Fetal Arrythmia
Introduction

Arrhythmias

- Can occur as soon as the heart starts to beat
- End on a final irreversible arrhythmia when we die

They are noted

- in about 2% of the pregnancies
- account for 10 to 20% of the referrals to fetal cardiology

The first heartbeat occurs by 3 weeks post-conception when the heart is only a primitive tubular structure. Major morphological remodeling occurs simultaneously with the development of the cardiac conduction system resulting by 7 weeks of gestation in a 4CH with synchronous contraction of the atrial and ventricular chambers at a rate of approximately 110 bpm.

Progressively, the SN acts as the primary pacemaker and the heart rate reaches 170 bpm by 9 to 10 weeks. At 11–14 weeks, it averages 150–170 bpm. Later on in gestation, heart rate slowly decreases. Between 20 and 40 weeks of gestation, the heart rate is regular, with a range from 110 to 180 bpm and a maximal beat-to-beat variation of 15 bpm.

Normal impulse formation and conduction

- The impulse propagates from the **sinoatrial node** along atrial muscle fibers toward the **atrioventricular junction**
  - stimulates the atrial myocardium to contract.

- **Within the AV node,**
  - The electrical impulse is **physiologically delayed**
  - Functions as a **filter** against the propagation of abnormally fast atrial rates or very premature atrial beats to the ventricles.

- **After crossing the AV node,**
  - The **bundle of His**, the **right bundle branch**, the **left bundle branch**
  - **Purkinje fibers** to the endocardial surfaces of both ventricles.

- The electrical depolarization spreads quickly from one ventricular myofiber to the next
  - so that both ventricles function as synchronous contractile units.
Abnormal impulse generation and conduction

- Cardiac cells in the specialized fibers of
  - Atria,
  - AV junction
  - His – Purkinje system

- May manifest **automaticity** outside the SA node.
  - They are called **latent pacemakers** as they are physiologically suppressed by the more rapid rate of the SA node
  - Consequently, these ectopic pacemakers **do not normally initiate the heartbeat**.
Abnormal impulse generation

There are two mechanisms of spontaneous impulse initiation that may lead to arrhythmias,

- **Automaticity** - *Automatic arrhythmias of the sinus node*
  - occur when the SA node fires at an abnormally
    - fast (sinus tachycardia),
    - slow rate (sinus bradycardia),
  - but it is still the dominant pacemaker
  - Persistent fetal sinus arrhythmia is usually associated with a precipitating factor
    - stress
    - maternal antibody mediated fetal thyroxicosis
    - hypoxia
    - acidosis
There are two mechanisms of spontaneous impulse initiation that may lead to arrhythmias,

*Triggered activity - Ectopic automatic rhythms*

- occur when the dominant pacemaker shifts from the sinus node to a latent pacemaker,
  - When the intrinsic rate of the SA node < ectopic pacemaker,
    - the intrinsic rate of the ectopic pacemaker increases above the normal SA rate
      - atrial ectopic tachycardia
      - junctional ectopic tachycardia
      - ventricular tachycardia
  - When the normal sinus impulse is prevented from conduction throughout the heart (AV block), leaving an ectopic pacemaker free to fire at its own, slower intrinsic rate.
Abnormal impulse conduction

- **Reentry**
  - propagation of an impulse through myocardial tissue already activated by the same impulse in a circular movement.
  - Reentry is the underlying mechanism of
    - atrial flutter
    - reentrant supraventricular tachycardia (SVT).

- **Blockage**
  - of the propagating cardiac impulse occurs when it arrives in regions of the heart that are not excitable, because
    - the tissue is still in the refractory period after a recent depolarization (e.g. 2:1 AV conduction ratio during atrial flutter)
    - the tissue is functionally abnormal (e.g. Replacement by scar tissue).
Intrauterine investigation of fetal rhythm and AV conduction

- **External fetal heart rate monitoring**
  - readily available
  - able to provide continuous monitoring over long periods of time
  - Ineffective at high rates
- **Conventional ECG**
- **Transmaternal fetal ECG**
  - a real-time fetal ECG is not obtainable due to the parasitic electrical field generated by the maternal heart and abdominal muscles.
  - May provide useful information
    - cardiac time intervals such as PR, QRS, and QT duration during a stable cardiac rhythm
    - it does not allow the analysis of individual cardiac cycles.
Intrauterine investigation of fetal rhythm and AV conduction

