# Fetal Arrhythmia

18

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**Abstract**

Fetal arrhythmias present as an irregular cardiac rhythm and abnormal heart rate. Despite the theoretical advantage of fetoplacental circulation, rapid progression to hydrops is found in fetuses with tachyarrhythmia or bradyarrhythmia due to the limited heart rate reserve. Accurate diagnosis is essential for appropriate management of fetal arrhythmias, but this can be challenging since fetal electrocardiography is unavailable. Echocardiography plays a pivotal role in diagnosis and management of fetal arrhythmias. Most cases of fetal arrhythmia have a structurally normal heart with isolated premature contractions that often spontaneously resolve without medical treatment. There is a clear clinical consensus that maternal transplacental antiarrhythmic therapy for fetal tachyarrhythmia is effective. Complete atrioventricular block is irreversible. Dexamethasone and intravenous immunoglobulin have been used to prevent myocardial inflammation, but recent studies have not shown efficacy of these drugs for fetal bradyarrhythmias. Long QT syndrome manifests in several heart rate patterns and is associated with cardiac arrest and sudden death. Maternal intravenous magnesium is effective for ventricular tachycardia or torsades de pointes. This chapter reviews the different types of fetal arrhythmias and gives an overview of the current diagnostic techniques and treatment strategies.

**Keywords**

Bradycardia · Fetal arrhythmia · Fetal treatment · Prenatal diagnosis · Tachycardia

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## 18.1 Clinical Features

Arrhythmias present as an irregular cardiac rhythm, a slow or fast heart rate, or a combination of abnormal rhythm and rate. Fetal arrhythmias are observed in 1–2% of pregnancies at mid- to third trimester. Most cases evaluated for an irregular rhythm have isolated premature contractions with a structurally normal heart. Premature contractions usually spontaneously resolve without medical treatment [1]. In contrast, fetal tachyarrhythmias or bradyarrhythmias are rare but life-threatening, and medical treatment is usually necessary in utero and after birth. Most cases of congenital heart disease, even complex heart defect, can adapt in the fetal circulation. Despite the theoretical advantage of fetoplacental circulation, the circulatory reserve of an unborn child is negatively influenced by a variety of environmental and intrinsic factors, including the limited heart rate reserve. Because of the limited pump reserve of immature hearts, any significant change in heart rate leads to a decline in cardiac output, impaired cardiac filling, and venous congestion, the severity of which depends on arrhythmia characteristics and myocardial properties. Persistent tachyarrhythmias and bradyarrhythmias are among the more common cardiac causes of fetal hydrops, prematurity, and perinatal death [2, 3]. Hemodynamic evaluation using Doppler echocardiography permits elucidation of the electrophysiological mechanism and improves the accuracy of diagnosis. This method can also be used to understand fetal cardiac pathophysiology and to assess fetal conditions and monitor the effect of antiarrhythmic treatment. Abnormal venous Doppler sonography findings are common and severe in fetuses with tachy- or bradyarrhythmias and indicate elevation of central venous pressure. An increase in wall stress will result in cardiac remodeling and hypertrophy, which increases myocardial oxygen consumption and aggravates myocardial dysfunction. To overcome the reduction in ventricular compliance, end-diastolic filling pressure and hydrostatic central venous pressure increase to improve cardiac output [4]. As a result, rapid progression to hydrops occurs in fetuses with tachy- or bradyarrhythmias.

## 18.2 Diagnosis

### 18.2.1 Fetal Echocardiography

Accurate diagnosis is essential for management of fetal arrhythmias, and electrocardiography is the main diagnostic tool for arrhythmias in neonates, children, and adults. Noninvasive fetal electrocardiography is available at a few institutions but is too time-consuming to use in general clinical settings due to sensitivity and difficulty with analysis. Fetal echocardiography is usually used to assess fetal heart rate and rhythm, using 2D, M-mode, or pulsed Doppler imaging [5]. M-mode imaging is useful for simultaneous recording of atrial and ventricular systolic wall motions [6]. The relationship between atrial and ventricular contractions can be

shown, and heart rate can be measured. Simultaneous pulse wave Doppler evaluation of the superior vena cava and ascending aorta (SVC/aAo Doppler) is used to examine the sequence and time relationship of blood flow events secondary to atrial and ventricular contractions [7]. The beginning of the retrograde SVC wave reflects onset of atrial systole (A-wave), whereas onset of aortic forward flow marks the beginning of ventricular systole (V-wave). Diagnosis of fetal arrhythmias is made using the relationship between the A-wave and V-wave (Fig. 18.1). Each atrial event is followed by a ventricular event within a normal atrioventricular (AV) time interval, which confirms normal 1:1 AV conduction [8]. Fetal echocardiography is not an electrical assessment but a mechanical assessment. Hence, repolarization abnormalities such as long QT syndrome cannot be confirmed solely by echocardiography.

