# Maternal physiology in pregnancy and fetal circulation

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# Endocrine changes during pregnancy

# Introduction

During pregnancy, a woman's body undergoes numerous physiological changes, including significant alterations in the endocrine system. The endocrine system plays a crucial role in regulating hormones, which are chemical messengers that control various bodily functions. These hormonal changes are essential for supporting and maintaining a healthy pregnancy, facilitating the growth and development of the fetus as well as facilitating subsequent lactation.

# HCG

Human Chorionic Gonadotropin (HCG): HCG is one of the earliest hormones to surge during pregnancy. Produced by the cells forming the placenta, HCG supports the corpus luteum, which in turn produces progesterone during the first trimester. This hormone is also the basis for most pregnancy tests, as it can be detected in the urine shortly after conception. Elevated HCG levels are essential for maintaining the uterine lining and supporting early fetal development.

- Origin: First expressed by trophoblast cells, then secreted by the syncytiotrophoblast after implantation (~6–10 days post-fertilization).

- Detection:
  - Serum β-hCG becomes detectable approximately 8–9 days post-ovulation, serving as the basis for most pregnancy tests.

  - Urine tests detect later, around 10 days after fertilization (or ~14 days LMP) .

**β HCG detection :**

**1.Urinary pregnancy test :**
1week after missed period

**2.Blood HCG :**
Quantitative : at time of missed period
± doubling
Qualitative : at time of missed period
( just +ve or –ve )

# Physiological Actions of hCG

1. Corpus luteum support: Stimulates progesterone production until placenta maintenance is ensured at ~10–12 weeks .
2. Suppresses uterine contractility, supports fetal organogenesis and umbilical development .
3. Increases regulatory T cells (Tregs) in early pregnancy.
4. Promotes maternal immune tolerance of the fetus.

# ?Clinical Uses

## 01. Confirming Pregnancy

## 02.

**Monitoring Pregnancy Progress**

- Rising slowly or plateauing → ⚠️ ectopic or miscarriage
- Rapidly increasing above normal → ⚠️ molar pregnancy

## 03.

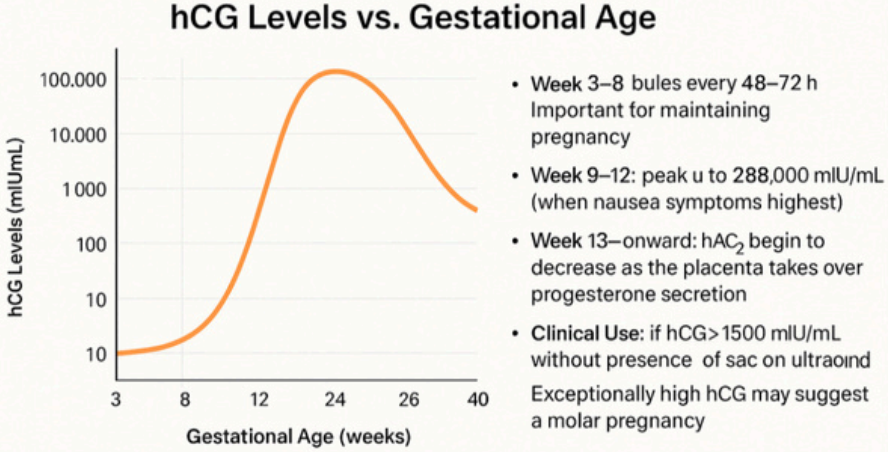
- **Tumor marker & pathology:**

Elevated in gestational trophoblastic disease and choriocarcinoma; used in monitoring molar pregnancy and other pathologies .

### Important Thresholds

- 1,500 mIU/mL: Should see gestational sac on TVUS
- 6,000 mIU/mL: Yolk sac visible
- 10,000 mIU/mL: Fetal heartbeat likely detectable

# hCG Levels Throughout Pregnancy




**Early Pregnancy (Weeks 3–8):**

hCG doubles every 48–72 hours, important to sustain corpus luteum function.
Useful for early pregnancy detection.

**Peak Phase (Weeks 8–12):**

hCG peaks up to ~288,000 mIU/mL.
This coincides with worst nausea/vomiting symptoms due to high hormone levels.

**Decline Phase (After Week 12–13):**

hCG decreases as the placenta takes over progesterone secretion.
Still detectable throughout pregnancy, but not as high.

**Clinical Use:**

If hCG >1500 mIU/mL but no gestational sac is seen on ultrasound, suspect ectopic pregnancy.
Exceptionally high hCG levels may suggest molar pregnancy (like trophoblastic disease).

# Progesterone:
# "The Hormone of Pregnancy Maintenance"

- Source:
  - Early pregnancy (Up to 6–7 weeks) : Secreted by corpus luteum, maintained by hCG.
  - After 7 weeks: Placenta takes over → called the "luteo-placental shift."
  - Myometrium receives progesterone directly from → Venous blood draining the placenta

- Functions:
  - Maintains endometrial lining (decidua) for implantation.
  - Prevents uterine contractions → maintains uterine quiescence → prevents miscarriage.
  - Suppresses gap junction formation and placental CRH expression.
  - Establishes immune tolerance for fetal tissues
  - Inhibits actions of estrogen, cytokines, and prostaglandins.

- Clinical Notes:
  - Low progesterone = threatened miscarriage.
  - Measured in some IVF or high-risk cases.
  - Progesterone supplements may be used in luteal phase defects or recurrent pregnancy loss.

# Progesterone:
# “The Hormone of Pregnancy Maintenance”

- High progesterone (>25 ng/mL) = Healthy pregnancy

Very high chance of viable intrauterine pregnancy

- Low progesterone (<5 ng/mL) = High risk of miscarriage or ectopic.

Used to evaluate early pregnancy viability.

- Progesterone supplementation is used to prevent preterm labor

Based on ACOG guidelines — used especially if there’s previous preterm birth.

- What week the hormone increases ? (Week 7–9 = placental takeover)

# Progesterone:
# “The Hormone of Pregnancy Maintenance”

**How is Progesterone Measured?**
1. Blood test
2. Usually done in the first trimester
3. Helps assess pregnancy viability

**Effect of Progesterone on the Uterus:**
1.Stabilizes the endometrium (prevents shedding)
2.Inhibits uterine contractions → reduces risk of early pregnancy loss

**When Is Progesterone Prescribed?**
1. History of miscarriage
2. Short cervix on ultrasound
3. Risk of preterm labor (especially if previous preterm delivery)

# Estrogen  in Pregnancy

- Both fetus and placenta are essential in synthesizing:
  - Estrone (E1)
  - Estradiol (E2)
  - Estriol (E3)

- Estriol (E3) is the most abundant estrogen in human pregnancy > source of fetus and placenta

# Physiological Roles of Estrogens in Pregnancy

- Increase uterine blood flow → support uterine growth.
- Prepare breast tissue for lactation.
- Stimulate liver to produce hormone-binding globulins.

Collaborates with cortisol for fetal lung surfactant production

# Clinical Importance of Estriol (E3)

- Estriol (E3) is the most abundant estrogen in pregnancy.
- Sudden decline in maternal estriol may signal fetal compromise.

(Sudden drop in maternal estriol → may indicate fetal compromise particularly significant if fetus is neurologically intact)

- Anencephalic fetuses: very low estriol due to underdeveloped Hypothalamic–Pituitary–Adrenal axis .
- Estriol level reflects fetal adrenal and liver function → used in fetal monitoring.

# Prolactin in Pregnancy

- Prolactin: peptide hormone from anterior pituitary (MW ~20,000).
- Non-pregnant level ~10 ng/mL.
- In pregnancy: levels increase due to rising maternal estrogen.

  Estrogen stimulates pituitary lactotrophs to produce more prolactin.
- Main function: stimulates postpartum milk production.
- Fetal pituitary prolactin (2nd half of pregnancy) may aid fetal adrenal growth.

# Pituitary Gland

**Note : Endocrine Changes in Maternal Hypothalamus**

**Likely influenced by estrogen stimulation → Drives downstream pituitary changes**

- **Anterior Pituitary Enlargement**

  Pituitary Gland Growth in Pregnancy

  - Enlarges 2–3 times in size
  - Due to hyperplasia and hypertrophy of lactotroph cells
  - Plasma prolactin levels increase in parallel with pituitary size
  - Other cell types:
    - Somatotrophs & Gonadotrophs ↓
    - Thyrotrophs & Corticotrophs → (unchanged)

# Posterior Pituitary in Pregnancy

- Size ↓ during pregnancy

- Oxytocin:
  - Maternal oxytocin levels ↑ progressively
  - Follows estradiol and progesterone levels
  - Oxytocin receptors in uterus ↑ 100-fold by term→ Especially in the myometrium

# Thyroid Gland

## Thyroid Physiology During Pregnancy

### 01

- The thyroid gland undergoes moderate enlargement during pregnancy.

### 02

- This is not due to elevated TSH (which stays unchanged).

### 03

- Enlargement is linked to hCG's TSH-like action, especially during the first trimester when hCG is highest.

### 04

- This action may cause abnormally low TSH levels in early pregnancy.

# Thyroid Hormones in Circulation

- **Two primary active forms:**

1. Thyroxine (T4): More abundant, less potent, serves as prohormone for T3.
2. Triiodothyronine (T3): Less abundant, more potent metabolically.

Most circulating T4/T3 is protein-bound, with only free hormone being biologically active.

# Binding Proteins and Hormone Availability

- ~85% of T4 binds to Thyroxine-Binding Globulin (TBG).
- The rest binds to thyroxine-binding prealbumin.
- Estrogen ↑ → TBG increases → total T4 and T3 increase.
- Free (active) hormone levels remain unchanged.

# Thyroid Function Evaluation in Pregnancy

- Use the Free T4 Index → it adjusts total T4 for protein binding.
- Reference ranges remain same as in nonpregnant state.
- Measuring total T4 alone is misleading due to estrogen-induced rise in TBG.

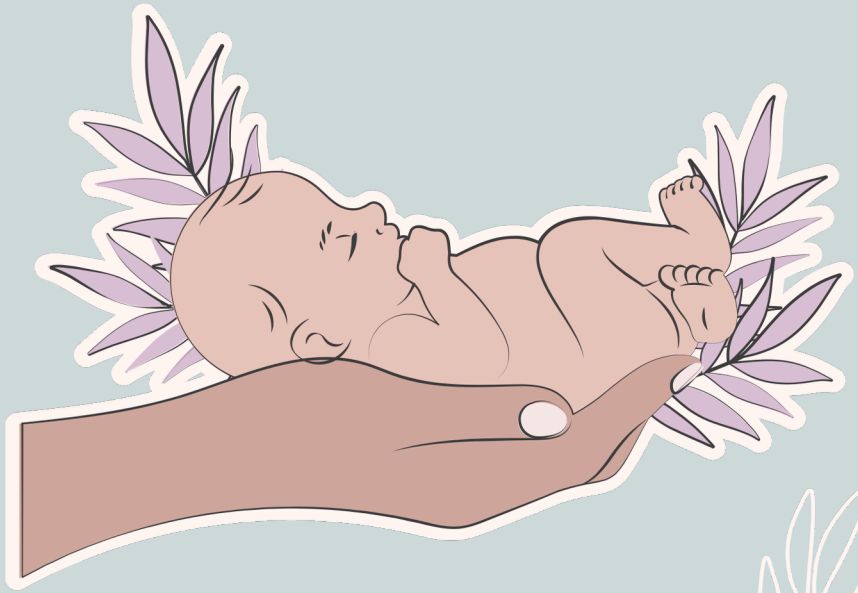
# Maternal-Fetal Thyroid Hormone Transfer

- Only minimal amounts of thyroid hormone cross the placenta.
- Transferred hormone is largely converted to reverse T3 (rT3) → inactive.
- Fetus synthesizes its own thyroid hormones starting in the 2nd trimester.

