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Fetal echocardiography and color Doppler flow imaging: the Rotterdam experience

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ABSTRACT

The adjunctive role of Doppler color flow mapping in the evaluation of cardiac structures and function was studied in 440 normal fetuses between 17 and 22 weeks of gestation (median, 20 weeks) and in 73 fetuses with suspected congenital heart disease between 16 and 38 weeks of gestation (median, 28 weeks). Flow through atrioventricular and arterial valves was generally easy to identify and identification was successful in approximately 90% of the normal fetuses. Flow in the pulmonary veins and through the foramen ovale was visualized in approximately 60% of normal cases. In the group with suspected congenital heart disease, Doppler color flow imaging provided additional information on both cardiac structure and function in 34 fetuses, on function alone in 13 fetuses and on structure alone in 20 fetuses. No additional information was collected in six fetuses.

INTRODUCTION

Fetal echocardiography is now a well-established technique in the diagnosis of normal and abnormal cardiac anatomy. Recently, a number of papers has appeared on the significance of Doppler color flow imaging as an additional tool in this area of prenatal diagnosis[1–4].

The objective of the present prospective study was to determine the adjunctive role of Doppler color flow imaging in the evaluation of anatomy and hemodynamics in fetal congenital heart disease.

MATERIAL AND METHODS

Doppler color flow imaging techniques were used in 68 patients with suspected structural and/or functional congenital heart disease between 16 and 38 weeks of gestation (median, 28 weeks). Five were twin pregnancies resulting in 73 fetuses being examined. A total of 440 fetuses from patients with a known increased risk of congenital heart disease served as controls. Here, the gestational age varied between 17 and 22 weeks (median, 20 weeks).

The study was effectively performed during two separate periods, due to availability of equipment. During the first period (1988), a Sonotron Vingmed CFM 700 with a 3 MHz probe was used, and 180 at-risk patients and 16 suspected abnormal patients were seen. For the second period (1990–91), a Toshiba SSA 270A with a curved-linear 3.75 MHz probe or phased-array 5 MHz was used, and 220 at-risk patients and 52 referred anomalies were investigated.

Attempts were made in all patients to document Doppler color flow patterns through atrioventricular and arterial valves, venous return to right and left atria, flow across the foramen ovale, the aortic arch and ductus arteriosus. The study was performed in a cross-sectional manner with only one patient (case 24) being seen more than once.

Structural information was defined as that information which was acquired only as a result of the Doppler color flow imaging overlay. Functional information was defined as that which was obtained by using pulsed and, where necessary, continuous wave Doppler as an adjunct to the Doppler color flow imaging. Within the patients with congenital heart disease four groups resulted:

(1) Group I consisted of 31 pregnancies and 34 fetuses in whom both structural and functional information was obtained with Doppler color flow imaging.

(2) Group II comprised 12 pregnancies and 13 fetuses in whom only functional information was obtained.

(3) Group III was made up of 19 pregnancies and 20 fetuses where structural information was collected.

(4) Group IV consisted of six pregnancies and seven fetuses where neither structural nor functional information was obtained.

All fetuses with structural heart disease with or without other anomalies underwent chromosomal analysis using either amniocentesis or cordocentesis. One fetus was already known to have an inversion of chromosome 20

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(46,XY,inv(20)(p13 p11.2) and a ventricular septal defect.

RESULTS

Fetuses without congenital heart disease

Flow through atrioventricular (Figure 1) and arterial valves was generally easy to identify and was successful in around 90% of the fetuses. Doppler color flow imaging allowed rapid visualization of the size and position of the great vessels and ductus arteriosus. This was even so when real-time visualization was not possible. Flow through the aortic arch was difficult to visualize when the fetal spine or shoulders obstructed the ultrasound beam. Flow in the inferior vena cava was visualized in over 90% of the cases, as it was usually possible to angle the transducer in many directions. However, the success rate of only around 60% in visualizing pulmonary venous and foramen ovale flow was mainly due to the unfavorable fetal position and/or obstruction due to fetal ribs or spine. These structures were frequently at an angle of up to 90° to the ultrasound beam. No information was obtained in around 12% of the patients due to severe maternal obesity and, therefore, extreme depth of the fetal structures. Scanning time was generally shortened, especially in normal hearts when using color flow imaging, as a rapid impression of flow profiles could be achieved immediately following the morphological analysis.

