Pulmonary Vascular Resistance as Assessed by Bicycle Stress Echocardiography in Patients With Atrial Septal Defect Type Secundum

Alexander Van De Bruaene, MD; Andre La Gerche, MD; David L. Prior, MD, PhD; Jens-Uwe Voigt, MD, PhD; Marion Delcroix, MD, PhD; Werner Budts, MD, PhD

Background—Volume overload of the pulmonary circulation in patients with atrial septal defect (ASD)-type secundum causes pulmonary vascular disease over a long period of time. This study aimed at (1) evaluating pulmonary vascular resistance (PVR) and (2) investigating the relation between PVR and exercise capacity in patients with open and closed ASD.

Methods and Results—Forty-six patients with ASD-type secundum (18 open, 28 closed) and 20 age-matched controls were enrolled. All underwent standard and symptom-limited bicycle stress echocardiography and cardiopulmonary exercise testing. PVR was calculated as the slope of a pulmonary artery systolic pressure (PAP)-flow plot or as the ratio of PAP to cardiac output (total PVR). The slope of PAP-flow was higher in patients with open (5.1±2.2 versus 3.0±0.8 mm Hg/L per min; \(P=0.002\)) and closed ASD (4.0±1.7 versus 3.0±0.8 mm Hg/L per min) compared with controls. In patients with an open ASD, total PVR did not change from rest to peak exercise. In patients with a closed ASD and controls, total PVR decreased from rest to peak exercise. However, in patients with an ASD closed later in life (≥34 years, median age at repair), the slope of PAP-flow was higher (5.1±1.4 versus 3.0±0.8 mm Hg/L per min; \(P<0.0001\)), but total PVR did not change from rest to peak exercise. Peak oxygen consumption correlated inversely with the slope of the PAP-flow plots in patients with open (\(P=0.013\)) and closed ASD (\(P=0.005\)).

Conclusions—In patients with an open ASD, the slope of PAP-flow was higher and total PVR did not change from rest to peak exercise. In patients with an ASD closed later in life, the slope of PAP-flow was higher and total PVR did not change from rest to peak exercise, indicating altered pulmonary hemodynamics in these patients. (Circ Cardiovasc Imaging. 2011;4:237-245.)

Key Words: atrial septal defect ■ bicycle stress echocardiography ■ pulmonary vascular disease

It is generally accepted that long-standing volume overload of the right ventricle and pulmonary circulation in patients with atrial septal defect (ASD)-type secundum causes pulmonary vascular disease over a long period of time.\(^1\) However, noninvasive assessment of mild pulmonary vascular disease remains difficult. The pulmonary vasculature is able to adapt to a higher cardiac output by diminishing pulmonary vascular resistance (PVR), as has been previously described.\(^2\) However, the persistently elevated pulmonary blood flow in ASD patients also causes shear stress on the endothelium, inducing progressive pulmonary vascular lesions.\(^1\) The earlier stages of shunt-induced pulmonary vascular disease, as first described by Heath and Edwards, present with medial hypertrophy and/or intimal proliferation and are largely reversible after closure of the defect.\(^3,4\) When occlusive fibrotic lesions have developed, closure of the ASD later in life, although still feasible, may not result in complete normalization of pulmonary artery pressures and has been shown to be associated with worse outcome.\(^5-7\) Studies have suggested that an abnormal increase in pulmonary artery pressures during exercise reflects mild pulmonary vascular disease and limits exercise capacity.\(^8-11\) However, pulmonary artery pressures are defined by both cardiac output and PVR.\(^12-14\) Using bicycle stress echocardiography, PVR can be estimated either as a ratio of pulmonary artery systolic pressure (PAP) and cardiac output at each stage (total PVR) or by using linear regression analysis of the pressure-flow plots (slope of a PAP-flow plot).\(^2,13\) This study aimed at (1) evaluating PVR in patients with open and closed ASD and (2) investigating the relation between the slope of the PAP-flow plots with functional capacity as assessed by cardiopulmonary exercise testing in ASD patients.

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Methods

Study Population
In total, 18 patients with open ASD, all scheduled for repair (mean age 39±19 years), 28 with closed (mean age 41±17 years) ASD-
type secundum and 20 age-matched healthy volunteers (mean age 42±11 years) gave informed consent to participate in the study. Patients were consecutively enrolled in the outpatient clinic of congenital heart disease of the University Hospitals of Leuven, Belgium from March to November 2009. Patients younger than 17 and patients with known coronary artery disease, significant valvular diseases other than mild tricuspid or mitral regurgitation, pulmonary disease, a history of pulmonary embolism, or concomitant congenital heart disease were excluded from the study. Patients with a history of arrhythmias or current arrhythmias also were excluded. None of the healthy volunteers were competitive athletes. The University Hospitals Leuven Institutional Review Board approved the study, and informed consent was obtained from all patients.

