CONGENITAL HEART DISEASE

Evaluation of fetal arrhythmias from simultaneous pulsed wave Doppler in pulmonary artery and vein

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Objective: To evaluate the clinical application of simultaneous recordings of pulsed wave Doppler (PWD) signals in pulmonary artery and vein as alternative sampling site for assessment of arrhythmias in the fetus.

Design: Prospective, cross-sectional study.

Setting: Tertiary referral centre for fetal cardiology.

Patients and methods: From July 1999 to July 2005 PWD was used in pulmonary vessels to assess fetal arrhythmias at 15–40 weeks' gestation. Sample volume placement in the peripheral lung vessels was guided by colour flow mapping on a four-chamber section of the fetal heart. Atrial and ventricular systoles were identified from the pulmonary venous and arterial signals respectively. M-mode recordings were used for comparison.

Outcome measures: Diagnosis of fetal arrhythmias.

Results: Of 129 cases, 15 had supraventricular tachycardia, 12 with 1:1 atrioventricular conduction and 3 with atrial flutter and 2:1 block. There were 96 cases of atrial and 7 of ventricular premature beats, 2 of sinus bradycardia, 8 of variable degree heart block and 1 of ventricular tachycardia. PWD was diagnostic in 119 cases. PWD was better than M mode for diagnosis of premature beats and added information about mechanisms of tachycardia. Both methods facilitated interpretation of all arrhythmia patterns, although PWD was of less practical value in cases of complete heart block.

Conclusion: Simultaneous PWD recording of pulmonary vessels in the fetus allows accurate diagnosis of arrhythmias. It is easily obtained with standard ultrasound equipment and adds to the armamentarium of diagnostic techniques for assessment of rhythm abnormalities prenatally.

M-mode imaging (M mode) and pulsed wave Doppler (PWD) echocardiography are standard techniques used to assess cardiac rhythm in the fetus.^{1–6} Each method has advantages and limitations, which are influenced by image resolution, fetal position and complexity of the arrhythmia. Newer techniques such as tissue velocity imaging and magnetocardiography have also been used,^{7,8} but neither is widely available, restricting their clinical applicability. We have used simultaneous sampling of the peripheral pulmonary artery (PA) and vein (PV) to study PWD patterns and the atrioventricular (AV) interval during sinus rhythm (SR) in the fetus from 15 weeks' gestation to term (unpublished). In this study we evaluate the use of this technique for diagnosis of abnormal rhythms in the human fetus.

METHODS

This study includes all cases of fetal arrhythmia investigated by PWD recordings of the PA and PV at a tertiary referral centre (July 1999 to July 2005). We used an HDI 5000 (Advanced Technology Laboratories, Bothell, WA, USA) or SSD 5500-PHD (Aloka, Tokyo, Japan) ultrasound system with 3–7.5 MHz probes, as appropriate. Fetal echocardiography (FE) was performed owing to suspected arrhythmia or structural abnormality. All patients gave verbal informed consent before FE. Cardiac anatomy was assessed using the sequential segmental approach.^{9–10} For PWD assessment, the pulmonary vessels were identified on grey scale and colour flow mapping on a four-chamber view, using preset colour optimisation for low velocities. The sample volume size was adjusted to allow simultaneous recording from a branch PA and PV in the inner two-thirds of lung parenchyma. All recordings were obtained in the absence of fetal body or breathing movements. Atrial ("A")

and ventricular systole ("V") were identified from pulmonary venous and arterial traces, respectively. The venous trace shows a characteristic triphasic waveform where ventricular systole ("S"), early diastole ("D") and atrial systole ("A") are identified separately¹¹ (fig 1A).

Bradycardia and tachycardia were defined as heart rate <100 bpm and >180 bpm, respectively. Cardiac rhythm was analysed at the time of FE to enable clinical decision making. PWD recordings were stored digitally and in S-VHS format for further analysis if necessary. To validate interpretation of the arrhythmia using this new Doppler approach, M-mode recordings were also obtained in all cases early in the study period. After the initial learning curve, however, we no longer used M mode for diagnosing isolated premature beats as these were more readily identified by PWD, but we continued to record M mode in all other cases.

