HEMODYNAMICS OF NORMAL PREGNANCY AND PREECLAMPSIA

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HOW MEASURE HEMODYNAMIC CHANGES

Swan-Ganz catheter

TTE

Advanced echo

CMR

Invasive vs noninvasive

Use of contrast

Influenced by preload, afterload

Patient position

Small numbers of patients

"Moving target"
PREGNANCY: SYSTOLIC FUNCTION

Ability of heart to generate force and shorten

Surrogates:

• Cardiac output = Stroke Volume x HR
• Fractional shortening
• Ejection fraction
• Strain
• Stroke work

Van Oppen et al. Obstet Gynecol 1996; 87: 310
[nice review of multiple studies]
Savu, O et al Circ Cardiovasc Imag 2012; 5: 289
MEASUREMENT OF EJECTION FRACTION

Visual Estimate

Simpson’s Rule

• Infinite # disks
• EDV-ESV/EDV = EF

Studies variable

• ↑ EF, ↓ EF, no Δ

Preload afterload sensitive

\[
\frac{170.2 \text{ ml} - 73.1 \text{ ml}}{170.2 \text{ ml}} = 59\%
\]
ECHO ASSESSMENT OF HEMODYNAMICS NORMAL PREGNANCY

Graphs showing changes in cardiac output, stroke volume, heart rate, total vascular resistance, and mean arterial pressure across different trimesters (Control, Trim 1, Trim 2, Trim 3, Postpartum) with statistical significance marked by asterisks (*), hash marks (#), and ampersands (&).
MYOCARDIAL MECHANICS STRAIN

LONGITUDINAL CONTRACTION

RADIAL CONTRACTION
LONGITUDINAL STRAIN

Global longitudinal strain using speckle tracking
Significant increase in EDV, CO increased, CI
But EF diminished
Ventricle more spherical
Global longitudinal strain decreased
They find decrease in all strain measures, may reflect that speckle tracking angle independent

RT3DE

No geometrical assumptions
Unaffected by foreshortening
More accurate and reproducible compared to other imaging modalities
Lower temporal resolution
Less published data on normal values
Image quality dependent
RT3DE PREGNANCY

43 normal pregnant women T1, T2, T3
LV mass increased
EF decreased
Ventricle more spherical
E/e’ increased but remained within normal limits (upper normal)
DETERMINANTS OF DIASTOLIC FUNCTION

- PRELOAD
- LV GEOMETRY
- RV-LV INTERACTION
- PERICARDIUM
- MYOCARDIAL STIFFNESS
- Atrial Contraction
- ACTIVE LV RELAXATION
- HEART RATE
IS THIS NORMAL OR ABNORMAL?
DIASTOLIC FUNCTION IN NORMAL PREGNANCY

E wave increased in pregnancy vs post partum

Increased blood volume

E/A ratio decreases

? Worse diastolic fxn

E/e’ remains normal throughout asymptomatic pregnancy

NI LVEDP

Fok et al. Ultrasound Obstet Gynecol 2006 28: 789
## PREECLAMPSIA: DIAGNOSIS

<table>
<thead>
<tr>
<th>Blood Pressure</th>
<th>≥ 140/90 mm Hg on at least 2 occasions at least 4 hours apart after 20 weeks gestation (newly diagnosed)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>≥160/110 mm Hg on two occasions</td>
</tr>
<tr>
<td><strong>AND...</strong></td>
<td></td>
</tr>
<tr>
<td>Proteinureia</td>
<td>≥300 mg per 24 hr urine or Protein/creatinine ratio ≥ 0.3 mg/dl</td>
</tr>
<tr>
<td><strong>OR...</strong></td>
<td></td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>Platelet count &lt; 100,000/microliter</td>
</tr>
<tr>
<td>Renal insufficiency</td>
<td>Serum creatinine concentration &gt; 1.1 mg/dl or doubling of serum creatinine concentration in absence of other renal disease</td>
</tr>
<tr>
<td>Impaired liver function</td>
<td>Transaminases &gt; twice control</td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td></td>
</tr>
<tr>
<td>CNS/visual symptoms</td>
<td></td>
</tr>
</tbody>
</table>
PATHOGENESIS PREECLAMPSIA

