Fetal Arrhythmia Diagnosis

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Disclosures

- No relevant disclosures

Overview

- Background and clinical implications
- General principles of fetal arrhythmia diagnosis
- Specific Diagnoses
  - Irregular Rhythm
  - Tachycardia
  - Bradycardia

Fetal Arrhythmias: Background

- Heart begins to beat at 22 days of gestation
- By 6 weeks post-conception, AV synchrony can be demonstrated
- Normal fetal heart rate is age-dependent
  - 6 weeks – 100 bpm
  - 9 weeks – 170 bpm
  - 14 weeks – 150 bpm
  - 20 weeks to term – 140 bpm
- Beat-to-beat variation 5-15 bpm

Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

American Heart Association

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Diagnosis and Treatment of Fetal Cardiac Disease: A Scientific Statement From the American Heart Association

Mary T. Donofrio, Anita J. Moon-Grady, Lisa K. Hornberger, Joshua A. Copel, Mark S. Sklansky, Alfred Abuhamad, Bettina F. Cuneo, James C. Huhta, Richard A. Jonas, Anita Krishnan, Stephanie Lacey, Wesley Lee, Erik C. Michelfelder, Sr, Gwen R. Rempel, Norman H. Silverman, Thomas L. Spray, Janette F. Strasburger, Wayne Tworetzky and Jack Rychik on behalf of the American Heart Association Adults With Congenital Heart Disease Joint Committee of the Council on Cardiovascular Disease in the Young and Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and Council on Cardiovascular and Stroke Nursing

Fetal Arrhythmias: Background

- Incidence of fetal arrhythmias is 1-2% of all pregnancies
- Account for 10-20% of referrals to fetal cardiologists
- Usually detected by routine Doppler in the OB office
- Most arrhythmias detected after 20 weeks
- 50% spontaneously resolve by evaluation
- 90% atrial or ventricular ectopy

Sustained arrhythmia can result in hydrops or demise

- 10% of total beats = sustained arrhythmia
  - Risk of hydrops or demise is 5-25% in untreated sustained arrhythmia
- Treatment itself has risks
  - Every antiarrhythmic agent has the potential to cause pro-arrhythmia and potential mortality

Arrhythmia can be associated with:

- Structurally normal heart
- Congenital heart disease
  - Heterotaxy
  - Ebstein's anomaly
- Tumors and ventricular wall defects

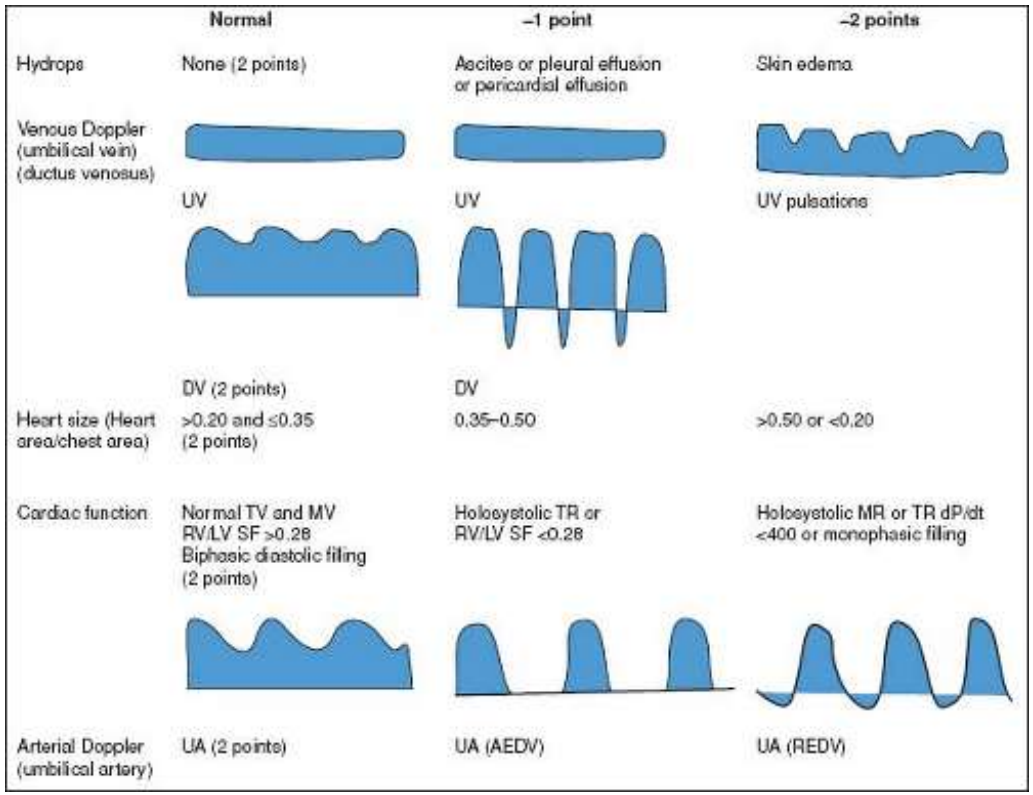
Fetal Arrhythmia Evaluation

- Assess fetal well-being
- Assess anatomy
- Diagnose rhythm

Fetal Arrhythmia Evaluation: Fetal Well Being

- Assess Anatomy and Degree of Heart Failure

Cardiovascular Profile Score

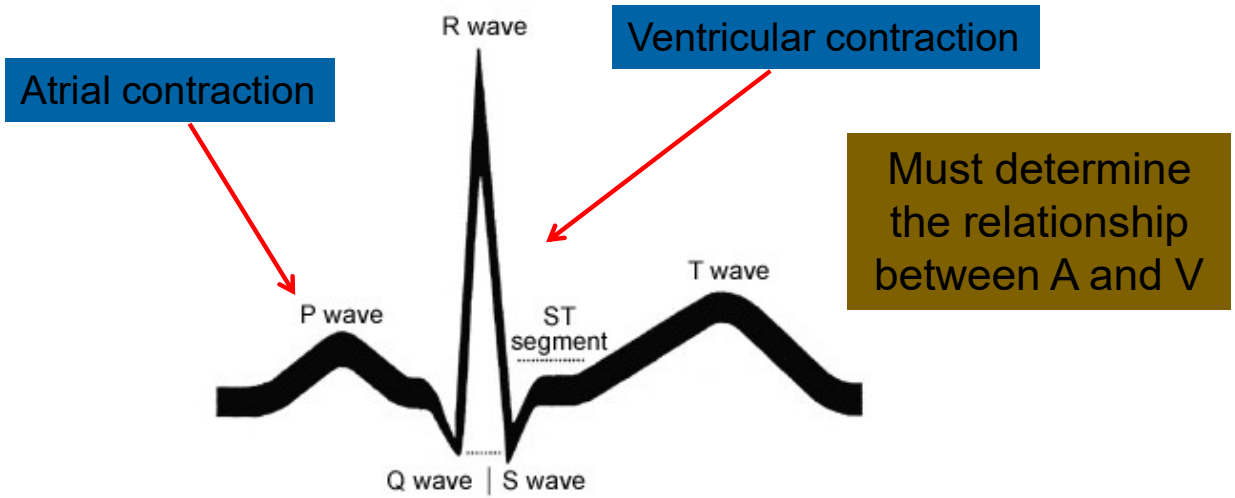


	Normal	–1 point	–2 points
Hydrops	None (2 points)	Ascites or pleural effusion or pericardial effusion	Skin edema
Venous Doppler (umbilical vein) (ductus venosus)	UV	UV	UV pulsations
	DV (2 points)	DV	
Heart size (Heart area/chest area)	>0.20 and ≤0.35 (2 points)	0.35–0.50	>0.50 or <0.20
Cardiac function	Normal TV and MV RV/LV SF >0.28 Biphasic diastolic filling (2 points)	Holosystolic TR or RV/LV SF <0.28	Holosystolic MR or TR dP/dt <400 or monophasic filling
Arterial Doppler (umbilical artery)	UA (2 points)	UA (AEDV)	UA (REDV)

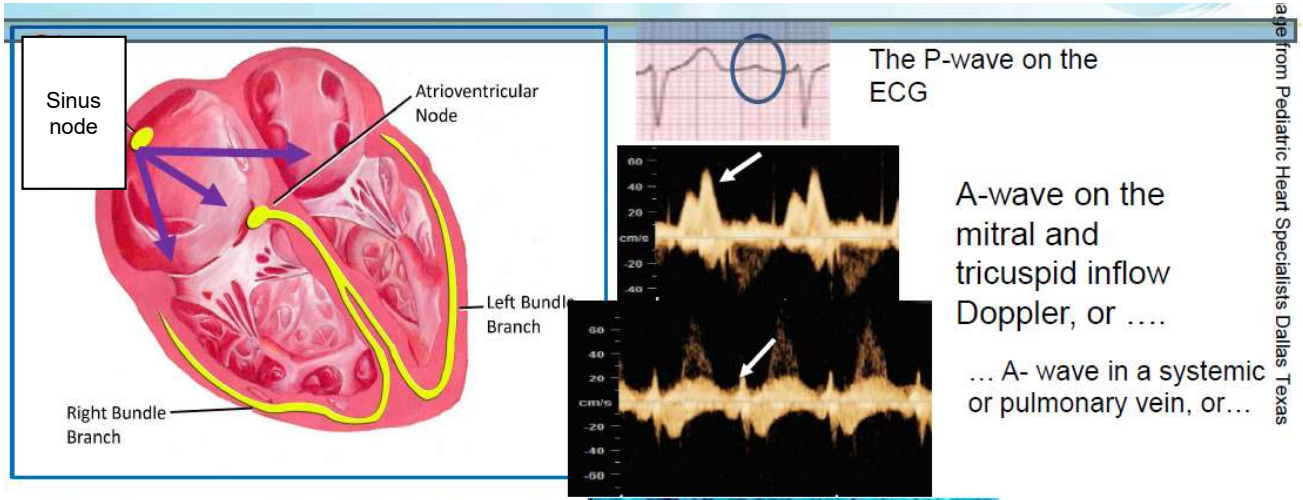
Fetal Arrhythmias Evaluation: Diagnosis

Why is diagnosing a fetal arrhythmia so tricky?

- No EKG – must make assumptions

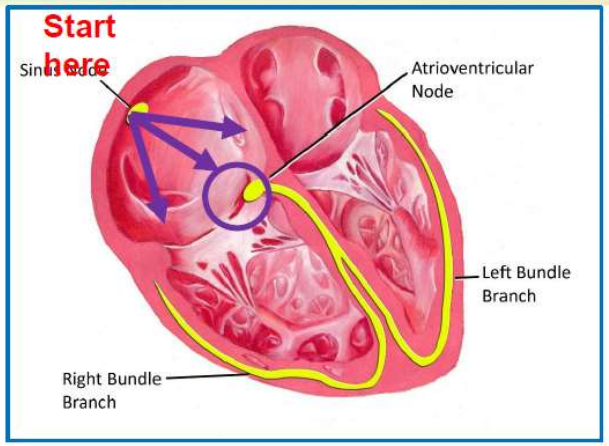


The normal cardiac conduction system

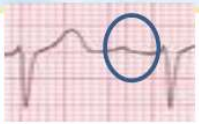


- The pacemaker region of the heart is the sinus node located in the right atrium
- It fires off and the impulse spread across the right and left atria and they contract
- Atrial contractions correspond to:

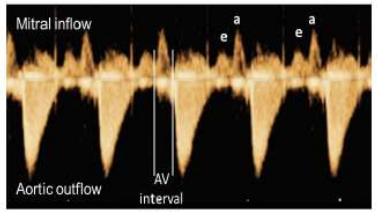
The normal cardiac conduction system



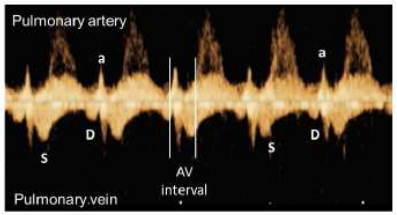
- The impulses are slowed at the AV node.
- This slowing corresponds to the PR or AV or mechanical PR interval
- If the AV node is abnormal, impulses are delayed (1° AV block), intermittently or not at all conducted (2° and 3° AV block



The PR interval on ECG



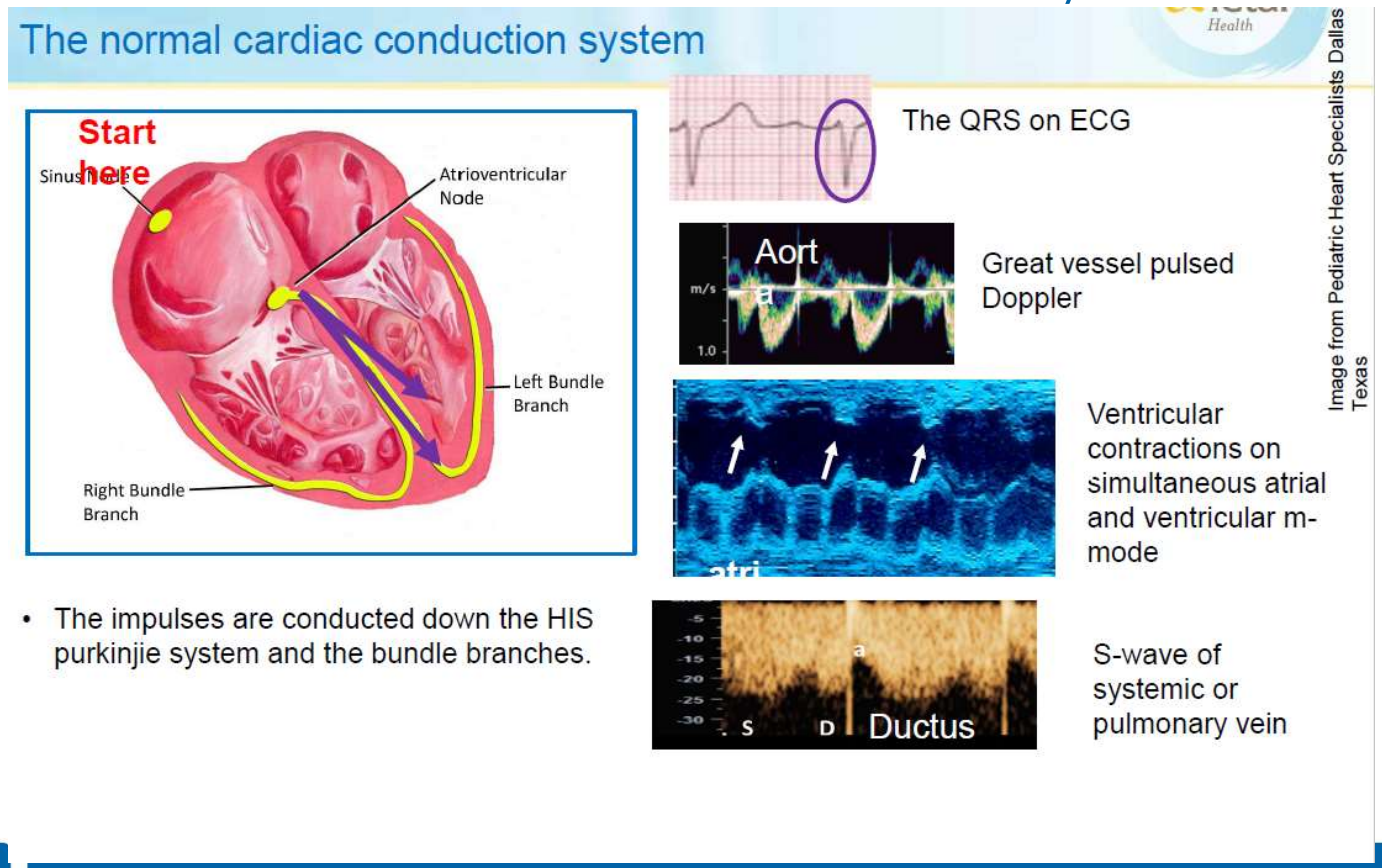
The AV interval… on mitral inflow and aortic outflow….



…or any systemic vein and artery

Image from Pediatric Heart Spe
Dallas Texas

The normal cardiac conduction system

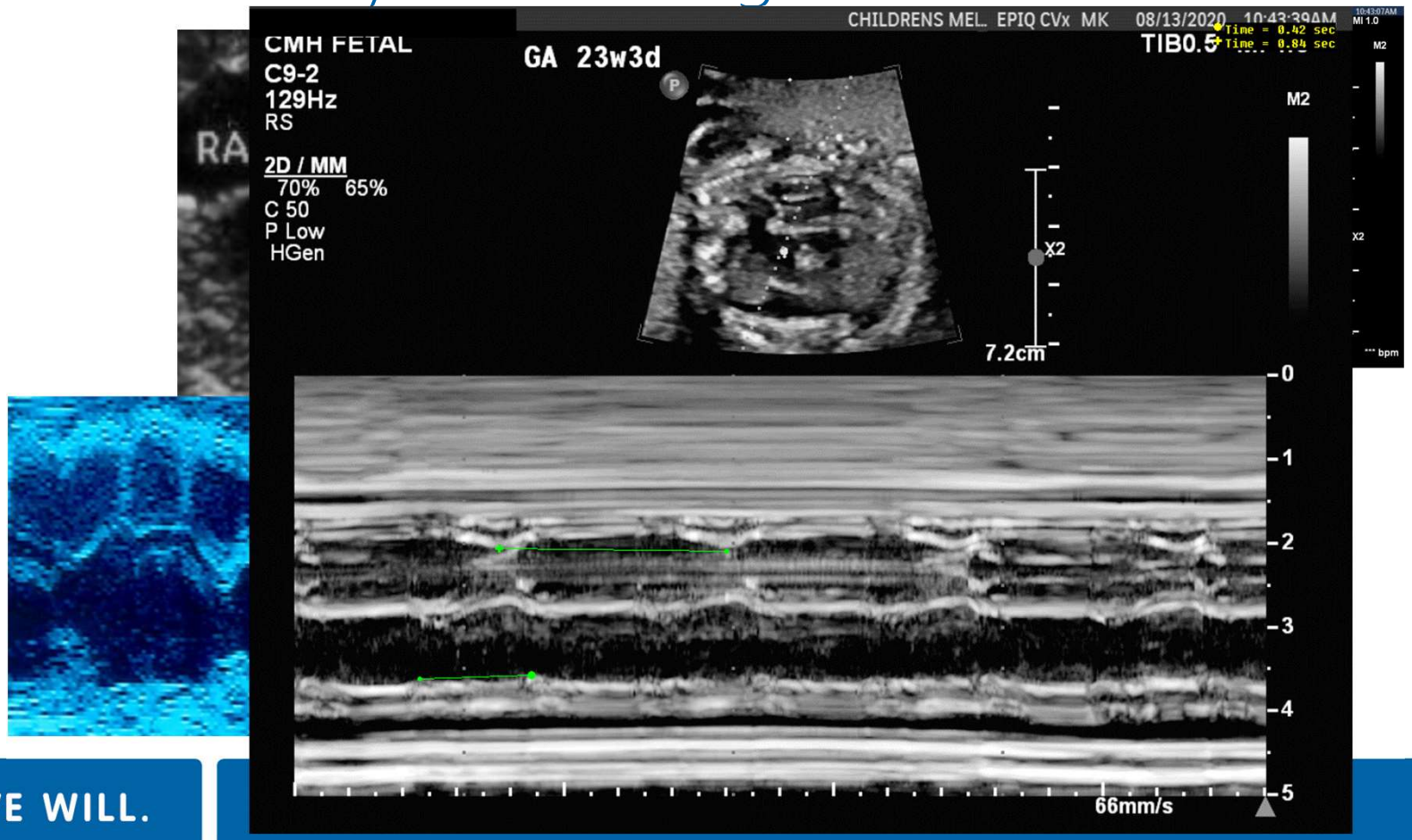


- The impulses are conducted down the HIS purkinjie system and the bundle branches.

