Effects of Balloon Pulmonary Valvuloplasty on Atrial Shunting

— A Patient With Pulmonary Valve Stenosis and A Large Secundum Atrial Septal Defect —

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Percutaneous balloon valvuloplasty has been established as an effective treatment for pulmonary valve stenosis (PS) in children and adults. However, there are few reports of the use of this technique in patients with other cardiac anomalies. We report the performance of balloon pulmonary valvuloplasty in a 72-year-old woman with a large atrial septal defect (ASD). This patient had a PS with a right ventricular pressure of 108/18 mmHg and a pulmonary arterial pressure of 42/21 mmHg, corresponding to a 66 mmHg pressure gradient. The ASD measured 32×27 mm. Balloon pulmonary valvuloplasty reduced the pressure gradient to 5 mmHg immediately after the procedure and to 2 mmHg 40 days after the procedure. The left-to-right shunt ratio was reduced from 17% before to 12% immediately after and to 36% 40 days after the procedure. In contrast, the right-to-left shunt ratio showed a transient increase from 16% before to 28% immediately after the procedure before decreasing to 11% 40 days after the procedure. The arterial blood oxygen tension was 53 Torr before, 46 Torr immediately after and 55 Torr 40 days after the procedure. The grade of heart failure decreased from New York Heart Association class IV before to class II in the chronic stage. These findings indicated that balloon pulmonary valvuloplasty in a patient with ASD transiently increased the right-to-left shunting associated with a decrease in oxygen tension, but that the change in the chronic stage was slight. In our patient with ASD valvuloplasty was useful in relieving the symptoms associated with elevated right ventricular pressure but could not reduce the hypoxemia caused by right-to-left shunting. In conclusion, valvuloplasty in patients with ASD should be considered as a preoperative treatment with the aim of reducing the risk of surgery and to treat symptoms in patients who refuse to undergo surgery.

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Key Words: Atrial septal defect; Atrial shunting; Elderly patient; Percutaneous balloon pulmonary valvuloplasty

Since its landmark description in 1982, percutaneous balloon pulmonary valvuloplasty (PBPV) has replaced surgery as the initial treatment of choice in patients of all ages with pure pulmonary valve stenosis (PS)1−3. However, other congenital anomalies, such as atrial septal defect (ASD) or ventricular septal defect (VSD), require complete surgical repair, and PBPV is rarely performed in such patients. Lau et al4 recently reported successful PBPV in 4 patients with PS and a small secundum ASD but did not assess the effect of PBPV on atrial shunting.

We performed PBPV in a 72-year-old woman with a large secundum ASD. In this paper, we report the acute and chronic effects of PBPV on atrial shunting.

Case Report

A 72-year-old woman who had had a heart murmur since early childhood was admitted to our clinic for worsening exertional dyspnea with repeated leg edema.
Balloon Pulmonary Valvuloplasty in A Patient With Atrial Septal Defect

Fig 1. Left: transesophageal view of the horizontal plane. The size and location of the atrial septal defect are shown in detail. Right: transesophageal pulsed Doppler valvuloplasty. The shunting flow is bidirectional before valvuloplasty, but is predominantly from left to right after valvuloplasty. LA, left atrium; RA, right atrium.

Table 1 Hemodynamics Before, Immediately After and 40 Days After PBPV

<table>
<thead>
<tr>
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<th>Before</th>
<th>Immediately after</th>
<th>40 days after</th>
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<tr>
<td>HR</td>
<td>75</td>
<td>83</td>
<td>75</td>
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<tr>
<td>PCWP</td>
<td>(20)</td>
<td>(14)</td>
<td>(12)</td>
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<tr>
<td>PAP</td>
<td>(42/21)</td>
<td>(37/18)</td>
<td>(24/14)</td>
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<tr>
<td>RVP</td>
<td>108/18</td>
<td>42/18</td>
<td>42/14</td>
</tr>
<tr>
<td>RAP</td>
<td>(20)</td>
<td>(17)</td>
<td>(10)</td>
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<tr>
<td>LAP</td>
<td>(20)</td>
<td>(13)</td>
<td>(11)</td>
</tr>
<tr>
<td>AoP</td>
<td>112/67(85)</td>
<td>103/68(84)</td>
<td>96/55(70)</td>
</tr>
<tr>
<td>LVP</td>
<td>113/17</td>
<td>103/13</td>
<td>95/8</td>
</tr>
<tr>
<td>LR shunt</td>
<td>17</td>
<td>12</td>
<td>36</td>
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<tr>
<td>RL shunt</td>
<td>16</td>
<td>28</td>
<td>11</td>
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<tr>
<td>Qp</td>
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<tr>
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<tr>
<td>PVR</td>
<td>193</td>
<td>349</td>
<td>219</td>
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PBPV, percutaneous balloon pulmonary valvuloplasty; HR, heart rate (beats/min); PCWP, mean pulmonary capillary wedge pressure (mmHg); PAP, pulmonary arterial pressure (mmHg); RVP, right ventricular pressure (mmHg); RAP, right atrial mean pressure (mmHg); LAP, left atrial mean pressure (mmHg); AoP, aortic pressure (mmHg); LVP, left ventricular pressure (mmHg); LR shunt, left-to-right shunt ratio (%); RL shunt, right-to-left shunt ratio (%); Qp, pulmonary blood flow (L/min); Ql, systemic blood flow (L/min); PVR, pulmonary vascular resistance (dyne/sec/cm⁻²).

and cyanosis that had persisted for several years despite treatment with diuretics.

