PEDIATRIC CARDIOLOGY

Regional Ventricular Wall Motion Abnormalities in Tricuspid Atresia After the Fontan Procedure

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Objectives. The purpose of this study was to determine whether wall motion abnormalities are present before or after the Fontan procedure in patients with a univentricular heart of the left ventricular type with an absent right atrioventricular valve connection (tricuspid atresia) and to assess the impact of such abnormalities on ventricular performance and clinical outcome.

Background. Normal systolic and diastolic ventricular function is critical for a successful Fontan repair. However, there have been no previous studies addressing the relation between regional ventricular function and hemodynamic factors.

Methods. Thirty-seven pediatric patients were studied with biplane ventricular cineangiography. There were 20 male and 17 female patients whose mean age at the time of the Fontan operation was 6.5 ± 3.5 years (range 2.5 to 15.6). Eighteen patients were studied preoperatively, 25 at >1 year postoperatively and 6 serially. Wall motion was assessed by a centerline

method. Normal ranges for wall motion and other variables were established from 25 normal subjects.

Results. Wall motion abnormalities were observed in 2 of 18 patients preoperatively and in 11 of 25 patients postoperatively. Age at operation and ventricular volumes did not differ between postoperative patients who had normal (group I, 14 patients) or abnormal (group II, 11 patients) wall motion. However, ventricular mass and the mass/volume ratio were significantly greater and systolic variables and cardiac index were significantly lower in group II versus group I. Two patients in group I were considered to have a clinically poor outcome (persistent heart failure), and five in group II had heart failure, including one who died late.

Conclusions. These observations suggest that postoperative regional wall motion abnormalities in this setting are not rare, may be related to excessive hypertrophy and may contribute to cardiac dysfunction and a poor clinical outcome.

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Many studies (1–12) have described abnormal hemodynamic variables before and after the Fontan procedure in patients with a univentricular connection and an absent right atrioventricular (AV) valve connection (tricuspid atresia). Factors contributing to these morphologic and functional abnormalities include preoperative chronic volume overload (1,2), cyanosis (3), perioperative myocardial damage (4), postoperative unbalanced ventricular hypertrophy (5–8) and elevated systemic vascular resistance (1,7–9). These studies (1–12) have identified both contractile and filling compromise before and after the Fontan operation. However, such abnormalities, as measured by indexes of global ventricular function, may be affected by abnormalities of regional function. In this regard, Gibson et al. (13) observed abnormal ventricular wall motion in patients with a univentricular

heart, regardless of age, volume overload or degree of cyanosis. Using the same technique, Penny and Redington (14) recently reported that such abnormalities were also observed after the Fontan procedure. Although these observations suggested that wall motion abnormalities in this setting were not rare, the relation to hemodynamic variables and clinical outcome remains uncertain. In this study, using ventricular cineangiography, we attempted to elucidate the relation among such wall motion abnormalities, cardiac performance and ventricular hypertrophy before and after creation of an atrial to pulmonary connection and atrial separation.

Methods

Study patients. From January 1984 to June 1991, 61 patients with tricuspid atresia with normally related great arteries (AV and ventriculoarterial concordance), with or without a bidirectional caval to pulmonary connection, underwent a Fontan-type procedure (right atrial to pulmonary artery or right ventricle anastomosis). There were three early deaths (5%). From this study group, 37 patients (61%) had an acceptable selective ventricular cineangiogram for volumetric and wall motion analysis. All patients were considered suitable candidates for an atrial to pulmonary

connection on the basis of preoperative hemodynamic data and conventional criteria (15). There were 20 male and 17 female patients whose mean age at operation was 6.5 ± 3.5 years (range 2.5 to 15.6). Thirty-four patients (92%) had one or more previous palliative procedures: 33 with a systemic to pulmonary shunt; 6 with a caval to pulmonary shunt; 5 with both; 1 patient each with pulmonary artery banding and Blalock-Hanlon procedure. Twenty-five patients had an atrial to pulmonary artery anastomosis, and the remaining 12 had an atrial to ventricular anastomosis. Coronary artery abnormalities and significant mitral regurgitation were not observed in this patient group.