- **Fetal magnetocardiography (fMCG)**
  - allows recording of the fetal heart magnetic field instead of the traditional electric field recorded by ECG.
  - the best modality to analyze the fetal heart rhythm
  - restricted to select centers due to its high cost
Intrauterine investigation of fetal rhythm and AV conduction

- Ultrasound imaging of the fetal heart
  - 2D US
  - M-mode
  - Tissue Doppler imaging
    - can be utilized to help characterize wall motion
  - Pulsed wave Doppler
    - SVC/ Asc. aorta Doppler
    - Left ventricular outflow tract
Echocardiographic assessment of the fetal atrioventricular conduction system

- Stepwise interpretation of the fetal heart rhythm is based on the
  - determination of rhythm origin
  - determination of regularity
  - relationship between atrial and ventricular events
  - Rate

- Electrophysiological ‘normality ’
  - regular and normocardic fetal heart rate
  - with a normal 1:1 AV relationship
Echocardiographic assessment of fetal arrhythmias

- Irregular rhythm
  - In at least 90% of an unselected pregnancy population
    - can originate from atria/AV junction/ventricle
    - brief, isolated, and clinically benign events,
    - typically presenting as occasional ‘skipped beats’ due to isolated PACs

- Abnormal rates
  - prolonged or persistent
    - Bradycardia (heart rate < 100 beats per minute)
    - Tachycardia (heart rate > 180 beats per minute)
Irregular rhythm

- **Premature atrial contractions**
  - 90% of irregular rhythm in fetuses
  - The ventricular contraction occurs
    - prematurely if the PAC is conducted,
    - missing if the PAC is non-conducted to the ventricles.
    - In both situations, the ventricular rate is irregular
      - unless non-conducted PACs occur in a bigeminal pattern
  - Etiology
    - thyroid disease consumption of stimulants ??
  - 1-3% will develop a tachyarrhythmia
  - mostly benign and remain self-limited with a spontaneous resolution
    - after the diagnosis of the arrhythmia before birth in 95% of fetuses
    - by 1 year of age in 95% of children
  - perform a detailed fetal cardiac ultrasound
    - If the PACs are very frequent (>5 per minute, bigeminy, trigeminy),
    - Persisting for more than 3 weeks
    - associated with signs of cardiac failure or extracardiac anomalies, it is recommended
  - CHD is identified in only 0.3–2%

Irregular rhythm

- Premature junctional and ventricular contractions
  - Very rare
  - difficult to diagnose
  - PJC
    - simultaneous premature atrial and ventricular wall motion.
  - PVC
    - ventricular wall motion, which is not preceded by atrial contraction.

- Isolated PJC, PVC
  - benign prognosis
  - cardiovascular compromise can occur if sustained junctional or ventricular tachycardia develops

- M-mode recording PVC
  - the atrial rhythm (A–A) is regular,
  - the ventricular rhythm (V–V) is regularly irregular
    - due to prematurity of every alternating ventricular beat (ventricular bigeminy).
  - The average ventricular rate remains normal, despite the fact that only every second atrial beat is conducted (indicated with arrows).
Fetal bradydysrhythmia

Bradycardia

- Defined as
  - an area of the heart that depolarizes slower than the normal range for age for at least three successive beats.
  - A ventricular rate in fetuses <110 bpm
- <5% of arrhythmia referral in fetuses
- Results from
  - An abnormally slow atrial pacemaker activity with a normal 1:1 AV conduction,
  - results from different forms of conduction block at the AV junction
- Bradydysrhythmia may be
  - an isolated rhythm disorder,
  - associated with structural fetal heart disease.
- Most common causes of a sustained slow heart rate are
  - complete heart block, persistent ventricular rate <60 bpm
  - sinus bradycardia, rates between 60 and 80 bpm
  - blocked atrial bigeminy.

