Pattern of Pulmonary Venous Flow in Patients with Atrial Septal Defect

Tahsin A Al KInani, FIBMS (med), FIBMS (card)
Lecturer, College of Medicine, ThiQar University; Consultant Cardiologist, Nassirriya

نمط التدفق في الوريد الرئوي لدى المرضى المصابين بالفتحة الولادية بين الاذينين

الغاية من الدراسة: تلقت دراسة نمط التدفق الوريدي لدى المرضى المصابين بالفتحة الولادية بين الاذينين القليل من الاهتمام. سعينا لدراسة هذا النمط في محاولة لتسليط الضوء على الفسلجة المرضية له
المرضى والأساليب: تم سبر الوريد الرئوي الاعلى الايسر باستخدام فحص الدوبلر النبضي خلال فحص القلب بالأمواج فوق الصوتية عن طريق المرئ في ٣٢ مريضا على التوالي مصابين بالفتحة الثانوية او الجيبية\الوريدية بين الاذينين مع نبض جيبي طبيعي. تم تسجيل عدد من المعلمات شملت النمط الموجي، السعة القصوى لموجة S و D و مدى موجة الانعكاس الاذيني (موجة A).
النتائج: في جميع المرضى الاثنين والثلاثين، لوحظ و جود نمط فريد من التدفق الدموي يتميز بوجود موجة منفردة متواصلة تقدمية شاملة الانقباض و الانبساط دون انقطاع، وتنتهي في بداية الانقباض الاذيني. كانت ذروة سرعة التدفق أعلى من الطبيعي، بدءا من ٤٠,٥ و حتى ١٠٨ سم في الثانية (متوسط±أم ٧٤,١١±١٥,٩٦)، في حين بلغت سرعة الانعكاس الاذيني (A) 5.0_-2.4_م/ثا (متوسط±أم 2.75±3.6_)، وهو أقل من المعدل الطبيعي.
الخلاصة:عيب الحاجز الاذيني يؤدي الى تغيير نمط تدفق الدم في الدورة الدموية، و يتمثل ذلك بنمط أحادي الموجة مع تسارع التدفق، و يمكن أن يساعد ذلك في تشخيص الحالة.

Objective:
the pulmonary venous flow pattern in patients with ASD has received little attention. We sought to study this pattern and attempt to shed light on its pathophysiology
Patients and methods: the left upper pulmonary vein was interrogated by pulse wave Doppler during trans-esophageal echocardiography in 32 consecutive patients with secundumASD or sinus venosus ASD in sinus rhythm;we observed several parameters including the waveform pattern, the maximal amplitude of the S and D waves, and the magnitude of the atrial reversal wave (a wave).
Results: in all 32 patients, a unique waveform pattern was noted, characterized by a single continuous antegrade pattern spanning systole and diastole without interruption, ending at the onset of atrial contraction. The peak flow velocity was exaggerated, ranging from 40.5 to 108 cm/sec (mean ± SD 74.11 ± 15.96). The atrial reversal wave had a peak velocity of _2.4-_5.0 (mean ± SD _3.6±2.75) which is lower than normal.
Conclusion: atrial septal defect causes an altered pattern of blood flow in the pulmonary veins, characterized by a single waveform pattern with increased **velocity of flow and could aid in the diagnosis of the condition.**

Introduction

In patients with atrial septal defect (ASD), the pulmonary venous flow (PVF) pattern has been recently studied by few investigators [1-4]. Saric et al[1] described a unique flow pattern in patients with ASD, which returned to normal after surgical closure.Dass et al[2] described the same pattern of flow using trans-thoracic echocardiography. The normal pulmonary venous flow waveform consists of an antegrade systolic wave (S wave), an antegrade diastolic wave (D wave), and an atrial reversal wave (A wave) (figure 1) [5].