Normal control subjects. The 25 children who served as control subjects were undergoing cardiac catheterization and were found to have hemodynamically insignificant lesions not affecting left ventricular volume and pressure variables. All 25 had a small ductus arteriosus (pulmonary/systemic flow ratio <1.5) and measurements performed 30 min after closure by transcatheter technique. There were 3 male and 22 female patients with a mean age of 4.1 ± 2.2 years (range 1.4 to 9.3).

Cardiac catheterization and angiographic studies. Adequate ventriculograms were obtained in 18 patients preoperatively, in 25 patients >1 year postoperatively and in 6 patients both preoperatively and postoperatively. The interval between preoperative catheterization and operation was 6.4 ± 4.6 months (range 1 day to 1.4 years) and an average of 2.5 ± 1.3 (range 1 to 5.3) years postoperatively. All patients had a standard electrocardiogram (ECG) before catheterization.

Ventricular mass and volumes were measured from biplane cineventriculograms in the right anterior and axial left anterior oblique projections at 60 frames/s, and cavity borders were traced manually at end-diastolic and end-systole during sinus rhythm. Ventricular end-diastolic and end-systolic volumes were calculated by the modified Simpson's rule and were corrected by regression equations established in our laboratory (16) and indexed for body surface area as the end-diastolic volume index and end-systolic volume index. Ventricular mass was calculated as previously described by Rackley et al. (17) and Sano et al. (9) and indexed to body surface area (wall mass index), expressed as g/m².

Ejection fraction and heart rate–corrected mean normalized systolic ejection rate were determined as systolic ejection phase variables. The heart rate–corrected mean normalized systolic ejection rate (MNSERc) was calculated as MNSERc = (EDV − ESV)/(EDV × SETc), where EDV and ESV are the end-diastolic and end-systolic volumes, respectively, and SETc the corrected systolic ejection time, which was obtained by dividing by the square root of the RR interval. Systemic vascular resistance index (SVRI) was calculated as SVRI = [(mAP − mRA)/CI] × 80 and expressed as dynes·s·cm^{-5}·m², where CI is the cardiac index, and mAP and mRA are the mean aortic and right atrial pressures, respectively.

Regional wall motion was analyzed by a centerline

method established by Sheehan et al. (18). In this technique, wall motion is determined clockwise from 100 chords drawn perpendicular to a centerline constructed midway between the end-diastolic and end-systolic counters. The length of each chord is normalized to yield a shortening fraction by dividing by the length of the end-diastolic perimeter (Fig. 1 and 2). In this study, end-diastolic and end-systolic outlines were superimposed at midaorta and rotated until the lines from the center of mass to the midaorta along the long axes were aligned (19). Hypokinesia was defined as present when there were >10 consecutive contiguous chords whose shortening fraction differed by >2 SD below the mean shortening fraction of normal. Mitral valve (chords 75 to 100 in the right anterior oblique projection and chords 1 to 25 in the axial left anterior oblique projection) and septal areas surrounding the position of the ventricular septal defect (chords 75 to 100 in the axial left anterior oblique projection) were not included in the evaluation. Measurements were processed on an Angiographic Ventricular Dynamics System (Siemens-Elema AB).

Statistical analysis. Data are expressed as mean value ± 1 SD. Statistically significant differences among the three groups were determined by a one-way analysis of variance (ANOVA), followed by a Bonferroni method if the ANOVA probability value was < 0.05. A chi-square test was used for comparison of categoric data.

Results

Preoperative analysis. End-diastolic and end-systolic volume indexes, stroke volume index, wall motion index and cardiac index were significantly higher than values in control subjects, reflecting the adaptive response to the volume overload (Table 1). All systolic ejection phase variables and systemic vascular resistance index were significantly decreased, whereas the mass/volume ratio remained within the normal range. Abnormal wall motion was observed in two patients (Patients 8 and 18). Both had anterolateral hypokinesia. No specific ECG findings were defined in any patient with abnormal wall motion.

