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Original Article

Cardiopulmonary haemodynamics after sieve-shaped atrial septal defect repair: a multicenter study

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Abstract: Objective: The study aimed to collect case data on cardiopulmonary bypass (CPB) sieve-shaped (S-S) and non-sieve-shaped (N-S-S) atrial septal defects (ASDs). Methods: We analysed and summarized the postoperative blood flow in the cardiopulmonary system. We retrospectively collected 86 patients who underwent CPB S-S and N-S-S ASD repair. The data collected included sex, age, CPB time, ASD area, percentage change in ventricular value (PVV) (preoperative/postoperative), left ventricular wall thickness, ejection fraction (EF) (preoperative/postoperative), fluid inflow value, pulmonary arterial pressure/pulmonary venous pressure, percentage change in total lung resistance (PTLR) (preoperative/postoperative) for statistical analysis and comparison. Results: There were 86 eligible patients in this study, 37 and 49 of whom had S-S and N-S-S ASDs, respectively. The PVV, PTLR, and pulmonary arterial pressure/pulmonary venous pressure (postoperative) were significantly different between the S-S and N-S-S groups. The mean PTLR in the S-S and N-S-S groups was 0.78±0.24 and 0.62±0.28, respectively. The mean PVV in the S-S group was 11.53±7.63, and that in the N-S-S group was 16.47±9.71. Multivariate analysis revealed PVV (OR, 0.143; 95% CI, 0.026-0.790; P=0.026), PTLR (OR, 0.156; 95% CI, 0.049-0.500; P=0.002), and pulmonary arterial pressure/pulmonary venous pressure (postoperative) (OR, 9.014; 95% CI, 2.480-32.755; P=0.001) as significant factors. The rate of pulmonary infection absence postoperatively in the S-S group was 76.52%, and that in the N-S-S group was 42.75%. Conclusion: Due to the differences in heart structure between the S-S and N-S-S groups, the haemodynamic index (PVV and PTLR, postoperative pulmonary arterial pressure/pulmonary venous pressure) changes after S-S ASD repair were less than those after N-S-S ASD repair, so the postoperative pulmonary infection rate was higher after N-S-S ASD repair. The pulmonary infection rate was low after S-S ASD repair, and drugs should be reasonably administered to prevent infection.

Keywords: Sieve-shaped atrial septal defects, cardiopulmonary bypass, haemodynamics

Introduction

Atrial septal defects (ASDs) include several types of atrial trafficking, which allows blood to be shunted between the left atrium and right atrium. The presence of shunts at the atrial level can cause corresponding haemodynamic abnormalities [1-3] (**Figures 1** and **2**). Blood flow in children is shunted from left to right. Shunting leads to excessive volume in the right heart, which leads to reconstruction of the conductive beam and may also increase patient susceptibility to arrhythmias and conduction disorders [4, 5]. The effect of ASD repair is related to the age of the patient, the type and size of ASD, and other factors (such as pulmonary hypertension and cerebral thrombosis) [6, 7].

The foetus manifests genetic mutations, leading to the occurrence of ASD. At the same time, the type of ASD is different due to genetic changes, including sieve-shaped (S-S) ASDs and non-sieve-shaped (N-S-S) ASDs [8-10]. The incidence of S-S ASDs is relatively low, and there have been few related clinical studies. Thus, a unified understanding of the clinical data, diagnoses and treatments is lacking. Currently, there is no systematic research on this type of ASD [11]. The S-S type is different from the N-S-S type, and the fluid dynamics generated by these morphological differences

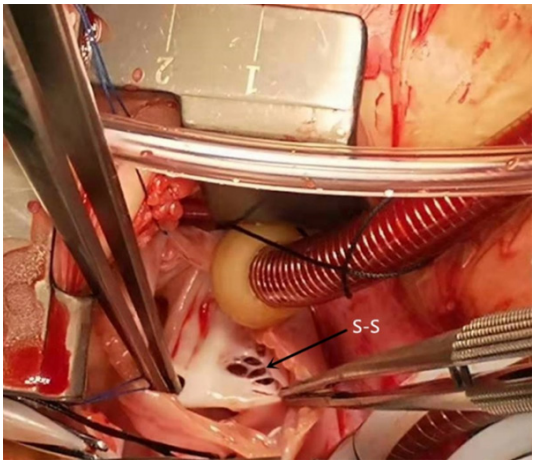

Figure 1. During cardiopulmonary bypass, the atrial septal defect showed sieve-like changes. S-S: sieve-shaped atrial septal defect.

affects heart and lung function and has an important effect on the postoperative recovery of ASD patients [12]. In the patients we observed during follow-up, there were differences in postoperative recovery effects; thus, this clinical study is of great significance and may provide guidance for clinical treatment.

Materials and methods

This study was approved by the hospital ethics committee (No.: ks2563). The patients in this study signed a written informed consent form before relevant reports and photographs were obtained.

Case sample selection

This was a retrospective study of 86 patients who underwent S-S and N-S-S ASD repair with cardiopulmonary bypass (CPB) at the First People's Hospital of Guangyuan City and the Affiliated Hospital of North Sichuan Medical College from April 2016 to October 2019.