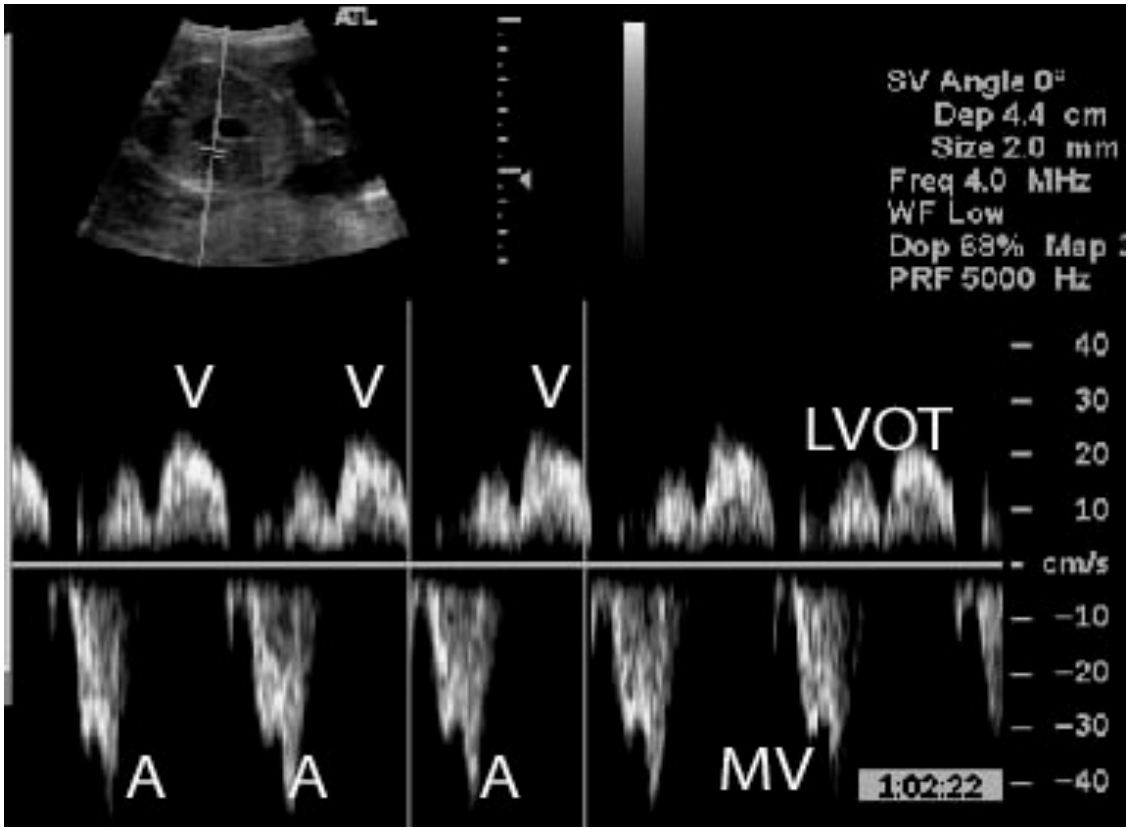
Fetal Arrhythmias: Diagnosis

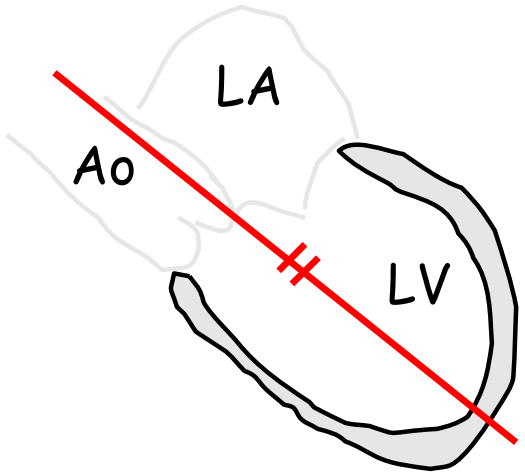
- M-mode
- Pulsed Doppler
  - LV inflow & outflow
  - SVC and ascending aortic flow
  - Pulmonary vein and pulmonary artery flow
- Tissue Doppler – atrial & ventricular
- Magnetocardiography

Fetal Arrhythmia Diagnosis: M-Mode



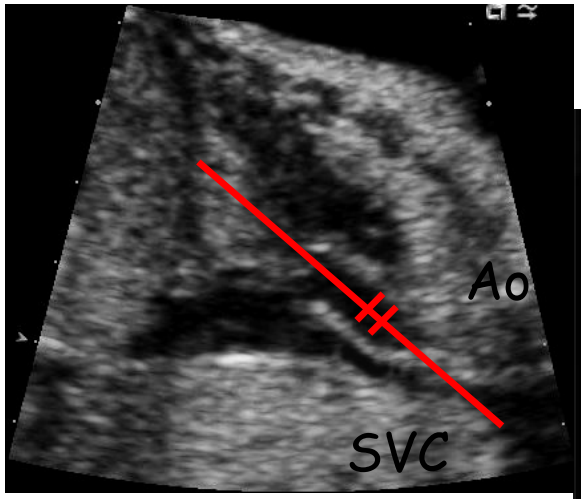
Fetal Arrhythmia Diagnosis: Pulsed Doppler



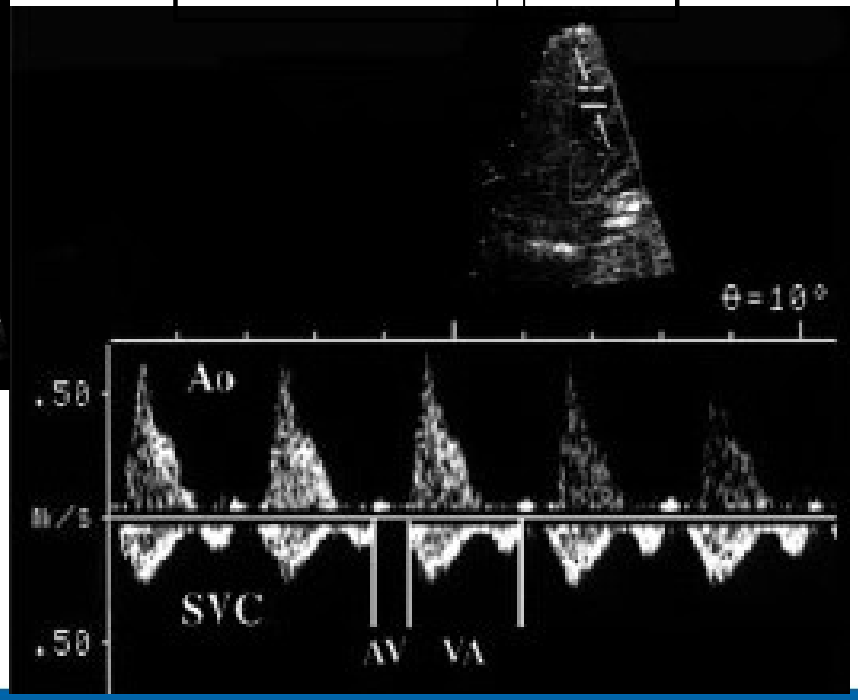


Pulsed Doppler
LV inflow-outflow

Fetal Arrhythmia Diagnosis: Pulsed Doppler

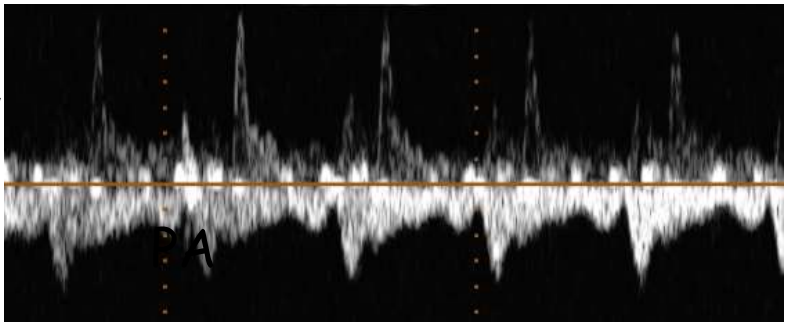


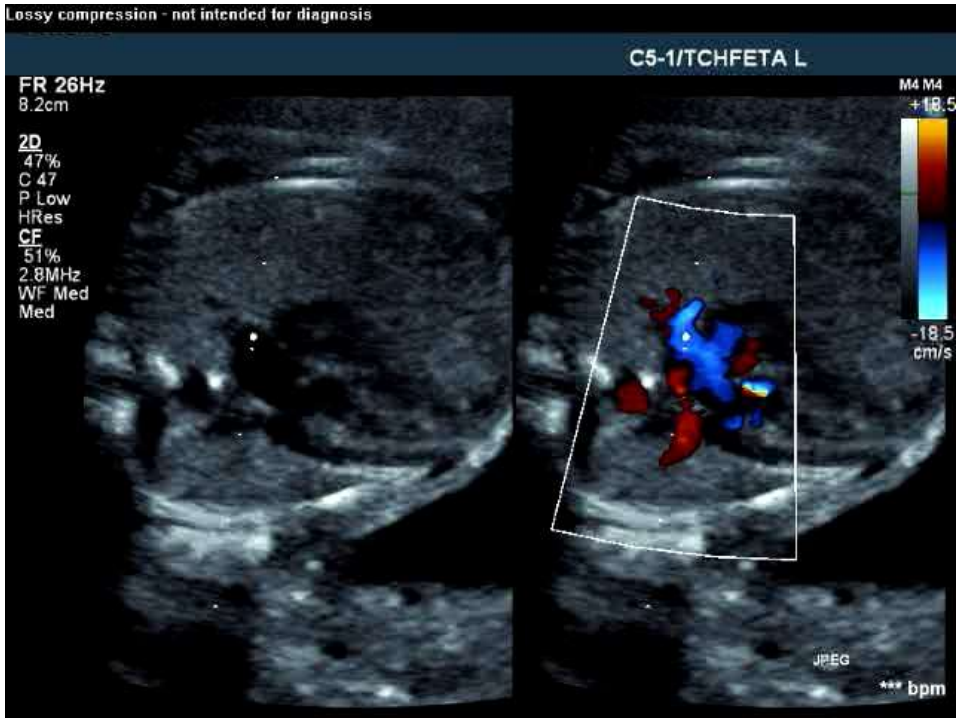
SVC-Ao Doppler



Fetal Arrhythmia Diagnosis: Pulsed Doppler

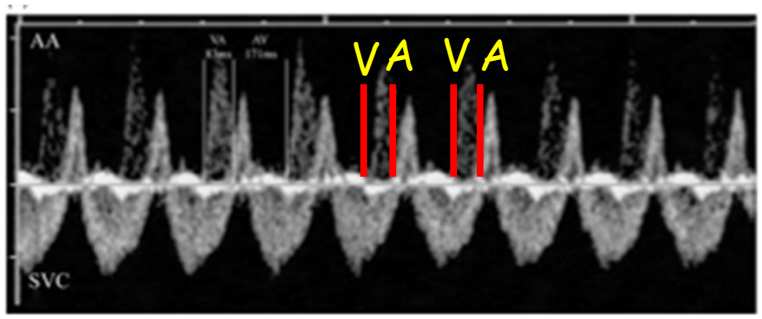
Pulmonary vein and pulmonary artery Doppler



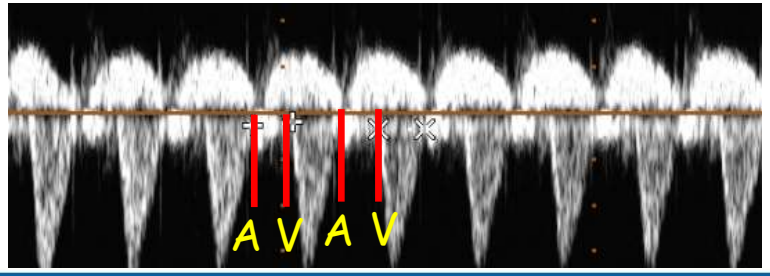


Fetal Arrhythmia Diagnosis: VA relationship

- The timing of the atrioventricular (AV) and ventriculoarterial (VA) relation is important in order to distinguish the mechanism of tachycardia.
  - Short VA time –



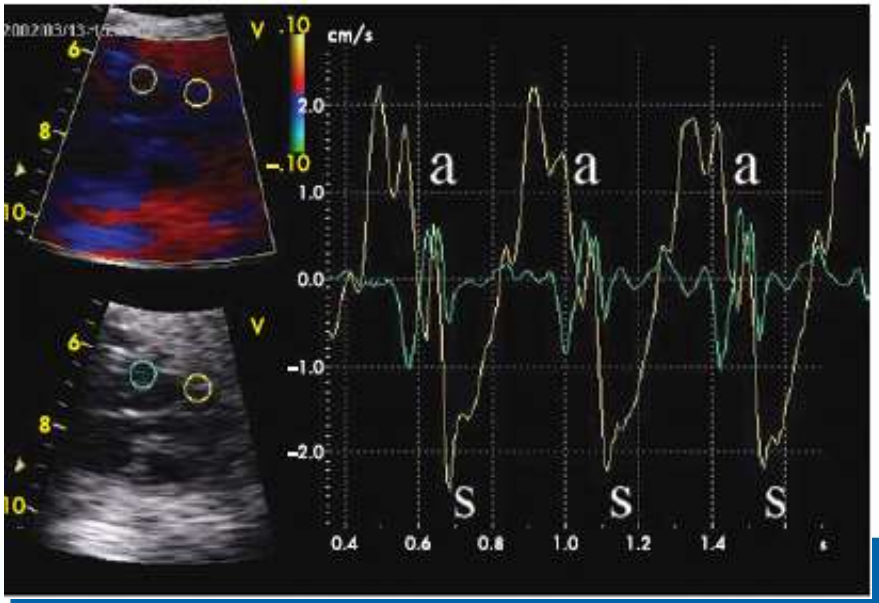
  - Long VA time -



Fetal Tachyarrhythmias

Tissue Doppler Imaging

Simultaneous sampling of atrial and ventricular wall velocities to yield precise temporal analysis of atrial and ventricular events.