**Right Heart Catheterization**

Both patients with open ASD, those with open ASD scheduled for repair, and patients with closed ASD underwent right heart catheterization with assessment of pulmonary artery pressures. Shunt ratio was calculated using the Fick method.

**Bicycle Stress Echocardiography**

Echocardiography was performed with a Vivid 7 or 9 ultrasound system (General Electric Vingmed Ultrasound, Horten, Norway) equipped with a 3-MHz transducer and tissue Doppler imaging technology. Before exercise, a complete resting echocardiography study was performed with the patient in the supine position in all standard views. Two-dimensional, Doppler, and tissue Doppler images were digitized for off-line analysis using dedicated software (EchoPac BT08, General Electric Vingmed Ultrasound, Horten, Norway).

Exercise echocardiography was performed on a semisupine ergometer (Easystress, Ecogito Medical sprl, Lierre, Belgium). The exercise table was tilted laterally from 20° to 30° to the left. The protocol started at 25 watts with an increment of 25 watts every 2 minutes or until the maximum tolerated load. Blood pressure and 12-lead ECG were recorded at rest and every 2 minutes during exercise. A single observer performed the on-line and the off-line analyses. All measurements were made in triplicate, and results are presented as means.

**HemodynamicCalculations**

Right ventricular (RV) and left ventricular (LV) stroke volume and cardiac output were calculated from the flow velocity time integral in the outflow tract of both ventricles. LV ejection fraction was calculated using end-systolic and end-diastolic areas measured from the apical 4- and 2-chamber views. RV function was assessed using the RV area shortening fraction and the tricuspid annular plane systolic excursion. Mitral Doppler inflow patterns were obtained from tricuspid regurgitant velocities using the simplified Bernoulli equation (4 times velocity squared). During exercise echocardiography, LV and RV cardiac output were recorded at each stage. PAP was calculated using the Fick method.

**Cardiopulmonary Exercise Test**

Supine exercise testing was performed on a bicycle ergometer (Ergometrics 800S, Ergometrics, Bitz, Germany). The initial workload of 20W was increased by 20W every minute until exhaustion. A 12-lead ECG (Max Personal Exercise Testing, Marquette, WI), blood pressure, and respiratory data through breath-by-breath anal-

<table>
<thead>
<tr>
<th>Table 1. Patient Characteristics</th>
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<tr>
<td></td>
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<tr>
<td>Age at visit (years)</td>
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<tr>
<td>Age at repair (years)</td>
</tr>
<tr>
<td>Sex (male:female)</td>
</tr>
<tr>
<td>Defect size (mm)</td>
</tr>
<tr>
<td>sPAP (mm Hg)*</td>
</tr>
<tr>
<td>mPAP (mm Hg)*</td>
</tr>
<tr>
<td>Shunt ratio (Qp:Qs)*</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
</tr>
<tr>
<td>Syst BP (mm Hg)</td>
</tr>
<tr>
<td>Diast BP (mm Hg)</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
</tr>
</tbody>
</table>

sPAP indicates systolic pulmonary artery pressure; mPAP, mean pulmonary artery pressure; BMI, body mass index; Syst BP, systolic blood pressure; Diast BP, diastolic blood pressure.

Statistical Analysis

We analyzed the data using SPSS for Windows (Version 16, SPSS, Chicago, IL). Descriptive data for continuous variables are presented as means±SD or as medians with ranges, when appropriate. Descriptive data for discrete variables are presented as frequencies or percentages. Continuous variables were evaluated between subgroups using 1-way ANOVA analysis and, if significant, subgroups were compared using independent t test analysis. Proportions were evaluated between subgroups using χ² analysis. The change in total PVR from rest to peak exercise was evaluated using a paired t test analysis. Patients with a closed ASD were stratified according to median age at repair to compare early and late defect closure. Pearson correlation coefficient was used to estimate the correlation between peak VO₂ and the slope of the PAP-flow plots. A Poone adjusted mean slope for each subgroup was calculated, allowing for similar linear or mildly nonlinear data from different subjects to be grouped together for graphical analysis while effectively minimizing intersubject variabilities that could confound the qualitative relationship.¹⁹ Intra- and interobserver variability testing was performed for baseline and peak measurements of PAP and RV cardiac output. Intraobserver variability was assessed by repeated analysis of 10 data sets at least 2 months after the initial analysis and blinded to the initial results. Assessment of interobserver variability was performed by a second observer. The agreement between the 2 measurements was expressed using the 95% confidence interval and determined as the mean bias±1.96 SD. Inter- and intraobserver reliability also was assessed using the intraclass correlation coefficient.¹⁹ All tests were 2-sided, and P<0.05 was considered statistically significant.