RESULTS

One hundred and twenty-nine fetuses had rhythm abnormalities. Median gestational age at diagnosis was 31 weeks (range 15–40). One hundred and twenty-one had normal cardiac anatomy, but eight had an associated structural defect: aortic coarctation (two), ventricular septal defect (two), tetralogy of Fallot (one), partial anomalous pulmonary venous connection (one), right and left isomerism (one each). At presentation, five had hydrops fetalis: two with complete heart block, three with tachycardia (intermittent in one). Another four fetuses with tachyarrhythmia had ascites, pericardial or pleural effusions.

Abbreviations: AV, atrioventricular; FE, fetal echocardiography; PA, pulmonary artery; PWD, pulsed wave Doppler; SR, sinus rhythm; PV, pulmonary vein; SVT, supraventricular tachycardia; VA, ventriculo-atrial

Figure 1 (A) Pulsed wave Doppler recording of pulmonary vessels during sinus rhythm where atrial systole corresponds to reduction of PV flow to the LA. (B–D) Tachycardia with 1:1 atrioventricular (AV) conduction. (B) Characteristic pattern observed with short ventriculo-atrial (VA) tachycardia (~225 bpm, VA interval = 45 ms, AV interval = 225 ms, VA/AV ratio = 0.2). PV flow is detected below the baseline towards the LA and above the baseline as retrograde flow to the lungs (to and fro pattern). (C) Long VA tachycardia resembled sinus rhythm (~190 bpm, VA interval = 209 ms, AV = 109 ms, VA/AV ratio = 1.92). (D) Atrial ectopics (arrows) precede a short VA tachycardia (heart rate ~210–220 bpm, VA interval = 130 ms, AV interval = 150 ms, VA/AV ratio = 0.87). PA, pulmonary artery; PV, pulmonary vein; LA, left atrium; AE, atrial ectopic. In PV signal: A/S, atrial/ventricular systole; D, early diastole. In PA signal: V, ventricular systole.

Figure 2 Composite pulsed wave Doppler (PWD) recording of pulmonary vessels (A) and M mode (B) from the same fetus with atrial flutter and 2:1 atrioventricular block (see text). The vertical dashed lines show the corresponding atrial and ventricular activity both on PWD signal and M mode. HR, heart rate; PA, pulmonary artery; PV, pulmonary vein; LA, left atrium; V, ventricular systole, f, flutter wave.

Premature beats were the most common arrhythmia (96 of atrial and seven of ventricular origin). Heart rate was usually normal, including two cases of atrial trigeminy. Six others had bradycardia (persistent in two) due to blocked atrial bigeminy. There were 10 additional cases of bradycardia, eight with different degrees of AV block and two with sinus bradycardia. One fetus presenting with frequent atrial ectopics developed sustained tachycardia 2 weeks later. Sixteen fetuses had tachycardia, one with ventricular and 15 with supraventricular tachycardia (SVT). Of these, 12 showed 1:1 AV conduction and three had atrial flutter associated with AV block.

In the absence of fetal breathing movements, recordings were obtained in less than 5 minutes in most cases. The diagnosis was established with PWD in 119 of 129 cases. In five cases with atrial premature beats (infrequent or occurring during active "breathing" movements) and in one with complete heart block (17-week hydropic fetus), the diagnosis was established on M mode and no further attempts to perform PWD were made. In another four, initial attempts to interrogate the pulmonary vessels were non-diagnostic. One fetus had intermittent heart block. Two other term fetuses (one atrial flutter, one SVT with 1:1 AV conduction) were delivered shortly afterwards and no further attempts to record PWD were made. In the fetus with ventricular tachycardia, functional pulmonary atresia during the tachyarrhythmia precluded the use of this technique.

Supraventricular tachycardias

SVT with 1:1 AV conduction was documented by PWD in 11/12 cases seen, including one at 16 weeks. Heart rates varied from 190 to 350 bpm. Measurements of the AV and ventriculo-atrial (VA) intervals allowed the arrhythmia to be classified as either short or long VA interval, analogous but not identical to short or long RP tachycardia on the ECG. Eight showed a short VA tachycardia (VA/AV ratio <1) (fig 1B) and two showed a long VA interval (VA/AV ratio >1) (fig 1C). In three cases, initiation of SVT triggered by atrial premature contraction was documented (fig 1D).