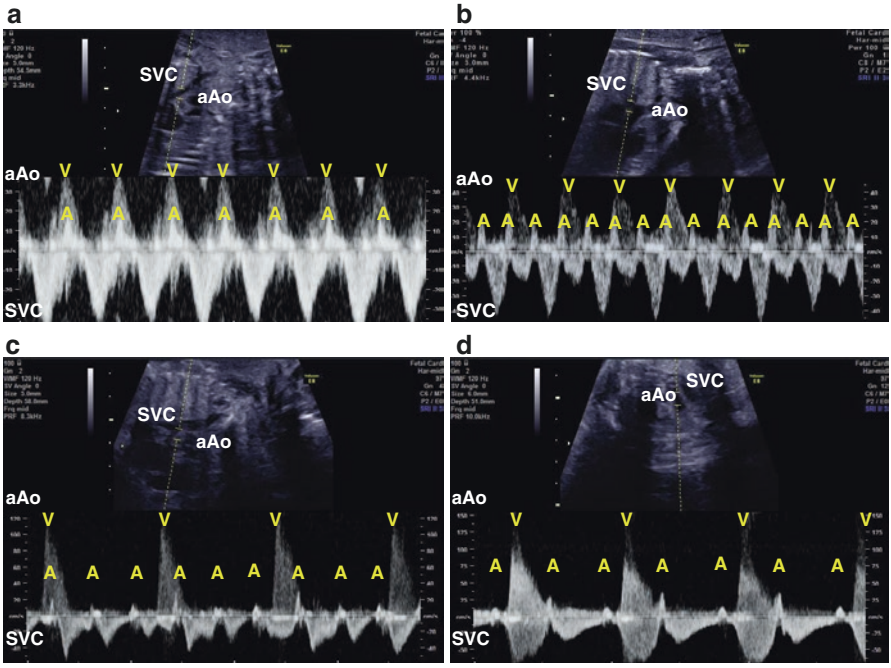


**Fig. 18.1** Two-dimensional imaging showing a segment of the ascending aorta (aAo) adjacent to the superior vena cava (SVC) draining into the right atrium. Pulsed Doppler echocardiography shows aortic ejection waves (V-waves) recorded above the zero velocity line. Anterograde venous flow in the SVC in the opposite direction is shown below the line, and small venous retrograde waves (A-waves) caused by right atrial contraction are above the same line. (**a**) Fetal tachyarrhythmia with a short ventriculoatrial pattern, suggesting atrioventricular reentrant tachycardia. (**b**) Fetal tachyarrhythmias with 2:1 atrioventricular conduction, suggesting atrial flutter. (**c**) Fetal bradyarrhythmia with a regular normal atrial rhythm and rate but with the ventricles beating independently at a much slower rate, suggesting complete atrioventricular block. (**d**) Fetal bradyarrhythmia with 2:1 atrioventricular conduction, suggesting congenital second-degree atrioventricular block or functional atrioventricular block due to long QT syndrome

### 18.2.2 Cardiotocography

Cardiotocography traces fetal heart rate based on pulsed Doppler technology and is usually used for evaluating antepartum and intrapartum fetal well-being [9]. Similar to Holter electrocardiogram, cardiotocography can display uninterrupted segments of recorded time during normal rhythm or arrhythmias. In particular, cardiotocography plays an important role in management of fetal tachyarrhythmia, since it clearly shows the fetal heart rate baseline and frequency of fetal tachyarrhythmias, making it easy to evaluate the effects of fetal treatment. In addition, the clinical features of fetal ectopic atrial tachycardia (EAT) can be shown by cardiotocography. Fetal EAT has slow baseline changes that are referred to as a "warm-up and cool-down" phenomenon [10]. Onset and termination are sudden in most cases of reentrant supraventricular tachycardia (SVT) and atrial flutter (AFL) (Fig. 18.2a, b), whereas in EAT the shift of the pacemaker from the sinus node to the ectopic focus is more gradual. Thus, EAT shows a gradual increase after onset of tachycardia and a gradual decrease before termination of tachycardia (Fig. 18.2c). Another feature of fetal EAT on cardiotocography is short-term variability with acceleration during tachyarrhythmia [11].