Fetal bradydysrhythmia

- Bradycardia
  - fall in fetal heart rate
    - reduction in fetal cardiac output
    - sustained fetal bradydysrhythmia may compromise the fetal circulation
    - important in fetuses affected by associated structural cardiac defects,
      - may limit their cardiac performance further.
  - Due to the reduced compliance of fetal ventricles, diastolic ventricular filling in the fetal heart depends to a larger degree on the atrial contraction than postnatally.
  - Worst Scenario
    - sustained fetal bradydysrhythmia
    - with a very slow ventricular rate of less than 50 bpm
    - Concomitant complete atrioventricular block

Fetal bradydysrhythmia
Clinical presentation

- **Sinus bradycardia**
  - The most common cause of bradycardia.
  - Fetal heart rate is regular and slow
    - < 100 beats per minute
  - Atrial and ventricular activities are associated in a 1:1 fashion.
- **Transient**
  - Increased vagal discharge in the fetus, possibly resulting from the pressure applied to the maternal abdomen by the transducer.

*SVC/aorta Doppler recording of sinoatrial bradycardia.*
- Abnormally slow, but regular atrial (A) and ventricular (V) rates that occur in a normal 1:1 AV relationship.
Sustained sinus bradycardia,
  - needs to be evaluated
  - may be found in the seriously sick fetus and will commonly be associated with other signs of impending fetal demise such as loss of fetal movements or fetal hydrops

- Maternal hypothermia
- Sick sinus
- Long QT syndrome
  - Long-QT syndrome is a heterogeneous genetic disorder caused by mutations in several genes that encode different ion channel proteins, most of them potassium channel proteins.

- Moderate bradycardia is also found in the fetus with frequent premature atrial contraction which are blocked at the level of the atrioventricular node.
- AV block – 2nd, 3rd degree
- familial idiopathic atrial fibrillation with slow ventricular response.
Fetal bradydysrhythmia
Clinical presentation

- **Sustained Bigeminy PAC’s with blocked premature**
  - 60-100 beats per minute.

- **SVC/aorta Doppler recording of atrial bigeminy.**
  - Normal SA node impulse (A) and premature atrial contraction (A2= PAC) alternate.
  - PAC occurs prematurely enough to regularly fail conduction to the ventricles.

- **Careful echocardiographic investigation is required to distinguish**
  - benign and transient cause of bradyarrhythmia
  - life-threatening high-degree AV block or sinus bradycardia
Fetal bradydysrhythmia
Clinical presentation

- Congenital AV Block
  - 1st degree
    - AV conduction time increased
    - Gestational age-matched reference values of AV time intervals
  - 2nd degree
    - Mobitz Type I (Wenckebach)
    - Mobitz Type II
Fetal bradydysrhythmia
Clinical presentation

- **3rd degree**
- **Complete heart block**
  - The slowest fetal heart rate will be noted if complete AV block is present.
    - complete dissociation of atrial and ventricular contractions with normal atrial, but slow ventricular rates
    - once in about 20 000 newborns.
    - The incidence may be higher in prenatal life,
      - some fetuses with complete heart block will not survive to term.
  - Complete heart block may result from
    - a lack of fusion between nodal tissue and the His bundle, which initially develop separately,
    - secondary interruption of the atrioventricular conduction axis.
Fetal bradydysrhythmia

- **Isolated complete heart block**
  - an immunological disorder
  - Mothers of affected fetuses often have connective tissue disease
    - Sjögren’s syndrome
    - Systemic lupus erythematosus
  - Almost all of them are positive for autoantibodies
    - cross the placental barrier
    - react with fetal cardiac tissue,
    - SSA/Ro or SSB/La ribonucleoproteins located on the cell surface
      - involved in the normal developmental apoptosis of cardiac cells
      - the anti-Ro antibodies, with consequent impairment of the physiological apoptosis, attraction of macrophages, and production of cytokines.
      - Inflammation, fibrosis, and calcification in the conducting system
      - heart block and/or endocardial fibroelastosis


Fetal bradydysrhythmia

- **Isolated complete heart block**
  - The risk for a woman with known anti-SSA/Ro or anti-SSB/La antibodies to have a child with complete heart block is only about
    - 2 – 5 %, and
  - After having had one child with complete heart block
    - the recurrence rate is just 15 – 20% in subsequent pregnancies.
  - Commonly, isolated complete heart block
    - develops between 18 and 24 weeks of gestation
    - progression from second-degree to complete heart block has been observed in some cases.

