RHYTHM ABNORMALITIES OF THE FETUS

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Heart 2007;**93**:1294–1300. doi: 10.1136/hrt.2005.069369

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The myocardium begins to contract rhythmically by 3 weeks post-conception as a consequence of the activity of spontaneously depolarising myocardial pacemaker cells of the embryonic heart, and its maturation continues even into the postnatal period. While the exact timing of onset of the atrioventricular (AV) electromechanical relationship remains speculation in humans, by 6 weeks post-conception AV synchrony can be demonstrated using standard Doppler techniques. By 5–6 weeks the normal mean fetal heart rate is 110 beats/min (bpm). With further growth and maturation of the conduction system, including definition of the sinoatrial node as the primary cardiac pacemaker with its highest intrinsic rate of spontaneous depolarisation, there is a subsequent increase in the rate to 170 bpm by 9–10 weeks. The rise in heart rate is followed by a decrease to 150 bpm by 14 weeks, likely as a consequence of increasing parasympathetic control and improved myocardial contractility. By 20 weeks the average (SD) fetal heart rate is 140 (20) bpm with a gradual decrease to 130 (20) bpm by term. In the healthy fetus the heart rate is regular, usually remains between 110 and 180 bpm, and has a beat-to-beat variation of 5–15 bpm.

FETAL RHYTHM ABNORMALITIES

Fetal rhythm abnormalities, which include fetal heart rates that are irregular, too fast or too slow, occur in up to 2% of pregnancies and account for 10–20% of the referrals to fetal cardiologists. They are usually identified by the obstetrical clinician who detects an abnormal fetal heart rate or rhythm using a Doppler ''listening device'' at routine assessment of the pregnant mother. While such clinical assessments begin at 12–14 weeks, most fetal arrhythmias are detected only after 20 weeks. The vast majority of affected pregnancies have isolated premature atrial contractions which may have even spontaneously resolved by fetal echocardiographic assessment. Less than 10% of referrals for fetal rhythm abnormalities have a sustained tachyarrhythmia or bradyarrhythmia considered to be of clinical significance, as they may indicate severe systemic disease or may have the potential to compromise the fetal circulation themselves. For such abnormalities prenatal diagnosis with the potential for prenatal, perinatal and neonatal intervention may be critical and may ultimately improve outcome. This article will review the diagnostic modalities currently employed in the assessment of fetal rhythm disturbances, the types of arrhythmias encountered before birth, as well as the management and outcome of fetal arrhythmias with and without intervention.

ASSESSMENT OF FETAL RHYTHM

Accurate diagnosis of rhythm and conduction abnormalities can be challenging given the lack of conventional ECGs. While fetal electrocardiography has been described, the low p wave voltage, frequent inability to document heart rates above and below a normal range, particularly at rates that are similar to the mothers, limitations in fetal signal acquisition between 28–34 weeks, and use of signal averaging make assessment of the majority of fetal arrhythmias by electrocardiography at present not feasible or practical.

Most centres currently rely on echocardiographic modalities to define the fetal rhythm which include M mode, and pulse and tissue Doppler (fig 1). The echocardiographic diagnosis of fetal arrhythmias involves the assessment of the chronological relationships between atrial and ventricular contractions, with assumptions about their electrophysiological relationships. In M mode assessment, a simultaneous recording of atrial and ventricular wall motion is acquired. The quality of the assessment is significantly affected by image resolution and fetal position. Pulsed Doppler interrogation through the left ventricular inflow and outflow tract, with evaluation of the relationship between the a wave during atrial contraction and the outflow signal, can provide additional information although it is limited when atrial contraction occurs against a closed AV valve such as may occur in AV block. More recently, Fouron and colleagues described the use of a