- Placental abnormalities lead to release of antiangiogenic factors such as sFlt1, soluble endoglin leads to endothelial dysfunction
- ↑ vascular permeability, vasoconstriction,
- activation of the coagulation system,
- micorangiopathic hemolysis hypertension
- proteinuria
PREECLAMPSIA AND CVD RISK

Brown MC et al. EurJEpidemiol 2013 28: 1

> Double the Risk
WHAT'S THE LINK?  MARKER OR CAUSE?

**SHARED RISK FACTORS**
- Hypertension
- Diabetes
- Obesity
- Renal disease
- Family hx htn or dm
- African American

**SHARED VASCULAR FACTORS**
- Pathologic lesions
- Endothelial dysfunction
- Vasomotor dysfunction
## CHANGES IN LV FUNCTION IN MOTHERS WITH PREECLAMPSIA

<table>
<thead>
<tr>
<th>Timing of study</th>
<th>Systolic function</th>
<th>Diastolic function</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cardiac output</td>
<td>EF</td>
</tr>
<tr>
<td>Early pregnancy</td>
<td>↑</td>
<td>~ or ↓</td>
</tr>
<tr>
<td>Late pregnancy</td>
<td>↓</td>
<td>~ or ↓</td>
</tr>
<tr>
<td>≤ 1 yr pp</td>
<td>↓</td>
<td></td>
</tr>
<tr>
<td>&gt; 1 yr pp</td>
<td>~</td>
<td></td>
</tr>
</tbody>
</table>

2D Imaging (EP and LP)
- ventricles bigger than controls
- EF’s similar
- RWT bigger than controls
- E/e’ : EP > LP > control
- e’ velocities decreased

3D Imaging
- LV volumes bigger and more spherical than controls
- EP volumes bigger than LP
- EF ↓ in PE but less in LP
- LVMi: EP > LP > control

Speckle Tracking: strain reduced

More eccentric hypertrophy
Worse myocardial compliance
Greater filling pressures

Central Pressure and Brachial Pressure are not the same

Pulse Pressure Amplification (PPA) = \frac{\text{Brachial Pulse Pressure (BPP)}}{\text{Central Pulse Pressure (CPP)}}
PULSE-WAVE ANALYSIS

Enables analysis of the aortic pressure waveform, which is a combination of:

- Forward waveform from the left ventricle
- Reflected wave from the arterial tree
- Can be used to assess arterial stiffness
PULSE-WAVE ANALYSIS

Augmentation Index = $\frac{AP}{PP}$

In normal pregnancy cardiac output increases, SVR decreases.
The left atrium enlarges and there is an increase in mass.
There are changes in diastolic function but they may be physiologic and related to the tachycardia and volume load of pregnancy.

In preeclampsia women are hypertensive, peripheral vascular resistance is increased, there is an increase in LV mass. Left ventricular end diastolic pressure is increased, the left atrium enlarges and there are changes in diastolic function and subtle changes in systolic function.
### Sample Size to Detect 10% Change

<table>
<thead>
<tr>
<th>Parameter</th>
<th>2D Echo</th>
<th>CMR</th>
<th>Sample Size</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEDD</td>
<td>509</td>
<td>261</td>
<td>509</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>LV mass</td>
<td>898</td>
<td>35</td>
<td>898</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>LVEF</td>
<td>609</td>
<td>91</td>
<td>609</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

- LVEDD 2D Echo 509 vs 261 by CMR
- LV mass by Echo 898 vs 35 by CMR
- LVEF by Echo 609 vs 91 by CMR

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Strohm et al. JMRI 2001; 13: 367
Grothus et al AmJCardiology 2002; 90:29
Bellenger et al JCMR 2000; 2: 271
BLIND MEN AND AN ELEPHANT

