Introduction to Fetal Medicine

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Fetal Cardiology

Important in evaluation of high risk pregnancies.
Information obtainable in > 95% of patients attempted.
Allows for assessment of developmental cardiovascular physiology.

Best clinical management depends on strong collaboration between subspecialists:

- perinatology
- genetics
- obstetrics
- neonatology
- ultrasonography
- pediatric cardiology
- internal medicine
- cardiac surgery

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Fetal Circulation

Placenta: low resistance circuit, organ of gas exchange, nutrient supply.

Lungs: high resistance, non-functional, breathing important.

Brain development is primary!

Shunt pathways:
- Foramen ovale
- Ductus arteriosus
- Ductus venosus
Fetal Circulation

Shunt Pathways:

**Ductus venosus**: bypasses fetal liver

**Foramen ovale**: R-L shunt across atrial septum

**Ductus arteriosus**: bypasses high resistance (non-aerated) lungs
Transitional circulation

- Separation from low resistance placenta
  - $\Rightarrow$ increased SVR
  - low flow constricts ductus venosus
- First breath expands lungs
  - $\Rightarrow$ decreased PVR
  - increased pulmonary blood flow
  - increased LA pressure closes PFO
  - increased PaO2 $\Rightarrow$ constricts PDA
Fetal Echocardiography

First observations of normal cardiac anatomy utilizing M-mode by Winsberg in 1972.


High resolution cross-sectional scanners allow real-time directed utilization of:

- Two-dimensional imaging
- Pulsed & color flow Doppler
- M-mode
Diagnostic capabilities

- Cardiac ultrastructure
  2 - dimensional, M - mode
- Vascular & intracardiac flow patterns
  Color, pulsed & continuous wave Doppler
- Cardiac rate and rhythm
  M – mode & Doppler evaluation of electromechanical events.
- Myocardial function
Indications

**Fetal factors:**

- IUGR
- Arrhythmia
- Hydrops fetalis
- Abnormal genetic screen
- Extracardiac anomalies – nuchal translucency
- Diminished fetal movement
- Abnormal 4-chamber screen
Indications

Maternal factors:

CHD (risk increases from ~1% to 4-5%)

Poly/oligo - hydramnios

Diabetes

Collagen vascular disease

Teratogen exposure

Pre - eclampsia

Advanced parental age
Indications

Familial factors:

CHD

Genetic syndromes;

Marfan

Noonan

Ellis van Crevald

Hypertrophic cardiomyopathy

Tuberous sclerosis
Fetal echocardiography

- No known adverse fetal effects
- Optimal timing – 16 -22 wks
  - Diagnosis *possible* at 12-14 wks
- No uniformly accepted approach
  - 4-chamber screen
  - Addition of great vessels/outflow tracts
  - Association with increased nuchal translucency
    (>99%ile >>> 3-5x risk of CHD)
Technique

Establish fetal lie, complete level II.
Cardiac & abdominal situs.
Fetal heart rate and rhythm.

Four chamber view.

(92% sensitivity, 99% specificity)

Segmental approach for venous and arterial connections and Doppler flow patterns:
Systemic, pulmonary veins
AV valves
LV, RV outflow tracts
Aortic, ductal arch
Four Chamber view
LV outflow tract
RV outflow tract
Systemic venous confluence
Aortic and Ductal arch
Aortic arch
Doppler flow patterns
Sinus rhythm – Doppler
AV Canal Defect
AV Canal
Complex single ventricle (Db1 inlet/db1 outlet LV)
Complex single ventricle
TV dysplasia
Intracardiac rhabdomyoma
Arrhythmias

Isolated extrasystoles

Sustained arrhythmia:

Any irregular rhythm, or any regular rhythm outside the normal fetal range of 100 - 160 bpm, and not associated with uterine contraction.
Arrhythmias

Indications for Fetal Arrhythmia Evaluation

Suspected arrhythmia
Non-immune hydrops fetalis
  (esp heterotaxy syndromes, corrected transposition)
Fetal cardiac tumors
Maternal collagen vascular disease
Maternal medications/toxins that may predispose fetus to arrhythmia
Arrhythmias

Isolated extrasystoles (benign)

Tachycardia:
- SVT: > 90 % reentry (AVRT)
- Atrial flutter / fibrillation
- Ventricular tachycardia (rare)

Bradycardia:
- High degree AV block associated with collagen vascular disease or complex CHD.

