

# Fetal echocardiography: New insight into fetal cardiology—A review

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## Summary

Recent advance in echocardiography has permitted detailed imaging of the fetal heart, facilitating the prenatal diagnosis of congenital heart disease and the diagnosis and treatment of fetal arrhythmias. New advance in Doppler echocardiography has provided important information about the physiology of normal and abnormal fetal circulation.

From 1980 to 1986, we performed echocardiography on 564 fetuses, including 254 normal and 310 high risk pregnant. Results are discussed in terms of the method and accuracy of the prenatal diagnosis of congenital heart disease, the recognition of and intrauterine treatment of fetal arrhythmias, the diagnosis of fetal heart failure, the assessment of fetal circulation by Doppler echocardiography, the management of the fetus with cardiac problems, and the ethical aspects of fetal cardiology.

## Key words

Fetal echocardiography    Prenatal diagnosis    Congenital heart disease    Fetal arrhythmias    Fetal circulation

## Introduction

Echocardiography has become increasingly important as a means for diagnosing congenital heart disease in infants and children. Being painless and harmless, it can be repeated as often as necessary. It is ideal for making anatomical and structural evaluations of the heart in children, particularly in newborn infants who are seriously ill. Recent advance in echocardiography and improvement in the resolution of

ultrasonographic techniques have aided the precise evaluation of the anatomical structure of the fetus and fetal heart successfully and safely. Fetal echocardiography was introduced in the early 1980's<sup>1-6)</sup>, and has facilitated the prenatal diagnosis of congenital heart disease<sup>2,3,6,7)</sup> and the diagnosis and treatment of fetal arrhythmias<sup>8)</sup>. Recently new advance in Doppler echocardiography have provided exciting information on fetal blood flow and the physiology of normal and abnormal fetal circulation<sup>9)</sup>.

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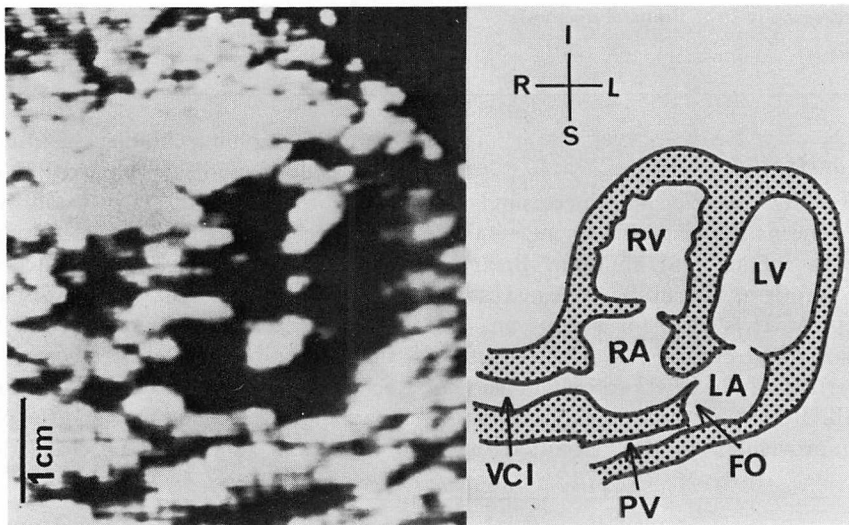
**I. Evaluation of the structure of the fetal heart and the prenatal diagnosis of congenital heart disease**

We have utilized fetal echocardiography, including two-dimensional and M-mode echocardiography since 1980<sup>6)</sup> and Doppler echocardiography since 1984 for 564 fetuses consisted of 254 normal pregnancies and 310 high risk pregnancies. Their gestational ages ranged from 16 to 42 weeks. The pregnancy at high risk for heart disease in the newborn included intrauterine growth retardation, fetal arrhythmia, hydrops fetalis, a family history of congenital heart disease, exposure to teratogens such as rubella or maternal diabetes, and maternal systemic lupus erythematosus (SLE).

We used a commercially available two-dimensional ultrasonographic apparatus, with 3.5 and 5 MHz transducers. Since 1984, we have employed a pulsed Doppler echocardiography and a Doppler color flow mapping system. After determining the fetal position using a linear array

two-dimensional echocardiograph, the fetal cardiac structures were evaluated by four standard sections: consisting of the four-chamber, the left ventricular longaxis, the great arterial, and the aortic arch views. These are similar to the standard sections used in postnatal echocardiography. Our success rate in obtaining good echocardiographic recordings was 98% for the four chamber-view, and 84% for the LV long axis-view. The optimum timing during pregnancy for obtaining good echocardiographic recordings is about 25 weeks' gestation. Later in gestation, notably after 35 weeks, it becomes difficult to do so because of maternal obesity, or interference with the ultrasonic beam by fetal bone.

The four-chamber view of the heart (**Fig. 1**) is the easiest to interpret and record. Anatomic landmarks include the flap of the foramen ovale in the left atrium, the inferior vena cava entering the right atrium, the moderator band or trabeculation of the right ventricle, and the insertion of the septal leaflet of the tricuspid valve closer than the mitral valve insertion to the apex of the



**Fig. 1. Four-chamber view of a fetus of 27 weeks gestation.**

The right ventricle and the left ventricle are nearly in equal size. The flap of the foramen ovale in the left atrium, the inferior vena cava entering the right atrium, rough trabeculation of the right ventricle, the pulmonary vein entering the left atrium, and insertion of the septal leaflet of the tricuspid valve closer to the apex than that of the mitral valve, are recognized.

