Atrial Septal Defect in the Aged

By Manuel Rodstein, M.D., Frederic D. Zeman, M.D., and Isadore E. Gerber, M.D.

The less obvious diseases occurring in the aged are often overlooked in clinical practice and have been given little mention in medical literature. Atrial septal defect has a high incidence in youth and middle age. Among individuals over the age of 50 it is the most common major congenital defect. The refinement of diagnostic criteria is made imperative by the fact that rapidly progressive cardiac failure (of relatively recent origin) may occur in cases with atrial septal defect over the age of 60. Ellis has shown that such individuals may be successfully operated upon, with closure of the defects, and symptomatic improvement. In the aged a well-defined clinical picture is often present, and may be seen with minimal impairment of cardiac function and no significant cardiac symptoms. In view of these considerations we are presenting the clinical and pathologic findings in two cases over the age of 70. One of these survived to the age of 84 years, and is the oldest case of atrial septal defect yet reported. We have combined the findings in these two cases with those in 16 other cases (65 years of age or older) reported by other authors with postmortem findings in order to determine common clinical features and explain the reasons for prolonged survival.

Case Reports

Case 1

An 84-year-old man had a history of dyspnea for 8 years, congestive heart failure for 2 years, and repeated attacks of bronchopneumonia and anginal pain. His blood pressure was 215/90; basal systolic and diastolic murmurs were present, no cyanosis was found. Enlargement of the right ventricle, of the right atrium, and of the pulmonary artery was present on x-ray (fig. 1), and a hilar dance was noted on fluoroscopy (figs. 2 and 3). Regular sinus rhythm with right bundle-branch block was present in his electrocardiogram. The clinical diagnosis of atrial septal defect was made. After 7 years of observation death resulted from congestive heart failure. At postmortem examination a high atrial septal defect 2 cm. in diameter was found in a 520-Gm. heart with hypertrophy of the right ventricle and atrium, and dilatation and severe atheromatosis of the pulmonary artery (fig. 4).

Case 2

A 72-year-old woman gave a history of dyspnea for over 18 years, congestive heart failure for over 9 years, recurrent attacks of bronchopneumonia, and persistent cyanosis. Her blood pressure was 205/95. A basal systolic murmur was constantly observed. An enlarged right atrium, right ventricle, and pulmonary artery were present on x-ray; hilar dance was not noted. Atrial flutter and right bundle-branch block were noted electrocardiographically. The clinical diagnosis of atrial septal defect was made. Death was due to congestive heart failure and bronchopneumonia. At postmortem examination a 2.5 by 3 cm. foramen ovale was found with the pulmonary vein from the right upper lobe emptying into the right atrium (fig. 5). The right atrium and ventricle were hypertrophied; the pulmonary artery was dilated.

Discussion

We have collected from the literature 16 cases of atrial septal defect coming to postmortem examination over the age of 65 and combined their data with our two cases (tables 1 and 2). We have omitted published cases in which clinical and postmortem data were not adequate. We have not included cases in which another congenital cardiac defect predominated, such as that of Adams and Hudson. In this case a 79-year-old woman, who died of cholangiohepatitis without any antecedent signs or symptoms of cardiac disease, was found to have a predominant Ebstein’s anomaly with an atrial septal defect 1.7 cm. in diameter of the ostium secundum type.

The age of the group ranged from 67 to 84 years for the men and from 70 to 82 for the women, with an average age of 75 years.
### Table 1

