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Relationship Between Pulmonary Venous and Transtricuspid Flows in Patients with Chronic Rheumatic Heart Disease

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KRONİK ROMATİZMAL KALP HASTALIĞINDA PULMONER VENÖZ AKIM VE TRİKÜSPİD AKIMLARI ARASINDAKİ İLİŞKİ

Pulmoner venöz akım velositeleri sol atrium fonksiyonu ve mitral kapak akım velositesi ile ilişkilidir. Pulmoner venöz akım değerlendirmeleri çeşitli kalp hastalıklarında sol ventrikül fonksiyonlarını karakterize etmeye yardım eder. Pulmoner venöz akım velositeleri sol ventrikül fonksiyonunu değerlendirmek için sınırlı sayıda çalışmada kullanılmakla beraber, triküspid akım velositesi ve sağ atrium fonksiyonunu değerlendirmek için şimdiye değin kullanılmadı. Çalışmada 21 kronik romatizmal kalp hastalığı olan olguda pulmoner venöz akımı etkileyen faktörler ve pulmoner venöz akım velositelerinin triküspid kapak akım velositeleri ve sağ atrium fonksiyonları ile ilişkisi araştırıldı. Pulmoner ven diastolik akım hızı ve akım zaman süresi, ortalama triküspid basıncı ile, pulmoner ven sistolik akım hızı ortalama triküspid basıncı ile sistolik pulmoner ven akım süresi ise ortalama triküspid basıncı ve erken diastoldeki en yüksek triküspid akım hızı ile korelasyon içerisinde idi. Atrial kontraksiyondaki reserve pulmoner venöz akım süresi atrial kasılmadaki en yüksek triküspid akım hızı, sağ atriyum maximum çapı, sağ atriyum maximum alanı, sağ atriyum ejeksiyon fraksiyonu ile uyumlu iken, akım hızı triküspid akım hızı, izovolumetrik relaxasyon zamanı ve ortalama basınç ile korele bulundu. Bu sonuçlar pulmoner venöz akım velosite ve akım sürelerinin sağ ventrikül diastolik dolumu, sağ atrium alan ve fonksiyonu ile bağımlı olduklarını göstermektedir.

Anahtar kelimeler: *Pulmoner ven akım hızı, triküsid kapak akım hızı, kronik romatizmal kalp hastalığı, sağ atriyum.*

Pulmonary venous flow is pulsatile and has been related to the left atrial pressure, mitral valve function and left atrial compliance [1,2]. It is not a common finding that pulmonary venous flow velocities reflect the functions of the pulmonary vascular resistance. Tricuspid flow velocity obtained with pulsed Doppler echocardiograpy is being increasingly used for the indirect evaluation of right ventricular diastolic function. Abnormal pulmonary venous flow patterns have been described for various conditions incuding constrictive pericarditis, dilated cardiomyopathy, arrhythmias and pulmonary venous obstruction. Pulmonary venous flow patterns have also been described for mitral stenosis; they have helped to assess the severity of mitral regurgitation and functional effects of mitral regurgitation and they have been used to estimate mean left atrial pressure [1-5], but have not been used to determine right ventricular function.

The aim of this study was to determine the relative importance of several proposed factors that could influence pulmonary venous flow velocity. To do this, two dimensional anatomic and Doppler tricuspid, pulmonary venous flow velocity data were compared in patients with chronic rheumatic heart disease.

PATIENTS and METHOD

Twenty-one children with choronic rheumatic heart disease were studied. Ages ranged from 7 to 16 (mean: 11.8) years. Nine of them were male and 12 female. The underlying heart diseases were mitral valve disease in 13 patients (mitral regurgitation), aortic valve disease in 3 patients (aortic regurgitation) and mixed left-sided heart disease (aortic plus mitral regurgitation) in 5 patients. Mean duration of our patients was 4.9 years. All patients were in sinus rhythm. All children enrolled in this study had normal renal function as assessed by determinations of normal serum creatinine concentration. Acute phase reactants were normal.

The control group consisted of 14 healthy children with a functional murmur. Ages ranged from 6 to 15 years (mean age 11.3 years). Six of them were male and 8 female.

A complete M mode and two-dimensional and Doppler echocardiographic examination was performed. A Toshiba non-imaging Doppler with a 3 mHz transducer for continuous and pulsed wave Doppler echocardiography was used for examinations. The length of the sample volume

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used was 5 mm. The unit was also equipped with a M-mode transducer operating at 5.25 mHz. Children were studied resting calmly in the supine position. No premedication was used.

