Echocardiograph Pulmonary Venous Flow Patterns in Congenital Heart Defects with Increased Pulmonary Flow

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Summary

Objective: **To describe pulmonary venous flow patterns using transthoracic echocardiograms on children suffering from different congenital heart defects with increased pulmonary flow.**

Methods: **Prospective study and consecutive selection of children suffering from congenital heart defects with increased pulmonary flow. The transthoracic, apical view, Doppler echocardiogram was used, positioning the sample-volume at the lower left pulmonary vein, 4mm from its junction with the left atrium. The data analyzed included: dominant systolic or diastolic pulmonary venous flow and atrial contraction waveform characteristics, designated as "A" for absent and "R" for reversed.**

Results: **The study included twenty-nine patients with a mean age of 29.9 ± 58.9 months, suffering from the following congenital heart conditions: interatrial and interventricular communication defects, patent ductus arteriosus, atrioventricular septal defects, total transposition of the great arteries and truncus arteriosus. All the patients presented a continuous pattern of high velocity pulmonary venous flow. Nine patients presented a dominant systolic waveform (31%), eighteen presented a dominant diastolic wave form (62%) and 2 patients had systolic and diastolic wave forms of equal amplitude (7%). Six patients (21%) presented a R atrial contraction waveform and 23 (79%) presented an A atrial contraction waveform.**

Conclusion: **Congenital heart diseases with increased pulmonary flow present a continuous pattern of high velocity pulmonary venous flow with alterations mainly in the atrial contraction reversal pattern.**

Key words: **Pulmonary veins; heart defects, congenital; echocardiography.**

Introduction

The increased pulmonary flow observed in some congenital heart defects can alter pulmonary venous flow patterns on a Doppler assessment. According to some previous studies, these conditions present increased systolic and diastolic waveform velocities and a slower or nonexistent atrial contraction waveform[1-3].

Recently, some theories have been formulated to explain these alterations in interatrial communication.[1] However, other studies demonstrate that this phenomenon is not exclusive to this defect, as it is observed in other situations that also cause increased pulmonary flow[2,3].

The objective of this study was to investigate and describe the pulmonary flow pattern obtained during Doppler studies for various heart defects that cause increased pulmonary flow.

Methods

A consecutive and prospective selection of patients with increased pulmonary flow defects that had been detected on a conventional transthoracic echocardiography was conducted (table 1). The first 18 patients were selected from a study that analyzed the Qp/Qs ratio and pulmonary vascular resistance (PVR) using catheterization[4]. Eleven other patients were added later in order to exclusively analyze pulmonary venous flow characteristics.

The echocardiographic study was conducted with commercially available equipment, complete with 2.5 MHz and 5.0 MHz transducers. A two dimensional study was conducted for anatomical definition and a pulse-wave Doppler was used to map the flows in color for analysis. In all the tests the pulse-wave Doppler sample volume was placed in the lower left pulmonary vein, 4mm from its junction with the left atrium as described previously[5]. The tests were recorded on video tapes for later analysis.

Qualitative analysis of the flow patterns was conducted, placing emphasis on determining the component with the greatest amplitude, that is, whether the systolic (S) or diastolic (D) waveform was dominant or if the amplitude of both was similar or equal (SD). The atrial contraction waveform presence and direction were also determined and have been designated as R when the flow was reversed or flowing in the opposite direction of the dominant flow, as expected in normal situations, and A when it was absent or undetectable.

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Table 1 - Patient data from the study, distributed by diagnosis, pulmonary flow patterns observed during the Doppler study and hemodynamic data obtained during catheterization

		Pulmonary flow pattern on the pulse-wave Doppler		Catheterization Data	
Patient	Diagnosis	Dominant Wave	Atrial Contraction Wave	Qp/Qs	PVR
01	IVC	D	R	4.2	1.1
02	IVC + IAC	S	A	2.9	4.9
03	TGA + IVC	S	A	3.8	3.5
04	AVSD+ PDA	D	A	4.7	3.4
05	IVC + PFO	D	A	2.6	4.0
06	PDA+ IAC	S	A	2.7	4.8
07	PDA+ IAC	D	A	4.7	3.5
08	IVC	S	A	1.2	1.5
09	PDA	D	R	1.2	1.4
10	IVC	D	A	1.5	9.5
11	AVSD	S	A	6.2	1.5
12	AVSD	S	A	4.9	3.6
13	PDA	D	A	1.7	8.3
14	AVSD	S	R	2.6	8.3
15	TA I	S-D	R	2.1	8.1
16	PDA + IAC	D	A	3.6	5.3
17	DORV	D	R	2.6	8.0
18	IVC + IAC	D	A	4.1	6.0
19	IVC + PFO	D	A	-	-
20	IAC	D	A	-	-
21	PDA	S	A	-	-
22	PDA + E	D	A	-	-
23	PDA	D	A	-	-
24	IVC	S-D	A	-	-
25	AVSD	D	A	-	-
26	IVC	S	R	-	-
27	IVC	D	A	-	-
28	IVC	D	A	-	
29	IVC	D	A	-	-