**Clinical Characteristics**

<table>
<thead>
<tr>
<th>Author</th>
<th>Age</th>
<th>Sex</th>
<th>Duration of congestive heart failure</th>
<th>History of broncho pneumonia</th>
<th>Angina</th>
<th>Duration of dyspnea</th>
<th>R.P.</th>
<th>Cyanosis</th>
<th>Basal diastolic murmur</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rodstein, M. et al. (Reported in this paper)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1) 84 M</td>
<td></td>
<td></td>
<td>2 yr.</td>
<td>+</td>
<td>+</td>
<td>8 yr.</td>
<td>215/90</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>2) 72 F</td>
<td></td>
<td></td>
<td>Over 9 yr.</td>
<td>+</td>
<td>0</td>
<td>Over 18 yr.</td>
<td>205/95</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Ellis, F. R., et al.</td>
<td>82</td>
<td>F</td>
<td>Terminal</td>
<td>0</td>
<td>0</td>
<td>8 yr.</td>
<td>145/100</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Rosenthal, L.</td>
<td>81</td>
<td>F</td>
<td>4 yr.</td>
<td>0</td>
<td>+</td>
<td>6 yr.</td>
<td>180/120</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Stannus, D. G. et al.</td>
<td>70</td>
<td>M</td>
<td>14 yr.</td>
<td>0</td>
<td>0</td>
<td>14 yr.</td>
<td>122/65</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Tarnower, H. et al.</td>
<td>77</td>
<td>F</td>
<td>2 wk.</td>
<td>0</td>
<td>+</td>
<td>6 wk.</td>
<td>170/110</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Colmers, R. A.</td>
<td></td>
<td></td>
<td>one episode</td>
<td>0</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1) 68 M</td>
<td></td>
<td></td>
<td>8 yr.</td>
<td>0</td>
<td>0</td>
<td>8 yr.</td>
<td>150/90</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>2) 78 M</td>
<td></td>
<td></td>
<td>8 yr.</td>
<td>0</td>
<td>0</td>
<td>50 yr.</td>
<td>110/68</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>3) 72 M</td>
<td></td>
<td></td>
<td>8 yr.</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Askey, J. M. et al.</td>
<td>72</td>
<td>M</td>
<td>3 yr.</td>
<td>0</td>
<td>+</td>
<td>15 yr.</td>
<td></td>
<td></td>
<td>slight 0</td>
</tr>
<tr>
<td>Pepper, D. S. et al.</td>
<td>77</td>
<td>F</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>6 mo.</td>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Sailer, S.</td>
<td>67</td>
<td>M</td>
<td>5 mo.</td>
<td>0</td>
<td>0</td>
<td>Over 5 yr.</td>
<td>104/80</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Tinney, W. S., Jr.</td>
<td>76</td>
<td>M</td>
<td>Terminal</td>
<td>0</td>
<td>0</td>
<td>3 wk.</td>
<td>144/64</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Erickson, C. W. et al.</td>
<td>68</td>
<td>M</td>
<td>14 yr.</td>
<td>0</td>
<td>0</td>
<td>14 yr.</td>
<td>155/90</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Espino-Vela, J.</td>
<td>71</td>
<td>F</td>
<td>Marked, duration not noted</td>
<td>—</td>
<td>0</td>
<td>Marked, duration not noted</td>
<td>—</td>
<td></td>
<td>Very slight 0</td>
</tr>
<tr>
<td>Case Records #40341*</td>
<td>74</td>
<td>M</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2 yr.</td>
<td>140/90</td>
<td>None</td>
<td>0</td>
</tr>
<tr>
<td>Mass General Hospital</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case Records #42092*</td>
<td>83</td>
<td>F</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1 yr.</td>
<td>110/60</td>
<td>None</td>
<td>0</td>
</tr>
<tr>
<td>Mass General Hospital</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case Records #44411*</td>
<td>70</td>
<td>F</td>
<td>Several years</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>180/80</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Mass General Hospital</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Eight were female and 10 were male. This incidence is at variance with the female preponderance found in younger groups: Campbell and associates\textsuperscript{18} found a 7 to 1 ratio in those over 20 years of age. This phenomenon, which is even more striking in view of the general female preponderance at advanced ages, may perhaps be explained in the same way Wood\textsuperscript{19} explained the predominance of women in cases of atrial septal defect with pulmonary hypertension. He considered it related to the occurrence of pulmonary emboli during pregnancy. Since pulmonary hypertension has an adverse effect on prognosis, more men might be expected to survive to advanced ages. Of the five cases between 60 and 64 operated on by Ellis, however, four were female.\textsuperscript{2}

The history of repeated attacks of bronchopneumonia in four of the group has been noted as common in atrial septal defect and attributed to the susceptibility of the hyperemic lung to minor upper respiratory infections.\textsuperscript{1, 20} Successful treatment of bronchopneumonia with antibiotics may in part be responsible for the prolonged survival of some of our cases, particularly case 1, in which at least five attacks were observed in 7 years.