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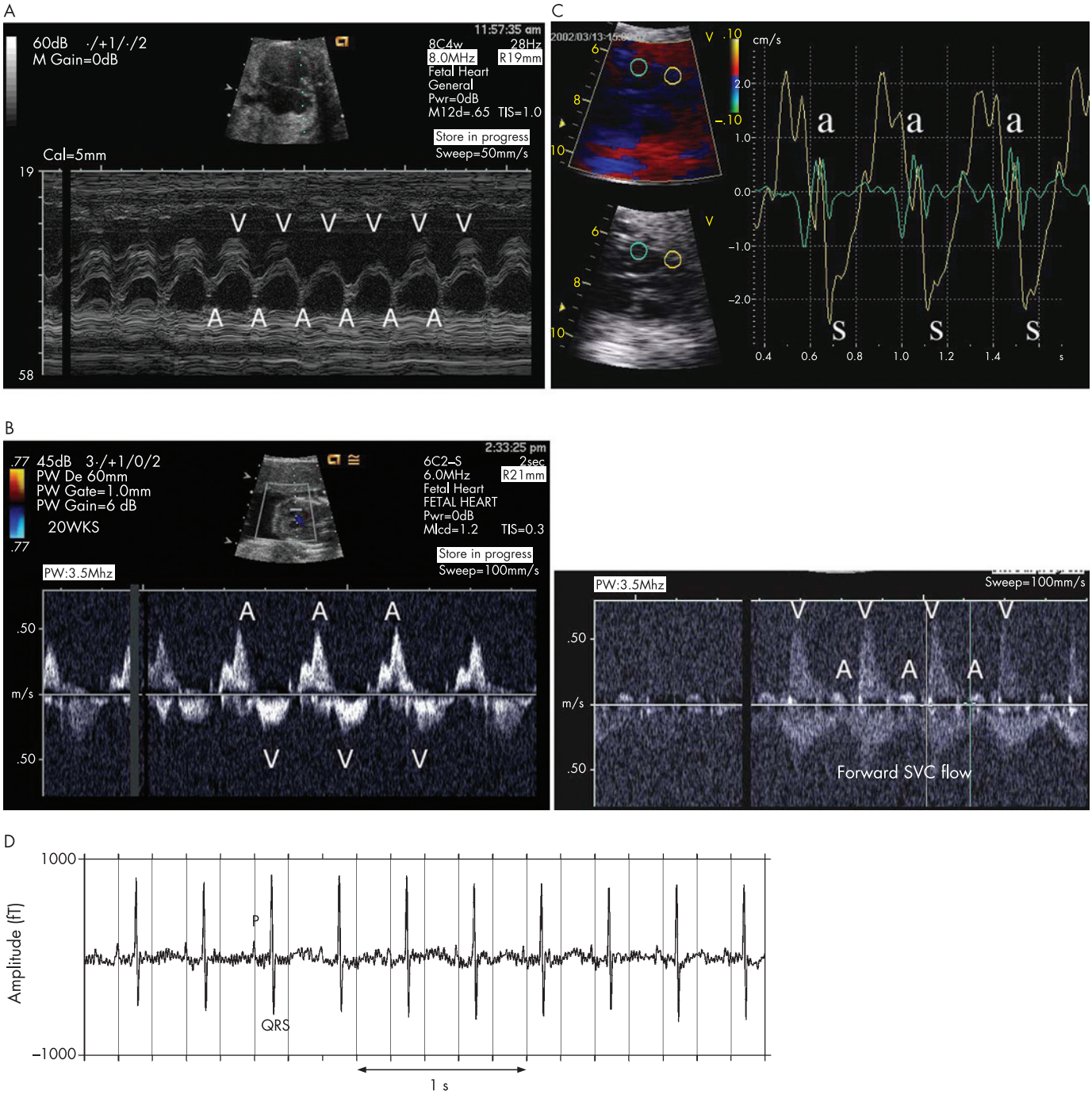

Figure 1 Techniques employed in the assessment of the fetal heart rate and rhythm. (A) M mode assessment in which the beam is directed through a portion of the right atrium (A) and right ventricle (V) demonstrating 1:1 mechanical atrial and ventricular relationship. (B) These Doppler spectra demonstrate Doppler assessment of fetal rhythm with simultaneous sampling of the left ventricular inflow (A: flow during atrial systole) and outflow (V: ventricular outflow) (left) and superior vena cava (SVC) and aorta (right). A: retrograde flow in SVC during atrial systole; V: forward flow in the ascending aorta during ventricular systole. (C) Left, four chamber, long axis view from which simultaneous tissue velocity data are obtained for left atrial (green) and left ventricular (yellow) wall motion. Right, the tissue velocity curves demonstrate a normal 1:1 relationship between atrial and ventricular contractions. (D) Magnetocardiogram obtained in a fetus with a normal atrial rhythm. Both P and QRS waves can be identified. (Image kindly contributed by Professor Ronald Wakai Director of the Biomagnetism Laboratory, UW-Madison.)

simultaneous superior vena cava and ascending aorta Doppler assessment obtained in a sagittal view through the fetal thorax.[1] With this technique, which demonstrates the relationship between atrial contraction (blood flow reversal in the superior vena cava) and ventricular contraction (forward pulsatile flow in the ascending aorta), A–V and ventriculo-atrial time intervals can be defined. Tissue velocity imaging has recently emerged as another technique for fetal arrhythmia assessment.[2] Raw data acquired from high frame rate two-dimensional tissue velocity images, in which either atria and ventricle are simultaneously sampled, provide precise temporal data of atrial and ventricular events. Tissue velocity imaging has been shown to permit

acquisition of such data more readily and consistently than M mode and Doppler techniques.

