Obstetric Critical Care Symposium

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Cardiovascular Changes of Pregnancy

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Objectives

1. Describe **hormonal** mechanisms that drive cardiovascular adaptation during pregnancy
2. Describe and quantify the **hemodynamic changes** of pregnancy (plasma volume, cardiac output, systemic vascular resistance)
3. Describe the **structural cardiac adaptations** to pregnancy
4. Describe the **peak hemodynamic stress** of the labor and delivery period
5. Describe the cardiovascular changes of the immediate **postpartum** period

Background

- Cardiovascular disease in pregnancy is on the rise due to advanced maternal age, cardiovascular risk factors, and successful management of congenital heart disease conditions
- This is the most common cause of indirect maternal mortality in pregnancy, affecting 1-2% of pregnancies, accounting for 15.5% of maternal deaths in the US
- Up to 1/3 of women with cardiovascular disease use cardiac medications during pregnancy

Hormonal mechanisms that drive cardiovascular adaptation during pregnancy

- 3 main hormonal mechanisms:
  - Estrogen
  - Progesterone
  - Prolactin

Estrogen

- **Increased plasma volume**
  - Activates RAAS (renin angiotensin aldosterone system) → increases plasma volume by up to 40—50% above pre-pregnancy values → sodium and water retention
- **Vasodilation** (both estrogen and progesterone)
  - 30-50% reduction in systemic vascular resistance (SVR) by the second trimester
- **Increased heart rate (HR)**
  - In late pregnancy, estrogen increases sinoatrial node automaticity and HR through ER alpha mediated mechanisms
  - HR reaches a maximum of 10-20 bpm above baseline in the 3rd trimester

Progesterone

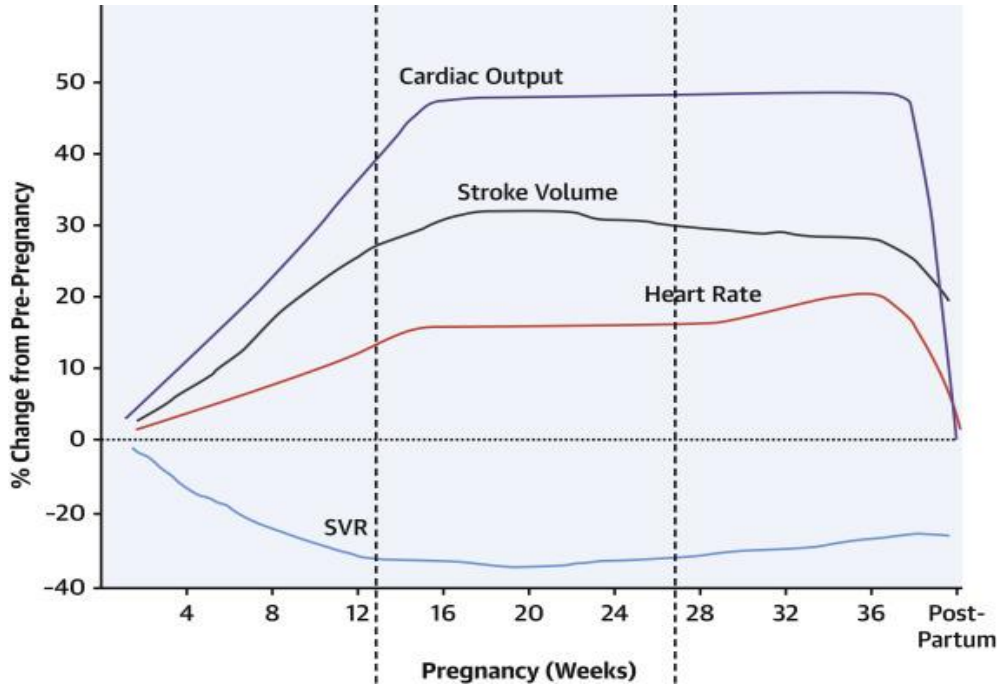
- **Vasodilation**
  - Increases nitric oxide synthesis in vascular endothelial cells→30-50% reduction in the SVR by the second trimester
- **Direct vascular muscle relaxation**
- **Plasma volume expansion**
  - Increases plasma and extracellular fluid volume independently of estrogen
  - With estrogen, synergistically expands plasma volume through both extracellular fluid volume expansion and reduced transcapillary escape rate of albumin