**Results**

**Patient Characteristics**

Characteristics of patients with open ASD, closed ASD, and controls are summarized in Tables 1, 2 and 3. Age, body mass index, heart rate, systolic blood pressure, and diastolic blood pressure at rest were not statistically different between
Table 2. Bicycle Exercise

<table>
<thead>
<tr>
<th>Type of Repair</th>
<th>Exercise capacity (watt)</th>
<th>Peak syst BP (mm Hg)</th>
<th>Peak diast BP (mm Hg)</th>
<th>Peak heart rate (bpm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Open</td>
<td>118 ± 46</td>
<td>172 ± 24</td>
<td>86 ± 16</td>
<td>134 ± 32</td>
</tr>
<tr>
<td>Closed</td>
<td>130 ± 61</td>
<td>168 ± 25</td>
<td>77 ± 12</td>
<td>129 ± 27</td>
</tr>
<tr>
<td>Control</td>
<td>179 ± 52*†</td>
<td>192 ± 24*†</td>
<td>93 ± 14*†</td>
<td>149 ± 21*</td>
</tr>
</tbody>
</table>

*P<0.05 vs control.
†P<0.05 vs open ASD.

ASD indicates atrial septal defect; syst BP indicates systolic blood pressure; diast BP, diastolic blood pressure.

Table 3. Demographics of Patients With a Closed ASD

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Type of Repair</th>
<th>Age at Repair (Years)</th>
<th>Interval Between Repair and Study (Years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Male</td>
<td>Surgical</td>
<td>18.6</td>
<td>11.2</td>
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<tr>
<td>2</td>
<td>Female</td>
<td>Device</td>
<td>18.4</td>
<td>0.6</td>
</tr>
<tr>
<td>3</td>
<td>Female</td>
<td>Surgical</td>
<td>3.1</td>
<td>15.9</td>
</tr>
<tr>
<td>4</td>
<td>Male</td>
<td>Surgical</td>
<td>28.8</td>
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</tr>
<tr>
<td>5</td>
<td>Female</td>
<td>Surgical</td>
<td>13.2</td>
<td>11.2</td>
</tr>
<tr>
<td>6</td>
<td>Female</td>
<td>Surgical</td>
<td>4.4</td>
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<tr>
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<td>Surgical</td>
<td>1.7</td>
<td>26.7</td>
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<tr>
<td>8</td>
<td>Male</td>
<td>Device</td>
<td>23.8</td>
<td>1.9</td>
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<tr>
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<td>Female</td>
<td>Surgical</td>
<td>7.3</td>
<td>18.9</td>
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<tr>
<td>10</td>
<td>Female</td>
<td>Surgical</td>
<td>15.6</td>
<td>7.7</td>
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<tr>
<td>11</td>
<td>Female</td>
<td>Device</td>
<td>32.5</td>
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<td>12</td>
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<tr>
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<td>14</td>
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<tr>
<td>15</td>
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<td>17</td>
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<td>Device</td>
<td>42.4</td>
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<tr>
<td>18</td>
<td>Female</td>
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<td>19</td>
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<td>21</td>
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<td>Device</td>
<td>52.2</td>
<td>5.2</td>
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<td>22</td>
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<td>Device</td>
<td>56.0</td>
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<tr>
<td>23</td>
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<td>Device</td>
<td>66.3</td>
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<td>24</td>
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<td>Device</td>
<td>57.3</td>
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<tr>
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<td>Female</td>
<td>Device</td>
<td>54.9</td>
<td>1.9</td>
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<tr>
<td>26</td>
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<td>Device</td>
<td>47.5</td>
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<tr>
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<td>Device</td>
<td>51.0</td>
<td>5.5</td>
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<tr>
<td>28</td>
<td>Female</td>
<td>Device</td>
<td>43.5</td>
<td>10.0</td>
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</table>

ASD indicates atrial septal defect.

