INTRODUCTION
Secundum atrial septal defect (ASD), the third most common congenital cardiac pathology in adults, after bicuspid aortic valve and mitral valve prolapse constitutes 9-10% of congenital heart diseases and is more frequently observed in women than men.1 Although patients with ASD can only live up to 35-49 years with medical treatment, asymptomatic patients who live up to 80 years are also reported.2 But long life is disputable in patients older than 45 years who are operated. 3 Although most of the ASD patients in their twenties are asymptomatic, a decrease in effort tolerance can occur in thirties and forties related to right heart deficiency and arrhythmia. It is beneficial to perform the procedure before these complications develop. Surgical treatment decreases pulmonary arterial pressure and prevents the development of right heart deficiency by removing pressure overload on right.

Atrial septal defect repair; our early and mid-phase results

Sedat Ozcan1, Ali Ümit Yener2, M. Turgut Alper Ozkan3

ABSTRACT
Objective: Atrial septal defect is one of the most commonly encountered congenital heart diseases in adults. The effect of age of the patient to the surgery is disputable. The purpose of this report was to evaluate surgical repair in patients with ASD who are operated in our clinic.

Methods: Total 40 patients were subjected to surgical repair due to ASD in Van Yuksek Ihtisas Education and Research Hospital between February 2006 and April 2009. Twenty seven of the patients were female and 13 were male, their ages differed between 8 and 71 and mean age of the patients was 33.70±14.04.

Result: Operative mortality did not occur. Two of our patients had coronary arterial disease in addition to ASD. ASD repair was performed together with coronary bypass surgery. Closing of ASD resulted in an increase in left ventricular ejection fraction, and a decrease in pulmonary arterial pressure and cardiothoracic ratio. Recovery in the functional capacity was observed post-surgery according to NYHA.

Conclusion: In this series, surgical results of the patients of various ages, with ASD closed were positive.

KEY WORDS: Atrial Septal Defect, Age, Surgical Repair, Adult Congenital Heart Disease.

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INTRODUCTION
Secundum atrial septal defect (ASD), the third most common congenital cardiac pathology in adults, after bicuspid aortic valve and mitral valve prolapse constitutes 9-10% of congenital heart diseases and is more frequently observed in women than men.1 Although patients with ASD can only live up to 35-49 years with medical treatment, asymptomatic patients who live up to 80 years are also reported.2 But long life is disputable in patients older than 45 years who are operated. 3 Although most of the ASD patients in their twenties are asymptomatic, a decrease in effort tolerance can occur in thirties and forties related to right heart deficiency and arrhythmia. It is beneficial to perform the procedure before these complications develop. Surgical treatment decreases pulmonary arterial pressure and prevents the development of right heart deficiency by removing pressure overload on right.

Although ASD can be closed with percutaneous transcatheter procedure, surgery may be required due to complications such as residual shunt, dislocation and vascular complications. Mortality of the surgery differs between 1-2% and post-surgery recurrence is below 2%.4

METHODS
Forty patients were operated in Van Yuksek Ihtisas Education Research Hospital, due to ASD
between February 2006 and April 2009. After pre-surgery physical examination, routine check-ups and echocardiography evaluations of patients, right heart catheterization was performed. Coronary angiography was also performed to patients older than 50 years in order to determine whether they had coronary arterial disease. The ratio of pulmonary flow to systemic flow was higher than 1.5 in all the operated patients.

Perfusion was started by performing aortic and bicaval venous cannulation after median sternotomy. Following cross clamp, diastolic cardiac arrest was induced by administration of antegrade cardioplegia and myocardium was protected by repeating this every 20-25 minutes. Vent cannula was inserted through right superior pulmonary vein for left heart decompression. Right atriotomy was performed by tightening snares around caval cannula and applying moderate hypothermia (28-32°C). Pericardial patch was preferred in cases where patch was required.

Pericardium was used after it became rigid by soaking it for 20 minutes in specially prepared 0.6% glutaraldehyde solution. Patients were followed between 3 months and 2 years (Mean 15 months). Pre- and post-surgery New York Heart Association (NYHA) functional classification, cardiothoracic rates and pulmonary arterial pressures were compared. Student t-test and chi-square test were used for statistical analysis. P<0.005 value was accepted as significant.