Where IVC - Interventricular communication; PDA - Patent ductus arteriosus; AVSD - Atrioventricular septal defect; IAC - Interatrial communication; TA I - Truncus arteriosus type I; TGA - Transposition of the great arteries; PFO - Patent Foramen ovale; DORV - Double outlet right ventricle; E - Einsenmenger complex. D - Diastolic; S - Systolic; R - reversed; A - absent; Qp/Qs - pulmonary flow/systemic flow ratio; PVR - Pulmonary vascular resistance.

Before beginning the study, the investigation protocol was approved by the Research Ethics Committee of the institution.

Statistical analysis - Fisher's exact test was used to analyze the relation between: R or A atrial contraction waveform and the dominant venous flow waveform for the entire group, as well as PVR (<3W or ≥ 3W) and the Qp/Qs ratio (< 1.5 or ≥ 1.5) in the first 18 patients.

Variations were considered significant when they were less than or equal to 0.05 or 5% ($p \leq 0.05$).

Original Article

Results

Twenty-nine patients were included in the study with a mean age of 29.9 ± 58.9 months. The congenital defects encountered were: interventricular communication in 13 patients, which was isolated in 9 patients and associated with total transposition of the great arteries in one patient, with interatrial communication (IAC) in two patients and patent Foramen ovale in two patients; one patient had isolated IAC; nine patients had patent ductus arteriosus, which was isolated in 5 patients and associated with ICA in 3 patients and with atrioventricular septal defect (AVSD) in 1 patient; four patients had isolated AVSD; one patient had a double outlet right ventricle and one patient had truncus arteriosus (Table 1).

In all cases the pulse-wave Doppler study of the lower left pulmonary vein showed negative deflections in the opposite direction of the transducer location and continuous recording of the systolic and diastolic waves, with no clear return to the outer contour baseline. Nevertheless, it was possible to determine the dominant systolic or diastolic waveform from the velocity of the pulmonary venous flow waves. Analysis of the dominant flow waveform revealed that the maximum velocity was systolic in 9 patients (31%), diastolic in 18 patients (62%) and had equal systolic and diastolic amplitudes in 2 patients (7%). Patient number 22 who was diagnosed with patent ductus arteriosus and Eisenmenger's complex, presented a clearer dominant diastolic component and better defined contours than the other patients. The atrial contraction waveform was considered A in 23 patients (79%) and R in 6 patients (21%). (Table 1, Figures 1 and 2). In 8 of the 27 cases where the atrial contraction waveform was not detected, a deflection in the same direction as the systolic and diastolic waveforms was observed in the location corresponding to the atrial contraction waveform as defined by the electrocardiogram (Figure 3). From the 9 patients with dominant systolic flow, 2 presented a R atrial contraction waveform and 7 presented an A atrial waveform; from the 18 patients with a dominant diastolic waveform, 15 presented an A atrial contraction waveform and 3 presented a R atrial contraction waveform (NS; p = 1.0). The two patients with systolic and diastolic waveforms of equal amplitude were excluded from the statistical analysis, one with a R atrial contraction waveform and the other with an A atrial contraction waveform. In the 18 patients studied by catheterization, PVR was <3W in 2 patients with A atrial contraction and 2 patients with R atrial contraction; PVR was ≥ 3W in 3 patients with R atrial contraction and 11 with A atrial contraction (NS; p = 0.5).

In respect to the Qp/Qs ratio, this relation was <1.5 in 1 patient with A atrial contraction and 1 with R atrial contraction; Qp/Qs was ≥1.5 in 4 patients with R atrial contraction and 12 patients with A atrial contraction (NS; p = 0.5).

Only five of the heart defects presented a R atrial contraction pattern: AVSD, PDA, DORV and truncus arteriosus, with one case each and IVC, with two cases.

Discussion

Pulmonary venous flow pattern alterations have been described in association with different heart defects. Agata et al[6] analyzed flow variations during the first hours after birth

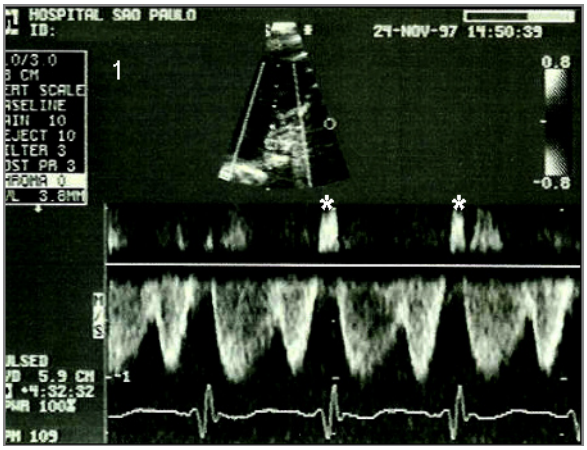

Fig. 1 - Echocardiograph image showing continuous pulmonary venous flow, with systolic and diastolic waveforms of equal amplitude and the reversed atrial contraction pattern.