Clinical and Echocardiographic Variables During Exercise

Maximal exercise capacity and peak systolic and diastolic blood pressures in patients with open and closed ASD were significantly lower compared with controls. Patients with a closed ASD had significantly lower peak heart rate compared with the control group (Table 2). Data obtained from standard echocardiography at rest and peak exercise are summarized in Table 4. Tricuspid annular systolic excursion and RV dimensions were higher in patients with an open ASD compared with patients with a closed ASD and controls.

Behavior of PVR During Exercise in the Control Group

Total PVR decreased significantly from rest to peak exercise (5.4±1.4 to 3.8±0.9 mm Hg/L per min; P<0.0001) (Figure 1A). There was a strong linear relationship between PAP and change in RV cardiac output (Figure 2). The slope of the PAP-flow plot was 3.0±0.8 mm Hg/L per min (Figure 1B, black line).

Behavior of PVR During Exercise in Patients With an Open ASD

In patients with an open ASD, total PVR did not change significantly from rest to peak exercise (4.0±1.4 to 4.4±1.1 mm Hg/L per min; P=0.050) (Figure 1A). Moreover, the slope of the PAP-flow plot was highly linear (Figure 1B, black line).
Behavior of PVR in Patients With a Closed ASD
In patients with a closed ASD, total PVR decreased significantly from rest to peak exercise (5.6 ± 2.1 to 4.7 ± 1.3 mm Hg/L per min; \( P = 0.009 \)) (Figure 1A). However, the slope of the PAP-plot was significantly higher compared with controls (4.0 ± 1.7 versus 3.0 ± 0.8 mm Hg/L per min; \( P = 0.016 \)) (Figure 1B, blue line). When reviewing the dataset in more detail, patients with an ASD closed at 34 years of age (median age at repair) presented with a significant decrease in total PVR from rest to peak exercise (5.5 ± 1.7 to 3.8 ± 1.1 mm Hg/L per min; \( P = 0.002 \)) and had a slope of the PAP-flow plot similar to controls (2.9 ± 1.3 mm Hg/L per min; \( P = 0.895 \)). In contrast, patients with an ASD closed ≥ 34 years of age did not present with a significant decrease in total PVR from rest to peak exercise (6.2 ± 2.3 to 5.6 ± 0.8 mm Hg/L per min; \( P = 0.414 \)) and had a higher slope of the PAP-flow plot compared with controls (5.1 ± 1.4 mm Hg/L per min; \( P < 0.0001 \)) (Figure 3A and 3B), which can be clearly appreciated after the Poon adjustment for intersubject variability (Figure 2).

Intra- and Interobserver Variability
There was good agreement in measurements of cardiac output (CO) and PAP between 2 blinded observers. Mean bias ± limits of agreement for CO measures were −6.0 ± 9.0% at rest and −0.7 ± 5.5% at peak exercise. Corresponding measures for PAP at rest were 7.7 ± 18.6% and at peak exercise were −3.6 ± 8.2%. The interobserver intraclass correlation coefficient was 0.981 (95% confidence interval [CI] 0.926 to 0.995) for CO at rest and 0.972 (95% CI 0.891 to 0.993) at peak exercise. The corresponding intraclass correlation coefficient for PAP at rest was 0.869 (95% CI 0.560 to 0.966) and at peak exercise was 0.937 (95% CI 0.768 to 0.984). Intraobserver variability for rest and peak measures combined were −2.9 ± 6.2% for CO and −0.5 ± 7.0% for PAP. Intraoberserver intraclass correlation coefficient for rest and peak measures combined was 0.991 (95% CI 0.964 to 0.998) for CO and 0.958 (95% CI 0.842 to 0.990).

Cardiopulmonary Exercise Test: Relationship Between Dynamic PVR and Peak VO₂
Peak oxygen consumption was measured by a separate exercise test. Peak VO₂ was 26.7 ± 10.6 mL/kg per min (79 ± 19% of the expected value) in patients with open ASD.
and 24.8±8.5 mL/kg per min (88±18% of the expected value) in patients with a closed ASD. There was no statistical difference between both groups in terms of expected peak VO₂ \((P=0.105)\). However, peak VO₂ correlated inversely with the slope of the PAP-flow plots in patients with open ASD \((R=-0.643; P=0.013)\) and patients with a closed ASD \((R=-0.529; P=0.005)\) (Figure 4).