**RESULTS**

Twenty seven of our patients were female and 13 were male, their ages were between 8 and 71 and mean age was 33.70±14.04 (Table-I). Main complaints of our patients were difficulty in breathing, tachycardia and chest pain and there were no symptoms in three patients. Atrial fibrillation (AF) was present in the ECG of two patients older than 50 years. Pulmonary venous return anomaly was present in three patients with sinus venosus type defect. Coronary arterial disease was detected by coronary angiography of 58 and 63 years old patients. ASD closure and coronary bypass was performed in the same session (LIMA-LAD) to both. The ratio of pulmonary blood flow to systemic blood flow varied between 1.5 and 3.6 and mean shunt ratio was 2.1±0.5. The size of atrial septal defects varied between 1.7 cm and 4.3 cm and mean defect size was 2.5±0.6 cm.

While the repair was performed with patch graft in 8 patients, primary repair was performed in 32 patients. Also Devega or Kay annuloplasty was performed in three patients with advanced tricuspid insufficiency. Total perfusion duration of patients varied between 28 and 75 minutes and mean duration was 41.17±4.5 minutes; and cross-clamp durations varied between 14 and 58 minutes and mean duration was 23.78±10.3.

In post-operative period an increase in EF occurred in all of our patients. While pre-operative functional capacity mean class (NYHA) was 2.3±0.4, post-operative value was 1.2±0.3 and it was statistically significant (P<0.05). Significant recovery occurred in NYHA Functional capacity and pulmonary arterial pressure of our patients in post-operative period (P<0.05) (Table-II).

**DISCUSSION**

Although growth retardation in patients with atrial septal defect draws attention, their early childhood period generally passes without symptoms. But in medium and large defects, symptoms such as effort intolerance can start in

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### Table-I: Patient distribution according to age groups.

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>No. of patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>8-15</td>
<td>4</td>
<td>10</td>
</tr>
<tr>
<td>16-20</td>
<td>6</td>
<td>15</td>
</tr>
<tr>
<td>21-30</td>
<td>5</td>
<td>12.5</td>
</tr>
<tr>
<td>31-40</td>
<td>8</td>
<td>20</td>
</tr>
<tr>
<td>41-50</td>
<td>9</td>
<td>22.5</td>
</tr>
<tr>
<td>50 and above</td>
<td>8</td>
<td>20</td>
</tr>
</tbody>
</table>

### Table-II: Comparison of pre- and post-surgery functional capacity, pulmonary arterial pressure and cardiothoracic rate.

<table>
<thead>
<tr>
<th></th>
<th>Pre-operative value</th>
<th>Post-operative value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean NYHA</td>
<td>2.3±0.4</td>
<td>1.2±0.3</td>
</tr>
<tr>
<td>Functional capacity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean pulmonary arterial pressure (mmHg)</td>
<td>49.75±14.12</td>
<td>34.2±10.9</td>
</tr>
<tr>
<td>Mean cardiothoracic rate (%)</td>
<td>68±6</td>
<td>54±4</td>
</tr>
</tbody>
</table>
childhood and become evident again in twenties; and other symptoms start in thirties. Cardiac insufficiency rarely encountered in infancy and early childhood is more frequent after the age of 45 years. Deaths in patients with ASD result generally from right ventricular insufficiency or tachycardia. Left ventricular insufficiency can also develop related to right ventricular dysfunction and can be one of the causes for mortality. All ASD patients with a shunt ratio of 1.5 or over should be operated to prevent complications like pulmonary hypertension and right ventricular insufficiency related to volume overload.

Defects in interatrial septum are defined as primum or secundum depending on the region. Primum type defects are included in the present day in another anatomopathologic classification as incomplete atioventricular canal defect.6,7 Whatever the anatomical localization of ASD may be in interatrial septum, its physiological effects originates from flow of blood from left atrium to right atrium. Relative compliances of two ventricles rather than the size of ASD are important on the size of shunt. Shunt is minimal during infancy because both ventricles are hypertrophied and relatively noncompliant. As the compliance of right ventricle will be relatively more than the left ventricle with the child grows, shunt will grow towards right.