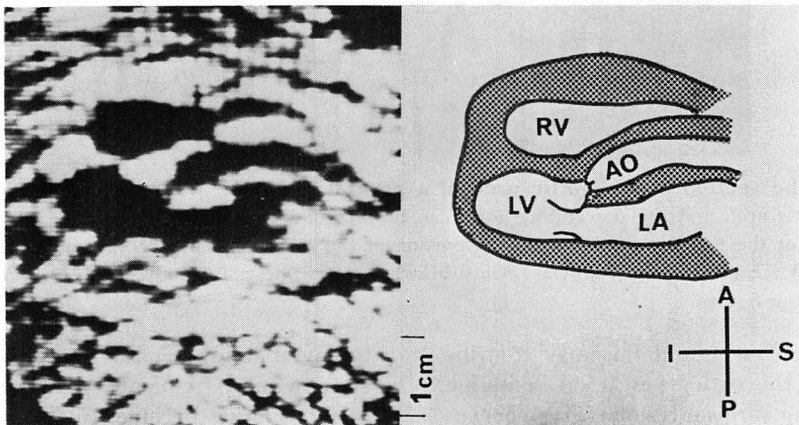
FO=foramen ovale; LA=left atrium; LV=left ventricle; PV=pulmonary vein; RA=right atrium; RV=right ventricle; VCI=vena cava inferior; I=inferior; L=left; R=right; S=superior.

heart. The aorta is demonstrated as an arch.

In the left ventricular long-axis view, the mitral valve connects with the semilunar valve with fibrous continuity (**Fig. 2**). In the section of the great arteries, the aorta is seen on the right posterior side of the pulmonary artery (**Fig. 3**). The ductus arteriosus is usually well demonstrated, which is widely open and connects with the descending aorta (**Fig. 3**). The aortic arch can also be recorded more clearly in a fetus than in a newborn infant (**Fig. 4**). The physiological narrowing of the isthmus or the preductal portion of the aorta is clearly recognizable<sup>10,11</sup>.

The incidence of congenital heart disease studied by fetal echocardiography in our series was 13 (2.3%) in all 564 cases; 3.5% in 310 high risk pregnancies, and 0.8% in 254 normal pregnancies. Types of congenital heart disease consisted of asplenia and polysplenia, complete endocardial cushion defect, tetralogy of Fallot, the coarctation complex, transposition of the great arteries, pulmonary atresia, double outlet right ventricle, hypoplastic left heart syndrome, large ventricular septal defect with large atrial septal defect, and ectopia cordis. These were all detected by fetal echocardiography, except for one patient diagnosed postnatally as having a small

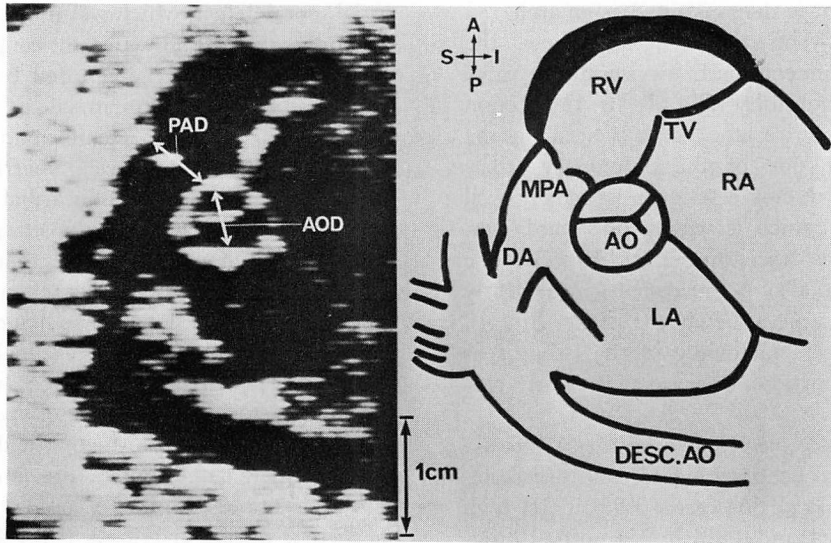
atrial septal defect, which was not evaluated during pregnancy. The types of congenital heart disease which can be diagnosed by fetal echocardiography are summarized in **Table 1**<sup>12</sup>. Some major structural malformations such as atrioventricular valve atresia, ventricular hypoplasia, single ventricle, endocardial cushion defect, and great arterial malposition are easily diagnosed. Some cases with total or partial anomalous pulmonary venous return are difficult to be evaluated. Lesions not so hemodynamically important to the fetus, such as small ventricular septal defect, atrial septal defect, and mild or moderate semilunar valve stenosis, are more difficult to be detected. Normally the ductus arteriosus is widely patent in fetal life, and this cannot be differentiated from the congenital anomaly, persistent ductus arteriosus (**Fig. 4**). However, most of the major cardiac lesions, particularly those which may require early medical or surgical treatment during the neonatal period, can be evaluated by fetal echocardiography (**Figs. 5~7**). This facilitates delivery of an infant in a specialized hospital covered with a well-trained medical team for early appropriate treatment. For infants with critical ductus-dependent lesions such as pulmonary atresia or aortic atresia, treatment with E type prostaglandins



**Fig. 2.** Left ventricular long-axis view of a normal fetus.

The atrio-ventricular valve connecting with the great artery with fibrous continuity is the mitral valve. The aorta is demonstrated as an arch.

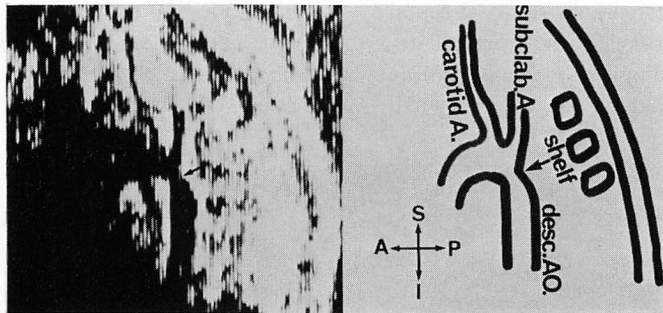
AO=aorta; A=anterior; P=posterior. Others; see Fig. 1.



**Fig. 3.** The section of the great arteries of a normal fetus.

The aorta is situated in the right posterior side of the pulmonary artery. The ductus arteriosus is widely patent and connects with the descending aorta. The right ventricle and the main pulmonary artery are dilated.

