Evaluation of Right Ventricular Systolic Pressure During Incremental Exercise by Doppler Echocardiography in Adults With Atrial Septal Defect*

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Study objectives: Pulmonary hypertension is the most important complication in patients with atrial septal defect (ASD), but its role in limiting exercise has not been examined. This study sought to evaluate exercise performance in adults with ASD and determine the contribution of elevated pulmonary artery pressure in limiting exercise capacity.

Design: We used Doppler echocardiography during exercise in 10 adults (aged 34 to 70 years) with isolated ASD (New York Heart Association class I, II) and an equal number of matched control subjects. Incremental exercise was performed on an electrically braked upright cycle ergometer. Expired gases and Ve were measured breath-by-breath. Two-dimensional and Doppler echocardiographic images were obtained at rest prior to exercise to determine ASD size, stroke volume (SV), shunt ratio (Qp:Qs), right ventricular outflow tract (RVOT) size, and right ventricular systolic pressure at rest (RVSPr). Doppler echocardiography was repeated at peak exercise to measure right ventricular systolic pressure during exercise (RVSPEx).

Results: Resting echocardiography revealed that RVOT was larger (21±4 vs 35±8 mm, mean±SD; p=0.0009) and RVSPr tended to be higher (17±8 vs 31±8 mm Hg; p=0.08) in ASD; however, left ventricular SV was not different (64±23 vs 58±23 mL; p>0.05), compared with control subjects. Despite normal resting left ventricular function, ASD patients had a significant reduction in maximum oxygen uptake (Vo2max) (22.9±5.4 vs 17.3±4.2 mL/kg/min; p=0.005). RVSPEx was higher (19±8 vs 51±10 mm Hg; p=0.001) and the mean RVSPEx-Vo2 slope (1±2 vs 18±3 mm Hg/L/min; p=0.003) and intercept (17±4 vs 27±4 mm Hg; p=0.05) were higher in the ASD group. Vo2max correlated inversely with both RVSPr (r=-0.69; p=0.007) and RVSPEx (r=-0.67; p=0.01).

Conclusion: These findings suggest that adults with ASD have reduced exercise performance, which may be associated with an abnormal increase in pulmonary artery pressure during exercise.

CHEST 1998; 113:1459-65

Key words: atrial septal defect; echocardiography; exercise; pulmonary hypertension

Abbreviations: ASD=atrial septal defect; BR=breathing reserve; CI=confidence interval; HR=heart rate; LVOT=left ventricular outflow tract; MPA=main pulmonary artery; NYHA=New York Heart Association; Qp=pulmonary cardiac output; Qp:Qs=shunt ratio; Qs=systemic cardiac output; RA=right atrial; RVOT=right ventricular outflow tract; RVSPr=right ventricular systolic pressure; RVSPEx=right ventricular systolic pressure during exercise; RVSP=right ventricular systolic pressure at rest; SaO2=arterial saturation of oxygen; SV=systemic stroke volume; TR=tricuspid regurgitation; VCO2=carbon dioxide output; Ve=minute ventilation; Vo2=oxyegn uptake; Vo2max=maximum oxygen uptake

Isolated atrial septal defect (ASD) is the second most frequently encountered form of congenital heart disease after childhood, accounting for approximately 22% of cases.1 Although some patients with uncorrected ASD survive to an advanced age,2,3 overall life expectancy is thought to be shortened.4

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Pulmonary hypertension, the most important complication, is associated with increased morbidity and mortality.5,7 At present, a majority of adults with ASD are advised to undergo surgical repair before the onset of pulmonary hypertension in order to
increase longevity and limit the deterioration of functional capacity. Once pulmonary hypertension develops, irreversible right ventricular failure may result. Detecting pulmonary hypertension during exercise, before the onset of sustained resting pulmonary hypertension, may be of value when considering operative repair in adults with ASD.

Doppler echocardiography provides an accurate noninvasive approach to determine right ventricular systolic pressure (RVSP) at rest and has been validated against direct measurements of pulmonary systolic pressure during exercise. This technique has not been utilized in this patient population, however. We conducted a prospective, controlled study to determine if adults with uncorrected ASD develop pulmonary hypertension during exercise, and to examine the importance of elevated pulmonary artery pressure during exercise in limiting overall performance.