The occurrence of angina in four of the group, a symptom not reported in young and middle-aged persons with atrial septal defects, may be attributed to coronary artery sclerosis in the presence of marked right ventricular hypertrophy.

The occurrence of dyspnea of many years' duration before the onset of overt congestive heart failure in 10 of the group has been noted as a common finding in atrial septal defect at all ages, and is attributed to the reduced air space in the lungs due to the excessive occupation of space by hyperemic blood vessels secondary to greatly increased pulmonary blood flow.\textsuperscript{21}

Cyanosis of appreciable duration and intensity was noted in only two cases. We have not considered significant the description of slight cyanosis in pre-terminal cases with associated mitral stenosis and in severe congestive heart failure as described in the aged cases of Askey and Kahler\textsuperscript{8} and Espino Vela.\textsuperscript{13} Slight cyanosis is frequently seen in cases of uncomplicated mitral stenosis in failure and is attributable to peripheral circulatory stasis. The rarity of cyanosis is surprising, since Campbell's group\textsuperscript{18} found a considerable incidence of central cyanosis in their cases in the fourth and fifth decades of life. It may be attributed to pulmonary hypertension not sufficient to cause a reversal of the principal atrial shunt to a right-to-left direction. Ellis and associates\textsuperscript{2} noted no case over 60 years of age with a predominant right-to-left shunt.

In the case of Erickson and Willius,\textsuperscript{12} cyanosis may be attributed to the huge size of the atrial septal defect, with a consequent pooling of atrial blood and marked bidirectional shunting.\textsuperscript{22} Persistent cyanosis in our case 2 may have been due to long-standing heart failure and to anomalous pulmonary venous drainage into the right atrium. Campbell and co-

*Figure 1*

*Case 1. Chest film of 31-year-old man, July 7, 1952, 6 years before death, showing marked enlargement of the pulmonary artery and its main branches and absence of the aortic knob.*
### Table 2