Postoperative analysis. Of the 25 patients, 14 (group I) had normal ventricular wall motion, whereas 11 (group II) had abnormal wall motion (Table 2). These abnormal segments (all hypokinetic) were found in the anterolateral region in four patients, the apical region in four patients, the diaphragmatic region in two patients and the posterolateral region in one patient. Volumetric variables in group I were not different from those in control subjects. However, wall motion index and the mass/volume ratio were significantly increased (Table 3). Additionally, a low ejection fraction and elevated systemic vascular resistance index were present in group I compared with values in control subjects, further contributing to the decreased cardiac index. Similar findings were observed in group II, although end-systolic volume index, stroke volume index and heart rate–corrected mean normalized systolic ejection rate were also significantly
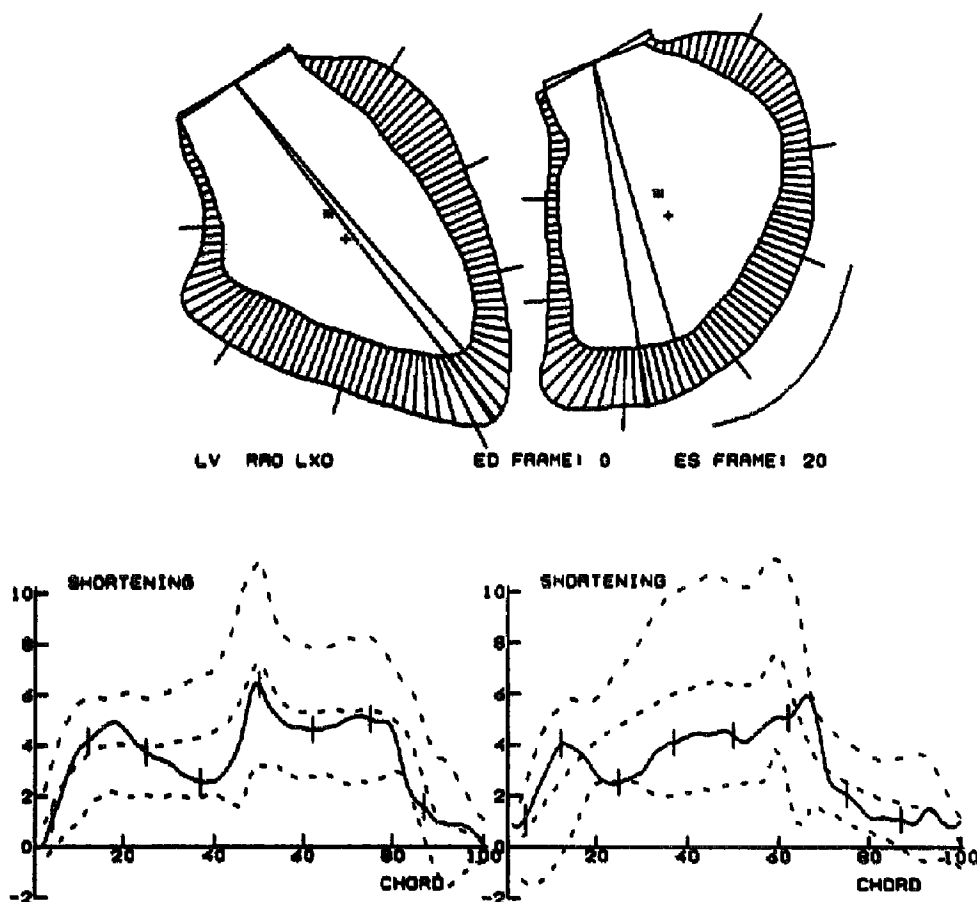
Figure 1. Patient 36: normal wall motion. Top panel, Left ventricular (LV) end-diastolic (ED) and end-systolic (ES) tracings from the right anterior oblique (RAO) (left) and axial left anterior oblique (LXO) projections (right). Motion is measured clockwise along 100 chords constructed perpendicular to the centerline. Bottom panel, Normalized motion of each chord for each projection. Solid line shows motion along each chord from the patient. Dashed lines show the mean value ± 2 SD from normal subjects. This patient has no apparent abnormal segments.

decreased compared with values in control subjects. The incidence of wall motion abnormalities was not significantly different between patients with a good or poor outcome.