Fetuses with congenital heart disease (first period)

The study design during this first period served effectively as our 'learning-curve'. However, the patients fell into our categories as shown in Table 1. In the Group I fetuses both structural and functional information was obtained in, for example, cases with Ebstein's anomaly, by virtue of defining the site (color flow imaging) and severity (continuous and/or pulsed wave Doppler) of the tricuspid insufficiency. In the category Group II, Doppler color flow imaging easily defined atrioventricular valvular regurgitation in cases of fetal hydrops without structural heart disease. The two fetuses in Group III demonstrated only structural information with Doppler flow imaging because of an unsuitable fetal position or structure too deep to obtain reliable functional data.

Fetuses with congenital heart disease (second period)

Group I fetuses (Table 2)

In most cases the superimposition of Doppler color flow imaging confirmed the structural and hemodynamic suspicions already found with conventional real-time imaging, e.g. confirmation of retrograde left heart filling in cases 21, 27, 31 with hypoplastic left heart (Figure 2), or quantification of assumed tricuspid insufficiency in case 22 with Ebstein's anomaly.

Abbreviations in Tables 1–5:

APVD	anomalous pulmonary venous drainage
AS	aortic stenosis
ASA	atrial septal aneurysm
ASD	atrial septal defect
AV	aortic valve
AVV	atrioventricular valve
CAVSD	complete atrioventricular septal defect
CCHB	complete congenital heart block
CCTGA	congenitally corrected transposition of great arteries
COFS	cerulo-oculo-fascio-skeletal syndrome
DORV	double outlet right ventricle
EFE	endomyocardial fibroelastosis
HLHS	hypoplastic left heart syndrome
HRH	hypoplastic right heart
IUD	intrauterine death
IUGR	intrauterine growth retardation
IVS	intraventricular septum
LV	left ventricle
MCA	multiple congenital anomalies
MI	mitral valve insufficiency
MV	mitral valve
NND	neonatal death
PA	pulmonary atresia
PI	pulmonary insufficiency
PS	pulmonary stenosis
PV	pulmonary valve
RV	right ventricle
SVC	superior vena cava
TA	tricuspid atresia
TGA	transposition great arteries
TI	tricuspid valve insufficiency
TOF	tetralogy of Fallot
TOP	termination of pregnancy
TS	tricuspid stenosis
TV	tricuspid valve
VSD	ventricular septal defect

Table 1 Fetuses with congenital heart disease from the first study period (cases 1–16)

Classification	*n*	*Condition*	*Number of patients*
Group I	10	HLHS	1
		Ebstein anomaly	2
		complex CHD	1
		CAVSD	2
		VSD	4
Group II	4	cardiomyopathy	2
		fetal hydrops	2
Group III	2	HLHS	1
		TOF	1

In some cases with a ventricular septal defect (cases 19, 25, 30), the direction of the shunt (left-to-right) was established. Pulmonary valve patency (cases 18, 23, 30, 34) and atresia (cases 22, 26) were distinguished with Doppler color flow imaging. Where atrial septum aneurysm was diagnosed, the presence (case 35) or absence (case 36) of flow across the aneurysmal structure was demonstrated. In case 17, the small size of the fetus and co-existing multiple anomalies resulted in a hypoplastic right heart being mistaken for the left heart.