2D Echo
Cardiac Hemodynamics
Pregnancy

RT3DE

Arterial stiffness (PWA)
Patient Factors

Strain Analysis
Patient Position

Timing of Imaging
UNIFYING THEORY PRECLAMPSIA

- Placental factors
- Preplacental factors
- Fetal factors

Preclampsia
THANK YOU!
PATHOGENESIS PREECLAMPSIA

- Placental abnormalities lead to release of antiangiogenic factors such as sFlt1, soluble endoglin leads to endothelial dysfunction
- ↑ vascular permeability, vasoconstriction,
- activation of the coagulation system,
- micorangiopathic hemolysis hypertension
- proteinuria
ECHO IN PREGNANCY: DIASTOLIC FUNCTION

Normal
Grade 1: abnormal relaxation
Grade 2: pseudonormal
Grade 3: severe (restricted pattern)
Not Validated in Pregnancy

Nagueh et al. JASE 2009 22: 107
Sohn et al. JACC 1997
## Changes in Diastolic Function

<table>
<thead>
<tr>
<th></th>
<th>Early [ n=100]</th>
<th>Late Preg[32]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sep e’ cm/sec</td>
<td>12</td>
<td>9</td>
</tr>
<tr>
<td>Lat e’ cm/sec</td>
<td>13.9</td>
<td>11.4</td>
</tr>
<tr>
<td>Sep a’ cm/sec</td>
<td>5.7</td>
<td>6.5</td>
</tr>
<tr>
<td>Lat a’ cm/sec</td>
<td>4.6</td>
<td>5.4</td>
</tr>
<tr>
<td>E/A m/sec</td>
<td>1.5</td>
<td>1.42</td>
</tr>
<tr>
<td>E/e’ sep cm/s</td>
<td>8</td>
<td>9</td>
</tr>
</tbody>
</table>

THANK YOU

CHANGING MEDICINE. FOR GOOD.
At the University of Illinois Hospital, our commitment to moving medicine forward through discovery and innovation means you will find the greatest advancements in medicine.
MRI Gold-Standard for Myocardial Volumes and EF

- Observer 1 LV Volume (ml) vs. Observer 2 LV Volume (ml)
  - Equation: $y = 1.06x + 3.8$
  - $r^2 = 0.990$

- Systolic LV Mass (g) vs. Diastolic LV Mass (g)
  - Equation: $y = 1.01x - 3.2$
  - $r^2 = 0.990$

- MRI EF (%) vs. Radionuclide EF (%)
  - Equation: $y = 0.82x + 10.2$
  - $r^2 = 0.87$
MRI Gold-Standard for Myocardial Volumes and EF

- Observer 1 LV Volume (ml)
  - Linear equation: \( y = 1.06x + 3.8 \)
  - \( r^2 = 0.990 \)

- Systolic LV Mass (g)
  - Linear equation: \( y = 1.01x - 3.2 \)
  - \( r^2 = 0.990 \)

- MRI EF (%)
  - Linear equation: \( y = 0.82x + 10.2 \)
  - \( r^2 = 0.87 \)

LCE/NHLBI/NIH
# INTERSTUDY DIFFERENCE 2D ECHO VS CMR

<table>
<thead>
<tr>
<th>Parameter</th>
<th>2D Echo</th>
<th>CMR</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV EDD</td>
<td>6.1±17</td>
<td>5.0±6.4</td>
<td>NS</td>
</tr>
<tr>
<td>LV post wall</td>
<td>13.6±2.7</td>
<td>11.0±13.5</td>
<td>NS</td>
</tr>
<tr>
<td>LV EF</td>
<td>24.4±17.9</td>
<td>16.9±19.4</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>LV mass</td>
<td>27.1±20</td>
<td>4.1±2.8</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Strohm et al. JMRI 2001; 13: 367
Grothus et al AmJCardiology 2002; 90:29
Bellenger et al JCMR 2000; 2: 271
SEVERITY OF PREECLAMPSIA AND CVD RISK