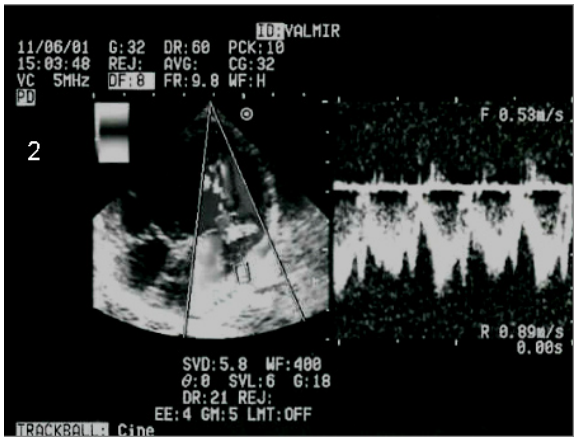

Fig. 2 - Echocardiograph image showing continuous pulmonary venous flow, with a dominant systolic waveform and no reverse atrial contraction flow.

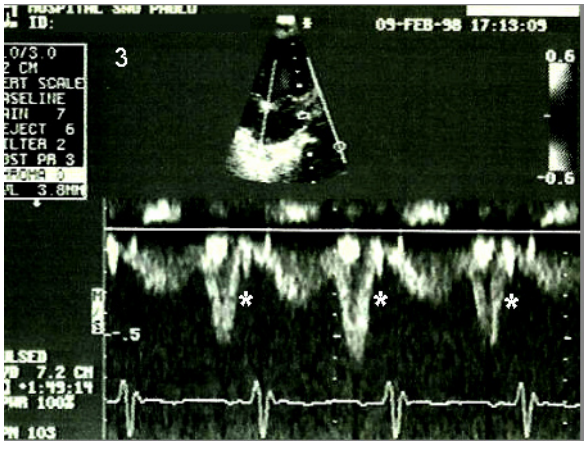

Fig. 3 - Echocardiograph image showing continuous pulmonary venous flow, with a dominant diastolic waveform and the image assumed to be the atrial contraction waveform in the same direction as the dominant flow.

and reported that the pattern observed 1, 4 and 8 hours after birth were continuous, with greater amplitude and no velocity deceleration or reversal during atrial contraction. The authors explain that this phenomenon is the result of increased pulmonary arterial flow due to the diminished pulmonary vascular resistance and increased flow produced by patent ductus arteriosus. A similar study conducted later by Hong & Choi observed greater pulmonary venous flow velocity during the first few hours after birth in comparison to the velocities obtained from term fetuses.[3] This flow pattern was continuous and of high velocity; however, it was possible to clearly define the systolic and diastolic waveforms that up to the end of the first week after birth presented a progressive deceleration, and then reached normal velocities for the age. According to the authors, the atrial contraction waveform was rarely observed before the first week of life. After this timeframe there was a progressive interruption of the continuous flow pattern and it was observed more frequently, suggesting that this flow pattern can be due to three factors: the absolute increase of pulmonary venous flow, the shunt across the arterial canal and the low capacitance of the pulmonary venous system.

Saric et al[7], conducted transesophageal echocardiograms on 22 patients diagnosed with interatrial communication and observed a continuous pulmonary venous flow pattern comprised of a single anterograde waveform, with a diminished or nonexistent reversed atrial contraction waveform that normalized after surgical closure of the defect. The authors explain that these alterations are a result of the constant flow from the left to right atrium during the entire cardiac cycle[7], keeping in mind that the continuous venous flow pattern from the pulmonary veins is similar to interatrial communication flow. The same atrial relationship caused by the defect would be responsible for the diminished or nonexistent reversed atrial contraction waveform. Therefore, the pulmonary venous flow in these patients would be less dependent on the intraventricular pressure variations; there exists, during the atrial contraction, a preferred flow directed towards the right atrium in relation to the pulmonary vein itself. Chockalingam et al[1] recently confirmed these same upper right pulmonary vein flow alterations in patients with interatrial communication using a transthoracic echocardiogram[1].

While this explanation is satisfactory for interatrial communication, it cannot be applied to the cases of patent ductus arteriosus[6], interventricular communication[2] or other heart defects[5].