DA=ductus arteriosus; MPA=main pulmonary artery; TV=tricuspid valve. Others: see Figs. 1 and 2.



**Fig. 4.** The section of the aortic arch of a normal fetus.

The aortic arch and the descending aorta can be recorded clearly in a fetus. The physiological narrowing of the isthmus or the preductal portion of the aorta is clearly recognizable.

carotid A.=carotid artery; subclav. A.=subclavian artery; desc. AO.=descending aorta. Others: see preceding figures.

should be started early, and this may contribute to keeping the patients in good condition and to improving surgical results (Figs. 5~7).

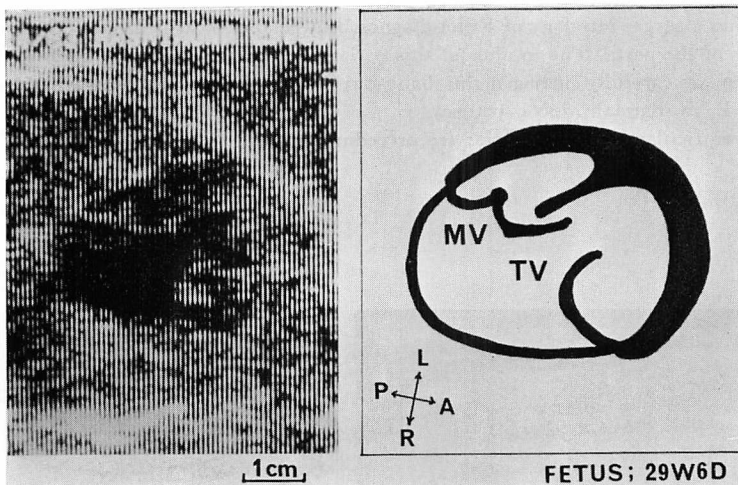
## II. Recognition and treatment of fetal arrhythmias

Fetal echocardiography may also be applied

to investigating fetal arrhythmias<sup>13-17</sup>. These are now more frequently recognized since heart monitoring has become widely used in obstetrical practice. In our experience the incidence of fetal arrhythmias was 3% among 564 fetuses, including normal and high risk pregnancies. In high risk pregnancies it was much greater,

**Table 1. Congenital heart disease demonstrated by fetal echocardiography**

POSSIBLE	DIFFICULT	IMPOSSIBLE
Single ventricle	D-transposition of the great arteries	Patent ductus arteriosus
Endocardial cushion defect	Double-outlet right ventricle	Mild or moderate pulmonary stenosis
Large ventricular septal defect	Tetralogy of Fallot	Small ventricular septal defect
Ebstein's disease	Coarctation of the aorta	
Tricuspid atresia	Interruption of the aortic arch	
Mitral atresia	Pulmonary atresia	
Aortic atresia	Truncus arteriosus	
Cardiomyopathy	L-transposition of the great arteries	
Complete AV block	Atrial septal defect	
2:1 AV block	Anomalies of pulmonary venous return	
Atrial flutter		

**Fig. 5. Hypoplastic left heart syndrome.**

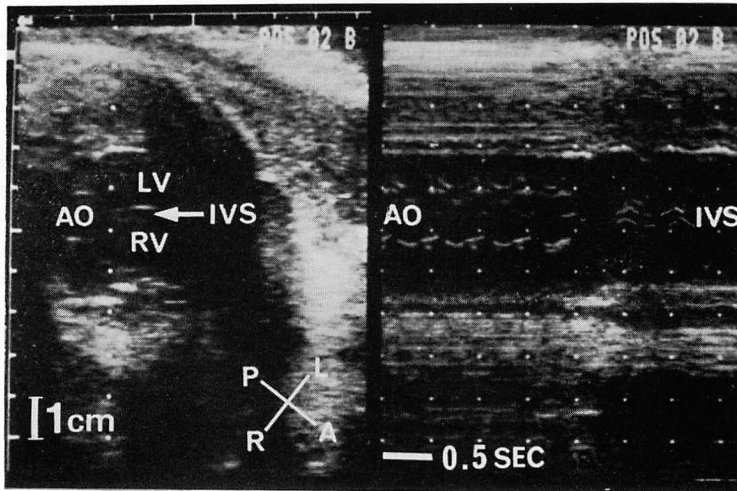
This patient has mitral atresia and aortic atresia. The fetal echocardiogram (four-chamber view) demonstrates a hypoplastic left ventricle and a tiny left atrium. The right atrium and the right ventricle are dilated. A small ventricular septal defect is recognized. Soon after birth, we administered prostaglandin  $E_1$  for this patient, and surgery was performed when he was 7 days of age, but the result was unsuccessful.

MV=mitral valve; TV=tricuspid valve.

being 4.8%.

Fetal arrhythmias were evaluated by the atrial and ventricular wall motions using dual M-mode echocardiography, or by assessing ventricular inflow and regurgitant flow in the atrioventricular valve by pulsed Doppler echo-

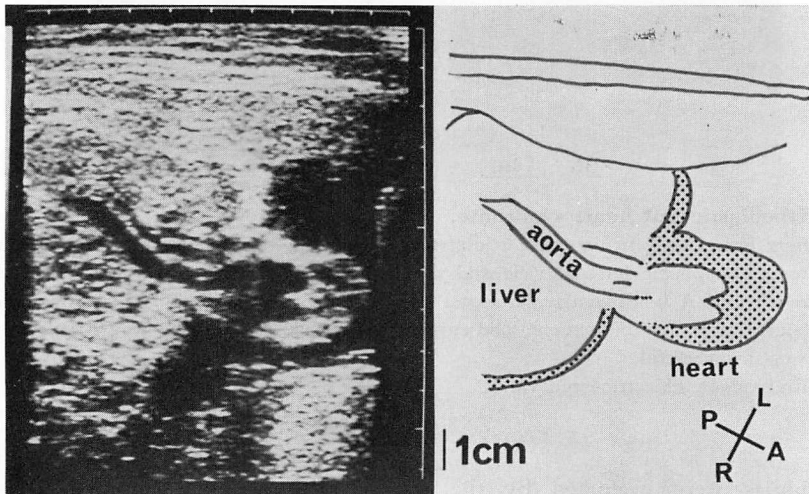
cardiography. **Fig. 8** shows the echocardiograms of a fetus revealing an atrial rate of 480 per min and a ventricular rate of 240 per min. Accordingly atrial flutter was diagnosed. Three cases of fetal atrial flutter have been successfully treated in utero by administering digitalis to



