Maternal Cardiac Disease In Pregnancy

August 25, 2017
PREGNANCY ECHO CONFERENCE
Maternal Physiology

Cardiac Output = HR x SV
Non-pregnant: 4.5 L/min
Pregnant: 6.0 L/min
Increase most acute in first 10 weeks
(increase in SV:10cc to 75 cc, blood volume: 50%)
Gradual increase to 24 weeks then plateau
(increase in HR)
Uterus: first trimester 3% CO
third trimester 17% CO
Maternal Physiology

LABOR: Increase CO by additional 40% pain (regional anesthesia mitigates increase) 
contractions (autotransfusion 300-500 cc) 
elevated BP

POST PARTUM: 10-20% increase CO 
autotransfusion from contracted uterus 
relief of IVC compression 
followed by rapid diuresis
Maternal Hemodynamics

<table>
<thead>
<tr>
<th></th>
<th>Non-Pregnant</th>
<th>Term Pregnancy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiac Output</strong></td>
<td>4.3 L/min</td>
<td>6.2 L/min</td>
</tr>
<tr>
<td><strong>Heart Rate</strong></td>
<td>71 bpm</td>
<td>83 bpm</td>
</tr>
<tr>
<td><strong>SVR</strong></td>
<td>1530 mmHg</td>
<td>1210 mmHg</td>
</tr>
<tr>
<td><strong>PVR</strong></td>
<td>119 mmHg</td>
<td>78 mmHg</td>
</tr>
<tr>
<td><strong>Colloid Oncotic</strong></td>
<td>21 mmHg</td>
<td>18 mmHg</td>
</tr>
<tr>
<td><strong>Pressure</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>MAP</strong></td>
<td>86</td>
<td>90</td>
</tr>
<tr>
<td><strong>PCWP</strong></td>
<td>6.3</td>
<td>7.5</td>
</tr>
<tr>
<td><strong>CVP</strong></td>
<td>3.7</td>
<td>3.6</td>
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</table>

S.Clark et al Am J Ob/Gyn 1989
Types of Cardiac Disease

Congenital
  The majority of patients seen
  Survival of children with CHD
Rheumatic Heart Disease
  Increasingly rare in developed countries
Peripartum Cardiomyopathy
In General . . .

Pregnancy has a favorable maternal and fetal outcome in patients with congenital heart disease.
EXCEPTIONS . . .

Severe Mitral Stenosis / Aortic Stenosis

Pulmonary Hypertension

Peripartum Cardiomyopathy
Beware

Signs of worsening heart disease may be interpreted as “normal pregnancy”

Increase HR, dyspnea, decrease exercise tolerance, edema, JVD, murmurs

Atypical: chest pain, orthopnea, arrhythmias
Beware

“Corrected” congenital heart disease

Almost always some “residual disease”
Risk of arrhythmias and decreased LV fxn.
# Low Risk Lesions (<1%)

<table>
<thead>
<tr>
<th></th>
<th>Mild AS</th>
<th>MVP</th>
<th>Mild / mod PS</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VSD</td>
<td>Mild AR</td>
<td>MR</td>
<td>Repaired non-cyanotic <strong><strong>CHD</strong></strong></td>
</tr>
<tr>
<td>PDA</td>
<td></td>
<td>Mild / Mod MS</td>
<td></td>
</tr>
</tbody>
</table>
Left to Right Shunts

- VSD, ASD, PDA
- Back leak of blood from systemic to pulmonary circulation
- Increase in CO balanced by decreased PVR
- Promotes systemic (forward) flow
- Pregnancy, Labor, post partum well tolerated
Left to Right Shunts

• EXCEPT:
  • If there is large shunt
    – pulmonary HTN
    – Arrhythmias
    – ventricular dysfunction
Labor Management

Avoid HTN to promote systemic flow.
Fluid balance: avoid fluid overload
## Moderate Risk Lesions (10%)

<table>
<thead>
<tr>
<th>Large left-right shunt</th>
<th>Moderate AS</th>
<th>Moderate MS</th>
<th>Severe PS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prior MI</td>
<td>Coartation of Aorta</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uncorrected Tetraology of Fallot</td>
<td>Marfans Syndrome (normal ao)</td>
<td></td>
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</table>
High Risk Lesions (25-50%)

Severe pulmonary HTN
Eisenmenger’s Syndrome (prolonged L-R shunt)
Complex cyanotic cardiac dz
Marfan Syndrome with aortic root >4.5 cm
Severe AS or MS
NYHA class III-IV
Prior peripartum cardiomyopathy
Eisenmenger / Pulmonary HTN

Consider pregnancy termination, but is not without risks

Oxygen, pulmonary vasodilating drugs, limit activity

Assisted second stage better outcomes than C/S
Marfan Syndrome

Autosomal Dominant Inheritance
Genetic counseling imperative
Aortic dissection / Rupture
SAB / SPTB
Prophylactic replacement of aortic root >4.0 cm
Beta Blockers (HR and BP)
If dilated root c/s recommended.
LV Outflow Obstruction

• Aortic Stenosis (bicuspid valve)
• Mild – moderate well tolerated
• Severe (aortic valve area < 1.0 cm)
  – 10% risk of maternal morbidity
  – Mortality rare
  – Ideally valve correction prior to pregnancy
LV Outflow Obstruction

Labor and postpartum:
IVF management. Increased fluid volume
Avoid hypoperfusion of coronary vessels
MI
Peripartum Cardiomyopathy

IDENTIFY THE PHENOTYPE (>36 wks)

- idiopathic
- viral
- hypertension
- CHD

Prognosis for future pregnancies depends on return of LV fxn. EF<40% = 50% mortality rate
“SBE prophylaxis is not recommended for NSVD or C/S in the absence of infection, except possibly for the small subset of patients at highest potential risk for adverse cardiac outcomes who are undergoing vaginal delivery”

Joint statement from AHA and ACC
Heart Transplant

Acute / chronic rejection not increased
(watch medication levels)
Prednisone, cyclosporine, azathioprine

Risks: infection PTB, low birth weight, preeclampsia

C/S for obstetric indications

Risks highest immediate post partum

Arrhythmias / sinus tachycardia (surgical trauma/ ischemia / loss of vagal nerve)
SBE Prophylaxis

SBE prophylaxis be considered in the highest risk patients

-Cyanotic cardiac disease
-Prosthetic valves

SBE prophylaxis administered 30-60 minutes before anticipated delivery
SUMMARY

Counseling is crucial

  Maternal, fetal, inheritance, recurrence

Multidisciplinary approach

Understand basic physiology of pregnancy

Understand basic physiology of lesions

Do NOT underestimate low risk lesions and “repaired” lesions

Cesarean section not always better option

Pregnancy termination poses risks