Our results demonstrate that even though the pulmonary venous flow was continuous, the diastolic velocity component was dominant, a phenomenon previously observed in symptomatic patients diagnosed with interventricular communication and a Qp/Qs ratio ≥1.2, which appears to be related to the increased pulmonary flow and reduced left atrial complacency that increases the left atrial venous pressure "v" waveform[2].

In a previous publication we proposed a new echocardiograph signal to detect increased pulmonary flow[5], characterized by the dilation of the lower left pulmonary vein, which, in these situations, is easily seen on a transthoracic study both through the two dimensional test and the color flow mapping. At that time, it was noted that the pulse-wave Doppler volume sample can be placed in the vein without difficulty improving, obviously, the quality of the waveform images obtained with clearly defined contours when compared to the images using the right upper pulmonary vein, in which in situations of increased pulmonary flow, the ample dispersion of the blood flow can enter the left atrium without adequate definition of the wave contours or maximum velocity. In that study, it was observed that the velocity, and particularly the time – velocity integrals of the pulmonary venous flow were elevated in situations of increased flow when compared to normal individuals (25.0 ± 4.6 cm and 14.8 ± 2.1 cm, respectively, $p = 0.0001$)[5].

In the present study, the pulmonary venous flow waveform components' characteristics were studied for different clinical situations and heart defects. In all cases, a continuous venous flow pattern with increased velocity and a dominant diastolic component was observed. In relation to the atrial contraction waveform, it was reversed in only 7% of the cases. This atrial contraction waveform characteristic was not associated with dominant systolic or diastolic wave flows, PVR or the Qp/Qs ratio.

It is not yet known if the volume sample location in the pulmonary vein, approximately 4mm from its junction with the left atrium, can cause the atrial contraction component to diminish or disappear even in normal individuals[8]. However, studies that have analyzed pulmonary venous flow in the upper right pulmonary vein at its junction with the left atrium also present this characteristic[1-3,6].

An interesting aspect observed in 8 patients in this study, is the image that is assumed to be atrial contraction that presents flow in the same direction as the dominant venous flow (figure 3). This is a paradoxal situation which is completely different from the normal reversal pattern. Even though all the contours of this image are related to atrial electrical activation on the electrocardiogram, the origin is difficult to explain in a mechanical event such as atrial contraction. A possible explanation was previously suggested by Smallhorn et al[8] in an echocardiography study involving patients with different hemodynamic situations, including reduced and increased pulmonary flows and post-surgery conditions of different types of pulmonary cavity anatomosis, while studying lower left pulmonary vein flows. The authors indicate that in this situation, artifacts originating from the atrial wall are very common and can lead to a false interpretation of the atrial contraction wave[8].

With the exception of the reverse "a" wave, caused by atrial contraction, currently there is no consensus regarding the etiology of other pulmonary venous flow waveforms. Experimental studies in animals show that these flow waves could be secondary to pulmonary arterial pulse transmission ("forward-traveling compression wave or *vis-a-tergo*") through the capillary bed[9-13] or analogous to systemic venous flow, that exclusively rely on pressure variations ("backward-traveling expansion wave or *vis-a-fronte*") in the left atrium or ventricle[14-18]. Probably the first systolic anterograde component is associated with a suction effect from the left atrium and ventricle during ventricular contraction, whereas the second systolic anterograde component could be the result of pulse pressure propagation from the right ventricle[19-22]. Usually the systolic pulmonary venous flow component is related to

atrial relaxation and to factors such as: pressure level, atrium complacency, whereas the diastolic component, particularly the transmitral filling pattern isrelated to atrial pressure, ventricular relaxation and myocardial viscoelastic forces[23].

The preload increase produced by intravenous liquid infusion increases the systolic, diastolic and atrial contraction velocities of pulmonary venous flow in humans and animals[23,24], in a model similar to congenital heart defects with increased pulmonary flow. Therefore, the continuous flow observed in pulmonary veins could be the exclusive result of a mechanical factor related to the elevated blood volume and venous pressure during the cardiac cycle, increasing the gradient between the pulmonary veins and left atrium, with no direct relationship to the factors that determine normal flow patterns, or in other words, does not depend on ventricular function. However, this elevated preload should also cause the atrial contraction waveform to increase, unless the elevated pulmonary venous pressure in these situations surpasses the pressure in the left atrium, even during the atrial contraction timeframe, allowing the maintenance of the anterograde flow during the entire cardiac cycle.

The results of the present study show that in situations of increased pulmonary flow associated with some congenital heart defects, the pulmonary venous flow presents significant variations in relation to the normal pattern, characterized mainly by the absence of the biphasic pattern, with no return to the baseline between the systolic and diastolic waveforms and the tendency to lose the reverse component during atrial contraction.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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