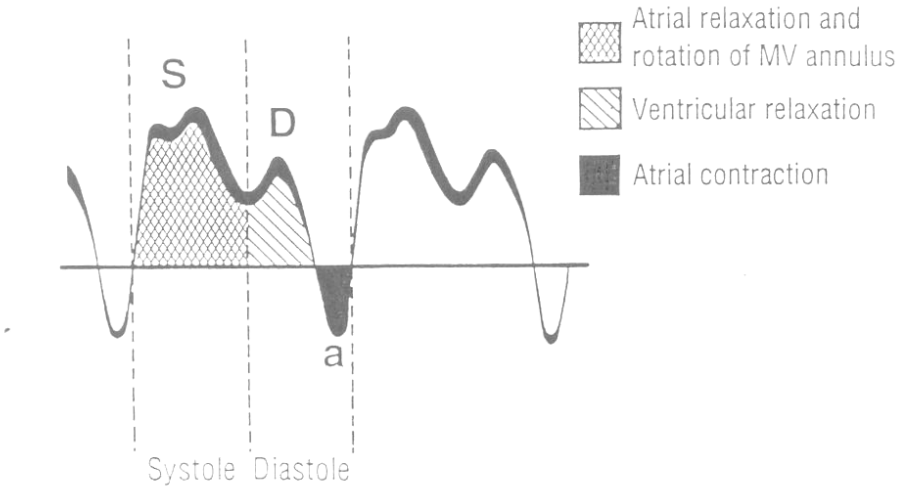

Figure 1: Schematic summary of the physiologic basis for the S, D, and awaveforms.

Normally, blood starts to flow into the left atrium (LA) immediately after the end of atrial systole, the flow velocity peaks at the mid of systole, driven by the pressure gradient between the pulmonary veins and the left atrium, which is augmented by the downward movement of the mitral annulus during ventricular systole. It slows down towards the end of ventricular systole as the LA becomes full of blood, thereby producing the upstroke and descent of the S wave, respectively [5]. As the mitral valve opens, blood starts to flow back again into the LA as the latter empties itself into the LV through the open mitral valve, producing the D wave. During atrial contraction, blood flow into the pulmonary vein is transiently reversed due to temporary elevation of LA pressure (a wave) [5, 6].

In patients with ASD, there is an alteration in the driving hemodynamics which may lead to a change in the pattern of pulmonary blood flow. In this paper, we sought to investigate this changing pattern.

PATIENTS AND METHODS

Study population:

As of January 2010 till October 2010, 32 consecutive patients seen in the consultation clinic in Nassiriya Heart Centerwere included in the study. Their ages ranged from 12-65 years. There were 10 (31.25%) males and 21 (68.75%) females. All patients had hemodynamically significant ASDs, as revealed by clinical, radiologic, and echocardiographic assessment.

Echocardiographic data:
A comprehensive transthoracic echocardiographic examination was performed in all patients. The presence, site, location and hemodynamic significance of ASD were ascertained by transthoracic echocardiography. During transesophageal echocardiography (TEE), all patients were studied in the left lateral decubitus position. TEE was performed according to a standard method using Evnisor CHD echocardiographic machine (Philips Medical Systems, Andover, USA). The patient was consciously sedated using midazolam intravenously, and after application of local anesthesia to the oral cavity (xylocaine spray), the probe was introduced into the patient's esophagus and standard views were obtained. After identifying the ASD and assuring its site, the left upper pulmonary vein was sampled. The best view of the vein was obtained by rotating the transducer till a longitudinal section of the vein was in view. The vein was placed in the center of the displayed section and sampled with color flow Doppler to assist positioning the pulsed wave sample volume, which was placed within 1 cm of the pulmonary vein-left atrial junction. The signal gain was optimized to record the flow waves while avoiding background noise. A small sample volume was used. The pulse repetition frequency was set at the maximal possible value for the depth of the sample volume, and wall motion filter was set at the minimal value. A simultaneous ECG tracing was continuously displayed on the echocardiographic monitor.
All data were recorded on the device memory and were analysed offline including the defect size, the magnitude of RA and RV dilatation, the pattern and timing of flow, and peak velocity.
Exclusion criteria: Excluded from the study were patients not in sinus rhythm, those with severe mitral regurgitation (MR), patients with left ventricular (LV) dysfunction, those with osteumprimum ASD, and patients with an associated defect at a level other than the atrial septum.
All data were recorded as mean ±S.D, the obtained values were compared with standard normal values.

RESULTS

Table (1) shows the echocardiographic findings in patients with atrial septal defect.

	Range	***Mean ±SD***
LAD (cm)	2 – 4.03	3.03±0.55
RVD (cm)	1.9 – 4.69	3.1±0.75
LVIDD (cm)	2.7 – 5.7	4.31±0.63
LVIDS (cm)	1.6 – 3.7	2.8±0.48
FS %	26.7 – 55.1	36.09 ± 6.7
EF%	54.3 – 80.9	62.02± 8.37

Table 1: echocardiographic findings in 32 patients with ASD. LAD: left atrial diameter. RVD: right ventricular diameter. LVIDD: left ventricular diastolic internal diameter. LVIDS: left ventricular systolic internal diameter. FS: fractional shortening. EF: ejection fraction.

