following occlusion are risk factors for acute left ventricular dysfunction following ASD closure. A more detailed study involving more patients is necessary.

Our study has several limitations, primarily the small number of patients. The left ventricular diastolic function of our patients appeared to be preserved but does not represent those of all elderly patients. We measured LVEDP once at 15 minutes after closure because of time limitations. Our study lost E/e' data because it relied on a retrospective review of medical records, which might have affected the comparisons between the parameters measured before and after ASD closure. We did not propose any criteria pertaining to either left ventricular diastolic dysfunction following ASD closure or preconditioning before closure because none of our patients experienced cardiac events after closure. Therefore, additional controlled studies should be conducted to predict the risk of LV failure following ASD closure.

Conclusion

LVEDP significantly increased immediately following transcatheter ASD closure in adults and decreased rapidly over time. LVEDP 15 minutes after device closure remained higher than that before closure. LVEDP following closure was not correlated with age, Qp/Qs, or E/e' at baseline; however, it was correlated with LVEDP at both baseline and immediately following closure. Therefore, ASDs with high LVEDPs should be closed with caution.

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