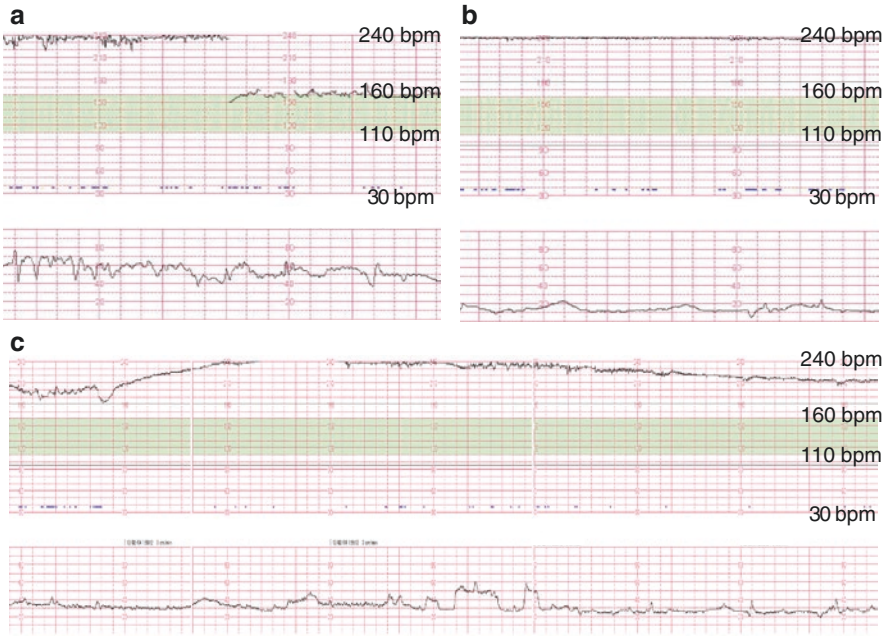


**Fig. 18.2** Fetal cardiotocography showing (**a**) atrioventricular reentrant tachycardia with sudden termination of sinus rhythm, (**b**) atrial flutter with a flat baseline, (**c**) the fetal baseline accelerating gradually from 200 to over 240 bpm and decelerating gradually from 240 to 200 bpm, indicating a "warm-up and cool-down" phenomenon of ectopic atrial tachycardia

### 18.2.3 Fetal Magnetocardiography

Fetal magnetocardiography (fMCG) is used to assess electromagnetic characteristics of fetal cardiac conduction. Measurement must be performed in a shielded room that excludes magnetic interference from environmental sources [12]. Because of the requirement for specialized equipment and expertise, fMCG is currently performed in only a few institutions worldwide. fMCG captures the P-wave, PR interval, QRS interval, ST-T waves, QT interval, and RR interval in most fetuses at >24 weeks of gestation (Fig. 18.3a) [13, 14] and may be especially useful for analyzing complex rhythm and rate patterns such as irregular, multiple, or transient arrhythmias and for providing a more accurate differential diagnosis of tachy- and bradyarrhythmia. No other current method can detect repolarization abnormalities such as T-wave alternans [15]. Although fMCG currently has limited availability, use of this technique is reasonable for assessment of cardiac conduction and rhythm in fetuses with a known or suspected disease of the conduction system.