Over the past decade, magnetocardiography has been applied to obtain electrophysiological information in fetuses. This modality records the magnetic field created by the electrical activity of the fetal heart with generation of waveforms that are very similar to those observed by electrocardiography[3] (fig1D). Limitations of this technique include need for a magnetically shielded room, use of signal averaging, and lack of availability of the technology in many centres.

EXTRASYSTOLES

Premature atrial contractions (PACs) are usually identified in third trimester fetuses and their frequency may be highly variable. They may be conducted or blocked at the AV node. When there is atrial bigeminy, even with AV conduction, the Doppler listening devices may not detect the Doppler signal of the ventricular output following the extrasystole. Such pregnancies are often referred for intermittent or persistent fetal bradycardia. While in isolation these are usually benign, resolving just before or shortly after birth, 1–3% of affected fetuses have intermittent runs of supraventricular tachycardia which can lead to compromise. Even if rare, PACs identified in the fetus with cardiomegaly, evidence of ventricular systolic dysfunction, AV valve regurgitation and/or hydrops suggests the possibility of supraventricular tachycardia. As such, ongoing weekly or biweekly obstetrical ultrasound assessment is warranted until they have resolved or until delivery. Less than 10% of affected fetuses have associated structural heart disease, including lesions associated with significant tricuspid or mitral insufficiency with atrial dilation and intracardiac tumours; in such fetuses, the minor haemodynamic alterations that occur with the extrasystoles may not be tolerated.

Premature ventricular contractions in the fetus are rare. When detected, they too may be benign, but exclusion of myocardial disease, intracardiac tumours and any evidence of cardiovascular compromise is warranted. Serial prenatal assessment and further evaluation after birth is always indicated to confirm the nature of the ectopy and exclude intermittent runs of more sinister tachyarrhythmias.

FETAL TACHYARRHYTHMIAS

Fetal tachycardia is diagnosed when the ventricular rate exceeds 180 bpm. Most tachyarrhythmias demonstrated after birth have also been diagnosed in utero. The majority of fetal tachycardias are supraventricular in origin, of which supraventricular tachycardia (SVT) associated with an AV accessory pathway is most common. Supraventricular tachyarrhythmias can be divided into those with a short ventricular–atrial (V–A) interval, those with a long V–A interval, those in whom the V and A are superimposed, and atrial re-entrant tachycardias.[1] While not defined in the majority of articles that review experience with fetal tachyarrhythmias, Fouron has demonstrated the importance of defining the mechanism in planning the most appropriate and effective management strategy.[1] Fetal SVT due to an accessory AV pathway is associated with ventricular rates typically of 230–280 bpm, and a shorter V–A interval relative to the A–V interval (so-called short V–A tachycardia) with 1:1 atrial-ventricular conduction (fig 2). At birth, 10% of affected fetuses have Wolff–Parkinson–White syndrome. So-called long V–A tachycardias with a shorter A–V interval relative to the V–A interval documented in utero include sinus tachycardia, ectopic atrial tachycardia (EAT) and permanent junctional reciprocating tachycardia (PJRT). As is true after birth, differences in timing and nature of onset and offset and the presence of beat-to-beat variability can assist in differentiating between these forms of long V–A SVTs. Junctional ectopic tachycardia is very rarely encountered prenatally and is suspected when the a wave is superimposed on the v wave. Finally, fetal atrial flutter is usually identified late in gestation. In the fetus atrial flutter is associated with atrial rates ranging from 300–550 bpm with variable A–V