Physical examination revealed a blood pressure of 102/64 mmHg and an irregular heart rate of 54 beats/min. A loud pulmonic ejection click and a grade 4/6 systolic ejection murmur were heard at the left sternal border. The S2 sound was widely split but there was no respiratory change. Arterial blood gas analysis showed a low oxygen tension of 53 Torr in room air. An electrocardiogram showed atrial fibrillation, complete right bundle branch block, and right ventricular hypertrophy. A transthoracic echocardiogram revealed pulmonary valve stenosis and right ventricular hypertrophy together with an atrial septal defect. Color Doppler flow imaging disclosed a moderate tricuspid regurgitant jet that was directed toward the atrial septum. Continuous-wave Doppler examination demonstrated a velocity of 4 m/sec across the pulmonary valve, corresponding to an instantaneous peak systolic pressure gradient of 64 mmHg. Transesophageal echocardiography revealed an atrial septal defect measuring 32×27 mm and pulsed Doppler examination showed a bidirectional shunting flow at the center of the defect (Fig 1). Cardiac catheterization demonstrated PS with a transpulmonic systolic pressure gradient of 66 mmHg, a right ventricular pressure of 108/18 mmHg, and a pulmonary arterial pressure of 42/21 mmHg. The right and left atrial mean pressures were 20 mmHg. Systemic and pulmonary blood flows calculated with the Fick method using the Autoaerobics System (Model R1500S, Anima INC, Tokyo) for oxygen consumption measurement were similar, and the shunt ratios were 17% for the left-to-right shunt and 16% for the right-to-left shunt (Table 1). The right ventricular angiogram showed systolic doming of the pulmonary valve and infundibular pulmonary stenosis. On angiography the size of pulmonary valve annulus was measured as 20 mm.

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Our recommended treatment option was surgical repair of both PS and ASD, but the patient refused surgery because of her age. We therefore decided to perform PBPV to reduce the right ventricular pressure overload. It was hoped that this treatment would relieve the right ventricular failure and decrease right-to-left shunting, thus alleviating the patient's symptoms. Catheterization for PBPV was performed after 2 months' medical treatment resulted in no symptom improvement. Written informed consent was obtained from the patient and the person in charge of the old people's home where she lived. An exchange guide wire (0.038 inch; Cook, IN) was placed in the left pulmonary artery and a Mansfield balloon (20 × 40 mm; Mansfield Scientific, Boston, MA) was passed across the pulmonary valve. The balloon was briefly inflated manually with contrast material to a pressure of 50 psi. There was no change in any subjective or objective findings during the procedure. Simultaneous recordings of the aortic and the right ventricular pressures before and during PBPV are shown in Fig 2. We did not measure right ventricular pressure during the procedure, but aortic pressure did not fall, although hypotension is usually noted during this procedure. Immediately after PBPV, the right ventricular systolic pressure decreased from 108 to 42 mmHg, which resulted in a decrease in the transpulmonary pressure gradient to 5 mmHg. An increase in the right-to-left shunt ratio to 28% and a decrease in the left-to-right shunt ratio to 12% resulted in a reduction of arterial blood oxygen tension from 53 to 46 Torr.

During follow-up after PBPV, the exertional dyspnea decreased and the repeated leg edema disappeared without diuretics. Catheterization was repeated 40 days after PBPV and showed a right ventricular pressure of 38/14 mmHg and a transpulmonic systolic pressure gradient of 2 mmHg. The left-to-right shunt ratio increased to 36% and the right-to-left shunt ratio decreased to 11%. Transesophageal pulsed Doppler examination showed the dominant left-to-right shunting flow, but right-to-left shunting flow was also still apparent (Fig 1). The degree of tricuspid regurgitation was less than before PBPV. There was a slight increase in arterial blood oxygen tension from 53 to 55 Torr, but the arterial oxygen tension during nasal oxygen inhalation (3 L/min) increased from 66 Torr before PBPV to 78 Torr during the follow-up period.

The patient is now 75 years old and is doing well without diuretics. Repeated Doppler echocardiography 3 years after PBPV revealed a small increase in a transpulmonic systolic pressure gradient to 15 mmHg, but bidirectional shunting flow through the atrial defect and the pulmonary-systemic flow ratio calculated from an echocardiogram were similar to the values 40 days after PBPV.

**Discussion**

We report the case of an elderly patient with a large secundum ASD in whom PBPV was safely performed and resulted in improvement of symptoms. There are few reports of PBPV in patients with PS and ASD. Lau et al performed PBPV in 4 patients with a small secundum ASD, but they did not describe the effects of the procedure on atrial shunting. We will discuss the effects of PBPV on atrial shunting and the clinical implications of PBPV in patients with other cardiac anomalies.