Termination of pregnancy was carried out in two cases (10%) and intrauterine or early postpartum death

Table 2 Information obtained by color Doppler flow imaging on both cardiac structure and function in congenital heart disease (Group I, cases 17–36)

Case	*Gestational age* (weeks)	*Conventional ultrasound*	*Color flow imaging*		*Outcome*
			Structural	*Functional*	
17	16	MCA, dextrocardia, HLHS?	R-AVV + pulmonary trunk visualized	flow pulmonary trunk < flow ascending aorta	TOP, dextrocardia HRH + PA
18	23	TOF	VSD + overriding aorta	antegrade flow through stenotic pulmonary valve	DORV + PS
19	> 23	MCA, VSD	L→R shunt VSD	L→R shunt VSD	TOP, MCA, VSD
20	25	hydrops, ASA	ASA	R→L obstruction, ASA, TI	neonatal death, dysmorphic, dysplastic TV
21	25	HLHS	retrograde flow ductus → ascending aorta → LV. No forward flow through left heart	as for structural	neonatal death, HLHS
22	> 25	ascites, Ebstein + PA	Ebstein + PA	massive TI, no pulmonary artery flow	IUD, Ebstein, PA, 2° ASD
23	28	twins: 1 × normal, 1 × IUGR + PS	normal PS	normal PS + PI	alive and well IUD, macerated
24	28 > 29 > 31	polyhydramnios polyhydramnios thick IVS, AS?	heart too deep heart too deep normal connections	heart too deep heart too deep gradient distal and proximal AV	 alive, dysmorphic AS?, cardiomyopathy, Noonan
25	> 29	MCA, VSD, small LV, great vessels?	L→R shunt VSD	L→R shunt VSD	alive, MCA, DORV, PA
26	> 30	TA + PA	no flow TV + PV >> flow pulmonary veins	no flow TV + PV normal flow left heart	NND, TA + PA
27	> 31	IUGR, HLHS	HLHS	reduced, absent flow MV, no flow in ascending aorta	IUD, no autopsy
28	32	hydrops, VSD, dysplastic TV	VSD	severe TI	IUD; 3 × VSD dysplastic TV
29	32	twins: 1 × normal 1 × giant, non-contractile RV ?Ebstein	 normal disturbed flow TV + PV	 normal severe TS, TI, mild PI	 alive and well NND, PI, EFE
30	> 32	IUGR + TOF	L→R shunt VSD, antegrade flow PV	antegrade flow PV	alive, TOF
31	33	HLHS	HLHS	absent left heart flow, disturbed pulmonary artery flow	NND, HLHS
32	35	echodense RV, fibroelastosis, ?abnormal TV	normal	mild TI	alive, echodense RV, 'physiological variant'
33	> 35	?right heart pathology	normal flow	normal flow	alive, normal heart
34	> 35	MCA, VSD, dysplastic PV, abnormal MV	VSD, abnormal PV + MV	PS + PI, MI, TI	NND, trisomy 18, MCA, VSD, dysplastic PV, abnormal foramen ovale
35	> 37	ASA, > right heart, normal connections		normal flow across ASA	alive, normal heart
36	> 37	hydrops ASA, right heart >>	ASA	no flow through TV, no flow across ASA	alive, restrictive foramen ovale, mild valvular AS

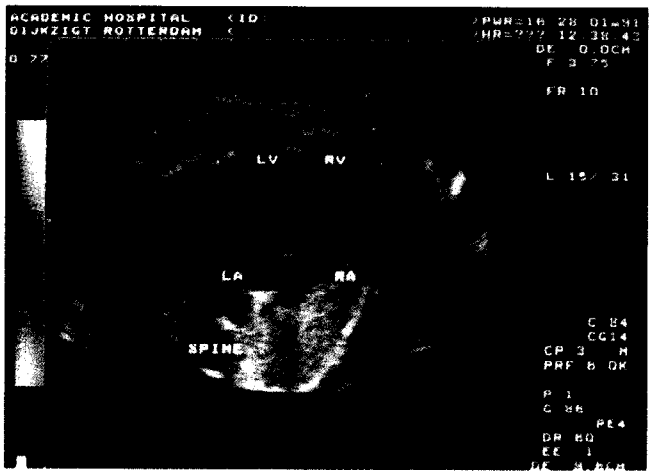

Figure 1 Normal color fetal four-chamber view: RA, right atrium; RV, right ventricle; LA, left atrium; LV, left ventricle