Meta Regression
5 Case Control 10 Cohort Studies Pre-Eclampsia

Mild RR 2
Moderate RR 2.99
Severe RR 5.36

111,175 W with Pre-Eclampsia vs 2,259,579 W without

MacDonald S et al. AmHJ 2008
ECHO IN PREGNANCY: DIASTOLIC FUNCTION

Is this decreased diastolic function?
  - Tachycardia shortens diastole which increases A wave
  - Increased LV mass decreases compliance

Or is this normal diastolic function?
  - Volume load
  - Hyperdynamic circulation
PULSE-WAVE ANALYSIS

Provides a measure of central blood pressure and systemic arterial stiffness

• Augmentation pressure
• Augmentation index

Factors that may affect results include:

• Age
• Obesity
• Cigarette smoking
• Caffeine
PWA PREECLAMPSIA
# Inter-Study-Difference for 2D-Echo and MRI (Percent Difference From Baseline Value)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>2D-echo</th>
<th>MRI</th>
<th>P-values</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV-EDD</td>
<td>6.1 ± 17.9</td>
<td>5.0 ± 6.4</td>
<td>n.s.</td>
</tr>
<tr>
<td>LV-PW</td>
<td>13.6 ± 2.7</td>
<td>11.0 ± 13.5</td>
<td>n.s.</td>
</tr>
<tr>
<td>LV-EF</td>
<td>24.4 ± 17.9</td>
<td>16.9 ± 19.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVM</td>
<td>27.1 ± 20</td>
<td>4.1 ± 2.8</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

LV-EDD = left ventricular end-diastolic diameter  
LV-PW = left ventricular posterior wall  
LV-EF = left ventricular ejection fraction  
LVM = left ventricular mass

Strohm et al. JMRI 2001; 13: 367  
See also Grothues et al. Am J Cardiol 2002; 90: 29  
Bellenger et al. JCMR 2000; 2: 271
MRI Gold-Standard for Myocardial Volumes and EF

Observer 2 LV Volume (ml)

Observer 1 LV Volume (ml)

Diastolic LV Mass (g)

Systolic LV Mass (g)

MRI EF (%)

Radionuclide EF (%)

y = 1.06 x + 3.8
r² = 0.990

y = 1.01 x - 3.2
r² = 0.990

y = 0.82 x + 10.2
r² = 0.87

LCE/NHLBI/NIH
Aortic pressure waveform provides information about patient physiology and risk.

Contribution of wave reflection and arterial stiffness to hypertension.

Individualized treatment strategy.

**AP = Augmentation Pressure**

Contribution of reflected wave to pulse pressure.
Contribution of wave reflection and arterial stiffness to hypertension

Individualized treatment strategy

Aortic pressure waveform contains important information about patient physiology and risk

**Pulse Pressure Amplification (PPA)** = \(
\frac{\text{Brachial Pulse Pressure (BPP)}}{\text{Central Pulse Pressure (CPP)}}
\)
PRE-ECLAMPSIA AND CNS RISK

Brown MC et al. EurJEpidemiol 2013 28: 1

Almost Doubles Risk
SEVERITY OF PREECLAMPSIA AND CVD RISK

MacDonald S et al. AmHJ 2008

111,175 W with Pre-Eclampsia vs 2,259,579 W without
## PREECLAMPSIA: RISK FACTORS ABN 1 YR PP

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Pre-eclampsia</th>
<th>Normotensive</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight</td>
<td>77.3</td>
<td>71.8</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>BMI</td>
<td>29</td>
<td>26</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>% Obese</td>
<td>38.6</td>
<td>18.6</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>MetSynd</td>
<td>18.6%</td>
<td>5.7%</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>BP systolic</td>
<td>120</td>
<td>111.3</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>BP diastolic</td>
<td>81.5</td>
<td>72.2</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Glucose</td>
<td>4.93</td>
<td>4.81</td>
<td>NS</td>
</tr>
<tr>
<td>Insulin Level</td>
<td>63.6</td>
<td>44.7</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LDL Chol</td>
<td>2.71</td>
<td>2.37</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Smith, et al. AJOG 2009; 200:58
LIFETIME CUMULATIVE SURVIVAL AFTER DIAGNOSIS PRE-ELAMPSIA

FIGURE 1. Life table cumulative estimates of maternal survival after preeclampsia. “Preeclampsia, subsequently, normotensive” refers to women with preeclampsia who had at least one subsequent normotensive birth. “Preeclampsia, subsequent status unknown” refers to all other women with preeclampsia.

