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Original Article

Intracardiac Shunts and Role of Tissue Doppler Imaging in Diagnosis and Discrimination

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Abstract

Background: *We sought to assess right ventricular (RV) systolic and diastolic functions via tissue Doppler imaging (TDI) in order to discriminate right-to-left (bidirectional) from left-to-right intracardiac shunts.*

Methods: *A tissue Doppler velocity study via Doppler echocardiography was performed in 20 patients with left-to-right shunt (without evidence of significant pulmonary hypertension) and 20 patients with right-to-left shunt or bidirectional shunt (with significant pulmonary hypertension) or Eisenmenger´s complex and 20 healthy subjects as the control group. RV myocardial performance index (MPI), S wave velocity, E wave velocity, isovolumic relaxation time (IVRT), and isovolumic contraction time (IVCT) from the lateral tricuspid annulus were measured using TDI.*

Results: *In the patients with left-to-right shunt, the tissue Doppler parameters showed higher S-wave, peak systolic(Sa)/ early contraction(Ea) , Sa/IVRT, and Sa/IVCT values; and in the patients with right-to-left or bidirectional shunt tissue, the Doppler parameters showed higher MPI and MPI/Sa value with a high specificity and sensitivity.*

Conclusion: *We conclude that an evaluation of MPI, S wave, E wave, IVRT, and IVCT via tissue Doppler echocardiography is a useful index for the discrimination of right-to-left from left-to-right and bidirectional intracardiac shunts.*

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Introduction

The right ventricle (RV) is a structurally and functionally complex chamber. This chamber propels systemic venous blood returning from the right atrium through the pulmonary vascular bed and maintains hemodynamic stability.[1-3] An

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assessment of RV function is highly important in patients with congenital heart disease. The loss of RV contractile function and pulmonary dysfunction is the main cause of exercise intolerance in patients with congestive heart failure. RV dysfunction may also cause serious problems in maintaining an adequate cardiac output after surgical correction of congenital heart disease. Tissue Doppler imaging (TDI) has provided a new insight into RV function assessment.[4-7] The purpose of this investigation was to evaluate RV systolic and diastolic functions via TDI for the discrimination of RV pressure overload from RV volume overload.

Methods

Twenty patients (12 female, average age 45±17 years) with echocardiographic signs of pulmonary hypertension (pulmonary artery systolic pressure [PASP]>70mmHg, pulmonary vascular resistance [PVR]>4 WOOD), and right-to-left or bidirectional shunt (8 patients with primum type atrial septal defect [ASD], 5 patients with patent ductus arteriosus [PDA], 3 patients with common atrio-ventricular [AV] canal, and 4 patients with inlet ventricular septal effect [VSD]) were enrolled as group I. Another 20 patients (10 female, average age 32±15 years) without echocardiographic signs of significant pulmonary hypertension (PVR<2 WOOD, 30<PASP<50 mmHg) and left-to-right shunt (7 patients with secundum type ASD, 5 patients with sinus venous type ASD, 5 patients with perimembranous VSD, and 3 patients with small PDA) were enrolled as group II. The third group consisted of 20 healthy subjects (10 female, mean age 35±16) enrolled as controls (PASP<30). The shunt direction was evaluated by the presence of pulmonary hypertension and contrast echocardiography. Exclusion criteria were hemodynamically significant left-sided valvular heart disease, left ventricle systolic dysfunction, and any rhythm other than sinus rhythm. All the patients underwent standard echocardiography and TDI. RV ejection fractions (RV EF) were estimated using Simpson's or modified Simpson's methods.[6]