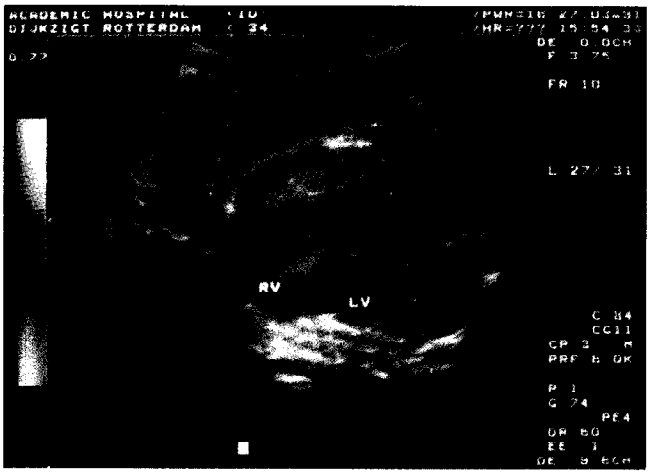

Figure 2 Color four-chamber view in case 31 with hypoplastic left heart. Note absence of flow into the left ventricle, LV, and normal flow into the right ventricle, RV

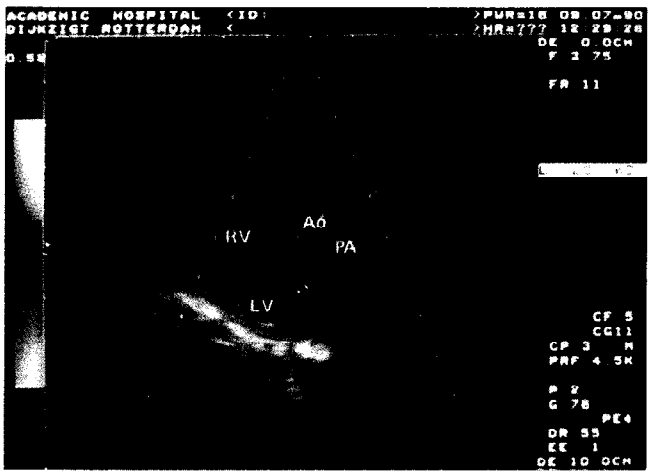

Figure 3 Case 56 with double outlet right ventricle. Arrows indicate flow from the small left ventricle (LV) through the ventricular septal defect, 'bypassing' the pulmonary artery (PA) and exiting the aorta (Ao) from the right ventricle (RV)

occurred in ten (50%). There are eight survivors with heart disease (40%) over 1 month old and the two normal co-fetuses from the twin pregnancies have also survived. A chromosomal anomaly (trisomy 18) was present in one case.

Group II fetuses (Table 3)

Structural information using Doppler color flow imaging was not obtained in this group for various reasons. In some fetuses no structural disease was present (cases 37,39 and 40) or the cardiac structures were situated too deep (cases 38, 42). In others this was due to the advanced gestation, and/or positional inaccessibility of the fetal heart. However, functional information, i.e. demonstra-

Table 3 Information obtained by color Doppler flow imaging on cardiac function alone in congenital heart disease (Group II: cases 37–44)

Case	*Gestational age* (weeks)	*Conventional ultrasound*	*Color flow imaging*		*Outcome*
			Structural	*Functional*	
37	18	MCA, hydrops	—	TI, MI	TOP, MCA
38	> 21	critical AS + EFE	heart too deep	decreased flow MV + AV	TOP, critical AS, EFE
39	25	twin–twin transfusion: 1 × normal 1 × hydrops	 normal normal	 normal MI + TI	 normal dysplastic TV?
40	> 30	CCHB	normal	normal flow, >> velocities	alive, early postpartum pacemaker
41	> 30	CCHB, CCTGA, narrow aorta	no information	TI + antegrade aortic flow	NND, CCHB, CCTGA, functional aortic atresia
42	32	MCA, VSD, PS	no information	PS + PI	trisomy 18, IUD, MCA, VSD. Great vessels not mentioned in autopsy report
43	33	large VSD, malposition IVS	no information	normal flow profiles through all vessels	alive, MCA, pentalogy of Cantrell
44	> 35	complex CHD, HLHS, AVSD	no information	antegrade flow through PA + aorta	NND, HLHS, AVSD, functional aortic atresia

tion of mitral and tricuspid valve insufficiency in cases (37, 39) of fetal hydrops was achieved. Two fetuses (cases 41, 44) both demonstrated antegrade flow through the ascending aorta at 30 and 35 weeks of gestation, respectively, although both died within 2 days of birth with functional aortic atresia.