**Laboratory and Postmortem Findings**

<table>
<thead>
<tr>
<th>Author</th>
<th>ECG</th>
<th>Rhythm</th>
<th>Fluoroscopy</th>
<th>Chest x-ray</th>
<th>Cause of death</th>
<th>Heart weight, Gm.</th>
<th>Description of defect</th>
<th>Mitral stenosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rodstein, M. et al.</td>
<td>1) Rt BBB</td>
<td>RSR</td>
<td>Hilar dance</td>
<td>Same</td>
<td>CHF</td>
<td>520</td>
<td>Simple CHF</td>
<td>0</td>
</tr>
<tr>
<td>(Reported in this paper)</td>
<td>2) Rt BBB</td>
<td>Atrial flutter</td>
<td>—</td>
<td>Same</td>
<td>CHF</td>
<td>—</td>
<td>Broncho-pneumonia</td>
<td>0</td>
</tr>
<tr>
<td>Ellis, F. R. et al.</td>
<td>—</td>
<td>—</td>
<td>Iris dance</td>
<td>—</td>
<td>Typical Pulmonary ulcer</td>
<td>525</td>
<td>CHF</td>
<td>2.5 x 3 cm. anterior and above foramen ovale</td>
</tr>
<tr>
<td>Rosenthal, L.</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>CHF</td>
<td>250</td>
<td>CHF</td>
<td>2 cm. diam.</td>
</tr>
<tr>
<td>Stannus, D. G. et al.</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>CHF</td>
<td>3.3 x 2.8 cm.</td>
<td>CHF</td>
<td>0</td>
</tr>
<tr>
<td>Tarnower, H. et al.</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>CHF</td>
<td>4 cm. foramen ovale</td>
<td>CHF</td>
<td>0</td>
</tr>
<tr>
<td>Colmers, R. A.</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>CHF</td>
<td>635</td>
<td>CHF</td>
<td>2.5 diam. ovale secundum</td>
</tr>
<tr>
<td>Askey, J. M. et al.</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>CHF and Uremia</td>
<td>849</td>
<td>CHF</td>
<td>Large-lower edge over mitral and tricuspid valves</td>
</tr>
<tr>
<td>Pepper, D. S. et al.</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Sulfone block</td>
<td>280</td>
<td>0.8 cm. diam.</td>
<td>0</td>
</tr>
<tr>
<td>Sailer, S.</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>CHF</td>
<td>700</td>
<td>3 cm. diam.—lower edge over tricuspid valve</td>
<td>0</td>
</tr>
<tr>
<td>Tinney, W. S. Jr.</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>CHF</td>
<td>800</td>
<td>4.5 cm. foramen ovale</td>
<td>0</td>
</tr>
<tr>
<td>Erickson, C. W. et al.</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>CHF</td>
<td>880</td>
<td>10 x 7 cm. foramen ovale</td>
<td>0</td>
</tr>
<tr>
<td>Espino-Vela, J.</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>RV, CHF</td>
<td>3.5 cm. diam.</td>
<td>Paradoxic emboli</td>
<td>0</td>
</tr>
<tr>
<td>Case Records #40541</td>
<td>Normal (No BBB)</td>
<td>RSR</td>
<td>—</td>
<td>—</td>
<td>Paradoxic emboli</td>
<td>250</td>
<td>&quot;Large patent foramen ovale&quot;</td>
<td>0</td>
</tr>
<tr>
<td>Mass. General Hosp.</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Pulmonary emboli</td>
<td>0.5 cm. foramen ovale</td>
<td>Paradoxic emboli</td>
<td>0</td>
</tr>
<tr>
<td>Case Records #42092</td>
<td>Normal (No BBB)</td>
<td>Atrial fibr.</td>
<td>—</td>
<td>Normal</td>
<td>Pulmonary emboli</td>
<td>0.5 cm. foramen ovale</td>
<td>Paradoxic emboli</td>
<td>0</td>
</tr>
<tr>
<td>Mass. General Hosp.</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Multiple paradoxic emboli</td>
<td>2 cm. foramen ovale</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Case Records #4441</td>
<td>Normal leads</td>
<td>RSR</td>
<td>—</td>
<td>—</td>
<td>Multiple paradoxic emboli</td>
<td>—</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>
workers\textsuperscript{18} thought that this causes an additional large fixed left-to-right shunt that cannot become smaller with exercise. Wood\textsuperscript{29} included hemi-anomalous pulmonary venous drainage into the right side of the heart among the causes of severe pulmonary hypertension and cyanosis. On the other hand, Guntheroth et al.\textsuperscript{24} did not find cyanosis in 10 such cases studied with cardiac catheterization ranging in age from 2 to 40 years. Levinson et al.\textsuperscript{25} demonstrated only slight pulmonary hypertension on catheterization of such cases.

The absence of significant pulmonary hypertension in many elderly patients with atrial septal defect must be regarded as an important factor in their survival. The factors that cause pulmonary hypertension are repeated respiratory infections, pulmonary thrombosis and pulmonary embolism, particularly associated with pregnancy, and a failure of the pulmonary vascular resistance to fall low enough after birth to prevent slight pulmonary hypertension from increased flow.\textsuperscript{19} Kelly and Lyon\textsuperscript{29} have reported pulmonary hypertension in only seven of 19 patients over the age of 45 years; and in two over the age of 70 it was absent on cardiac catheterization. Coulsed and Littler\textsuperscript{26} found a pulmonary artery pressure of 50/20 in a patient aged 68. Ellis et al.\textsuperscript{2} reported that 20 of 38 cases 40 years of age and over had systolic pulmonary artery pressures less than 60 mm. of mercury. Patients with atrial septal defect who develop high pulmonary pressures die at an average of 36 years of age.\textsuperscript{21} Those who do not, develop considerable intolerance to effort as a rule between 50 and 60 years of age; if they remain in regular sinus rhythm effort, intolerance may not be manifest until after the age of 60.