TABLE 5-1

## HORMONES AND TRANSMITTERS OF PREGNANCY AND PARTURITION

| Hormone/Transmitter | Source | Function(s) | Clinical Comments |
|---|---|---|---|
| Human chorionic gonadotropin (hCG) | Placental trophoblastic tissue | Prevents regression of (rescues) the corpus luteum of pregnancy; increases T-cells that affect immunity | A likely regulator of a process that provides immune tolerance for the fetus; other trophic activities |
| Human placental lactogen (hPL) | Placenta | Antagonizes maternal glucose use so more is available for the fetus | Low values found in pregnancy loss; normal levels may increase risk of gestational diabetes |
| Corticotropin-releasing hormone (CRH) | Placenta | Stimulates fetal adrenocorticotropic hormone (ACTH) secretion, which allows the fetal adrenal to secrete DHEA-S for progesterone production; CRH may facilitate vasodilation | Fetal cortisol stimulates placental CRH release and fetal ACTH secretion; elevated levels may predict an increased risk of preterm birth |
| Prolactin | Maternal and fetal (late pregnancy) anterior pituitary glands | Stimulation of postpartum milk production | May play a role in fetal adrenal growth, as well as fluid and electrolyte membrane transfer |



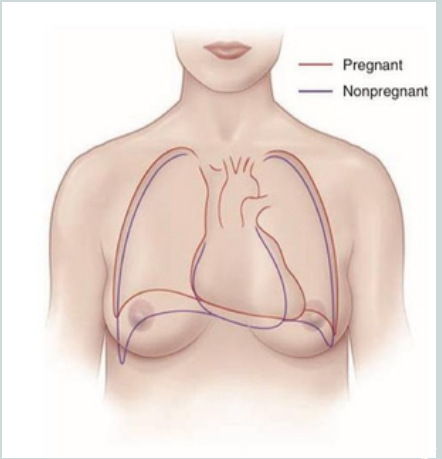
02

# Cardiovascular and hemodynamic changes

# General concept

- Thematernal physiologicchanges that occurduring pregnancy are directlylinked tothe specific metabolic demands of the fetus. The numerous physiologic adaptations of pregnancy are not the result of a single factor or event; rather, they are the culmination of the biochemicalinteractionsthatoccurbetweenthreedistinct interacting systems: maternal, fetal, and placental.

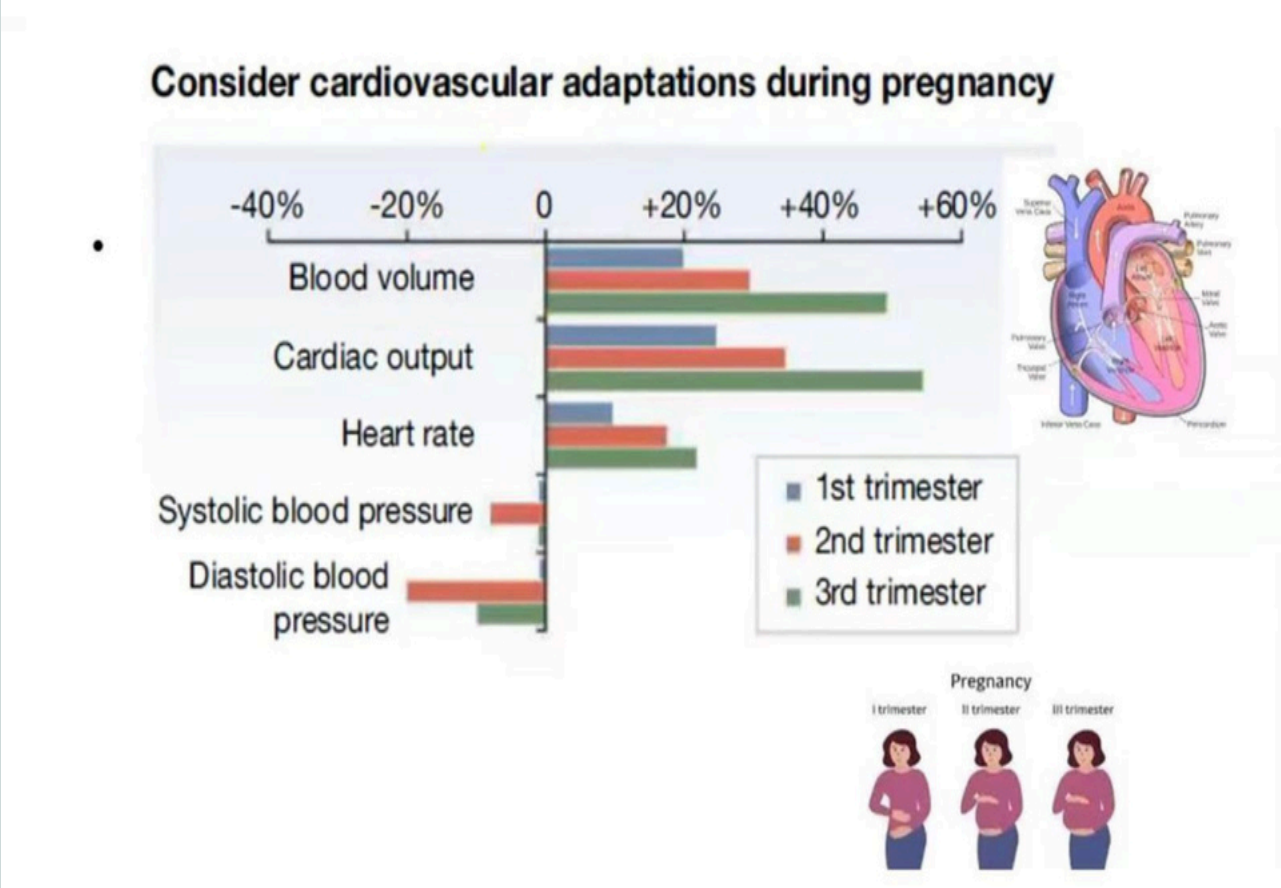
- The earliest and most dramatic changes in maternal physiology are cardiovascular.
- 'These changes improve fetal oxygenation and nutrition.
- *these changes devided into:

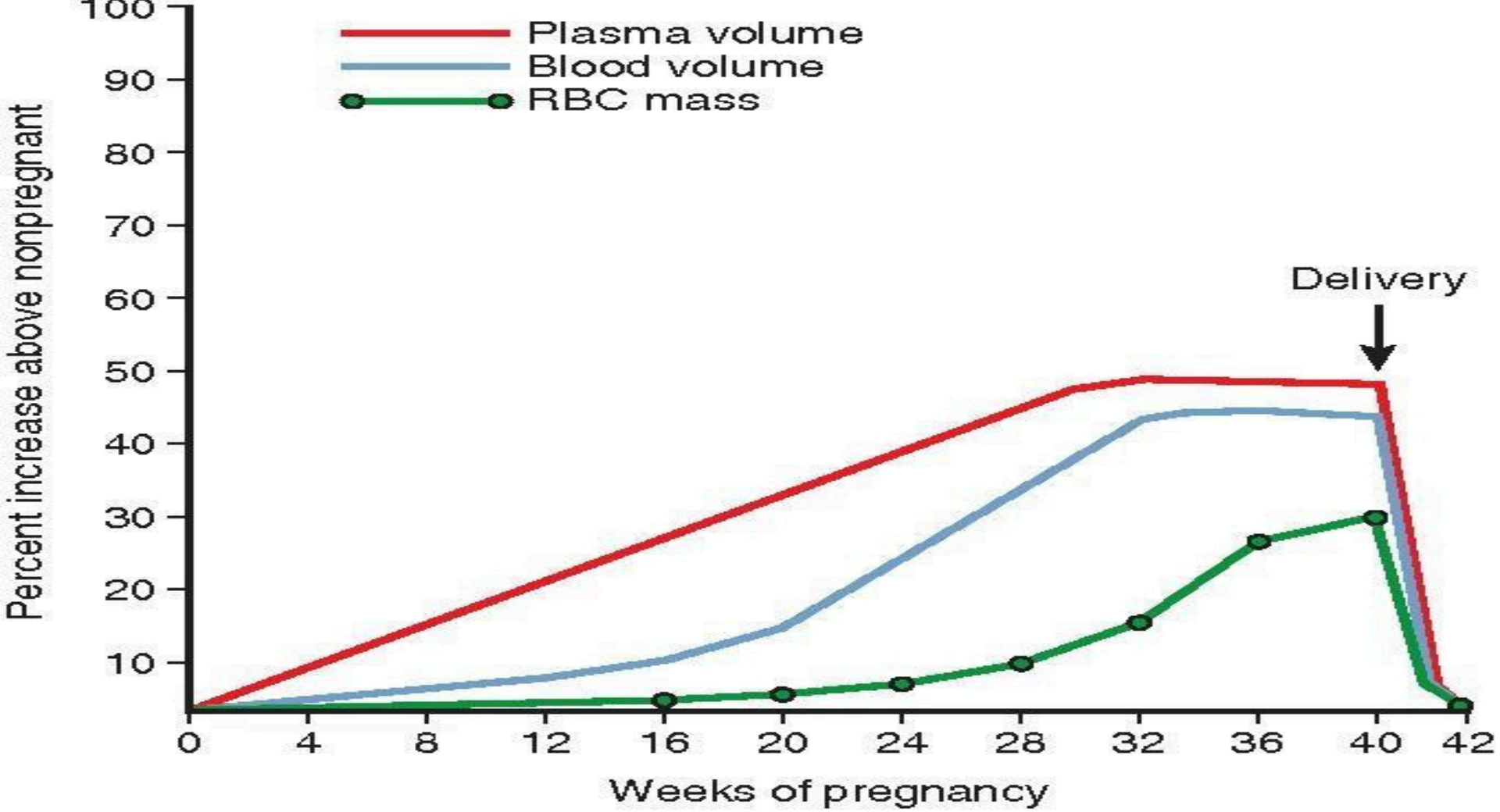



# 1.ANATOMIC CHANGES:

During pregnancy, the heart is displaced upward and to the left and assumes a more horizontal position as its apex is moved laterally. These position changes are the result of diaphragmatic elevation caused by displacement of abdominal viscera by the enlarging uterus. In addition, ventricular muscle mass increases and both the left ventricle and atrium increase in size parallel with an increase in circulating blood volume.

# 2-Functional changes

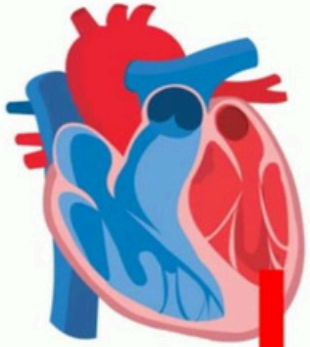



Plasma volume
Blood volume
RBC mass
Delivery
Percent increase above nonpregnant
100
90
80
70
60
50
40
30
20
10
0
4
8
12
16
20
24
28
32
36
40
42
Weeks of pregnancy

# 2.FUNCTIONAL CHANGES:

** The primary functional change in the cardiovascular system in pregnancy is **a marked increase in cardiac output.**

- **# Blood volume:**
- During pregnancy, the total blood volume increases by about 1.5 liters, mainly to supply the demands of the new vascular bed and to compensate for blood loss occurring at delivery, also to cope with the very large increases in blood flow to organs which require little extra oxygen, the skin ,uterus,breast and the kidneys. Of this, around one liter of blood is contained within the uterus and maternal blood spaces of the placenta. Increase in blood volume is, therefore, more marked in multiple pregnancies and in iron deficient states.
- Expansion of plasma volume occurs by 10–15 % at 6–12 weeks of gestation.
- Red cell mass (driven by an increase in maternal erythropoietin production) also increases, but relatively less, compared with the increase in plasma volume, the net result being a dip in hemoglobin concentration. Thus, there is dilutional anemia.



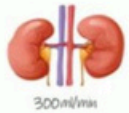
**CARDIAC OUTPUT** = **STROKE VOLUME** x **HEART RATE**

↑ ↑ ↑

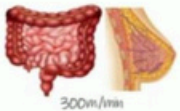
40% 25-30% 10%

4.5 L/min → 6 L/min

80 bpm → 90 bpm





**# Heart rate:** -Beginning to raise in the 1st trimester.The degree of HR increase is depending on the
pt, however its likely that this increase will not exceed 100b/min.