**Discussion**

This study showed that in patients with an open ASD, total PVR did not decrease during exercise (in contrast to controls) and that the slope of the PAP-flow plots was higher than in controls. In patients with an ASD closed later in life (≥34 years of age), total PVR did not decrease during exercise and the slope of the PAP-flow plots was higher than in controls. The slope of the PAP-flow plot was related with peak VO₂ in patients with open and closed ASD.

Patients with ASD-type secundum present with a left-to-right shunt, causing volume overload of the right heart (with increased RV dimensions and higher tricuspid annular systolic excursion) and pulmonary circulation. The subsequent shear stress on the endothelium of the pulmonary vasculature may lead to the development of irreversible occlusive vascular lesions over a long period of time.\(^3\) Although the indication for late ASD closure has long been a matter of debate, several studies have shown that closure of the defect, even after the age of 40, reduces mortality and morbidity.\(^{20–22}\) It also has been demonstrated that, even in older patients, cardiac remodeling occurs after ASD closure, although pulmonary artery pressures often remain elevated.\(^{5,6,23–26}\)

**Behavior of PVR During Exercise in the Control Group**

In this study, total PVR decreased significantly (−41.2%) in controls from rest to peak exercise, indicating normal adaptation of the pulmonary vascular bed to increased RV CO during exercise. The pulmonary vascular bed is a low-resistance system that is capable of accommodating large amounts of blood flow at low levels of pulmonary artery pressure. However, a further decrease in PVR (generally 30% to 50% in humans), albeit more modest than the decrease in systemic vascular resistance, has been described during exercise.\(^2\) This decrease in PVR during exercise can be attributed to a combination of passive recruitment of perfusion and ventilation of the upper lung segments (with increasing pulmonary artery pressures and ventilation) and distension of the pulmonary vessels in combination with active reduction in pulmonary vascular tone. Because our patients were exercising in a semisupine position, ventilation and pressure distribution in the lung was distributed more equally, resulting in less pulmonary vascular recruitment during exercise. Consequently, the smaller expected decrease in PVR during semisupine exercise normally should be more related with vessel distension and changes in pulmonary vascular tone, with a complex interplay of vasoconstrictive, and vasodilator influences of endothelial and neurohumoral factors.\(^{27–29}\)

The slope of pressure-flow plots has been used to evaluate PVR in healthy individuals and specific patient populations.\(^{12,13}\) Moreover, the effect of specific pulmonary hypertension therapy on the slope of PAP-flow already has been evaluated successfully.\(^{30,31}\) Although the slope of a pressure-
flow plot represents a single value throughout exercise, ignoring a slightly curvilinear relationship, it is preferable to an isolated PVR measurement at rest. A complete evaluation of the pulmonary hemodynamics, including RV CO, pulmonary artery pressures, PVR, and left atrial pressures at each stage may provide useful pathophysiological information, but the slope of PAP-flow represents a simple and relatively accurate measure of PVR that integrates pulmonary artery pressure, CO, and total PVR and may prove useful for evaluating the effect of therapy affecting the pulmonary vasculature. Our data indicated that individuals in the control group were able to increase pulmonary blood flow 1 L/min with a 3 mm Hg increase in PAP.

Behavior of PVR During Exercise in Patients With an Open ASD
Patients with open ASD had a higher RV CO, but a similar PAP at rest when compared to patients with a closed ASD or controls, resulting in a lower total PVR at rest. Interestingly, they were unable to further decrease total PVR from rest to peak exercise. Because pulmonary artery pressure at peak exercise is determined by the change in CO, PVR, and left atrial pressures, the higher PAP at peak exercise observed in patients with an open ASD are most likely because of a combination of higher RV CO (mainly determined by a higher stroke volume) and the inability of the pulmonary vasculature to further decrease PVR, a phenomenon already described in competitive athletes. The lower PVR at rest probably represents an adaptation to increased RV CO in these patients. Interestingly, the synchronous rise in RV and LV CO from rest to peak exercise in patients with an open ASD suggests that the pressure gradient between left and right atrium persists during exercise.

In this study, the slope of the PAP-flow plot of patients with an open ASD was higher compared with controls, confirming the differential behavior of total PVR from rest to peak exercise. A steeper slope of the PAP-flow plot suggests an inability to lower PVR during exercise.