We used a commercially reliable ultrasound system (GE Vivid Seven) equipped with a multi frequency phased array transducer and pulsed Doppler tissue imaging technique for transthoracic echocardiography (TTE). All the patients were in stable hemodynamic condition, and tracings were recorded during end expiration. The tricuspid annulus systolic and diastolic velocities and the time interval were acquired in apical 4-chamber views at the junction of the right ventricle free wall and the anterior leaflet of the tricuspid valve via TDI. The acoustic power, filter, and gain were adjusted for detecting myocardial velocities. All the recordings were made at a sweep speed of 50 and 100 mm/s with a simultaneous superimposed ECG.[8,9] The peak systolic (Sa) and 2 diastolic waves: early (Ea) and atrial contraction (Aa), the time between the end of Sa and the beginning of Ea (isovolumic relaxation time [IVRT]), the time between the end of Aa and the beginning of Sa (isovolumic contraction time [IVCT]), and ejection time (duration of Sa) were obtained by placing a sample volume with a fixed length of 0.5 cm at the junction of the RV free wall and the anterior leaflet of the tricuspid valve in the 2-D four chamber view via DTI (Figure 1).[10,11]

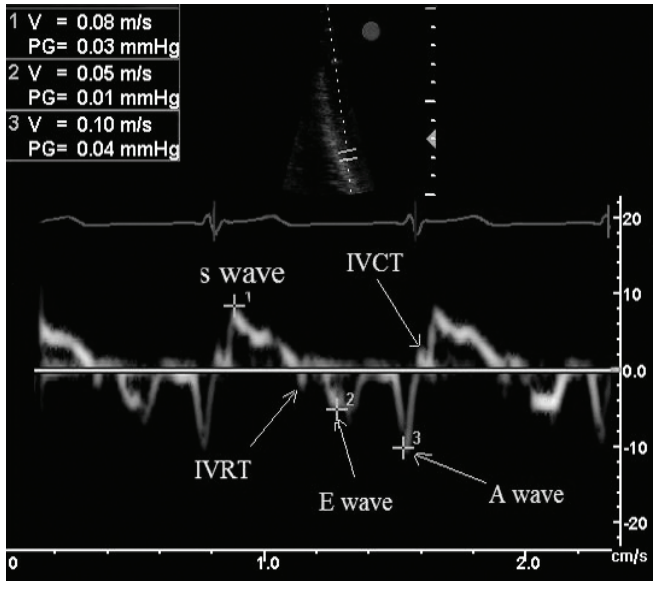

Figure 1. Illustration of pulsed tissue Doppler imaging of tricuspid valve S wave, peak systolic velocity at the anterior leaflet of tricuspid valve; E wave, peak early diastolic velocity at the anterior leaflet of tricuspid valve; A ware, a positive wave toward the left atrium at late diastole; IVRT, the time between the end of S wave and the beginning of E wave; IVCT, the time between the end of A wave and the beginning of S wave

The myocardial performance index (MPI) was calculated as (a-b/b), where a is the interval from the onset of IVCT to the end of IVRT and b is the ventricular ejection time (Figure 2).[12]

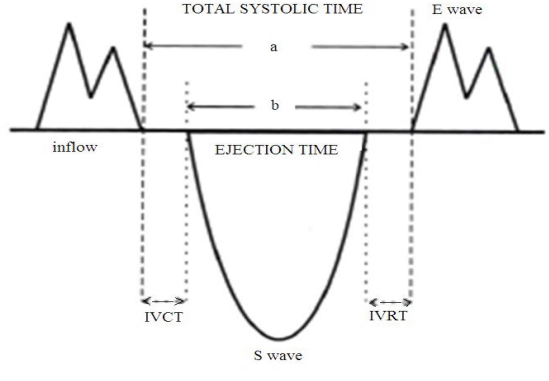

Figure 2. Myocardial performance index was calculated as (a-b/b) E wave, peak early diastolic velocity at the anterior leaflet of tricuspid valve; IVCT, Isovolumic contraction time; IVRT, Isovolumic relaxation time; S wave, peak systolic velocity at the anterior leaflet of tricuspid valve

A commercially available statistical program (SPSS

10.1 and 11.1) was used. Pearson's correlation and linear regression were plotted to show certain relationships.