**Fig. 6. Tetralogy of Fallot.**

This fetus has a severe tetralogy of Fallot diagnosed by fetal echocardiography which demonstrates the overriding of the aorta. The mother of this patient has phenylketonuria. Because of severe tetralogy of Fallot, we carefully managed this baby during delivery and after birth, and administered prostaglandin E<sub>1</sub> to maintain ductal patency.

IVS=interventricular septum. Others: see preceding figures.



**Fig. 7. Ectopia cordis.**

A routine obstetrical ultrasound examination at the 31 weeks gestation demonstrates the fetal heart extruding from the fetal thorax and moving freely in the amniotic fluid. The intracardiac abnormalities include transposition of the great arteries and complete endocardial cushion defect.

the mothers. One of two cases with fetal supra-ventricular tachycardia was successfully treated by maternal digoxin administration, and in the

other, following unsuccessful treatment by digoxin, it resolved spontaneously during pregnancy (Table 2).

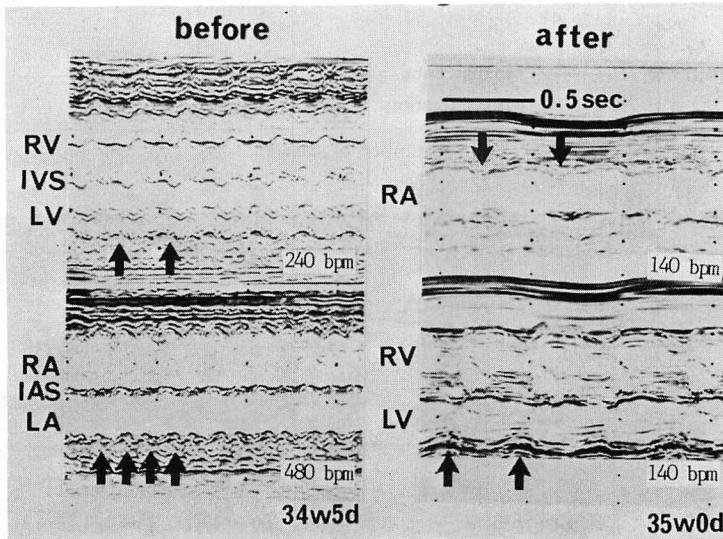
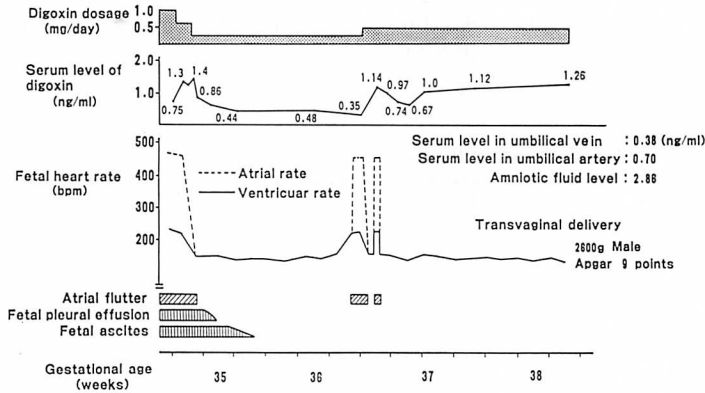


Fig. 8. Transplacental digitalization for fetal atrial flutter.

The dual M-mode echocardiogram of a fetus at 34 weeks gestational age (left) demonstrates an atrial rate of 480 beats per min and a ventricular rate of 240 per min. Atrial flutter was diagnosed. Digoxin was administered to the mother while the fetal heart rate was continuously monitored. Five hours after the third administration of medication, the fetal heart rate dropped from 240 to 135 per min (right). Examination by echograms then showed massive pleural effusion in the fetus. We therefore administered furosemide to the mother but there was no evidence of improvement. Following detection of a rupture of the membranes and confirmation by a positive shake-test, we performed delivery by Caesarian section. The digoxin concentration in the maternal venous blood was 1.4 ng/ml; in the amniotic fluid, 2.8 ng/ml; and in the umbilical venous blood, 0.38 ng/ml. No congenital heart disease was found and the baby has been doing well on digitalis for 7 months, and has never manifested tachycardia.

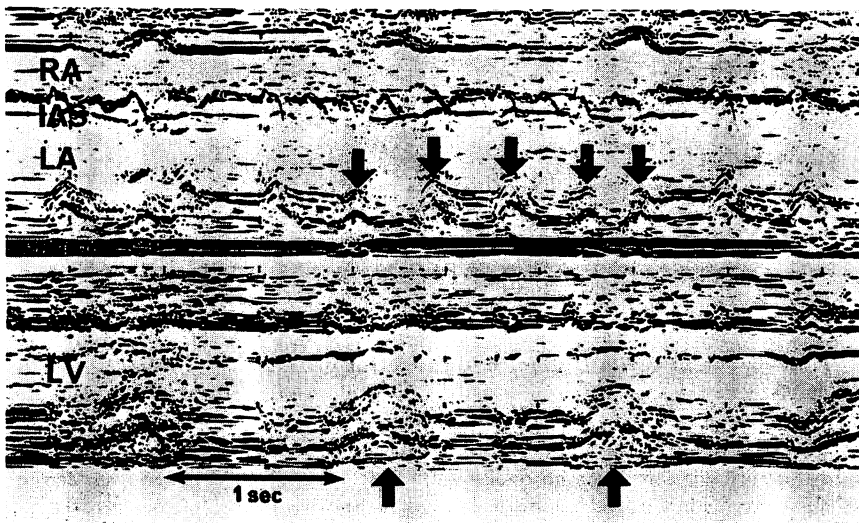
If fetal tachycardia becomes sustained, intrauterine cardiac failure can ensue within hours. Therefore, once it is correctly identified, attempts should be made to control the tachycardia prenatally by administering appropriate drugs to the mother. Success has been achieved using digoxin, verapamil, and procainamide<sup>8,12,13,16,17</sup>. When bradycardia is encountered with rates