Prolactin

- At physiological levels, promotes **vasodilation** and **BP reduction** through nitric oxide pathways
- Has been implicated in increased preload and blood volume expansion of pregnancy (unclear why)

Hemodynamic changes of pregnancy

- Plasma volume
- Cardiac output
- Systemic vascular resistance



Hemodynamic changes of pregnancy

- Plasma volume ⬆

Increased circulating plasma volume

- How: estrogen, progesterone, and RAAS activation→ water and sodium retention
- Begins as early as **6th week** of pregnancy, rising rapidly until mid-pregnancy, then continues to rise but at a slower rate
- **Increases by 45-50%** (over 1L) above pre-pregnancy values, peaking late in the third trimester around 32-38 weeks gestation
- RBC mass also increases, but much less rapidly than plasma volume, resulting in **hemodilution/physiological anemia**
- **Decreased blood viscosity** → lower resistance to blood flow (+ vasodilation)→ reduced BP despite increased cardiac output

Hemodynamic changes of pregnancy

- Cardiac output (CO) 

Increased cardiac output

- CO (cardiac output) = HR (heart rate) x SV (stroke volume)
- How:
  - Plasma volume expansion
  - Increased heart rate
- Increases by up to 30-50% above pre-pregnancy values, peaking in early 3rd trimester

Increased cardiac output (cont.)

- Timeline:
  - Early (1st trimester): **Increased stroke volume**
  - Later (2nd and 3rd trimesters): **HR increases** by 10-20 bpm, reaching max by 3rd trimester, becoming **predominant driver** of continued CO elevation
- Keep in mind:
  - Preload reduction may occur due to compression of the IVC by the gravid uterus (3rd trimester) → reducing venous blood return to the R heart → potentially reduced CO in supine position