Funai EF et al. Epidemiology 2005; Mar 16 (2): 206
WHAT’S THE LINK? MARKER OR CAUSE?

**SHARED RISK FACTORS**
- Hypertension
- Diabetes
- Obesity
- Renal disease
- Family hx htn or dm
- African American

**SHARED VASCULAR FACTORS**
- Pathologic lesions
- Endothelial dysfunction
- Vasomotor dysfunction
GESTATIONAL HTN AND CV RISK

GESTATIONAL HTN

HTN  HR 2.7
  • Younger age

All CVD  HR 1.59

IHD  HR 1.67

MI  HR 1.85

MI death  HR 4.49

CHF  HR 2.47

CNS disease  HR 1.67

Increased DM, CKD

Männistö T et al. Circ 2013; 127:681
FUTURE RISK FROM GDM, GHTN, PRE-ECLAMPSIA

Risk rivals that associated with smoking and family history

Predict CV risk earlier than conventional screening tools like Framingham where age is predominant determinant and few women high risk before age 70
ECHO IN PREGNANCY: ADVANTAGES

Cheap, Quick, Familiar
Safe
Structure
Function
Hemodynamics
Pulmonary pressure
EVALUATION OF HEMODYNAMICS
ECHO IN PREGNANCY: DISADVANTAGES

Good imaging more difficult
- Heart more horizontal
- Patient more tachypneic
- Subcostal views?
- Contrast use?

No “normal values”

Influenced by preload, afterload, HR, hormones

**“steady state”**
ECHO ASSESSMENT OF HEMODYNAMICS NORMAL PREGNANCY

![Graph showing hemodynamics comparison between Control, T1, T2, T3, and PP. The graph illustrates changes in MAP and TVR across these time points.](image)
CHIRP STUDY

34 women imaged by both TTE and CMR early in 3rd trimester and 3 mo post partum

Mean age 29, BMI 24, mean gestational age 34 weeks ± 16 days at T3 imaging

CMR done in half LLD position; TTE done in LLD position

Increase in LV mass by both techniques

? Show figure 1

TTE tended to underestimate and had wider confidence limits

LA volumes increased, no change in aortic diameter

Diastolic parameters: increase in A velocity, decreased E/A ratio DT, IVRT and E/e’ did not change significantly

HR increased by 20%
SUPINE VS LLD POSITION

Rossi et al: 5 nonpregnant women, 6 pregnant women at mid pregnancy and 8 healthy pregnant women at 32 weeks underwent CMR in supine vs LLD position

T2 increase in LVEF, SV, LA atrial diameters increased with change from supine to LLD position

T3 changes even more important

Recommended imaging in the LLD position

Right heart changes seen at 20 weeks but not 32 weeks

Show figure 2
EFFECTS OF MATERNAL POSITION

Nelson et al. 14 normal weight and 9 obese women were imaged 12-16 weeks, 26-30 weeks, 32-36 weeks and 3 months post partum

BMI in normal group 22.7 ± 1.7 at baseline

BMI in obese group  29.4 ± 2.2 obese group was younger

Normal group: no change in T1, stroke volume increased in LLD in comparison with supine T2

In obese no difference –hypothesize that cushioning effect protects against aortocaval compression.
HOW MEASURE LA SIZE: VOLUME

Biplane Area Length
\[ \frac{8}{3} \pi [(A_1)(A_2)/(L)] \]

* (L) is the shortest of A4C or A2C length

Simpson’s Rule
Sum of volume of individual disks
Volume = \( \frac{\pi}{4} (h) \sum (D_1)(D_2) \)