There were significant volumetric and hemodynamic differences between groups I and II (Table 3). Although age at operation and end-diastolic and end-systolic volume indexes were not different between groups I and II, the wall motion index and mass/volume ratio were significantly greater in group II than in group I. Similarly, all systolic variables and cardiac index in group II were significantly decreased, and systemic vascular resistance index was significantly increased compared with values in group I. Left ventricular end-diastolic pressure was not different among the patient and normal groups. There was no difference in either total cardiopulmonary bypass or aortic cross-clamp times between groups I and II. Two patients in group I were considered to have a clinically poor outcome (persistent heart failure), and five in group II had a poor outcome, including late death in one (Patient 17) (p = 0.08).

Various ECG abnormalities were observed postoperatively in Groups I and II. However, no finding was correlated to the presence of a localized wall motion abnormality. Right bundle branch block was defined in five patients, and two of these (Patients 17 and 18) had abnormal motion. No relation was found in three patients who had surgical closure

of a ventricular septal defect and the presence of wall motion abnormalities.

In the six patients with both preoperative and postoperative studies, wall motion abnormalities were found in two, appearing in one after the procedure (Patient 17) and in the other before and after the procedure (Patient 18).

Discussion

Wall motion analysis in pediatric patients. The assessment of regional ventricular function has been well established in adults as a useful adjunct to hemodynamic evaluation in patients with ischemic heart disease (18,19) or cardiomyopathy (20). Although the importance of wall motion analysis has been recognized even for pediatric patients, few studies have been performed (13,14) because of the frequent complex ventricular topology and the need for established normal ranges. In this regard, echocardiographic normal ranges have been established for wall motion in neonates and children for a variety of congenital lesions (21–24). Although our "normal" population was not without cardiac lesions, volumetric and hemodynamic variables from this cohort correlated closely with previous normal reports in this age population (1,5,9,25), suggesting that these asso-

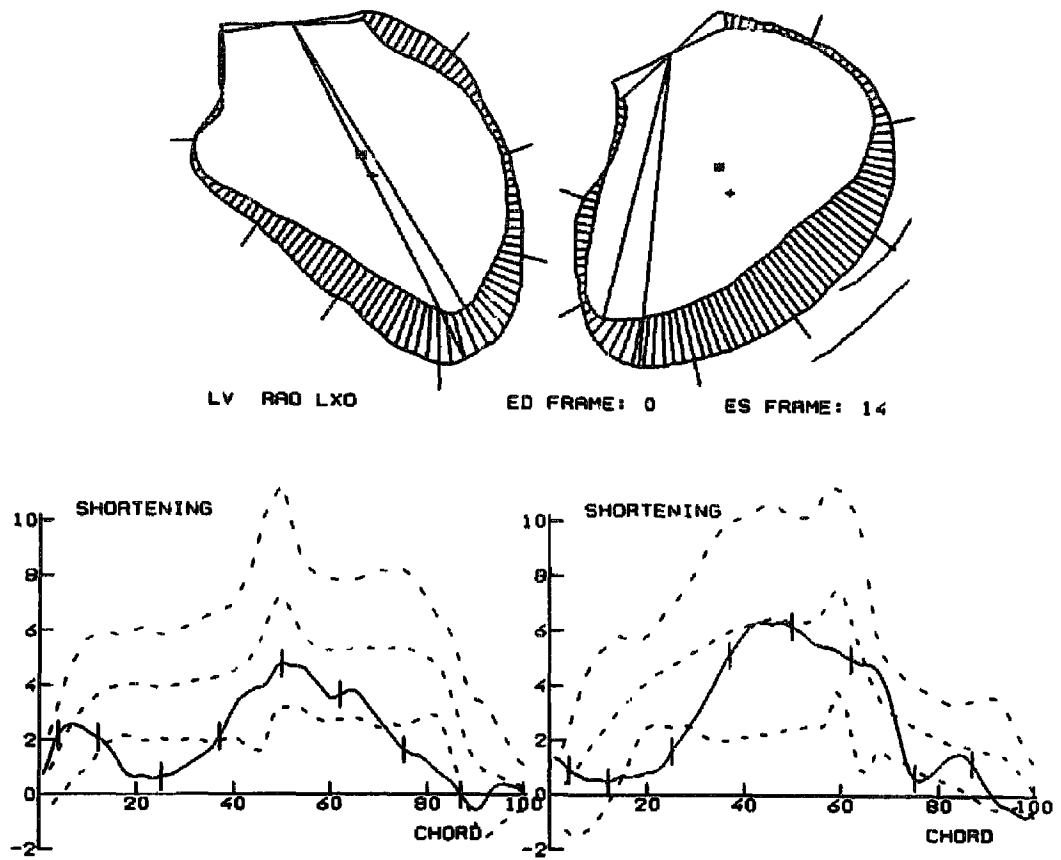LV   RAO LXO   ED FRAME: 0   ES FRAME: 14