Fetal bradydysrhythmia

- **Complete heart block and structural heart disease**
  - Complete heart block associated with structural heart disease is mainly seen in fetuses with complex cardiac lesions.
    - Hearts with left atrial isomerism
      - bilateral left atrial morphology
      - lack a normal AV node which is a right atrial structure,
    - discordant AV connection
      - the inversion of the ventricles often leads to disruption of the AV conduction axis.
    - ventricular non-compaction
      - this form of cardiomyopathy may worsen cardiac function further, ü
      - none of the affected fetuses survived the neonatal period.
  - Associated structural heart disease is seen in
    - 30% of newborns with congenital complete heart block

Prevention and prenatal treatment of complete heart block

The effectiveness of CHB prenatal treatment is controversial.

- The rationale of such treatment is
  - To reduce damage to the conduction system and the myocardium through anti-inflammatory agents
    - Both dexamethasone and betamethasone have been used;
    - in favor of long-term treatment are not available,
    - concerns about fetal and maternal side effects of steroid therapy have been raised.

<table>
<thead>
<tr>
<th>Study</th>
<th>Intervention</th>
<th>Number of Participants</th>
<th>Outcomes</th>
</tr>
</thead>
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<tr>
<td>Izmirly et al</td>
<td>FS use in cases with second-degree AV Block (R)</td>
<td>20 treated, 16 untreated</td>
<td>Higher percentage reverting to first-degree AV block or normal sinus rhythm in treated group (P = 0.053)</td>
</tr>
<tr>
<td>Eliasson et al</td>
<td>FS use in cases with hydrops fetalis (R)</td>
<td>27 treated, 10 untreated</td>
<td>Lower 6-month mortality in treated group (P = 0.059)</td>
</tr>
<tr>
<td>Jaeggi et al</td>
<td>FS use in cases at diagnosis of heart block (R)</td>
<td>21 treated, 16 untreated</td>
<td>Lower 1 year mortality in treated group (P &lt; 0.02)</td>
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<tr>
<td>Eliasson</td>
<td>FS use in cases with second- and third-degree AV block (R)</td>
<td>67 treated, 108 untreated</td>
<td>No significant difference in mortality between groups</td>
</tr>
<tr>
<td>Buyon et al</td>
<td>IVIG 400 mg/kg q3weeks from GW 12–24 in mothers with previous cardiac NL child (P)</td>
<td>33 treated</td>
<td>6 cases of cardiac NL (18% recurrence rate)</td>
</tr>
<tr>
<td>Pisoni et al</td>
<td>HCG exposure throughout pregnancy in mothers with previous cardiac NL child (R)</td>
<td>40 treated, 217 untreated</td>
<td>Decreased recurrence rate of cardiac NL in treated group (P = 0.050)</td>
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FS indicates fluorinated steroids; AV, atrioventricular; IVIG, intravenous immunoglobulin; HCG, hydroxychloroquine; R, retrospective analysis; P. prospective study; GW, weeks of gestation.

Fetal bradydysrhythmia

**Prevention and prenatal treatment of complete heart block**

- increase the ventricular heart rate to above 60 bpm in cases with a very low (<55 bpm) ventricular escape rate
  - administration of beta-agonists.
    - Salbutamol / terbutaline
      - to increase the fetal cardiac output through an increase in heart rate associated with a reduction in peripheral resistance.
    - if administered at high dosages
      - maximum of 40 mg/day for salbutamol
      - maximum of 30 mg/day for terbutaline
    - are effective in increasing the heart rate by 5–10 bpm.
  - Common maternal side effects
    - include tremors, tachycardia, and sweating, which usually disappear with continuation of therapy.
  - Serious effects
    - pulmonary edema, myocardial ischemia, and arrhythmias have been described.