Right ventricular and right atrial M-mode recordings were obtained from a parasternal long-axis view. Using an apical transducer position, two-dimensional images of the right ventricle and atrium were obtained at a frame rate of either 45 or 55 frames rate /sec in orthogonal apical two or four chamber views. Tricuspid flow velocity was obtained with pulsed wave technique from an apical transducer position by placing and 3 mm sample volume between the tips of the tricuspid leaflets. Pulmonary venous flow velocity was obtained from an apical or modified apical transducer positon using a 5 mm sample volume placed 1 to 2 cm proximal to the left atrium in the rigth superior pulmonary vein. For the measurement of the right ventricle isovolemic relaxation time (IVRT), tricuspid flow velocities were recorded together from an apical transducer position using continious and Doppler techniques and a paper speed of 100 mm/sec [2,7].

Echocardiographic data: Maximum and minimum right atrium areas and dimensions were identified from the four chamber views. Atrial ejection fraction (EF) for both views were calculated as right atrial volume (max)-right atrial volume (min)/right atrial volume (max) and were also averaged. Atrial fractional shortening was calculated as right atrial dimension (max)-right atrial dimension (min)/right dimension (max)X 100.

The tricuspid flow velocity variables measured are shown in Figure I. These include IVRT, peak tricuspid velocity in ealy diastole (E) peak tricuspid flow velocity at atrial contraction (A), tricuspid acceleration time (Tri at), and tricuspid deceleration time (Tri dt).

The E/A ratio was calculated in all patients. Valvular regurgitation was graded using Doppler criteria [7].

The pulmonary venous flow velocity variables measured are shown in figure 2. These include peak forward velocity (PVs) and velocity time integral (PVs VTI) during ventricular systole, peak forward velocity (PVd) and velocity integral (PVd VTI) during ventricular diastole and peak reserve flow velocity (PVa) ad velocity time integral (PVa VTI) during atrial contraction.

Statistical analysis: All values were expressed as mean ± SD (standard deviation). Statistical analysis with SPSS correlation coefficients were calculated to relate echocardiographic parameters. P value <0.05 was considered significant. Comparison of the control and patient groups were performed by using t test.

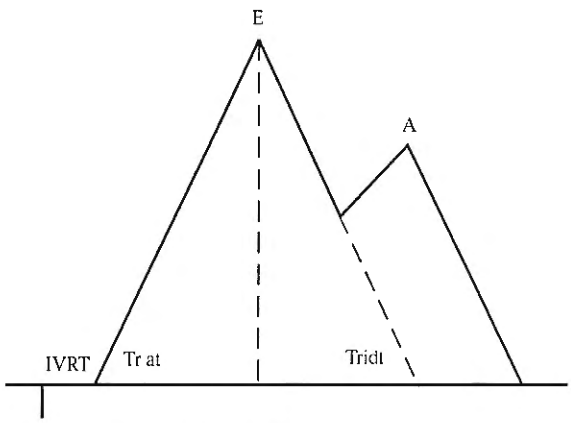

Figure 1. Schema of tricuspid flow velocity. The tricuspid variables measured in this study included peak tricuspid flow velocity in early diastole (E), peak tricuspid flow velocity at atrial contraction (A), tricuspid acceleration time (Tr at), tricuspid deceleration time (Tr dt), and the time interval between aortic valve closure click and the start of tricuspid flow (IVRT).

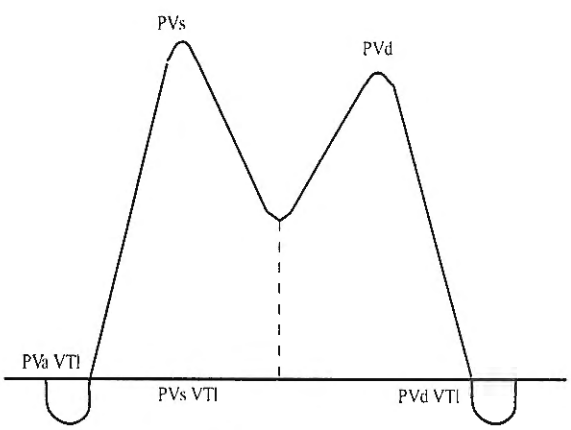

Figure 2. Schema of pulmonary venous flow velocity. Flow above the zero baseline represents forward flow into the left atrium. Flow below the zero baseline represents reserve flow associated with atrial contraction. Variables measured included peak pulmonary venous flow velocity during ventricular diastole (PVd), peak reserve pulmonary venous flow velocity associated with atrial contraction (PVa), the velocity time integral of pulmonary venous flow during ventricular systole (PVs VTI), the velocity time integral of pulmonary venous flow during ventricular diastole (PVd VTI), the velocity time integral of pulmonary venous flow during at the time o atrial contraction (PVa VTI).