TEE measurements:
A correct diagnosis of the presence and type of shunt was made in all 32 patients with a proved ASD, 24 patients were found to have an ostiumsecundum, 7 a sinus venosus defect, and one had an aneurysm of the interatrial septum with a patent foramen ovale (PFO). Of the 32 patients with ASD, 11 patients had mild MR, with a mean mosaic jet area of 0.78±0.52 cm^2.
The shunt was predominantly left-to-right in all 32 patients, as assessed by TEE.
Maximum defect diameter in these 32 patients varied from 0.9 to 3.66 cm (mean 2.14±0.77).
Pulmonary venous flow velocitypattern in ASD:
Table (2) shows pulmonary venous flow velocities in patients with ASD. In all patients, the antegrade PVF 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 (fig 2).

Table 2: TEE findings in 32 patients with ASD.

Parameter	*Range*	*Mean ± SD*
Single continuous wave (cm/sec)	40.5 – 108	74.11 ±15.96
Atrial reversal wave (cm/sec)	-2.4 _ -5.0	-3.26±2.75

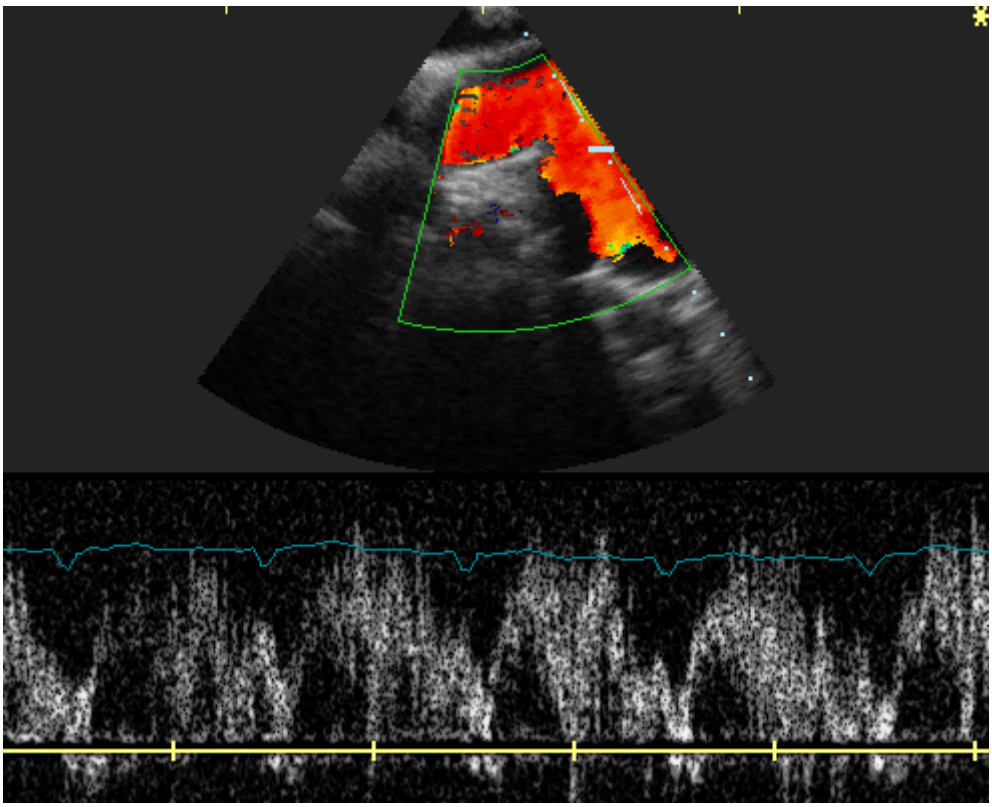

Figure 2: pulsed wave Doppler for the left upper pulmonary vein in a patient with ASD. Note the single wave pattern and the low amplitude AR waves

DISCUSSION:

In our patients with ASD who underwent TEE, a peculiar pattern of PVF was seen. Instead of showing two antegrade waves (systolic & diastolic) that is seen in the normal individual[5,6], there was a single wave that started early in systole and merged with the diastolic wave, ending at the onset of the atrial reversal wave, thereby producing a single wave pattern.

This phenomenon has not been previously studied in great detail. There are only few small studies showing the same findings [1-4]. In one study[3], it was found that the pulmonary venous flow pattern reverted to normal in three patients who were re-examined after surgical closure of the defect. None of our patients underwent TEE postoperatively to confirm their findings.

This finding, which is a fairly novel one, would be expected to cast further light onto the pathophysiology of the interatrial shunt in patients with ASD.