Symptom improvement was the result not of an increase in arterial blood oxygen tension, but of a
reduction in right ventricular pressure because arterial blood oxygen tension was unchanged after PBPV. We expected before PBPV that a reduction in right ventricular pressure might ameliorate the right ventricular failure caused by the elevated pressure and the hypoxemia caused by the right-to-left shunting. Unfortunately, in our patient, arterial blood oxygen tension in room air immediately after the procedure decreased from 53 to 46 Torr, and even on follow-up the increase was only slight (2 Torr).

The decrease in oxygen tension immediately after the procedure may be the result of an increase in right-to-left shunting. A transient increase in right-to-left shunting through a patent foramen ovale during this procedure has been noted previously in an adult patient\(^7\) and this interpretation is supported by the unchanged aortic pressure during the procedure (Fig 2) and the increased right-to-left shunt ratio immediately after the procedure (Table 1). The increased right-to-left shunting may be caused by an elevated right atrial pressure associated with acute occlusion of the right ventricular outflow. In addition, the exaggerated tricuspid regurgitant flow across the ASD during the procedure could also play a part in the development of right-to-left shunting.\(^\) Increased right-to-left shunting during the procedure should cause cyanosis, but we did not notice any change in the patient's appearance, perhaps because the balloon was only inflated for a very short time (less than 10 sec).

The disappointing increase in arterial blood oxygen tension during follow-up may be mostly attributed to the residual right-to-left shunt associated with the high pulmonary vascular resistance (Table 1 and Fig 1). However, pulmonary dysfunction might also be responsible for the low arterial oxygen tension. Thus, the low oxygen tension of pulmonary venous blood (79 Torr) in the present case suggests impaired oxygenation in the lung.

We did not note any clear residual infundibular PS after PBPV in our patient. Infundibular stenosis is usually more severe in elderly patients because the severity of infundibular hypertrophy is dependent on the duration of high wall stress and the age of onset.\(^8\) We cannot determine whether the absence of marked infundibular stenosis is attributable to the successful technique or the right ventricular dysfunction caused by long-term high pressure loading and hypoxemia. A small pulmonary blood flow remaining even 40 days after PBPV might support the hypothesis that the absence of the residual infundibular stenosis is due to right ventricular dysfunction.

Combined PS and ASD or VSD is not rare. A study of 433 patients with PS aged 1–36 years revealed 164 cases of isolated PS (37%), 132 cases of PS combined with ASD (31%), and 137 cases of PS combined with VSD (32%)\(^9\) However, PS is a rare cardiac anomaly in elderly patients. Resnekov\(^10\) reported an incidence of 11% in adults with congenital heart disease, but in an autopsy study of 3,000 subjects over the age of 60 no pulmonary valve organic stenosis was found.\(^11\) The natural course of PS is dependent on the severity of stenosis, and to our knowledge the oldest patient with moderate or severe PS was aged 76 years.\(^12\) The oldest reported patient with ASD was 87 years,\(^13\) but there has been no report of combined ASD and PS in any patient over 70 years. Our patient is the oldest person to have PS combined with ASD.\(^14\)–\(^16\)

The best treatment for these patients is surgical closure of the septal defect combined with relief of right ventricular pressure overload. ASD can be surgically closed with low mortality and excellent long-term results in children and in young adults.\(^17,18\) Although good surgical results have also been reported in elderly patients\(^18\)–\(^21\) as a result of the development of methods that protect the myocardium from surgical injury, complications appear to be more frequent than in younger patients.\(^22\) Mortality in adult and elderly patients operated on for ASD ranges from 0% to 12.6%;\(^17,21,23\) and the incidence of major complications ranges from 24% to 67%.\(^18,24\) The best results are achieved in the absence of other diseases or complications of ASD. Moreover, progression from normal preoperative vascular resistance to pulmonary vascular disease\(^25\) and the onset of left ventricular failure, probably due to left ventricle hypoplasia, have been noted after surgical closure of ASD.\(^17\) High pulmonary vascular resistance plays a key role both in the deterioration and in the response to surgical treatment. In ASD combined with PS the pulmonary hemodynamics and shunt dynamics may be modified, making preoperative evaluation difficult. In our patient, the small increase in pulmonary blood flow despite relief of right ventricular outflow obstruction could be associated with concealed right ventricular dysfunction and/or pulmonary obstructive vascular damage.

Although we chose PBPV because our patient stoutly refused surgical treatment, the first choice of treatment in all such cases should be surgery. However, in patients with other cardiac anomalies or who refused surgery because of old age or significant extracardiac disease, PBPV should be considered. The successful repair of PS with PBPV could relieve symptoms and improve quality of life. Although longer term follow-up studies are necessary to establish the

Clinical Implications
usefulness of PBPV in such patients, the indications for PBPV might be extended in the near future.

References