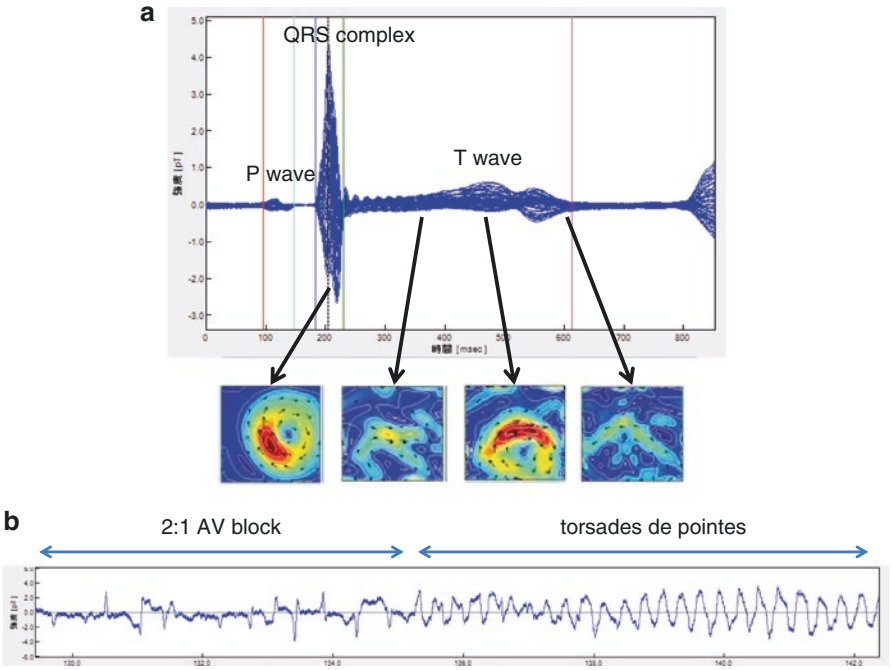


**Fig. 18.3** Fetal magnetocardiography showing (**a**) accurate P-wave, PR interval, QRS interval, ST-T waves, QT interval, and RR interval evaluated using the signal-averaged method, and (**b**) consecutive recording of 2:1 atrioventricular (AV) block and torsades de pointes due to long QT syndrome

## 18.3 Management

### 18.3.1 Premature Contractions

Most fetuses evaluated for an irregular rhythm have a structurally normal heart with isolated premature contractions that disappear during pregnancy or shortly after birth. Premature atrial contraction (PAC) accounts for most patients with an irregular heartbeat at any age. PAC manifests as premature P-waves with abnormal P-wave axes and with an AV conduction that may be normal, aberrant, or blocked. In echocardiography, PAC is detected as a shorter than normal atrial interval. If AV conduction is normal, the premature atrial event is followed by a timely related premature ventricular event. If the PAC is premature enough to prevent conduction across the refractory AV node, no ventricular event is observed, which manifests as a skipped heartbeat. Before birth, PACs have been associated with a less than 1% risk of fetal tachyarrhythmias, although a higher risk has been suggested for atrial bigeminy and couplets [16]. PACs usually spontaneously resolve without medical treatment. Premature ventricular contraction (PVC) is uncommon in utero. PVC manifests in a premature QRS complex that is not preceded by a P-wave. In echocardiography, PVC is not preceded by an atrial beat, whereas atrial intervals are usually normal and regular. Isolated PVCs are typically benign and self-limited and require no treatment. PVCs secondary to ventricular aneurysm or cardiomyopathy should be noted. The American Heart Association statement recommends fetal echocardiography to assess cardiac structure and function and to determine the mechanism of arrhythmia if the fetus presents with frequent ectopic beats [17]. Fetal heart rate should be monitored weekly until PACs or PVCs have resolved.

### 18.3.2 Fetal Tachyarrhythmias

Detection of a fast heart rate >180 bpm in a fetus constitutes a medical emergency because it carries a significant risk of fetal heart failure, hydrops, and death. Possible mechanisms include SVT, AFL, and VT. SVT and AFL account for 90% of fetal tachyarrhythmias, and both are readily distinguished by echocardiography. Secondary sinus tachycardia due to hyperthyroidism should be excluded. A variety of fetal and maternal conditions may cause sustained sinus tachycardia, including distress, anemia, and infection. The underlying cause of tachycardia should be treated.

#### 18.3.2.1 Atrioventricular Reentrant Tachycardia

Atrioventricular reentrant tachycardia (AVRT), the most common mechanism of fetal SVT, involves a reentrant circuit that uses the AV node to conduct from the atria to the ventricles and a fast-conducting accessory pathway to propagate the ventricular impulse back to the atria [18]. Heart rate in AVRT usually ranges from 220 to 300 bpm. AVRT starts suddenly with a PAC and terminates with AV block. Most hearts are structurally normal, but Ebstein's anomaly is a well-known