Inclusion criteria: patients with stable vital signs before surgery (blood pressure: 80-110/50-90 mmHg; heart rate: 100-140 beats/min); patients with stable sinus heart rate and spontaneous breathing; patients who were conscious; patients with a simple ASD and no other related basic diseases; patients who underwent ASD repair performed by CPB extracorporeal circulation through a small right chest incision; patients who were successfully repaired without an atrial septal residual shunt; and patients with normal preoperative brain natriuretic peptide (BNP), troponin, and myocardial enzyme levels [13].

Exclusion criteria: patients with a right-to-left shunt in the heart before surgery; children older than ten years; patients with severe organ failure after surgery [14]; and patients with incomplete postoperative follow-up data.

Data collection

Basic patient data were collected, colour Doppler ultrasound was performed before and after the surgery, and the cardiac index of the patients before and after the operation was strictly measured [15, 16]. Based on the patients' medical history data, sex, age, CPB time, ASD area, percentage change in ventricular value (PVV), left ventricular wall thickness, cardiac ejection fraction (EF) before/after surgery, fluid inflow value, pulmonary arterial pressure/pulmonary venous pressure, and percentage change in total lung resistance (PTLR) were analysed [17]. According to the follow-up procedures, the respiratory infection symptoms, if any, and the time of onset were recorded.

Treatment

Both groups were treated with right thoracic minimally invasive surgery [18]. The patient was placed in the left decubitus position and administered with endotracheal intubation under general anaesthesia. The right chest pad was raised 30-45 degrees, the right arm was lifted up to the head for fixation, and routine disinfection and draping were performed. An arc-shaped incision was made between 3 cm from the right edge of the sternum to the axillary front and along the lower edge of the right breast to the muscular layer, and the subcutaneous tissue and breast were pushed to the head side to enter the chest cavity between the fourth rib. The intercostals were opened, the chest was opened, the right lung was pressed posteriorly, and the pericardium and right phrenic nerve were exposed. The thymus was separated forward, part of the thymus was removed, and the pericardium was cut longitudinally approximately 2 cm from the front of the right phrenic nerve (up to the pericardial fold of the ascending aorta, down to the front of the diaphragm was cut forward). The pericardium was then suspended, followed by systemic heparinization and routine establishment of CPB.

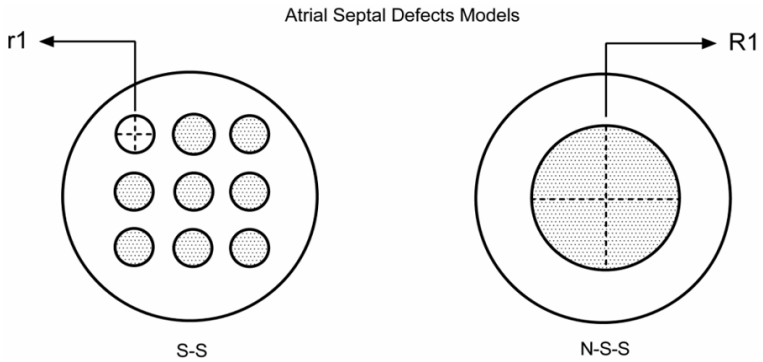

Figure 2. Schematic diagram of the S-S and N-S-S. According to Poiseuille's law, the blood flow is proportional to the fourth power of the cross-sectional radius. When the flow resistance is constant, the greater the radius, the greater the flow and the greater the load on the right ventricular system. It is considered that when R1>r1, and the radius of N-S-S is greater than that of S-S, there is a difference between them. S-S: sieve-shaped atrial septal defects; N-S-S: non-sieve-shaped atrial septal defects.

The superior and inferior vena cava and aorta were blocked, and the head was positioned downward at 30 degrees. The anterior wall of the right atrium was cut to expose the atrial septum. According to the size and type of defect, direct sutures, autologous pericardial patches or polyester patches were selected to repair ASDs. After the repair, the tricuspid valve was checked to ensure it was completely closed. The superior and inferior vena cavas were opened, the right atrial incision was sutured, and the cardiac cavity was exhausted. Transitional CPB was initiated. After complete haemostasis and pericardial suturing, a drainage tube was placed in the right thoracic cavity, and the chest was routinely closed. The amount of fluid in and out of patients was controlled according to body weight after surgery, and cardiac and diuretic drugs were used as appropriate.

Follow-up

Patients were followed up with a mailed questionnaire or telephone call by contacting the referring cardiologist or general practitioner.

Statistics

The obtained data were analysed by SPSS 24.0 statistical analysis software (SPSS Inc., Chicago, Illinois). Quantitative data conforming to a normal distribution are expressed as the mean ± standard deviation ($\bar{x}$ ± sd), and independent samples/paired samples were selected for the t-test. Count data are expressed as proportions or percentages, and the χ^2 test or Fisher's exact probability method was used for statistical analysis. Binary logistic regression analysis was employed to explore the relevant factors associated with lung infection, and the Pearson correlation test was performed to detect the correlations of the variables. The Kaplan-Meier (K-M) curve was used to understand the occurrence of lung infection in patients. P< 0.05 was considered for significant differences.