of less than 100 per min, sinus bradycardia can be differentiated from complete heart block by examining M-mode echocardiographic tracings (Fig. 9). Fetal bradycardias in our series are summarized in Table 3. Three cases had 2 to 1 A-V block associated with congenital heart disease, such as endocardial cushion defect. Four cases, two of which died in

**Table 2. Fetal tachyarrhythmias**

Fetal arrhythmia	In-utero therapy	Postnatal ECG	Outcome
Supraventricular tachycardia (2)		PAT Sinus rhythm with normal heart rate	Alive Alive
Tachyarrhythmia (1)		Chaotic atrial rhythm	Alive
Atrial flutter (3)	Transplacental digitalization	Sinus rhythm with normal heart rate	Alive

( ) = number of case(s).



**Fig. 9. Complete AV block.**

The dual M-mode echocardiogram in this patient shows that the ventricular rate is slow at 55 per min and the atrial rate is 160. The fetus demonstrated severe hydrops fetalis and heart failure. We injected isoproterenol into the amniotic fluid, however, there was no response. Following Caesarian delivery, we successfully implanted a permanent pacemaker.



utero had complete heart block. Two of them received pacemaker implantation after birth. Fetuses with A-V block and severe heart failure have extremely poor prognosis. To date, no effective treatment has been developed for this condition. If the fetus is near full term and shows evidence of lung maturity, early delivery will be necessary and pacemaker implantation may be required immediately after birth.

Other arrhythmias, such as ectopic beats, are commonly observed but rarely important in terms of morbidity or mortality. These are rarely associated with structural heart disease, however, careful examination is necessary for their exclusion<sup>14</sup>.

### III. Recognition of fetal heart failure

Fetal heart failure may result in intrauterine fetal death, however, it is sometimes difficult to evaluate before birth (Fig. 10). Recently, hydrops fetalis has been recognized using fetal echocardiography<sup>18</sup>. With the declining frequency of Rh isoimmunization, nonimmune hydrops fetalis has become the important type of hydrops. Nonimmune hydrops may be caused by fetal anemia, intrauterine hypoproteinuria, chromosomal anomalies, congenital infection or fetal heart failure<sup>19</sup>. We recognized 20 cases

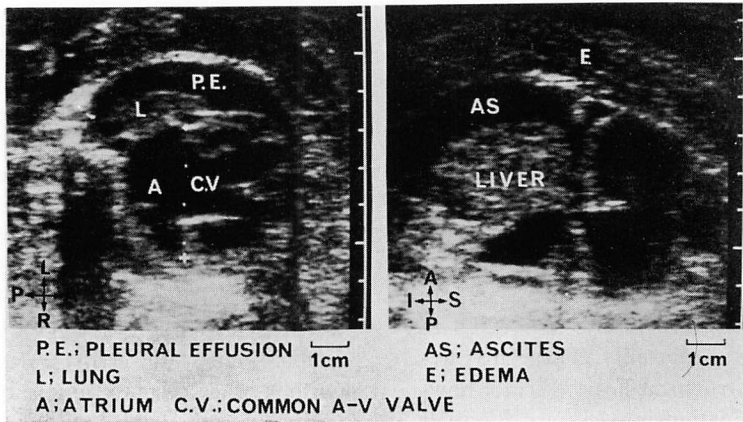
with fetal hydrops in our series, in which 8(4%) cases had fetal arrhythmias and/or congenital heart disease. These suggest the presence of fetal heart failure which is of relatively high incidence in fetal hydrops. Hydrops caused by cardiac problems was observed in two fetuses with atrial flutter, and in three cases with congenital complete AV block. Among three fetuses with polysplenic syndrome, endocardial cushion defect, or double outlet right ventricle, two were associated with 2:1 AV block and hydrops fetalis. Thus, fetal arrhythmias may be an important cause of nonimmune hydrops fetalis.

Evidence of fetal hydrops consists of fetal pleural effusion, ascites, or tissue edema, all of which are easily recognizable by routine obstetrical ultrasonography (Fig. 11). However, these may be present in a variety of conditions other than cardiac diseases. If hydrops fetalis is identified by fetal ultrasonography, one must search for structural cardiac anomalies or dysrhythmias.

Another means of evaluating fetal heart failure is to measure the total cardiac dimension (TCD) of the fetal heart<sup>4</sup>, which is the distance between the anterior wall of the right ventricle and the posterior wall of the left ventricle at the level of both artioventricular valves in the four-