Behavior of PVR in Patients With a Closed ASD
Although more modestly, total PVR also decreased significantly from rest to peak exercise in patients with a closed ASD. However, when reviewing the data in more detail, this group consisted of patients who either had early or late defect closure. Patients who underwent ASD repair before age 34 years showed a significant decrease in total PVR, as controls did, which may indicate that early defect closure prevented the development of pulmonary vascular disease. However, patients who underwent late ASD closure showed no decrease in total PVR from rest to peak exercise. One could argue that the lack of decrease in total PVR in older patients may be caused by increased left heart filling pressures, but a
similar E/E' at peak exercise suggested that the difference is unlikely to be caused by decreased LV compliance and increased left atrial pressures. These results are consistent with an earlier study evaluating PVR during exercise after surgical ASD closure in patients already presenting with pulmonary hypertension before repair.\(^3\) The lack of decrease in PVR in patients with an ASD closed later in life in this study led to an exacerbated increase in PAPs during exercise, affecting exercise capacity, as has been described before.\(^3\)

This may be because of structural remodeling of the pulmonary vasculature with stiffening of the lung vessels\(^3\),\(^5\) whether or not in combination with an increase in pulmonary vascular tone.\(^3\),\(^6\)

Finally, the slope of the PAP-flow plots of patients with an ASD closed at later age was also higher compared with controls, indicating the inability to further decrease PVR during exercise.

Clinical Implications
This study indicated that patients who underwent ASD closure later in life were unable to decrease PVR during exercise in contrast to patients who underwent early ASD repair, despite both having normal PAP at rest. Indeed, age at repair is known as an independent predictor of late outcome,\(^2\) and late closure is associated with incomplete remodeling.\(^5\)–\(^7\)

Bicycle stress echocardiography may identify patients with altered pulmonary vascular hemodynamics that could not be detected at rest. A steeper PAP-flow plot may be related to limitations in exercise capacity as indicated by the relation with peak VO\(_2\). Because elevated pulmonary pressures before repair predicted late atrial arrhythmia in patients with ASD-type secundum,\(^4\) the current findings of steeper PAP-flow plots in patients with an ASD closed later in life raise the question whether lowering the slope of the PAP-flow plot in these patients would improve functional capacity and even outcome.

Limitations
It is important to underline a few limitations of the study. First, there was a sex difference between the control group and patients with a closed ASD. Female sex has been associated with elevated PAPs (but not with PVR) at rest and during exercise.\(^4\) However, the slope of PAP-flow plots has shown to be sex independent.\(^4\)

Moreover, none of the female patients included in the study had echocardiographic signs suggestive of diastolic dysfunction at rest and at peak exercise, making it less probable that left atrial pressures would behave differently between the subgroups during exercise.
exercise. Although still possible, diastolic dysfunction as a cause of elevated PAPs seems less plausible. Second, we used noninvasive echocardiographic parameters that have been validated against invasive measurements at rest and during exercise. However, the ratio of PAP to CO has not been validated invasively and does not include measurements of left and right atrial pressures. To enhance tricuspid regurgitation Doppler signals, we used agitated geloplasma. It also must be noted that behavior of left atrial pressure during exercise is controversial in the literature, even when using invasive measurements. Third, if the recovery of the ability to decrease PVR during exercise were a lengthy process, a bias may be present because the time interval between repair and stress testing was shorter in patients closed later in life.

Conclusions

In patients with an open ASD, the slope of the PAP-flow plots was higher and total PVR did not change from rest to peak exercise. In patients with an ASD closed later in life, the slopes of the PAP-flow plots were higher and total PVR did not change from rest to peak exercise, indicating altered pulmonary hemodynamics in these patients. Further research is needed to determine whether patients with steeper PAP-flow plots have worse outcome, and whether normalizing PVR during exercise, if possible, would change prognosis.

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Disclosures

None.

References


**CLINICAL PERSPECTIVE**

By measuring pulmonary artery pressures and cardiac output during bicycle stress echocardiography, a pulmonary artery pressure (PAP)-flow relationship can be constructed for each patient. The slope of the PAP-flow plot represents the response of the pulmonary vasculature to increased flow and may identify patients with altered pulmonary hemodynamics which would have been missed at rest. This study showed steeper PAP-flow plots in patients with an atrial septal defect closed later in life, indicating that longstanding volume overload of the pulmonary circulation causes subclinical pulmonary vascular lesions with a decrease in cross-sectional area of the pulmonary vessels. The relation between the slope of the PAP-flow plot and peak oxygen consumption raises the question whether lowering the slope of the PAP-flow plot would increase exercise capacity in these patients.
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