Results

Clinical data

Over a mean follow-up time of 21.4±6.3 months (range: 1.5 to 40.6 months), there was 0 death. According to the inclusion and exclusion criteria, 113 patients were enrolled; 27 patients were excluded, and the remaining 86 patients were included. Among them, 37 patients were S-S type and 49 patients were N-S-S type. The average age of the patients in the two groups was 3.6±2.2 years, and 40 of the patients (46.5%) were male. The average extracorporeal circulation time was 65.8±25.2 min, the ASD area was 9.5±3.2 mm^2, the PVV was 10.8± 7.2%, and the left ventricular wall thickness was 8.0±5.2 mm. The EF preoperative/postoperative was 95.2±70.2, the fluid inflow value was 115.6±70.2 mL, the pulmonary arterial pressure/pulmonary venous pressure (preoperative) was 6.2±3.2, the pulmonary arterial pressure/pulmonary venous pressure (postoperative) was 4.5±2.8, and the PTLR was 0.9± 0.3. There were no statistically significant differences in sex, age, CPB time, ASD area, left ventricular wall thickness, preoperative/postoperative EF, fluid inflow value, and pulmonary artery pressure/pulmonary venous pressure (preoperative) between the two groups (P> 0.05). There were statistically significant differences between the two groups in the PVV, pulmonary arterial pressure/pulmonary venous pressure (postoperative), and PTLR (P<0.05).

Table 1. Comparison of clinical data between the two groups of patients

Variable	S-S (n=37)	N-S-S (n=49)	t-value/ χ^2-value	*P*-value
Age (year, x ± sd)	3.2±2.5	3.5±1.8	-0.121	0.225
Male (n, %)	17,46.0	23,47.0	-0.362	0.421
CPB time (min)	72.4±22.1	68.2±25.2	0.564	0.412
Atrial septal defect area (mm^2)	9.3±2.5	8.3±2.8	0.252	0.215
PVV (%)	9.8±7.2	20.3±12.2	-1.232	0.032
Left ventricular wall thickness (mm)	7.6±5.6	8.3±5.1	-0.754	0.413
EF before/after surgery (%)	96.3±70.2	86.3±56.1	0.564	0.096
Fluid inflow value (mL)	106.2±50.2	110.6±61.3	-0.121	0.125
Pulmonary arterial pressure/pulmonary venous pressure (%)				
Before surgery	6.3±3.2	5.9±4.0	0.060	0.528
After surgery	5.3±2.0	3.5±2.3	0.204	0.026
PTLR (WU)	0.9±0.3	0.6±0.3	0.233	<0.001

Note: CPB: cardiopulmonary bypass; PVV: percentage change in ventricular value; EF: ejection fraction; PTLR: percentage change in total lung resistance; S-S: sieve-shaped atrial septal defects; N-S-S: non-sieve-shaped atrial septal defects.

Table 2. Pearson correlation test of pulmonary arterial pressure/pulmonary venous pressure (postoperative) with PTLR

	X3	X4
X3		
Pearson correlation	1	0.413
Saliency (two-tailed)		0.015
n	37	37
X4		
Pearson correlation	0.413	1
Saliency (two-tailed)	0.015	
n	37	37

Note: X3: pulmonary arterial pressure/pulmonary venous pressure (postoperative); X4: PTLR. PTLR: percentage change in total lung resistance.

The comparison of clinical data between the two groups of patients is shown in **Table 1**.

Pearson correlation analysis

Univariate analysis confirmed that there were significant differences in the PVV, pulmonary arterial pressure/pulmonary venous pressure (postoperative), and PTLR between the two groups (**Table 1**). To clarify the correlation between the above variables, Pearson test was performed. The above variables conformed to the normal distribution (or mildly skewed distribution) such that they could be included in the study. Testing showed that there were no significant correlations among the PVV, pulmonary arterial pressure/pulmonary venous pressure (postoperative) and PTLR, but there was a significant correlation between the pulmonary arterial pressure/pulmonary venous pressure (postoperative) and PTLR.

Pearson correlation test of pulmonary arterial pressure/pulmonary venous pressure (postoperative) and PTLR

Pulmonary arterial pressure/pulmonary venous pressure (postoperative) and PTLR data were included in the Pearson correlation test, and the results showed a significance value of 0.015; specifically, the chance of no correlation was 1.5%, indicating that the correlation between the two groups of variables was very significant. The Pearson correlation coefficient was 0.413, indicating that the two group variables were positively correlated, and the correlation was relatively strong (**Table 2**).

Pearson correlation test between PVV and PTLR

PVV and PTLR data were included in the Pearson correlation test, and the results revealed a significance value of 0.983, i.e., a chance of no correlation of 98.3%, indicating no significant correlation between the two groups of variables. The Pearson correlation coefficient was 0.004, indicating that the two groups of variables had a weak correlation (P<0.05; **Table 3**).