Figure 2. Patient 34: abnormal wall motion. A significant hypokinetic segment was observed in the right anterior oblique projection (chords 15 to 38), in the anterolateral area. Abbreviations and symbols as in Figure 1.

ciated lesions did not influence left ventricular function and wall motion.

Wall motion abnormalities and hemodynamic variables in tricuspid atresia. Previous studies have reported the presence of wall motion abnormalities in patients with a univentricular heart before (13) and after creation of an atrial to pulmonary connection (14). In patients with abnormal wall motion, systolic ejection variables were significantly more depressed than in those with normal wall motion, although ventricular volumes were not different. Ventricular mass and the mass/volume ratio in patients with abnormal wall motion were also significantly increased compared with values in patients with normal wall motion or control subjects. These data suggest that postoperative wall motion abnormalities may relate to excessive ventricular hypertrophy and contribute to ejection or filling dysfunction.

In patients with a univentricular connection, a single dominant ventricular chamber functions as the only effective pump for both systemic and pulmonary circulations. Preoperatively, ventricular volume and mass are significantly increased compared with values in normal subjects (1,2,7,8) but are compensated for with a normal mass/volume ratio. After creation of an atrial to pulmonary anastomosis and atrial separation, ventricular volume overload resolves abruptly, and chamber volume rapidly decreases. However, the hypertrophied ventricular mass does not decrease in parallel, and the mass/volume ratio increases compared with

that in normal subjects (1,7,8). In patients with a poor outcome, the increased mass/volume ratio is found preoperatively, further increasing postoperatively and contributing to diastolic filling abnormalities (5,6,8,12,26). This increased mass/volume ratio, reflecting excessive hypertrophy, may combine with postoperative wall motion abnormalities, contributing to a poorer outcome.

It is not clear whether the excessive ventricular hypertrophy (increased mass/volume ratio) is directly related to the wall motion abnormality or whether there are other influencing factors. Although we did not perform myocardial perfusion studies, ECG findings suggest that these abnormal segments were not related to myocardial ischemia, corresponding to a territory of a coronary artery. Preoperative chronic volume overload and hypoxemia contribute to the environment for myocardial stress and compromised coronary flow and remain an important element leading to myocardial dysfunction in this setting (27). Furthermore, perioperative myocardial damage during cardioplegia may be enhanced in patients with hypertrophied ventricles (4), although no differences were observed in total bypass or aortic cross-clamp times in this study. A recent study