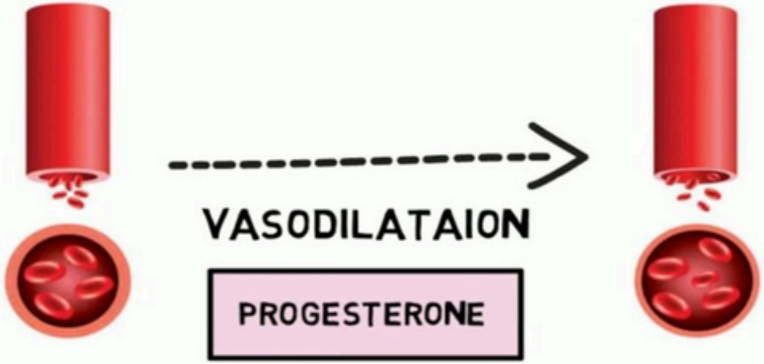
## # Stroke volume:

These changes in stroke volume are due to alterations in circulating blood volume and systemic vascular resistance. Circulating blood volume begins increasing by 6 to 8 weeks' gestation and reaches a peak increase of 45% by 32 weeks' gestation. Systemic vascular resistance
decreases because of a combination of the smooth muscle- relaxing effect of progesterone, increased production of vasodilatory substances **(prostaglandins, nitric oxide, atrial natriuretic peptide),** and arteriovenous shunting to the uteroplacental circulation.

## # Cardiac output: CO=SV*HR

Overall, cardiac output increases 30% to 50%, with 50% of that increase occurring by 8 weeks' gestation. In the first half of pregnancy, cardiac output rises as a result of increased stroke volume, and in the latter half of pregnancy, as a result of increased maternal heart rate while the stroke volume returns to near-normal, nonpregnant levels..

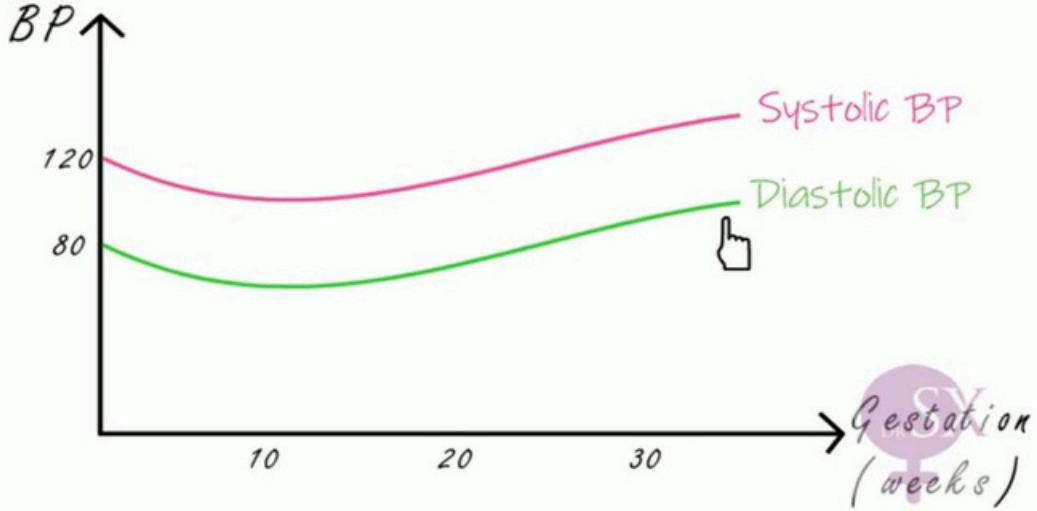
PERIPHERAL VASCULAR RESISTANCE ↓ 20-30%




BLOOD PRESSURE ↓

BP = CO X SVR

↓ SVR → ↓ BP



## # Systemic vascular resistance :

- Systemic vasodilation begins at approximately five weeks of gestation and SVR drops progressively throughout pregnancy.
- The decline in SVR can be attributed to the high-flow, low-resistance circuit in the uteroplacental circulation and vasodilatation.
- The factors responsible for vasodilatation are incompletely understood, but one of the major findings is decreased vascular responsiveness to the pressor effects of angiotensin II and norepinephrine and also due to the effect of the dominant hormone in pregnancy which is **progesterone** .

## # Blood pressure: BP=CO*SVR

-Arterial pressure usually declines to a nadir at 24 to 26 weeks gestation and rises thereafter. Diastolic pressure decreases more than systolic >>> **decreased MAP**.

**-Changes in posture affect arterial blood pressure.**

## # Central venous pressure and EF:

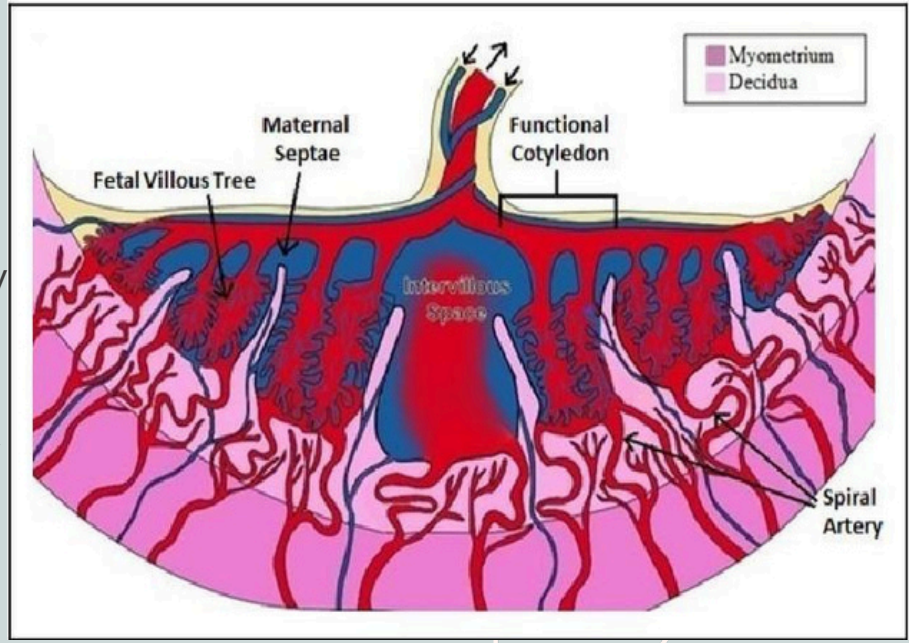
- There is a lot of blood during pregnancy, the venous return as a consequency will be increased but the heart of pregnant women will be compensated .How?? There is a ventricular remodeling in the cardiac myocytes so the CVP and EF they both remain same during pregnancy. **(UNCHANGED)**

— central venous pressure remains in the normal range throughout pregnancy due to the reduction in cardiac afterload induced by the substantial decrease in both SVR and pulmonary vascular resistance.

# Uteroplacental circulation:

The uteroplacentalcirculationsupportsfetal growth and development.

During pregnancy, uterine vascular tone is relaxed due to the varying effects of variety of factors, including nitric oxide, renin-angiotensin, estrogen,progesterone,and prostacyclin, as well as uterine spiral artery dilation due to vascular remodeling in response todecidualnatural killer(NK)cell activity and extravillouscytotrophoblast inv as ion



# Supine Hypotension Syndrome

-occurs within 3 to 10 minutes of lying supine

-Late in pregnancy, when the mother assumes the supine position, the gravid uterus compresses the inferior vena cava and decreases venous return to the heart. This results in decreased cardiac output and symptoms of dizziness, light-headedness, and syncope. This significant arterial hypotension resulting from inferior vena cava compression is known as supine hypotensive syndrome or inferior vena cava syndrome. Therefore, it is not recommended that women remain in the supine position for any prolonged period of time in the latter part of pregnancy. When patients describe symptoms of the supine hypotensive syndrome, there is no need to proceed with additiona cardiac or pulmonary workup

-The earliest sign of developing supine hypotension is an increase in maternal heart rate and a decrease in pulse pressure, indicating significantly reduced venous return, these alterations are the best indicators of an impending attack, many individuals remain asymptomatic

# SUPINE HYPOVENTILATION SYNDROME

Compression of IVC

↓ Venous Return

↓ Cardiac output

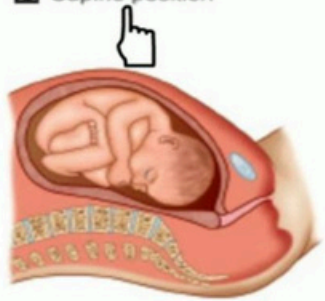
↓ BP

+ Fetal distress

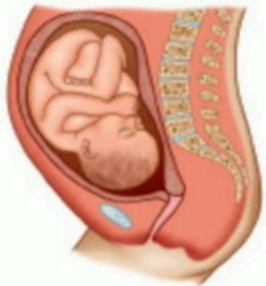
A Supine position

B Lateral position

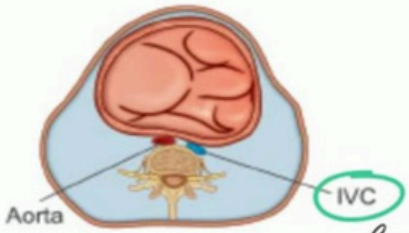
LEFT LATERAL POSITION

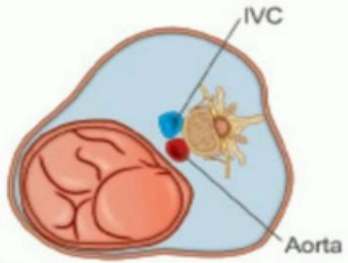


Side view



Top view




Compression of IVC

*A reduction in placental perfusion may result in non-reassuring changes in the fetal heart rate, even with no or minimal decrease in upper extremity maternal BP. Therefore, it is important to avoid the supine position even in symptom-free patients and to ensure that pregnant patients are positioned in the left lateral tilt position for procedures (e.g. labor and delivery, surgery, nonstress test, ultrasound), particularly after 20 weeks of gestation.

The venous system is a high-fow, low-resistance circulation during pregnancy . While antecubital venous pressure remains unchanged, in the supine position, femoral venous pressure rises steadily, from approximately 8 mm Hg early in pregnancy to 24 mm Hg at term. Venous blood flow in the legs is retarded except when the lateral recumbent position is assumed. This tendency toward blood stagnation in the lower extremities during later pregnancy is attributable to occlusion of the pelvic veins and inferior vena cava by the enlarged uterus. The elevated venous pressure returns to normal when the pregnant woman lies on her side and immediately after delivery. These alterations contribute to the dependent edema frequently experienced and to development of hemorrhoids and varicose veins in the legs and vulva. These changes also predispose to deep-vein thrombosis and pulmonary embolism.

# INTRAPARTUM CHANGES

- Significant hemodynamic changes can occur intrapartum duetomultiplefactors,suchaspain,uterinecontractions, exertion, uterine involution, hemorrhage, infection, and administrationofmedications,suchasforanesthesia, analgesia, or tocolysis (eg, terbutaline, nifedipine).

# 1) Cardiac output

- In patients without epidural anesthesia, basal cardiac output between contractions increases by12 percentaboveprelabor levels . During contractions, cardiac output increases progressively as labor advances, increasing by a mean value of 34 percent above prelabor levels at full dilation.The increase isdue to blood forcedinto the systemic circulation from the uterine sinusoids with each uterine contraction as well as pain, thereby increasing preload . While epidural anesthesia reduces the increase due to pain, the increase associated with uterine contractions persists.
- Inthe second stage, exertionassociated withpushingincreases cardiac output up to 50 percent above the prelabour level.

# 2) Blood pressure

• During each uterine contraction, systolic and diastolic BP can
increase 15- 25% and 10-15%, respectively. The rise in systemic BPdependsuponthe durationandintensityofthecontractions, theparturient'sposition(changes areminimizedintheleft
lateral position), and the degree of pain and anxiety experienced

• The increases in arterial pressure associated with each uterine contraction aremirroredbyariseinpressureintheamniotic fluid,intrathoracicveins, cerebrospinalfluid,andextradural compartment. • Pushing during the second stage alters the BP and heart rate inasimilar waytotheValsalvamaneuver

# The hemodynamic changes resulting from a Valsalva maneuver vary with its different phases:

- **During phase 1** (onset of the maneuver), left ventricular output transiently increases.
- **During phase 2** (straining phase), venous return, right and left ventricular volumes, stroke volumes, mean arterial pressure, and pulse pressure decrease accompanied by a reflex increase in heart rate.
- **During phase 3** (release of Valsalva), which only lasts for a few cardiac cycles, left ventricular volume is further reduced.
- **During phase 4,** stroke volume andarterial pressure increase accompanied by reflex slowing of the heart rate (the overshoot).