Table 3. Pearson correlation test between PVV and PTLR

	X2	X4
X2		
Pearson correlation	1	0.004
Saliency (two-tailed)		0.983
n	37	37
X4		
Pearson correlation	0.004	1
Saliency (two-tailed)	0.983	
n	37	37

Note: X2: PVV; X4: PTLR. PVV: percentage change in ventricular value; PTLR: percentage change in total lung resistance.

Table 4. Pearson correlation test between PVV and pulmonary arterial pressure/pulmonary venous pressure (postoperative)

	X2	X3
X2		
Pearson correlation	1	0.060
Saliency (two-tailed)		0.735
n	37	37
X3		
Pearson correlation	0.060	1
Saliency (two-tailed)	0.735	
n	37	37

Note: X2: PVV; X3: pulmonary arterial pressure/pulmonary venous pressure (postoperative). PVV: percentage change in ventricular value.

Pearson's correlation test of PVV and pulmonary arterial pressure/pulmonary venous pressure (postoperative)

PVV and pulmonary arterial pressure/pulmonary venous pressure (postoperative) data were included in the Pearson correlation test, and the results showed a significance value of 0.735, with a chance of no correlation of 73.5%, indicating that the correlation between the two groups of variables was less significant. A Pearson correlation coefficient of 0.060 indicated that the two groups of variables had a weak correlation (P<0.05; **Table 4**).

PVV and PTLR group scatter plot

There was no significant linear correlation between the PVV and PTLR in the S-S and N-S-S groups (**Figure 3**).

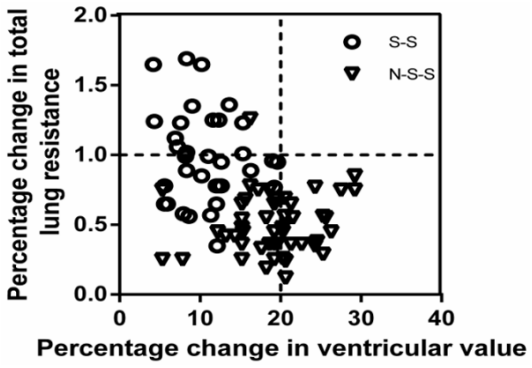

Figure 3. S-S and N-S-S groups in PVV and PTLR scatter plot diagram. S-S: sieve-shaped atrial septal defects; N-S-S: non-sieve-shaped atrial septal defects; PVV: percentage change in ventricular value; PTLR: percentage change in total lung resistance.

PVV and PTLR follow-up results at 12 months

According to the PVV and PTLR in the S-S and N-S-S groups, the mean PTLR in the S-S and N-S-S groups at 12 months after the operation was 0.78±0.24 and 0.62±0.28, respectively. The mean PVV in the S-S group at 12 months postoperation was 11.53±7.63, and the mean PVV in the N-S-S group at 12 months postoperation was 16.47±9.71. The changes in the PVV and PTLR between the two groups were significantly different (P<0.05; **Figures 4** and **5**).

Binary logistic regression analysis of PVV, PTLR, and pulmonary artery pressure/pulmonary venous pressure (postoperative)

The results revealed significant differences in the PVV, PTLR, and pulmonary arterial pressure/pulmonary venous pressure (postoperative) between the two groups of patients. The maximum likelihood ratio method and stepwise binary logistic regression analysis (entry: 0.05, removal: 0.10) were used to incorporate the above variables into the model (**Table 5**).

K-M curves of pulmonary infection after S-S and N-S-S ASD repair by CPB

According to the postoperative CPB effect analysis of the S-S and N-S-S groups, the time of postoperative pulmonary infection was determined to obtain the postoperative infection rate curve. The rate of pulmonary infection absence 12 months after the operation in the

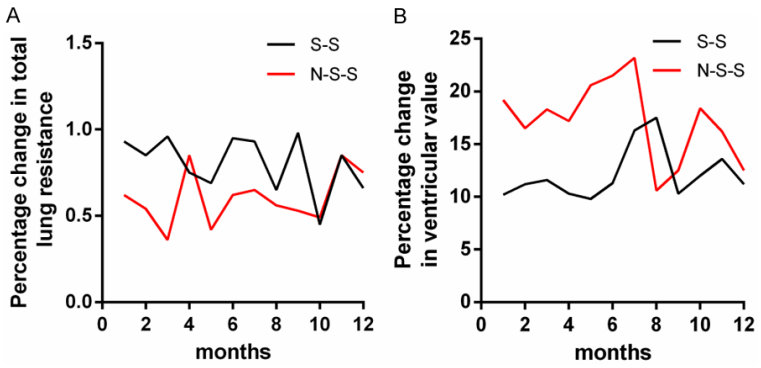

Figure 4. Changes in the two observation indicators in the two groups within 12 months. A: Line chart of changes in PTLR in the S-S and N-S-S groups at 12 months after surgery; B: Line chart of changes in PVV in the S-S and N-S-S groups at 12 months after surgery; S-S: sieve-shaped atrial septal defects; N-S-S: non-sieve-shaped atrial septal defects.