Fetal tachyarrhythmia

- **Tachycardia is defined as**
  - an area of the heart that depolarizes faster than the normal range for age for at least three consecutive beats,
  - above 180 bpm in the fetus between 20 and 40 weeks of gestation

- **Fetal tachycardia**
  - evaluation and consultation
    - presence of sustained tachyarrhythmia or an intermittent arrhythmia that is frequent and prolonged (occurring >50 percent of the time) puts the fetus at risk for cardiovascular failure

- **Echocardiographic signs of hemodynamic compromise**
  - **Early**
    - bialtrial enlargement
    - atrioventricular valve regurgitation
  - **Late**
    - cardiomegaly
    - decreased systolic function,
    - hydrops fetalis
    - can appear within 24 hours of onset of sustained tachycardia

- **Associated maternal complications due to**
  - severe polyhydramnios,
  - Preterm contractions and labor,
  - premature rupture of the membranes

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Fetal tachyarrhythmias

- Sinus tachycardia,
- Supraventricular tachycardia (SVT)
- Atrial flutter
- Ventricular tachyarrhythmia

According to electrophysiological levels

- Atrial tachycardia
  - atrial flutter,
  - atrial ectopic tachycardia
- Conduction system tachycardia
  - Atrioventricular reentry tachycardia via an apparent or ‘concealed’ accessory pathway,
- Permanent junctional reciprocant tachycardia,
- Atrioventricular nodal reentry tachycardia
- Ventricular tachycardia.

- Extrasystoles,
  - supraventricular origin is by the most frequent cause,
  - AV nodal and ventricular origins being infrequent in healthy fetuses and infants.
- In fetuses
  - 70 % are paroxysmal AV reentry tachycardia,
  - 24 % are primary atrial tachycardias (mostly atrial flutter),
  - 6 % sinus tachycardia

Fetal tachyarrhythmia

- **Sinus tachycardia**
  - usually slower than AET and PJRT
  - atrial rates of 180 – 200 bpm
  - normal 1:1 AV conduction,
  - some variability of the fetal heart rate
  - Prolonged sinus tachycardia
    - fetal distress,
    - anemia,
    - infections,
    - elevated maternal catecholamine levels due to anxiety or pain
    - Maternal β-stimulation,
    - fetal thyrotoxicosis.
  - The importance of sinus tachycardia is recognizing and treating the underlying cause.
Fetal tachyarrhythmia

- Ventricular tachycardia and junctional ectopic tachycardia are exceptional causes of fetal tachyarrhythmias.
  - VT is very rare in fetuses
    - <1% of all tachyarrhythmias
  - An underlying structural heart disease is present in approximately half of the pediatric cases
    - hypertrophic cardiomyopathy,
    - long QT syndrome,
    - right ventricular dysplasia,
    - left ventricle noncompaction,
    - congenital cardiac malformation
  - If there is no retrograde conduction across the AV node or an accessory pathway, the ventricular rate will exceed the atrial rate during ventricular or junctional tachycardia.
  - If there is retrograde 1:1 VA conduction, these arrhythmias become difficult to discern from SVT.
  - Treatment
    - beta-blockers, flecainide, sotalol, lidocaine, and amiodarone,
    - but due to the very limited number of cases, success rate of treatment is not clearly established
    - and a first-line agent remains to be established.

Atrial flutter

- atrial rate > 300 bpm
- every second or third atrial beat is conducted to the ventricles
- ventricular response rates between 150-250 bpm
Fetal tachyarrhythmia

Supraventricular Tachycardia (SVT)

1:3,700 pregnancies

It accounts
- 5 to 10% of all fetal arrhythmias,
- up to 90% of all tachycardias
- >50% of the clinically significant

Characterized by
- Regular heart rate between 220–260 bpm
- Can be sustained for hours or days, but more commonly intermittent.

Fetal tachyarrhythmia

- SVT encompasses three different arrhythmia mechanisms:
  - AV re-entrant tachycardia
    - related to fast retrograde accessory pathway conduction
    - Most commonly
  - Permanent junctional reciprocating tachycardia (PJRT)
    - related to slow retrograde pathway conduction
  - atrial ectopic tachycardia (AET)
    - due to enhanced atrial focal automaticity.