Increase of left to right shunt in patients with ASD results probably in an increase in right ventricular dysfunction and troponin levels. Because right ventricular dysfunction and resultant subacute myocardial necrosis may develop in patients with ASD; even elderly ASD patients should be operated.8 The prevalence of AF in ASD patients is strongly related to the level of mitral and tricuspid regurgitation.

Mitral regurgitation is more frequent in ostium primum type ASD patients. But it can also be found in ostium secundum and sinus venosus type defects. Mitral insufficiency increases in patients with ASD due to age related deterioration in geometry of left ventricle, shortening in chord, fibrous and myxomatous degeneration.9

The relationship between tricuspid insufficiency and ASD is not as strong as the relationship between mitral insufficiency and ASD. But in adult patients with ASD, this condition might not be seen. Moderate or serious tricuspid regurgitation may be found in one fourth of the patients. This can be related to abnormal leaflet structure or myxomatous degeneration. But it is most frequently related to annular dilatation or hemodynamic disorder of right ventricle. Mitral or tricuspid valve insufficiency should be repaired while ASD is closed in order to decrease development of late AF that might occur as a result of these.9

Pulmonary vascular disease develops at last in 20-25% of the children with ASD. If it doesn't develop until the age of 20, the probability of development later is lower. While right ventricular dysfunction and tricuspid insufficiency develops in elderly patients, degree of left to right shunt may decrease. But in some of the patients degree of left to right shunt may increase due to decrease in hypertension and left ventricular compliance.

Post-operative complications increase in patients with repaired ASD over the age of 50 years. Complications developed generally in ASD repairs performed in children and young adults because chronic AF causes changes in atrial myocardium, persistence of chronic AF post-surgery should not be regarded as a surprise as in two patients in our series.11

The prevalence of AF is related to advanced age. left atrial expansion and mitral and tricuspid insufficiency degree. Gender, anatomical type, defect size, pulmonary and systemic flow ratio, pulmonary arterial pressure, right ventricle size and ventricle systolic function do not have any relationship with late phase developed AF.9

Sinus venous type ASDs forms 9% of all ASDs and frequently progress with the abnormal return of right pulmonary veins as in three patients in our series. Sinus venous type ASD, together with other congenital heart diseases are seen more frequently and are differentiated by high pulmonary pressure and resistance. It is reported that closure of sinus venous type ASDs with pericardial patch via lateral cavotomy decreases post-operative sinus node dysfunction and constriction of pulmonary vein and caval vein.12

Reasons that worsen the natural course and increase the risk of mortality in patients with ASD are congestive cardiac insufficiency caused by left ventricular dysfunction, recurrent pulmonary and paradoxical emboli, arrhythmia and recurrent pulmonary infections. Pulmonary vascular disease ultimately causes shunt to reverse and hypoxia to develop. This reversal of shunt may be discontinuous depending on the tricuspid regurgitation and right ventricle function.

In ASD patients with large shunts, end-diastolic compliance of left ventricle tends to decrease. Volume increase in right ventricle due to shunt, destroys left ventricle dilatation. Shunt flow from
left atrium to right atrium is seen from mid systole to early diastole and during atrial contraction. Right ventricle volume increase might be higher than left ventricle volume increase during early diastole and atrial contraction.13

Left ventricle dysfunction in secundum type ASD patients is seen especially over age of 50 years. The reason of this is hypoplastic state of left ventricle due to filling deficiency and the decrease in compliance.12

Age for elective closure of ASD is 4-5 years; there isn't any benefit for operating earlier. But postponing the closure to advanced ages causes atrial arrhythmia due to irreversible changes caused by long term volume load on right atrium and right ventricle. IF cardiac insufficiency secondary to mitral valve insufficiency is present in primum type ASD patients, an earlier operation is required.5

As in our series surgical closure of ASD can be successfully performed at every age if irreversible changes do not develop in pulmonary vascular bed. Following surgery a clear recovery occurs in the NYHA functional classification and a distinct decrease occurs in pulmonary arterial pressure and cardiothoracic ratios.

REFERENCES

Authors Contribution:

SÖ, AUY: Conceived, designed and did statistical analysis & editing of manuscript.

MTAO: Did data collection and manuscript writing.