# HEMATOLOGICAL CHANGES

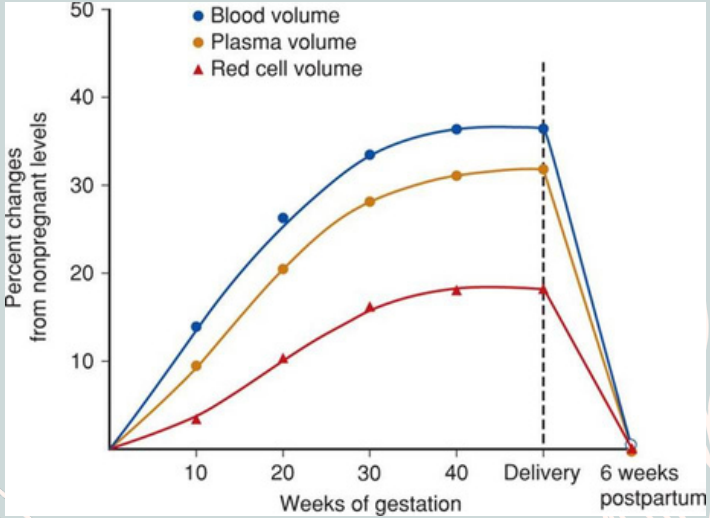
# Normal physiological changes

Pregnancy-induced hypervolemia serves several functions.

1. First, it meets the increased metabolic demands of the enlarged uterus and its greatly hypertrophied vascular system.
2. It also provides abundant nutrients and elements to support the rapidly growing placenta and fetus and waste removal
3. The expanded intravascular volume protects the mother, and in turn the fetus, against the effects of impaired venous return in the supine and erect positions. Last, it protect the mother from excessive blood loss during delivery

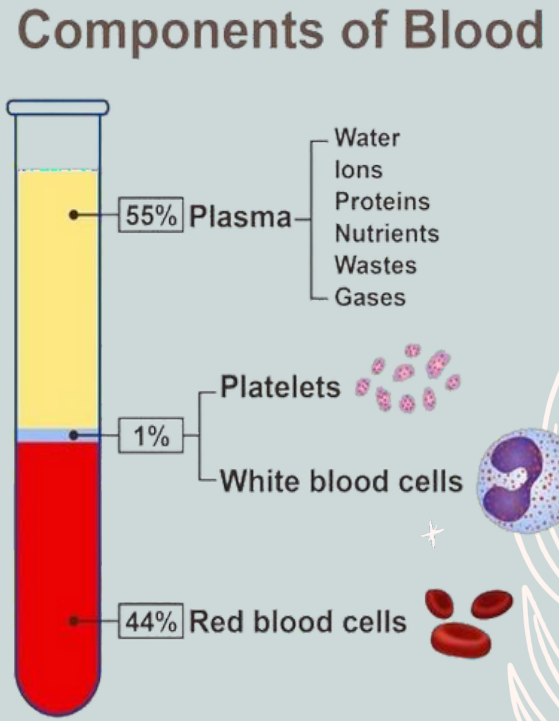
- Maternal blood volume begins to arise during the first trimester. By 12 menstrual weeks, plasma volume expands by approximately 15 percent compared with that prior to pregnancy

. Maternal blood volume grows most rapidly during the midtrimester, rises at a much slower rate during the third trimester, and reaches a plateau during the last several weeks of pregnancy . Blood volume arise  even more dramatically in twin gestations.



- During blood volume expansion, plasma volume and erythrocyte mass increase. resultant physiologicanemia

- Mild neutrophilia
- Mild thrombocytopenia
- Increased procoagulant factors
- Decreased natural anticoagulants
- Diminished fibrinolysis



# Hematologic changes of specific blood Components during pregnancy

## 1) Plasma volume

- Plasma volume increases by 10-15% at 6 to 12 weeks of gestation, expands rapidly until 30 to 34 weeks, and then plateaus or decreases slightly through term The total **gain at term averages 1100 to 1600 mL** and results in a total plasma volume of 4700 to 5200 mL, which is 30-50% above that in nonpregnant women Total plasma volume expansion is accompanied by retention of sodium and water, which is distributed among the fetus, amniotic fluid, and extracellular and intracellular spaces
- Triggered by systemic vasodilation and hormonal changes (↑ renin, ↓ atrial natriuretic peptide)

The hypothesis that vascular changes precede expansion of the plasma volume is also supported by the observation that increasing sodium intake does not lead to further volume expansion

# 2) Red blood cells

- ✓ Red blood cell (RBC) mass begins to increase at 8 to 10 weeks of gestation, steadily rises, and **reaches levels 20-30% higher** than in nonpregnant females by the end of pregnancy
- ✓ The increased RBC mass is accompanied by a **slight increase in the mean corpuscular volume** MCV in healthy pregnant people . However, the **increase in mass is smaller than the increase in plasma volume,which** **contributes to the physiologic anemia of pregnancy.**
- ✓ The **major mediator** of increased RBC mass is an increase in **erythropoietin**, which stimulates RBC production. **Erythropoietin levels increase by 50percent**
- ✓ The increase in RBC mass requires sufficient iron, folate, and vitamin B12; thus, individuals with deficiencies of iron or these vitamins will have blunted increases in RBC mass and are likely to develop more severe anemia

# Dilutional/physiologic anemia

the normal pregnancies , relative expansion of plasma volume relative to the increase in RBC mass is associated with a modest decrease in hemoglobin concentration ,which is referred to as physiologic or dilutioal anemia of pregnancy

The greatest disproportion between the rates at which plasma and RBCs are added to the maternal circulation occurs during the late second to early third trimester ,thus the lowest hemoglobin concentration is typically measured at 28 to 36 weeks
nearer to term , hemoglobin concentration increases due to cessation of plasma expansion and continuing increase in RBC mass

# Dilutional/physiologic anemia

*thresholds for diagnosing anemia in pregnancy are:*

- CDC criteria : For the first and third trimesters, anemia is defined as Hb less than 11 g/dL or Hct less than 33%. In the second trimester, it's defined as Hb less than 10.5 g/dL or Hct less than 32%

- The WHO – **Anemia** in pregnancy is defined as a hemoglobin level <110 g/L (<**11** g/dL) or a hematocrit <6.83 mmol/L (<33 percent). **Severe anemia** is defined as a hemoglobin level <70 g/L (<7 g/dL). **Very severe** anemia is defined as hemoglobin <40 g/L (<4 g/dL).

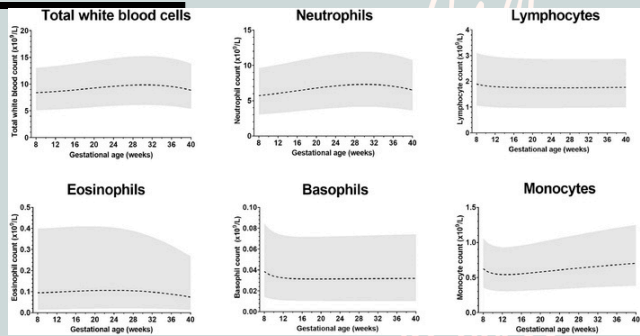
# 3) White blood cells

An increase in WBC associated with fever, a large number of immature WBC forms, or any blasts in the peripheral blood are not normal and should be evaluated promptly.

The neutrophil count begins to increase in the second month of pregnancy and plateaus in the second or third trimester, at which time WBC counts range is 9000-15,000 cells/microL

Mean WBC counts in laboring patients were 10,000 to 16,000 cells/microL

- ✓ The absolute lymphocyte count and the relative numbers of T and B lymphocytes **do not change**
- ✓ The monocyte count is generally stable.
- ✓ The basophil count may **decrease**slightly.
- ✓ The eosinophil count may increase slightly
- ✓



# 4) Platelets(normalrange150,000-450,000)

- Platelet count declines as pregnancy progresses but in the vast majority of uncomplicated pregnancies, the platelet count remains >=100,000/microL and returns to the prepregnancy baseline level by several weeks postpartum.

• The most common cause of a decline in platelet count is a normal physiologic response (hemodilution) referred to as gestational thrombocytopenia (GT; also called incidental thrombocytopenia of pregnancy). GT is a diagnosis of exclusion and may recur in subsequent pregnancies. We generally do not evaluate patients with a mild decrease in platelet count during pregnancy if they are asymptomatic and their platelet count is >=100,000/microL

---

• Moderate to severe thrombocytopenia (platelet count <100,000/microL) is rare in pregnancy, but when it occurs, it may be a medical emergency.

• Possible causes include:

- Immune thrombocytopenia,
- Preeclampsia with severe features,
- Sepsis with disseminated intravascular coagulation
- HELLP syndrome (syndrome of hemolysis, elevated liver enzymes, and low platelets)

# Coagulation and fibrinolysis

Normal pregnancy is a prothrombotic state ( hypercoagulable )

The shift in the balance between the hemostatic and fibrinolytic systems serves to prevent excessive hemorrhage during placental separation. Compared with nonpregnant females, pregnant people have a marked increase in some coagulation factors, reduced fibrinolysis, and increased platelet reactivity. Consequently, there is increased risk for thromboembolic complications. While these changes increase the risk of thrombosis, they are not an indication for intervention.

➢ Increased procoagulant factors:

Procoagulant factors fibrinogen (factor I), factors II, VII, VIII, and X.

• The prohemostatic von Willebrand factor (VWF) can increase substantially during pregnancy. Studies have reported that VWF increases by two- to fourfold during pregnancy, peaks within 24 hours postpartum, and returns to baseline by one month postpartum

# Coagulation and fibrinolysis

## Reduced anticoagulant factors

- Anticoagulant protein S decreases physiologically in nearly all pregnant people(ifpatient develops venous thromboembolism andinherited thrombophiliais suspected, testing forproteinSlevel shouldbedelayedtill after delivery)
- antithrombin (AT) levels, protein C,  factor V, factor XI remain unchanged

## Reduced fibrinolysis

- Activity of fibrinolytic inhibitors increases, including **thrombin activatable fibrinolyticinhibitor**, **plasminogenactivatorinhibitor-1(PAI-1), andPAI-2** .PAI-1 levels increase markedly since it is partly derived from the placenta and decidua.