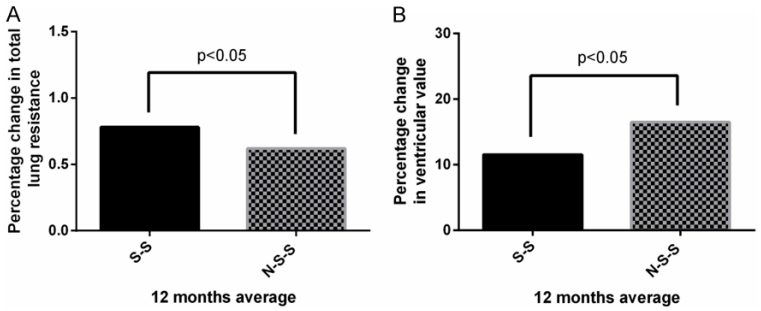

Figure 5. The average value of the two observation indexes in the two groups at 12 months. A: Histogram of the mean 12-month postoperative PTLR in the S-S and N-S-S groups; B: Histogram of the mean 12-month postoperative PVV in the S-S and N-S-S groups. S-S: sieve-shaped atrial septal defects; N-S-S: non-sieve-shaped atrial septal defects; PVV: percentage change in ventricular value; PTLR: percentage change in total lung resistance.

S-S group was 76.52%, and that in the N-S-S group was 42.75%. The pulmonary infection rate in the N-S-S group was significantly higher than that in the S-S group. The results showed that there was a significant difference in the distribution of infection between the two groups (χ^2=4.976, P=0.026). The pulmonary infection occurred later after N-S-S repair, and the incidence of lung infection after surgery was higher in the N-S-S group (**Figure 6**).

Discussion

Normally, the left atrial pressure is higher than the right atrial pressure; thus, a left-to-right shunt occurs in the presence of an ASD, and the shunt mainly depends on the pressure difference and defect area of the left and right atria. When pulmonary circulating blood flow exceeds the capacity limit of the pulmonary vascular bed, volume-induced pulmonary hypertension can occur. Long-term pulmonary hypertension can lead to pulmonary intimal hyperplasia, thickening of the tube wall, and resistance-induced pulmonary hypertension.

The univariate analysis confirmed that the PVV, PTLR and pulmonary arterial pressure/pulmonary venous pressure were significantly different between the S-S and N-S-S groups after surgery. At the same time, after 12 months of follow-up, the time points of pulmonary infection in the two groups were also different (P< 0.05). Logistic regression analysis showed that the above three observation factors had certain effects on the incidence of postoperative infection in both groups and were independent factors (P<0.05). The above analysis also confirmed that different types of ASD after the same surgical treatment will indeed lead to different postoperative pulmonary infections and affect the postoperative effect. Frost AE et al. considered that the abnormal structure of the atrial septum was related to the pressure balance of the pulmonary arteriovenous system [19]. Since the basic conditions of the two groups of patients were the same, abnormalities in the internal structure of the heart and differences in hemodynamics were the root causes of the differences in the above three indicators and postoperative effects [20].

Relationship between cardiac structural differences and PVV

S-S and N-S-S ASDs have some differences in morphology and structure, leading to some differences in preoperative atrial septal blood flow. Yoo et al. have shown that different ASD forms may result in changes in the myocardial velocity, myocardial function index (MPI) and isovolumetric acceleration postoperatively due to structural differences [21]. Blood flow refers

Table 5. Analysis of related independent factors of pulmonary infection after ASD

Variable	Single-factor analysis (*P*-value)	Binary logistic regression analysis		
		B-value	OR-value (95% CI)	*P*-value
PVV	0.032	-1.948	0.143 (0.026-0.790)	0.026
PTLR	0.000	-1.855	0.156 (0.049-0.500)	0.002
Pulmonary arterial pressure/pulmonary venous pressure (postoperative)	0.026	2.199	9.014 (2.480-32.755)	0.001

Note: PVV: percentage change in ventricular value; PTLR: percentage change in total lung resistance; ASD: atrial septal defect.

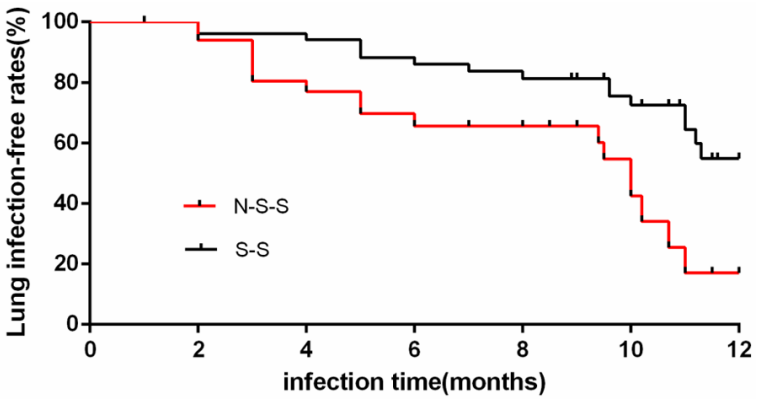

Figure 6. Rate curves of pulmonary infection at 12 months after surgery for the S-S and N-S-S groups. S-S: sieve-shaped atrial septal defects; N-S-S: non-sieve-shaped atrial septal defects.