4 chamber view – green – left atrium and yellow is simultaneous LV 1:1 relationship

Fetal Tachyarrhythmias

Magnetocardiography

- Records the magnetic fields generated by the electrical activity of the fetal heart

- Sensors are positioned several centimeters above maternal abdomen in a magnetically-shielded room

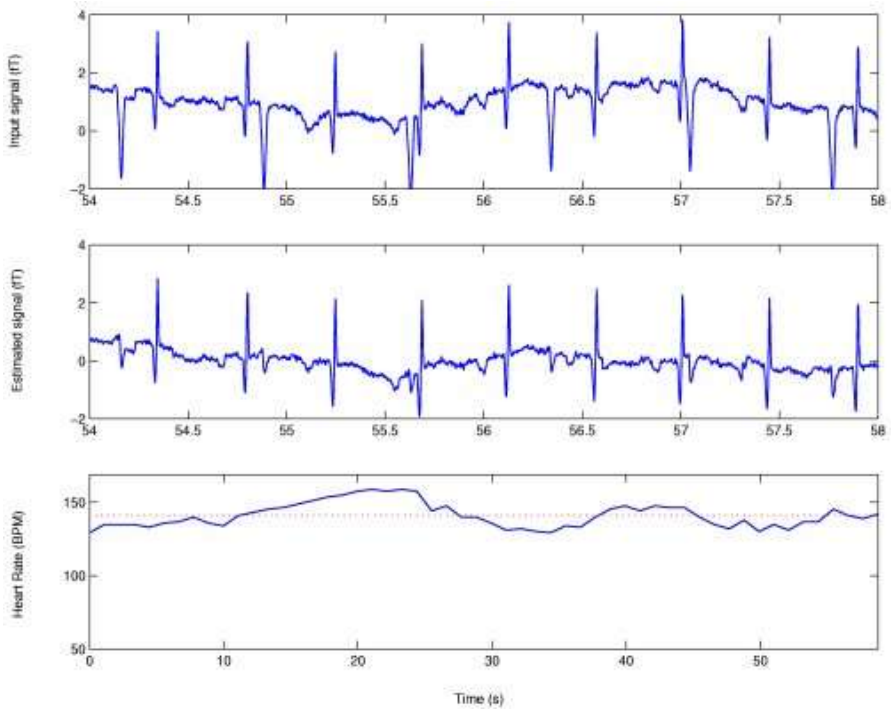
- High technical prerequisites

- Allows for more precise timing measurements of PR, QRS, and QT

Fetal Tachyarrhythmias

Magnetocardiography

Maternal HR removed



Echo v. fMCG

Echo	MCG
Accurate measurement of atrial and ventricular rates and approximation (mechanical consequences of) PR interval from 14-40 weeks GA	Accurate measurements of all cardiac intervals (QT, PR, QRS duration) from 18-40 week GA
Assess cardiac morphology and "function" (cardiac size, valve insufficiency, effusions, venous and arterial Doppler)	Morphology of P, QRS and T waves; repolarization abnormalities
HR assessment during echo	Beat to beat HR over several hours
Detects a therapeutic response to transplacental therapy	Electrophysiological signs of medication effect, toxicity and proarrhythmia
Ubiquitous and cost effective	Only in Wisconsin and Arkansas
Can even train fellows to do them	Only Ron REALLY knows how it works
You can take some impressive pictures	You can take some impressive pictures

Fetal Arrhythmias by type

- Irregular:
  - Premature Atrial Contractions (PACs)
  - Premature ventricular contractions
  - 2nd degree Heart block*
- Tachyarrhythmia
  - Atrioventricular reentrant tachycardia via an accessory pathway
  - Sinus tachycardia
  - Atrial Flutter (AF)
  - Ectopic Atrial Tachycardia (EAT)
  - Permanent Junctional Reciprocating Tachycardia (PJRT)*
  - Junctional Ectopic Tachycardia (JET)*
- Bradyarrhythmia
  - Sinus Bradycardia
  - Heart block
  - Long QT Syndrome

Fetal Arrhythmia Diagnosis by AV Relationship

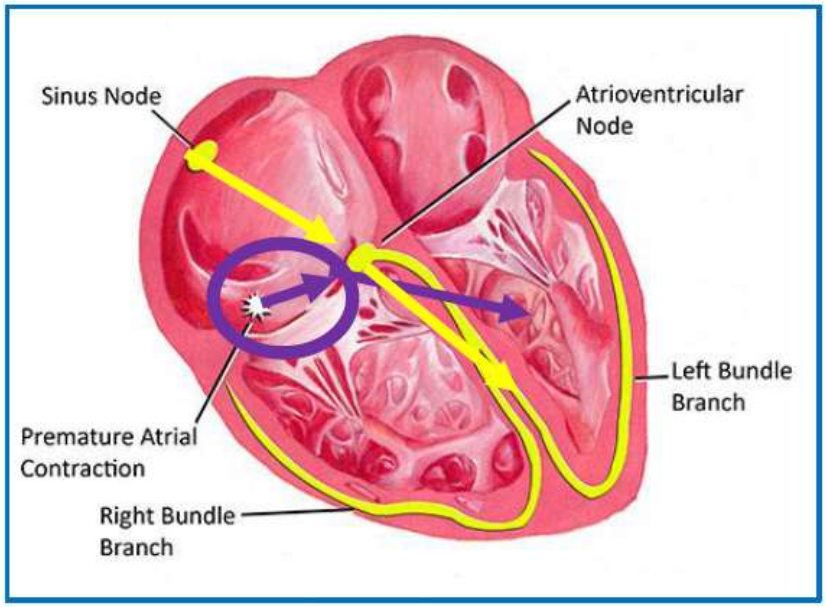
	A=V	A>V	A<V
Normal (130-170)	Sinus	Atrial flutter (4:1 block)	Accelerated ventricular rhythm; JET
Bradycardia (< 3rd% GA)	Sinus	AV Block BAB	Sinus node dysfunction and accelerated ventricular rhythm
Tachycardia (180-300)	Sinus SVT PJRT	Atrial Flutter CAT or AET	Ventricular tachycardia
Irregular Rhythm		Intermittent AVB Type 2, 2° AVB Atrial ectopy	Ventricular ectopy

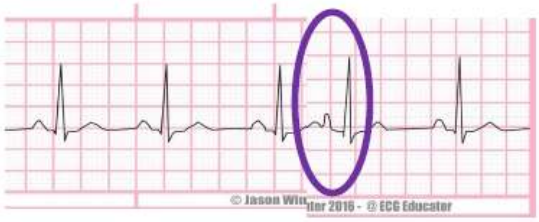
Fetal Irregular Rhythm:
Premature Atrial Contractions

- Most common referral for irregular HR
  - 90% of referrals for arrhythmia
- Usually > 30 weeks gestation
- Usually benign - associated with SVT in ~1%
- Can be associated with structural disease
  - Ebstein anomaly
  - Tumors
  - Ventricular aneurysm
  - Atrial septal aneurysms

Fetal Irregular Rhythm:

Premature Atrial Contractions



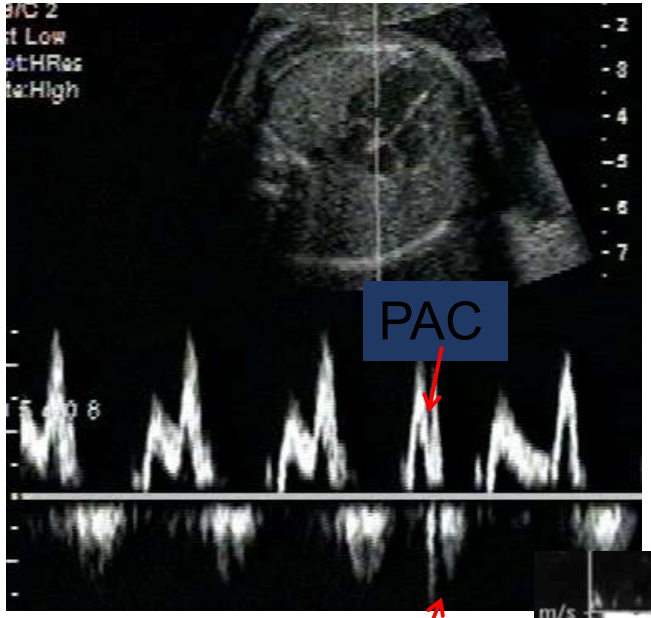


Fetal Irregular Rhythm:

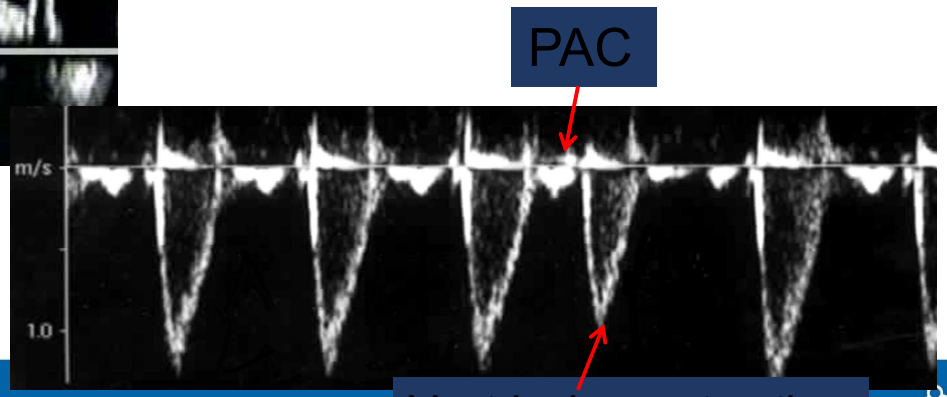
Premature Atrial Contractions

Fetal Irregular Rhythm:

Premature Atrial Contractions



Conducted PACs

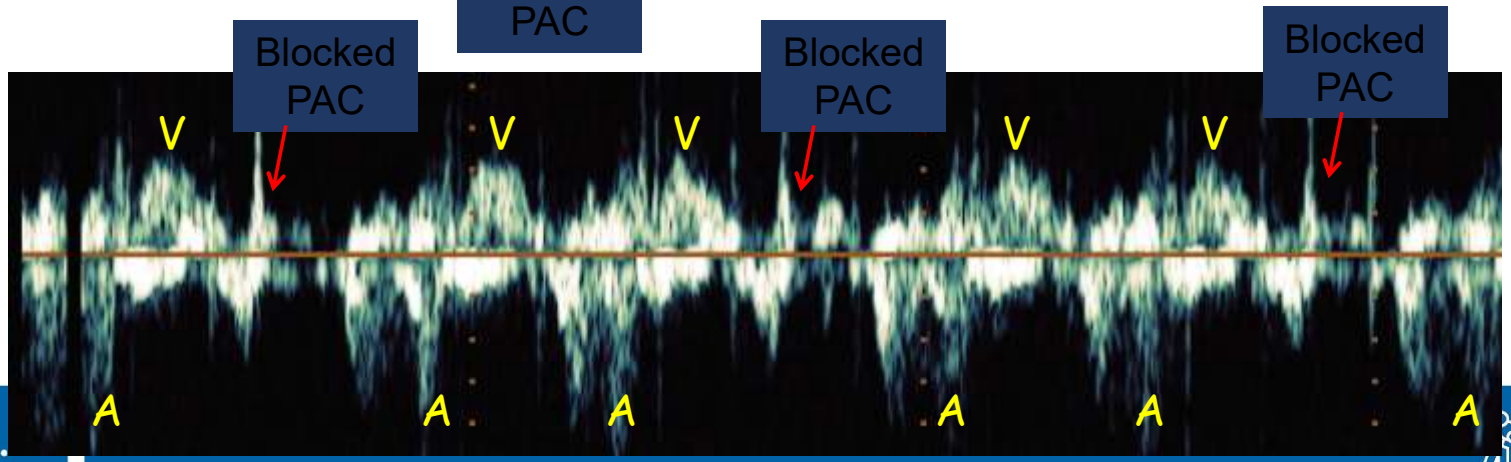


Fetal Irregular Rhythm:

Premature Atrial Contractions

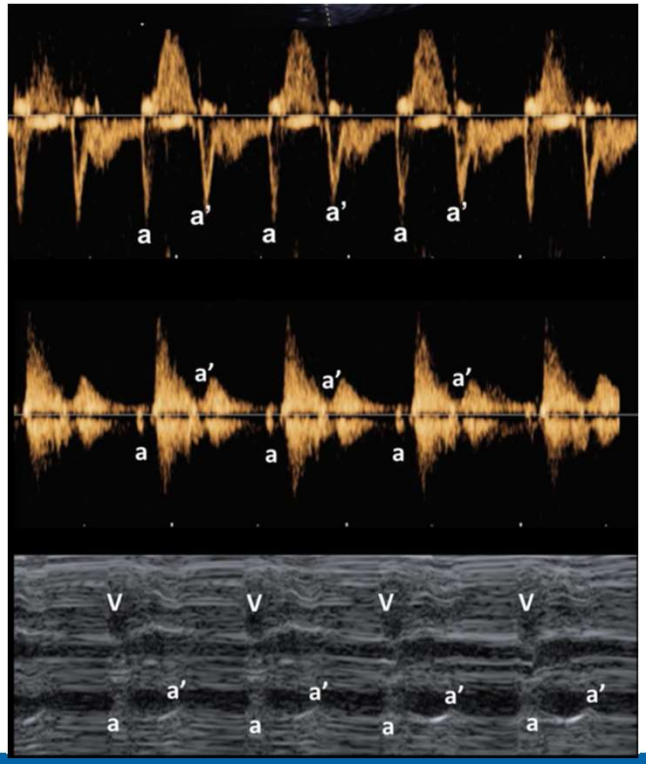
Blocked PAC

Non-conducted PACs



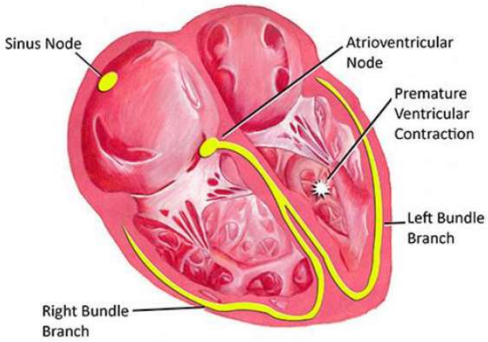
Fetal Irregular Rhythm:
Blocked atrial Bigeminy

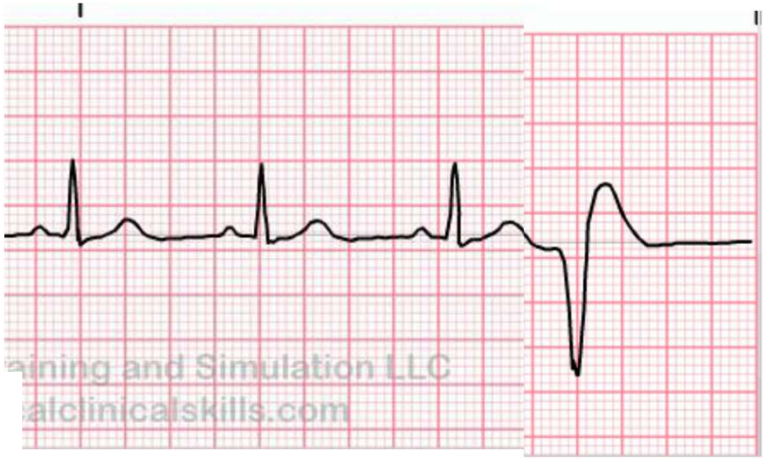
Distance Between a-a' is less than the distance from a' - a



Fetal Irregular Rhythm:

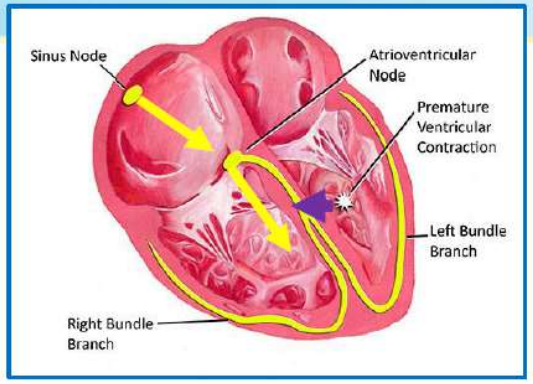
Premature Ventricular Contractions



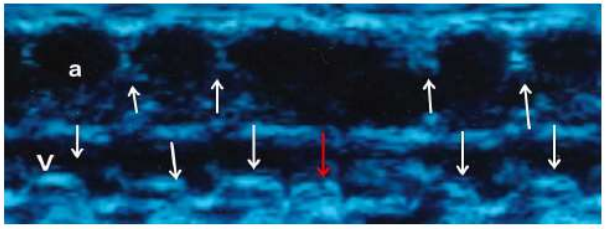


Fetal Irregular Rhythm:

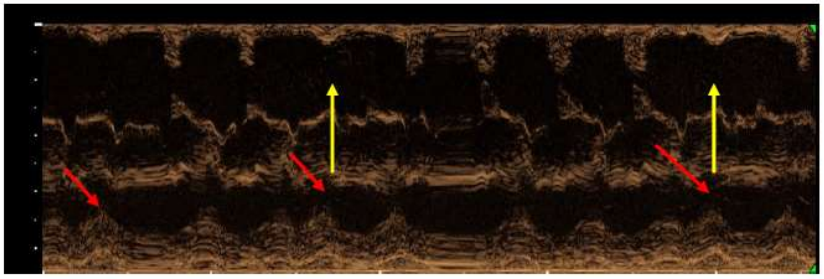
Premature Ventricular Contractions



M-mode: PVC with no retrograde conduction to atrium



M-Mode: PVC with retrograde conduction to atrium



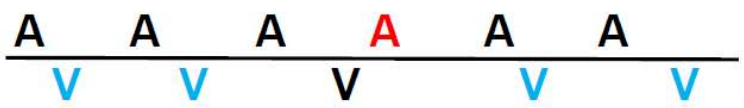
Fetal Irregular Rhythm:

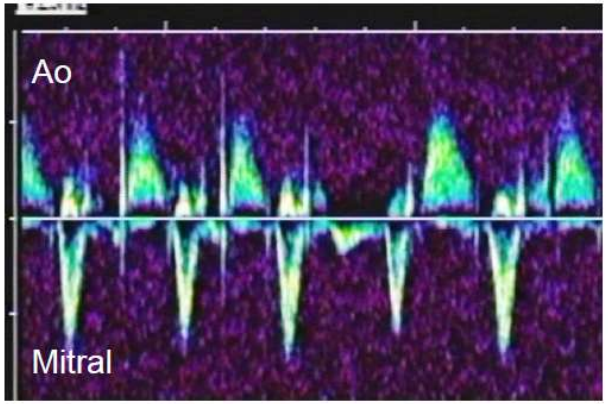
Premature Ventricular Contractions

Management of Irregular Rhythm (not second degree AV block

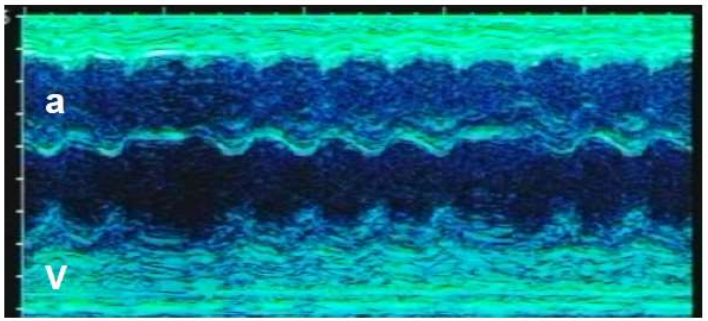
Atrial Ectopy	Ventricular Ectopy
1. Confirm normal structure	1. Confirm normal structure
2. Weekly FHR auscultation	2. Consider infectious etiology
3. Monthly fetal echo if ectopy persists	3. R/o Tumors and diverticulum, consider LQTS
4. Postnatal ECG if ectopy persists	4. Weekly FHR auscultation; consider home Doppler
	5. Monthly fetal echo if ectopy persists
	6. Postnatal ECG