association with accessory pathways. Fetal echocardiography shows a short ventriculoatrial (VA) pattern because atrial contraction occurs soon after ventricular contraction (Fig. 18.1a). Because of the near simultaneous atrial and ventricular contractions, the AV valves are closed during atrial systole, and there is pronounced A-wave flow reversal in the precordial veins and ductus venosus. fMCG can detect a delta wave in utero if there is anterograde pathway conduction during sinus rhythm (Wolff-Parkinson-White syndrome). In retrospective studies, 40% of fetuses with AVRT presented with hydrops, and this was associated with perinatal mortality of 21–27%. In contrast, the rate of perinatal mortality was <5% for cases without hydrops. After delivery, medical treatment must be reassessed relative to the antiarrhythmic drug used in utero, the duration since the last recurrence, and the mechanism of clinical tachycardia. Prophylactic antiarrhythmic treatment is often used to prevent AVRT recurrence during the first 6 months or longer because approximately 50% of AVRT cases have recurrence in the neonatal period [2, 19].

#### 18.3.2.2 Ectopic Atrial Tachycardia

EAT accounts for 10–15% of cases of fetal SVT. EAT is caused by automaticity and arises from an ectopic focus within the atria. Cardiotocography is useful for detection of unique heart rate changes with a "warm-up and cool-down" phenomenon [10]. Although EAT is usually 1:1, conduction delay with AV block may be seen. EAT is more refractory to pharmacological treatment than AVRT, resulting in congestive cardiomyopathy [20]. Short-term antiarrhythmic treatment is often required, but EAT usually resolves before 6 months of life.

#### 18.3.2.3 Atrial Flutter

AFL accounts for 30% of fetal tachyarrhythmias and is often associated with accessory AV pathways and reentrant SVT [2]. AFL is sustained by a macro-reentrant circuit that is confined to the AV ring. The atrial rate of AFL usually ranges from 400 to 540 bpm, which is commonly associated with 2:1 AV conduction and a ventricular rate of 200–270 bpm (Fig. 18.2b). A normal ventricular rate is occasionally observed in AFL with slower 3:1 or 4:1 AV conduction. In the absence of structural heart disease, AFL is almost exclusively observed in fetuses during the third trimester or at birth. If AFL persists to birth, sinus rhythm can be restored by synchronized electrical cardioversion. Neonatal recurrence of AFL is uncommon and long-term treatment is rarely required [18].

#### 18.3.2.4 Fetal Treatment for SVT and AFL

A randomized control study has not been performed, but there is a clear clinical consensus on the efficacy of maternal transplacental antiarrhythmic therapy for fetal tachyarrhythmias [17]. Rapid pharmacologic cardioversion to a normal sinus rhythm is most pressing for hydropic fetuses with incessant tachyarrhythmia. Possible medications to treat fetal SVT and AFL until birth include maternal digoxin, sotalol, or flecainide alone or in combination. Oral administration of antiarrhythmic agents is recommended. Direct treatment of the fetus by intramuscular

injection may have a role in more rapidly restoring sinus rhythm in the hydropic fetus, but experience with this route is quite limited. The goal of fetal treatment is establishment of sufficient sinus rhythm or heart rate to allow resolution of hydrops or ventricular dysfunction. Management of fetal tachyarrhythmias depends on gestational age, the presence and degree of fetal compromise, hydrops or other risk factors, and potential fetal and maternal risks of fetal therapy and early delivery.

A flowchart for decisions on treatment of fetal tachyarrhythmias at the National Cerebral and Cardiovascular Center is shown in Fig. 18.4. Pharmacological treatment is recommended for all but the near-term fetus with sustained SVT or AFL (occurring more than 50% of the time) or with fetal hydrops or cardiac dysfunction. In contrast, treatment of intermittent tachyarrhythmia (less than 50% of the time) is likely to include close observation because heart failure rarely develops [21]. The treatment protocol depends on fetal and maternal risk analysis, with little data to support a specific treatment protocol that is likely to be most effective and to carry the lowest risk. In most cases at our center, digoxin is used as first-line therapy, a combination of digoxin and sotalol as second-line therapy, and a combination of digoxin and flecainide as third-line therapy. Fetal treatment is continued until delivery, even when cardioversion has been achieved, because approximately 15% of fetal tachyarrhythmias recur in utero [18].