# skin changes

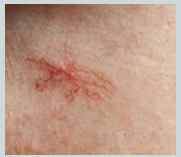
# 1) Hyper pigmentation

**These are the most noticeable and affect over 90% of pregnant women due to increase Levels of melanocyte-stimulating hormone, and estrogen and progesterone also are reported to have melanocyte-stimulating effects.**

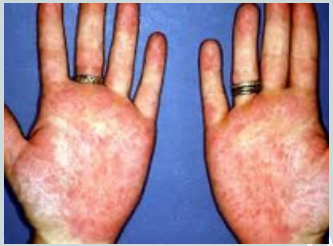
**Of specific sites, the pigmented skin line in the midline of the anterior abdominal wall-the linea alba- takes on dark brown-black pigmentation to form the linea nigra. Occasionally, irregular brownish patches of varying size appear on the face and neck, giving rise to chloasma or melasma gravidarum-the mask of pregnancy. Pigmentation of the areolae and genital skin also may be accentuated. After delivery, these pigmentary changes usually disappear or at least regress considerably. Oral contraceptives may cause similar alterations**

# 2) Vascular Changes

**They are likely the consequence of hyperestrogenemia. In addition to these discrete lesions, increased cutaneous blood flow in** pregnancy



- **Spider Angiomas, called vascular spiders, Small, dilated blood vessels with radiating capillaries are particularly common on the face, neck, upper chest, and arms.**
- **Palmar erythema is Redness on the palms (especially the thenar and hypothenar areas). Seen in ~2/3 of white and ~1/3 of Black pregnant women.**



**3) Hair and Nail Changes**

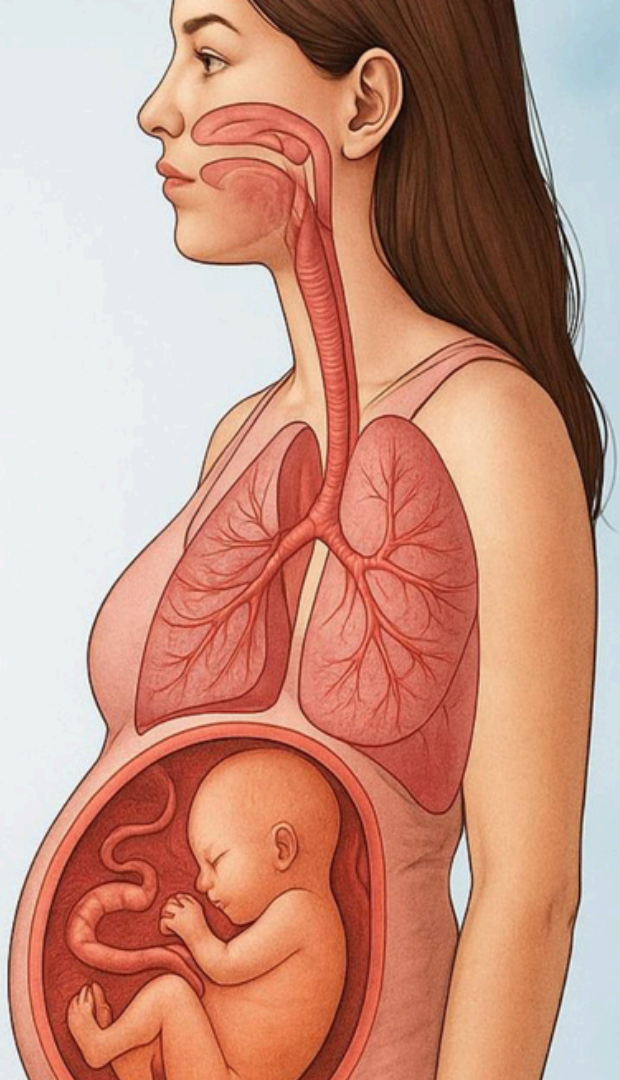
**Hair**

- **Increased hair growth during pregnancy**

**Nails**

- **May grow faster.**

# Respiratory System Changes During Pregnancy



- Respiratory changes during pregnancy are important to accommodate and meet the demands of mother and foetus, there are changes in all lung volumes, changes in the upper airway respiratory tract, and breathing pattern.

# Anatomical Changes

- The diaphragm is elevated by about 4cm due to the enlarged uterus.
- Ligaments connecting ribs to sternum become lax during pregnancy. The subcostal angle increases from 68 in early pregnancy to 103 in late pregnancy. Chest circumference increase from 5-7cm and this is associated with lower chest compliance.

# Blood gases

- Oxygen consumption increases by 20% to 40% during pregnancy, as the oxygen demand of the growing fetus, placenta, and increased metabolic activity of the maternal organs all increase the pregnant woman's overall oxygen requirements.

# Blood Gases

- Progesterone stimulates respiration and can lead to hyperventilation (exhaling more than inhaling). Due to this, the arterial partial pressure of oxygen ($PaO_2$) increases to 105 mmHg, while the arterial partial pressure of carbon dioxide ($PaCO_2$) decreases to approximately 30 mmHg. This blood gas change results in slight respiratory alkalosis metabolically compensated by increased bicarbonate excretion by the kidneys to approximately 20 mEq/L. The oxyhemoglobin dissociation curve is shifted to the right, favoring oxygen dissociation and facilitating oxygen transfer across the placenta.

| *Investigations* | *Normal values* | |
|---|---|---|
| | *Pregnant* | *Non-pregnant* |
| pH | 7.40–7.47 | 7.35–7.45 |
| $pCO_2$, mmHg (kPa) | ≤ 30 (3.6–4.3) | 35–40 (4.7–6.0) |
| $pO_2$, mmHg (kPa) | 100–104 (12.6–14.0) | 90–100 (10.6–14.0) |
| Base excess | No change | +2 to –2 |
| Bicarbonate (mmol/l) | 18–22 | 20–28 |

- Functional residual capacity is the sum of expiratory reserve volume and residual volume. Throughout pregnancy, due to the enlarging uterus, the resting position of the diaphragm shifts up approximately 5 cm, leading to bibasilar alveolar collapse and basilar atelectasis, thus decreasing the expiratory reserve volume and functional residual capacity. Vital capacity remains unchanged, as reduced expiratory reserve volume is accompanied by an increased inspiratory reserve volume. Increased progesterone concentrations, beginning in the first trimester, cause an increase in tidal volume by approximately 30-50%. The product of tidal volume and respiratory rate is minute ventilation, which increase by 30-50%. The respiratory rate remains unchanged from the nonpregnant state.

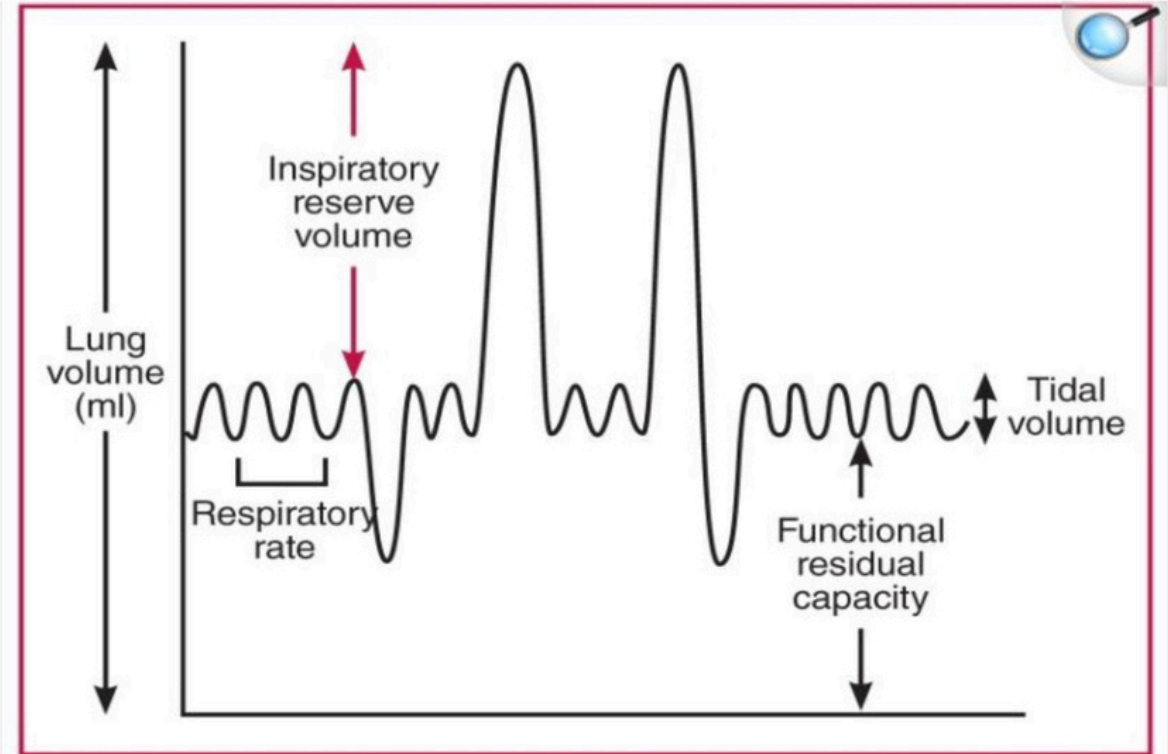
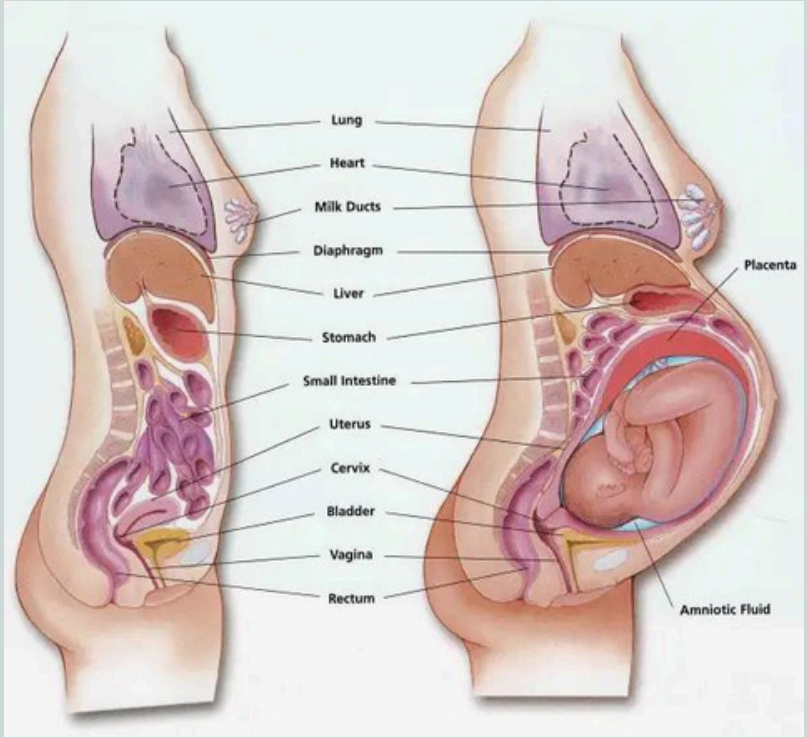



**Fig. 1.** Physiological changes in respiratory function in pregnancy.

- Pregnancy may also be accompanied by a subjective feeling of breathlessness without hypoxia. This is physiological and is most common in the third trimester but may start at any time during gestation. Classically, the breathlessness is present at rest or while talking and may paradoxically improve during mild activity.

- During labor, minute ventilation increases by as much as 140-200% depending on the stage of labor, leading to an even more pronounced decrease in $PaCO_2$.
- Metabolic oxygen consumption rises during childbirth due to uterine contractions, sympathetic activity, and maternal Valsalva maneuvers to deliver the fetus.
- As oxygen demand outpaces oxygen delivery during active labor, anaerobic metabolism ensues, and lactic acid production occurs.

# Gastrointestinal changes



# Oropharynx and Taste:

## Orophary nx

• The mucous membrane lining the oropharynx is responsive to the hormonal changes related to pregnancy. The gingiva is primarily affected, while the teeth, tongue, and salivary glands are spared, although excessive salivation during pregnancy (ptyalism) has been described.

## Taste

• Most studies suggest that taste perception changes during pregnancy. The etiology is unknown, and the direction of taste change varies among studies.

# Gingiva

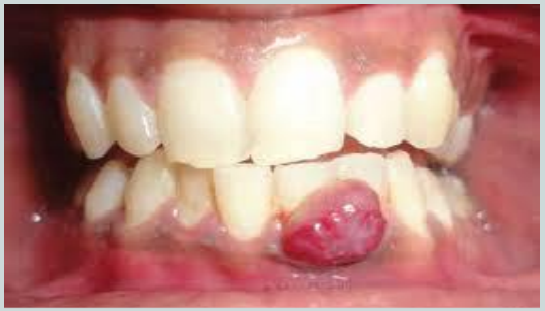
- Enlargement and blunting of the interdental papillae of the gingiva may result in gingivitis.

- Elevated circulating estrogen and progesterone levels are implicated in increasing vascular permeability and decreasing immune resistance, thereby increasing susceptibility to gingivitis.

# *Pyogenic granuloma of pregnancy*

Pyogenic granulomas (also known as **lobular capillary hemangioma**, pregnancy tumor or epulis, and granuloma gravidarum) are benign, vascular tumors with friable surfaces that develop over a few days to weeks in early pregnancy.

The **oral mucosa, lip, and tongue** are common sites of occurrence



# Ptyalismor sialorrheagravidarum

- Ptyalism or sialorrhea of pregnancy is an oral pathological condition consisting of ***excessive salivation*** that typically begins in the first trimester.
- Symptoms generally abate in the second trimester but can continue to term. Salivary volumes range from **1.5 L to2 L per day** .Reported incidences range widely from0.08 to35 percent and depend upon the definition used (eg, inclusion of patients with pseudo-sialorrhea).Ptyalism can be bothersome, but it is not associated with increased maternal or perinatal morbidity.