The PWD pattern of short and long VA tachycardia differed. Short VA SVT had VA/AV ratios <1 and a pattern distinct from that observed during SR (figs 1B and D). Heart rates varied from 200 to 250 bpm. Characteristically, the PWD signal in the PV showed variable degree of summation of the "S" and "D" waves (forward velocities). In seven cases reversal of flow during atrial systole was seen. To some extent, this masked the arterial forward flow signal during SVT (fig 1B), owing to their close temporal relationship: venous waveforms were easily detected, whereas signals from the peripheral PA had to be optimised, usually by adjusting the sample volume. Conversely, in the two cases with long VA mechanism (VA/AV ~ 2 in both cases, heart rate 190–210 bpm), the "A" wave preceded the "V" wave and the signal resembled that seen during SR (fig 1C). In one, the tachyarrhythmia was intermittent and alternated with episodes of bradycardia (~ 80 bpm) owing to atrial bigeminy. This was compatible with an atrial ectopic tachycardia (fig 1C). The second case with long VA mechanism was referred at 14 weeks' gestation owing to an increased nuchal translucency. The heart appeared structurally normal with heart rate ~ 200 bpm. A persistent long VA tachycardia was diagnosed at 20 weeks.

In the remaining case with 1:1 AV conduction, there were intermittent, short-lived but frequent periods of tachycardia (heart rate 260–350 bpm). Neither PWD nor M mode allowed distinction between SVT (re-entrant or ectopic) and atrial flutter with 1:1 conduction.

Three cases of atrial flutter (2:1 AV conduction) were found. A PWD recording was obtained in two. A monophasic forward venous waveform was present. Atrial activity ("flutter waves", rate of ~ 450 –500 bpm) was timed in between forward velocity waveforms (corresponding to return to baseline), with no obvious flow reversal. Venous waveforms alternated between lower and higher amplitude signals. The latter preceded a "V" wave on the PA trace. Every other "flutter wave" was not followed by a ventricular contraction, characterising a 2:1 pattern (figs 2A and B).

Figure 3 Pulsed wave Doppler recording of pulmonary vessels from four fetuses with atrial (A–C) and ventricular ectopics (D). A ventricular waveform ("V") is seen in the arterial signal with a conducted beat (A) but not with a blocked one (B, C). The distance between two vertical bars (C, D) represents the A–A interval in sinus rhythm. Note a non-compensatory pause with premature atrial contraction (C) and a compensatory pause with the ventricular ectopic (D). Ectopic-related arterial waveforms were of reduced amplitude (A, D). In (B) the post-extrasystolic arterial waveform shows a higher amplitude. A/V, atrial/ventricular waveforms, AE/VE, atrial/ventricular ectopic; PA, pulmonary artery.

Figure 4 Composite pulsed wave Doppler (PWD) recording of pulmonary vessels (A) and M mode (B) from the same fetus with bradycardia (~80 bpm) due to blocked atrial bigeminy. PWD from other fetuses (C–E) with atrial trigeminy (C), 2:1 atrioventricular (AV) block (D) and first-degree AV block (E). In (C) note a regular pattern of two atrial/sinus contractions followed by a non-conducted ectopic, heart rate ~100–110 bpm. In (D) every other “A” wave is not followed by a “V” wave. In (E) the distance between asterisks shows the AV interval ~200 ms. A, atrial systole; AE, atrial ectopic; V, ventricular systole.

Premature beats

Atrial ectopics were a common arrhythmia promptly recognised by an early “A” wave (early notch or retrograde wave) disrupting the regular A–A timing during SR (figs 3A–C). They were more easily documented by PWD than M mode, irrespective of fetal position. The morphology of “S” and “D” waves following the premature contraction varied: the later the ectopic, the more resemblance to a sinus beat. A pattern of atrial bigeminy with ventricular bradycardia was present in six cases, heart rate ~80 bpm (figs 4A and B). Two fetuses had non-conducted atrial trigeminy, heart rates ~100–110 bpm (fig 4C).