A P-value less than 0.05 was considered significant. For the assessment of inter-observer variability, the mean value of the first observer was compared with that of the second observer, who was unaware of the first observer's result. The mean difference between their measurements was calculated, and the percentage of the variability was derived as the absolute difference between the measurements divided by the mean of the two observations. Intra-observer variability was also calculated using this method. Receiver-operator characteristic curves were analyzed to select the optimal cut-off values. The study protocol was approved by the Institutional Review Board of Shahid Beheshti University of Medical Sciences, Tehran, Iran.

Results

PASP was calculated according to the values obtained from the echocardiographic studies of the right heart (tricuspid regurgitation peak gradient [TRPG] + right atrium pressure [RAP]) except in the patients that were catheterized. In these patients, PASP was directly measured. RAP was estimated by the diameter of the inferior vena cava and respiratory response. For the evaluation of pulmonary vascular resistance, the following formula was employed:

[TR peak velocity/RVOT (VTI)]×10, where TR is tricuspid regurgitation, RVOT is right ventricular outflow tract, and VTI is time-velocity integral. The different diagnoses of the patients in group I (bidirectional or right-to-left shunt) and group II (left-to-right intracardiac shunt) are listed in Table 1.

The basic characteristics and standard echocardiographic parameters of the groups are listed in Table 2. In groups I and II, left ventricle ejection fractions were within normal limits. The patients with pulmonary hypertension (group I) had a high incidence of lower RV ejection fraction and lower RV stroke volume. On the other hand, RV stroke volume and RV ejection fraction in the patients with left-to-right shunt (group II) were much higher than those in the other groups.

The analysis of the tissue Doppler parameters showed that Sa and Aa velocities in group II (left-to-right shunt) were greater than Sm in group I (bidirectional or right-to-left shunt) and the control group (III) ($P< 0.0001$, $P=0.018$).

IVCT, IVRT, and MPI in group I were greater than those in the other two groups ($P <0.0001$).

Ea velocity in group I was lower than that in the other two groups ($P <0.0120$).

Sa/Ea in group II was significantly greater than that in the other two groups ($P<0.0001$).

MPI/Sa in group I was significantly greater than that in the other two groups ($P<0.0001$).

Sa/IVRT and Sa/IVCT in group II were significantly greater than those in the other two groups ($P<0.0001$).

In the patients with left-to-right shunt (group II), the RV Sa/Ea value was>1.25 with a sensitivity of 80% and specificity of 75%.

In the patients with right-to-left or bidirectional shunt (group I), the MPI/Sa value was >0.045 with a sensitivity of 85% and specificity of 83%.

In the patients with left-to-right shunt, Sa/IVRT was> 0.23 with a sensitivity of 80% and specificity of 80%.

In the patients with left-to-right shunt (group II), Sa/IVCT was >0.24 with a sensitivity of 83% and specificity of 84%.

The RV Sa/Ea value >1.25, Sa/IVRT value >0.23, and Sa/IVCT value >0.24 were useful to identify left-to-right shunt (RV volume overload) from right-to-left or bidirectional shunt (RV pressure overload); and the MPI/Sa value >0.045 was useful to identify right-to-left shunt or bidirectional shunt (RV pressure overload) from left-to-right shunt (RV volume overload).

Table 1. Diagnosis of patients with left to right shunt and bidirectional or (right to left) shunt

Groups	Diagnosis			
Right to left or bidirectional shunt	Inlet VSD 4	Common AV Canal 3	PDA 5	Primum ASD 8
Left to right shunt	Perimembranous VSD 5	Sinus venous ASD 5	Secondum ASD 7	Small PDA 3

VSD, Ventricular septal defect; AV, Atrio-ventricular; PDA, Patent ductus arteriosus; ASD, Atrial septal defect

Table 2. Basic characteristics and echocardiographic parameters of the three groups