Termination of pregnancy was performed in two cases (25%) and intrauterine or early postpartum death occurred in three cases (37.5%). There are three survivors with heart disease (37.5%), one of whom underwent pacemaker implantation during the second day of life. The co-existing normal twin (case 39) has also survived. An abnormal karyotype (trisomy 18) was present in one case.

Table 4 Information obtained by color Doppler flow imaging on cardiac structure alone in congenital heart disease (Group III: cases 45–62)

Case	*Gestational age* (weeks)	*Conventional ultrasound*	*Color flow imaging*		*Outcome*
			Structural	*Functional*	
45	16	MCA, CAVSD	L→R shunt through CAVSD	—	TOP, macerated, autopsy impossible
46	> 18	MCA, HLHS	HLHS	—	IUD, autopsy refused
47	> 19	twins:		—	
		1 × normal	normal	—	normal
		1 × VSD	unclear result (technical)	—	no obvious VSD (*in utero* closure?)
48	20	MCA, dextrocardia, single ventricle, single great vessel	single ventricle and great vessel	—	TOP, univentricular heart, truncus, ASD, partial APVD
49	21	MCA, HLHS?	right heart identified, probable HLHS	—	45XO, TOP, MCA, TOF
50	21	inversion chromosome 20, VSD	R→L shunt through VSD	—	alive, closure VSD
51	23	MCA, TOF + PA	L→R shunt through VSD, flow only through aorta	—	trisomy 18, TOP, MCA, TOF, PA
52	> 25	MCA, CAVSD, HLHS, DORV	single flow through AVSD, ?DORV	—	trisomy 18, TOP, no autopsy
53	29	MCA, TOF	shunt through VSD, absent pulmonary flow	—	trisomy 13, NND, no postmortem
54	> 29	marked polyhydramnios, irregular bradycardia, CAVSD, DORV?	R→L shunt through AVSD to right-sided aorta	—	NND, situs inversus, CAVSD, normal great vessels
55	31	hydrops, cardiomegaly	confirmed normal connections	—	NND, no obvious malformations
56	> 31	IUGR, HLHS, DORV, hypoplastic PA	bidirectional shunt through VSD, flow through VSD to anterior ascending aorta, absent pulmonary flow	—	NND, MCA, HLHS, DORV
57	> 32	MCA, VSD?	confirmation technically unavailable	—	IUD, MCA, no postmortem
58	33	MCA, TOF, PA	small LV, dilated overriding aorta	—	IUD, MCA, TOF, absent pulmonary artery and ductus arteriosus
59	> 33	IUGR, CAVSD, absent SVC, ?abnormal pulmonary veins, HLHS	HLHS	—	NND, CAVSD, HLHS, persistent R SVC
60	33	HLHS	normal right heart flow, retrograde flow left heart	—	HLHS, still alive at age 6 weeks
61	> 35	IUGR, marked poly-hydramnios, MCA, VSD	shunt through VSD technically not measurable (depth)	—	trisomy 18, IUD, MCA, VSD
62	36	echodense liver, right heart >>	confirmed normal connections	—	alive, echodense liver, etiology unknown, presumably affecting fetal venous return

Group III fetuses (Table 4)

In this group only structural information was collected. Functional information was not obtained for various reasons. In case 50 the right-to-left shunt through a ventricular septal defect had been clearly identified with Doppler color flow imaging, so no functional studies were considered necessary. In some cases, early gestation (cases 45, 46) or late gestation (cases 58, 59 and 61) hampered further investigation. Sometimes (cases 51, 52, 54, 61) polyhydramnios had deleterious effects on the depths that could be reached even using continuous wave Doppler. In case 56 with double outlet right ventricle, a bidirectional shunt through the ventricular septal defect was observed with blood flow 'bypassing' the atretic pulmonary artery to exit the heart through the anterior positioned aorta (Figure 3). However, functional information was not obtainable due to the fetal position. In cases 47 and 57 a temporary machine failure interfered with the study after structural information had been obtained. In the remainder, external circumstances prevented further investigation, i.e. maternal labor and/or uterine contractions, relative dyspnea and maternal (psychological) distress.