to the volume of blood flowing through a section of a blood vessel per unit time and the volume velocity. In the recirculation system, the relationship between blood flow, blood flow resistance, and blood pressure is as follows: Q= (p2-p1)/R [22], where p2-p1 represents the pressure difference across the blood vessel, R represents blood flow resistance, and Q represents blood flow. Blood flow is directly proportional to the pressure difference between the two ends of the pipeline and inversely proportional to the resistance of the pipeline to liquid flow. The relationship between them can be described by Poiseuille's law, Q=π*r^4*Δp/(8 ηL) [23], where η is the blood viscosity, L is the length of the blood vessel, and r is the radius of the blood vessel. The flow resistance R is inversely proportional to the fourth power of the pipe radius r. This shows that the influence of the pipe radius on the flow resistance is very large [24].

However, in actual S-S ASDs, the size of the sieve holes varies, and the number of sieve holes is significantly less than the theoretical value of the number of sieve holes with the same pressure. Therefore, the flow rate through S-S ASDs is significantly lower than that through N-S-S ASDs. Ventricular septal and right ventricular work causes insufficient left ventricular dilation. Left ventricular compressive deformation, left ventricular morphological flattening, decreased dilatation, and reductions in the left ventricular end-diastolic anteroposterior diameter (LVEDD) and left ventricular end-systolic anteroposterior diameter (LVESD) occur. After ASD repair, as the postoperative haemodynamic disorder is corrected, the blood returning to the left atrium increases, and the right ventricular volume load is reduced. The reduction or disappearance of abnormal ventricular septal movements, combined with increased left ventricular work, reduces the pressure on the left ventricle, progressively changes the size of the left ventricle, and increases left ventricular dilatation. We found that left-to-right blood flow was lower after S-S ASD repair, with decreased left ventricular deformation, and the PVV was lower than that after N-S-S ASD repair (**Figure 7**).

Therefore, in the case of constant pressure on the left and right sides of the ASD, because the resistance of S-S ASDs is large, the shunt flow through S-S ASDs is small. In the long term, because N-S-S ASDs have more blood flowing to the right heart than S-S ASDs, causing the left heart to contract and relax more, there is a greater change in the heart after blood flow returns to normal after repairing the heart. Tashiro et al. showed that the left ventricle increased in size with time after ASD closure, but the left ventricular size in elderly patients did not reach the level observed in young patients. In addition to left ventricular insufficiencies, diastolic dysfunction may also lead to late left atrial enlargement after ASD closure in elderly patients [25].

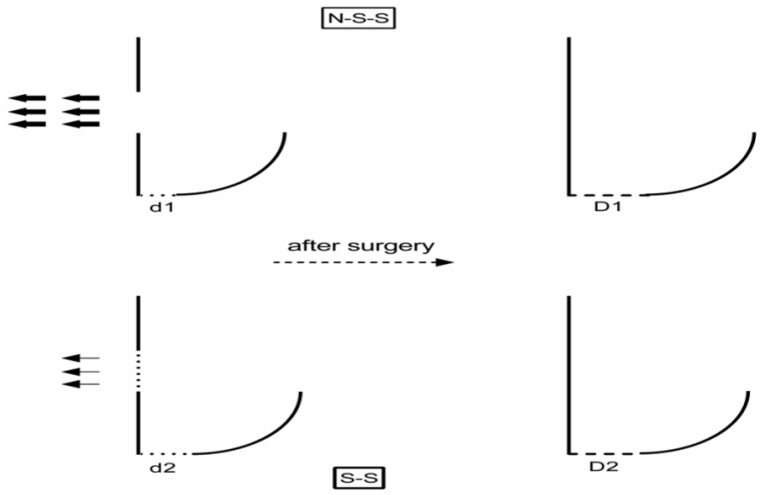

Figure 7. Postoperative ventricular changes in the S-S and N-S-S groups. d1<d2, D1≈D2, D1-d1>D2-d2. S-S: sieve-shaped atrial septal defects; N-S-S: non-sieve-shaped atrial septal defects.

rate of pulmonary infection in ASD patients may be improved, but it is still a difficult problem in patients with severe disease [31]. In this study, we found that the PTLR differed between the S-S and N-S-S groups.

The TLR (Wood units) was calculated as the mean pulmonary arterial pressure (mmHg)/pulmonary circulation blood flow (L/min), and the PVR (Wood units) was calculated as follows: [mean pulmonary arterial pressure (mmHg)-mean pulmonary capillary pressure (mmHg)]/pulmonary circulating blood flow (L/min).

Heart structure differences and PTLR

Pulmonary arterial hypertension (PAH) is an important indicator for evaluating the condition of the pulmonary vascular bed in patients with ASD, the indication for surgery, and the postoperative follow-up assessment of recovery. The total lung resistance (TLR) is a measure of the pulmonary vascular bed and is one of the most important indicators for evaluating PAH [26]. Previous studies have suggested that in patients with an ASD complicated by PAH, the pulmonary artery systolic blood pressure decreases during the postoperative follow-up, suggesting that the pathological changes in the pulmonary vascular bed, caused by the increased volume load before surgery, gradually return to normal after surgery; however, these pathological changes require a long transition period, during which the pulmonary arterial pressure may even increase slightly [27]. However, related research indicates that remodelling and changes in the right heart are not completely reversible for pulmonary vascular abnormalities in patients treated with advanced ASD repair, which is related to the different types of ASDs and longer development times, leading to hemodynamic changes [28-30].