Ventricular premature beats, identified as early “V” waves in the PA recordings were documented in seven cases. The A–A interval remained regular but the venous waveform pattern that followed the ventricular extrasystole differed from sinus beats (fig 3D).

Sinus bradycardia

Two cases were found of sinus bradycardia with a baseline fetal heart rate of 90–95 bpm. The PWD pattern showed a regular sequence of atrial and ventricular contraction, the only abnormal finding being the slower heart rate.

Heart block

Second-degree heart block with 2:1 AV conduction was documented by PWD in three fetuses (fig 4D). Maternal anti-SSA antibodies were positive in two cases. In one fetus this

progressed from 2:1 to 3:1 AV block (ventricular rate ~40 bpm). The newborn developed complete heart block, requiring cardiac pacing. In the other, the rhythm alternated between first- and second-degree block. In this case, PWD (fig 4E) and M mode offered complementary diagnostic information. No pacing has been required to date. Maternal antibodies were negative in the third case. The AV block resolved in utero, and the newborn was in SR with a normal QT interval.

In three documented cases of third-degree block, the arterial trace clearly showed the slower idioventricular rhythm (rates ~60 bpm). However, recognition and timing of a regular pattern of atrial contractions was easier on M mode, where they are identified as single waveforms, in contrast to the non-uniform appearance on PV signal, where “A” waves are superimposed at random on the underlying venous signal (“S” and “D” waves). To facilitate PWD interpretation, recordings were made at a slower speed.

Postnatal documentation

In most cases seen in this series, the arrhythmia was no longer present postnatally. Fetuses with isolated atrial ectopics, the commonest type of arrhythmia, were not routinely followed up after birth. Atrial bigeminy and trigeminy resolved in utero in all but one case, in whom a postnatal ECG confirmed the nature of the arrhythmia. Prenatal control of tachyarrhythmias was achieved in most cases (including short VA tachycardia). In those in whom the arrhythmia persisted or recurred

postnatally, the diagnosis was confirmed by 12-lead ECG or 24-hour ECG monitoring. The fetus with 1:1 tachycardia rates of 260–350 had short periods of apparent atrial flutter as a neonate. One intrauterine death occurred in a hydropic fetus with atrial flutter. Antenatal diagnosis of persistent AV block was recorded postnatally in all newborns.

DISCUSSION

We have documented patterns of arrhythmias by recording simultaneous PWD in the peripheral PA and PV in the fetus. This provides an alternative method for assessment of abnormal cardiac rhythms prenatally.

Analysis of cardiac rhythm is based on the ability to record atrial and ventricular contractions simultaneously. Traditionally in the fetus, M-mode and Doppler echocardiography have been used to identify these events.^{1–4} PWD Doppler sampling can be performed at various sites, including the left ventricular inflow–outflow tract area,² inferior vena cava–descending aorta⁵ and superior vena cava–ascending aorta.⁶ More recently, tissue velocity imaging, fetal electrocardiography and magnetocardiography have also been used to assess fetal cardiac rhythms.^{7–8, 12–14}

Conventional M-mode and Doppler techniques when used for assessment of AV conduction are inevitably dependent on fetal position and familiarity with their interpretation. Additionally, defining AV and VA intervals on M mode can be difficult as onset and offset times can be unclear. Newer techniques are being investigated, but the apparatus needed to perform them is available only in research centres.^{7–8, 12–14} Magnetocardiography also requires a dedicated area isolated from magnetic fields, and its feasibility in an unshielded clinical setting has only recently been reported.¹⁵ Although magnetocardiography can be performed from mid-gestation onwards,⁸ fetal ECG is frequently unsuccessful before 32 weeks¹² and both are based on signal averaging. This makes analysis of irregular patterns, such as those of premature beats, more difficult.

Contrary to other Doppler methods, using PWD in pulmonary vessels is relatively independent of fetal position¹¹ owing to the multiplicity and different orientation of these vessels in the lung parenchyma, which allow a wide range of insonation windows through the fetal chest. Thus, irrespective of fetal position, it was possible to obtain the PWD signal. Additionally, this Doppler approach is not dependent on image resolution of intracardiac structures and can be applied at the level of the four-chamber view—a standard obstetric section plane. To obtain diagnostic M-mode recordings, good resolution B-mode images are often necessary.