**Materials and Methods**

**Study Population**

Ten patients with isolated ASD (New York Heart Association [NYHA] functional class I, II), with a mean age of 52.9±11.2 years (range, 34 to 70 years), and an equal number of matched healthy control subjects gave informed consent to participate in the study. All study subjects (patients and controls) were selected from the echocardiographic laboratory database (UpBeat Systems Inc; Montreal, Canada) of the Sir Mortimer B. Davis-Jewish General Hospital. Between September 1989 and April 1993, 10,725 patients were enrolled in this database. From this group, 63 individuals with ASD were identified. Twenty patients who had previously undergone surgical repair were excluded. An additional 17 patients were excluded because of known coronary artery disease, other valvular cardiac abnormalities, or vascular, orthopedic, neurologic, or other systemic disease; seven patients were excluded because of age >70 years, and six were classified as NYHA class III or IV. Of the 13 remaining ASD patients who fulfilled all entry criteria, 10 agreed to participate in the study. Ten consecutive patients from this database who had a normal echocardiogram, no clinical history of cardiopulmonary disease, and matched ASD patient characteristics with respect to age, sex, height, and smoking history, were recruited as control subjects. None of the ASD patients or controls had any evidence of pulmonary stenosis by echocardiography. This study was approved by our institutional review board for human studies.

Height and weight were measured, and resting spirometry (DS 560; Collins; Braintree, Mass) performed on all study participants within two weeks of the exercise study.

**Exercise Protocol**

Exercise studies were performed at the Sir Mortimer B. Davis-Jewish General Hospital Cardiopulmonary Exercise Laboratory. Subjects were seated on an upright cycle ergometer (CPE 2000; Medical Graphics Corp; St. Paul, Minn). A mouthpiece and noseclips were placed and a 12-lead ECG was performed.

Ve, VO2, and VCO2 were measured breath by breath using a commercially available metabolic cart (Cardio II System; Medical Graphics Corp). The pneumotachograph was calibrated using a 3-L syringe at five different flow rates, with errors of ±2% accepted. A zirconia cell O2 analyzer and single-beam infrared CO2 analyzer were calibrated with room air and a 5% CO2/12% O2 gas. The phase delay between volume and expired gas fraction measurements was assessed with the wave form analyzer to ensure a correct cross integral.

Heart rate (HR) and BP were measured at rest and during each minute of exercise. A pulse oximeter (Si Med S100c; Bothell, Wash) with continuous readout was placed on an index finger. The lowest value for arterial saturation of oxygen (SaO2) during each minute of exercise was recorded. A 12-lead ECG was performed each minute during exercise.

All patients and control subjects exercised on room air. A 5-min period of rest preceded incremental exercise until exhaustion on a continuous ramp at a rate of 10 W/min.

**Echocardiography**

Supine resting and upright resting and exercise echocardiographic studies were performed on all subjects with an ultrasonograph (Hewlett-Packard 1500; Hewlett-Packard; Andover, Mass), equipped with a standard imaging 2.5-MHz probe. Before exercise, a complete resting echocardiographic study was performed in the supine position in all standard views to determine cardiac chamber and great vessel dimensions, cardiac output, and shunt ratios. All echocardiograms were recorded on high-quality VHS videocassettes and interpreted blindly, without knowledge of patient identity. The subjects were positioned in the left lateral decubitus position for parasternal and apical views and dorsal decubitus for subcostal images. Images were obtained during held indinopression to enhance the definition of the right ventricular outflow tract (RVOT) and main pulmonary artery (MPA).

Cardiac dimensions were obtained by parasternal long-axis M-mode echocardiography, according to the American Society of Echocardiography standards. These dimensions included left ventricular end-diastolic diameter, interventricular septal wall and posterior wall thickness, right ventricular outlet diastolic diameter, and left ventricular end-systolic diameter. Right ventricular inlet diameter was obtained by the apical four-chamber view, just below the tricuspid valve leaflet insertion. Atrial septal defect dimensions were calculated by color flow imaging in the subcostal views. Each parameter was measured off-line in triplicate and averaged.