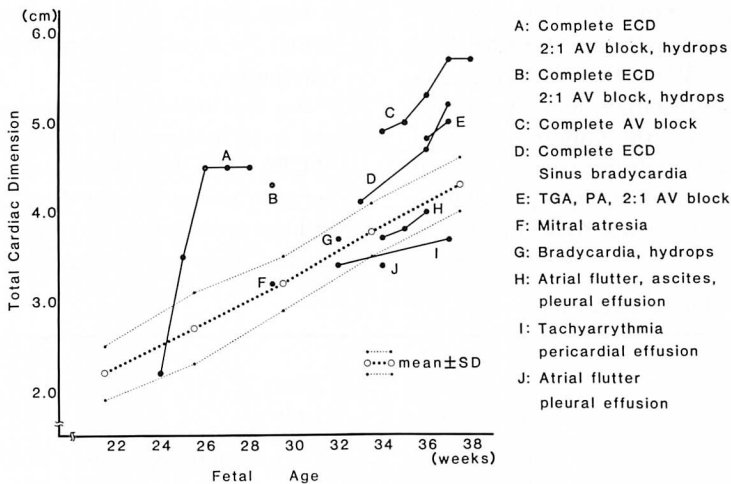
Table 3. Fetal bradycardias

Fetal arrhythmia	CHD	Fetal age (weeks)	Fetal hydrops	Outcome
2:1 AV block (3)	ECD	24	+	Intrauterine fetal death
	ECD	29	+	Death at 1 day
	DORV	36	-	Permanent pacemaker implant.
Complete AV block (4)	-	17	+	Intrauterine fetal death
	-	20 (Maternal SLE)	+	Transvenous pacing Death at 9 days
	-	25	+	Intrauterine fetal death
	-	34	-	Permanent pacemaker implant.
Sinus bradycardia (4)	-	20	-	Alive
	ECD	31	-	Death at 9 months
	TOF	32	+	Intrauterine fetal death
	ECD	36	-	Death at 3 days
SA block (1)	-	39	-	Alive



**Fig. 10. Hydrops fetalis.**

This fetus (33W) had polysplenic syndrome diagnosed postnatally. Before birth, however, the diagnosis of complete type of endocardial cushion defect was suspected by fetal echocardiography. Pleural effusion and ascites were recognized, which suggested the presence of heart failure.



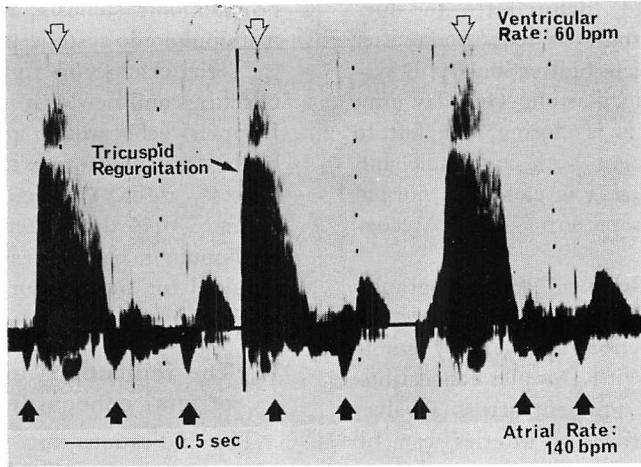
**Fig. 11. Total cardiac dimension (TCD) in normal and abnormal fetal hearts.**

Normal values of TCD of the fetus are indicated by means and standard deviations. Most of the fetuses with heart failure demonstrate increased TCDs.

chamber view. This dimension increases with fetal age, and its normal values are shown in **Fig. 11**. In fetal heart failure, the total cardiac dimension usually increases over the standard deviation of normal values. The presence of fetal hydrops and an increased total cardiac dimension are important indicators of fetal heart failure<sup>6)</sup>.

#### IV. Assessment of the physiology of fetal circulation: Fetal Doppler echocardiography

Two-dimensional scanning provides anatomical information and recently developed Doppler echocardiography provides new information concerning blood flow of normal and



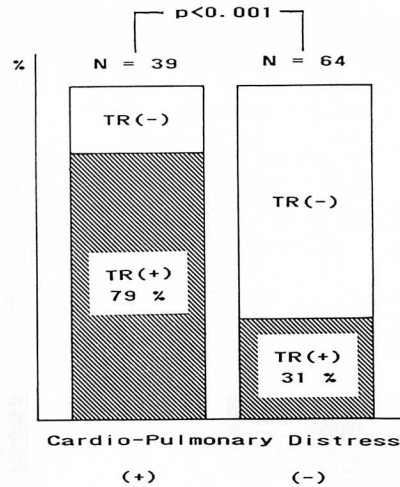
**Fig. 12. Doppler echocardiogram in a fetus (21w 6d) with complete AV block.**

The fetus, whose mother suffered from systemic lupus erythematosus, revealed complete AV block and hydrops fetalis. The large Doppler signals suggest the presence of tricuspid regurgitation (white arrows), with a ventricular rate of 60 per min. The small Doppler signals (black arrows) show atrioventricular inflow with an atrial rate of 140.

abnormal fetal circulation<sup>20-23</sup>). In the past, such information was obtained from experimental animal studies. Now, with Doppler echocardiography we can study human fetal circulation. Doppler echocardiography is sufficiently sensitive for evaluating valvular regurgitation, shunt flow and its direction, flow volume and cardiac output, and pressure gradients across the stenotic valves. We can also determine pressures in the ventricles, the pulmonary artery, and the aorta, in some cases (Fig. 12).

Fig. 13 shows the incidence of tricuspid regurgitation in fetuses and in newborns. In the presence of fetal distress or cardiopulmonary distress manifested by hypoxia, arrhythmias or heart failure, atrioventricular valve regurgitation, particularly tricuspid regurgitation, may develop. Such regurgitation may suggest the presence of myocardial dysfunction<sup>24</sup>. This regurgitation may resolve with the resolution of cardiopulmonary distress.

The physiology of the ductus arteriosus is quite interesting<sup>25</sup>. With Doppler echocardiography the flow pattern in the ductus arteriosus of humans can be known noninvasively before



**Fig. 13. Incidence of tricuspid regurgitation in fetuses and newborns.**

If fetal distress or cardiopulmonary distress such as hypoxia, arrhythmias or heart failure is present, atrioventricular valve regurgitation, particularly tricuspid regurgitation may frequently appear. Such regurgitation may suggest the presence of myocardial dysfunction.

and after birth (Fig. 14). Before birth, the ductal shunt is mainly right to left. Soon after birth, however, it directs bidirectionally in systole and left to right in diastole. One day after birth, when the ductus is closing, the left to right shunt is prominent both in systole and diastole. After the ductus is closed, a normal pulmonary artery flow pattern is easily recognizable.