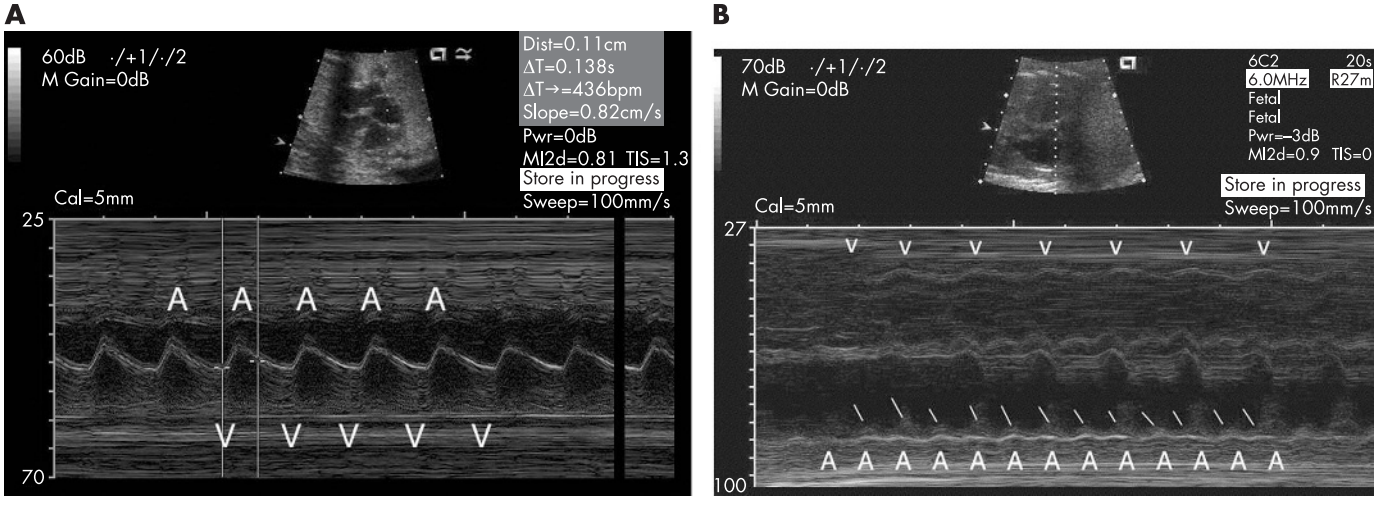

Figure 2 Examples of fetal supraventricular tachyarrhythmias as assessed by echocardiographic techniques. (A) This 25 week gestational age fetus had intermittent long ventriculo-atrial (V–A) supraventricular tachycardia at a rate of 230 bpm. The 1:1 atrioventricular (A–V) relationship is demonstrated by M mode (left), but the longer V–A relative to A–V relationship is more clearly defined by simultaneous superior vena cava–aorta (SVC–AO) Doppler (right). Postnatally a diagnosis of ectopic atrial tachycardia was confirmed. ''A'' denotes retrograde flow in the SVC during atrial systole and V denotes flow in the ascending aorta with ventricular systole. (B) This is an M mode tracing obtained in a 32 week fetus with incessant atrial flutter and significant cardiomegaly which was associated with an atrial (A) rate of 400 bpm and a ventricular (V) rate of 200 bpm.