Table 1. Preoperative Data on the 18 Patients Studied Preoperatively

Pt No.	Age at Op. (yr)	Age at Study (yr)	Abnormal Segment	EDVI (ml/m²)	ESVI (ml/m²)	SVI (ml/m²)	WMI (g/m²)	WMI (g/m³)	WMI/EDVI (g/ml)	HR (beats/min)	CI (liters/min per m²)	EF (%)	MNSERc (EDV/s)	LVEDP (mm Hg)	SVRI (dynes·s·cm⁻⁵·m²)	ECG Findings	Clinical Outcome
1	13.4	12.3	—	92	35	57	116	1.26	77	4.4	62	1.64	3	1,438	LAD, BAE, LVH, flat T (V₄–V₆)	Poor
2	5.6	4.5	—	93	59	34	163	0.84	130	4.5	37	1.16	6	1,182	LAD, RAE, LVH	Good
3	2.8	2.2	—	126	77	49	100	0.79	90	4.5	39	1.13	8	996	LAD, flat T (V₅, V₆)	Poor
4	3.1	2.8	—	83	29	54	60	1.11	106	5.7	64	1.52	6	954	LAD, LVH	Good
5	3.5	3.0	—	115	54	61	89	0.77	99	5.5	53	1.98	6	944	LAD, LVH	Good
6	3.8	3.8	—	97	49	47	84	0.87	96	4.6	49	1.37	8	1,478	LAD, LVH, flat T (I, V₆)	Good
7	5.1	4.7	—	112	47	64	141	1.26	96	5.8	58	1.59	8	1,487	BAE, LVH	Good
8	5.5	5.3	Antlat	109	74	35	94	0.86	100	3.5	32	0.99	3	1,531	LAD, BAE, LVH	Good
9	2.8	2.3	—	111	51	59	94	0.83	95	5.7	53	1.68	5	716	LAD, LVH	Good
10	8.5	6.1	—	133	55	78	146	1.09	86	6.7	59	1.41	7	812	LAD	Poor
11	3.6	3.0	—	181	45	75	79	0.78	119	6.1	55	1.44	4	997	LAD, LVH, neg T (V₅, V₆)	Good
12	3.3	3.1	—	138	62	75	102	0.74	99	7.3	55	1.29	5	784	LAD, RAE, LVH	Good
13	2.5	2.0	—	98	35	55	83	0.92	103	5.7	61	1.75	8	912	LAD, BAE, LVH	Good
14	11.5	11.5	—	174	90	84	146	0.72	86	7.2	48	1.34	5	633	LAD, BAE, LVH	Good
15	2.8	2.5	—	64	21	43	88	1.57	130	5.6	67	1.82	8	1,057	RAE, LVH	Poor
16	2.5	1.8	—	139	75	64	116	0.83	110	7.1	46	1.28	5	710	LAD, RAE, LVH	Good
17	4.4	4.1	—	126	69	56	117	0.93	120	6.8	45	1.27	3	859	LAD, RAE, LVH	Poor
18	13.4	12.9	Antlat	118	72	45	142	1.20	86	3.9	39	0.88	3	1,272	LVH, neg T (V₅, V₆)	Poor
Mean	5.5	4.9		112	56	56	105	0.96	100	5.6	51	1.36	6	1,022		
±1 SD	±3.7	±3.6		±25*	±19*	±14*	±25*	±0.21	±15	±1.2*	±10*	±0.26†	±2	±268*		

*p < 0.001 versus values in control subjects. †p < 0.05 versus values in control subjects. Antlat = anterolateral; BAE = biatrial enlargement; CI = cardiac index; ECG = electrocardiographic; EDV = end-diastolic volume; EDVI = end-diastolic volume index; EF = ejection fraction; ESVI = end-systolic volume index; HR = heart rate; LAD = left axis deviation; LVEDP = left ventricular end-diastolic pressure; LVH = left ventricular hypertrophy; MNSERc = corrected mean normalized systolic ejection rate; neg = negative; Op. = operation; Pt = patient; RAE = right atrial enlargement; SVI = stroke volume index; SVRI = systemic vascular resistance index; T = T wave; WMI = wall mass index.

