

Pulmonary Venous Flow in Large, Uncomplicated Atrial Septal Defect

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Background: The pulmonary venous flow velocity pattern (PVFVP) in atrial septal defect (ASD) has not been previously studied in detail. Normally, PVFVP is primarily determined by the left heart performance. We hypothesized that the impact of left-sided heart dynamics on PVFVP is diminished in patients with ASD because of the presence of a left-to-right shunt into the low-resistance right side of the heart. **Methods and Results:** Transesophageal echocardiography was performed in 19 adults and 3 children with a large, uncomplicated secundum ASD (maximum diameter 0.6 to 3.0 cm). All patients were in normal sinus rhythm with an average heart rate of 78 bpm in adults and 116 bpm in children. In 21 subjects the antegrade PVFVP lacked distinct systolic (S) and diastolic (D) waves. Instead, we observed a single continuous antegrade wave extending from

the beginning of systole to the onset of atrial contraction. Furthermore, the amplitude of the atrial reversal (AR) wave was smaller than in historical controls. In 3 patients in whom ASD was surgically repaired, we observed an immediate return of distinct S and D waves postoperatively. This confirmed that PVFVP abnormality was indeed the result of the ASD. Also a large increase in the AR wave amplitude (46 ± 15 cm/s) was noted postoperatively.

Conclusions: This previously unrecognized PVFVP comprising a single continuous antegrade wave and a diminished AR wave sheds new light on the hemodynamics of ASDs. Its presence may also alert the echocardiographer to the possibility of an ASD when the septal defect cannot be visualized directly. (J Am Soc Echocardiogr 2001;14:386-90.)

INTRODUCTION

Atrial septal defect (ASD) is one of the most prevalent forms of congenital heart disease. On the basis of modern echocardiographic screening methods, it has been estimated that the prevalence of ASD is about 24 per 10,000 live births. The majority of these echocardiographically detected ASDs are small and close spontaneously.¹ Nonetheless, ASD still ranks among the most common congenital defects after the age of 20 years. Only the bicuspid aortic valve is encountered more frequently than ASD in that age group.²

Although congenital defects can occur in any portion of the atrial septum, they are most often found

in the area of fossa ovalis and are referred to as *secundum* ASDs. In a much smaller proportion of cases, the ASD involves either the lower portion of the atrial septum (primum ASD) or the septal areas around the superior vena cava (sinus venosus ASD) and the coronary sinus.

Because the right side of the heart has a higher compliance and a lower resistance than the left side after birth, ASD most commonly leads to a left-to-right shunt at the level of the atria. The shunting of the left atrial blood leads to volume overload of the right side of the heart and subsequent right atrial and right ventricular dilatation.

Persons with untreated ASD have a shortened life span.^{3,4} Timely diagnosis is important as early surgical closure improves survival and diminishes morbidity.⁵ Two-dimensional echocardiography (2D) may reveal the actual defect in the atrial septum as well as the dilatation of right-sided heart chambers. In addition, color and spectral Doppler are used to characterize the flow through the defect, to visualize the augmentation of blood flow through the pulmonary valve, and to estimate pulmonary artery pressures.

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Transesophageal echocardiography (TEE) is superior to transthoracic echocardiography in the visualization of ASD. It allows for a detailed exploration of the systemic and pulmonary venous return to the heart, which may be necessary for (1) evaluation of possible sinus venous ASD and the anomalous pulmonary venous return, and (2) estimation of left atrial pressures by pulmonary venous flow (PVF) velocity analysis.

Echocardiographic data on PVF velocity patterns in patients with ASD are scarce.⁶ We hypothesized that altered left atrial hemodynamics imposed by the left-to-right shunt in large, uncomplicated ASD lead to changes in PVF. Such changes, in turn, could help elucidate the hemodynamics of ASD and aid in its diagnosis.

METHODS

Patients

We evaluated PVF in 22 patients (9 males, 13 females) with large, uncomplicated secundum ASD. There were 3 children, all aged 3 years, and 19 adults, whose ages ranged from 18 to 83 years (mean \pm SD: 51 \pm 18 years).

With the use of commercially available echocardiographic equipment, routine TEE was performed in all patients. There were no clinical or echocardiographic signs of significant valvular or myocardial abnormalities, increased pulmonary resistance, or right-to-left shunting.

A multiplane probe was used in the adults and a biplane probe in the children. In 6 patients, the study was performed just before surgery while the patient was under general anesthesia. For the remaining 16 patients, the study was performed in the echocardiography laboratory with or without conscious sedation.

Three patients underwent surgical closure of the ASD, providing an opportunity to study PVF before as well as immediately after the ASD closure.

Pulmonary Veins

Two-dimensional echocardiography and color flow mapping were used to identify the location of pulmonary veins. The left upper pulmonary vein was visualized adjacent to the left atrial appendage at about 30°. By further increasing the angle, and by leftward rotation and deeper insertion of the probe, the left lower pulmonary vein was brought into view.

The right lower pulmonary vein was imaged at 0° after a rightward rotation and a deeper insertion of the probe to the level of the lower left atrium. The right upper pulmonary vein was brought into view at 90° after an extreme rightward rotation of the probe past the superior vena cava.