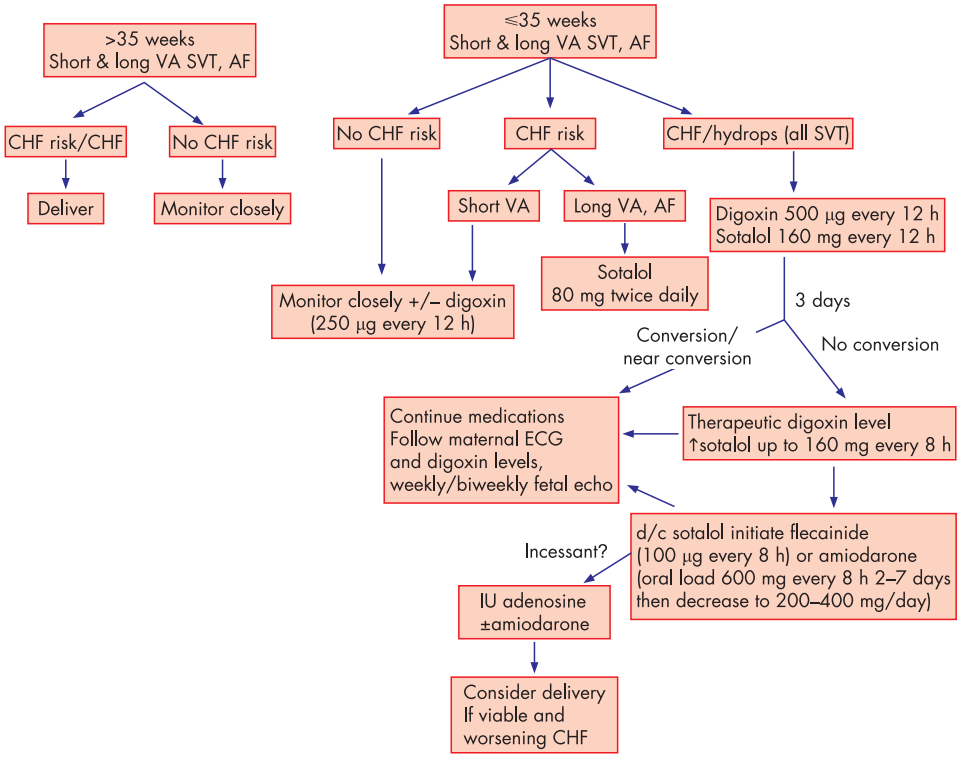

Figure 3 Potential management algorithm for fetal supraventricular tachycardias. AF, atrial flutter; CHF, congestive heart failure (hydrops); SVT, supraventricular tachycardia; VA, ventriculo-atrial.

conduction and thus ventricular rates. The very high atrial rates and slower ventricular rates are usually appreciated in the two-dimensional images but are typically confirmed through M mode tracings or systemic venous Doppler demonstration of the atrial wave rates.

Hydrops fetalis, a severe manifestation of fetal heart failure, is identified at presentation or evolves in 40–50% of fetuses with SVT. SVT results in reduced diastolic filling time which, combined with the normal limitations of relaxation and reduced compliance of the fetal myocardium, leads to increasing atrial and central venous pressures, manifested as increasing a wave (atrial systole) blood flow reversal in the systemic and pulmonary veins. The reversal of blood flow in atrial systole is worsened when the atrium contracts against a closed AV valve as occurs in atrial flutter with AV block. This ultimately leads to increased hydrostatic pressure, increased extravasation of plasma proteins into the interstitial space, and may eventually result in hepatic congestion with subsequent impairment in serum albumen production. Furthermore, elevated central venous pressure with increasing a wave reversal ultimately breeches the ductus venosus and impedes umbilical venous flow, resulting in the evolution of placental oedema and eventual placental dysfunction which leads to fetal hypoxaemia.

In order to reverse or even prevent the evolution of hydrops in fetal SVT, management strategies have evolved over the past two decades to treat the dysrhythmias when necessary. Treatment is largely reserved for fetuses with heart failure or those in whom the risk of developing heart failure is high. Fetuses at highest risk of developing heart failure are those with more incessant SVT, those with earlier onset of SVT (<32 weeks) and those with structural heart disease, the latter of which occurs in up to 10% of supraventricular tachyarrhythmias.[4] The actual ventricular rate and mechanism has not to date been clearly identified as a risk factor in the development of heart failure, although some, including long V–A SVTs, can be more resistant to treatment.

Most fetal SVTs can be treated successfully through maternal/transplacental administration of antiarrhythmia medications. This requires consideration of the changes that occur in pregnancy, including altered maternal gastric emptying and increased maternal renal clearance, which may necessitate the use of higher doses than typically used in the non-pregnant adult to achieve a therapeutic effect in the fetus. The distribution of the drug in the pregnant woman, the placenta and fetus, and the impact of fetal and placental hydrops on its distribution, have not been the subject of a definitive study. Finally, the influence of the medications on the placenta and the myocardial depressant effects of the medications must be considered in planning treatment, particularly in the fetus with severe ventricular dysfunction. As is true in paediatric SVT, no one medication effectively works for all fetal SVT, even SVT of the same mechanism. Many different antiarrhythmic medications have been reported in the treatment of fetal SVT including digoxin, propranolol, flecainide, sotalol, propafenone, verapamil and amiodarone. In the absence of hydrops, many choose to initiate digoxin treatment, particularly for short V–A SVT and atrial flutter. Digoxin alone has been associated with an 80–85% success rate in the treatment of fetal SVT and 60–65% in the treatment of atrial flutter in the absence of fetal heart failure.[5 6]

Successful treatment of fetal SVT and atrial flutter in the presence of hydrops has been shown to require a median of at least two medications (usually digoxin and another medication) and to take many more days to achieve success. This has led some groups to initiate stronger antiarrhythmic therapy at