Table 2. Postoperative Clinical Characteristics of the Study Patients

Pt No.	Age at Op. (yr)	Age at Study (yr)	Abnormal Segment	EDVI (ml/m²)	ESVI (ml/m²)	SVI (ml/m²)	WMI (g/m²)	WMI/EDVI (g/ml)	HR (beats/min)	CI (liters/min per m²)	EF (%)	MNSERc (EDV/s)	LVEDP (mm Hg)	SVRI (dynes·s·cm⁻⁵·m²)	ECG Findings	Clinical Outcome
1	12.5	13.5	—	78	37	40	124	1.58	90	3.7	52	1.58	10	1,578	RAE, LVH, neg T (I, aVL, V₄–V₆)	Poor
2	5.6	7.3	—	51	21	30	52	1.01	100	3.0	59	1.81	4	1,760	LAD, BAE	Good
3	2.8	3.9	—	77	25	51	88	1.15	90	4.7	67	1.15	6	1,362	RAE, LVH, neg T (I, aVL, V₄–V₆)	Poor
4	3.1	4.1	—	54	17	36	77	1.43	90	3.3	67	1.43	4	1,261	LAD, BAE	Good
17	3.7	4.7	Postlat	65	42	23	75	1.14	120	2.8	36	1.14	2	1,657	LAD, BAE, IRBBB	Poor
18	13.5	15.1	Antlat	40	21	19	100	2.50	93	1.7	47	2.50	4	2,824	RAE, CRBBB, neg T (V₅–V₆)	Poor
19	5.4	9.2	—	62	32	30	81	1.29	72	2.2	49	1.29	5	2,727	LAD, BAE	Good
20	4.1	8.8	—	58	20	37	84	1.43	90	3.4	64	1.43	6	1,635	LAD, BAE, IRBBB	Good
21	6.8	9.1	—	60	30	29	58	0.97	72	2.1	49	0.97	5	3,086	LAD, BAE	Good
22	9.0	10.0	—	91	39	51	66	0.73	72	3.7	57	0.73	5	1,427	LAD, BAE, IRBBB	Good
23	5.9	9.2	—	66	38	28	108	1.62	72	2.0	42	1.62	3	2,880	LVH	Good
24	3.1	4.9	—	66	40	26	68	1.02	97	2.6	40	1.02	5	2,400	LAD, BAE	Good
25	5.0	8.5	—	72	34	37	101	1.40	85	3.2	52	1.40	8	2,275	LAD, RAE, LVH, neg T (I, aVL, V₄–V₆)	Good
26	15.6	17.7	—	69	30	38	102	1.47	90	3.5	56	1.47	5	2,217	LAD, RAE, neg T (I, II, V₄–V₆)	Good
27	8.0	11.4	—	53	22	31	63	1.17	85	2.7	59	1.17	5	2,222	LAD, BAE, neg T (V₅, V₆)	Good
28	6.7	11.3	—	80	35	45	109	1.35	90	4.1	56	1.35	5	1,424	LAD, IRBBB	Good
29	10.3	14.3	Apical	66	37	28	93	1.39	72	2.1	43	1.39	5	2,743	neg T (V₅–V₆)	Good
30	3.2	5.2	Antlat	57	31	25	87	1.52	110	2.8	45	1.52	7	2,229	LVH	Good
31	12.0	15.3	Diaph	65	35	30	92	1.40	85	2.6	46	1.40	7	2,000	LAD, RAE, neg T (I, aVL, V₄–V₆)	Good
32	6.3	7.4	Diaph	59	35	24	105	1.76	106	2.6	41	1.76	2	2,708	LAD, RAE, neg T (I, II, V₄–V₆)	Good
33	7.1	11.0	Apical	64	42	21	96	1.48	90	2.0	34	1.48	10	3,600	LAD, RAE, LVH, neg T (V₅, V₆)	Good
34	10.5	11.9	Antlat	61	36	24	132	1.67	95	2.3	40	1.67	4	2,957	LAD, BAE, LVH, neg T (I, aVL, V₄–V₆)	Poor
35	7.4	9.5	Antlat	54	22	31	119	2.19	75	2.4	58	2.19	2	2,564	LAD, BAE, LVH, neg T (I, aVL, V₄–V₆)	Poor
36	7.4	12.7	Apical	55	22	32	125	2.26	82	2.7	59	2.26	5	1,985	LAD, RAE, flat T (V₅, V₆)	Poor
37	9.1	10.7	Apical	72	40	31	114	1.57	120	3.8	44	1.57	8	2,147	LAD, RAE	Good

CRBBB = complete right bundle branch block; Diaph = diaphragmatic; IRBBB = incomplete right bundle branch block; Postlat = posterolateral. Other abbreviations as in Table 1.