Once the location of the pulmonary vein was determined by 2D echocardiography and color mapping, a pulsed Doppler sample volume was placed into the ostium of the pulmonary vein approximately 1 cm from the left atrium. At least 2 different pulmonary veins were visualized in each patient. Spectral tracings of the pulmonary veins were recorded on a standard VHS tape for off-line analysis.

Atrial Septal Defect

The diagnosis of ASD was established by 2D echocardiography and confirmed by color flow mapping and pulsed Doppler spectral analysis at the level of the atrial septum. Statistical analysis was performed with the Student *t* test (2-tailed) for comparison between group means. The difference between groups was considered significant when the *P* value was less than .05.

RESULTS

Atrial Septal Defect Findings

The maximal ASD diameter ranged from 0.6 to 3.0 cm (mean \pm SD: 1.5 \pm 0.8 cm). In all patients, the flow across the ASD was a typical continuous flow with a wave of acceleration at end diastole associated with atrial contraction. The direction of the shunt was from the left atrium to the right atrium in all patients.

Heart Rate

All patients were in normal sinus rhythm. The average heart rate in adult patients was 78 \pm 14 bpm. In the 3 children, the average heart rate was 116 \pm 15 bpm.

Pulmonary Venous Flow Velocity Pattern

In all but one patient, a characteristic pulsed Doppler PVF velocity pattern could be observed in the pulmonary veins. The pattern consisted of a single antegrade wave (lasting from the onset of ventricular systole to the onset of atrial contraction) and a retrograde wave (after atrial contraction), the amplitude of which was smaller than those in historical controls. No significant difference was found in the PVF velocity pattern in left versus right or superior versus inferior pulmonary veins.

Antegrade Flow Velocity Component

The antegrade flow velocity component in 21 of our 22 patients with ASD lacked the distinct systolic and diastolic waves that are observed in healthy persons (Figure 1, A). We refer to this component as the continuous antegrade wave (CAW). The peak CAW veloc-

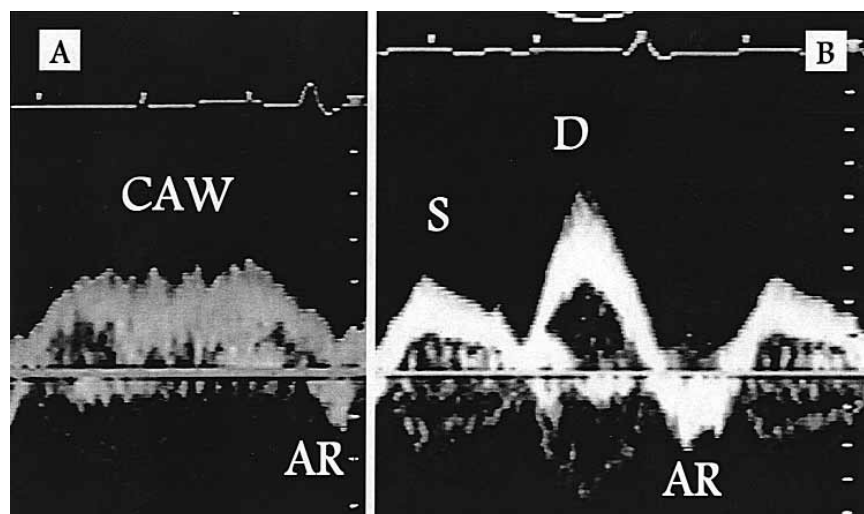


Figure 1 Pulmonary venous flow velocity pattern in a 30-year-old man with a large atrial septal defect (maximum diameter 1.3 cm) before (**A**) and after (**B**) surgical closure. Note the absence of distinct systolic (*S*) and diastolic (*D*) waves and the presence of a continuous antegrade wave (*CAW*) in **A** compared with the clear separation between the *S* and *D* waves in **B**. Also note the increase in the peak atrial reversal velocity from 24 cm/s preoperatively to 45 cm/s postoperatively. Each tick on the vertical axis represents 20 cm/s.

ity among all study patients ranged between 40 and 89 cm/s (mean \pm SD: 63 ± 15 cm/s).

Retrograde Flow Velocity Component

Although all study patients were in normal sinus rhythm, the usual reversal of flow from the left atrium into the pulmonary veins with atrial contraction (AR wave) was either absent or small. The peak AR velocity ranged between 0 and 26 cm/s (mean \pm SD: 14 ± 7 cm/s).

Postoperative Studies

In those patients in whom the ASD was surgically repaired, the postoperative PVF velocity pattern showed distinct systolic and diastolic antegrade waves and a larger retrograde velocity after atrial contraction (Figure 1, *B*).

The average postoperative AR velocity increased to 46 ± 15 cm/s. This was statistically significant (2-tailed *P* value = .04) when compared with preoperative values in the same patients, though only a small number of patients underwent surgery in this study.