Fetal tachyarrhythmia

- Determination of the type of SVT is based on the assessment of the AV relationship and other specific characteristics
  - Sinus tachycardia and ventricular tachycardia should be ruled out since management and prognosis differ from nonsinus SVT.
  - Provoking factors for nonsinus SVT in the fetus have to be looked for
    - Co-existing CHD,
    - Hyperthyroidism,
    - Maternal caffeine, alcohol, or nicotine consumption.
      - These last causes are among the most frequent ones [49].
Fetal tachyarrhythmia

- They are distinguished based on their arrhythmia pattern and VA time relationship.
  - AV reentry, short VA tachyarrhythmia
    - retrograde atrial activation proceeds across a fast conducting accessory pathway
    - therefore occurs shortly after the ventricular contraction.
  - In long VA SVT,
    - the atrial contraction closely precedes the ventricular contraction.
    - This activation pattern is typically seen during AET, PJRT, and sinus tachycardia.
      - Sinus tachycardia is the most common cause.
In utero diagnosis of fetal tachyarrhythmia

- The majority of fetal tachyarrhythmias detected during routine obstetric examination in the second and third trimesters of pregnancy.

- If an intensive noninvasive and invasive search for an underlying disease is unsuccessful,
  - paroxysmal supraventricular tachyarrhythmia should always be taken into consideration,
  - particularly if signs of congestive heart is present.
  - repeated sonographic heart rate monitoring
  - long-term cardiotocography carried out several times per day
Fetal tachyarrhythmia

- Treatment
  - Four options are available for fetuses diagnosed with tachycardias:
    - Delivery and plan for postnatal treatment
    - Transplasental fetal treatment through administration of antiarrhythmic drugs to the mother
    - Direct (invasive) fetal treatment, with delivery of the antiarrhythmic drug directly into the fetal circulation
    - Close monitoring with no active intervention.

- As a general rule, preterm delivery of a sick hydropic fetus should be avoided
  - most experts agree on not treating a fetus with an intermittent tachycardia and no signs of fetal heart failure.

Fetal tachyarrhythmia

Things to remember

- Maternal administration of antiarrhythmic drugs is effective in the majority of cases.

- If maternal administration of antiarrhythmic drugs fails, then direct fetal therapy or delivery should be considered.
  - Hydrops reduces the transplacental transfer of the drug.

- Hydrops represents a poor prognostic sign.
  - Associated with a decrease in survival rate (73% vs. 96% in nonhydropic fetuses), a
  - Higher incidence of preexcitation on neonatal electrocardiography (ECG) (16% vs. 4%),
  - And a higher chance of long-term postnatal antiarrhythmic therapy.

Fetal tachyarrhythmia

The optimum approach depends on the following factors

- **Tachycardia rate**
  - Rates >220 bpm are most likely to progress to hydrops,
  - rates <200 to 220 bpm are much less likely to have hemodynamic consequences.

- **Persistence of the tachycardia**
  - SVT present >50 percent of the day is likely to lead to hydrops
  - tachycardia <20 percent of the day is usually well-tolerated.
  - Intermittent short bursts of tachycardia are very well tolerated and do not mandate treatment.

- **Gestational age**
  - Delivery for postnatal treatment is preferable as gestational age increases and prematurity risks decrease.

- **Presence/absence of hydrops**
  - Prior to pulmonary maturity, sonographic evidence of developing hydrops mandates treatment to slow the FHR and improve cardiac performance.
  - Delivery followed by postnatal treatment is less desirable in this setting because the combination of hydrops and prematurity is associated with very high morbidity and mortality.
  - Nonhydropic preterm fetuses with frequent and/or long periods of SVT are often treated since successful cardioversion is less likely after hydrops has developed.

- **Congenital heart disease**
  - If structural anomalies are present, the postnatal management and prognosis of these anomalies need to be taken into account.

- **Maternal factors**
  - Preeclampsia or mirror syndrome places the mother at risk of severe sequelae, and is an indication for intervention.

Fetal tachyarrhythmia

- Initial maternal and fetal monitoring
  - maternal assessment
    - medical history (especially cardiac history),
    - medication history,
    - ECG,
    - blood pressure,
    - laboratory tests (serum electrolytes, tests of renal and hepatic function, urine protein, and platelet count).
  - fetal
    - observation to document baseline status
Transplasental Fetal Antiarrhythmic Therapy

- The choice of the drug will depend mostly on the state of the fetus (signs of heart failure, fetal hydrops) as well as on the type of SVT.
- Since no large prospective randomized controlled trial (RCT) has been undertaken, there is to date no agreement on the best antiarrhythmic.