In the present study, pulmonary PWD recordings established the diagnosis of fetal arrhythmia in most cases. This was verified using M mode as a comparative method in all cases of complex arrhythmias and postnatally whenever available. In practice, the technique offered a rapid means of establishing the precise diagnosis of premature beats, tachyarrhythmias (short and long VA mechanisms and atrial flutter) and bradycardias (sinus, atrial bigeminy and trigeminy and incomplete heart block).

The AV interval on ultrasound acts as a surrogate for the PR interval on the ECG. The relationship VA to AV interval gives insight into mechanisms of tachycardia.^{6–16} A short VA interval (VA/AV ratio <1) was considered compatible with an AV re-entrant mechanism associated with a fast conducting VA pathway (RP interval < PR interval), whereas a long VA tachycardia (VA/AV ratio >1) may reflect sinus tachycardia, atrial ectopic tachycardia or a re-entrant mechanism due to a slow conducting VA pathway. Two possible mechanisms were thought to account for the long VA tachycardias seen here, the

distinction being made on natural history (persistence, long RP tachycardia) or coexistence of multiple atrial ectopic beats as well as runs of atrial bigeminy (atrial ectopic tachycardia). Identification of mechanisms of tachycardia may influence drug treatment, if this is needed. In our series, however, we treated the patient with persistent long VA tachycardia with flecainide—our preferred treatment option for SVT over this time period, despite a different mechanism of tachycardia being identified. Our decision was based on our experience in monitoring the use of flecainide in pregnancy and not on the mechanism of SVT. The arrhythmia was partially controlled (rate reduced from 210 to 180 bpm) and digoxin was added subsequently. At the end of pregnancy, the rhythm was regular (rate ~150–160 bpm). The neonatal ECG showed sinus rhythm. In retrospect, treatment with a different drug such as sotalol might have been preferable.

The ability quickly to obtain a diagnostic signal in cases of atrial premature beats was of considerable clinical utility as this is the most common arrhythmia seen prenatally. After characterising and validating the arrhythmia pattern by comparison with M mode, we no longer use M mode if a diagnostic Doppler pattern can be obtained. It was also easy to teach sonographers and obstetricians how to obtain and interpret the signal, thus minimising the number of referrals to the fetal cardiologist for a specialised scan.

Fetal breathing movements may interfere with the interpretation of the signal. This is overcome by waiting a few minutes for a period of fetal quiet. A second and perhaps more important limitation was encountered when assessing bradyarrhythmias with a very low ventricular rate in association with a non-regular relation between atrial and ventricular activities such as in cases of complete AV block. In these cases, identification and timing of atrial activity as seen on PWD was hampered by difficulty in recognising the “A” wave against a background of low or absent venous velocities in different phases of the cardiac cycle. To some extent, this could be minimised by recording the Doppler signal at low speed. In cases of complete heart block, however, M mode proved to be more practical.

Simultaneous use of PWD in pulmonary vessels is an easy and quick method to assess fetal cardiac rhythm and allows precise diagnoses of cardiac arrhythmias. It adds to the armamentarium and complements other diagnostic tools available to those dealing with complex abnormalities of fetal cardiac rhythm.

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REFERENCES

- 1 **Allan LD**, Anderson RH, Sullivan ID, *et al*. Evaluation of fetal arrhythmias by echocardiography. *Br Heart J* 1983;**50**:240–5.
- 2 **Stewart PA**, Tonge HM, Wladimiroff JW. Arrhythmia and structural abnormalities of the fetal heart. *Br Heart J* 1983;**50**:550–4.
- 3 **DeVore GR**, Siassi B, Platt LD. Fetal echocardiography. III. The diagnosis of cardiac arrhythmias using real-time-directed M-mode ultrasound. *Am J Obstet Gynecol* 1983;**146**:792–9.
- 4 **Kleinman CS**, Donnerstein RL, Jaffe CC, *et al*. Fetal echocardiography. A tool for evaluation of in utero cardiac arrhythmias and monitoring of in utero therapy: analysis of 71 patients. *Am J Cardiol* 1983;**51**:237–43.