Two-dimensional Doppler echocardiography (Doppler color flow mapping) can indicate the flow pattern, flow direction, and velocity simultaneously (Fig. 15). With Doppler echocardiography, the pressure gradient across a valve, or between two ventricles or arteries, can be determined noninvasively. The fetus or newborn in cardiopulmonary distress frequently has tricuspid regurgitation. Therefore, we can record the flow velocity of the regurgitant flow of the tricuspid valve, and can estimate right ventricular systolic pressure by applying a simplified Bernoulli's equation. It is nearly the same as pulmonic systolic pressure if there is

no pulmonic stenosis; therefore, we can estimate pulmonic systolic pressure. Fig. 15 shows the serial changes in right ventricular pressure of fetuses and newborn infants as estimated by Doppler echocardiography before and after birth. In a newborn without cardiopulmonary distress, right ventricular pressure declines within 3 or 4 days after birth; however, with cardiopulmonary distress, much more time is required for the pressure to decline to similar levels.

### V. The indications and ethical problems of fetal echocardiography

The indications for fetal echocardiography are summarized in Table 4. There is no evidence that ultrasonography is harmful to the human fetus; however, frequent examinations of the fetus should be avoided. Since obstetric ultrasound examination has become a standard practice in the management of normal gestations, the detection of congenital cardiac lesions and other cardiac problems has become more com-

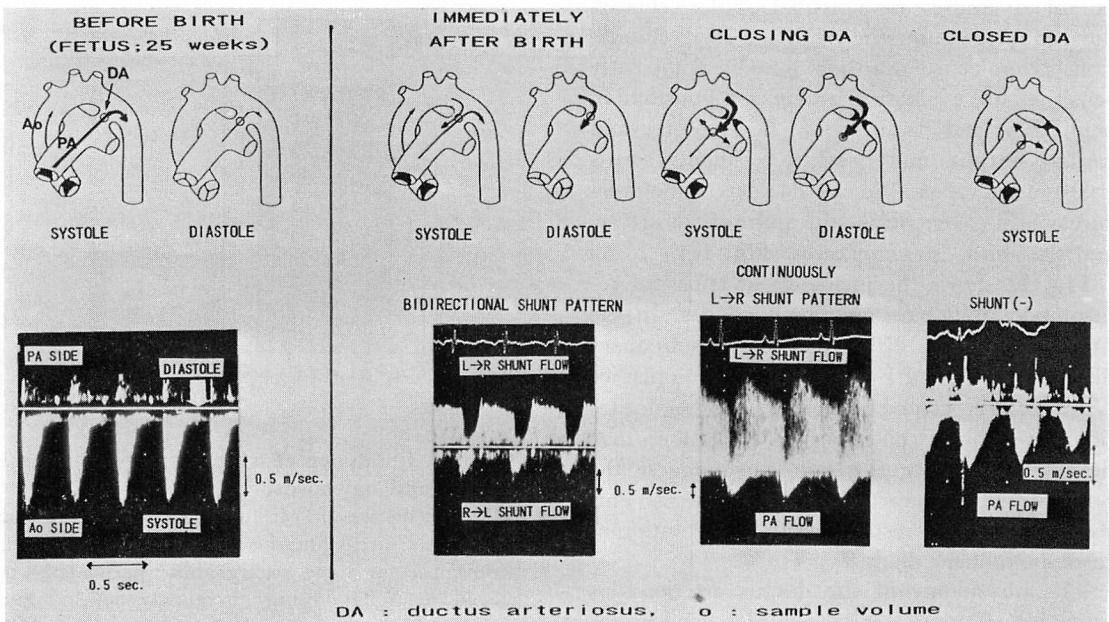
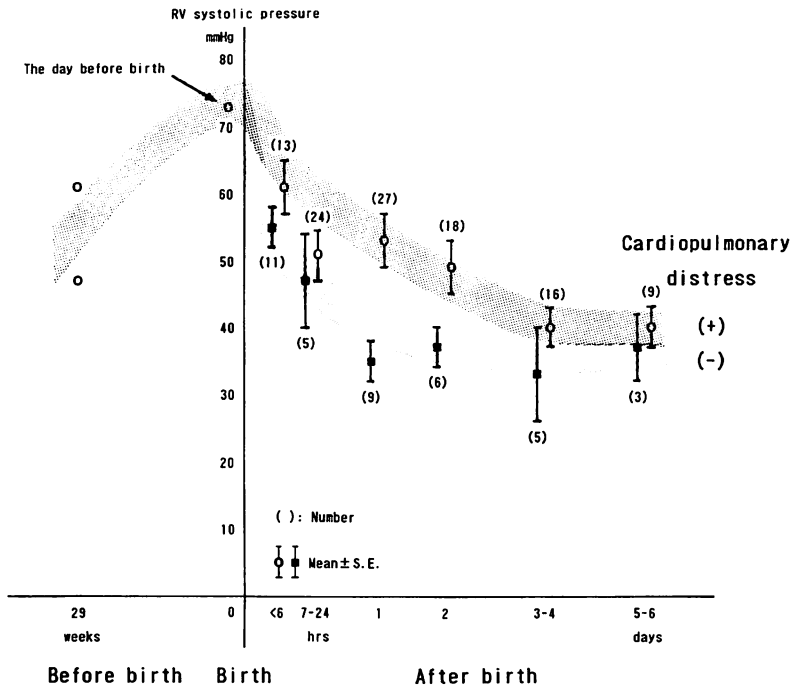


Fig. 14. Flow patterns in the ductus arteriosus of fetuses and newborns evaluated by Doppler echocardiography.



**Fig. 15. The changes in right ventricular systolic pressure.**

Right ventricular systolic pressure is assumed by the velocity of tricuspid regurgitant flow detected by Doppler echocardiography. If a newborn has no cardiopulmonary distress, right ventricular pressure declines within 3 or 4 days after birth. If a baby has cardiopulmonary distress, much more time is required for the pressure to decline to normal levels.

mon. Accordingly referrals from obstetricians to our department of pediatric cardiology have been increasing.