First-line agents
- Digoxin
- Sotalol
- Flecainide

Second-line antiarrhythmics
- Propafenone
- Amiodarone
- Adenosine

In Hydropic fetuses
- The conversion rate decreases to less than 25 % with digoxin
- flecainide + digoxin
- sotalol + digoxin

Fetal tachyarrhythmia

**Digoxin**

- Based on its safety profile and efficacy, digoxin is the initial drug of choice
  - either administered orally or intravenously to the mother
  - Or via direct intramuscular fetal injection

- **Rapid Loading dose**
  - 1 to 2 mg
  - which can be given in three doses: 0.5 mg, 0.25 mg, and 0.25 mg
  - over 18 to 24 hours,
  - followed by a digoxin level.
    - Additional doses are given if the digoxin level is low.
  - The target level is 1 to 2 ng/mL.

- **Maintenance dose**
  - determined by titrating to the fetal response, which might take several days.
  - higher in pregnant women than nonpregnant
  - 0.5 to 0.75 mg daily given in divided doses.

- **Daily ECGs to monitor**
  - P-R prolongation
  - T wave changes

- **Direct fetal intramuscular injection of digoxin combined with transplacental therapy**
  - appears to shorten the time to initial conversion of SVT and to sustain sinus rhythm in the fetus with SVT complicated by hydrops fetalis.

Fetal tachyarrhythmia

- **Flecainide**
  - 50 mg, 100 mg, and 150 mg tablets;
  - pregnancy class C
    - Initial dose 3x100
    - Maintenance 250-300mg
  - cleared by the kidney
  - three times daily dosing during pregnancy
  - Continuous maternal cardiac monitoring for 48 hours or for the first five to six doses
  - daily ECGs

- **Sotalol**
  - 80 mg, 120 mg, and 160 mg tablets;
  - pregnancy class B
    - Initial dose 3x80
    - Maintainance 250-300mg
  - cleared by the kidney
  - three times daily dosing during pregnancy
  - Continuous maternal cardiac monitoring for 48 hours or for the first five to six doses
  - daily ECGs