High resolution images of the left ventricular outflow tract (LVOT), immediately below the insertion of the aortic valve leaflets were obtained by freeze-frame two-dimensional echocardiography in the left parasternal window. The LVOT area was calculated as d^2·0.785, where d is the LVOT diameter. Using the same calculations, the RVOT area and proximal MPA area were measured, respectively, immediately below the pulmonic valve leaflet insertion and distal to the pulmonic valve leaflets. The pulsed Doppler cursor was carefully positioned parallel to the RVOT and MPA to measure flow velocity time integral at both sites and obtain the right ventricular stroke volume (SV). The pulmonary cardiac output (Qp) was calculated as the average of SV obtained at the RVOT and the MPA, multiplied by the resting HR for each patient. The LVOT flow was measured by the apical four-chamber view, immediately below the aortic valve leaflets. The systemic cardiac output (Qs) was obtained by multiplying the LVOT area by the LVOT velocity time integral and HR. For patients in sinus rhythm, each parameter was measured in triplicate and averaged; for patients with atrial fibrillation, seven measurements were averaged.

The RVSP was calculated from the tricuspid regurgitation (TR) jet obtained under color flow imaging guidance. In all patients,
the best TR Doppler signal quality was obtained from the apical or modified low left parasternal four-chamber view. The chest was carefully marked with a felt-tip pen at the site providing the best TR Doppler signal for quick localization during exercise testing. In the patients with atrial fibrillation, the largest and most complete pansystolic TR signals were considered for analysis. Technically adequate TR Doppler signals were obtained at rest in eight patients and six control subjects, and during exercise in seven patients and seven control subjects; complete rest and exercise data were available for six patients and six control subjects. Using the simplified Bernoulli formula (peak gradient=4V², where V is the peak TR velocity into the right atrium), the RVSP was obtained by adding the peak TR gradient to the estimated right atrial (RA) pressure. RA pressure was estimated at rest by the response of the inferior vena cava to deep inspiration and was assumed to be constant throughout exercise. With the trailing-edge to leading-edge technique, maximum inferior vena cava diameters before inspiration and minimum diameters after inspiration were measured in the subcostal view within 2 cm of the entrance to the right atrium. When the diameter of the inferior vena cava decreased by less than 50% after deep inspiration, RA pressure was defined as 15 mm Hg; when the diameter decreased by more than 50%, RA pressure was defined as 5 mm Hg. All patients and control subjects showed more than a 50% decrease in vena cava diameter at rest. This technique, which assumes that the estimated RA pressure remains constant during exercise, has been validated against simultaneous measurements of pulmonary artery systolic pressure during incremental exercise.

Data Analysis

Resting ventilatory and gas exchange data were averaged over the last 2 min of rest. During exercise, they were averaged over contiguous 30-s intervals. VE, V̇O₂, and V̇CO₂ were calculated from standard formulas. V̇O₂max was defined as the highest V̇O₂ measured during the last minute of the symptom-limited exercise test. The ventilatory threshold was defined as the VO₂ corresponding to the inflection point of the V̇CO₂ vs V̇O₂ plot or V-slope method, and was determined by two blinded physicians experienced in exercise testing. Predicted maximum HR was estimated as 220–[age in years]. The breathing reserve (BR) was calculated as the difference between maximum voluntary ventilation and maximum VE. A pulmonary mechanical limit to exercise was defined as a BR <11 L/min. A significant O₂ desaturation was defined by a decrease in SaO₂ ≥4%. Predicted values for VO₂ max and normal values for the BR were those of Hansen et al. A significant left-to-right shunt was defined as Qp:Qs ≤1.5. All values are expressed as mean±SD. Discrete rest and exercise variables were compared using Student’s two-tailed t test. Continuous data were analyzed by simple linear regression. Computations were made with the Statview 4.0 statistical program (Abacus Concepts; Berkeley, Calif). A p value of ≥0.05 was considered significant.