DISCUSSION

In healthy persons, the PVF velocity pattern consists of 2 large and distinct antegrade waves referred to as *S* (systolic) and *D* (diastolic) waves, and a small re-

trograde wave concomitant with atrial systole (atrial reversal or AR wave). The morphology of individual PVF velocity waves is not influenced by respiration⁷ and is primarily determined by the performance of the left side of the heart.⁸⁻¹⁰

The initial antegrade component (*S* wave) coincides with atrial relaxation and apical displacement of the mitral ring after ventricular contraction. The *S*-wave velocity peaks when the left atrial pressure reaches its nadir.¹¹ As the atrial stretching reaches its limit in late ventricular systole, the left atrial pressure rises, and the *S*-wave velocity gradually approaches zero.

Upon opening of the mitral valve in early ventricular diastole, the antegrade PVF resumes, and the *D* wave is inscribed on the pulsed Doppler spectral tracing. The PVF velocity *D* wave coincides with the mitral *E* wave.¹² The morphology of both *D* and *E* waves is determined by the diastolic properties of the left ventricle and the pressure gradient between the left atrium and the left ventricle in early diastole.

In young persons (aged < 41 years), the *D*-wave peak velocity is usually larger than that of the *S* wave (53 ± 10 versus 46 ± 10 cm/s). In middle-aged persons, the peak *S* and *D* velocities are similar and reach on average 53 ± 9 and 46 ± 10 cm/s, respectively. With aging, a gradual increase occurs in the *S*-wave velocities concomitant with the diminution of the *D*-wave velocities. For instance, in persons older

than 55 years, peak S and D velocities average 60 ± 10 and 38 ± 10 cm/s, respectively.^{13,14}

Because the pulmonary vein ostia lack valves, the blood is propelled with each atrial contraction into the pulmonary veins as well as into the left ventricle. The average peak velocity of the retrograde (AR) wave in healthy young persons (aged < 41 years) is 18 ± 3 cm/s. In middle-aged persons, it averages 22 ± 4 cm/s and rises to 25 ± 5 cm/s after the age of 55 years.^{13,14}

Atrial septal defect dramatically alters cardiac hemodynamics during both ventricular systole and diastole. The primary objective of this study was to determine whether this altered cardiac physiology leads to a unique PVF velocity pattern on TEE. We emphasize that we measured PVF velocities rather than volumetric flows. Direct flow measurements are not possible with TEE because the diameters of pulmonary veins cannot be measured accurately with this technique.

In patients with ASD, the entire pulmonary venous return does not cross the mitral valve (as is the case in healthy persons), but rather is divided between the flow across the mitral valve and the flow across the ASD.

Unlike healthy persons in whom the left atrium becomes a dead-end chamber during ventricular systole, patients with ASD have a left atrium that is in constant communication with the compliant, low-resistance right side of the heart throughout the cardiac cycle. This persistent egress of blood from the left atrium via the ASD throughout the cardiac cycle allows for a continuous flow into the left atrium from the pulmonary veins.

We hypothesized that this continuous inflow into the left atrium leads to the loss of distinct S and D waves and their replacement with a CAW. Indeed, the PVF velocity pattern in patients with large uncomplicated ASD resembles the pulsed Doppler velocity pattern recorded at the level of the septal defect during the corresponding part of the cardiac cycle.

The characteristic CAW was observed on Doppler echocardiography in 21 of our 22 patients. In one report from 1986, Takaya et al¹⁵ demonstrated a similar CAW velocity pattern by direct PVF velocity measurements during cardiac catheterization in some of their patients with secundum ASD. The preservation of distinct S and D waves in our remaining patient is probably explained by the small size (0.6 cm) of his ASD.

An AR wave with an amplitude often smaller than that reported in historical controls is another characteristic of the PVF velocity pattern, seen in our patients with a large ASD. In our study patients

(mean age 51 ± 18 years), the average peak AR velocity was 14 ± 7 cm/s, which is lower than the reported control values for any age group (see above).

In healthy persons, the atrial contraction ejects blood into the left ventricle and pulmonary veins, whereas in patients with a large ASD, atrial systole propels blood into an additional chamber, namely the right side of the heart. As a consequence, the PVF becomes less dependent on left ventricular pressure changes. The diminished amplitude of the AR wave in our study patients suggests that a preferential flow exists during atrial systole across the low-resistance ASD at the expense of retrograde flow into the pulmonary veins.

In patients in whom the ASD was surgically repaired, changes in both the antegrade and retrograde components of the PVF velocity pattern were seen. The antegrade velocity component revealed a return of separate S and D waves after the ASD closure. This confirms that the preoperative abnormality in antegrade PVF characterized by the CAW was indeed caused by the presence of the ASD.

After ASD closure, the amplitude of the AR wave increased significantly. This rise in the peak AR velocity is consistent with the loss of a low-resistance outflow path from the left atrium.

Conclusions

In uncomplicated, large ASDs, the normal biphasic pattern of antegrade PVF velocity is absent, and the normal PVF reversal with atrial contraction is often diminished. This previously unrecognized PVF pattern sheds new light on the hemodynamics of ASDs. In those instances in which the ASD may be difficult to visualize directly, the presence of this characteristic PVF velocity pattern may alert the clinician to the possibility of an ASD.

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