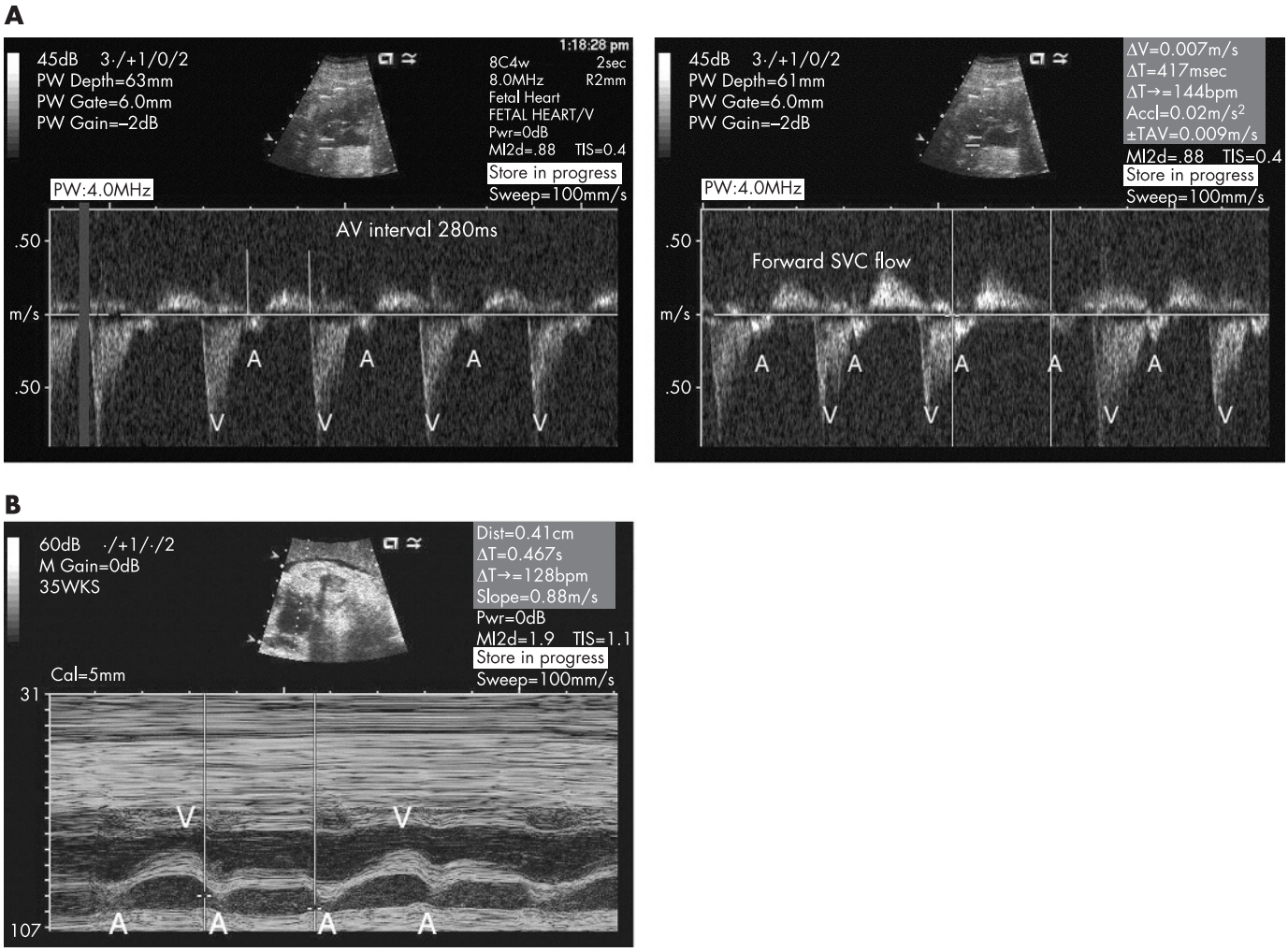

Figure 4 Examples of echocardiographic diagnosis of fetal atrioventricular (AV) block. (A) Simultaneous superior vena cava (SVC)–pulmonary artery (PA) Doppler spectra in a 32 week gestational age fetus with double-inlet ventricle associated with L-ventricular looping, transposition of the great arteries and pulmonary stenosis. A prolonged A–V interval of 280 ms (normal <130 ms) suggested the presence of first degree AV block which was present throughout the examination. Within 3 weeks there was progression to intermittent second degree (image on right), and by term there was complete AV block necessitating early pacemaker therapy. A: retrograde SVC flow in atrial systole; V: forward flow in the PA during ventricular systole. (B) This M mode tracing demonstrates the findings in a 30 week fetus with isolated AV block due to maternal autoantibodies. There is lack of AV synchrony with an atrial rate of 128 bpm and ventricular rate of 60 bpm. A: atrial systole; V, ventricular systole.