**RESULTS**

**Baseline Characteristics**

The baseline data for ASD patients and control subjects are presented in Table 1. A secundum-type ASD was present in six, a sinus venosus-type ASD in three, and a primum-type ASD in one. Seven ASD patients were in functional class I (NYHA), and three were in NYHA class II. No differences were found in age and height, however, weight was higher and FEV₁ tended to be lower in the ASD group. Two ASD patients had chronic atrial fibrillation; one of them was being treated with a β-blocker, while the other was receiving nifedipine. A third ASD patient was receiving prazosin for the treatment of hypertension. All of the other patients and control subjects were in normal sinus rhythm, and none were receiving β-blockers, calcium-channel blockers, digitalis, or other systemic vasodilators.

Baseline echocardiography demonstrated an increase in right ventricular size in the ASD group, and a significant left-to-right shunt (Qp:Qs≤1.5) in all but one of the ASD patients. Despite these abnormalities, SV was no different from that of control subjects (64±23 vs 55±23 mL; p>0.05), suggesting normal resting left ventricular function.

**Incremental Exercise**

V̇O₂max was reduced (22.9±5.4 vs 17.3±4.2 mL/min/kg; p=0.005) in ASD patients (Table 2). No subject in either group reached a pulmonary mechanical limit to exercise (BR <11 L/min) or sustained a drop in SaO₂ during exercise. In addition, no ischemic ECG changes or new arrhythmias occurred during exercise. Maximum HR tended to be lower in ASD patients, but this difference did not reach statistical significance. The trend remained after eliminating the ASD patient receiving a β-blocker (163±8 vs 144±23 beats/min; p=0.12). A ventilatory threshold was identified in all 10 control subjects and in nine ASD patients, but was not different in the two groups.

RVSP tended to be increased at rest, and was markedly higher at peak exercise in ASD patients (Tables 1 and 2). The mean RVSP-V̇O₂ slope (1±2 vs 18±3 mm Hg/L/min; p=0.003) and intercept (17±4

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<th>Table 1—Baseline Characteristics*</th>
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<td><strong>Control Subjects</strong></td>
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<td>RVSP, mm Hg</td>
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*p<0.05 compared with control subjects.

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*RVOT, SV, and Qs data not obtained for one control subject. RVSPr data not obtained for four control subjects and two patients. RVSPr=resting right ventricular systolic pressure.
vs 27±4 mm Hg; p=0.05) were higher in the ASD group (Fig 1). In all subjects, an inverse correlation was noted between VO$_2$max and RVSP at both rest (r=-0.69; p=0.007; Fig 2) and peak exercise (r=-0.67; p=0.01; Fig 3), but VO$_2$max did not correlate with shunt ratio, RVOT, ASD size, or maximum HR.

**DISCUSSION**

In the current investigation, 10 minimally symptomatic adults with ASD (NYHA class I, II) and normal resting left ventricular function demonstrated a significant reduction in VO$_2$max during incremental exercise compared with matched controls. RVSP, measured noninvasively by exercise Doppler echocardiography, tended to be increased at rest and rose inappropriately during exercise in patients with ASD, whereas no significant rise in RVSP occurred in control subjects. Peak RVSP during exercise (RVSPex) was inversely correlated with VO$_2$max in all patients. These findings suggest that (1) functional capacity is overestimated by subjective measures in this group; and (2) reduced exercise tolerance may be associated with an abnormal rise in pulmonary artery pressure during exercise.

Reports characterizing the physiologic response to exercise in adults with ASD are few, and they have mainly focused on postoperative patients. When compared with matched healthy control subjects, asymptomatic children with ASD may have significantly diminished maximal exercise endurance. In adults with corrected ASD, several studies have shown that exercise capacity remains normal if surgery was performed at an early age; however, a blunted HR response during exercise was found in some. If surgery is delayed beyond early childhood, most reports show exercise performance to be reduced; surgical repair may permit some improvement, however.