Fetal Irregular Rhythm: Mobitz Type I 2nd degree block



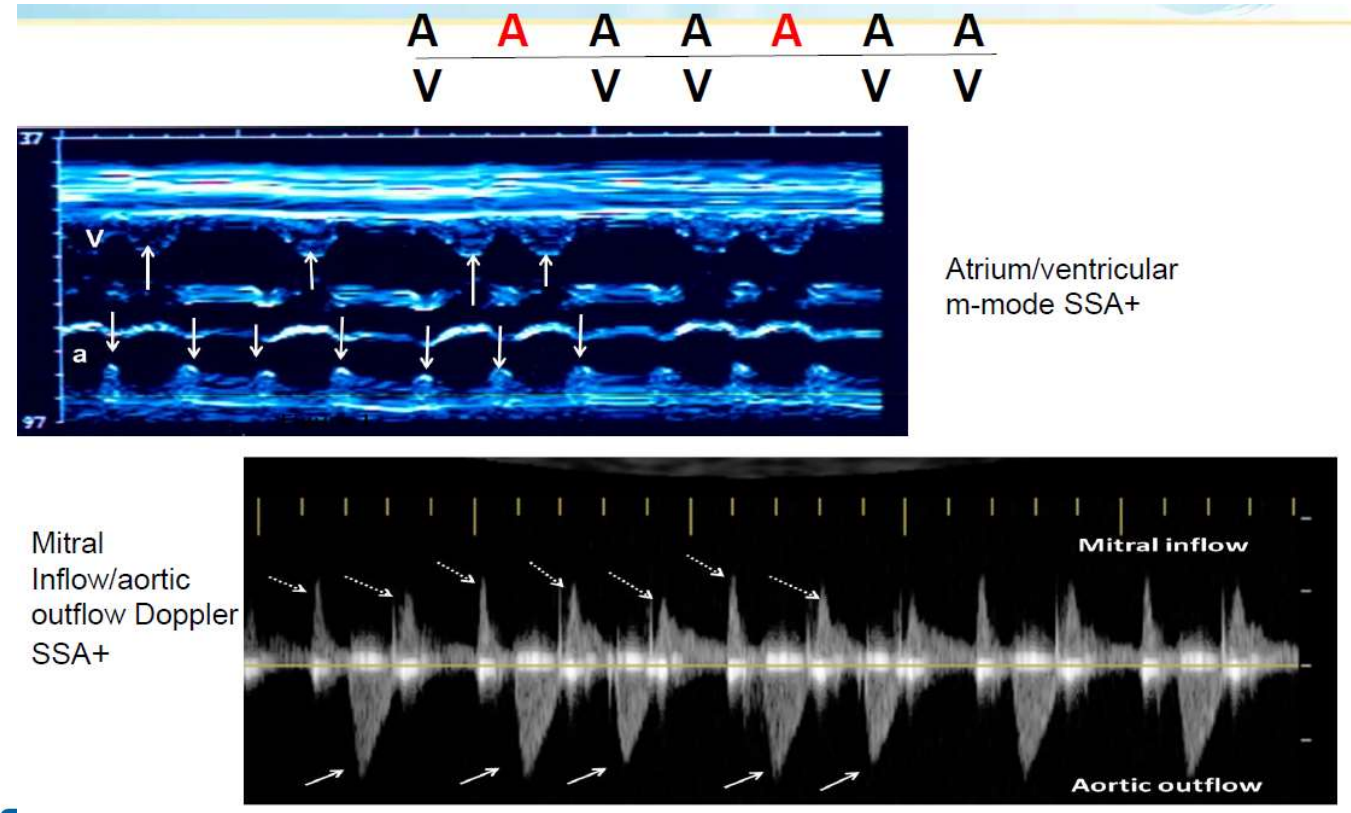


Mitral Inflow/aortic outflow Doppler SSA+



Atrium/ventricle M-mode SSA+

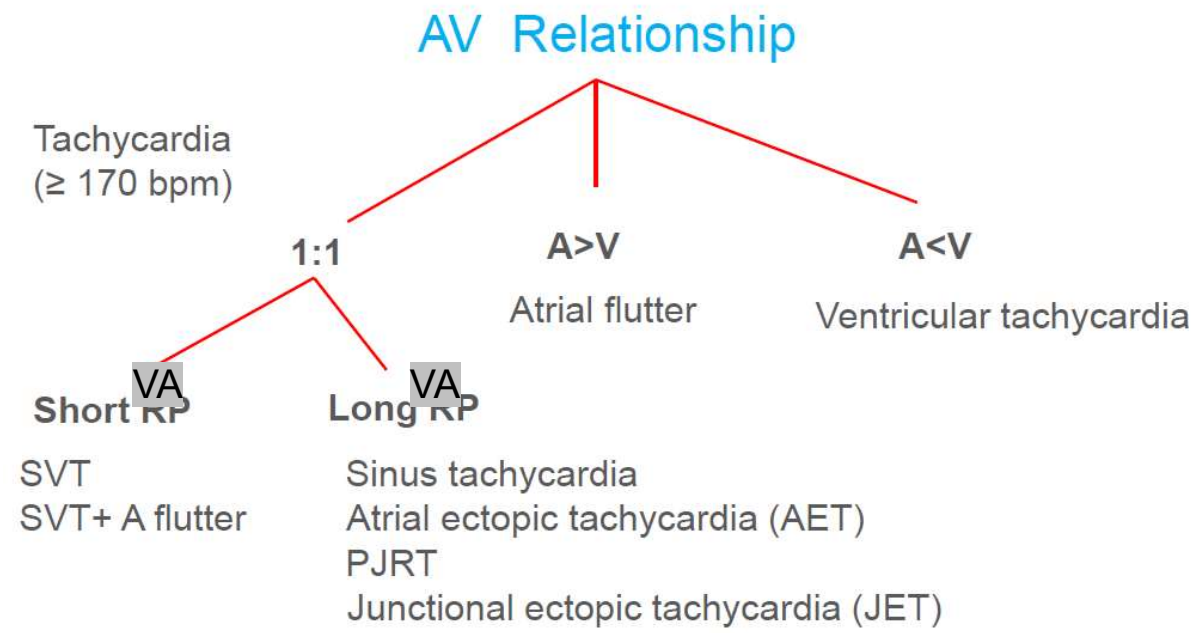
Fetal Irregular Rhythm: Intermittent Mobitz II AV block



Assessment of Fetal Tachycarrhythmia

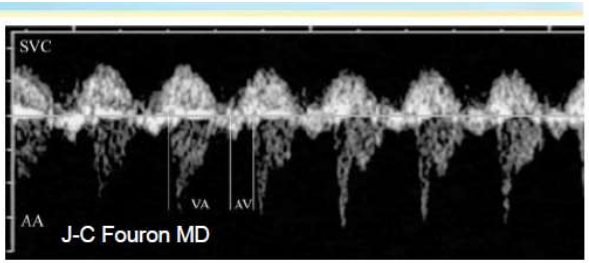
- Atrial flutter
- Ventricular Tachycardia
- SVT
  - 2 mechanisms: reentrant or automatic
- Sinus Tachycardia*

Assessment of Fetal Tachyarrhythmia

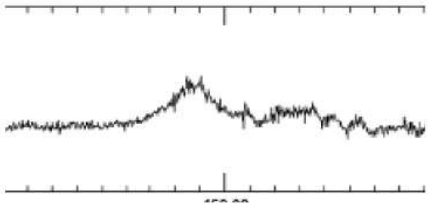


Assessment of Fetal Tachyarrhythmia

Short v. long VA Tachycardia



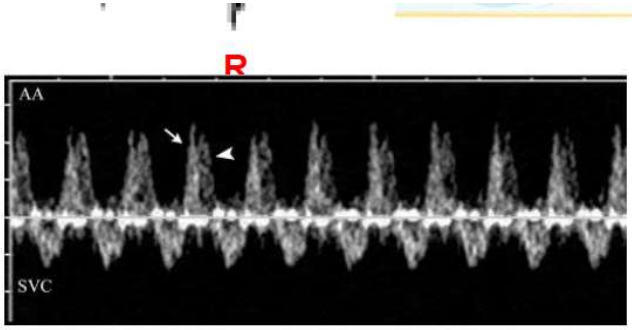
Automatic mechanism



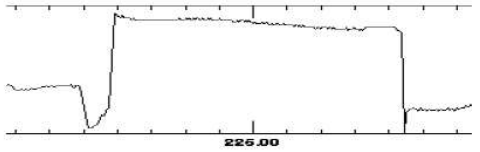
Sinus tachycardia
Atrial Ectopic Tachycardia
VT

Heart Rate

Time



Re-entrant mechanism



Junctional ectopic tachycardia(JET)
Permanent junctional reciprocating tachycardia (PJRT)
VT

Assessment of Fetal Tachyarrhythmia

Assessment of Fetal Tachyarrhythmia

Rhythm	Atrial Rate	A-A Interval	AV RELATIONSHIP	Ventricular Rate	Etiology
Sinus	180-200	Regular	1:1; long RP	180-200	Fetal anemia, infection; maternal hyperthyroid, stimulants
Atrial ectopic	180-220	Regular	Can be >1:1 If 1:1 long RP	180-220	Atrial tumors
SVT (re-entry)	220-280	Regular	1:1, short RP	220-280	Accessory connections
Atrial flutter	400-520	Irregular	2-4:1	180-260	Accessory connections
PJRT	220-280	Regular	1:1, long RP	220-280	Accessory connections
VT	110-200	Regular	Dissociated	280-340	LQTS, myocarditis, mat. SSA/SSB antibodies
Junctional ectopic					Maternal SSA antibodies

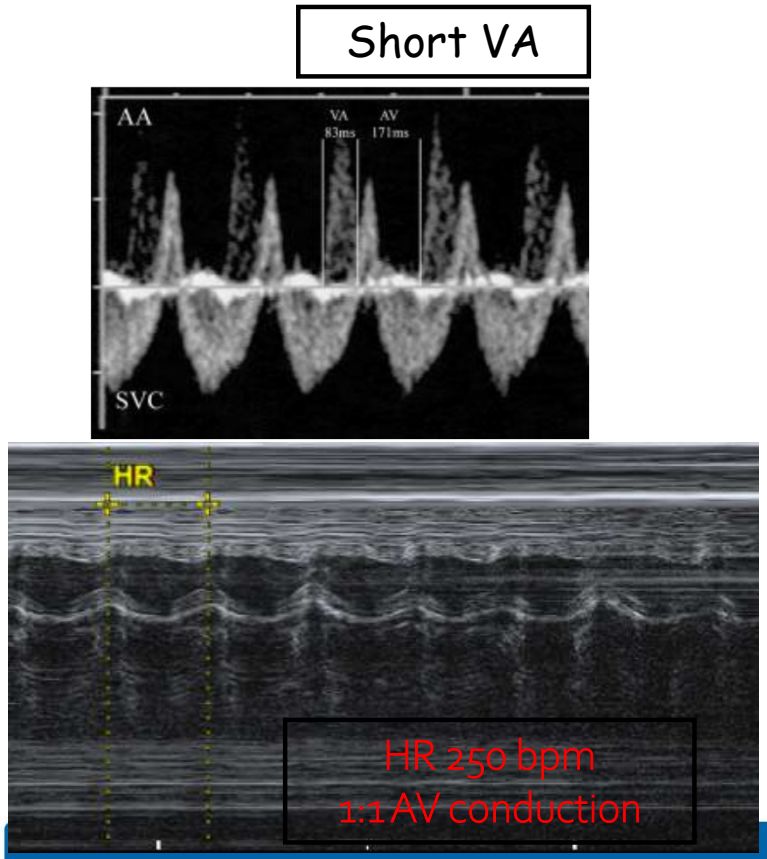
Fetal Tachyarrhythmias

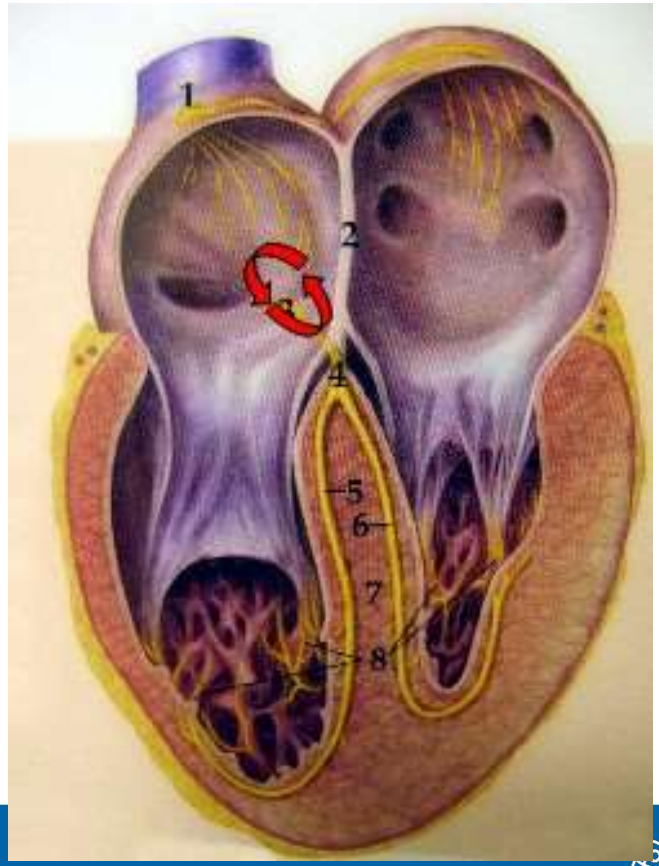
SVT with accessory AV pathway

- Most common fetal SVT
- Atrial rate 220 - >4000
- Associated with ventricular rates 220-280bpm
- Reentrant mechanism
  - initiated with an atrial premature beat
  - sudden onset/offset
  - constant rate
- 1:1 atrial-ventricular conduction
- Occurs in long and short VA
- Can be associated with Ebstein anomaly
- 10% have WPW after birth, 90% concealed pathway

Fetal Reentrant Tachyarrhythmias

SVT with accessory AV pathway





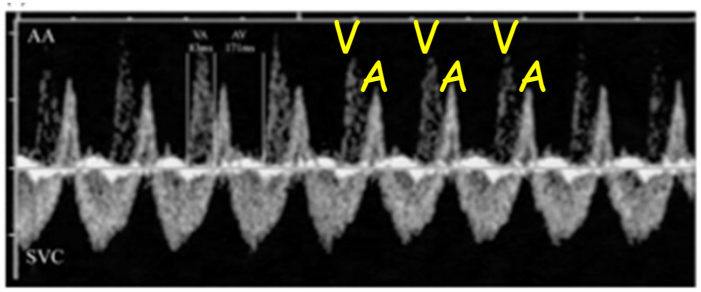
Fetal Tachyarrhythmias

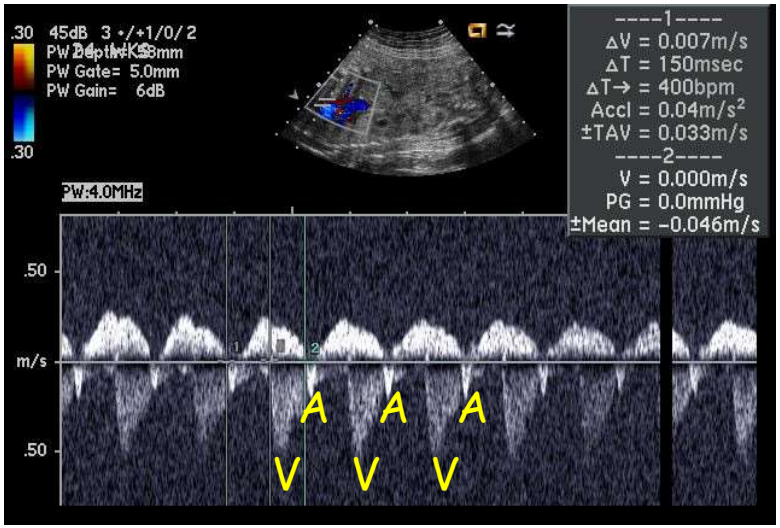
SVT with accessory pathway

Fetal ReentrantTachyarrhythmias

SVT with accessory AV pathway

SVC-Ao Dopplers
Short VA interval





SVT with 1:1 AV conduction, HR 230bpm

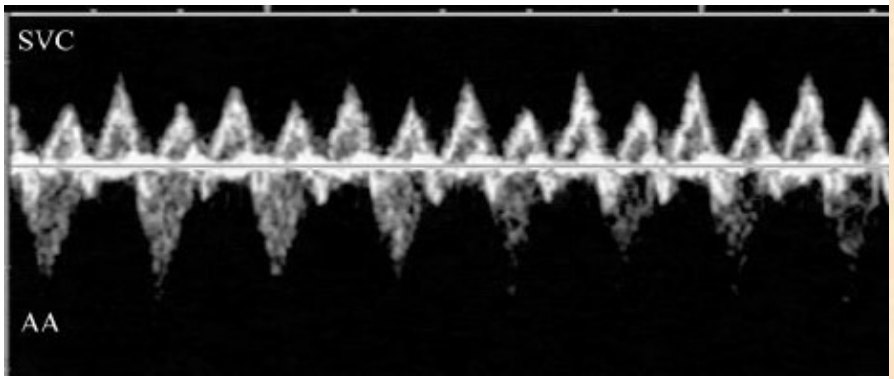
Fetal Reentrant Tachyarrhythmias

Atrial Flutter

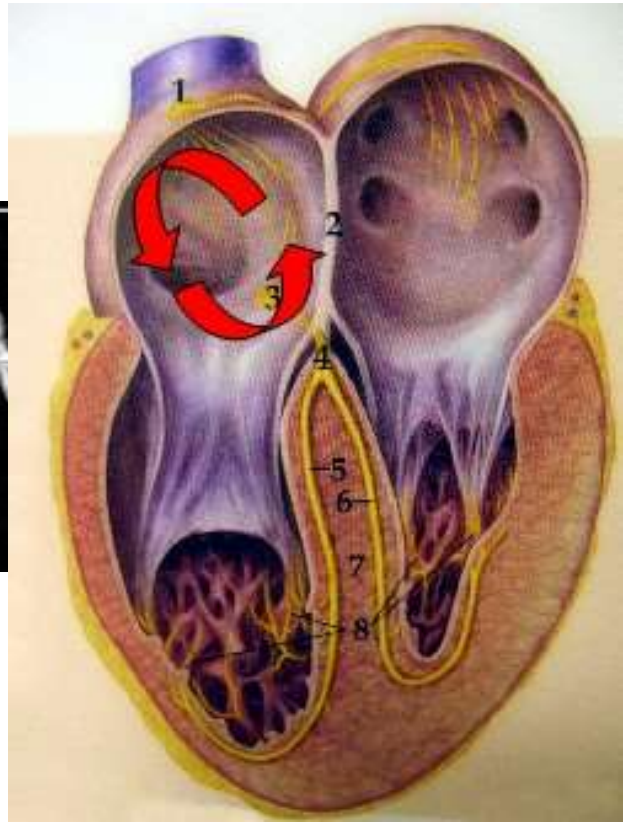
- Second most common fetal SVT
- Atrial rates of 350-500 bpm and variable ventricular rates (most often 2:1 A-V block)
- Atrial reentrant mechanism with sudden onset and offset
- Presentation often late in gestation
- Can be associated with structural CHD, particularly Ebstein's anomaly and other TVD with atrial enlargement