LA volume: early small ↓ than ↑ 5%

Lang et al. JASE 2005: 18: 1440
MEASUREMENT OF CARDIAC OUTPUT

CO = Stroke volume x HR

Increased bld volume 500 cc
Increased chamber size 5-10%

↑ 20-30% stroke volume

Reaches peak at end of 2nd trimester in some studies, 3rd in others

Increase in HR 10-20 bpm

Area LVOT: \( \pi R^2 = 4.48 \)

Flow LVOT: 0.172m
SV = 77 ml

Van Oppen et al. Obstet Gynecol 1996; 87: 310 [nice review of multiple studies]
GOLD STANDARD FOR ASSESSMENT OF VOLUME & MASS
ECHO ASSESSMENT OF LEFT SIDED HEMODYNAMICS NORMAL PREGNANCY

Bottom line
CO increased
Stroke work increased
EF no change (2D), decreased (3D)
LA increased
Mass increased, eccentric hypertrophy
More spherical shape
Peak systolic velocity septum increased
Average strain rate increased for all segments
RT 3DE
MYOCARDIAL MECHANICS: STRAIN

Radial Thickening

Longitudinal Thickening

Video Courtesy of Mayank Kansal, MD
TORSIONAL MECHANICS

Video Courtesy of Mayank Kansal, MD
SUMMARY STRAIN SLIDE
MRI GOLD STANDARD FOR EVALUATION OF MASS AND FUNCTION

Strohm et al. JMRI 2001; 13: 367
Grothus et al AmJCardiology 2002; 90:29
Bellenger et al JCMR 2000; 2: 271
CMR RESULTS:

14 normal

Stroke volume and cardiac output increase in pregnancy

CMR RESULTS

9 obese women

No significant positional change in CO in the obese

RT3DE: LA
RESULTS

Systolic, diastolic and MAP small decrease in the second trimester with slight increase in the third

TVR decreased reaching nadir in second trimester and did not significantly (?) change in third.

Cardiac output increased progressively initially due to an increase in SV and then late increase in HR
RT3DE CHAMBER QUANTIFICATION

CONG LA
HEMODYNAMIC SHIFTS

Data: Bonica et al 1994; Image: Ouzounian 2010
PREGNANCY: NORMAL DIASTOLIC FUNCTION

LV MASS INCREASES

PATHOGENESIS PREECLAMPSIA

- Placental abnormalities lead to release of antiangiogenic factors such as sFlt1, soluble endoglin leads to endothelial dysfunction
  - ↑ vascular permeability, vasoconstriction,
  - activation of the coagulation system,
  - microangiopathic hemolysis hypertension
  - proteinuria
PATHOGENESIS PREECLAMPSIA

Healthy Blood Vessel

Blood Vessel in Preeclampsia

- FLT-1
- PIGF
- VEGF
- sFLT-1
- ENDOGLIN (ENG)
- Transforming Growth Factor (TGF-β I)
- Transforming Growth Factor (TGF-β II)
- Soluble Endoglin (sEng)

Increased blood pressure
Diagram illustrating the relationship between various factors and preeclampsia. Genetic factors such as VEGF, PIGF, FLT-1, and sFLT-1 are shown to influence the placenta and blood vessels. Hypertension and other symptoms like proteinuria, edema, and renal insufficiency are also depicted. Anticoagulant and vasodilatory factors affect healthy endothelial cells, while procoagulant and vasoconstricting factors impact dysfunctional endothelial cells.
Early onset PE associated with abnormal uterine artery Doppler, fetal growth restriction more adverse neonatal and maternal outcomes

Late onset PE milder normal or slightly increased uterine resistance index more favorable outcomes

Hypothesis that hemodynamics and volume homeostasis different

Early low CO high TVR and late High CO low TVR
LA VOLUME AND FUNCTION DURING NORMOTENSIVE AND PREECLAMPTIC PREGNANCY
TTE PREECLAMPSIA

Dennis AT et al
Aortic pressure waveform

Wave reflection and arterial stiffness

AP = Augmentation Pressure

Contribution of reflected wave to pulse pressure