**Table 2—Exercise Results**

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<th>Control Subjects</th>
<th>ASD Patients</th>
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<tr>
<td>VO$_2$max, mL/min/kg</td>
<td>22.9±5.4</td>
<td>17.3±4.2</td>
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<tr>
<td>(p predicted)</td>
<td>(87±20)</td>
<td>(75±13)</td>
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<td>VT, mL/min/kg</td>
<td>11.7±2.4</td>
<td>11.5±1.7</td>
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<tr>
<td>(p predicted VO$_2$max)</td>
<td>(44±7)</td>
<td>(48±5)</td>
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<tr>
<td>Maximum HR, beats/min</td>
<td>158±9</td>
<td>147±20</td>
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<tr>
<td>(p predicted)</td>
<td>(95±15)</td>
<td>(87±17)</td>
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<tr>
<td>BR, L/min</td>
<td>58±17</td>
<td>53±14</td>
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<tr>
<td>Peak exercise SaO$_2$,</td>
<td>98±1</td>
<td>98±2</td>
</tr>
<tr>
<td>%</td>
<td></td>
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<tr>
<td>RVSPex, mm Hg</td>
<td>19±8</td>
<td>51±10</td>
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*A ventilatory threshold could not be determined for one ASD patient. Maximum HR not included for two ASD patients with atrial fibrillation during exercise. RVSPex was not obtained for four control subjects and three patients. VT=ventilatory threshold.*

$p<0.05$ compared with control subjects.

**Figure 1.** Plot of RVSP vs VO$_2$ at rest and peak exercise using individual data. ASD group represented by closed circles; control group by open circles. Complete data available for six patients and six control subjects. The mean RVSP-VO$_2$ slope (1±2 vs 18±3 mm Hg/L/min; p=0.003) and intercept (17±4 vs 27±4 mm Hg; p=0.05) were higher in the ASD group.

**Figure 2.** Curve fit of VO$_2$max vs right ventricular systolic pressure at rest (RVSPr) in all study subjects. ASD patients represented by closed circles; control group by open circles. Data not obtained for two patients and four control subjects. Slope=-0.4 mL/min/kg/mm Hg (95% confidence interval [CI], -0.6 to -0.1). Intercept=30.0 mL/min/kg (95% CI, 23.2 to 36.8). Correlation coefficient=-0.69 (p=0.007).
tests were performed on seven. Although preoperative VO2max was higher than in our group (23.2±1.9 vs 17.9±4.4 mL/min/kg), a 23% increase in VO2max occurred in six of seven patients after surgery.26

Both a chronotropic abnormality and pulmonary hypertension may have contributed to the exercise limitation in our patients. Although ASD patients were heavier than control subjects, deconditioning probably did not contribute to the reduction in VO2max since no difference in the ventilatory threshold was found between groups.28 Maximum HR tended to be lower in ASD patients, but this did not reach statistical significance. As mentioned previously, a blunted HR response to exercise has been described in patients with ASD.23,24 However, the mechanisms remain ill-defined. No correlation was identified between peak exercise HR and VO2max, suggesting that a chronotropic abnormality, if present, is probably not the major determinant of overall exercise performance. In two older studies of adults with uncorrected ASD,20,30 pulmonary vascular resistance appeared to be an important determinant of cardiac function during exercise. More recently, Hirata et al31 used radionuclide ventriculography during exercise in seven patients with ASD (mean age, 40±17 years) to demonstrate an association between reduced right ventricular ejection fraction during exercise and elevated resting pulmonary vascular resistance. Although suggestive of a pulmonary vascular limit to exercise, the submaximal exercise protocols used in these studies precluded a link with VO2max.

To our knowledge, the current investigation is the first report in which the abnormal elevation in pulmonary artery pressure during exercise was related to a reduction in VO2max in adults with uncorrected ASD. The absence of oxygen desaturation at peak exercise excludes shunt reversal as a mechanism limiting exercise in this group. This suggests that the abnormal rise in pulmonary artery pressure limited exercise by an inability to adequately augment systemic cardiac output. Although we were unable to simultaneously measure pulmonary artery pressure and systemic cardiac output during exercise using Doppler echocardiography, abnormal left ventricular performance in association with right heart dysfunction during exercise in ASD has been reported previously.32 Three possible mechanisms may have contributed to an abnormality in left ventricular performance during exercise: (1) reduced pulmonary venous return due to a poorly recruitable pulmonary circulation incapable of handling the large increase in blood flow demanded by exercise; (2) reduced blood flow from the left atrium to left ventricle because of augmented left-to-right interatrial shunting; and/or (3) interventricular septal shift due to right ventricular volume overload. Although all these mechanisms would limit systemic cardiac output by underfilling the left ventricle, one would expect left ventricular end-diastolic pressure to be reduced, unless the latter mechanism predominated.