- 5 **Chan FY**, Ghosh A, Tang M, *et al.* Simultaneous pulsed Doppler velocimetry of fetal aorta and inferior vena cava. Diagnosis of fetal congenital heart block; two case reports. *Eur J Obstet Gynecol Reprod Biol* 1990;**35**:89–95.
- 6 **Fouzon JC**, Fournier A, Proulx F, *et al.* Management of fetal tachyarrhythmia based on superior vena cava/aorta Doppler flow recordings. *Heart* 2002;**89**:1211–6.
- 7 **Rein AJ**, O'Donnell C, Geva T, *et al.* Use of tissue velocity imaging in the diagnosis of fetal cardiac arrhythmias. *Circulation* 2002;**106**:1827–33.
- 8 **Stinstra J**, Golbach E, van Leeuwen P, *et al.* Multicentre study of fetal cardiac time intervals using magnetocardiography. *Br J Obstet Gynaecol* 2002;**109**:1235–43.
- 9 **Shinebourne EA**, Macartney FJ, Anderson RH. Sequential chamber localization: a logical approach to diagnosis in congenital heart disease. *Br Heart J* 1976;**38**:327–40.
- 10 **Carvalho JS**, Ho SY, Shinebourne EA. Sequential segmental analysis in complex fetal cardiac abnormalities: a logical approach to diagnosis. *Ultrasound Obstet Gynecol* 2005;**26**:105–11.
- 11 **DeVore GR**, Horenstein J. Simultaneous Doppler recording of the pulmonary artery and vein: a new technique for the evaluation of a fetal arrhythmia. *J Ultrasound Med* 1993;**12**:669–71.
- 12 **Taylor MJ**, Smith MJ, Thomas M, *et al.* Non-invasive fetal electrocardiography in singleton and multiple pregnancies. *Br J Obstet Gynaecol* 2003;**110**:668–78.
- 13 **Wakai RT**, Strasburger JF, Li Z, *et al.* Magnetocardiographic rhythm patterns at initiation and termination of fetal supraventricular tachycardia. *Circulation* 2003;**107**:307–12.
- 14 **Sturm R**, Muller HP, Pasquarelli A, *et al.* Multi-channel magnetocardiography for detecting beat morphology variations in fetal arrhythmias. *Prenat Diagn* 2004;**24**:1–9.
- 15 **Brisinda D**, Comani S, Meloni AM, *et al.* Multichannel mapping of fetal magnetocardiogram in an unshielded hospital setting. *Prenat Diagn* 2005;**25**:376–82.
- 16 **Jaeggi E**, Fouzon JC, Fournier A, *et al.* Ventriculo-atrial time interval measured on M mode echocardiography: a determining element in diagnosis, treatment, and prognosis of fetal supraventricular tachycardia. *Heart* 1998;**79**:582–7.

IMAGES IN CARDIOLOGY

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Lung cancer in natural history crossing the pericardium: a rare echocardiographic view

A 70-year-old man, with an untreated and histologically undefined (of his own free will) lung cancer (diagnosed 5 years before) was admitted to our ambulatory for increasing dyspnoea and progressive exercise intolerance. Apical four-chamber echocardiographic findings (panel A) were a large and fixed bilobate mass on the left atrial (LA) lateral wall (that appeared firm) and a little pericardial serous effusion. Pulmonary artery pressure was 45/14 mm Hg. The thoracic

contrast-enhanced computed tomography (CT, panel B) showed a wide pulmonary cancer affecting the left main bronchus with lung collapse, the left pulmonary artery (completely obliterated) and the left atrium crossing the pericardium (by continuity; panel B, dotted arrows).

A left atrial mass, generally rare, is more infrequently due to a secondary cardiac tumour (because of the heart contractility and the high coronary flow velocity). In our patient, the atrial mass was uniform (an

overlapped thrombosis was unlikely) and bilobate (probably for an associated metastatisation through a left pulmonary vein). The echocardiographic view (with an unmistakable CT image) of metastatisation from the lung (the most common metastatic cardiac tumour) into the heart by continuity is rare, almost certainly, due to a prolonged natural history.

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