In the early stage of cardiac repair, the pressure of the pulmonary arteries increases slightly after N-S-S ASD repair, resulting in an increased probability of pulmonary congestion and an increased possibility of postoperative pulmonary infections. Nassif et al. showed that the PVR directly reflects the state of the pulmonary vasculature and is a more accurate parameter that reflects the condition of the pulmonary vascular bed. TLR refers to the resistance of the right ventricular blood flow to the left atrium and left ventricle through the pulmonary artery and its branches after draining from the right ventricle. The impact of left-to-right shunts on the pulmonary vascular network is manifold. The pulmonary vascular network is a low-resistance system with a large capillary network and strong blood reserve capacity. It can reduce lung resistance in the early stages to accommodate an increased volume load [32]. However, the increased volume load caused by the continuous left-to-right shunt can cause progressive damage to the pulmonary vascular intima. Early pathological changes include medial hypertrophy and endothelial cell proliferation. These changes are reversible after blocking the shunt. This study suggests that the TLR measured after ASD repair in the S-S and N-S-S groups is higher than that measured before surgery, with greater change in the N-S-S group than that in the S-S group (**Figure 8**). A possible explanation is that ASD patients have long-term left-to-right shunting and increased pulmonary circulation blood flow, which opens the pulmonary arteriovenous direct channel and capillary bed early in the disease. Rao and others involved in clinical trials have also described the possible mechanism [33]. Surgery blocked the left-to-right shunt, and the pulmonary circulation blood flow immediately decreased after

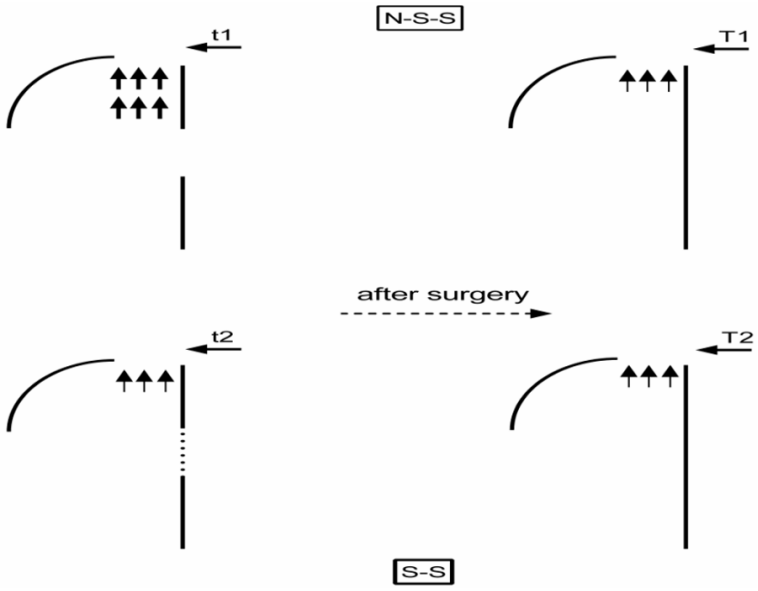

Figure 8. TLR changes after S-S and N-S-S. t1>t2, T1≈T2, so t1-T1>t2-T2. S-S: sieve-shaped atrial septal defects; N-S-S: non-sieve-shaped atrial septal defects.

ASD repair. The previous pathologically open pulmonary arteriovenous direct channel and capillary bed were closed again due to reduced volume, resulting in blood loss. Resistance to flow through the pulmonary circulation subsequently increased. PVR is blood flow-dependent, and it should not be assumed that PVR will necessarily decrease proportionally with the decrease in shunt flow and pulmonary blood flow. Therefore, the increase in PVR in a short time after surgery can be explained. Due to the difference in preoperative ASDs between the S-S and N-S-S groups, the left and right atrial pressures were the same in the two groups before surgery. Moreover, because of the greater atrial septal resistance in the S-S group, the blood flow to the right heart was lower, and the TLR before surgery was lower than that in the N-S-S group. The number of arteriovenous direct channels and capillary beds is relatively small, resulting in a return to normal pulmonary blood flow in the S-S group through pathological mechanisms in a short time after surgery, while in the N-S-S group, the preoperative TLR is relatively large, which leads to a larger increase in the postoperative TLR. Thus, there is a greater possibility of related pulmonary symptoms. The peak tricuspid regurgitation velocity/right ventricle outflow tract blood flow time-velocity integral (TRV2/TVIRVOT) and other ultrasound indicators reflect PVR, and these indicators have a good correlation with cardiac catheter measurements [34, 35]. Studies have shown that PVR decreases following ASD repair. However, when does PVR reach its peak and start to decrease after surgery? The cause of the transient increase in PVR after surgery needs more pathophysiological research.