Termination of pregnancy took place in five cases (29%) and intrauterine or early postpartum death occurred in nine cases (53%). There were three survivors above 1 month of age (18%). The co-existing normal twin (case 47) has also survived. An abnormal chromosome result (45,XO, trisomy 18 (three cases), inversion chromosome 20 (46,XY,inv(20)(p13 p11.2)) was determined in five cases.

Group IV fetuses (Table 5)

In this group no structural or functional information was acquired. In cases 63, 65 and 66, maternal obesity and abdominal striae were responsible for poor quality ultrasound imaging. In case 64 with a suspected ventricular septal defect, the septum remained perpendicular to the ultrasound beam. In case 67 maternal obesity and oligohydramnios hampered the examination. In case 68 advanced gestation (34 weeks) with shadowing from the ribs prevented further information being acquired although the diagnosis was correctly made with standard real-time imaging.

DISCUSSION

In this study the role of Doppler color flow imaging in establishing normal and abnormal fetal cardiac structure and function was assessed.

Normal morphology and function could be demonstrated at the inflow, atrioventricular and outflow levels in the vast majority of cases. In 12%, however, no flow information was obtained, which was mainly due to maternal obesity. Pulmonary venous and foramen ovale flow could only be visualized in 60% of the cases, mainly as a result of an unfavorable fetal position relative to the ultrasound beam and/or obstruction due to the fetal ribs or spine. Following identification of the normal cardiac anatomy using conventional two-dimensional real-time ultrasound, the addition of the Doppler color flow imaging overlay allowed rapid visualization of flow directions and therefore shortened the scanning time. This is important to safety, since Doppler color flow imaging displays lower peak intensities than pulsed Doppler[5].

Doppler color flow imaging contributed to both the morphological and functional aspects of congenital heart disease. Information was obtained on the presence or absence of flow, flow direction and the flow pattern. Doppler color flow imaging may provide unexpected information such as in case 56 (Table 4) (Figure 3) in which conventional ultrasound had shown a double outlet right ventricle with an anterior ascending aorta and a narrow pulmonary artery, whereas Doppler color

Table 5 No information obtained by color Doppler flow imaging in congenital heart disease (Group IV: cases 63–68)

Case	*Gestational age* (weeks)	*Conventional ultrasound*	*Color flow imaging*		*Outcome*
			Structural	*Functional*	
63	17	twins: 1 × normal 1 × omphalocele + TOF	— maternal obesity, small fetuses, no information acquired	— —	alive NND
64	19	MCA, ?VSD, mosaic trisomy 13	septum perpendicular to beam, no information obtained	—	TOP, MCA, no VSD
65	21	CCHB, echodense AV valves	no information acquired	—	NND, multiple organ failure, absent AV mode
66	21	MCA, ?VSD	no information acquired	—	alive, dysmorphic, dextrocardia, COFS syndrome, no VSD
67	> 21	oligohydramnios, omphalocele, MCA, ?? overriding aorta	no information acquired, maternal obesity and oligohydramnios	—	NND, MCA, no postmortem
68	34	TGA	differentiation great vessels not possible	—	alive, TGA