The management of fetal cases after the recognition of their cardiac lesions becomes a new problem.<sup>26)</sup> Currently, there is no established standard for this issue. We are using the guidelines indicated in **Table 5**. If the fetus has ductus dependent congenital heart disease such as pulmonary atresia, the hypoplastic left heart syndrome, or A-V block without severe heart failure, the mother should be referred to the institutions having specialized facilities where appropriate early medical or surgical management is available. If the fetus has a critical cardiac condition, such as congenital heart disease or complete heart block with severe heart failure,

preterm Caesarian delivery may be required after confirming evidence of lung maturity. Fetal tachyarrhythmias such as supraventricular tachycardia or atrial flutter should be treated by transplacental medication using digoxin or verapamil. Confirmation of a complicated congenital heart disease with severe malformation syndrome may permit selective abortion.

The diagnosis and management of fetal heart disease raise many important medical and ethical problems.<sup>27)</sup> In principle, we treat "a fetus as a patient". Questions for decision-making arise as to whether to abort the fetus or to change the timing or mode of delivery, or to select the alternative of intrauterine treatment. Currently, decisions are made after discussion, in agreement with the mother and the family, and with obste-

**Table 4. Indications for fetal echocardiography**

Fetal	1) Intrauterine growth retardation
	2) Fetal arrhythmia
	3) Somatic anomalies (ultrasound fetoscopy)
	4) Decreased fetal activity
	5) Abnormal genetic screen (amniocentesis)
	6) Hydrops fetalis
Maternal	1) Heart disease (congenital heart disease, myocardial disease)
	2) Drug exposure (eg, alcohol, narcotics, lithium, anticonvulsants, etc)
	3) Hydramnios
	4) Rubella
	5) Rh sensitization
	6) Metabolic disease (diabetes mellitus, phenylketonuria, etc)
	7) Collagen vascular disease (SLE)
Familial	8) Elderly gravida
	1) Congenital heart disease
	2) Genetic syndrome
	3) Anomaly syndrome

**Table 5. Management of fetal cardiac problems**

1.	Detectable in utero but treatable after delivery at term
	CHD (treated by PGE or surgery)
	Pulmonary atresia
	Coarctation of the aorta
	Hypoplastic left heart syndrome
	D-transposition of the great arteries
	Heart block without heart failure
2.	May require Caesarian delivery
	CHD requiring a preterm delivery in the presence of inadequate labor or fetal distress
	CHD associated with heart failure
	Heart block associated with heart failure
3.	May require treatment in utero
	Tachyarrhythmias (atrial flutter, SVT)
4.	Managed by selective abortion
	CHD associated with severe malformations (eg. anencephaly, porencephaly, renal agenesis, etc)
5.	Observation only; no treatment
	Most of CHD
	Arrhythmias (APC, VPC)

tricians and pediatricians according to the guidelines shown in **Table 5**.

In conclusion, this new technology has made the prenatal diagnosis of congenital heart dis-

ease possible and allows an improved outcome for patients with many types of congenital heart disease, because of early diagnosis and specific management. Fetal arrhythmias can also be

evaluated by fetal echocardiography and Doppler echocardiography, facilitating intrauterine treatment in certain cases. This technique also offers new insight into unknown aspects of normal and abnormal human fetal circulation, and contributes to research in fetal and neonatal physiology.

#### 胎児心エコー図：胎児心臓病学における新しいアプローチ

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最近の超音波機器の進歩により胎児心臓の描写が可能となり，心奇形の出生前診断や胎児不整脈を診断することが試みられている。われわれも1980年より564例の胎児（うち正常妊娠254，ハイリスク妊娠310）の心エコー図を検討してきた。これらの結果にもとづき今までの内外の主な研究結果をまじえて，以下の点につき概説する。

胎児心の構造診断は，左室長軸断面，四腔断面，大血管断面，大動脈弓断面の四つを基本断面とし，系統的にアプローチすることにより可能であった。564例中13例（2.3%）に先天性心疾患の出生前診断ができ，そのタイプは，心内膜床欠損，単心室，ファロー四徴，大動脈縮窄，大血管転位，左心形成不全症候群，肺動脈閉鎖，大きい心室中隔欠損などであった。1例の小さい心房中隔欠損が出生前に診断できなかつた。出生後すぐに重篤になる疾患のほとんどは出生前診断が可能であり，適切な施設で分娩させ，適切な内科的および外科的治療が敏速にできるようになった。

胎児の不整脈は心房壁と心室壁のMモード心エコー図を同時記録することにより，診断できた。上室性頻拍や心房細動などの頻拍性不整脈は6例に見られうち5例にジゴキシンによる経胎盤的治療を行い，3例に成功した。徐脈性不整脈は12例に診断され，半数に先天性心疾患を伴っていた。うち4例は子宮内死亡し，2例に出生後すぐにペースメーカー植え込みを行った。

胎児の心不全の診断や病態に関しては不明の点が多かったが，胎児心エコー図の導入により胎児水腫の存在や全心径（total cardiac dimension）の計測により可能となった。また胎児心不全の原因として前述の頻拍性不整脈や徐脈性不整脈，または一部の先天性心疾患が重要であり，胎児仮死や死亡の原因となることが分った。

胎児から新生児にいたる循環動態の変化は古くより興味をもたれ研究の対象となってきたが，主に動物実験によりなされていた。ドップラー心エコー図の導入によりヒトでの研究が可能となった。胎生期から新生児期にわたる動脈管の血流動態，肺血管抵抗の変化，cardio-pulmonary distressにおける房室弁逆流などについて検討したので報告した。

胎児心エコー図の導入により，胎児・新生児病学に新たな光が差し込まれ，今後，重要な心臓病学の一分野となるであろう。それに伴い新たな医学的ないし倫理的問題が生じてくる。重度の障害が発見された時，妊娠を継続するか，またはどのようにマネージするか，いつ分娩させるか，など多くの問題が新たに生じてきた。これらの問題に関しても現時点におけるconceptを述べた。

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