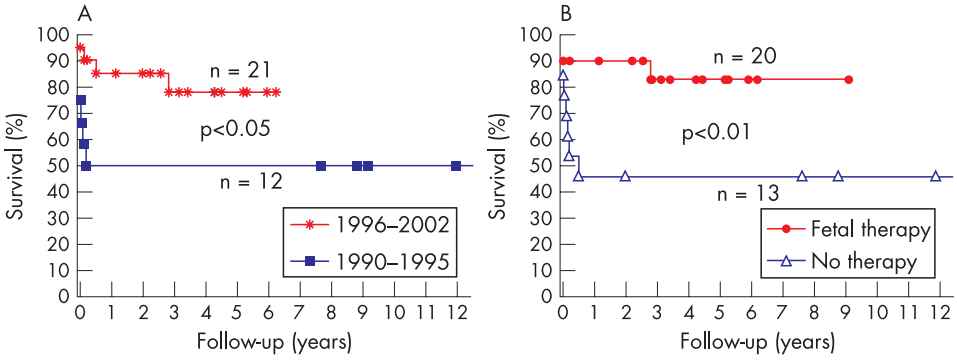

Figure 5 Kaplan–Meier curves demonstrating (A) era of diagnosis of fetal atrioventricular (AV) block and freedom from death, survival of fetuses/infants with a fetal diagnosis of isolated complete AV block associated with maternal autoantibodies, and (B) comparison of freedom from death with and without maternal/transplacental dexamethasone treatment. Improved survival was observed with a protocol guided approach which included the routine use of daily maternal steroid therapy. Reproduced with permission from Jaeggi *et al*, *Circulation* 2004;**110**:1542–8.

the onset of treatment in conjunction with digoxin to achieve success more rapidly and thus reduce the risk of mortality and even morbidity associated with haemodynamically significant fetal SVT.

Figure 3 provides a potential management algorithm that takes into consideration SVT mechanism and severity of fetal illness currently employed in the University of California fetal cardiovascular programme. With successful treatment, or even partial treatment, fetal hydrops can resolve resulting in a more viable infant at birth. In the absence of effective treatment, the mortality of the hydropic fetus with SVT approaches 50%. Even with successful treatment of the hydropic fetus, however, there is as much as a 10% risk of mortality and risk of morbidity which includes cerebral injury from thromboembolism, ischaemia and hypoxia. Rarely an incessant fetal SVT warrants an attempt at conversion with intraumbilical administration of antiarrhythmic medications including adenosine, digoxin and amiodarone. As long V–A SVT, particularly EAT and PJRT, have been shown to be more resistant to digoxin treatment, one might consider using a class III agent at the onset of therapy rather than after documentation of digoxin failure.[1] Finally, recent data in fetal pig models aimed towards providing metabolic support in fetal tachyarrhythmias suggest a potential role in the future for the maternal hyperglycaemic state in the acute resuscitation of the hydropic fetus with supraventricular tachycardia which, in providing metabolic support for the fetal myocardium, may ultimately be used coincident with initiation of antiarrhythmic treatment.

While as paediatric cardiologists we tend to focus on the health of the fetus in the treatment of fetal SVT, the mother's health is clearly of utmost importance. A cardiovascular assessment, preferably by an adult cardiologist, and cautious monitoring at the onset of and with changes in the medications is critical. In addition to serial ECGs and telemetry, monitoring drug concentrations where possible is helpful to determine whether therapeutic or toxic values have been achieved.

As is true for ventricular ectopic beats, ventricular tachycardia is very rare in the fetus. It may be associated with myocardial disease or observed in the presence of tumours. Rarely incessant ventricular tachycardia warrants maternal antiarrhythmic medication, including the use of β-blockade, lidocaine and amiodarone.

BRADYARRHYTHMIAS

Fetal bradycardia is usually diagnosed when the ventricular rate is <110 bpm. The most common cause of fetal bradycardia is sinus bradycardia, which can occur with vagal stimulation as is believed to occur with compression during ultrasound examinations, with fetal distress or with more serious systemic disease. Fetal bradycardia with brisk return to a normal heart rate is often observed during ultrasound examinations and suggests the fetus has reasonable reserve. Rarely sinus bradycardia may be a sign of long QT. Intermittent 2:1 AV block with no alterations in A–V conduction may assist in making this diagnosis.