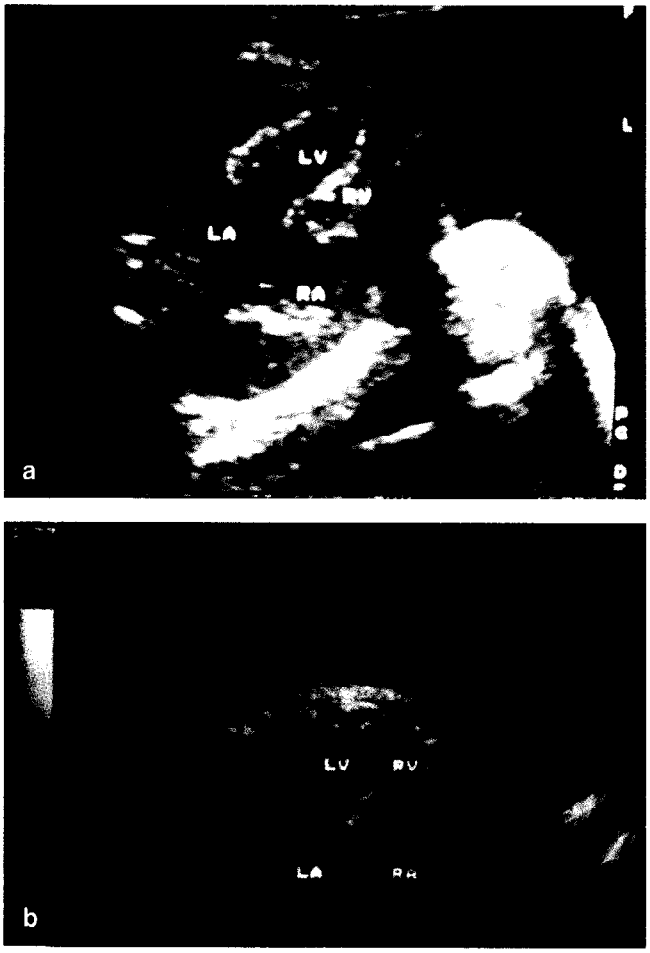

Figure 4 Case 26 with tricuspid and pulmonary atresia. (a) The conventional four-chamber view; (b) color overlay confirming absence of flow into the right ventricle (RV). RA, right atrium; LA, left atrium; LV, left ventricle

flow imaging demonstrated flow through the ventricular septal defect bypassing the pulmonary artery (which was atretic) and exiting via the anteriorly positioned ascending aorta. Cases 30 and 35 were of interest because antegrade flow through a narrowed hypoplastic ascending aorta was demonstrated at 32 and 35 weeks, respectively, while after birth functional atresia was present. Obviously, the prenatal presence of antegrade flow in a narrowed outflow tract vessel may not indicate postnatal patency.

In the presence of a conventionally diagnosed atretic valve, Doppler color flow imaging may confirm the diagnosis (Figure 4) by showing absence of flow through that valve. In Ebstein's anomaly, Doppler color flow imaging may help in establishing the direction of eccentric jets at the level of the dysplastic tricuspid valve (Figure 5) and therefore may aid the precise placement of the Doppler sample volume for quantification of the jets. Doppler color flow imaging may identify atrioventricular valve leakage in complete atrioventricular septal defects, although this was not apparent in any of our series. Doppler color flow imaging did not alter the original two-dimensional real-time diagnosis of congenital heart disease, although the functional aspects of the defect were often clarified by color imaging. Maternal