- The mechanism in pregnancy is not known. It is commonly associated with nausea and vomiting as well as hyperemesis gravidarum. Causes of ptyalism unrelated topregnancy include gastroesophageal reflux, medications (eg, clozapine), or irritants(eg, smoking)

- For patients with a potential identified cause, treatment or removal of the underlying cause reduces excessive salivation. For those with symptoms resulting only from pregnancy, treatment is mainly aimed at lessening symptoms.
  Patients have reported relief with frequent expectoration, chewing gum or using lozenges ,frequent sips of water, and/or antiemetics.

## Teeth:-

The effect of pregnancy on the initiation or progression of caries is not clear; pregnancy-related changes in the oral environment (salivary pH, oral flora) or in maternal diet and oral hygiene may increase the risk of caries

# ESOPHAGUS AND STOMACH

Stomach

- Reduced tone of the gastroesophageal junction sphincte
- Increases production of Gastrin hormone significantly, resulting inincreased stomach volume and decreased stomach pH

Gastric emptying is affected by pregnancy and it is prolonged during

labor if sedatives or
opioids are administered and in

patients with diabetes who have gastroparesis

# Gastroesophageal reflux

Gastroesophagealreflux(GERD,orheartburn) isreportedby40 to 85 percent of pregnant people .

Most studies describe an increasing prevalence of symptoms from the first to the third trimester, with relief postpartum .

While GERD symptoms can be severe, erosive gastropathy and

other complications are rare

GERD tends to recur in subsequent pregnancies, and similarly affects multiparous and nulliparous individuals.

The pathogenesis of GERD during pregnancy involves both mechanical and intrinsic factors that adversely affect lower esophageal sphincter tone . Lower esophageal sphincter pressure is below the lower limits of normal in all trimesters, returning to normal in the postpartum period.

•A study of pregnant people during early pregnancy and then six weeks after pregnancy termination also demonstrated a reduced response of the sphincter to injections with pentagastrin, edrophonium, and methacholine , or a protein meal. Thus, it appears that pregnancy is associated with both decreased lower esophageal sphincter pressure and inhibition of the adaptive responses of the sphincter

# Aspiration of gastric contents

Pregnant people are at increased risk of aspiration during labor, birth, and immediately postpartum dueto supine positioning, analgesia/anesthesia, and a combination of factors related to pregnancy(lower esophageal sphincter incompetence, gastroesophageal reflux, low gastric pH, distortion of the gastric anatomy due to the enlarging uterus, increased intra-abdominal pressure). Aspiration may also occur as a complication of intubation for general anesthesia for cesarean birth. Aspiration pneumonia, acute bronchospasm, or the acute respiratory distress syndrome may ensue.

# Liver

During pregnancy, absolute hepatic blood flow remains largely unaltered and hepatic Function remain normal.

Position: In late pregnancy, the liver becomes difficult to palpate because of the expanding uterus. As the enlarging uterus pushes the diaphragm upwards to a maximum of 4cm, the liver is forced further up into the chest as well. **A palpable liver is an abnormal finding** .
The biliary tract however isusually normal.

- The **PT and aPTT are unchanged or slightly reduced**
- **Serum fibrinogen is markedly increased(Esp in late pregnancy)**

# Blood chemistries

•**Serum albumin levels decrease** during the first trimester because of hemodilution , and this decrease becomes more accentuated with advancing gestation.

•**Serum alkaline phosphatase concentrations are significantly higher (**up to two to four times normal**)** in the third trimester, primarily due to placental synthesis of alkaline phosphatase •**Serum gamma-glutamyltranspeptidase is significantly reduced** and **5-nucleotidase is slightly increased**

**Blood chemical constituent changes during pregnancy**

| | Nonpregnant adult | First trimester | Second trimester | Third trimester |
|---|---|---|---|---|
| **Alanine aminotransferase** (unit/L) | 7 to 41 | 3 to 30 | 2 to 33 | 2 to 25 |
| Albumin (g/dL) | 4.1 to 5.3 | 3.1 to 5.1 | 2.6 to 4.5 | 2.3 to 4.2 |
| Alkaline phosphatase (unit/L) | 33 to 96 | 17 to 88 | 25 to 126 | 38 to 229 |
| **Alpha-1 antitrypsin** (mg/dL) | 100 to 200 | 225 to 323 | 273 to 391 | 327 to 487 |
| **Alpha-fetoprotein** (ng/mL) | - | - | Approximately 130 to 400 | Approximately 130 to 590 |
| Gamma-glutamyl transpeptidase (unit/L) | 9 to 58 | 2 to 23 | 4 to 22 | 3 to 26 |

**Serum total cholesterol and triglyceride concentrations increase markedly.** Values differ between studies, but the results of a large series are show in in the table below. •Since the increase in total cholesterol, LDL and triglycerides is physiologic, **treatment is not indicated**

**•Patients receiving statin therapy should stop it three months prior to conception**

**An increase** in **serum aminotransferase , bilirubin or fasting total bile acid concentrations** during pregnancy may be **pathologic and should be evaluated**

| Biomarker | Second trimester | Third trimester |
|---|---|---|
| Total triglyceride | 254 mg/dL | 415 mg/dL |
| Total cholesterol | 319 mg/dL | 380 mg/dL |
| LDL | 217 mg/dL | 251 mg/dL |
| HDL | 98 mg/dL | 95 mg/dL |

# Gallbladder

Pregnancy reduces gallbladder motility and increases the lithogenicity of bile (possibility of forming stones).

•OnU/S, fasting gallbladder and residual volume after contraction may be increased but the size of the common hepatic duct remains unchanged .

•Overall, studies have shown that pregnancy increases risk for gallstones.

# Pancreas

There isn't sufficient information on the effect of pregnancy on pancreatic secretion, but amylase levels have been reported to be normal or slightly increased.

•Acute pancreatitis in pregnant women is a rare but potentially serious complication, and even a life-threatening disease for the mother and fetus. The greatest risk of complications is associated with acute pancreatitis caused by hypertriglyceridemia .

•Most cases of acute pancreatitis in pregnancy are associated with gallstones and the incidence increases with advancing gestational

# Bowel, rectum, anus

Bloating and constipation: it is a frequent complaint among pregnant women, as some studies suggest a prevalence of 16% to 39% during pregnancy and postpartum (higher than base line of 7%)

•Suggested causes of bloating and constipation include **increased progesterone concentration** which decreases the activity of colonic smooth muscle and increases intestinal transit time. Progesterone is also thought to inhibit motilin release which isa stimulatory gastrointestinal hormone .

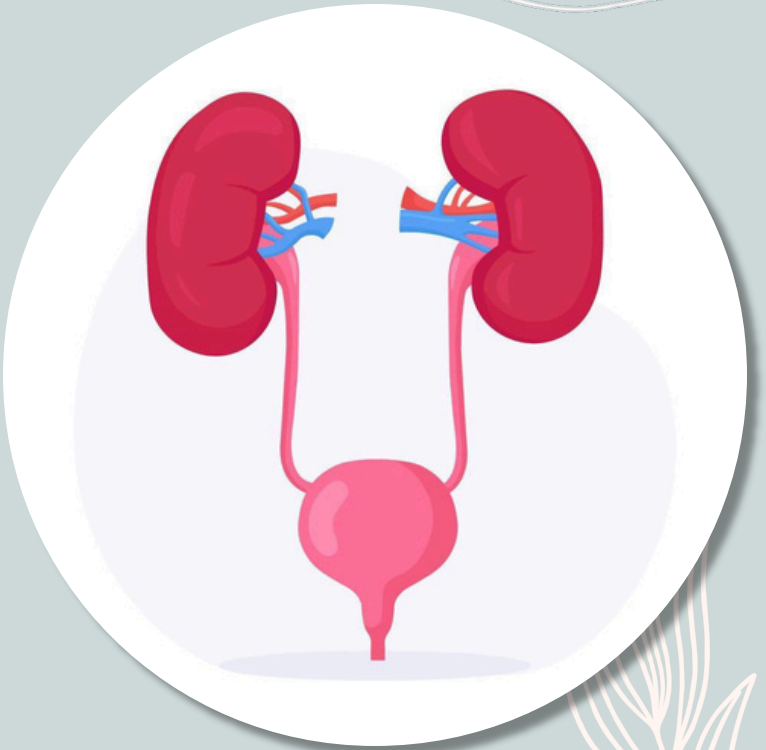
•Moreover, the g**ravid uterus mechanically impedes small bowel transit.**

**Hemorrhoidal disease** is particularly **frequent in the last trimester and postpartum .**

•Symptoms include pruritis, discomfort, and/or bleeding. And constipation which is prevalent in pregnancy exacerbates these symptoms .

•Treatment includes conservative management like application of anti inflammatory, anti pruritic and local anesthetic agents. Severe cases require surgery which can be safely performed during pregnancy.

# Renal &Urinary tract changes



# Renal changes

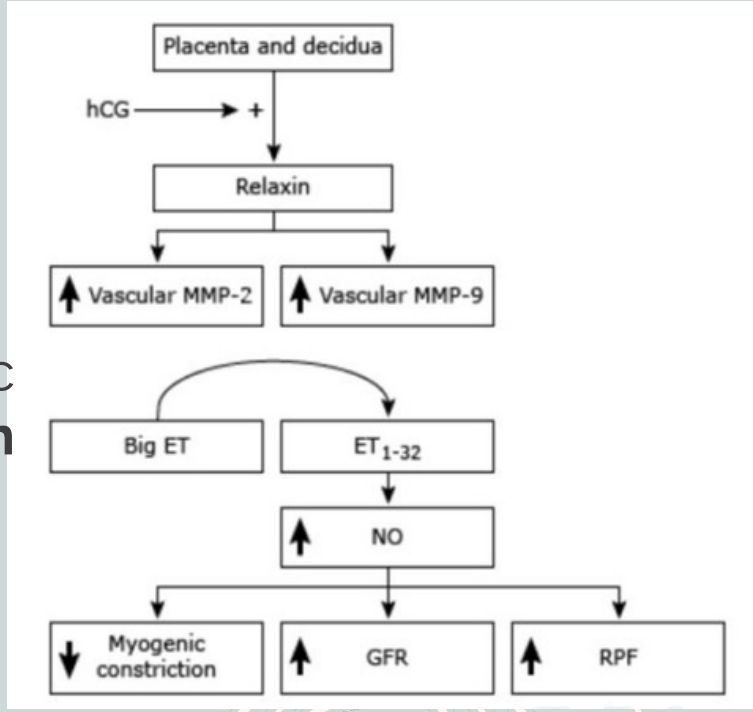
Pregnancy is associated with an increase in renal size ,renal plasma flow and GFR
Both kidney will increase in:

1.Length (1-1.5 )
2.The volumeincreaseby30%dueto:
*increase in renal blood flow (so hypertrophy occurs)
*dilationinpelviccalyces portion(hydronephrosis)
3. The size of collecting system (renal pelvises, caliceal system and ureters), due to progesterone and compression of uterus.

**Nochangesinnumber ofnephrons.
** The hypertrophy doesn't resolve until 3 months p os tpartu m.

# Mechanism of increased renal plasma flow in pregnancy

Normal pregnancy is characterized by **widespread vasodilation**, with **increased arterial compliance**, leading todecreased systemic vascular resistance, increased cardiac output,and a smalldecreasein blood pressure. These global hemodynamic changes include **increased renal perfusion and GFR.**



# Mechanism of increased GFR

1. Reducedvascularresponsiveness to vasopressor (eg:angiotensin 2 , norepinephrine,ADH )
2. Increased nitric oxide synthesis

3. Relaxin which is secreted in large amounts from placenta, Increases endothelinand nitric oxide.

All of them will cause vasodailation

# GFR, renal plasma flow, and creatinine

- **Renal plasma flow increases** by up to 80% by 12w of gestation and then decreases in the third trimester.
- **GFR increases within 1 month** of conception, peaking at 40-50% above baseline levels by the early second trimester, it then declines slightly toward term.
- The increase in GFR results in **decrease in serum creatinine** concentration in the first trimester, it levels off in second trimester and rises again in third trimester.
- Therefore, a **serum creatinine of >0.75** usually **reflects renal impairment** in pregnant women as a **small rise in creatinine reflects a marked reduction in renal function**.