Analysis of pulmonary blood flow differences and postoperative pulmonary infection

S-S and N-S-S ASDs showed a preoperative difference in shunt volume, and the blood flow of the left-to-right shunts in the N-S-S group was significantly higher than that in the S-S group, resulting in a better systemic blood volume and a relatively high post-load in the S-S group. After ASD repair, the blood volume of the pulmonary circulation was significantly reduced. The blood flow of the left-to-right shunts in the N-S-S group was significantly higher before surgery. After ASD repair, significantly more blood returned to the systemic circulation (much more than after S-S repair), so the post-load increased significantly [36]. Left heart valve regurgitation leads to different venous returns, increased pulmonary capillary pressure, intravascular fluid infiltration into the interstitial lung and alveoli, pulmonary oedema, and pulmonary infection [37] (**Figure 9**).

At the same time, excited β2 receptors increase the heart rate and cause myocardial oxygen consumption to increase. Increasing oxygen consumption also increases the potential risk of myocardial ischaemia in some high-risk patients, leading to the appearance of heart failure [38].

In our observations, we also found that the BNP level was abnormal in the two groups, but there was no significant relationship in the BNP level between the two groups. Notably, when a patient has a lung infection, the patient's BNP level increases accordingly, which indicates that after ASD repair, the patient's BNP level needs to be carefully screened to determine

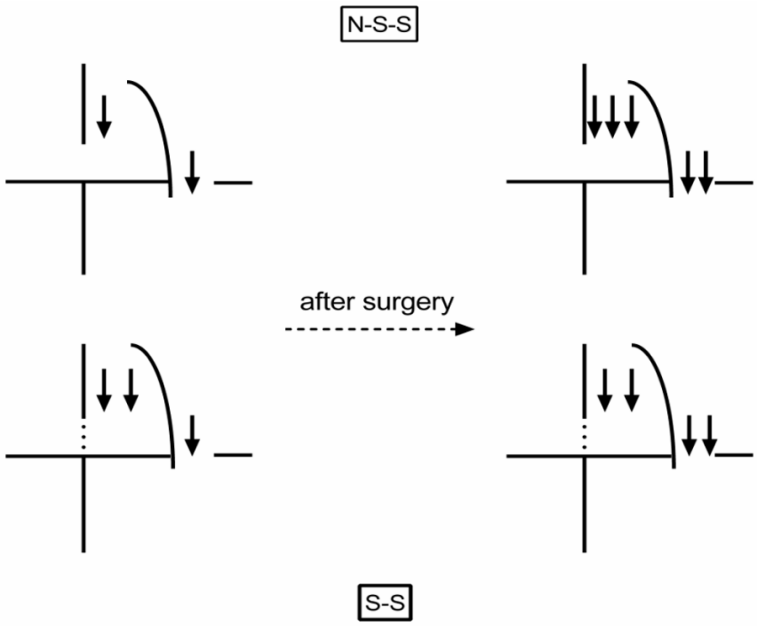

Figure 9. Changes in blood flow before and after surgery in the S-S and N-S-S groups. S-S: sieve-shaped atrial septal defects; N-S-S: non-sieve-shaped atrial septal defects.

the possibility of lung infection. Mekontso-Dessap et al. described the relationship between BNP, the internal environment and ventricular pressure [39]. BNP is mainly secreted from the ventricle and is mainly concentrated at the junction of the myocardial infarct and non-infarct areas. In such patients, the systemic neurohumoural system is activated, and the level of BNP, as a small part of the neurohumoural system, will increase significantly in the case of overloading of the left ventricle [40].

For the relief of pulmonary oedema, the following schemes are commonly used: sodium nitroprusside is a vasodilator that can effectively expand arteriovenous smooth muscle without affecting the duodenum, uterus, or myocardial contraction, and peripheral vascular resistance is significantly reduced after vasodilation. Deacetyllanoside injection is a digitalis-type positive inotropic drug that has a significant inhibitory effect on Na^+-K^+-ATPase activity on the myocardial cell membrane, which can increase myocardial contraction, improve cardiac output, and reduce heart load pulmonary oedema.

This study has several limitations. First, the sample size was small, and the follow-up time was short. Thus, further trials with larger patient populations and long-term follow-up are needed. Second, the S-S model is not necessarily applicable to all patients with S-S ASDs, so there is a certain deviation in fluid dynamics. Therefore, there may be a difference between the model and the real situation. Third, postoperative pulmonary infection is related not only to the difference in cardiopulmonary hemodynamics but also to the patient's own resistance and environment. Therefore, these results need to be confirmed by a large-sample-size research.

Conclusion

Due to the differences in heart structure between the S-S and N-S-S groups, the hemodynamic index (PVV, PTLR, and pulmonary arterial pressure/pulmonary venous pressure (postoperative)) changes in the S-S group were less than those in the N-S-S group, so the postoperative pulmonary infection rate was higher in the N-S-S group. The pulmonary infection rate was low after S-S ASD repair, and drugs should be used as appropriate to prevent infection.

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Disclosure of conflict of interest

None.

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