Hemodynamic changes of pregnancy

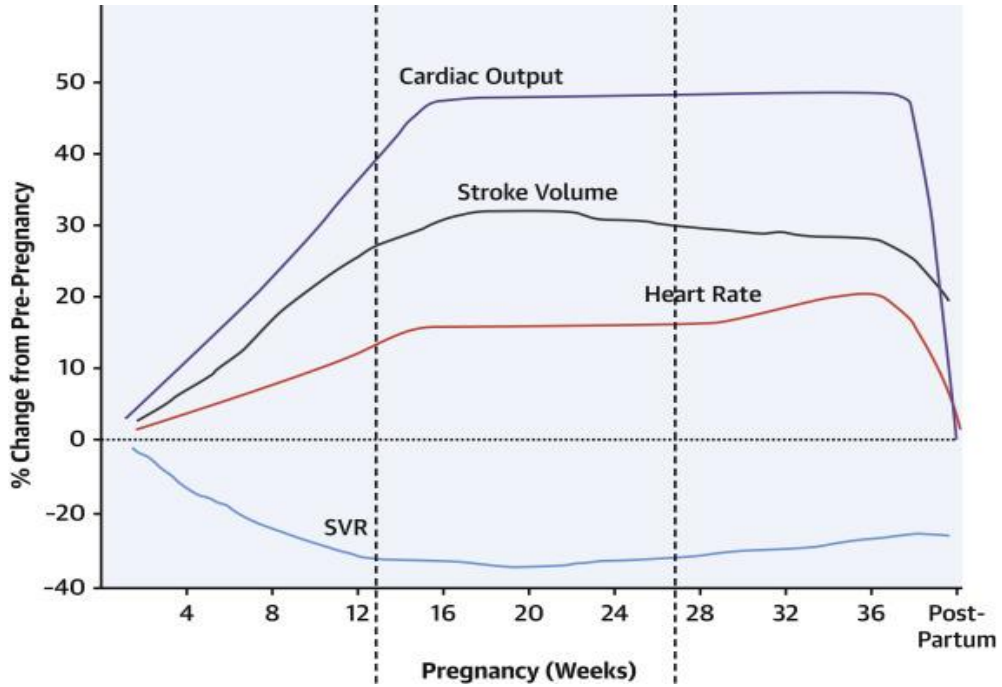
- Systemic vascular resistance (SVR) ⬇

Reduced systemic vascular resistance

- How:
  - Multiple vasodilatory mediators
- Falls 30-50% from pre-pregnancy values by the second trimester, followed by slight increase at the end of the 3rd trimester
- Structural vascular remodeling occurs to accommodate the expanded plasma volume