Fetal Reentrant Tachyarrhythmias

Atrial Flutter

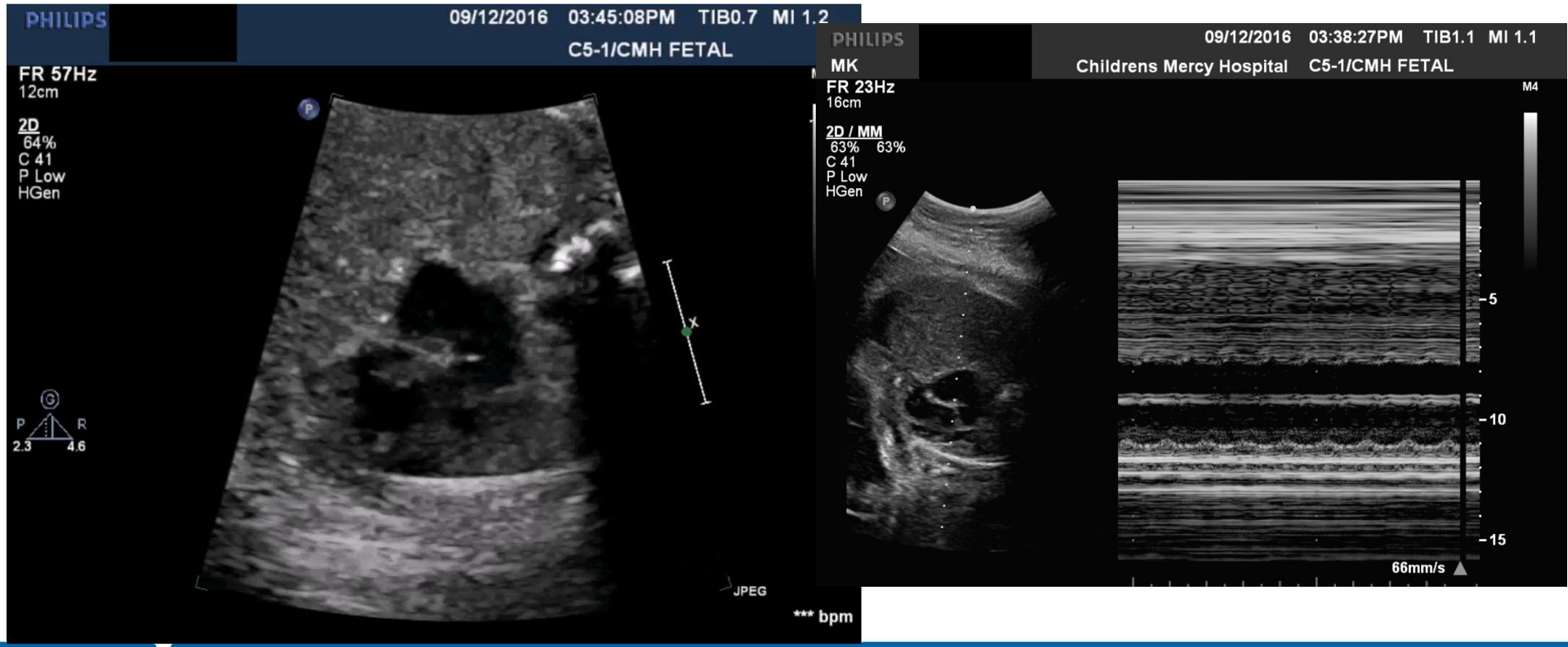


Atrial Flutter with 2:1 conduction
Atrial rate 300 bpm



Fetal Reentrant Tachyarrhythmias

Atrial Flutter

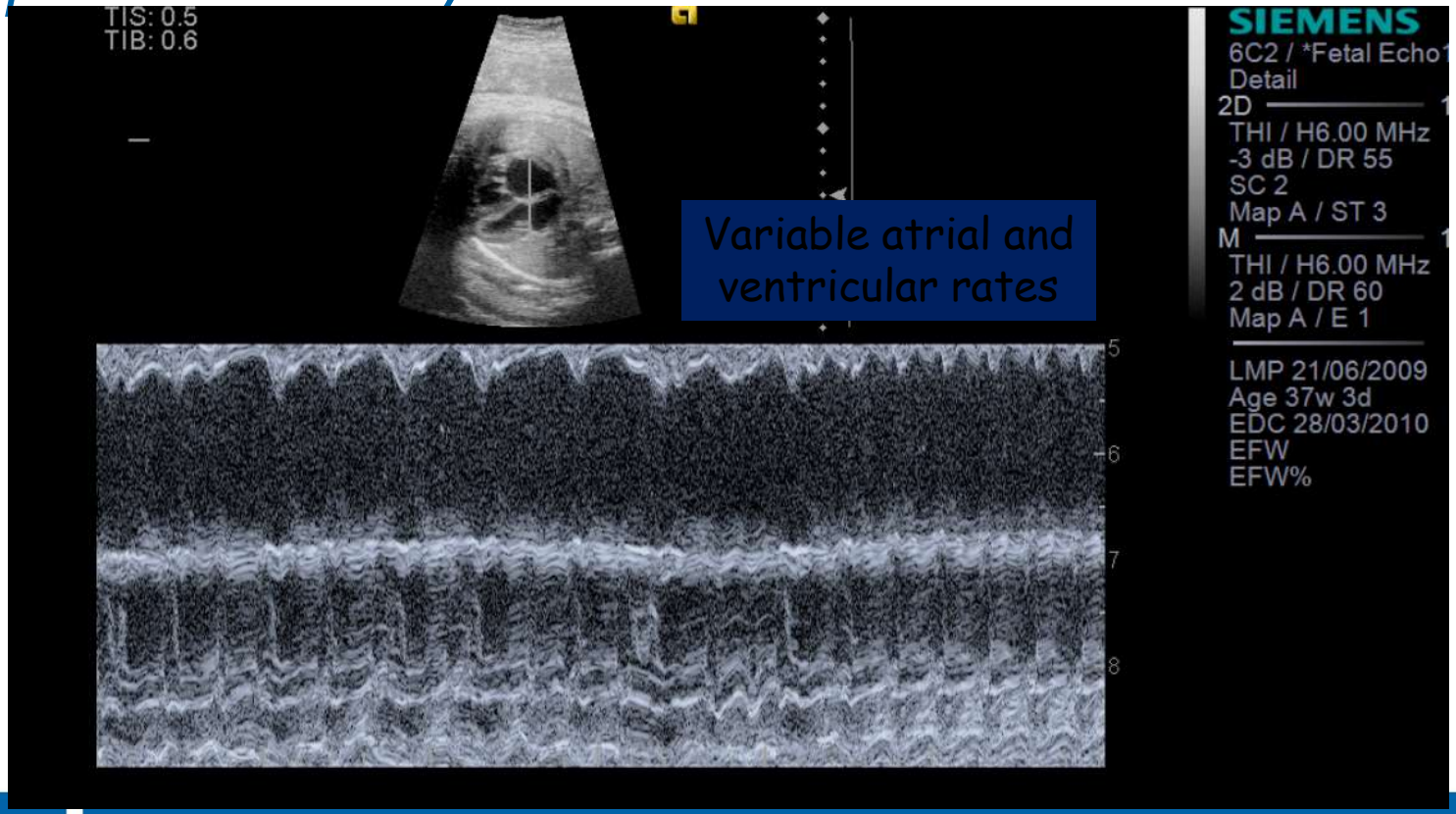


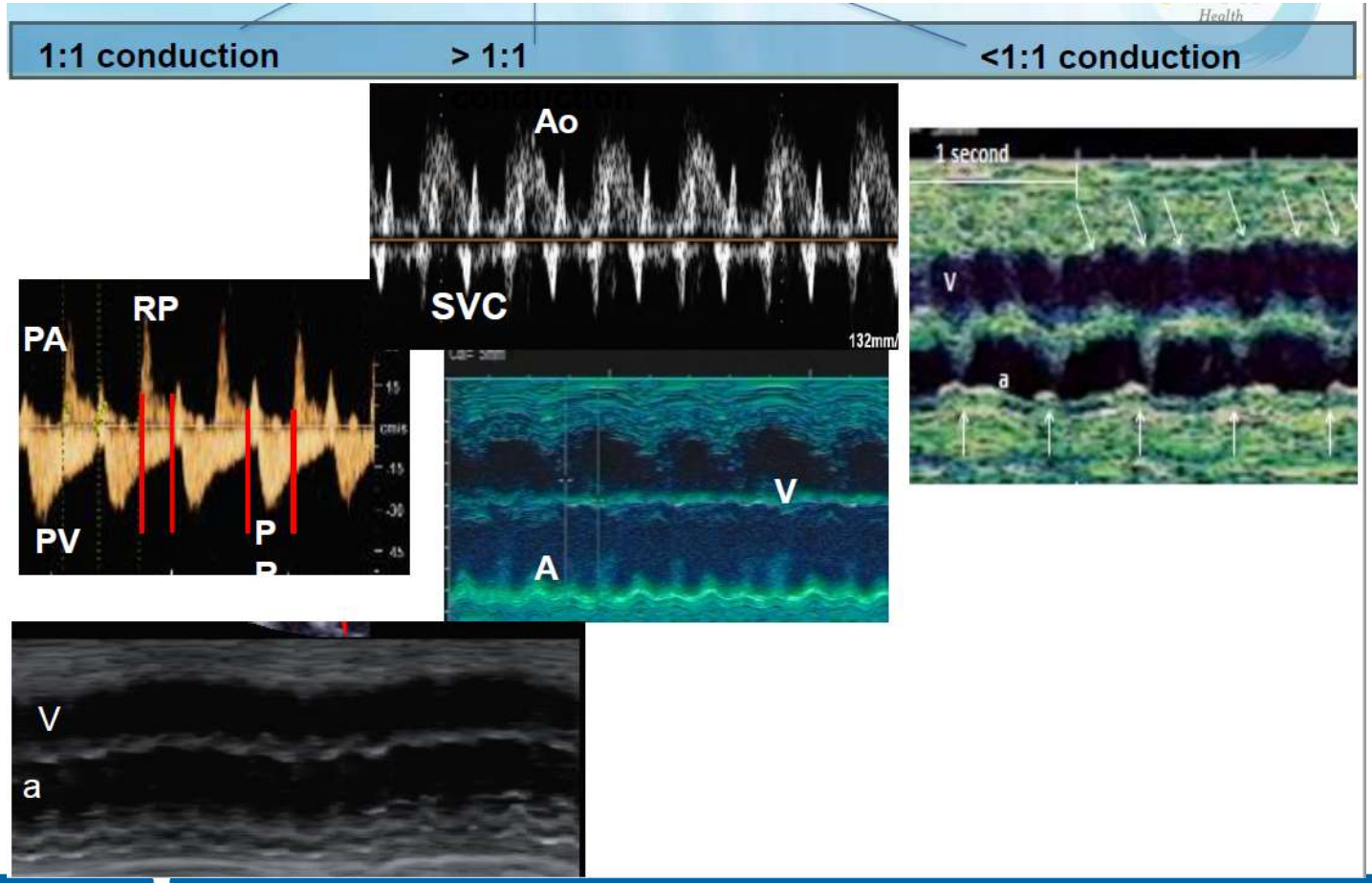
Fetal Automatic Tachyarrhythmias

- Atrial rate 180-210
- Ventricular rate 180-300
- Exhibits warm up and cool down with FHR variability
- ONLY occurs in LONG VA tachycardia
  - Sinus tach
  - JET
  - AET
- Challenging to treat

Fetal Automatic Tachyarrhythmias

Ectopic Atrial Tachycardia




1:1 conduction
> 1:1
<1:1 conduction
Ao
SVC
132mm/
PA
RP
PV
P
V
A
1 second
V
a
V
a

Etiologies of Tachycardia

- Sinus
  - Anemia
  - Hyperthyroidism
- SVT (1:1) or atrial flutter (2-3:1)
  - Accessory pathway
- VT
  - Accelerated ventricular rhythm
  - Fast VT

SVT v. VT

- SVT
  - Atrial rate = ventricular rate
  - 220-280 bpm: reentrant AVRT
  - 200-220: automatic AET
  - If atrial rate > ventricular rate think atrial flutter
- VT
  - AV disassociation
  - V rate > atrial rate
  - 180-200bpm: accelerated ventricular rhythm
    300 bpm: myocarditis, LQTS

PJRT and AET

- •18% of fetal tachycardias
- •Occurs 15-39 weeks of gestation
- •1:1 AV relation with long RP (VA)
- •FHR 170-220
- •Difficult to treat, rate control acceptable

Fetal Tachyarrhythmias

Atrial Flutter

Fetal Tachyarrhythmias

Ectopic Atrial Tachycardia

- Associated with ventricular rates of 200-300 bpm
- Automatic focus with gradual onset and offset & variable rates
- May be associated with atrial premature beats
- Longer V-A compared to A-V interval
- May be seen in CHD associated with atrial dilation and intracardiac tumors

Fetal Tachyarrhythmias

Ectopic Atrial Tachycardia

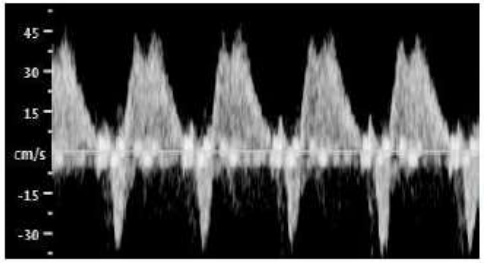
Fetal Tachyarrhythmias

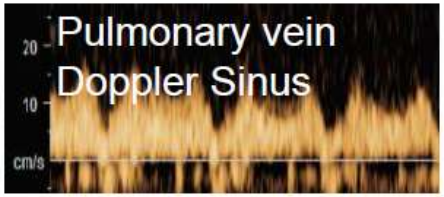
Junctional Ectopic Tachycardia

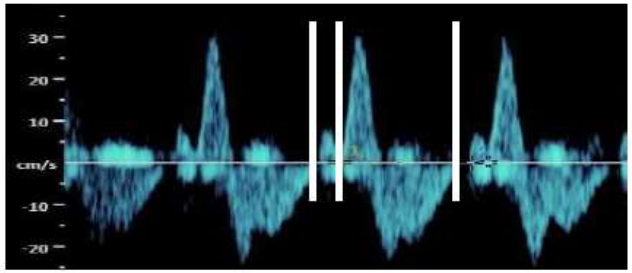
- 1% of fetal tachycardia
- Heart failure out of proportion to heart rate
- HR 150-210
- Typically automatic focus with gradual onset-offset
- There may be 1:1 V-A conduction or dissociation
- Simultaneous V and A waves (AVNRT may be in differential)
- Rare in the fetus –can spontaneously convert to sinus rhythm in months to years after birth

Echocardiographic Hallmarks of JET:

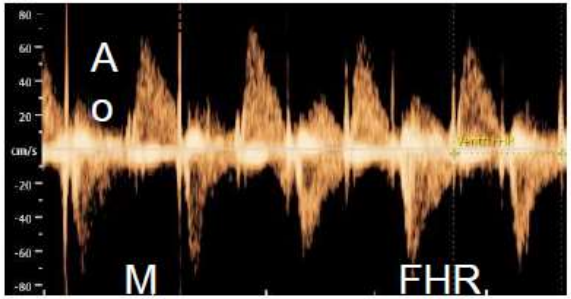
1. Abnormal flow in the systemic and pulmonary veins