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Non-Hydropic fetus

Transplacental Digoxin

Hydropic fetus

Transplacental Flecainide/Sotalol

Delivery for postnatal treatment

Direct Flecainide/Sotalol/Digoxin

Delivery for postnatal treatment

2nd line

Transplacental Digoxin + Flecainide/Sotalol

1st line

3rd line
Teşekkürler
<table>
<thead>
<tr>
<th>Arrhythmia</th>
<th>A rate</th>
<th>A-A interval</th>
<th>A-V relation</th>
<th>V rate</th>
<th>V-V interval</th>
<th>V-A interval</th>
<th>Incidence</th>
<th>Relevance, outcome</th>
</tr>
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<tr>
<td>Irregular rhythm</td>
<td>Isolated PAC, conducted</td>
<td>Normal</td>
<td>Irregular</td>
<td>1:1</td>
<td>Normal</td>
<td>Irregular</td>
<td>Variable</td>
<td>+++</td>
</tr>
<tr>
<td>Atrial trigeminy, conducted</td>
<td>Isolated PAC, blocked</td>
<td>Normal</td>
<td>Irregular</td>
<td>&gt;1:1</td>
<td>Normal</td>
<td>Irregular</td>
<td>+++</td>
<td>Minor, transient</td>
</tr>
<tr>
<td>Atrial trigeminy, blocked</td>
<td>Atrial bigeminy, conducted</td>
<td>Normal</td>
<td>Regularly irregular</td>
<td>1:1</td>
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</tr>
<tr>
<td>Atrial bigeminy, conducted</td>
<td>Isolated PVC (+VA block)</td>
<td>Normal</td>
<td>Regular</td>
<td>&lt;1:1</td>
<td>Normal</td>
<td>Irregular</td>
<td>Rare</td>
<td>Minor, transient</td>
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<tr>
<td>Ventricular bigeminy</td>
<td>Normal</td>
<td>Regular</td>
<td>1:2</td>
<td>Normal</td>
<td>Regularly irregular</td>
<td>Rare</td>
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<tr>
<td>Second degree AVB Wenckebach</td>
<td>Normal</td>
<td>Regular</td>
<td>&gt;1:1</td>
<td>Normal</td>
<td>Irregular</td>
<td>Rare</td>
<td>May progress</td>
<td></td>
</tr>
<tr>
<td>Bradycardia</td>
<td>Sinus</td>
<td>75–90</td>
<td>Regular</td>
<td>1:1</td>
<td>75–90</td>
<td>Regular</td>
<td>Long VA</td>
<td>+</td>
</tr>
<tr>
<td>Atrial bigeminy, blocked</td>
<td>Atrial bigeminy, blocked</td>
<td>Normal</td>
<td>Regularly irregular</td>
<td>2:1</td>
<td>65–90</td>
<td>Regular</td>
<td>Rare</td>
<td>Minor, transient</td>
</tr>
<tr>
<td>2:1 AV block</td>
<td>Normal</td>
<td>Regular</td>
<td>2:1</td>
<td>60–75</td>
<td>Regular</td>
<td>Rare</td>
<td>Major, may progress</td>
<td></td>
</tr>
<tr>
<td>Third degree AVB</td>
<td>Slow–normal</td>
<td>Regular</td>
<td>Dissociated</td>
<td>35–80</td>
<td>Regular</td>
<td>Rare</td>
<td>Major, irreversible</td>
<td></td>
</tr>
<tr>
<td>Tachycardia</td>
<td>Sinus</td>
<td>160–200</td>
<td>Regular</td>
<td>1:1</td>
<td>160–200</td>
<td>Regular</td>
<td>Long VA</td>
<td>Rare</td>
</tr>
<tr>
<td>AV reentry</td>
<td>Normal</td>
<td>Regular</td>
<td>1:1</td>
<td>190–280</td>
<td>Regular</td>
<td>Short VA</td>
<td>++</td>
<td>Major, treatable</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>Normal</td>
<td>Regular</td>
<td>Mainly 2:1</td>
<td>150–250</td>
<td>Mainly regular</td>
<td>Rare</td>
<td>Major, treatable</td>
<td></td>
</tr>
<tr>
<td>JET, PJRT</td>
<td>Normal</td>
<td>Regular</td>
<td>1:1</td>
<td>180–230</td>
<td>Regular</td>
<td>Long VA</td>
<td>Rare</td>
<td>Major, treatable</td>
</tr>
<tr>
<td>Ventricular (+VA block)</td>
<td>Slow–normal</td>
<td>Regular</td>
<td>&lt;1:1</td>
<td>160–260</td>
<td>Regularly irregular</td>
<td>Dissociated</td>
<td>Rare</td>
<td>Major, treatable</td>
</tr>
</tbody>
</table>

A, atrial; A→V, atrioventricular; JET, junctional ectopic tachycardia; PAC, premature atrial complex; PJRT, permanent junctional reciprocating tachycardia; PVC, premature ventricular complex; V, ventricular; V→A, ventriculatoatrial; VA block, absence of retrograde conduction via AV node or accessory pathway.

Incidence: +++ , detected in 1/10–1/1000 pregnancies; ++ , in 1/1000–1/10000 pregnancies; + , in 1/10000–1/100 000 pregnancies; rare, affects fewer than 1/100 000 pregnancies.
Bradycardia
irregular

AV activity associated, every 3rd atrial contraction premature, blocked at AV-node

allorhythmic (2:1) - PACs

AV delay increasing, atrial contraction repeatedly blocked at AV-node

2.° AV block (Wenckebach)

AV activity dissociated, frequent PVCs

complete AV block & PVCs
Bradycardia

**Regular AV Activity**
- AV activity associated, slow atrial and slow ventricular rate
  - Sinus bradycardia (LQTS?)

**Regular AV Activity Dissociated**
- AV activity dissociated, normal atrial and slow ventricular rate
  - Bigeminal PACs
  - Complete AV-block