Fetal AV block is another cause of fetal bradycardia. Usually the presenting form is that of complete AV block but occasionally second degree and, more recently—with documentation of A–V intervals and use of magnetocardiography—first degree AV block are diagnosed (fig 4). Fetal AV block is associated with maternal autoantibodies in approximately 45–48% of cases, structural heart disease in 45–48% of cases, and is isolated and of unclear aetiology in 4–10% of cases.[7] Isolated AV block with or without myocardial disease is usually associated with the transplacental passage of maternal autoantibodies, anti-SSA/Ro and/or anti-SSB/La. Deposition of the antibodies along the conduction system and myocardium results in a cascade of events with an inflammatory component ultimately evolving into fibrosis of involved tissue, including the AV node and endocardium. Although these antibodies are found in women with clinical autoimmune disease, including Sjogren syndrome and systemic lupus erythematosus, 70–80% of the mothers of affected fetuses have no clinical autoimmune disease at the time of AV block diagnosis.[8]

Fetal rhythm abnormalities: key points

- Evaluation of fetal arrhythmias is largely based on the assessment of the chronological relationship between atrial and ventricular contractions
- The majority of referrals for fetal arrhythmias represent benign atrial premature beats
- Accurate definition of the atrioventricular (AV) relationship permits delineation of the type of tachyarrhythmia and bradyarrhythmia and may assist in more appropriate management of affected pregnancies
- Most forms of fetal supraventricular tachycardia (SVT), even in the presence of hydrops, are treatable before birth through maternally administered medications
- Maternal autoantibody mediated fetal AV block and cardiomyopathy evolves as a consequence of the transplacental passage of maternal antibodies, the influence of which may be ameliorated through the use of maternally administered corticosteroids.

Maternal autoantibody mediated AV block is a prenatally acquired condition which likely requires a predisposing factor in the fetus such as variable antigen expression or a more rigorous fetal immune system, or external factors such as infection. Between 15–20% of fetuses with autoimmune mediated AV block have additional more diffuse myocardial disease which can lead to endocardial fibrosis and ventricular dysfunction.[8] The evolution of myocardial disease and ventricular rates of <50 bpm have been identified as important mortality risk factors in this disease. Given the initial role of inflammation, maternally administered corticosteroids, particularly fluorinated glucocorticoids such as dexamethasone, have been used in the treatment of this condition. Rare reports document the reversal of the severity of heart block. More recently it has been suggested that maternal dexamethasone treatment (4–5 mg/day) may reduce the incidence of more severe myocardial disease.[9]

Maternal and neonatal plasmaphoresis has had variable efficacy in this disease. Fetal ventricular pacing has been attempted largely in very compromised fetuses without success, likely as a consequence of the lack of A–V synchrony in the presence of severe myocardial dysfunction. Finally, we have suggested a potential role for intravenous γ-globulin, either administered to the mother or directly administered through cordocentesis to the fetus (thus reducing the mother's exposure) in the treatment of the fetus and neonate with reduced myocardial function suggesting more diffuse disease.

β-sympathomimetic treatment administered to the mother is also used, particularly in pregnancies where there is a low fetal ventricular rate (<55 bpm) or evidence of myocardial disease to support the function and improve the ventricular rate.

With close surveillance, use of maternally administered corticosteroids, delivery at 35–37 weeks or earlier with any evidence of fetal distress or cardiovascular compromise, aggressive neonatal cardiac intensive care management and early permanent pacemaker therapy, the mortality rate for prenatally diagnosed autoimmune mediated AV block has significantly decreased. The experience at the University of Toronto affiliated centres showed a decreased in the mortality from 45% in the years 1992–96 to 15% in 1996–2002[9] (fig 5). By adulthood, though, the majority of affected fetuses will require pacemaker therapy.[8] Need for pacemaker therapy and reintervention has been shown to be significantly greater than that for infants and children diagnosed after birth with AV block.

Fetal AV block may also be diagnosed in the presence of structural heart disease including heterotaxy syndrome, most commonly left atrial isomerism and lesions associated with L-ventricular looping. Despite improvements in the perinatal management of affected pregnancies, including use of β-sympathomimetic treatment, the outcome of AV block associated with structural heart disease is poor with a high incidence of fetal hydrops and low overall survival.[7 10]

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