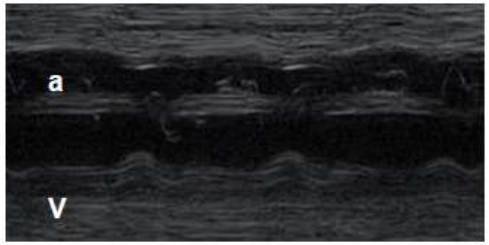


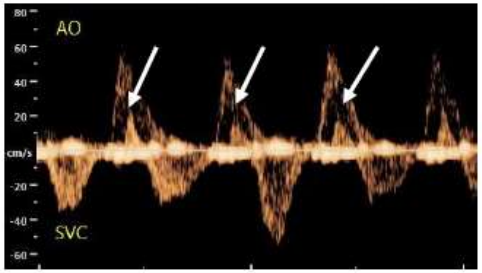


2. Monophasic AV filling even

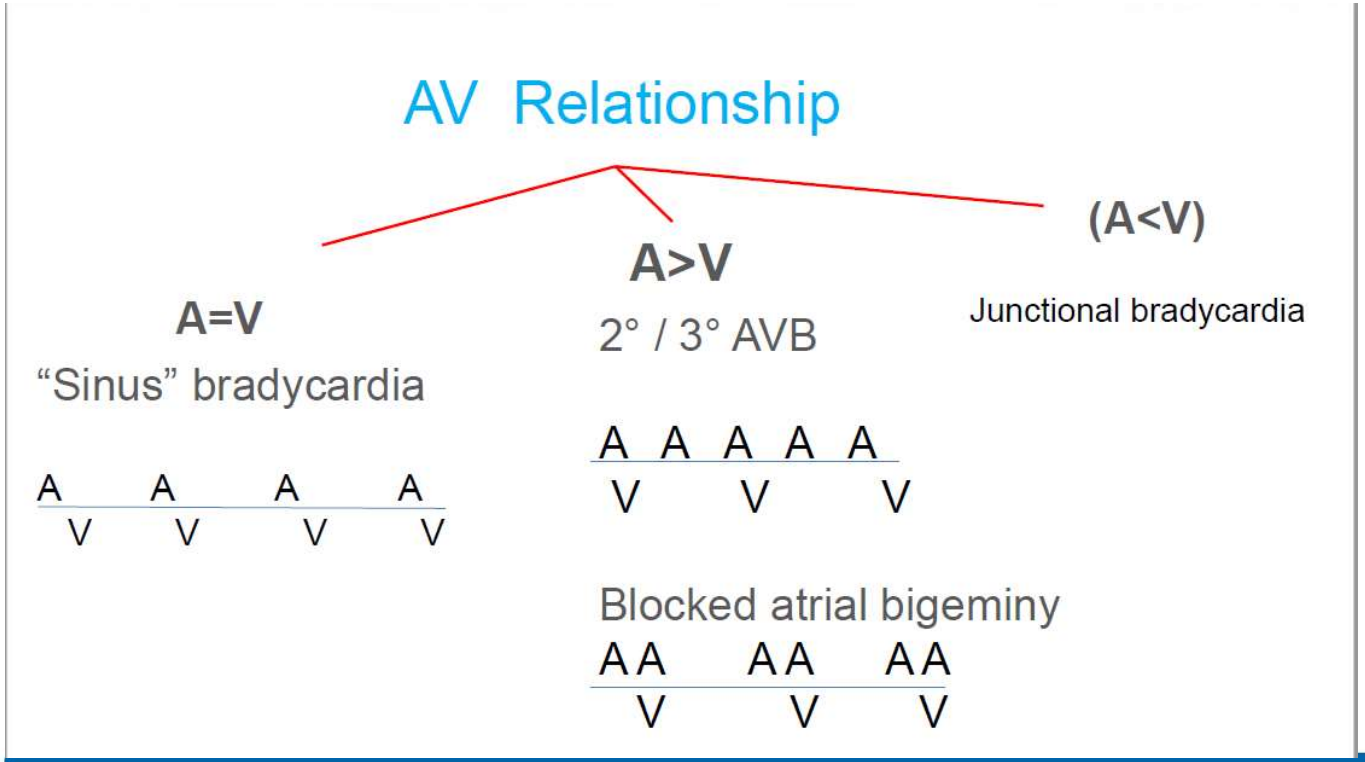


4. Simultaneous atrial and ventricular contractions





Assessment of Fetal Bradycardia

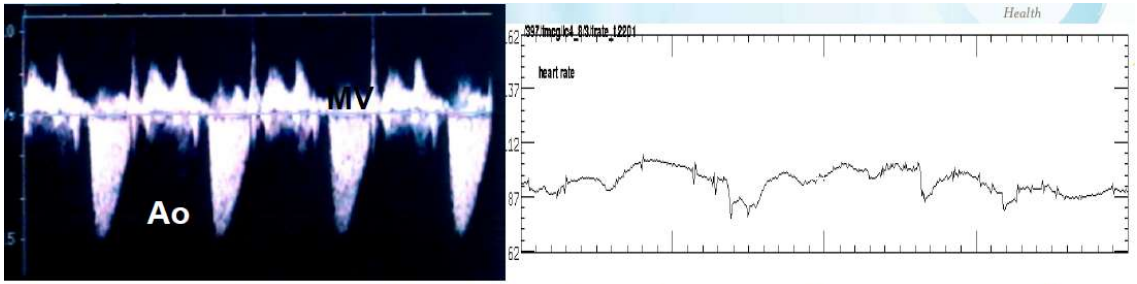


Assessment of Fetal Bradycardia: associated lesions

	Structural defect	Normal Structure
'Sinus"	• Heterotaxy • (ectopic multiple or absent sinus node)	• Chromosome/CNS abnormality • IUGR/Maternal medication/Distress • LQTS • Familial SB/"Sinus node "dysfunctior • Anti-SSA antibodies
Junctional		• Anti-SSA antibodies
AV Block	• L-transposition • Left atrial isomerism • Situs solitus and AV canal defect	• LQTS • Anti-SSA antibodies
Blocked atrial bigeminy	• Any defect	• No association

Fetal Bradyarrhythmias

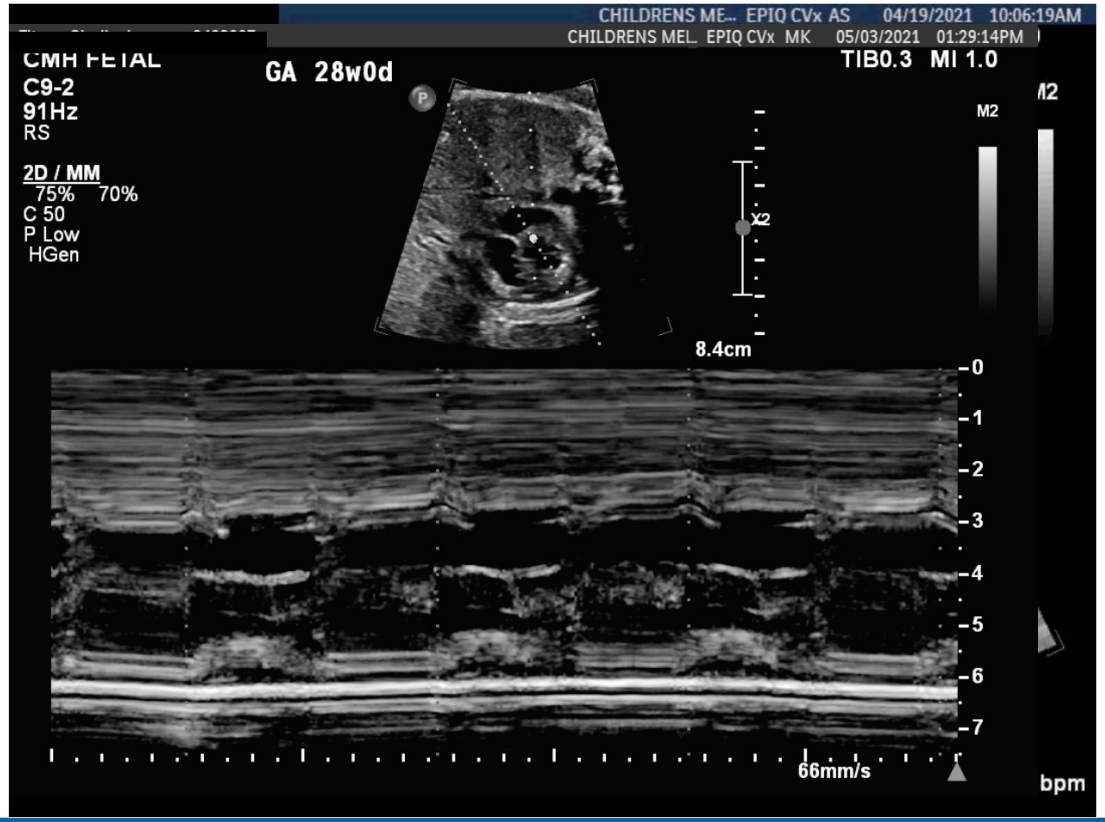
Sinus Bradycardia



- ***Genetic abnormalities*** **(dominant, recessive or sporadic mutation)**
  - Loss of function mutation in the α subunit of cardiac sodium channel (SCN5A) (Benson DW, et al. *J Clin Invest* 2003)
  - Mutation in pacemaker HCN4 ion channel (Milanesi R, et al. *New Eng J Med* 2006)
- ***Damage to a normal sinus node***
  - Viral or bacterial infection
  - Maternal SSA antibodies

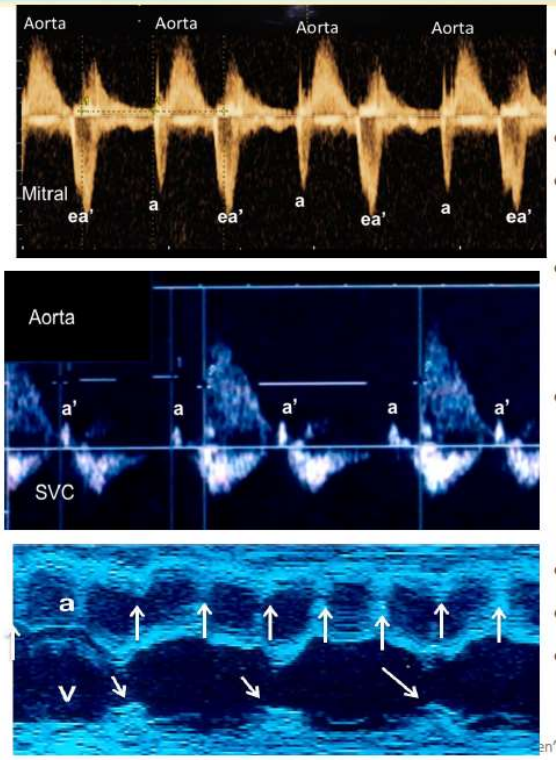
Fetal Bradyarrhythmias

Non-SSA-mediated Heart block (Heterotaxy polysplenia)



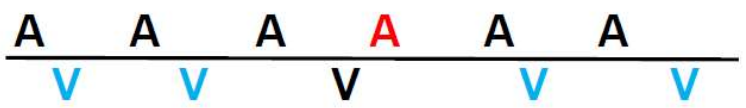
Fetal Bradyarrhythmias

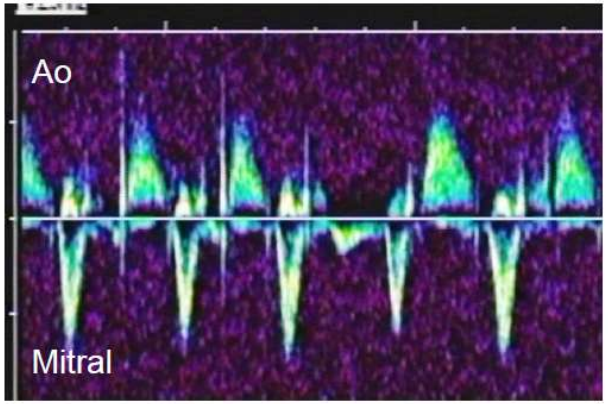
Anti SSA-mediated *Heart Block*



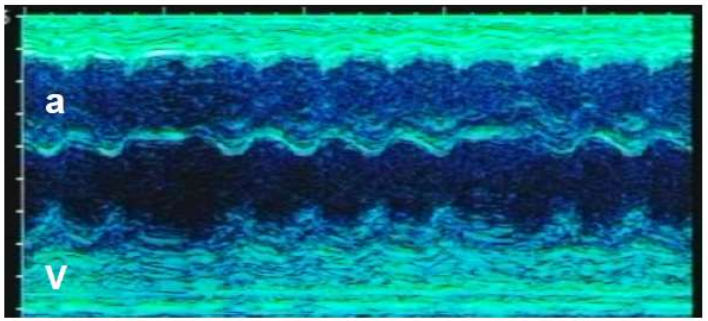
- If associated with complex structural defects can occur 13 weeks and beyond
- If mom anti-SSA +, AVB at 18-25 weeks
- 3° AVB is irreversible, 1° and 2° respond to Rx
- Occasionally first presentation is an irregular rhythm (Junctional ectopic tachycardia or type 1, 2° AV block
- CAVB has a guarded prognosis with mortality ranging from 5 % (L-transposition of the great vessels to 35% (anti-SSA +) to 95% (left atrial isomerism)
- Frequent follow-up is mandatory
- Treatment given for 2° AVB
- Treatment for 3° AVB should be considered