RESULTS

Table 1 shows right atrial maximal and minimal areas and dimensions, ejection fraction and fractional shortening from the apical four-chamber view. Tricuspid flow velocity variables are shown in Table 2 and pulmonary venous flow velocity variables are shown in table 3. There were 13 patients with mitral regurgitation, 3 patients with aortic regurgitation and 5 patients with aortic plus mitral regurgitation.

Relation between pulmonary venous flow velocities and other measured variables: These relations are shown in Tables 4 and 5. There were relations for pulmonary venous diastolic flow and VTI with mean pressure, pulmonary venous systolic flow velocity with mean presure and VTI with mean pressure and Ev. Pulmonary venous flow velocity reversal atrial contraction VTI with Av, RA dimension max, RA

Table 1: Echocardiographic two-dimensional variables for the study and control groups

	RA area max cm²	RA area min cm²	RA dimen. max mm	RA dimen. min mm	RA EF %	RA FS %
Patients n:21	13.68 ±3.69	8.69 2.19	36.47 ±6.15	25.94 ±4.67	56.71 ±6.39	31.85 ±6.29
Control n:14	11.74 ±2.09	6.53 ±1.53	33.09 ±3.19	23.86 ±3.16	59.05 ±6.98	31.85 ±6.29
	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05

RA: right atrium
EF: ejection fraction
FS: fractional shortening
dimen: dimension.

Table 2: Tricuspid velocity variables for the study and control groups.

	Mean pres mmHg	Ev cm/sec	Av cm/sec	E/A ratio	Tri at msec	Tri dt msec	IVRT msec
Patients n:21	0.77 ±0.38	70.8 ±18.2	48.0 ±16.4	1.53 ±0.43	97.42 ±18.27	138.0 ±22.23	64.42 ±7.42
Control n:21	0.46 ±0.08	75.9 ±17.8	46.3 ±8.71	1.68 ±0.42	102.0 ±27.47	128.8 ±32.38	61.14 ±6.27
	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05

Mean pres: mean pressure, Ev: peak tricuspid flow velocity in early diastole
Av: peak tricuspid flow velocity at atrial contraction, Tri at: Tricuspid acceleration time
Tri dt: Tricuspid deceleration time, IVRT: isovolumetric relaxation time

area max, RA EF, RA FS and pulmonary venous flow velocity reversal atrial contraction velocity with IVRT, Av, mean pressure. There were no statistically significant correlations between the other parameters of pulmonary venous velocities, tricuspid flow velocities parameters and right atrial parameters.

All parameters were compared with the control group. RA areas (min) were statistically significant. There was no significant difference between patients and the control group.

DISCUSSION

Blood flow from the the lung into the left ventricle involves pulmonary venous flow, left atrial contraction, and relaxation as well as flow across the mitral valve. By examining pulmonary vein velocities in conjunction with mitral velocities, a complete assessment can be made of the filling characteritics of the left side of the heart [1,2,4,8]. Studies of normal patterns of pulmonary venous flow using transthorasic Doppler echocardiography have demonstrated

Table 3: Pulmonary velocity variables for the study and control groups.

	PVa cm/sec	PVA VTI cm	PVs cm/sec	PVs VTI cm	PVd cm/sec	PVd VTI cm
Patients n:21	21.4 ±14	12.4 ±4.0	49.7 ±18.7	20.08 ±6.79	48.8 ±1.45	24.8 ±6.2
Control n:14	16.9 ±6.0	12.8 ±2.08	49.5 ±3.4	16.6 ±6.44	50.54 ±2.3	24.0 ±5.15
	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05

PVs: peak pulmonary venous flow velocity during ventricular systole
PVd: peak pulmonary venous velocity flow during ventricular diastole
PVa: peak reserve pulmonary venous flow velocity during atrial contraction
PVs VTI: the velocity time integral of pulmonary venous flow during ventricular systole
PVs VTI: the velocity time integral of pulmonary venous flow during ventricular diastole
PVs VTI: the velocity time integral of peak reserve pulmonary venous flow during atrial contraction

Table 4. Correlation coefficients between pulmonary venous velocity variables and echocardiographic variables.