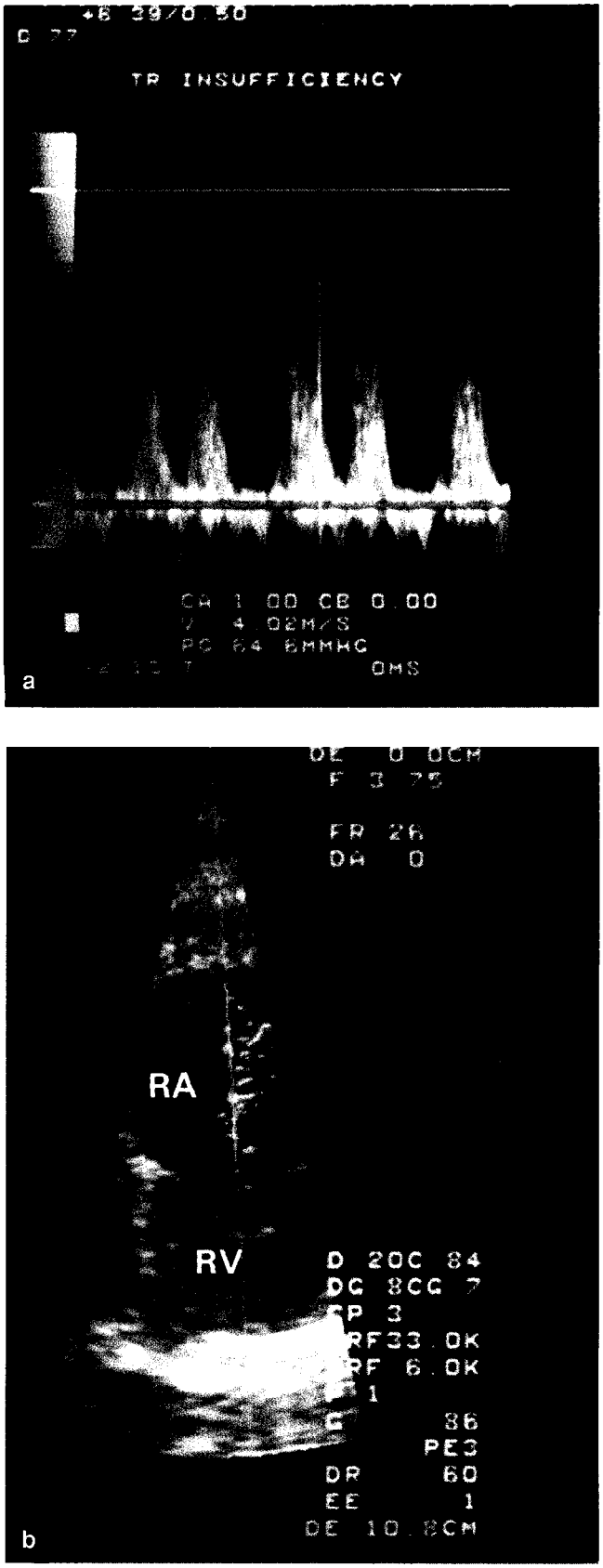

Figure 5 Case 28. (a) The continuous wave Doppler profile quantifying the tricuspid insufficiency (4.02 m/s); (b) the 'two-chamber' color view of right atrium (RA) and right ventricle (RV)

obesity and polyhydramnios may impose severe restrictions regarding the efficacy of the diagnosis of congenital

heart disease. This applies to all ultrasound modalities including Doppler color flow imaging.

In second-trimester fetuses, the minute size of cardiac structures and the possible combination of extracardiac anomalies may hamper precise definition of the disease. For instance, in case 17 (Table 2), using both conventional real-time and color Doppler imaging, a hypoplastic right heart was mistaken for the left heart. Similarly in late gestation, an unsuitable fetal position may present difficulties regarding the accurate visualization of cardiac structures.

Doppler color flow imaging has the potential to contribute to the obstetric management of cases with congenital heart disease, particularly in ongoing pregnancies to monitor changes in natural history, as well as the potential appearance of, and/or evolution of, fetal hydrops.

In this study 50% of the affected pregnancies were beyond 24 weeks of gestation which is the upper limit for legal termination of pregnancy in The Netherlands. Doppler color flow imaging did not really alter obstetric management in this series because the majority of cases presented with severe congenital heart disease frequently associated with additional anomalies.

The spectrum of fetal congenital heart disease is different to that seen postnatally with high incidences of chromosomal and/or multiple anomalies, as shown in this and other studies[6]. The classification of fetal heart disease based on postnatal groupings is, therefore, severely hampered and comparisons between prenatally and postnatally detected disease cannot be made easily.

It can be concluded that color Doppler flow imaging may provide additional information on fetal cardiac flow characteristics and may be a valuable adjunct in the assessment of congenital heart disease. However, the prognostic significance of this technique still needs to be determined in larger series of patients, studied longitudinally where possible.

These studies should continue to be performed in centers experienced in fetal echocardiography, as Doppler color flow imaging is obviously an integral part of fetal cardiac assessment.

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