Using hemodynamic recordings, Levin *et al.*[7] demonstrated that the left-to-right shunt in ASD occurs as a pulsatile flow associated with an LA-to-RA gradient. This gradient was shown to be at its maximum during the interval of the latter half of ventricular systole and the first portion of diastole. Using quantitative cineangiography, the authors also showed that the major shunt across the defect occurred during those intervals of prolonged pressure gradient. Therefore, normally blood flow towards the LA decelerates or ceases at the end of systole due to the rise in LA pressure and equilibration of pressures between the LA and pulmonary veins[8]. In ASD, however, the persistent pressure gradient between the LA and RA at the end of systole is translated into a pressure gradient between the pulmonary veins and RA. This gradient is expected to cancel the deceleration or cessation of flow that occurs normally towards the end of systole[9]. Instead, blood continues to flow through the pulmonary veins at the end of systole, producing an added flow wave that is superimposed on the original flow wave tracing, thereby merging the two waves together in a single and continuous wave. With this concept in mind, several questions still remain to be addressed. First, whether this pattern of PVF can be obtained in ASD of any size or even in patent foramen ovale, since many investigators found no correlation between the magnitude of the shunt and the size of the interatrialseptal defect [10]. Second, it is not known whether the same pattern of flow can be obtained in ostiumprimum ASD, which is usually associated with more significant mitral regurgitation, tricuspid regurgitation, and LV to RA shunt [11]. Our study did not include patients with ostiumprimum defect. Third, it would be interesting to see the pattern of PVF in case of ASD complicated by pulmonary hypertension or associated with pulmonary stenosis and RV failure. In these cases, the main determinant of the shunt magnitude, i.e. the compliance of the right ventricle [12], is reduced. Fourth, the effect of mitral regurgitation (which is a frequent association with ASD[13]) on the pattern of PVF needs further study, especially in cases of severe MR. Fifth, whether there is any correlation between the mean PVF velocity and the magnitude of the shunt or the size of the defect. And finally, the diagnostic significance of this finding also needs further elucidation. Saric*et al.* conclude that the finding of a single continuous wave pattern should alert the operator to the presence of an ASD when the defect cannot be visualized directly. However, we believe that to answer all the questions a larger study with a diverse patient population is needed.

References:

1. Saric M, Applebaum RM, Phoon K, Katz ES, Goldetein SA, Tunick PA, Kronzon I: pulmonary venous flow in large, uncomplicated atrial septal defects:Am SocEchocardiog 2001; 14: 368-390.
2. Dass S, Chockalingam V, Alagesan R, Subramanian T, Jaganathan V, Etangovan S, Rajesekar MA, Muthukumar D: pulmonary venous Doppler flow pattern in atrial defect: a new diagnostic marker. Echocardiography2005;1: 9-13.
3. Lam YY, Fang F, Yip GW, Li Za, Yang Y, Yu CM. New pulmonary vein Doppler echocardiographic index predicts significant interatrial shunting in secndum atrial septal defect. Int J Cardiol. 2011 (pubmed ahead of print: www.ncbi.nl)
4. Schroh AM, Lagheza LB, Doninguez PJ, Brandan V, Nento DE, Alvarez E, Farinelli CE: Pattern of pulmonary venous flow in patients with ostiumsecundum atrial septal defect. Eur J Echocardiogr2009; 2: 244-9.
5. Castello R, Pearson A, Lenzen P, Lavovitz AJ: Evaluation of pulmonary venous flow by transesophageal echocardiography in subjects with normal heart: A comparison with transthoracic echocardiography. J Am CollCardiol 1991; 18: 65-71.
6. Bartzokis T, Lee R, Yoeh TK: Transesophageal echocardiographic assessment of pulmonary venous flow patterns. AmSocEchocardiog 1991;4: 457-62.
7. Levin A, Spach MS, Boineau JP, Canet RV: Atrial pressure-flow dynamics in atrial septal defects (secundum type): Circulation 1968; 37: 475-488.
8. Munagala VK, Jacobsen SJ, Mahoney DW, Rodeheffer RJ, Bailey KR, Redfield MM: association of newer diastolic function parameters with age in healthy subjects: a population-based study. J Am SocEchocardiogr 2003;16: 1049-56.
9. Campbell: natural history of atrial septal defect. Br Heart J 1970; 32: 820-7.
10. Hoffman JIE, Rudolph AM, Danilowicz D: left-to-right atrial shunts in infants. Am J Cardiol 1972; 30: 868-73.
11. Smallhorn JF: cross-sectional echocardiographic assessment of atrioventricularseptal defect: basic morphology and preoperativerisk factors. Echocardiography 2001; 18: 415-28.
12. Joffe HS: effect of age on pressure flow dynamics in secundum atrial septal defect. Br Heart J 1984; 51: 469-74.
13. Leachman RD, Cokkinos DV, Cooley DA: Association of ostiumsecundum atrial septal defects with mitral valve prolapse. Am J Cardiol1976;38: 167-81,.