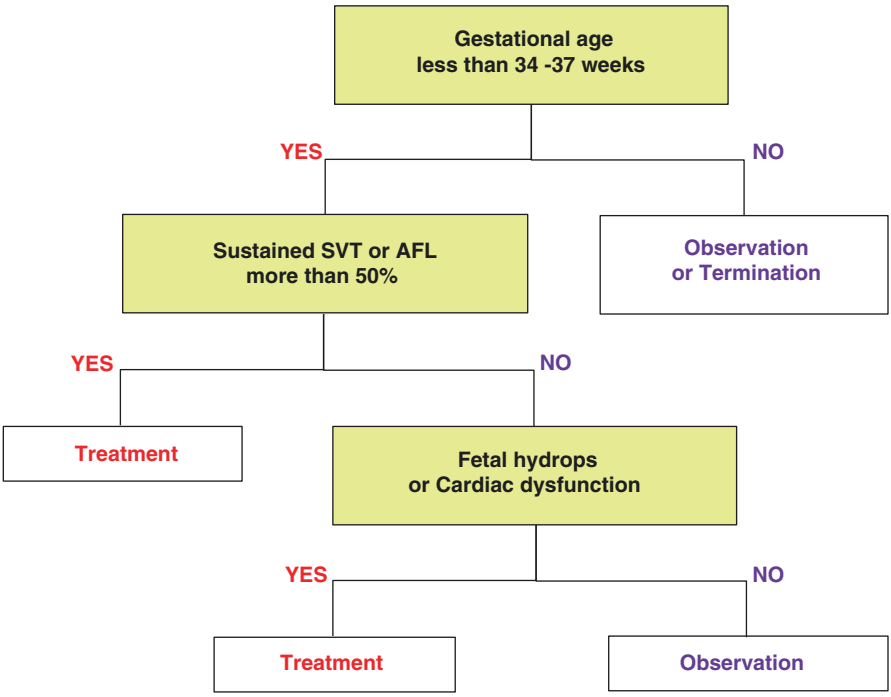


**Fig. 18.4** Flowchart for decisions on treatment for fetal tachyarrhythmias at the National Cerebral and Cardiovascular Center. *SVT* supraventricular tachycardia, *AFL* atrial flutter

Fetal treatment as first- and second-line antiarrhythmic therapy is still controversial [22]. For reentrant SVT, maternal digoxin administered orally or intravenously is used as first-line therapy in many centers because of its relatively safe profile. In some centers, sotalol or flecainide is used as primary therapy [23–26]. For AFL, sotalol is recommended because it is effective in converting 50–80% of cases [24]. These agents are all reasonable as first-line choices, but there is no study indicating which the best initial therapy is. Sotalol, flecainide, and amiodarone have often been used as second-line therapy. However, amiodarone has a more significant toxicity profile for the mother and fetus and should be reserved as third-line treatment for life-threatening tachyarrhythmias [27, 28]. The duration of therapy with amiodarone should be minimized with discontinuation after hydrops resolve. There are no randomized, multicenter, clinical trials of use of antiarrhythmic agents in fetal tachyarrhythmias. Therefore, treatment protocols remain center specific. In Japan, a multicenter clinical trial using antiarrhythmic agents (digoxin, sotalol and flecainide) in fetal SVT and AFL has been done (UMIN000004270, http://en.fetusjapan.jp/).

#### 18.3.2.5 Adverse Effects of Antiarrhythmic Medications

Relatively high doses of antiarrhythmic drugs are required during the second and third trimesters, since maternal circulating blood volume and renal clearance are both increased. There is limited information on maternal-fetal transfer of antiarrhythmic agents in humans. With the exception of sotalol and flecainide, most drugs have diminished transplacental transfer with fetal hydrops [29]. Nausea, fatigue, and loss of appetite are well-known adverse effects of digoxin, and sinus bradycardia or AV block are common effects or adverse effects with antiarrhythmic drugs. Use of combination therapies presents a greater risk of maternal and fetal complications than monotherapy. However, serious maternal adverse effects are rare in most reported series and generally resolve with discontinuation of therapy. For preventing proarrhythmia in the mother and fetus, it is important to keep the maternal serum potassium level at no less than 4.0 mEq/L. A maternal electrocardiogram is essential before treatment with QT-prolonging drugs such as sotalol, flecainide, and amiodarone, and close monitoring for maternal QTc interval is important. Frequent monitoring of drug levels and the maternal electrocardiogram are recommended to assess drug effects and toxicity, especially in dose escalation [17].