Atrial septal defect is the only congenital cardiac defect with a significant incidence of atrial fibrillation. Campbell et al.\textsuperscript{18} noted that

\textit{Figure 2}

\textit{Case 1. Electrocardiogram of 84-year-old man, August 4, 1953, 5 years before death, showing right bundle-branch block and regular sinus rhythm.}
death usually occurs within 5 to 10 years of its onset, after a period of reduced function. Wood found it occurring as early as 17 years of age in cases of atrial septal defects and accompanied by effort intolerance. He thought that the cause of failure was nearly always the onset of atrial fibrillation, which in previously uncomplicated cases occurs at an average age of 50. The atrial fibrillation results immediately in tricuspid incompetence (a matter of a few seconds). The tricuspid incompetence elevates the right atrial pressure during systole and makes the interatrial shunt diastolic rather than systolic, but does not alter materially the magnitude of the left-to-right shunt. The limited left ventricular filling and systemic output is further diminished by lack of atrial support, a constant effect of atrial fibrillation in any condition. The flow ratios remain much the same because the right ventricular output also falls from a combination of the atrial fibrillation and the tricuspid incompetence. The breakdown, which manifests itself in fatigue, high venous pressure, hepatic enlargement, and edema, is due to a combination of a low cardiac output with impaired renal flow and right ventricular failure with tricuspid incompetence. This
breakdown is very sudden but under medical treatment patients may expect another 10 years or more of life, and do not suffer unduly until toward the end; indeed, some of them remain remarkably well and continue their occupations despite their handicap.

So long as a patient with atrial septal defect remains in normal rhythm and is uncomplicated by pulmonary hypertension, which most are, they may continue almost symptom free to well over 60, or even into old age.

In this group electrocardiographic records were described in fourteen cases. Atrial fibrillation was present in eight, atrial flutter in two, atrioventricular nodal rhythm in one, and regular sinus rhythm in only three cases. The duration of the arrhythmias was noted in only three of the cases, 2⅔, 3, and 14 years. We assume that in the light of the above observations by Wood, atrial fibrillation probably developed late in life in these aged individuals.

Four of five of Ellis’s cases who developed rapidly progressive congestive heart failure between 60 and 64 years of age had atrial fibrillation.

Blood pressure determinations were available in 14 of the 18 cases. The pulse pressure exceeded 40 in all but two, and in eight systolic and diastolic hypertension was present. In younger cases a pulse pressure over 40 is rare and the systolic pressure rarely exceeds 120. Even lower levels are found in children. These differences may be accounted for by the increased incidence of hypertension in the aged and changes in aortic elasticity with aging could yield the widened pulse pressures.

Right bundle-branch block, complete or incomplete, is common in atrial septal defect and was present in almost all of these aged cases with defects of significant size, i.e., over 1 cm. in diameter. This has been attributed to diastolic overload of the right ventricle or to hypertrophy of the crista supraventricularis.

In all cases the chest roentgenograms showed typical findings when the defect exceeded 1 cm. in diameter. Cardiac fluoroscopy revealed a characteristic hilar dance in each of the three cases reported as being fluoroscopyed (expansile pulsation of the pulmonary arteries spreading to their smaller branches).

In six cases without mitral stenosis and with defects of significant size, the average heart weight was 730 Gm. This far exceeds the average weight of 469 Gm. reported by Roesler for such cases over the age of 14 years.

In three cases complicated by mitral stenosis an average heart weight of 688 Gm. was found. Schnitker found the reverse, i.e., a greater average heart weight in cases with associated mitral stenosis.

At postmortem examination the findings of right ventricular hypertrophy, right atrial and pulmonary artery dilatation, and small left ventricle and aorta were similar to those found in younger cases.

The location and type of septal defect was given in 15 of the cases. Two were of the rare ostium primum type. Eleven were of the secundum type located in the area of the fossa ovalis, and two were high atrial septal defects. The two high defects were our patient 1 who survived to 84 years, and Ellis’s patient, who reached 82 years, the two oldest cases in the literature. The defects were 2 and 2.5 cm. in diameter, respectively, which

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Figure 4

Case 1. Right side of heart of 84-year-old man, showing the arrow pointing to a 2 cm. atrial septal defect in the extreme superior portion of the interatrial septum. The probe in the fossa ovalis demonstrates a patent foramen ovale.
are smaller than those usually found in younger patients. The secundum lesions ranged up to 4.5 cm. in diameter. Watkins and Gross have noted this difference in size between high and mid-atrial defects. Ellis et al. noted that the defects in their operated cases over 60 years were smaller than those usually found in younger patients. Associated anomalous pulmonary vein drainage reported as common in high defects by Lewis were not noted.