# Laboratory tests changes

- Plasma**osmolalityfalls**from275-290to270mOsm/kg

- Plasma **sodium concentration falls 4-5 mEq/L below pre-pregnancy levels.**

- This hyponatremiais mediated by increased HCG which in turn produces thehyponatremiabythe releaseof relaxin.

- Aserum concentration ofsodium lowerthan 130mEq/L should prompt evaluation for pathological causes of hyponatremia(such as SIADH) and high levels should prompt evaluation forpossible diabetesinsipidus

# Proteinuria and glucosuria

• Urinary **proteinexcretionincreases** inpregnancyitrises to 150-200mg/day(so if urinary proteinis>300mg/day,itis considered abnormal but may be normal in twin pregnancies)

• Mechanisms include **increased GFR**, and **glomerular basement membrane pore size**, and **reduced tubular reabsorption** of filtered protein. • **Glucosuria**:seenin~50%ofpregnantpatients,itisprimarily due to decreased proximal tubular glucose reabsorption. • Other changes: chronic respiratory alkalosis, hypouricemia, and impaired tubular function.

# Urinary tract

• **Ureters**: dilationofuretersand renal pelvis isseen in up to 80% of pregnanciesandismoreprominentontheright.

• The dilated collecting system causes stasis and may increase risk-of pyelonephritis in pregnancy.

• **Factors that contribute to hydroureterand hydronephrosis:**

• Progesterone reduces ureteral tone, and peristalsis
• The Dextrorotated uterus kinks the right ureter
• Enlargedvesselsmaycompressureter
• Uterine enlargement

# Bladder and VUR

- **Bladder**: mucosa isedematousand hyperemic. Progesterone induces bladder wall relaxation, butthe enlarging uterus flattens the bladder and may decrease capacity.
- **Vesicoureteralreflux**: may occur in pregnancy due to bladder flaccidity and decreased intraureteralpressure.

Symptomsinclude : **Frequency and nocturia, dysuria, urgency and stress incontinence.**

# Other urinary symptoms

- **Urgencyandincontinence:**These symptomsmay bedueto uterine pressure onthebladder,hormonaleffectsonthe suspensory ligamentsof theurethra,and/oraltered neuromuscular function ofthe urethral striated sphincter.
- **Urinary retention:**The bladder and urethra inevitably experience some trauma during labor and delivery. The traumatic changes include mucosal congestion and submucosal hemorrhage, Bladder sensitivity/sensation is also decreased from trauma. As a result, detrusor atony, increased postvoidresidual urine, bladder overdistention, and urinary retention are common in the first few days after delivery.
- **Postpartum**: The pregnancy-induced physiologic changes described above return to the nonpregnantstate by four to six weeks following delivery. However, urinary incontinence may persist.

# FETAL CIRCUALATION

# Introduction

- The **fetal circulation**isthecirculatorysystem of a fetus.
- The term usually include the entire fetoplacental circulation, whichincludestheumbilicalcordandthebloodvesselswithin the placenta that carry fetal blood.
- Fetal circulation – **In the fetus, the placenta has the lowest vascular resistance**and receives 40 % of the fetal cardiac output, which **results in a low systemic pressure**
- In contrast, the lungs are filled with fluid, resulting in a high vascularresistanceandasaresultasignificantlyloweramount

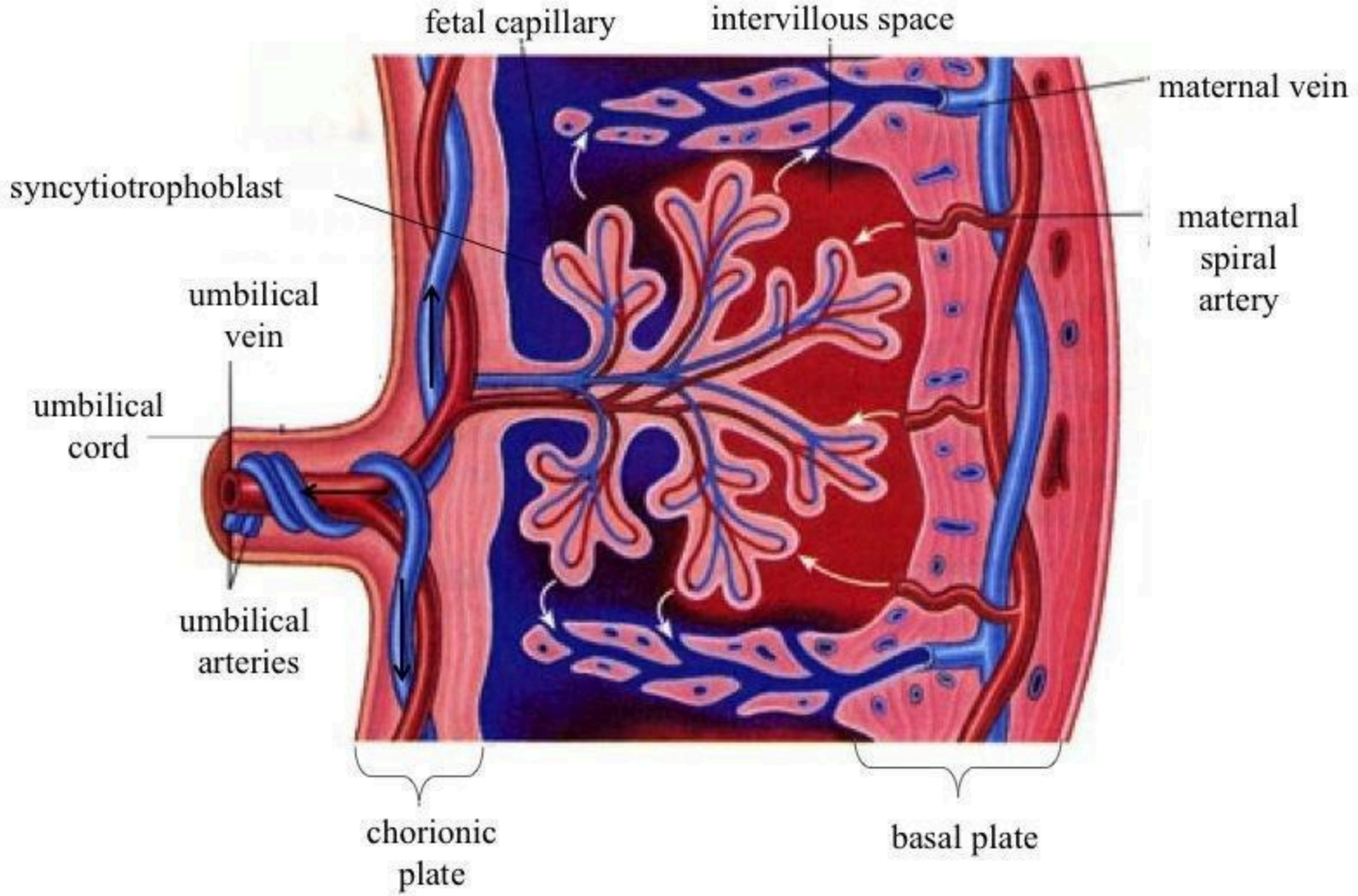
# The placenta

- The **placenta**is anorgan that connectsthedeveloping fetus to the uterine wall to:

1. allow nutrient uptake
2. provide thermoregulation to the fetus
3. waste elimination
4. gas exchange via the mother's blood supply
5. fight against internal infection
6. produce hormones to support pregnancy.

# Placental Role

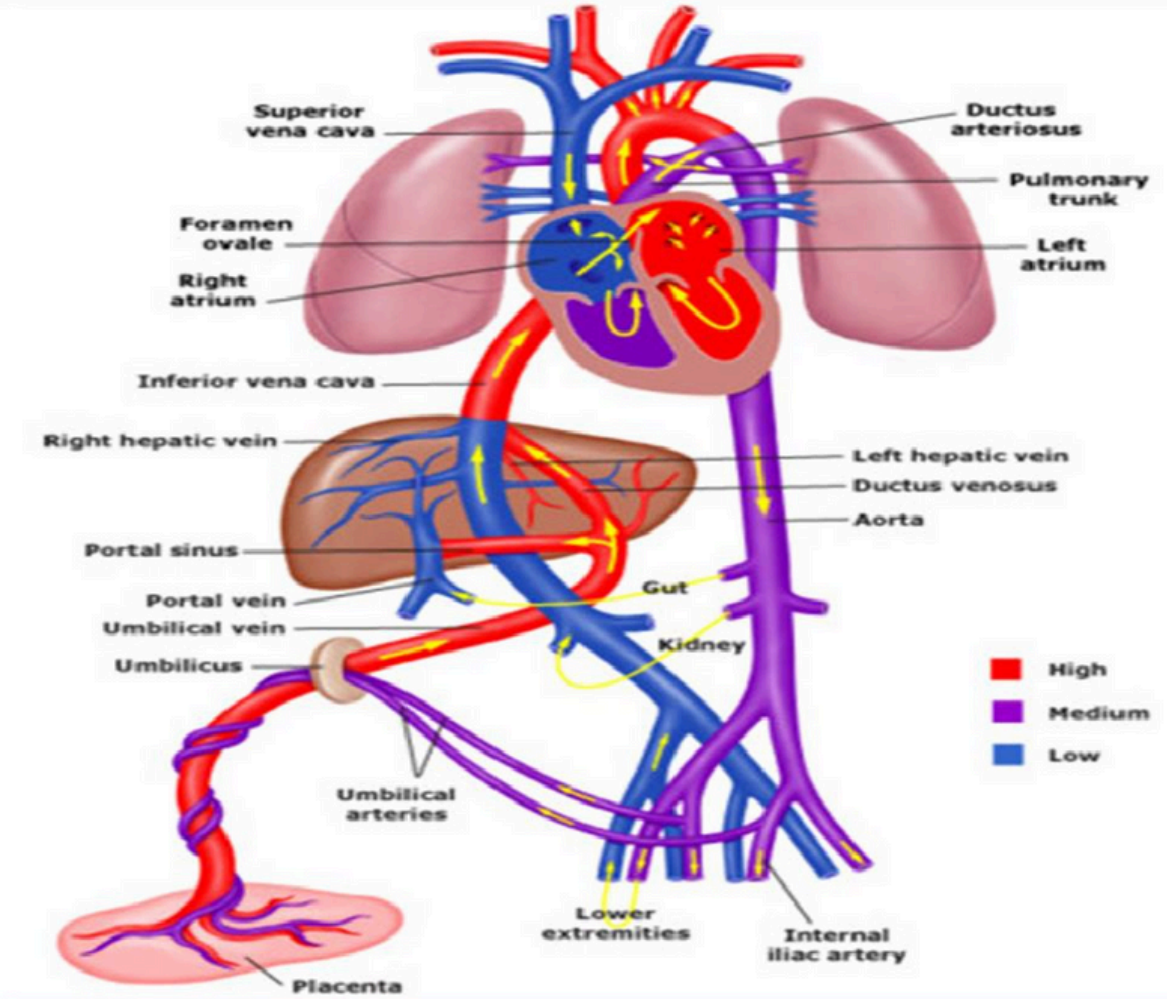
- The **core concept behindfetalcirculation** isthat fetal hemoglobin has a highe affinity for oxygen than adulthemoglobin, which allows diffusion of oxygen from the mother's circulatory system to the fetus.
- The mother's circulatory system is **not directly** connected to the fetus's, **so the placenta functions as the fetus's respiratory center** and **a site of filtration for plasma nutrients and wastes.**
- Water, glucose, amino acids, vitamins, and inorganic salts freely diffuse across the placenta along with oxygen.
- The umbilical arteries carry blood to the placenta, and the blood permeates the sponge-like material there ( villi ). Oxygen then diffuses from the placenta to the chorionic villus, an alveolus-like structure, where it is then carried to the umbilical vein