Fetal Bradyarrhythmia: Mobitz Type I 2nd degree block



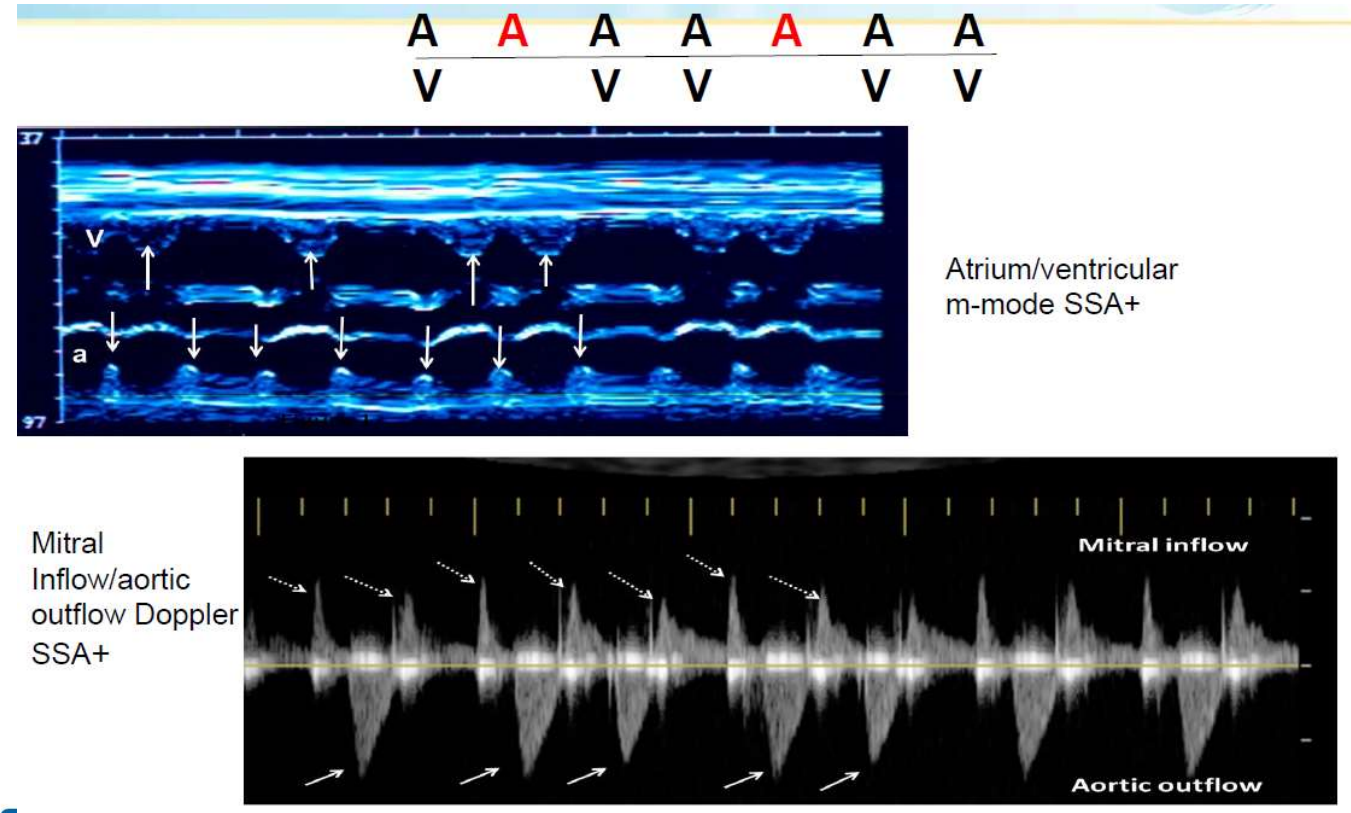


Mitral Inflow/aortic outflow Doppler SSA+

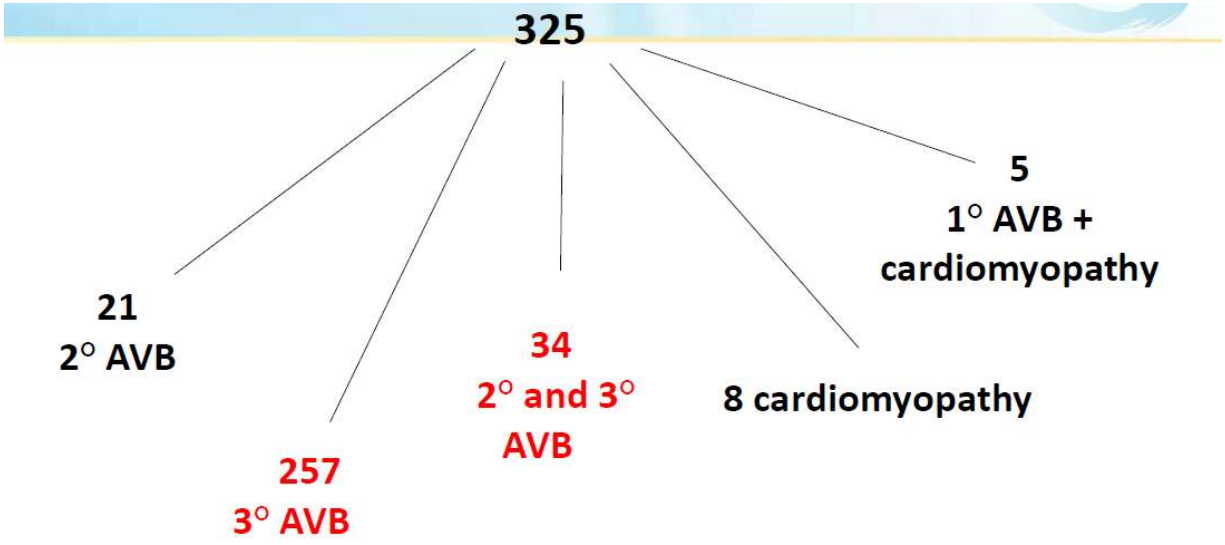


Atrium/ventricle M-mode SSA+

Fetal Irregular Rhythm: Intermittent Mobitz II AV block



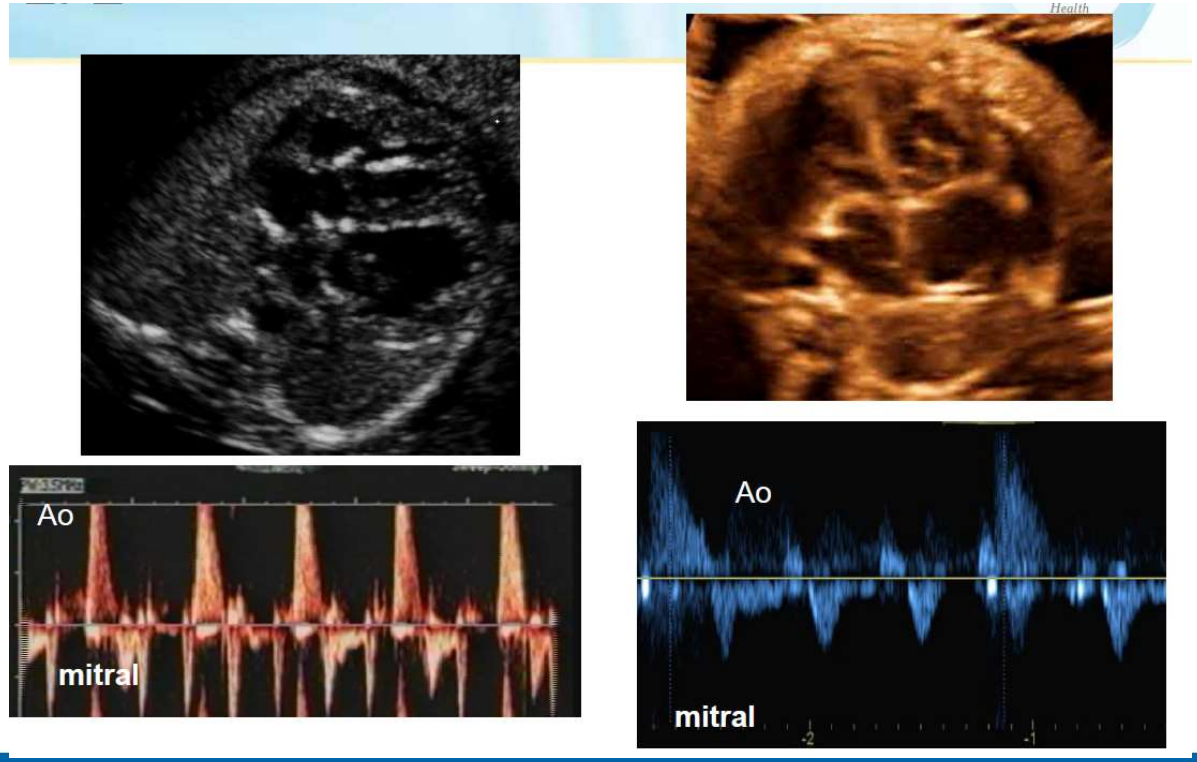
Fetal Bradyarrhythmia:
Presentation of SSA-mediated cardiac disease



96% Signature rhythm of AV Block

Izmirly PM. *Circulation* 2011

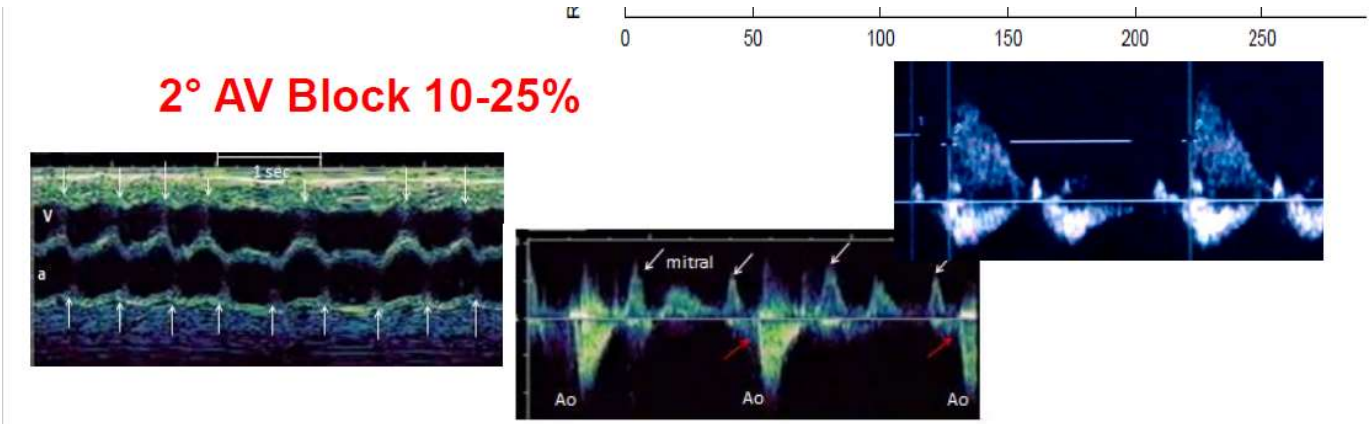
Fetal Bradyarrythmia:
Third Degree AV block and EFE

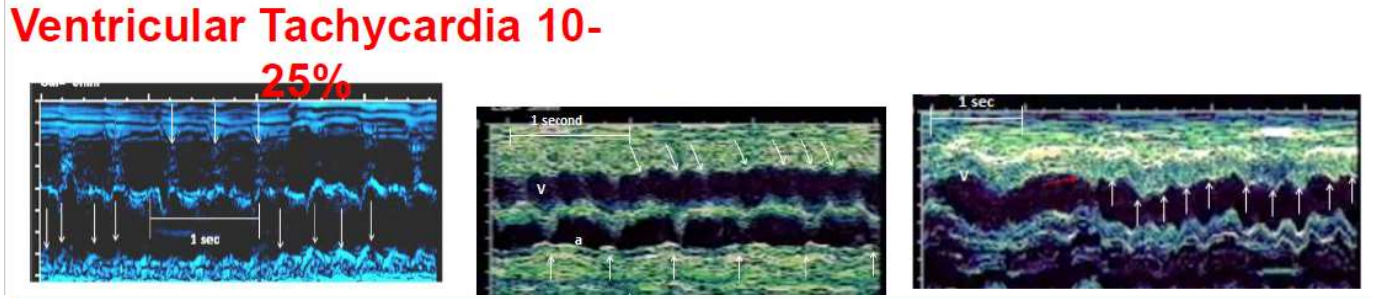


Fetal Bradyarrhythmia:
Long QT syndrome

- Genetic abnormality of the sodium and potassium channels regulating cardiac repolarization occurring in 1/2000 subjects
- •> 600 mutations in 12 susceptibility genes have been found
- •1/3 are novel mutations
- •25% are genetically elusive
- •10% of ostensibly normal IUFD and SIDS LQTS mutations
- •Only 1/7000 identified before birth

Fetal Bradyarrhythmia:
Presentation of Fetal LQTS

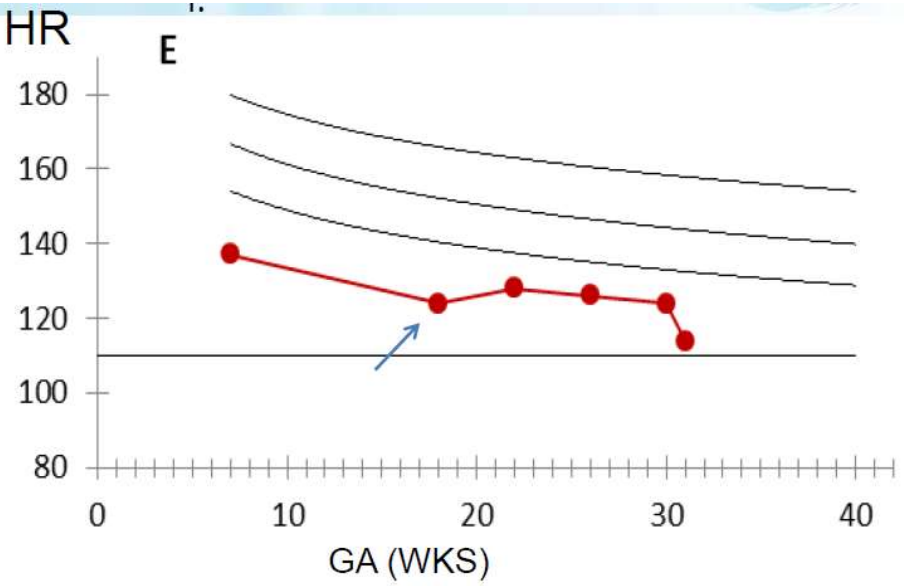




Fetal Bradyarrhythmmia Case Report:

"FHR a little lower than what I see at this GA"

- No family history of LQTS
- Parents QTcs normal
- No syncope, cardiac arrest
- OB history positive for 1 miscarriages at 23 weeks



Infant found to have KCNQ1 mutation G314D

Stayed Tuned for Parts 2 and 3…

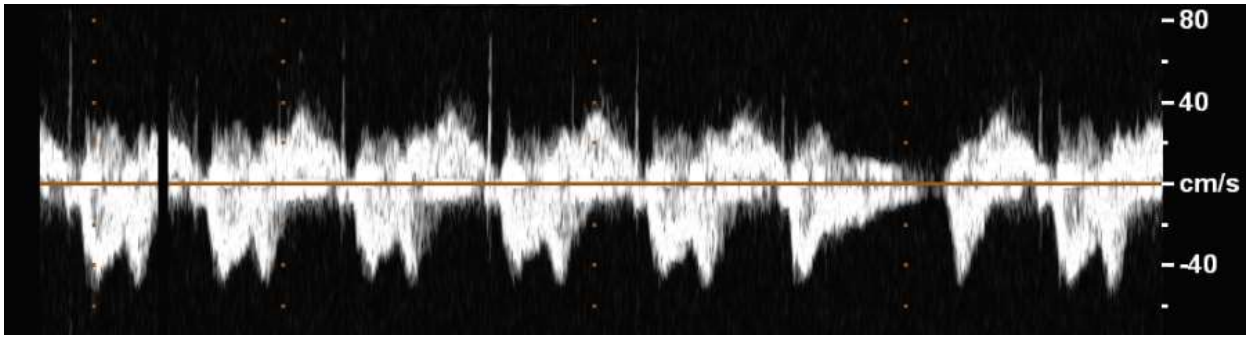
- January: Tachycardia
- February: Bradycardia

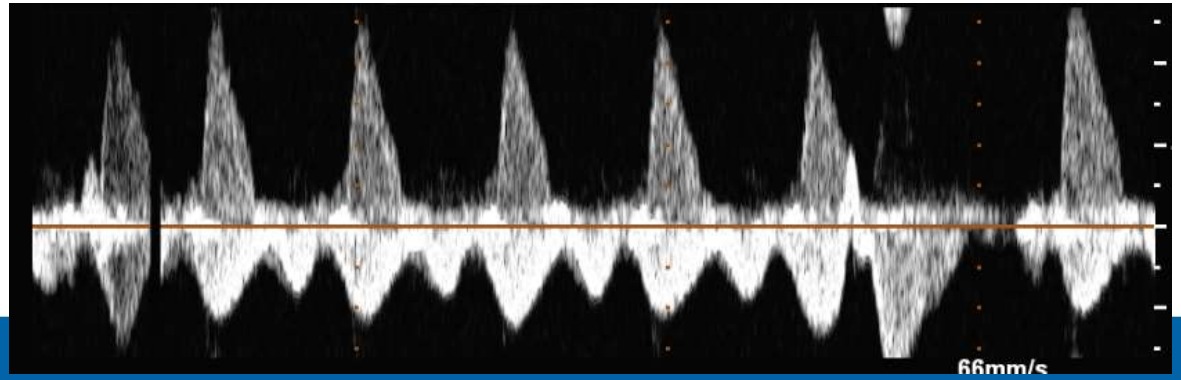
Acknowledgements

- Lisa Howley, MD and Bettina Cuneo, MD for some slides and figures
- Children's Mercy Fetal Health Center sonographers, nurses, and physicians
  - Case presentations and images made possible through their excellent work
- The families who allow us to care for them

Fetal Tachyarrhythmias:

Where is this Doppler tracing? What is the arrhythmia?

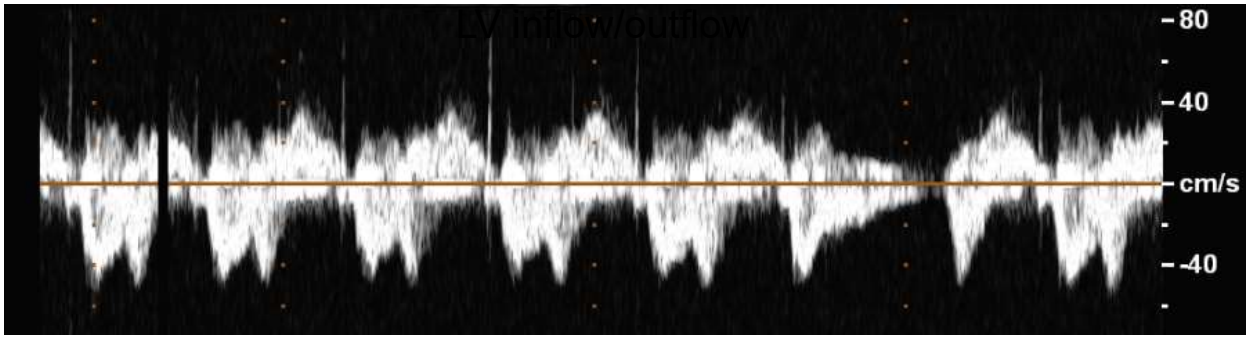


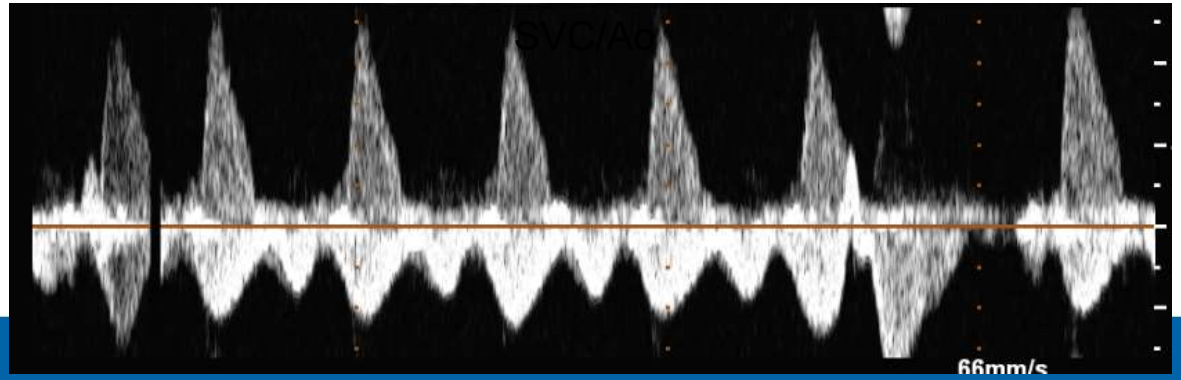


Fetal Tachyarrhythmias:

Where is this Doppler tracing? What is the arrhythm

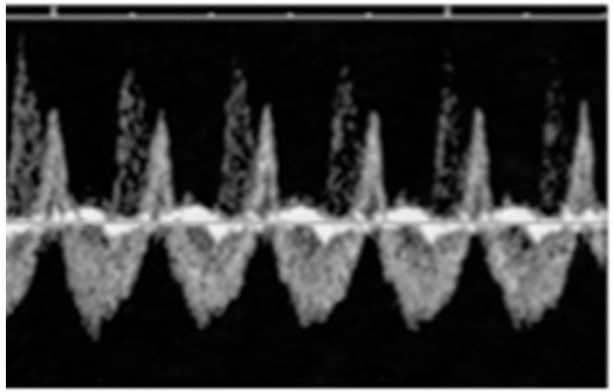
Blocked PACS





Fetal Tachyarrhythmias:

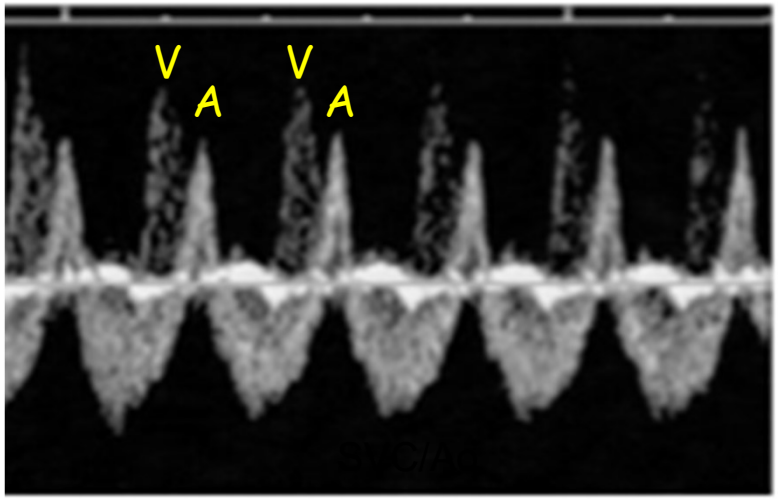
Where is this Doppler tracing? What is the arrhythmia?