	PVa	PVa VTI	PVs	PVs VTI	PVd	PVd VTI
RA dimension max.	0.095	0.142**	-0.013	0.002	0.002	0.048
RA dimension min.	0.048	0.072	-0.013	0.012	-0.023	0.026
RA area max.	0.033	0.154**	0.008	0.365	-0.012	0.357
RA area min.	0.035	0.080	0.005	-0.012	-0.008	0.02
RA EF	0.015	-0.221**	0.017	0.042	0.021	-0.003
RA FS	0.011	-0153**	0.014	0.028	0.011	-0.001

**: *Statistically significant*

Table 5: Correlation coefficients between pulmonary venous velocity variables and tricuspid valve velocity variables.

	PVa	PVa VTI	PVs	PVs VTI	PVd	PVd VTI
IVRT	-0.054*	-0.065	0.013	-0.011	-0.075	0.044
Ev	-0.004	-0.005	0.001	0.004*	-0.001	-0.003
Av	0.032*	-0.005*	0.001	0.001	0.001	-0.001
E/A	0.002	0.009	-0.002	-0.001	0.002	0.001
Tri at	-0.013	0.489	-0.069	0.067	-0.113	
Tri dt	0.006	.524	0.063	-0.013	0.104	-0.016
mean pres.	0.005*	0.004	-0.003*	0.004*	0.003*	-0.005*

*: *Statistically significant.*

that forward pulmonary venous flow biphasic, with a systolic and a diastolic peak followed by transient reversal of flow during atrial contraction. Others, however, have suggested that in some patients forward pulmonary venous flow may be triphasic, with the ventricular systolic component divided into early late phases. These normal flow patterns, however may be markedly altered by abnormalities in cardiac rhythm and function [2,5,8,9].

Pulmonary vein diastolic flow: As reported in previous transthoracic and transosephageal studies, pulmonary venous diastolic flow velocity and velocity time integral related with peak mitral flow velocity in early diastole (both variables) were also related to the left atrial maximum diameter, maximum volume and left ventricular and diastolic pressure. Patients with increased left ventricular diastolic pressure often have an enlarged atria and an increased atrial pressure (1,2,10,11). Pulmonary venous systolic flow is believed to occur as a result of the combination of the relaxation of the left atrium alters its contraction and the concomitant descent of the atriovenricular groove associated with left ventricular systole (1,2,4,5).

Pulmonary venous flow velocity previously has been investigated in patients with atrio-ventricular block, atrial fibrillation, and dilated cardiomyopathies. Without providing specific data on left size or function, these studies have also suggested that atrial size and function is an important determinant of pulmonary venous systolic flow. For example, in patients with atrial fibrillation pulmonary vein systolic flow velocity is reduced or absent. In patients with dilated cardiomyopathy, reduced pulmonary venous systolic filling is associated with an immobile mitral annulus or mild to moderate mitral regurgitation (1,2,5,8).

It is an unusual finding that there is close correlation between pulmonary venous flow velocity and the impairment in the indicators of diastolic function of the right ventricle, namely tricuspid flow velocity, the diameters and the areas of the right atrium, and ejection fraction. In contrast, a gradual decrease in the functions of right ventricle is a common finding in pathologies involving the mitral valve in which pulmonary vascular resistance is increased. Involvement of the mitral and aortic valves resulting in valvular stenosis and insufficiency in chronic rheumatic heart disease can impair the functions of right ventricle associated with the abolishment of pulmonary vascular resistance.

We investigated the correlation between the functions of right atrium, tricuspid flow velocity, pulmonary venous flow velocity in cases with chronic rheumatic valvular heart disease. We suggested that the function of the right ventricle is not markedly impaired in chronic rheumatic valvular heart disease as evident from the findings that differences in tricuspid flow velocities and the function of right atrium between patient and control groups were found to be not significant. However a significant correlation was found between flow velocities of the tricuspid valve, right atrium and pulmonary veins.

We concluded that diastolic function of the right ventricle and the function of the right atrium were not significantly impaired in chronic valvular heart diseases of rheumatic origin but that pulmonary venous velocities correlated with the functions of right atrium and the tricuspidal flow velocities.

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