*Hydrops indicates poor prognosis.*
Arrhythmias: M-mode

SVT

Atrial Flutter
Arrhythmias

Progression of fetal CHF:
- atrial dilation (AV valve regurgitation)
- liver engorgement
- peripheral edema &/or ascites
- polyhydramnios
- fetal demise
Consideration for intervention must incorporate: *in utero* and postnatal natural history of lesion. risk / benefit for both mother and fetus.

Arrhythmias:
- **sustained** vs intermittent
- transplacental (oral, IV) vs direct (PUBS)

**knowledge of electrophysiologic mechanism & typical postnatal response.**
Introduction to Fetal Medicine

**Therapeutics**

**Tachycardias:**
- SVT – **digoxin**, type IA (procainamide, quinidine)
  type IC (flecainide)
- Atrial fib / flutter - **digoxin**, type IA, type III
  (amiodarone)
- VT - type IB (lidocaine, mexilitene, amiodarone)

**Bradycardia / Heart Block:**
- Steroids – no clear benefit, **may** limit progression
- Plasmapheresis, pacemaker ????

**Early delivery?!?**
Does antenatal diagnosis make a difference?

**Obstetric decisions:**
- parental reassurance (~95% for ‘follow-up’ patients)
- amniocentesis, genetic counseling (20 - 38 % aneuploid)
- search for other anomalies
- frequency of follow – up
- ? termination
- time, mode, place of delivery
Does antenatal diagnosis make a difference?

- **Neonatal decisions:**
  - appropriate facility, staff
  - need for prostaglandin infusion – avoid circulatory collapse in duct dependant lesions
  - very difficult to prove/quantitate survival or outcomes benefit *except for*:
    - HLHS
    - Coarctation
    - TGA
  - Counseling !!!
Does antenatal diagnosis make a difference?

- Parental counseling
- Know local surgical results
  - Inter-stage morbidity and mortality
- Long term outcomes
  - Physical
  - Neurologic
  - Family dynamics
- Potential termination
- Allows families to prepare for challenges of ‘altered normality’
Fetal intervention

Fetal interventional catheterization:

1991-Maxwell, et al *in utero* balloon aortic valvuloplasty. 4 patients, 5 attempts; 1 survivor.

2004-Marshall, et al. 20 attempts for patients with fetal aortic stenosis, 14 technically successful

3 HLHS prevented ??

12 HLHS

5 demise: 3 *in utero*, 1 previable, 1 termination
Fetal Intervention
Fetal intervention

- Critical aortic stenosis / HLHS
  - >120 attempts (~10% fetal demise)
  - ~80% technically successful
  - ~33% get to 2 ventricle repair! - high rate of EFE (endocardial fibro-elastosis)
  - ALL need postnatal cardiac interventions

- HLHS w/intact atrial septum
  - 25 attempts (~10% fetal demise)
  - ~95% technical success
  - ~50% avoid emergent cath at birth
Future Directions

• Results are likely to improve
  – Better patient selection, timing
    • Pretty clear results for predicting AS > HLHS
  – Improved instrumentation
    • Robotics?
  – Experience - - learning curve

• Ethical issues
  – Can a pregnant woman really give informed consent??
  – What about dad??
  – Natural history of any disease process MUST be well understood before undertaking any fetal intervention!!
Two ventricles are better than one!

“Human subtlety will never devise an invention more beautiful, more simple or more direct than does Nature, because in her inventions, nothing is lacking and nothing is superfluous.”

Leonardo da Vinci