Variable	Group I (Right to left or bidirectional shunt) N=20	Group II (left to right shunt) N=20	Group III (control group) N=20	P value
Age (y)	45±17	32±15	35±16	0.0500
Men/women	8/12	10/10	10/10	-
LV EF (%)	53±3	55±5	60±5	< 0.0001
RV EF (%)	35±17	47±17	44±12	0.0010
PASP (mmHg)	82±11	40±10	21±8	0.0001
PVR (wood)	>4	<2	<1.7	< 0.0050
RV Sa (cm/s)	11±3	15.5±3	13±2	< 0.0001
RV Ea (cm/s)	10.5±3	11.5±3	12±3	0.0120
RV Aa (cm/s)	11±4/5	14±4	11±4	0.0180
RV IVCT (ms)	68±20	55±16	63±9	< 0.0001
RV IVRT (ms)	83±25	56±16	60±13	< 0.0001
RV MPI	0.57±0/2	0.44±0.16	0.4±0.05	< 0.0001
RV Sa/Ea	1.04	1.34	1.08	< 0.0001
RV MPI/Sa (cm/s)	0.054	0.038	0.03	< 0.0001
RV Sa/IVCT(cm/s²)	0.16	0.28	0.20	< 0.0001
RV Sa/IVRT (cm/s²)	0.13	0.27	0.20	< 0.0001
Qp/Qs	≤1	>1.4**	~1	< 0.0050

*Data are presented as mean±SD
**In PDA cases Qs/Qp >1.4
LV, Left ventricle; EF, Ejection fraction; RV, Right ventricle; PASP, Pulmonary arterial systolic pressure; PVR, Pulmonary vascular resistance; Sa, Peak systolic; Ea, Early contraction; Aa, Atrial contraction; IVCT, Isovolumic contraction time; IVRT, Isovolumic relaxation time; MPI, Myocardial performance index; Qp/Qs, pulmonary to systemic flow

Discussion

Diastolic RV dysfunction (lower tricuspid valve peak E velocity in TV inflow, lower E/A velocity, and prolonged RV IVRT and IVCT) and systolic RV dysfunction (lower TV peak S wave) have been demonstrated in patients with pulmonary hypertension and in those with symptomatic congestive heart failure, even in the absence of pulmonary hypertension, suggestive of a potential role for ventricular interdependence in impaired RV filling.[13] It must be noted that significant pulmonary hypertension leads to increased IVRT, IVCT, and MPI and a decreased S wave velocity. The present study was designed to assess the potential of TDI for the provision of new information to enable a differentiation between right-to-left shunt (RV pressure overload) and left-to-right shunt (RV volume overload).

According to the Frank- Starling law, a larger heart volume increases the initial length of the muscle fibers, which increases cardiac contractility and stroke volume. This can explain why RV Sa was much larger in the RV volume overload group than that in the other groups.

MPI was defined as the sum of IVRT and IVCT divided by ejection time [(IVRT+IVCT)/ ET].

However, pure RV volume overload had no significant effect on RV MPI. The prolongation of IVRT and IVCT, obtained by tissue Doppler from the lateral annulus of the tricuspid valve, was correlated with pulmonary hypertension. It seems that RV MPI/Sa>0.045 can be used to identify RV pressure overload with an acceptable sensitivity and specificity.

There were, however, some limitations in our study. There was a significant age difference between those in group 1 and the ones in the other two groups. We believe that this is because of the late appearance of right-to-left shunt and the longer time it requires to manifest itself. In group 2, we had three cases of small PDA. In these cases, we studied ratio of systemic flow to pulmonary (Qs/Qp) instead of Qp/Qs, which was more than 1.4/1 at all times. On the other hand, only in these cases we had an LV volume overload and not RV volume overload, although the shunt direction was still left-to-right. One more limiting factor in the present study was our low sample volume.

Conclusion

We conclude that an evaluation of MPI-Sa, Sa/Ea wave, MPI/Sa wave, Sa/IVCT, and Sa/IVRT values via TDI can be useful in the discrimination between RV pressure overload and RV volume overload.