Table 3. Postoperative Clinical Characteristics of the Three Study Groups

	Group I. Normal Wall Motion (n = 14)	Group II. Abnormal Wall Motion (n = 11)	Control Subjects (n = 25)	p Value Group I vs. Control Subjects	Group II vs. Control Subjects	Group I vs. Group II
Age at op. (yr)	6.7 ± 3.7	8.2 ± 3.2				NS
EDVI (ml/m²)	67 ± 12	60 ± 8	64 ± 14	NS	NS	NS
ESVI (ml/m²)	30 ± 8	33 ± 8	26 ± 8	NS	< 0.05	NS
SVI (ml/m²)	36 ± 8	26 ± 4	38 ± 6	NS	< 0.001	< 0.005
WMI (g/m²)	84 ± 22	101 ± 14	63 ± 16	< 0.001	< 0.001	< 0.05
WMI/EDVI (g/ml)	1.26 ± 0.26	1.72 ± 0.42	0.97 ± 0.19	< 0.001	< 0.001	< 0.005
HR (beats/min)	85 ± 10	95 ± 17	106 ± 15	< 0.001	NS	NS
CI (liters/min per m²)	3.2 ± 0.8	2.5 ± 0.6	4.0 ± 0.6	< 0.001	< 0.001	< 0.05
EF (%)	55 ± 8	45 ± 8	60 ± 5	< 0.05	< 0.001	< 0.01
MNSERc (EDV/s)	1.48 ± 0.34	1.18 ± 0.24	1.52 ± 0.23	NS	< 0.001	< 0.05
LVEDP (mm Hg)	6 ± 2	5 ± 3	5 ± 1	NS	NS	NS
SVRI (dynes·s·cm⁻⁵·m²)	2,032 ± 600	2,510 ± 525	1,556 ± 275	< 0.01	< 0.001	< 0.05
Total bypass time (min)	122 ± 35	143 ± 51				NS
Cross-clamp time (min)	38 ± 16	52 ± 34				NS

Values presented are mean value ± SD or p value. Abbreviations as in Table 1.

reported that postoperative ischemic lesions developed in pediatric patients after noncoronary artery surgery, suggesting that subclinical ischemia may occur and may be further enhanced in the presence of hypertrophy (28). Finally, the elevated systemic vascular resistance index found in these patients may also influence systolic function and induce ventricular hypertrophy, further compromising wall motion.

Study limitations. Penny and Redington (14) reported that incoordinate relaxation was more frequently observed after the Fontan procedure using a frame by frame analysis technique. The two-frame method used in this study does not evaluate such motion analysis during the ventricular filling phase; rather, our study identified systolic motion abnormalities. The increased mass/volume ratio noted here may also contribute to the observed filling dysfunction and induce incoordinate relaxation.

Among unresolved questions in wall motion studies are whether and how to correct for the translational motion of the heart within the chest and so distinguish it from the inward (or outward) motion of the ventricular walls. Previous studies in adults have not corrected for translational motion, but these artifacts, due to respiration or body movement, are perhaps more apparent in pediatric patients. Thus, in the present cohort we applied a realignment method of the end-diastolic and end-systolic counters in an attempt to adjust for this motion.

Geometric rotation of the heart can be induced by volume loading or a hypoplastic ventricle and may influence this analysis. To avoid such overestimations, only patients with normally related great arteries were enrolled, and a strict criterion for abnormal wall motion was used. Thus, minor abnormalities were not included in this study. However, such factors might influence the analysis and assessment of other forms of univentricular heart, and such interpretations are more difficult.

Conclusions. Although there were no significant differences between ventricular volumes after the Fontan procedure in these pediatric patients with tricuspid atresia, patients with abnormal wall motion had significantly increased ventricular mass and decreased systolic function compared with values in patients with normal wall motion. These observations suggest that postoperative regional wall motion abnormalities in this setting are not rare, may be related to excessive hypertrophy and may contribute to cardiac dysfunction.

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