Hemodynamic changes of pregnancy

- Plasma volume ⬆
- Cardiac output ⬆
- Systemic vascular resistance ⬇

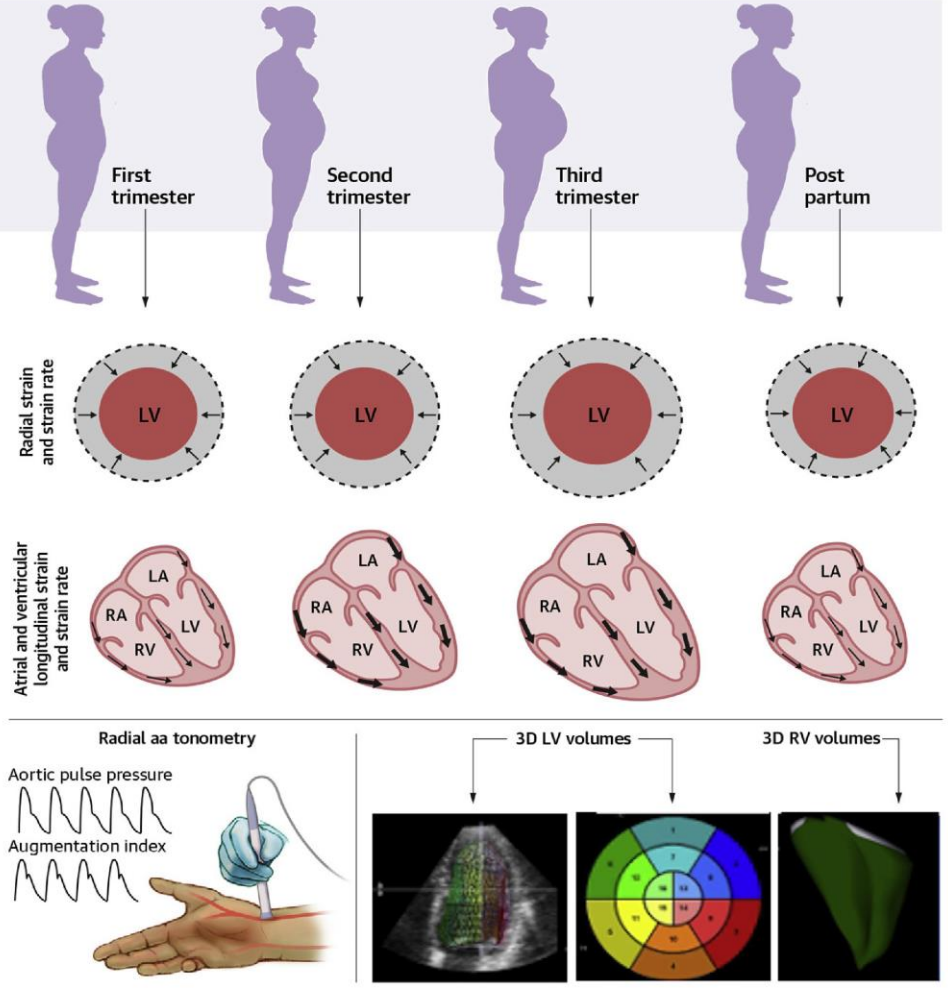


Structural cardiac adaptations to pregnancy

- Changes begin around 2nd trimester → onwards
- 4 chamber enlargement throughout pregnancy
- RV (right ventricle) and LV (left ventricle) mass increase
- LV and RV ejection fractions remain unchanged (unclear mechanism)
- All structural cardiac changes should resolve postpartum

CENTRAL ILLUSTRATION: Physiologic Cardiovascular Changes in Normal Pregnancy

- 45 healthy women were evaluated during normal pregnancy for cardiovascular changes:
  - 2D/Doppler
  - Speckle tracking strain
  - 3D echocardiography
  - Radial artery applanation tonometry



UNCHANGED

LV (Systolic)
- LV fractional shortening
- LV Vcfc
- LV circumferential strain and strain rate
- LV radial strain and strain rate
- Pulmonary aa. systolic pressure

LV (Diastolic)
- E/e'
- E-wave
- Deceleration time

DECREASED

- Aortic pulse pressure
- Augmentation index
- Systemic vascular resistance
- Mitral E/A ratio
- Mitral annular E'**

**Third trimester only

INCREASED

Left Heart (Systolic)
- IVS systolic thickness
- LVEDV, LVESV, LV stroke volume
- LVEF
- Longitudinal strain rate
- Ejection duration

Left Heart (Diastolic)
- LA volume
- Mitral inflow A-wave**
- PV S-wave
- PV atrial reversal wave
- LA longitudinal strain rate

Right Heart
- Annular S'
- RVEDV, RVESV (3D)
- RV stroke volume (3D)
- RV longitudinal strain rate
- RA area
- RA longitudinal strain rate
- Annular E'

Naqvi TZ, et al. JACC Adv. 2024;3(11):101360.

Peak hemodynamic stress of the labor and delivery period

- 1st stage
  - Contractions, pain, anxiety → **increased HR, BP, CO**
  - Each uterine contraction→**300-500 cc blood** into circulation
  - **Oxygen** consumption **increases 3x** during labor
- 2nd stage
  - Greatest hemodynamic changes when **patient is pushing**
  - **CO increases** ~50% during this stage
  - Valsalva during pushing can transiently reduce CO, BUT a compensatory increase in CO occurs with release of each Valsalva

Cardiovascular changes of the immediate postpartum period

- **CO increases** by nearly 50-80% above pre-labor values due to:
  - Sudden IVC decompression
  - Rapid autotransfusion (physiological redistribution of uteroplacental circulation back into maternal systemic circulation)
  - Redistribution of blood from the lower limbs
- **SVR increases** after placental delivery due to:
  - Estrogen and progesterone withdrawal
- Clinical vulnerability: increased susceptibility to pulmonary edema

Cardiovascular changes of the immediate postpartum period

- Oxytocin release
  - **HR and BP decrease** within 48 hours postpartum
  - Increased maternal atrial natriuretic peptide (ANP) in first week postpartum → **postpartum diuresis** to mobilize expanded volume

Resolution Timeline

- Most changes resolve within 2 weeks, but take up to 3-6 months, even a year, to get back to full baseline

Take Home Points

1. Hormonal changes drive increased plasma volume, heart rate, and vasodilation.
2. Plasma volume and cardiac output increase significantly, with a decline in systemic vascular resistance.
3. There are significant cardiac structural changes that resolve postpartum.
4. There is an increase in heart rate and cardiac output during labor/delivery, making this a very susceptible time to complications.
5. In the immediate postpartum period, we see rapid fluctuations in hemodynamics as the body returns to pre-pregnancy state.

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