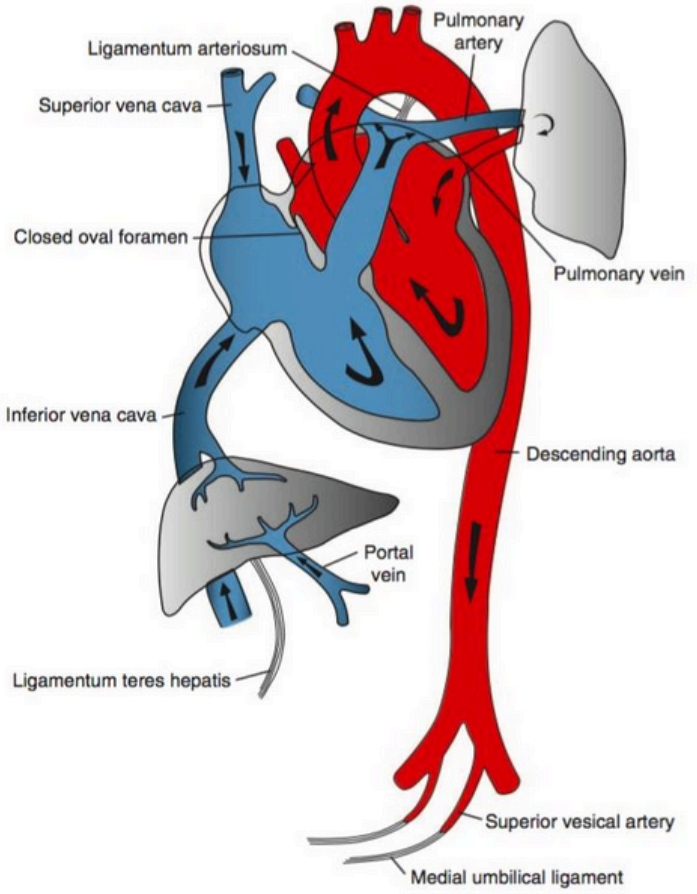

fetal capillary
intervillous space
maternal vein
syncytiotrophoblast
maternal spiral artery
umbilical vein
umbilical cord
umbilical arteries
chorionic plate
basal plate

# Special Structures

- **Foramenovale:** Connectionbetweentherightand left atria.
- **Ductus arteriosus:** Connection between the truncus pulmonalis and the aorta
- **Ductus venosus** shunts most of the left umbilical vein blood flow directly to the inferior vena cava. Thus, it allows oxygenatedbloodfrom theplacentatobypasstheliver.
- Thehypogastricarteries enterthe umbilical cord and are then known as the **umbilical arteries**.

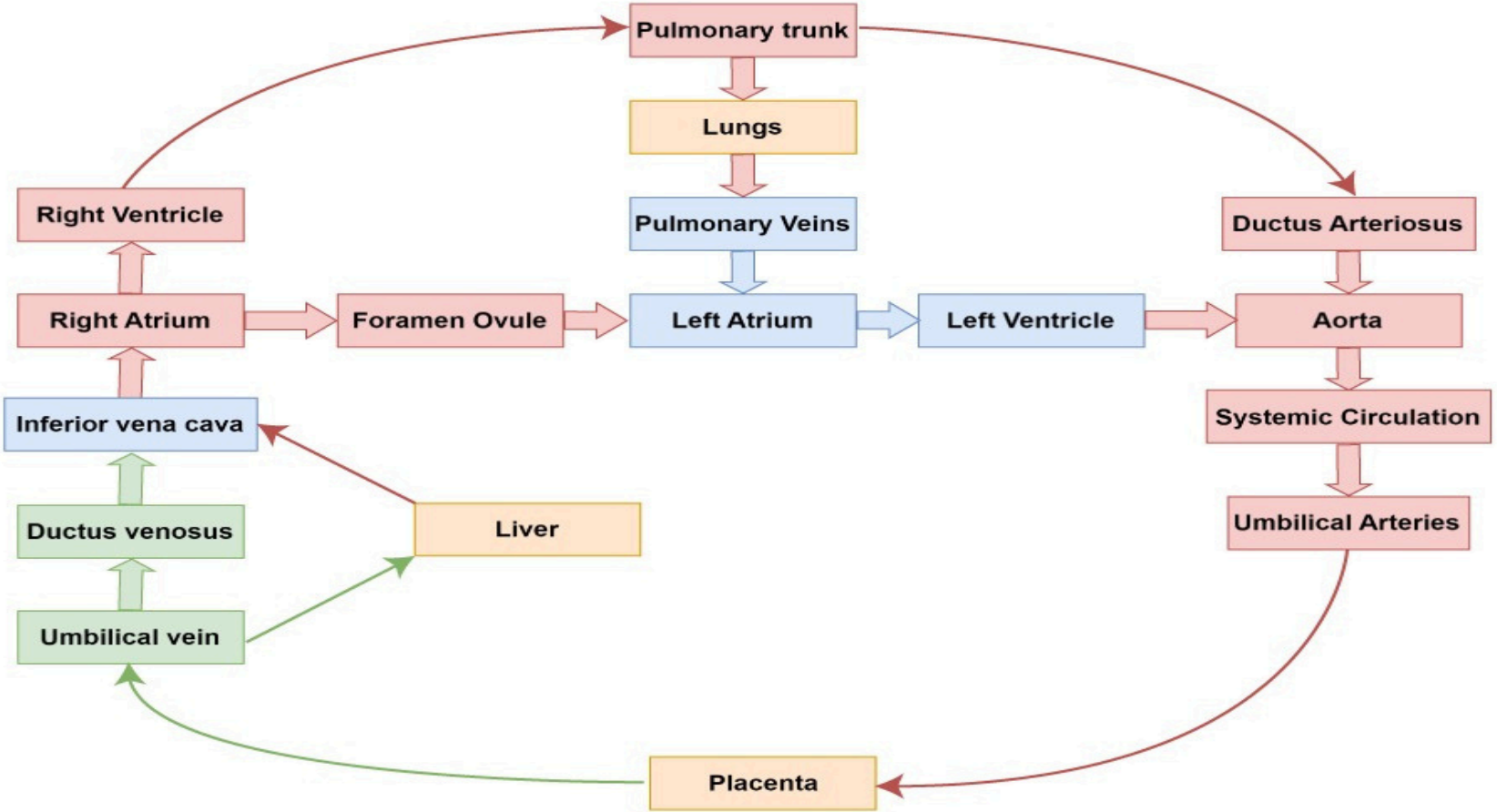
# Fetal circulation




Post Transition Circulation

# Fetal Circulation



# Circuit

- From the **placenta**, oxygenated blood flows through the **umbilical vein** and splits upon entering the abdomen of the fetus.
- The majority flows through the **ductus venosus into the inferior vena cava**, and then **the right atrium**; the remaining blood perfuses the liver.
- Blood originating from the ductus venosus enters the right atrium and, because of a streaming effect, is largely shunted through the **foramen ovale**into the **left side of the heart** and **aorta.**

- In contrast, less oxygenated blood from the superior vena cava and the inferiorvena cava distaltothe ductusvenous flows from the right atrium into the right ventricle with minimal mixing with
- the oxygenated blood originating from theductus venosus
  .
  Almost all the **right ventricular output** (90 percent) bypasses the lung and is **shunted through the patent ductus arteriosus**
- **to the descending aorta** distal to the origin of the carotid arteries.
  This **deoxygenated blood**is transported through the **aorta** and the **umbilical arteries** to the **placenta,**where it releases carbon dioxide and waste products and collects oxygen and

# Differential blood flow

- In thefetus,thebloodflowisstructuredsothatvitalorgans (eg, liver,heart, andbrain)receivebloodwitharelativelyhigh degree of oxygensaturation .

**1.LIVER**: receives blood directly from the umbilical vein without mixing with deoxygenated fetal blood.

**2.HEART AND BRAIN:–**Blood flowing through the coronary and carotid arteries has a high degree of oxygen saturation because oxygenated blood from the umbilical vein flows to the right atrium (via the ductus venosus and inferior vena cava) and is shunted through the foramen ovaleto the left side of the heart and aorta The deoxygenated blood is directed toward the right ventricle and shunted throughthe ductus arteriosus to the aorta, but distal to the origin of the carotid and coronary arteries

# TRANSITION AT DELIVERY

To successfully makethetransitionfrom intrauterine to extrauterinelife when the umbilical cord is clampedat birth,the neonatemust rapidlymakephysiologic changes incardiopulmonary function. **A successful transition is characterized by the following feature:**

**1.Alveolar fluid clearance**—Several mechanisms contribute to the clearance of alveolar fluid and lung aeration, including labor, initial breaths, and thoracic squeeze

**2.Circulatory changes** —With the clamping of the umbilical cord, the placenta with its low vascular resistance is removed from the neonatal circulation, resulting in a rise in neonatal systemic blood pressure.

**3.Lung expansion** —With the first effective breath, air movement begins as intrathoracic pressure falls, starting at pressures of less than -5 cm H O. Increasing inspiratory pressure expands the alveolar air spaces and establishes functional residual capacity (FRC).

Lung expansion also stimulates surfactant release, which reduces alveolar surface tension, increases compliance, and stabilizes the FRC

# Changes in the Fetal Circulation after birth

**Foramen ovale→Fossa ovalis**

**Ductus arteriosus→Ligamentum arteriosum**

**Ductus venosus →Ligamentum venosum**

**Umbilical arteries →Umbilical ligament**

**Umbilical vein →Ligamentum tere**

# DIFFICULTIES IN TRANSITION

- Althoughmost neonates successfully transitionbetweenintrauterine and extrauterinelife, about10 percentwill havesomedifficultyandrequire resuscitativeefforts atbirth

**Neonatal difficulties at birth include the following**:

1-Lack of respiratory effort

2-Blockage of the airways

3-Impaired lung function

4-Persistentincreasedpulmonaryvascularresistance (also referred to as persistent pulmonary hypertension or persistent fetal circulation)

# Risk factors

Thefollowingrisk factorsare associated with a greater likelihood of having difficultymaking a successful transition and of requiring resuscitation:

**1.Maternal conditions** –Advanced maternal age, maternal diabetes mellitus or hypertension, maternal substance use disorder, or previous history of stillbirth, fetal loss, or early neonatal death

**2.Neonatal conditions** –Prematurity, postmaturity, congenital anomalies, or multiple gestation

**3.Antepartum complications** –Placental anomalies (eg, placenta previa), or either oligohydramnios or polyhydramnios

**4.Delivery complications** –Transverse lie or breech presentation, chorioamnionitis, foulsmellingormeconium-stained amnioticfluid.

# Fetal vs. Infant Circulation

- Fetal
- Low pressure system
- Right to left shunting
- Lungs non-functional
- Increased pulmonary resistance
- Decreased systemic resistance

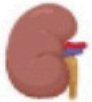
- Infant
- High pressure system
- Left to right blood flow
- Lungs functional
- Decreased pulmonary resistance
- Increased systemic resistance

## Gastrointestinal

↓ bowel motility -> constipation
↓ esophageal sphincter tone

## Renal

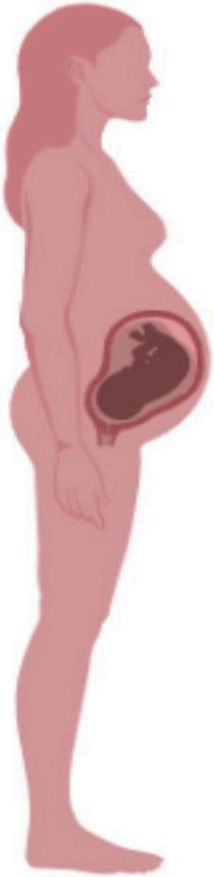
↑ renal plasma flow
↑ glomerular filtration rate
↑ urinary tract infections



↓ serum creatine
↓ urea nitrogen in the blood

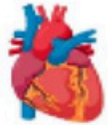
## Endocrine

↑ the level of estrogen and progesterone
↑ T3 and T4
↑ cortisol
↑ prolactin



## Cardiac

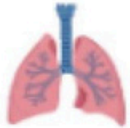
↑ heart rate
↑ cardiac output
↑ stroke volume



## Pulmonary

↑ ventilation per minute (VM)
↑ $PaO_2$
↑ $PAO_2$

↓ $PaCO_2$
↓ $PACO_2$



## Musculoskeletal

↑ lumbar lordosis
↑ joint laxity

## Hematological

↑ erythrocyte volume
↑ leukocytes
↑ risk of thromboembolism