#### 18.3.2.6 Ventricular Tachycardia

VT is a rare arrhythmia in fetuses. Fetal echocardiography shows tachycardia <200 bpm that is often incessant on presentation. VT often shows a short VA pattern similar to AVRT, which is most common in fetal arrhythmias. To distinguish VT from AVRT, close evaluation is important. The characteristic findings of VT are a ventricular rate higher than the atrial rate and dissociation between ventricular and atrial contractions. If fMCG shows a wide QRS in tachycardia, diagnosis of VT is more likely. In evaluation of fetal VT, possible causes include viral and anti-Ro antibody-mediated myocarditis, cardiac tumors, structural heart disease, and hereditary cardiomyopathy including long QT syndrome. Treatment and prognosis

depend on the VT mechanism and pattern, the hemodynamic impact, and associated conditions. Maternal intravenous magnesium is recommended as first-line treatment for fetal VT [30–32]. In addition, combination therapies of oral propranolol and mexiletine may be considered, even though there are no data showing which agent is most effective. In a case with suspected or confirmed long QT syndrome, drugs with QT-prolonging potential such as sotalol, flecainide, and amiodarone are contraindicated.

### 18.3.3 Fetal Bradyarrhythmias

Fetal bradyarrhythmias are defined by a heart rate <100 bpm. Occasional, brief sinus bradycardia is a benign physiologic response in which the rate of the sinus node is slower than normal for age. Prolonged or persistent bradycardia is of more concern and should trigger a more detailed assessment of the cause. The main mechanisms of fetal bradyarrhythmias include sinus bradycardia, complete AV block, and functional AV block due to nonconducted atrial bigeminy or long QT syndrome.

#### 18.3.3.1 Sinus Bradycardia

Sinus bradycardia is defined as a rhythm that originates from the sinus node but in which the rate is slow for age. A subsidiary pacemaker may become the dominant pacemaker if the rate of the sinus node decreases to less than that of the secondary pacemaker. In echocardiography, fetal sinus or atrial bradycardia resembles that of a normal rhythm, with the only difference being that the atrial and ventricular rates are slow for gestational age, usually in the range of 80–110 bpm. Sinus bradycardia is well tolerated but may be secondary to fetal distress, sinus node dysfunction, and long QT syndrome. Perinatal management of sinus bradycardia depends on the underlying cause and may include no treatment, anti-inflammatory medication for myocarditis, and postnatal therapy.

#### 18.3.3.2 Complete Atrioventricular Block

Complete AV block is defined as a complete failure of normal propagation of atrial impulses to the ventricles and is the most common congenital conduction abnormality before birth. Typical fetal echocardiography shows a regular normal atrial rhythm and rate, whereas the ventricles beat independently at a much slower rate of 40–80 bpm (Fig. 18.1c). In about half of fetal cases with congenital AV block, it is associated with major congenital heart defects (CHDs) such as left atrial isomerism and corrected transposition of the great arteries [33]. The prognosis of fetal bradyarrhythmia with CHD is still poor, with a nationwide survey in Japan showing neonatal and overall survival rates of 66% and 48%, respectively [3]. In fetal bradyarrhythmias associated with CHDs, a ventricular rate <55 bpm has significant effects on fetal myocardial dysfunction and fetal hydrops, resulting in a poor prognosis. However, all cases of corrected transposition of the great arteries or

ventricular rate ≥70 bpm survived. In the absence of structural heart disease, congenital AV block is strongly linked to the fetal transplacental passage of anti-Ro antibodies, which are prevalent in about 1–2% of pregnant women [34]. In 1–5% of exposed fetuses, the maternal antibodies lead to complications, including congenital AV block, sinus bradycardia, myocarditis, endocardial fibroelastosis, and dilated cardiomyopathy. Although fetuses with isolated congenital AV block often tolerate in utero circulation, the severe end of the disease spectrum includes low cardiac output, fetal hydrops, and death. Risk factors associated with perinatal death include gestational age, fetal hydrops, endocardial fibroelastosis, myocarditis, and ventricular rate <50–55 bpm [35, 36]. A nationwide survey in Japan showed that fetal hydrops was associated with a 14-fold increased risk of perinatal death, and myocardial dysfunction was a significant risk factor for a poor prognosis [37]. Interestingly, fetal heart rate and the presence of maternal antibodies were not associated with morbidity and mortality.