At rest, reduced systemic cardiac output in ASD patients has been noted in association with either normal33 or below-normal values for left ventricular end-diastolic pressure;32 during exercise, however, left ventricular end-diastolic pressure was elevated in some individuals.32 In a study using radionuclide cineangiography at rest and during exercise in 11 patients with ASD before and after surgical repair, an improvement in postoperative left ventricular function appeared to be related to a reduction in right ventricular volume overload and improved interventricular septal motion.27 It remains unknown whether the improvement in septal motion caused the left ventricle to function better or simply occurred as a consequence of improved left ventricular filling postoperatively. Clearly, more work is needed to elucidate the precise mechanisms that limit exercise in this patient population.

The importance of finding abnormal and possibly limiting elevations in pulmonary artery pressure during exercise in ASD lies in the decision regarding the timing of surgical closure. In young individuals with uncomplicated ASD, operative closure is usually recommended regardless of symptoms, because surgical mortality is low, medical complications can be obviated,25 and long-term survival returns to that of an age-matched control population.7 In older
patients, especially those older than 40 years at the time of surgical repair, the prognosis appears to be less favorable, with higher operative mortality7,8,34 and an increased incidence of late cardiac failure, stroke, and atrial fibrillation.7 However, long-term survival in patients over 40 years of age appears to be improved and the decline in functional status reduced more by operative repair than by medical management.8 In an older patient with severe pulmonary hypertension, the operative risks are higher and long-term outcome may not be improved.6,9 Once pulmonary hypertension is established, it may progress after surgery.35 However, a subgroup of affected patients may still be candidates for surgery if favorable pathologic changes are identified36 or if pulmonary vasodilatory capacity can be demonstrated.37 Once irreversible pulmonary hypertension has developed, isolated ASD repair may no longer be possible without concomitant lung transplantation.

Therefore, early detection of exercise-induced pulmonary hypertension can guide the decision about surgical repair. Until recently, the measurement of pulmonary artery pressure during exercise required the invasive method of right heart catheterization. In the absence of pulmonic valve stenosis, the measurement of RVSP by Doppler echocardiography can be used to closely approximate pulmonary artery systolic pressure at rest.9 This technique has also been validated during incremental exercise by studies with simultaneous hemodynamic monitoring.10,11 In fact, in 10 patients with chronic lung disease, Himelman et al.10 showed a very tight correlation (r=0.98; p<0.0001) between RVSP assessed by Doppler echocardiography and pulmonary artery systolic pressure measured by pulmonary artery flotation catheter at rest and exercise. Although Doppler echocardiography results were not verified by simultaneous hemodynamic monitoring in the current study, the methods used were identical to those of Himelman et al.,10 with the exception of saline enhancement. In addition, RVSP measurements for our control group, which showed small changes from rest to upright exercise, are consistent with prior reports,38-40 adding further support for the methods used in this study. We have shown that a technically adequate noninvasive assessment of RVSP during exercise can be made in a majority of patients with ASD. Saline contrast enhancement of the tricuspid insufficiency might have permitted determination of RVSP in a higher percentage of patients, however.

In conclusion, this study demonstrates that minimally symptomatic adults with isolated ASD have reduced exercise tolerance and abnormally high increases in pulmonary artery pressure with exercise, which may be causally linked. The detection of pulmonary hypertension during exercise in patients with ASD can be obtained by noninvasive Doppler echocardiography, and should be considered in all patients being evaluated for surgical repair. A larger, long-term prospective study with both pre- and postoperative exercise testing would likely shed more light on the appropriate indications and timing of surgical closure in adults with ASD, and could determine the value of exercise testing in ASD patients prior to surgical repair.

ACKNOWLEDGMENTS: The authors are indebted to Pierre Kupfer, William Klebansky, and Rosemary Mattosco for their technical assistance in the pulmonary function laboratory at S.M.B.D.-Jewish General Hospital. The authors also wish to thank Carole Daoust, RDCC, for her technical assistance.

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