Burnett and White, however, thought that in their series the size of the defect did not affect the length of life, provided it exceeded 1 cm. in diameter.

Defects less than 1 cm. in diameter are of no clinical significance except for the occurrence of paradoxical pulmonary emboli. In the usual etiology of this condition a large pulmonary infarct causes a rise in pulmonary resistance sufficient to cause reversal of flow through a patent foramen ovale or other septal defect, to a right-to-left direction. A subsequent embolus, usually from a thrombosed leg vein, may then pass through the defect into the arterial circulation. Such supposedly rare paradoxical emboli were present in three of this aged group. The foramina ovale were described as 2 cm., 0.5 cm., and large in diameter. The great majority of cases of paradoxical embolization probably have only probe-patent (0.2 cm.) or pencil-patent (0.7 cm.) foramina ovale preceding the first pulmonary embolization, and these become functionally patent only after the right atrial pressure rises subsequent to the embolization. Two of the three cases with paradoxical emboli in this group had no cardiac hypertrophy or other significant abnormalities indicative of a preceding significant atrial septal defect.

In a review series of 101 cases of paradoxical emboli, Gill and Dammin found 27 over the age of 60.

The leading cause of death was the same as that found in atrial septal defect at all ages, congestive heart failure.

The coexistence of mitral stenosis in six of the group is high, considering the present opinion of the rarity of the so-called Lutembacher syndrome. Kelly and Lyons thought that the high incidence in the early literature probably resulted from misinterpreting the fibrous and calcific beading of the mitral leaflets as mitral stenosis. Such changes are common in elderly subjects. McGinn and White noted that the average life span was less than that of either uncomplicated atrial septal defect or mitral stenosis. White thought that the mitral stenosis is of no aid to a person with atrial septal defect in dealing with the burden on his right heart and pulmonary artery. In severe mitral stenosis an atrial septal defect may be helpful only by preventing acute pulmonary edema with a
atrial septal defect in the aged

tachycardia. Most authors consider the relationship coincidental with mitral stenosis resulting from rheumatic fever.

Summary and Conclusions
The clinical and pathologic findings in two cases of atrial septal defect, a male aged 84 years and a woman aged 72 years, are presented. The findings in these cases are combined with those in 16 other cases of atrial septal defect confirmed at postmortem examination, over the age of 65 years, found in the literature.

The clinical picture of atrial septal defect in the aged is sufficiently similar to that found in younger individuals to permit of a high degree of clinical accuracy of diagnosis.

The differences found between the clinical and pathologic findings in the aged and those found in younger individuals were (1) higher incidence of angina in the aged, (2) higher pulse and blood pressures in the aged, (3) an increased incidence of atrial fibrillation and flutter in the aged, (4) less frequent cyanosis in the aged, (5) a higher incidence of males in the aged, (6) heart weight much increased in the aged, and (7) a lower incidence of pulmonary hypertension in the aged.

The two oldest cases both had defects high in the atrial septum above the fossa ovalis.

References

What we know and what we think is not a new fountain gushing fresh from the barren rock of the unknown at the stroke of the rod of our own intellect, it is a stream which flows by us and through us, fed by the far-off rivulets of long ago. As what we think and say to-day will mingle with and shape the thoughts of men in the years to come, so in the opinions and views which we are proud to hold to-day, we may, by looking back, trace the influence of the thoughts of those who have gone before. Tracking out how new thoughts are linked to old ones, seeing how an error cast into the stream of knowledge leaves a streak lasting through many changes of the ways of man, noting the struggles through which a truth now rising to the surface, now seemingly lost in the depths, eventually swims triumphant on the flood we may perhaps the better learn to appraise our present knowledge, and the more rightly judge which of the thoughts of to-day is on the direct line of progress, carrying the truth of yesterday on to that of to-morrow, and which is a mere fragment of the hour, floating conspicuous on the surface now but destined soon to sink, and later to be wholly forgot.—SIR M. FORSTER. Lectures on the History of Physiology. London, Cambridge University Press, 1901, p. 1.
Atrial Septal Defect in the Aged
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