Fetal Tachyarrhythmias:

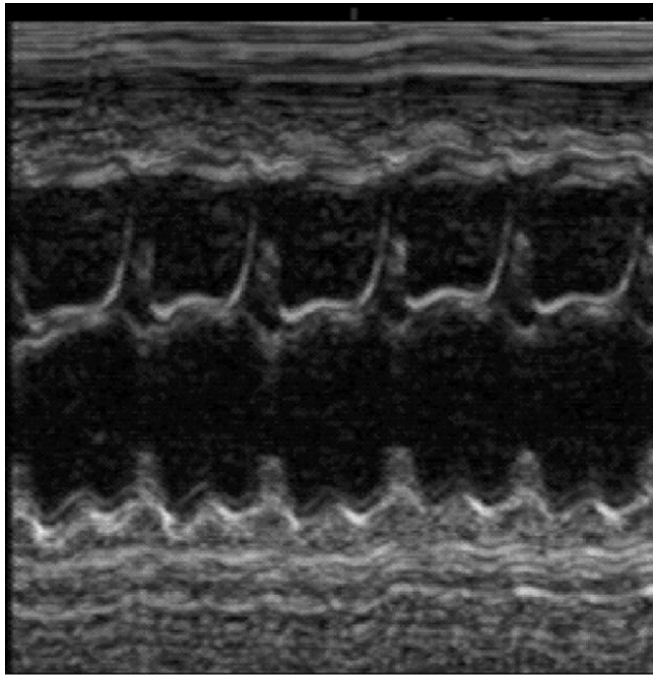
Where is this Doppler tracing? What is the arrhythmia?

Short VA tachycardia c/w accessory pathway



Fetal Tachyarrhythmias:

What is the arrhythmia?



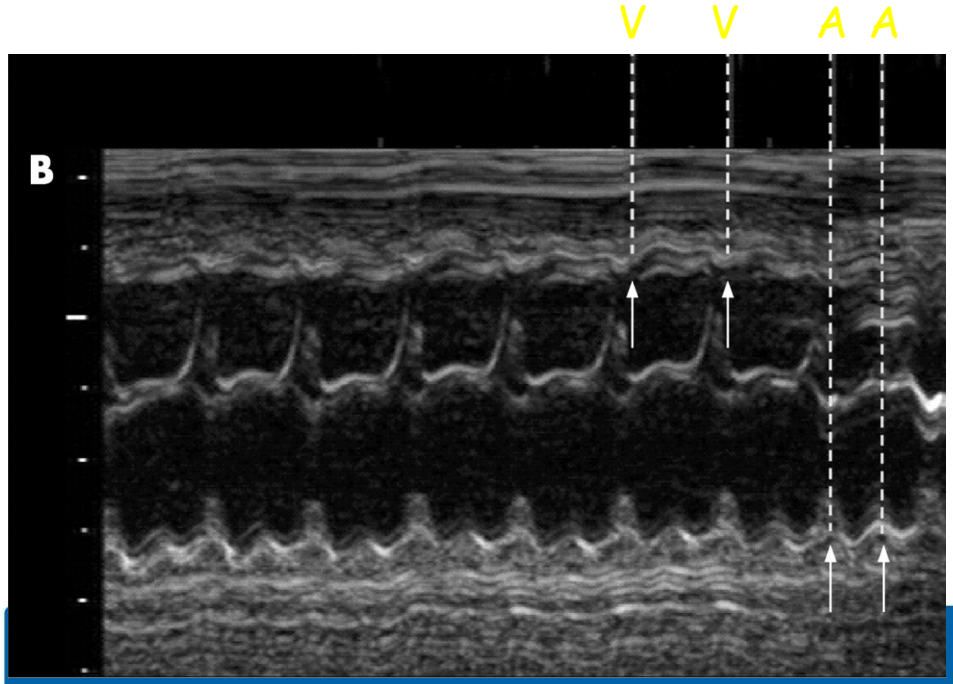
Ventricle

Atrium

Fetal Tachyarrhythmias:

What is the arrhythmia?

Atrial Flutter 2:1



Atrial rate 480 bpm

Ventricular rate 240 bpm

Fetal Supraventricular Tachycardia
Why Treat?

- Risk of evolving hydrops – SVT impedes ventricular filling due to short diastolic time resulting in increased venous pressure

- Risk of morbidity – premature delivery, tachycardia-induced cardiomyopathy

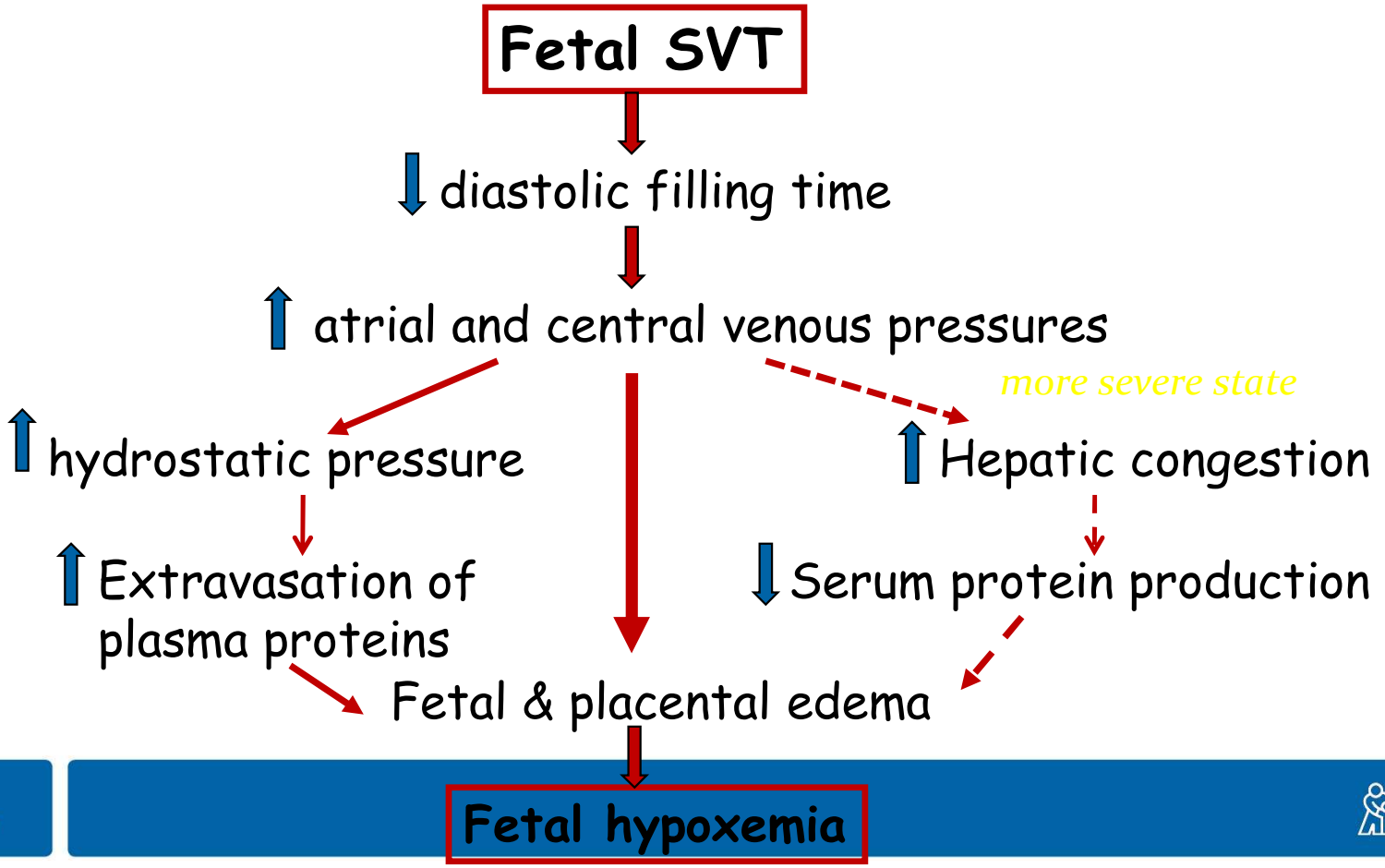
- Difficulty in peripartum monitoring – with incessant fetal SVT necessitating C/S in many institutions

Fetal Supraventricular Tachycardia

Evolution of Hydrops

Fetal Supraventricular Tachycardia

Evolution of Hydrops



Fetal Supraventricular Tachycardia *Why Treat?*

- Mortality associated with fetal hydrops
  - 23-55% without control (*Simpson & Sharland Heart 1998, Hansmann UOG 1991, Frohn-Mulder Prenat Diag 1995*)
  - 8-10% despite control (*Simpson Heart 1998, Krapp Heart 2003*)

- Morbidity associated with fetal hydrops
  - Preterm delivery
  - Prolonged ventilation
  - Prolonged NICU stay
  - Neurological insults in up to 40% (*Oudijk et al UOG 2004*)

Fetal Supraventricular Tachycardia *Treatment Facts*

- No universally applied treatment strategy exists
- No one medication does it all
- Short V-A tachycardia
  - Transplacental therapy successful for most fetuses, even with hydrops - success rates reported at 75-85%
  - Hydropic fetuses require median of 2 medications & take longer to respond *(Van Engelen et al JACC 1994)*

Fetal Supraventricular Tachycardia *Treatment Facts*

- Long V-A tachycardias more resistant to therapy (*Jaeggi, Fouron et al Heart 1998*)
- Atrial flutter - variable treatment success (*Jaeggi, Fouron J Peds 1998; Krapp et al Heart 2003*)
- Rare reports of fetal demise in the absence of CHF/hydrops with use of more potent antiarrhythmics (Flecainide, Sotalol and Amiodarone)

Fetal Supraventricular Tachycardia *Treatment Considerations*

- Transplacental drug therapy:
  - There are TWO patients – must think about drug effect on mother and fetus
  - Different drug levels in the fetus and mother
    - Pregnancy causes delayed maternal gastric emptying & increased renal clearance
    - In the setting of CHF and placental edema, limited ability of the medication to cross the placenta

Fetal Supraventricular Tachycardia *Who should we treat?*

Primary aim of therapy is to either ameliorate or prevent hydrops through cessation of the arrhythmia or reduction in ventricular rate

The "at risk fetus" *(Naheed et al JACC 1996)*

- Incessant SVT (>50% of exam)
- Earlier presentation (<32 weeks)
- Structural heart disease
- Evidence of cardiovascular compromise already present
- Rates >220 bpm

Fetal Supraventricular Tachycardia

Treatment Controversies

- Who & when to treat
  - Hydropic fetus – definitely yes
  - Nonhydropic but at-risk for hydrops - yes
  - Low risk for hydrops- unclear

- How to treat
  - Late gestation – deliver with postnatal treatment vs. prenatal treatment
  - Maternal/placental vs. direct fetal therapy
  - Medication options

Fetal Supraventricular Tachycardia

Maternal Digoxin Therapy

Loading Dose: 250-375 mcg PO 4x/day x 1 day
Maintenance Dose 250 mcg PO BID
Maternal Therapeutic level: 1.5-2.5ng/ml

- Fetal levels
  - without hydrops -70-100% of mother's level
  - with hydrops – very poor placental transfer
- Success with conversion of SVT :
  without hydrops 50-70%
  with hydrops <20%
- Not as effective in A flutter & long VA SVT

Fetal Supraventricular Tachycardia

Additional Antiarrhythmics

Sotalol

Flecainide

Amiodarone

Fetal Supraventricular Tachycardia

Maternal Sotalol Therapy

No loading dose

Maintenance dose 120-160 mg PO BID → TID if needed

No drug levels

- Class III agent (prolongs repolarization & action potential) & β blockade
- Fetal levels 70-100% of maternal levels within 48-72 hours
- Potential negative inotropic effect
- Proarrhythmic affect observed - QT prolongation
- Success with conversion of SVT :
  - without hydrops 75-90%
  - with hydrops 60-75%

Fetal Supraventricular Tachycardia

Maternal Flecainide Therapy

No loading dose

Maintenance dose 100mg po q 8 hours

Maternal Therapeutic level: 0.4-1mcg/ml

- Class Ic agent – fast sodium channel blockade (slows the conduction velocity in most cardiac pathways)
- Good transplacental transfer – fetus achieves 80% of maternal levels +/-hydrops
- **Proarrhythmic effect** observed (7.5%)
- Success with conversion of SVT (average 4-7 days);

Fetal Supraventricular Tachycardia

Maternal Amiodarone Therapy

Loading Dose: 1200mg IV infusion over 1 day or 200 mg PO Q4 hours over 1 day

Maintenance Dose 200mg PO Q6-8 hrs

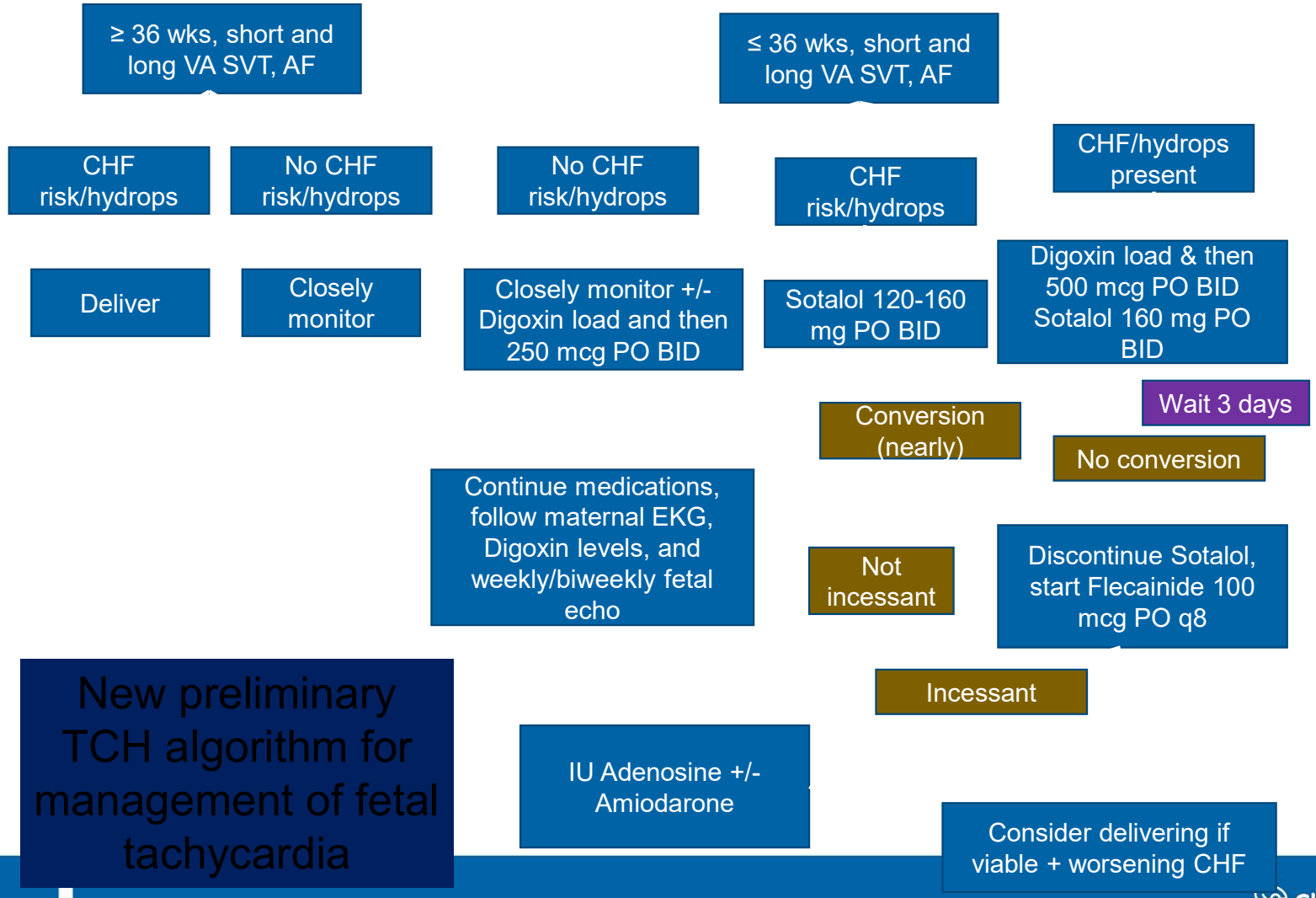
Maternal Therapeutic level: 1-2mcg/ml

- Class III agent (prolongs repolarization & action potential)
- Long elimination half-life (1-3 months)
- Fetal levels -10-40% of maternal levels, less with hydrops
- Less negative inotropic effect
- Success 50-60% *(Ito Peri Clin 1994)*, 93% reentrant SVT and 33% A flutter *(Strasburger Circ 2004)*
- Side Effects – hypothyroid (15-20%), ?neurodevelopmental delay

Fetal Supraventricular Tachycardia
Maternal Pretreatment Assessment

- Adult Cardiology assessment of mother:
  - Baseline EKG +/- echocardiogram
  - Baseline serum electrolytes
  - Family history

- Initiation of antiarrhythmic (IV or PO):
  - Maternal admission for telemetry until a steady/effective dose is achieved
  - Routine daily fetal evaluation for evidence of CHF/hydrops
  - Daily EKG until effective dose achieved



New preliminary TCH algorithm for management of fetal tachycardia

Fetal Supraventricular Tachycardia

Treatment Strategy

- If ≥36 weeks with fetal SVT:

  If CHF risk or CHF is present - deliver in 3° care center

  If no clear CHF risk (intermittent brief SVT, no CHD) monitor until delivery, deliver in 3° care center

- All fetal SVT without clear CHF risk and at 32-36 weeks
  - Monitor frequently until delivery
  - +/- initiation of Digoxin therapy