#### 18.3.3.3 Fetal Treatment for Fetal Bradyarrhythmias

There is currently no consensus on the indications of fetal treatment for isolated congenital AV block, and there is no treatment available to reverse complete AV block [38]. Dexamethasone, intravenous immunoglobulin, beta-sympathomimetics, and postnatal pacing have been used to prevent or treat severe immune-mediated myocardial inflammation, to augment cardiac output, and to improve the prognosis [39, 40]. A flowchart of the decision protocol for fetal bradyarrhythmias in fetuses without CHDs at the National Cerebral and Cardiovascular Center is shown in Fig. 18.5. Treatment of congenital AV block depends on the origin, presence, and degree of heart failure and the ventricular rate. When signs of fetal hydrops,

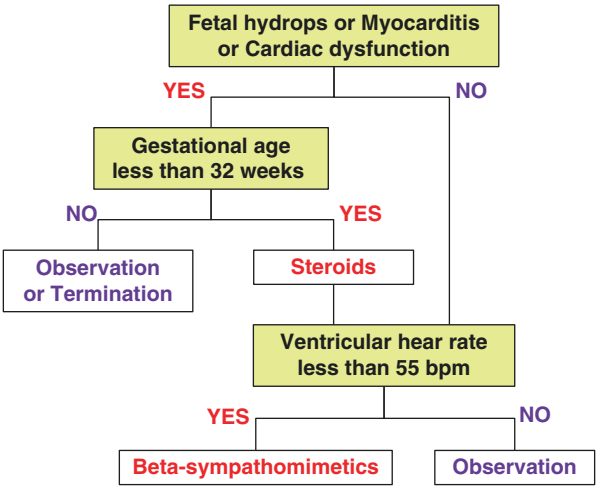


**Fig. 18.5** Flowchart of the decision protocol for fetal bradyarrhythmias in fetuses without CHDs at the National Cerebral and Cardiovascular Center

myocarditis, pericarditis, or cardiac dysfunction are observed, maternal dexamethasone (4 mg/day for 2 weeks; 2 mg/day to 30 weeks of gestation; 1 mg/day to 31 weeks of gestation; 0.5 mg/day to 32 weeks of gestation) is used from the time of diagnosis to 32 weeks of gestation. When fetal hydrops or severe cardiac dysfunction develops after 34 weeks of gestation, neonatal treatment should be considered. Possible treatment-related adverse events that may preclude routine use of high-dose steroids include fetal growth restriction, oligohydramnios, and maternal diabetes mellitus and central nervous system side effects [39]. Chronic prenatal steroid therapy for congenital AV block has no obvious impact on neurocognitive function at school age [41]. However, it is recommended that steroid use is limited to <10 weeks to avoid maternal and fetal adverse effects, especially fetal growth restriction and oligohydramnios [37]. We also use transplacental ritodrine hydrochloride infusion (usually 30–200 μg/min) to maintain fetal ventricular heart rate at >55 bpm. However, recent studies have not shown efficacy of this treatment for fetuses with or without CHDs [10, 35–37]. Several reports have suggested addition of maternal intravenous immunoglobulin (1 g/kg every 2–3 weeks) if endocardial fibroelastosis and ventricular dysfunction are detected [40]. The optimal timing for administration and repeated dosing intervals is unknown. Prospective, randomized trials or a registry is needed to establish definitive treatment recommendations for a fetus with congenital AV block.

#### 18.3.3.4 Long QT Syndrome

Long QT syndrome is an inherited ion channel disorder that manifests as several patterns of fetal arrhythmias [42, 43]. Sinus bradycardia is most common, usually in the range of 80–110 bpm. An irregular rhythm can also be caused by functional second-degree AV block (Figs. 18.1d and 18.3b), which is characterized by failure of AV conduction of atrial activity to the ventricle. Unlike in atrial bigeminy, the atrial rhythm in 2:1 AV block is fairly constant and 1:1 AV conduction recurs at slower atrial rates [16]. Long QT syndrome has the potential to cause VT or torsades de pointes (Fig. 18.3b), which is associated with fetal heart failure, hydrops, and sudden death, as in adults. Maternal intravenous magnesium is effective and recommended as first-line treatment for fetal VT or torsades de pointes due to long QT syndrome. Because of the predisposition of patients with long QT syndrome for VT-related cardiac arrests and sudden death, postnatal treatment with a long-acting beta-blocker with or without a pacemaker or implantable cardioverter-defibrillator is often required.

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